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STATUS OF EFFORTS TO IDENTIFY GULF WAR SYNDROME: MULTIPLE TOXIC EXPOSURES

HEARING

BEFORE THE

SUBCOMMITTEE ON HUMAN RESOURCES

OF THE

COMMITTEE ON GOVERNMENT REFORM AND OVERSIGHT HOUSE OF REPRESENTATIVES

ONE HUNDRED FIFTH CONGRESS

FIRST SESSION

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STATUS OF EFFORTS TO IDENTIFY GULF WAR SYNDROME: MULTIPLE TOXIC EXPOSURES

THURSDAY, JUNE 26, 1997

HOUSE OF REPRESENTATIVES,
SUBCOMMITTEE ON HUMAN RESOURCES,
COMMITTEE ON GOVERNMENT REFORM AND OVERSIGHT,
Washington, DC.

The subcommittee met, pursuant to notice, at 10:15 a.m., in room 2154, Rayburn House Office Building, Hon. Christopher Shays (chairman of the subcommittee) presiding.

Present: Representatives Shays, Pappas, Towns, Sanders, and Kucinich.

Staff present: Lawrence J. Halloran, staff director and counsel; Robert Newman, professional staff member; R. Jared Carpenter, clerk; Cherri Branson, minority counsel; and Ellen Rayner, minority chief clerk.

Mr. SHAYS. I welcome our witnesses and our guests, and we will begin this hearing.

In the course of these oversight hearings on Gulf war veterans' illnesses, we have delved deeply into complex scientific, clinical, military, and administrative issues. We are likely to do so again today as the subcommittee examines the possible synergistic effects of exposure to toxic cocktails, including low-level chemical weapons, pesticides, smoke from oil well fires, experimental drugs, depleted uranium, and biological agents.

Immersed in a sea of technical details, it is possible to lose sight of the larger question that still confronts us as a Nation 6 years after the war: Are sick veterans getting better?

Fortunately, testimony before this subcommittee from the General Accounting Office, GAO, Tuesday cut through the complexity and reasserted that simple, yet profound, important question as the moral, medical, and operational test of everything this Government does in the name of those it serves.

As directed by Congress last year, GAO evaluated the effectiveness of the clinical care and research programs for six Gulf war veterans. They found neither the Veterans' Affairs Department, VA, nor the Defense Department, DOD, can say whether the veterans on their health registries since 1992 are any better or worse today than when they were first examined. GAO also found the research effort reactive, predisposed to certain lines of inquiry, and highly unlikely to provide conclusive answers regarding the causes of Gulf war illnesses, and they found some official conclusions

about Gulf war illnesses by the Presidential Advisory Committee, the PAC, weakly supported or premature.

In short, 6 years after the war, when asked what progress has been made healing sick Gulf war veterans, VA and DOD cannot say where they have been and may never get where they are supposed to be going.

Part of the journey from cause to cure runs through the pools, clouds, and plumes of toxins in which Gulf war veterans lived and fought. It is a leg of the trip DOD and VA have never taken, too quickly dismissing the potential health hazards of many known exposures. Just as research into the effects of low-level chemical weapons was thwarted for 5 years by denials, inquiries into toxic effects of other agents, alone and in combination, have been dismissed or ignored.

It is simply not acceptable for VA and DOD to declare repeatedly "there is no evidence" of exposures or effects, when the evidence has never been sought.

Today, we will hear evidence of two ingredients of the toxic soup to which many Gulf war veterans were exposed: depleted uranium and mycoplasmas. No one claims either agent is the silver bullet causing the myriad of Gulf war illnesses, nor should anyone in the face of very real symptoms and very real suffering likely dismiss their potential for causing, enhancing, or accelerating the health effects of toxic exposures.

Depleted uranium is a heavy metal, like lead, which is highly toxic when ingested or inhaled. Mycoplasma infections may explain apparent transmission of illnesses to veterans' family members.

We asked VA and DOD witnesses to describe what is known about the extent and effects of exposures to these agents and how that knowledge is reflected in research, diagnosis, and treatment protocols. We also invited researchers familiar with the pathology and these agents to describe their work. The subcommittee appreciates the benefit of their views and their expertise.

The Gulf war veterans testifying today, like those who appeared here before, still travel the uncertain road they hope will lead to answers, good health, to the home they left to fight our desert battle. We are honored by their presence and we value their testimony.

Are sick Gulf war veterans getting better? Until the answer is yes, our work as a Congress and as a Nation remains unfinished, our debt to veterans unpaid.

At this time, the Chair would like to recognize a partner in this effort, Mr. Sanders from Vermont.

[The prepared statement of Hon. Christopher Shays follows:]

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Statement of Rep. Christopher Shays
June 26, 1997

In the course of these oversight hearings on Gulf War veterans' illnesses, we have delved deeply into complex scientific, clinical, military and administrative issues. We are likely to do so again today, as the Subcommittee examines the possible synergistic effects of exposure to "toxic cocktails" including low-level chemical weapons, pesticides, smoke from oil well fires, experimental drugs, depleted uranium, and biological agents.

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In short, six years after the war, when asked what progress has being made healing sick Gulf War veterans, VA and DoD can't say where they've been, and may never get where they're supposed to be going.

Part of the journey from cause to cure runs through the pools, clouds, and plumes of toxins in which Gulf War veterans lived and fought. It is a leg of the trip DoD and VA have never taken, too quickly dismissing the potential health hazards of many known exposures. Just as research into the effects of low-level chemical weapons was thwarted for five years by denials, inquiries into the toxic effects of other agents, alone and in combination, have been dismissed or ignored.

It is simply not acceptable for VA and DoD to declare repeatedly "there is no evidence" of exposures or effects, when the evidence has never been sought.

Today, we will hear evidence on two ingredients of the toxic soup to which many Gulf War veterans were exposed: depleted uranium and mycoplasmas. No one claims either agent is the "silver bullet" causing the myriad of Gulf War illnesses. Nor should anyone, in the face of very real symptoms and very real suffering, blithely dismiss their potential for causing, enhancing or accelerating the health effects of toxic exposures.

Depleted uranium is a heavy metal, like lead which is highly toxic when ingested or inhaled. Mycoplasmal infections may explain apparent transmission of illnesses to veterans' family members.

We asked VA and DoD witnesses to describe what is known about the extent and effects of exposures to these agents, and how that knowledge is reflected in research, diagnosis and treatment protocols. We also invited researchers familiar with the pathology and these agents to describe their work. The Subcommittee appreciates the benefit of their views and their expertise.

The Gulf War veterans testifying today, like those who appeared here before, still travel the uncertain road they hope will lead to answers, to good health, to the home they left to fight our desert battle. We are honored by their presence, and we value their testimony.

Are sick Gulf War veterans getting better?

Until the answer is yes, our work as a Congress and as a nation remains unfinished, our debt to veterans unpaid.

Mr. SANDERS. Thank you very much, Mr. Chairman, and I continue to applaud you and your staff for the outstanding work that you have done for a very long period in keeping this issue before the public eye and in trying to bring forth truths which, in fact, have been hidden for a number of years.

Mr. Chairman, within the last week or two, I think two important developments have occurred, which I want to very briefly mention. No. 1 was the release of a GAO report which basically concluded what many of us have been saying for a number of years, and that is that neither the Pentagon nor the Veterans' Administration have been doing a good job in helping us understand the cause of the problems or developing a treatment for the some 70,000 veterans who are hurting today. And that report, of course, did not come as a surprise to the members of this committee, because that is exactly the report that we have been making for a number of years.

Second, I submitted for the record a letter that was sent to the chairperson of the Presidential Advisory Committee that had the names of 86 members of the U.S. House of Representatives, and basically what that letter said to the Presidential Advisory Committee is that we, Members of Congress, disagree with the conclusion of your December 1996 report which suggests that stress and stress alone is the cause of Persian Gulf illnesses.

And I must tell you that we could have had many more signatures on that letter. I must tell you that it was not a partisan issue. Democrats, Republicans, conservatives, progressives all responded, because very few people today in the House of Representatives and, I expect, in the Senate as well and, I expect, within the veterans' community and, I expect, within the United States of America today accept the conclusion that only stress was the cause of the problems.

Is stress an important factor? Yes, it is. I happen to believe it is. But is it the only factor? No. And I think what we have been hearing, month after month after month, testimony before this committee is the role that chemicals, in one form or another, and the synergistic, the combined effect of chemicals, the role that they have played in causing illness, and it is impossible, in my view, to deny that conclusion any more.

Mr. Chairman, very briefly, the concern that I have and what the GAO had is the lack of focus and the lack of direction on the part of the DOD and the VA. In the letter that we sent to the Presidential Advisory Committee, we briefly summarized a dozen different studies by outstanding and well-known scientists and physicians who, in one way or another, point out the role that chemicals have played.

Interestingly, two of the studies were funded by the DOD itself. In 1995, the DOD, in one of their own studies at Fort Detrick, MD, concluded that pyridostigmine bromide, combined with DEET and pyrimethamine, have a synergistic effect, much more so than the additive effect on making rats sick, dying earlier than one would have expected, similar to the findings released by a Duke University study. A dozen different studies, and what the GAO is saying, where is it all going? In 5 years from now, in 10 years from now,

are we going to have more and more studies? So I would suggest this is not an academic exercise.

Now, the problems are many.

No. 1, I happen to believe, and I can understand it from a human nature point of view, that the DOD is not happy to acknowledge that after that smashing military victory in the Persian Gulf, a victory of enormous consequence, much better than anyone dreamed possible, that a two-bit despot like Saddam Hussein may have been able to cause yet so much damage. People do not want to acknowledge that.

No. 2: What about the role of pyridostigmine bromide? As we all know, the DOD received a waiver from the FDA, and I suspect that there is—and I am not here to criticize, in that sense, the DOD. We know that they want the best for our troops. We know the VA wants the best for our troops, but maybe there is a reluctance to investigate the fact that they themselves brought forth pyridostigmine bromide, administered it to hundreds of thousands of our troops, and maybe that is part of the problem.

And, No. 3, and maybe most significantly, there is a strong difference of opinion within the medical community; honest physicians, honest scientists disagree about what is called "multiple chemical sensitivity," and you have many physicians, I think, in the VA and the DOD who simply do not accept that diagnosis.

I will be curious to know from the DOD and the VA how many scientists they have on board who believe in the synergistic impact of chemicals, that chemicals can make us ill. And if you do not believe that, then you can have all the scientists you want peer-reviewing everything, and they are going to think, hey, this is quackery; this does not mean anything.

So I think those are some of the questions that we will want to explore today, and, Mr. Chairman, I simply congratulate you and your staff for the outstanding work that you have been doing.

[The letter referred to follows:]

Congress of the United States
Washington, DC 20515

June 20, 1997

Joyce C. Lashof
Committee Chair
Presidential Advisory Committee on
Gulf War Veterans' Illnesses

Dear Dr. Lashof:

In the December, 1996 Final Report of the Presidential Advisory Committee on Gulf War Illnesses, the Committee concluded that "current scientific evidence does not support a causal link between Gulf veterans' illnesses and exposures while in the Gulf region to the following environmental risk factors assessed by the Committee: pesticides, chemical and biological warfare agents, vaccines, pyridostigmine bromide, infectious diseases, depleted uranium, oil well fires and smoke, and petroleum products."

The Committee found rather that; "Stress manifests in diverse ways, and is likely to be an important contributing factor to the broad range of physical and psychological illnesses currently being reported by Gulf War veterans." Consequently, the Committee recommended that; "The entire federal research portfolio should place greater emphasis on basic and applied research on the physiologic effects of stress and stress-related disorders."

While in no way minimizing the role that stress may have played in causing or contributing to health problems experienced by some veterans, we are writing to ask you to reassess your conclusion that current scientific evidence does not support a causal link between the symptoms and illnesses reported by Gulf war veterans and their exposure to a variety of chemicals during their service in the Persian Gulf War. In fact, it is our belief that more and more scientific evidence suggests that a major cause of Persian Gulf illness is the synergistic effect of a wide variety of chemicals to which our soldiers were exposed. Our hope is that by reassessing your conclusion, you will recommend increased research into and treatment for the health effects of chemical exposures experienced in the Persian Gulf.

As you know, the Persian Gulf War theater was a chemical cesspool. It is now acknowledged that our troops were exposed to chemical warfare agents. There is debate and uncertainty as to the extent of that exposure but the Department of Defense confirms that at least 20,000 soldiers were exposed. Further, the Persian Gulf environment included widespread use of leaded petroleum for fuel and dust mitigation. There was also considerable use of pesticides, including pesticides which were sprayed on the uniforms of individual troops and on their skin.

Additionally, Persian Gulf troops were vaccinated against common infectious diseases, as

well as against two agents of biological warfare, anthrax and botulism toxin. Perhaps most importantly, as a result of a waiver from the FDA, the Department of Defense administered to Persian Gulf soldiers the investigational drug, pyridostigmine bromide, as an anti-nerve gas measure.

As you know, over the last several years there have been a number of scientific studies and research reviews which suggest that chemical exposures may have played a key role in the illnesses which tens of thousands of our Gulf veterans are suffering from. A brief description of a few of these studies follows:

Robert W. Haley, M.D., of the University of Texas Southwestern Medical Center published in January of 1997, "Scientific Findings on the Gulf War Syndrome and Action Plans Leading to Treatment for Veterans." This research project concluded that many veterans are suffering from three primary syndromes, due to subtle brain, spinal cord and nerve damage, but not due to stress. He concludes that the damage was caused by exposure to combinations of pyridostigmine bromide, DEET and pesticides. Different combinations of the chemicals appear to have caused the three different syndromes.

Mohamed Abou-Donia, a Duke Pharmacologist, and Tom Kurt, of The University of Texas Southwestern Medical Center in Dallas, published a study in the May, 1996 issue of Journal of Toxicology and Environmental Health. This study, conducted on hens, concluded that pyridostigmine bromide, in combination of DEET and permethrin caused neurological deficits in the test animals which are similar to those reported by Gulf War veterans.

Interestingly, in May of 1995, the DOD published its own study which concludes, "there is a significant increase in the lethal effect in rats given pyridostigmine bromide, permethrin and DEET simultaneously by gavage when compared to expected additive lethal effect of the individual compounds." This study, which received relatively little public notice when it was released, was recently published in the Journal of Toxicology and Environmental Health.

More recently, Dr. Abou-Donia conducted another research project with the VA Medical Center, Durham North Carolina. This research showed that when rats were given pyridostigmine bromide and then put in stressful conditions, pyridostigmine bromide was able to cross the blood-brain barrier, leading to suppressed AChE levels. The research forecasts that similar blood-brain barrier alterations in veterans may have contributed to neurological deficits of some Gulf War veterans who were exposed to these chemicals during the war.

Another study, conducted by Friedman, Kaufer, Shemer and others at the Department of Biological Chemistry, Life Sciences Institute, Hebrew University in Israel, presents evidence that stress may make the blood brain barrier permeable to PB. The Veterans Affairs, April, 1997 Report to Congress states that this study may explain the acute symptoms of individuals who took PB. This study was published in Nature Medicine in 1996.

Dr. Garth Nicolson of the University of Texas, Department of Tumor Biology, and Dr. Nancy Nicolson of the Rhodon Foundation for Biomedical Research have conducted research which indicates that many of the symptoms of Gulf War Syndrome may be caused by chronic pathogenic mycoplasma infections. The Nicolsons relate these infections to exposures to warfare

agents in the Gulf.

Dr. Satu Somani, PhD. of Southern Illinois University, School of Medicine concludes, in a statement before the House Subcommittee on Human Resources that in light of "experimental proof and historical evidence of symptoms such as impaired concentration and memory, headache, fatigue and depression of the workers who worked in organophosphate industry, I consider that the illness associated with Gulf War veterans may be due to low dose sarin exposure and intake of pyridostigmine and exposure to pesticides and other chemicals. The adverse effects of these were amplified by physical stress."

Dr. Myra B. Shayevitz, of the Northhampton VAMC, testified before the House Subcommittee on Human Resources that; "Experience at Northampton VAMC has led us to believe that the unexplained health problems of some Persian Gulf veterans may relate to the combination of chemical, physical and psychological stressors unique to the Desert Storm operation." In summary, Dr. Shayevitz testified that veterans seen at the VAMC facility complained of multi-system symptomology which is remarkably similar to the syndrome which has been labeled Multiple Chemical Sensitivity. Multiple Chemical Sensitivity is a disorder in which multiple symptoms occur in multiple systems or organs of the body as a result of exposure to chemicals.

Dr. Claudia Miller, assistant professor in allergy/immunology and environmental medicine at the University of Texas Health Science Center-San Antonio, consultant to the VA on the Gulf veterans' health problems, and a member of the VA's Persian Gulf Expert Scientific Advisory Committee, described the similarities between the Gulf veteran's symptoms and those of some civilians exposed to organophosphate pesticides, carbamate pesticides, or low levels of volatile organic chemical mixtures in a 1995 paper published in Archives of Environmental Health entitled, "Chemical Sensitivity Attributed to Pesticide Exposure Versus Remodeling." In testimony invited by your Committee, in several recently published papers, and in the second edition of the book Chemical Exposures: Low Levels and High Stakes, (co-authored by MIT Professor Nicholas A. Ashford, Ph.D., J.D.), she has presented compelling evidence that we may in fact be witnessing the emergence of a new mechanism or theory of disease, described as "toxicant-induced loss of tolerance."

Dr. Howard B. Urnovitz, PhD, has focused his research on how chemical and infectious agents interact to initiate and maintain a chronic disorder. He testified before the House Human Resources Committee that he became involved with Gulf War illnesses because the symptoms were similar to those of a dozen unexplained epidemics over the last 60 year. From his research survey, Dr. Urnovitz concluded, "Syndromes associated with organophosphate-induced delayed neuropathy could explain many of the observed and unexplained illnesses."

Dr. James I. Moss and Dr. Arthur Hume recently conducted research which focused on the possible interactions that might produce symptoms similar to those experienced by Persian Gulf veterans. Preliminary research on mice indicates that toxicity of pyridostigmine bromide increases when combined with caffeine or adrenaline.

In research on cockroaches conducted in 1993, Dr. Moss, when working for the Department of Agriculture, came to the conclusion that PB and DEET, when combined with each

other, were much more toxic than when used separately.

Dr. Frank H. Duffy, MD of the Department of Neurology Children's Hospital and Harvard Medical School, presented the following testimony to the House Human Resources Subcommittee: "Studies performed or funded by the US Army in the past clearly demonstrate, for both monkey and man, that exposure to the nerve agent, Sarin, can produce long term alteration of brain function. Levels of exposure capable of producing such late effects may not be recognizable by subjects, acutely, especially if they are unaware of what is happening and/or are distracted by other activities."

A study published in the Journal of Neurology, conducted by Jamal, Hansen, Aparcopoulos and Pedan focused upon evidence of peripheral and central nervous system dysfunction in veterans with Persian Gulf War illness that may have been caused by chemical exposure. The study concluded that there may have been a dysfunction in the nervous system of the veterans which were assessed, and that further studies were required to confirm and characterize this dysfunction.

Dr. William Rea of the Environmental Health Center in Dallas has treated over 60 Persian Gulf veterans, with a protocol that includes chemical-free environment, nutritional supplements, injection therapy and heat therapy. Dr. Rea concludes that neurotoxic environmental exposures and other personal exposures prior to and during deployment in the Gulf War theater of operations, including burning oil and smoke, pesticides, sand irritation, inoculations and nerve gas may have resulted in chronically deregulated immune and nonimmune detoxification systems, resulting in multi-system illness in veterans.

Dr. Mark A. Prendergast, of the Medical College of Georgia, and others, recently published a study in the journal Psychopharmacology, which suggests that exposures to low levels of nerve gas and some pesticides can lead to memory loss, a common complaint among Gulf War veterans.

The above references indicate that there is a wide array of scientific evidence available that leads to the conclusion that some Gulf War veterans are suffering illnesses related to chemical exposures in the Gulf. Moreover, many of the studies specifically link pyridostigmine bromide and pesticides with adverse health effects, similar to those our veterans are suffering from. While we agree that effects of stress must be considered and studied in order to better address the myriad of problems which Gulf War veterans face, we urge the Committee to now place your emphasis and focus on the role which chemical exposures played in the health problems of veterans who served in the Gulf War.

We would also like to express our concern that there is a feeling among the public that, for whatever reason, various agencies of the United States Government have been less than enthusiastic about addressing the issue of the relationship between chemicals and Persian Gulf illness.

As you know, it took over five years before the DOD and CIA publicly acknowledged that American troops were exposed to chemical warfare agents. The DOD, today, acknowledges that they do not yet know the full extent of the exposures.

A number of government researchers have either been fired or failed to receive support in investigating the possible relationship between chemicals and Persian Gulf illness:

Dr. James Moss, after concluding that PB and DEET when combined produce toxic effects on cockroaches, was terminated from his employment with the Department of Agriculture.

In 1993, in invited testimony before the Subcommittee on Oversight and Investigations of the Committee on Veterans Affairs, Dr. Claudia Miller called for a specialized research facility, an environmental medical unit, in order to test scientifically whether ill Gulf War veterans are sensitive to very low levels of common chemicals, as many of them now report. Although Congressional appropriations for half the costs of the facility were obtained through a bipartisan effort and DOD agreed to fund the remainder, DOD failed to implement the project. No such research facility currently exists that would allow physicians to diagnose or rule out chemical sensitivity in the veterans.

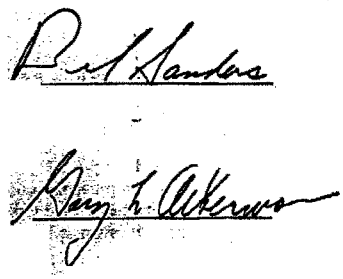
Dr. Myra Shayavitz, a VA physician, at Northampton Massachusetts VA hospital was given preliminary support by the VA for a treatment project based on the belief that Persian Gulf veterans were suffering from chemical exposures. Despite initial support, Dr. Shayavitz's research project was never funded and she eventually left the VA.

Jonathon Tucker, PhD, served on the Presidential Advisory Committee staff as the senior policy analyst responsible for investigating incidents of chemical and biological agents exposures from August to December, 1995. Dr. Tucker was summarily dismissed after aggressively attempting to understand the extent of chemical exposures in the Gulf.

In conclusion, we, the undersigned Members of Congress, urge the Presidential Advisory Committee to reevaluate the conclusions that were reached in the Final 1996 Final Report. We believe that the evidence is clear that exposure to a wide variety of chemicals in the Persian Gulf may be a significant factor in Persian Gulf illness.

We look forward to hearing your reply. Thank you for your consideration.

Sincerely Yours,



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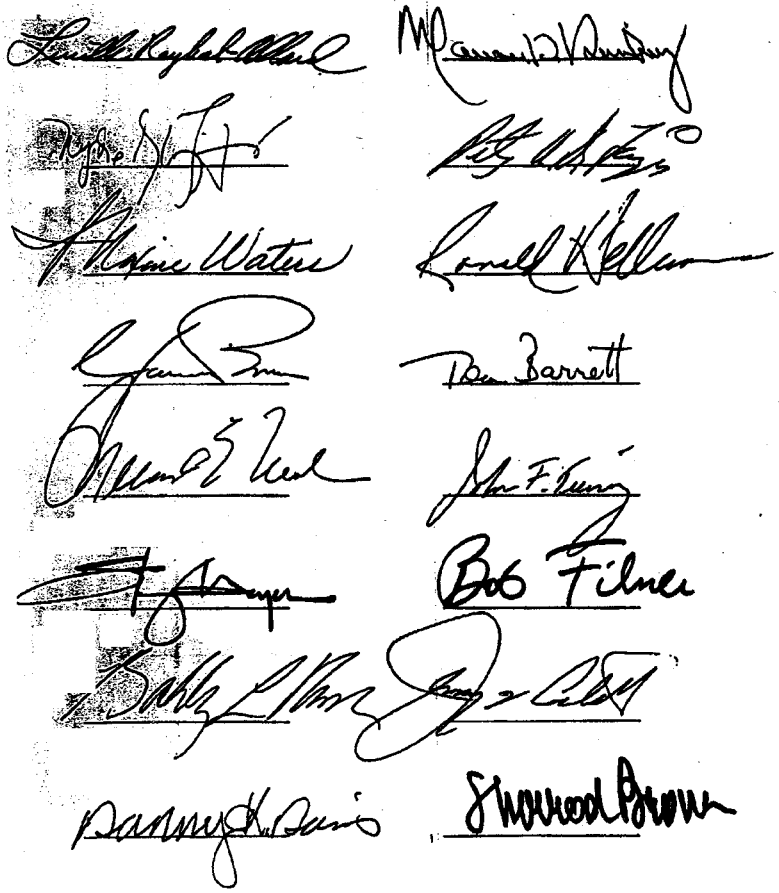


Dennis J. Kucinich

Patsy T. Mink

Gerald Kleczka

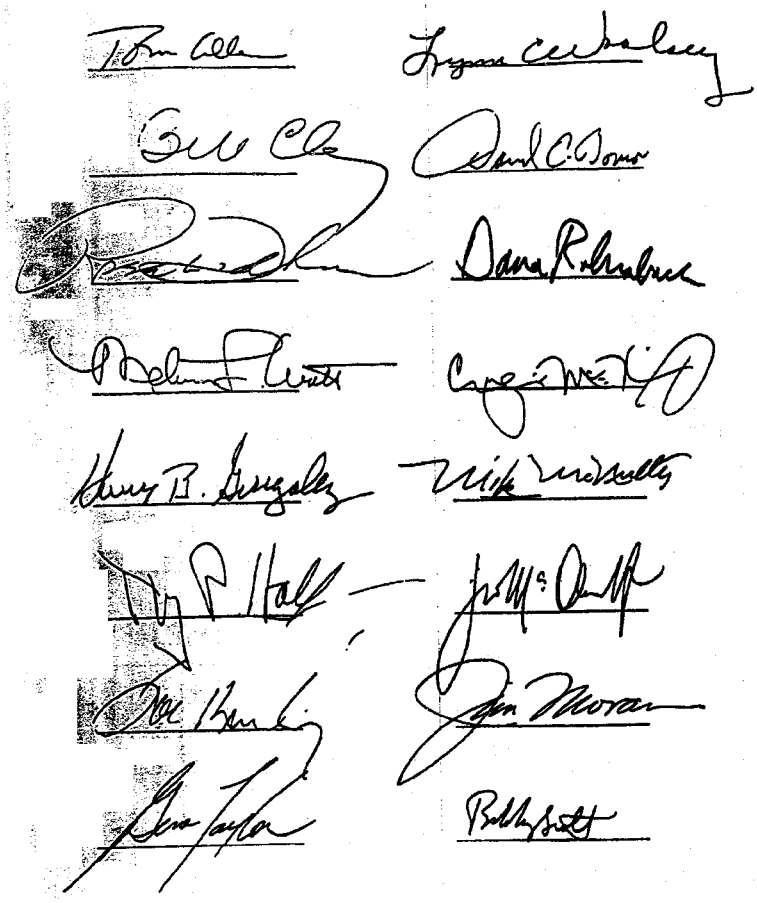
Mike Sabo

John Olver

Corrine Brown

Eliot L. Engel

Jack Metcalf



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Bill Paxiell

Tracey Kepter

Carolyn B. Maloney

Ellen Tauscher

Elizabeth Dole

Bob Wise

Peter King

Julia Carson

Lois M. Clayton

John E. Baldacci

Stephen Horn

Neil Abercrombie

Sander Levin	M. G. Martinez
Wayne T. Gilchrest	Darrell Issa
Maxine Waters	Jim Ramstad
Jim Leach	Christopher Shays
Bart Stupak	
Diana DeGette	

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Gregory Meeks

Gene Green

Jim Barcia

Barney Frank

Earl Blumenauer

Bud Cramer

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Congress of the United States
House of Representatives
Washington, DC 20515–1501

COMMITTEE ON BANKING AND FINANCIAL SERVICES
SUBCOMMITTEES:
HOUSING AND COMMUNITY OPPORTUNITY
DOMESTIC AND INTERNATIONAL MONETARY POLICY

COMMITTEE ON GOVERNMENT REFORM AND OVERSIGHT
SUBCOMMITTEES:
RANKING MINORITY MEMBER:
NATIONAL ECONOMIC GROWTH, NATURAL RESOURCES, AND REGULATORY AFFAIRS
HUMAN RESOURCES AND INTERGOVERNMENTAL RELATIONS

CHAIR: PROGRESSIVE CAUCUS

Members of Congress who have signed onto Sanders' June 20, 1997 letter to the Presidential Advisory Committee on Gulf War Illnesses, urging them to reassess their findings and recommendations

Bernie Sanders (VT)
Gary Ackerman (NY)
Lucille Roybal-Allard (CA)
Nydia Velazquez (NY)
Maxine Waters (CA)
Xavier Becerra (CA)
Richard Neal (MA)
Steny Hoyer (MD)
Bobby Rush (IL)
Danny Davis (IL)
Dennis Kucinich (OH)
Patsy Mink (HI)
Jerrold Nadler (NY)
Martin Olav Sabo (MN)
John Olver (MA)
Corrine Brown (FL)
Eliot Engel (NY)
Jack Metcalf (WA)
Thomas Allen (ME)
William Clay (MO)
Rosa DeLauro (CT)
Melvin Watt (NC)
Henry Gonzalez (TX)
Tony Hall (OH)
Joseph Kennedy (MA)
Gene Taylor (MS)
Bill Pascrell (NJ)
Carolyn Maloney (NY)
Ellen Tauscher (CA)
Robert Wise (WV)
Juanita Millender-McDonald (CA)
Julie Carson (IN)
James Traficant (OH)
Stephen Horn (CA)
Sander Levin (MI)
Wayne T Gilchrest (MD)
Jon Fox (PA)
Bart Stupak (MI)
Ileana Ros-Lehtinen (FL)
Susan Molinari (NY)
Jim Barcia (MI)
Barney Frank (MA)
Spencer Bachus (AL)
Luis Guitierrez (IL)
Jesse Jackson Jr. (IL)
Maurice Hinchey (NY)
Peter DeFazio (OR)
Ronald Dellums (CA)
Thomas Barrett (WI)
John Tierney (MA)
Bob Filner (CA)
Jerry Costello (IL)
Sherrod Brown (OH)
James McGovern (MA)
John Joseph Moakley (MA)
Major Owens (NY)
William Coyne (PA)
Nancy Pelosi (CA)
Chaka Fattah (PA)
Jose Serrano (NY)
Calvin Dooley (CA)
Lynn Woolsey (CA)
David Bonior (MI)
Dana Rohrabacher (CA)
Cynthia McKinney (GA)
Michael McNulty (NY)
Jim McDermott (WA)
James Moran (VA)
Robert Scott (VA)
Marcy Kaptur (OH)
Anna Eshoo (CA)
Elizabeth Furse (OR)
Paul McHale (PA)
William Jefferson (LA)
Eva Clayton (NC)
John Baldacci (ME)
Neil Abercrombie (HI)
Matthew G. Martinez (CA)
David Drier (CA)
Christopher Shays (CT)
Debbie Stabenow (MI)
Earl Blumenauer (OR)
Gene Green (TX)
Nick Rahall (WV
Zoe Lofgren (CA)
Robert Cramer (AL)

R-98%

Mr. SHAYS. I thank the gentleman. At this time, we have a Member of Congress, a distinguished Member of Congress, Jack Metcalf, who, while not a member of this committee, has been very active on this issue and very involved. We appreciate your involvement, and appreciate any testimony or statement that you would like to give.

STATEMENT OF HON. JACK METCALF, A REPRESENTATIVE IN CONGRESS FROM THE STATE OF WASHINGTON

Mr. METCALF. Thank you very much, Mr. Chairman, for your work and support and for the opportunity to speak to the subcommittee on this vital issue. I would like to have my entire statement entered in the record.

Mr. SHAYS. Without objection, so ordered, and I will use your point here as an excuse to do two business things and ask unanimous consent that all members of the subcommittee be permitted to place any opening statement in the record and that the record remain open 3 days and without objection, so ordered, and ask unanimous consent that all witnesses be permitted to include their written statement in the record and without objection, so ordered.

And does the ranking member mind if I just—OK. We welcome your statement now.

Mr. METCALF. Thank you very much. Gulf war illnesses have affected thousands of service personnel, both United States troops and those of our allies. In the beginning, the Department of Defense officially refused to recognize the possibility of serious illnesses related to operations in the Gulf that were not clearly the result of an identifiable source. However, reluctantly, in the past year there has been an increasing acknowledgement of events during the operation that could have potentially exposed troops to chemical and biological warfare agents.

Considering United States shipments of both chemical and biological material to Iraq as well as statements by Retired General Schwarzkopf and Secretary of State Albright and others regarding Iraq's development of biological weapons, it is difficult to understand how the Department of Defense can continue to deny the possibility that our troops could have been exposed to biologicals.

Additionally, I have a grave concern that the Government's unwillingness to seriously consider the cumulative health consequences, cumulative health consequences of exposures to multiple-risk factors has resulted in inadequate care for the sailors, soldiers, airmen, and Marines who put their lives on the line when their Nation called.

The most sobering experience I have had since I came to Congress has been to meet the sick young men and women that were in excellent health before their service in the Gulf. I have heard over and over their stories of multiple-risk-factor exposures.

Ed, a Marine scout sniper, was in outstanding health before his service in the Gulf, as evidenced by the award he received for attaining the maximum score on physical fitness tests. His performance as a Marine was continually commended. His health has steadily deteriorated since his return. As he related his story, what is clear is the complexity of the potential exposures.

He was seriously ill shortly after arrival in the Gulf, although the cause was unknown. He was ordered to take PB tablets and a botulinum vaccine. During his experiences, chemical alarms were continually sounding and blister agents were being detected. He and his team were breathing smoke from oil well fires, as well as smoke from burning tanks destroyed by depleted uranium rounds. He described a dark, foul rain that came from the north, its cause unknown. He was exposed to pesticides and other environmental hazards in the field.

The work done to date to help Ed and thousands like him is woefully deficient. The Department of Defense is quick to point out that the Government is funding 91 Gulf war medical research studies. A close look, however, reveals a sobering reality: Of those 91, only 3 are looking at issues associated with chemical weapon exposure, and only 2 are examining the health consequences of depleted uranium. What is truly amazing is that none of these three chemical weapons studies are even being done in this country.

Why are not the best and the brightest of our doctors and scientists working to find answers? The young men and women who serve this Nation deserve better.

Finally, I want to thank Dr. Garth Nicolson and Leonard Dietz for their testimonies today. When scientists with the stature of these researchers speak, we need to be listening. I can only hope that the public will do so, that the Pentagon and the public will do so.

We in Congress have a moral obligation to press for truthful answers and to ensure adequate health for our veterans and their family members who may be sick as a result of exposures in the Gulf.

Thank you very much, Mr. Chairman.

[The prepared statement of Hon. Jack Metcalf follows:]

Statement made by Congressman Jack Metcalf to the Subcommittee on Human Resources 6-26-97

Mr. Chairman:
Thank you for the opportunity to speak to the Sub-Committee on this vital issue.
I would like to have my entire statement entered into the record.

Gulf War Illnesses have affected thousands of service personnel, both United States troops and those of our allies. In the beginning, the Department of Defense officially refused to recognized the possibility of serious illnesses related to the operations in the gulf that were not clearly the result of an identifiable source. However, reluctantly in the past year, there has been an increasing acknowledgment of events during the operation that could have potentially exposed troops to chemical and biological warfare agents. Considering U.S. shipments of chemical and biological materials, as well as statements by retired General Schwarzkopf, Secretary of State Albright and others regarding Iraq's development of biological weapons, it is difficult to understand how the Department of Defense can continue to deny the possibility that our troops could have been exposed to biologicals.

Additionally, I have a grave concern that the government's unwillingness to seriously consider the **cumulative health consequences** of exposures to multiple risk factors, has resulted in inadequate care for the sailors, soldiers, airmen, and marines who put their lives on the line when their nation called.

The most sobering experience I have had since I came to Congress, has been to meet sick young men and women that were in excellent health before their service in the gulf. I have heard over and over their stories of multiple risk factor exposures.

Ed, a former Marine scout sniper, was in outstanding health before his service in the Gulf, as evidenced by the award he received for attaining the maximum score on the Physical Fitness Test. His performance as a Marine was continually commended. His health has steadily deteriorated since his return. As he related his story, what is clear is the complexity of potential exposures:

1. He was seriously ill shortly after arrival, although the cause is unknown.
2. He was ordered to take PB Tablets and the Botulinum Vaccine.
3. During his experiences chemical alarms were continually sounding and blister agents were being detected.
4. He and his team were breathing smoke from oil well fires, as well as smoke from burning tanks destroyed by depleted uranium rounds.
5. He described a "Dark-foul rain" that came from the north: its cause unknown.
6. He was exposed to pesticides and other environmental hazards in the field.

The work done to date to help Ed and the thousands like him is woefully deficient. The Department of Defense is quick to point out that the Government is funding 91 Gulf War medical research studies. A close look however reveals a sobering reality: Of those 91, only three are looking at issues associated with chemical weapon exposure, and only two are examining the health consequences of depleted uranium. What is truly amazing is that none of the three chemical weapons studies are even being done in this country! [redacted]. Why are not the best and brightest of our doctors and scientists working to find the answers? The young men and women who served this nation deserve better!

Finally, I want to thank Doctor Garth Nicolson and Leonard Dietz for their testimonies today. When scientists with the stature of these researchers speak, we need to be listening. I can only hope that the Pentagon will do so.

We in Congress have a moral obligation to press for truthful answers, and to insure adequate health care for our veterans and their family members who may be sick as a result of exposures in the Gulf.

Thank you Mr. Chairman.

Mr. SHAYS. I thank the gentleman for being here for his statement.

At this time, the Chair would like to recognize Mr. Towns, who truly is an equal partner in this process. I may have the gavel, but I consider us equal partners, and I particularly appreciate the fact that he is busy on the Commerce Committee but spends so much time as the ranking member on this subcommittee. Mr. Towns.

Mr. TOWNS. Thank you very much, Mr. Chairman. Let me begin by first thanking you for your kind words, and let me also thank you, as well as the majority staff and the minority staff, for arranging this hearing today on Persian Gulf war illness.

While I look forward to hearing the testimony of all of our witnesses, I am particularly interested in our treatment of the disorder known as multiple chemical sensitivity. Some people have questioned the VA's reluctance to recognize multiple chemical sensitivity as a compensable injury. However, this criticism ignores that the medical community is divided over whether MCS is a bona fide disease. The California Medical Association, the American Academy of Allergy and Immunology, the American College of Physicians, the American College of Medicine, and the Council on Scientific Affairs of the American Medical Association have all published position papers which question the existence of MCS, its diagnosis, and its treatments.

Additionally, the legal community is not unified on this issue, either. Courts have been divided over whether MCS is to be considered as an injury under State workers' compensation laws, and we in the Federal Government have not been consistent, either.

MCS has been classified as a disability under the Americans with Disabilities Act. MCS has also been recognized by the U.S. Department of Housing and Urban Development as a basis for seeking protection under the Federal housing discrimination laws, yet the Social Security Administration considers MCS on a case-by-case basis, and the Department of Veterans' Affairs does not recognize it at all.

In August 1997, the U.S. Agency for Toxic Substances and Disease Registry will publish an interim report on MCS. The Agency is composed of representatives from the Departments of Defense, Energy, Health and Human Services, and Veterans' Affairs, as well as the National Center for Environmental Health, the National Institute of Occupational Safety and Health, and the National Institute of Environmental Health Sciences, and the U.S. Environmental Protection Agency.

The report is expected to contain findings and recommendations which may affect the compensation policies of every Federal agency and provide some general agreement in the scientific and medical communities, which would lead to Federal recognition and also uniformity.

Mr. Chairman, I suggest that when the report is released, we hold a hearing on its findings. Additionally, I suggest that if the situation warrants, we consider legislation to require Federal benefit uniformity for all those who are disabled by multiple chemical sensitivity.

So I look forward to working with you, as I have done in the past, and I would like to also applaud you for staying with this

issue, because I think it is important that we do so, and at this time I yield back.

Mr. SHAYS. I thank the gentleman. In fact, we both are staying with this issue, obviously, along with Mr. Sanders. At this time, I am inviting to the table, recognizing our four witnesses, Col. Gilbert Roman, retired, Gulf war veteran—oh, I am sorry. Mr. Kucinich, I apologize. I did not see you walk in.

Mr. KUCINICH. Thank you very much, Mr. Chairman. I will be brief. I want to thank the Chair for his diligence in pursuing this issue over the past few years, and I have had a chance to look at testimony that has been presented to this committee, as well as the initial report which we received, and it is very apparent that there were many shortcomings in the approach that the Department of Defense used.

I would like to think that the United States of America has a Defense Department which is second to none in the world and that they really are dedicated to protecting the American people and assuring the security of Americans around the world and making sure that Americans' interests are protected.

But in this one case I think we have seen where despite perhaps some of the best intentions and some of the best people, it is quite possible some serious mistakes were made and those mistakes were repeated, that people went into the crisis affecting the Gulf war veterans with a theoretical forward which did not allow for the consideration of other possibilities other than post-traumatic stress or psychological conditions which can arise from people being separated from family and being in a certain environment, and the analysis was flawed from the beginning.

And so if we can, in these hearings, find a way to not just admit that possibility, but to remedy the injustice which has been done to the Gulf war veterans, then we can celebrate the unending possibilities of a democratic tradition which can include error and seek to create remedies which can overcome those errors.

Thank you very much, Mr. Chairman, for the work that you have done on this.

Mr. SHAYS. I thank the gentleman. At this time, we will recognize our four witnesses and ask them to stand to be sworn in: Col. Gilbert Roman, retired, Gulf war veteran, Denver, CO; Mr. Paul Canterbury, Gulf war veteran, Ashley, OH; Mr. Michael Stacy, Gulf war veteran, Inola, OK; and S/Sgt. Mark Zeller, Gulf war veteran, Fort Rucker, AL.

Gentlemen, we swear in all our witnesses, including Members of Congress. Raise your right hands.

[Witnesses sworn.]

Mr. SHAYS. Thank you. Please be seated. I note for the record that all four have answered in the affirmative.

We will begin in the order in which I called you, so we will just go right down the table. We are going to have a timer on, but you are free to run over the timer. We want to just keep track of how we are doing here, so I welcome you, Colonel.

STATEMENTS OF COL. GILBERT ROMAN, RETIRED, GULF WAR VETERAN, DENVER, CO; PAUL CANTERBURY, GULF WAR VETERAN, ASHLEY, OH; MICHAEL STACY, GULF WAR VETERAN, INOLA, OK; STAFF SGT. MARK ZELLER, GULF WAR VETERAN, FORT RUCKER, AL

Col. ROMAN. Thank you, sir. Mr. Chairman, distinguished members of the subcommittee, my fellow veterans, I am Gilbert D. Roman, Colonel, U.S. Army, retired, Reserve. I thank you for the opportunity to be here today.

I would like to start out with a newspaper item quotation, a very brief one, taken from the Army Times, 1994. It says, "Sick Gulf Vets Wary, Wait for Treatment." It goes on to quote, "We are committed to the treatment of the veterans of the Persian Gulf conflict who are experiencing problems as a result of their service," said Edwin Dorn, Under Secretary of Defense for Personnel and Readiness. "We are determined to fashion compensation for those who are too sick to work." Army Times, March 1994.

We are still waiting, sir. I am greatly saddened by recent newspaper accounts of what is not occurring in the dialog and discussion on this issue, because I see a continuing pattern of official DOD misinformation and negligence tantamount to malfeasance in office for ignoring testimony and documentation referring to the use or presence of chemicals and other biological agents our reports indicate were found in the theater of operations during Desert Shield/Desert Storm.

I arrived in the theater of operations on January 6, 1991—by the way, that would happen to be my birthday—after volunteering to serve in the Persian Gulf and being brought on active duty in December 1990. My primary responsibility as Colonel, Medical Service Corps, was the Deputy Commander of the 311th Evacuation Hospital——

Mr. SHAYS. Colonel, could you just slow down a little bit? We are not going to rush you.

Col. ROMAN. Are you sure?

Mr. SHAYS. Yes.

Col. ROMAN. OK.

Mr. SHAYS. Let me just say something to all of you. We learned early on that you are voices in the wilderness, with very few people listening.

Col. ROMAN. Thank you. Thank you.

Mr. SHAYS. And we decided that in almost every instance we would begin our hearings listening to those voices. So you are a very important voice, and you take your time.

Col. ROMAN. I took very serious that 5 minutes, though, that we were given.

Mr. SHAYS. Well, I want to explain to you, we would like you to have been aware of the 5 minutes. If you run over, we are just going to turn the light back on.

Col. ROMAN. Thank you, sir. My primary responsibility as Colonel, Medical Service Corps was as the Deputy Commander of the 311th Evacuation Hospital. I was responsible for operations, logistics, and security. In secondary assignments I was also the public affairs officer and liaison to the Ministry of Health in Abu Dhabi, United Arab Emirates, where the 311th was physically located.

We were also near Al Dafra and Al Bateen Air Force Bases where the United States Air Force flew daily sorties north. Also flying out of Al Bateen were daily air shuttles called the "Star Shuttle," which were either C–130's or C–141's that flew daily shuttles to Riyadh, Dharhan, King Khalid Military City, and other points in the Gulf operations.

During several of the official visits to these strategic military cities there were frequent SCUD attacks in SCUD Alley during which I often heard the chemical alarms. When I asked if these alarms meant chemicals, and I was a colonel, I was told that the chemical alarms had malfunctioned. I do not think they malfunctioned that often, sir.

My first time in Riyadh, I became ill. I was treated for nausea, headaches, vomiting, diarrhea, and a high temperature. My commander, a physician, was with me and treated me for the symptoms, which appeared to be food poisoning. There was nausea, headache, vomiting, and—I am bleeding; and the reason I am bleeding, sir, is because I have precancerous polyps—excuse me—that have not been treated in my nasal passages and colon. But if I can continue, I would appreciate it.

Mr. SHAYS. You may continue, and you may slow down.

Col. ROMAN. I am slowing down.

Mr. SHAYS. And we can also go to another witness and then come back to you.

Col. ROMAN. If I can just continue, I will be finished in a few minutes.

Mr. SHAYS. I just want to emphasize to you, though, just feel free to slow down.

Col. ROMAN. OK.

Mr. SHAYS. We just want to hear every word you have to say.

Col. ROMAN. Thank you, sir. This nausea, headache, vomiting, and flu-like symptoms continued throughout the time I was in the Persian Gulf, and I continued to treat it like food poisoning, with Immodium and 800 milligrams Motrin, the Army's blessed answer to all pain.

The rashes I had over my body while I was in the Gulf I thought were normal and expected, since I spent most of my days in the sand, wind, and the sun with all the attendant fleas, flies, and other desert parasites. A calamine lotion-like substance served to sooth but not relieve or get rid of the severe rashes that I experienced.

Life in the theater of operations was a constant adrenalin rush, with 3 or 4 hours of sleep in between. Headaches I began to experience attributed to fatigue and the lack of sleep were actually other things, as I found out later.

Upon returning home to the States and my discharge from active duty, I returned home, and the symptoms I experienced in the Persian Gulf continued after I got there, and they got progressively worse.

In 1993, I registered myself with the Washington, DC Veterans Hospital after receiving an invitation from the VA to come in for an examination because I was a Persian Gulf vet. The Washington, DC VA noted——

Mr. TOWNS. Mr. Chairman, may I make a suggestion that we allow him to go to the restroom and then return and allow someone else to testify and then let him come back and continue?

Col. ROMAN. OK. Thank you.

Mr. SHAYS. I think that is a good suggestion.

Col. ROMAN. I apologize. I just have not been able to stop these nosebleeds for a number of years now.

Mr. SHAYS. You know, you are apologizing to us, and we should be apologizing to you. Thank you, Colonel. We will see you back here. Mr. Canterbury.

Mr. CANTERBURY. Yes, sir.

Mr. SHAYS. We welcome your testimony; and, again, I just want to say we are in no rush.

Mr. CANTERBURY. Yes, sir.

Mr. SHAYS. So we welcome your testimony. You may begin.

Mr. CANTERBURY. Thank you. Hello. My name is Paul Canterbury, and I want to thank you for allowing me to come and testify before you.

Mr. SHAYS. I am sorry to interrupt. I want you to move the mic a little closer to you, and I want you to bring it down just a speck. There you go. Thank you.

Mr. CANTERBURY. I served in the U.S. Army at Fort Hood, TX from 1989 to 1992 in Delta Company, 57th Signal Battalion. I was sent to the Middle East as a private from September 1990 to April 1991. In August 1990, myself and my company went on alert and spent over 24 hours painting vehicles with the CARC paint, and I remember the fruity smelling odor. For several days after painting the battalion's vehicles, I felt very nauseous.

We were shipped to King Abdul Aziz Port. I stayed there for about 2 to 3 weeks. The facilities were pretty disgusting, filthy. There were not enough restrooms and showers to accommodate the amount of people who had to utilize them. They were not properly cleaned either.

On the port that I was at, food and water was rationed out to us. After a couple of weeks on the port, I began experiencing nausea, headaches, and diarrhea.

During the convoy to our first site in the desert, my condition became worse, with vomiting, migraines, and diarrhea. While setting up camp, I passed out and was taken to a field hospital and treated for what was then said as dysentery and dehydration. I was treated with pills and an IV.

After Christmas, my communication team supported the 18th Airborne Corps Main, where we were sent to King Khalid Military City, just days before the air campaign. KKMC was where I first heard chemical alarms and SCUD alerts. Hours before the air war started, we began taking the bromide tablets. During the first hours of the air war, we traveled in MOPP–4 at night to a city called Rafha, just miles from the Iraqi border.

I continued to take the bromide tablets for a total period of 8 to 9 days, three times a day, in front of a noncommissioned officer. At Rafha, we experienced many chemical alarms, and after the alarms were sounded, my platoon sergeant and my platoon leader would call for a private to unmask to see if it was all clear. I was one of those privates, and we were told we were expendable.

Sometime during this period, I was driving through Hafa-Albotin the day a SCUD landed. A soldier gave us the sign "GAS, GAS, GAS." I noticed a rainbow in the sky, and I questioned what that rainbow was caused from. Today, I still do want to know what it is, sir.

A day or two prior to the ground war, I went to Rafha to receive a shot. I was handed a piece of paper to sign and release the Army or the Government—I am not sure which—of any and all adverse side effects. The paper stated it was an experimental drug, which I do not remember the name. I was not allowed to refuse the shot. I was not allowed to receive the paper, but I was allowed to refuse to sign it.

After the shot was administered, I began noticing heart palpitations and tunnel vision. When the ground war started, we convoyed to Iraq and established a site. We were told by our first sergeant to turn in all live ammunition, and the only ones allowed to have it would be the guard points. Because of my lack of knowledge of the dangers of depleted uranium on destroyed tanks, armored vehicles, and bunkers, I did not protect myself with my MOPP gear while climbing on and in them.

In April 1991, I returned to Fort Hood, TX, and numerous times I reported to the troop medical clinic, complaining of heart palpitations, migraines, severe diarrhea, and muscle spasms. No tests were run, and I was always told to take a couple of days off and bed rest. Prior to getting out of the Army and my ETS physical, I stated those same problems I went to the TMC for.

They had me wear a heart monitor, and the results were that my heart was beating faster than normal, and I was told that it was nothing to worry about.

After I left the Army in 1992, I moved my family to Ohio. I first went to the VA Clinic in Columbus, OH, June 1994, to sign on the Persian Gulf Registry Exam. Upon completion of the exam, the attending physician stated to me, and I quote: "There is nothing wrong with you. It is all stress-related."

I believed him, and I thought from his opinion and my family's comments that there was nothing wrong with me. I later found out from a patient rep that the physician for the Persian Gulf Registry Exam had set various appointments for me, which my records indicate a no-show for all set appointments. To the best of my knowledge, I do not remember him setting those appointments for me. I was not aware of them.

As time went on, my symptoms had been increasing in number and seemed to be getting worse. I did nothing medically until July 1996, when I returned to the VA Clinic for another, a second Persian Gulf Registry Examination. After that time, a primary physician was established. She then started setting appointments, lab work, CAT scan of head, heart monitor, et cetera.

The problems I have had with the VA Clinic, outpatient clinic in Columbus, OH are numerous. One, not receiving test results. My appointments with my primary physician started out at about every 2 weeks, then they started going every couple of months. I had a problem with my physician personally walking me to the mental health clinic like I am a crazy person and I cannot find my own way.

I have a problem with a psychologist trying to hypnotize me for pain control. Stare at a black dot on the wall and listen to this tape.

On one occasion, after telling my physician my health has gotten worse, she told me this: Your lab work is normal. There is nothing to treat. There is no diagnosis. I can give you Tylenol or Motrin for your pain, but please note, before this time, she had been prescribing me meds such as Solodac and Hyproxin for my pain.

In November 1996, I admitted myself to the VA Hospital in Chillicothe, OH to get help for my health problems, depression, and suicidal tendencies. They diagnosed me with PTSD and Dysthymic Disorder.

In December 1996, I tried to commit suicide because of my declining health problems, which everyone said there was nothing wrong with me, and the breakup of my marriage. I was admitted to Knox County Community Hospital's psych ward for about a week.

In January 1997, I returned to my primary physician again, explaining everything that had happened, and I told her I had not worked for quite a while, and she said she could not give me a work excuse to turn in; she could not provide me with one. I asked for a referral to another medical facility, and she said she could not do that, either.

On my very first appointment with the physical therapist, she diagnosed me with fibromyalgia by having me push my arms this way, pull my arms that way, same with my feet. I do not see how this is possible.

In March 1997, I experienced bad blurred and double vision, and I went to an optometrist. His diagnosis was hypertropia, large vertical muscle imbalance, esophoria at near, accommodative deficiency. And vision therapy was recommended for treatment, prescription sunglasses, and bifocals.

May 12, 1997, I went to the VA Hospital in Washington, DC, and had numerous tests done on me, which I do not have the results of as of today. May 12 to 14, 1997, I went to Georgetown University Medical Center for further studies. No results as of today.

I was told that I would be at the VA Hospital in Washington, DC, between 10 to 14 days, but I was only there 6, 2 of which were on the weekend, and the first day nothing was done.

When I joined the Army, I signed a contract with the United States stating that if anything happened to me in an act of a war, peacekeeping process, what have you, if I die, if I become ill during my time in service, the United States would take care of me. I fulfilled my portion of that contract; now it is time for you to fulfill your portion of the contract.

In closing, I would like to say, due to the time restraints, I was not able to provide you with all the information I have knowledge of. Thank you, sir.

[The prepared statement of Mr. Canterbury follows:]

Detailed Report of Paul Canterbury from 1989 to Present

I would like to explain to the best of my ability, my experiences while in the Gulf Region and my experiences with the VA Hospitals.

I believe these statements to be as accurate and true as it can possibly be considering my memory losses.

I served in the US Army at Ft. Hood, TX. from 1989 to 1992. During my enlistment I was sent to the Middle East from Sept. 1990 to April 1991 with the rank of Private.

In August of 1990, my unit, Delta Co. 57th SIG BN, went on alert and prepared for deployment. While preparing for deployment I personally along with my unit spent well over 24 hours painting vehicles and equipment with the sand colored paint (C.A.R.C.). I'm not sure of any dangers from the fruity smelling paint, but I remember feeling pretty nauseous for several days after.

In September, 1990, 57th SIG BN deployed to King Abdul Aziz Port in Saudi Arabia. There we stayed for 2-3 weeks waiting for equipment to arrive in the country. There were literally thousands of troops housed in these Hugh buildings, and there was a lack of accommodations for this number of troops for any period of time. The sanitary conditions were unbelievable. There were not enough toilet and shower facilities for the number of people trying to utilize them. The facilities were not properly cleaned if at all. There was not enough food and water on the port, and because of that factor it was rationed.

After a couple of weeks on the port I began experiencing nauses, headaches and diarrhea. Days later equipment arrived and orders came in to move to the Desert. During the convoy to our first site, I became very ill, vomiting, a migraine and diarrhea.

Once we arrived at our site and started to make camp I passed out. I was then taken to a field Army Hospital, where I stayed several days and was given pills and IV's for treatment of Dysentery and Dehydration.

My job titles in the service were primary (31L10) Wire Systems Installer and Secondary (31D10) Transmissions Systems Operator. I was in the (LEN Platoon) Large Extension Node. Only 3, 31L10 of LEN Plt. carried the 31D10 job title. The three of us were sent to help Small Extension Node Commo. teams and would always end up back with the LEN. The LEN stayed at our first site for several months which during this time I was floating back and forth from the LEN and different SENS. Sometime after Christmas I went with a SEN Team to support 18th Airborne Corp. Main. This SEN team consisted of 6 people.

We went to K.K.M.C. (King Kalid Military City). We were at K.K.M.C. for a couple of days prior to the AIR campaign. It was during my stay there, I remember the first chemical alarms sounding and the first SCUD alerts. Just hours before the AIR campaign, we got orders to start taking the bromide tablets, and to move out to a new unoccupied territory during the first hours of the AIR war. Traveling in Mopp 4 at night, we ended up going to a city in Saudi Arabia, 7 miles from the Iraq border called Rafha. The Army took the airport next to the city the following morning.

I stayed with the SEN team until the LEN moved a couple miles up the pipeline. Back with the LEN once again, and for the rest of the time I spent in the Gulf, I continued to take the bromide tablets. I remember taking them 3 times a day every 8 hours and in the presence of a Non Commissioned Officer. In all I ended up taking a whole package and started another one before I was told it was OK to stop taking them.

The LEN stayed at this site until the ground war. At this site we experienced more chemical alarms. After the alarms our PLT LDR and PLT SGT. would call for a private to unmask. I was one of those privates. We were told at that time that we were expendable.

Also during this period, I was sent one day on a supply run to either Daharan or Dammon. We had to drive through Hafa-Albotin the day a SCUD landed. While driving through the city, soldiers were giving us the 'GAS, GAS, GAS' sign by waiving their arms back and forth in the air. I then pulled over and got into MOPP 4. Afterwards and still to this day I question what the rainbow in the sky was!!!!!! Speaking of questions, here's another one, but first keep in mind the flies were terrible in Saudi Arabia. Why were their no flies on the dead sheep near Rafha??????????

A day or so before the ground war we were sent to Rafha to receive shots. At the airport standing in line papers were passed out for us to sign. This paper stated we are getting a shot that was an experimental drug to protect us against chemicals Iraq was suspected to have. By signing this paper you are releasing the Army or Government for any adverse side effects. I was allowed to refuse to sign (which I did) but I was not allowed to refuse the shot or allowed to receive the paper stating the name of the drug the shot consisted of. I do not remember the name of the shot. I remember that after this time was when I first started to experience my heart racing and at the same time I get this tunnel vision.

The LEN left the site near Rafha when the ground war started. We convoyed to Iraq behind the French, 101, and a Armored Division. During the convoy I saw Apaches take out Iraqi tanks, and the aftermath of the combat ahead of us. I literally saw hundreds of Iraqi dear and thousands of POW's on the convoy to Iraq. Once the LEN reached its destination in Iraq, we established our site in that rainy, and rocky terrain. I don't know where in Iraq we were but we remained at this location until we returned to Saudi Arabia

in late March or early April. The day we established our site, a perimeter was set up with constatina wire and guard points were made. We were then told to turn in our live ammunition by our First Sgt. The only ones with live ammunition were the guard points for the time we remained in Iraq.

During the time in Iraq, I like many others were ignorant to the possible contamination of Depleted Uranium on destroyed Iraqi tanks, armored vehicles and bunkers. I climbed on and in them not protecting myself with my MOPP gear.

Somewhere around the 1st of April, 1991 all of Delta Company 57th SIG.BN slowly returned to Saudi Arabia to prepare for deployment back to Fort Hood, TX. We returned to Ft. Hood on April 15, 1991. After returning to Ft. Hood I attended the Troop Medical Clinic quite a few times complaining of heart palpitations, migraines, severe diarrhea, and muscle aches and spasms. No tests or treatment was recommended, just bed rest for a couple of days. Just prior to getting out of the Army, in my ETS physical, I stated the same problems that I went to the TMC for. They had me wear a heart monitor and the results were that my heart was beating faster than normal. I was told at that time it was nothing to worry about.

I got out of the Army on August 30, 1992. I then moved my family to Delaware, Ohio. My pregnant wife and 2 small children. At the time my wife conceived we were not advised we should wait a year after I had been home before having anymore children. Could something be wrong with my youngest daughter because she was conceived as soon as I returned back in the States?????????????????????????????????
Since being out of the Service my symptoms have worsened and more have appeared. I will attach a complete list of symptoms. I cannot honestly give an accurate chronological order of all my symptoms.

I first went to the V.A. outpatient clinic in Columbus, Ohio on June 28, 1994 to sign on the Persian Gulf Registry Exam. The attending Physician went through his exam and later stated to me and I quote "THERE'S NOTHING WRONG WITH YOU, IT'S ALL STRESS RELATED". I believed him and just thought from his opinion and my family's comments that there was nothing wrong with me. I though well maybe this is just a sign of getting older. I found out on Jan. 29, 1997 from the Patient Representative that the Physician for the P.G.R.E. set up various appointments for me which my records indicate a NO-SHOW for all appointments. From the best that I can remember I did not have any knowledge of said appointments. From the time of the P.G. exam to Nov, 1996, I did not seek medical attention other than with 2 bouts of kidney stones and a case of the chicken pox. From this time on I secluded myself from my family, friends and mostly my wife and children. I had lost total interest in family functions or social activities. I believe this is due to my experience in the Gulf, my current health conditions, and the beginning of my drug abuse. Because of secluding myself from my spouse and children, constant complaints of pain & fatigue, and the drug abuse the end result was separation and a divorce in the process.

As time went on my symptoms may have been increasing in numbers and seemed to be getting worse, not knowing my rights to be seen by Doctors at the V.A. Clinic I again returned to have another P.G. Registry Exam done in July, 1996. The attending Physician in turn started setting up appointments for me. A primary care team and Physician were established. I do not doubt that at first the Physician was setting up appointments with me for lab work, cat scan of head, heart monitor, etc, etc. The problems I've been having at the V.A. Clinic is I've not received results of these tests, medications not helping and the Physician walking me personally to the Mental Health Clinic like I'm some crazy person that can't find their own way. I also have a problem with Psychologists trying to hypnotize me by sitting me in a room to listen to a tape and stare at a black dot on the wall. This procedure was for pain control.. Another problem with my care at the V.A. Clinic is my appointments with my primary Physician being spread further apart. My last scheduled appointment was 1/8/97. My last appointment was on 1/21/97 and that was made by me on 1/16/97 when I walked in due to my being rushed to the emergency room at a local hospital due to my illnesses on 1/11/97. I waited until 1/16/97 to go the V.A. Clinic because I couldn't get enough strength to go. When I did go I was not able to see my primary Doctor. I really received no help on that date, just set up appointment with primary Doctor for 1/21/97.

In November, 1996, I admitted myself to the V.A. Hospital in Chillicothe, Ohio. I went there to seek medical attention for my health conditions and for a feeling of depression and suicidal tendencies. They diagnosed me with P.T.S.D.(with no test results) and Dysthmic Disorder.

December, 1996 I tried to commit suicide because of my current declining health problems and my personal issues. I was admitted to Knox County Community Hospital in Mt. Vernon, Ohio.

On 1/21/97 I went to see my primary Doctor again. I told her then that I have not been working and that my health has gotten worse since I was last seen. I requested a written work excuse, which she said she couldn't provide me with one. I requested a referral to another medical facility, which she said she couldn't do. Also in response to me telling her my health had worsened she responded by saying to me exactly this 'THERE IS NOTHING TO TREAT, YOUR PHYSICAL EXAM IS NORMAL, YOUR LAB WORK IS NORMAL, THERE IS NOT A DIAGNOSIS'. I can give you Tylenol or Motrin for you pain.

On 1/16/97 I went to the Persian Gulf coordinator in the building. I told her what had happened that day and for some reason she set up an appointment with a Doctor on 1/28/97 to talk to him. On 1/28/97 I went to my appointment with this Doctor with a list of my health problems and a list of requests. He sat and listened to my requests, and said I'm not the person who you need to talk to. He said the Patient Representative is the person whom you need to speak to. He then walked me and my records down to her office. She was not available at this time. He asked someone to page her. While waiting he took my records into another Doctors office and started going through my records and discussing me with him. As soon as I walked in the office and sat down to see what was

being said my records were closed. The Doctor got up and walked out. About a minute or so later the Patient Rep. walked up to us and I was introduced to her and we went into a room to talk. She said I just caught her leaving the building, could I be brief with her. I handed her my list of requests and ailments, she made copies and asked if she could call me the next day at home. I said yes and as we were leaving the room the Doctor walked by and stopped the patient Rep. I went on to the canteen for some lunch. My records remained in the Doctors office. I know this to be a fact because later that day I had my first scheduled appointment with a Doctor from Ohio State University. When arriving at the appointment she didn't have my records. I personally went to the first Doctor's office to get them and he said he didn't have them any longer. I then went to the second Doctor's office and he was not in, but my records laid on his desk opened. I took them back to the Physical Therapists' office and she said some paperwork was missing, could I go back out to some desk to see if they could print it up and bring them back to her. I did as she requested. At that appointment with her all she did was have me to remove my shirt, shoes and socks. She looked at me said you have dry skin, had me push this way, pull that way with my arms and legs. She said all right you have what is called Fibromyalgia. How can she come up with a diagnosis on one visit when all the others have come up with nothing to treat?
Now with talking with the Patient Representative on 1/29/97 who had my records in front of her while talking to me on the phone, she stated the my primary Doctor had notes down as Fibromyalgia (this was never discussed with me by her). I don't know but I'm concerned about the legitimacy of this diagnosis.

January, 1997 I filed claims with the V.A. Regional office in Cleveland, Ohio. They in turn set up a Comprehension & Pension Exam at Knox County Community Hospital for February 28, 1997 which I attended. Also in January, 1997 I sent off to St. Louis, Mo. for my complete, including medical and dental service records and to this date I have not received them. My case worker in the Cleveland, Ohio Regional office has said "They have not received theirs either. Their office gives St. Louis 120 days for these records to be sent and that it has been well over the allotted time. My claim is ready, but the hold up is those missing records.

In March, 1997 I began having problems with my eyes. What I was experiencing was blurred and double vision. Please keep in mind other than light sensitivity, I have never had any problems with my eyes, I have always had 20/20 vision. February, 1997 I went to an Optometrist and what he found is as follows:

Hypertropia (a condition where one eye turns upward or downward)
A Large Vertical Imbalance
Esophoria at near
A Accommodative Deficiency.

He then recommended Vision Therapy, Prescription Sunglasses and Progressive Multifocal (bifocals) correction.

On May 6th, 1997 I was sent to the Persian Gulf Referral Program in Washington D.C. I was at the V.A. Hospital from the 6th to the 12th and sent to Georgetown University Medical Center for a research program entitled, Central Nervous System Dysregulation in Fibromyalgia, Cronic Fatigue Syndrome, and Persian Gulf Syndrome.

I was told that the program at the D.C. V.A. Hospital would take between 10 to 14 days. I was there for only 6 days 2 of which were on a weekend. During this time I went through some lab work, and tests. **

I believe that there is a protocol or a standard set of tests to be done on Gulf War Vets, and I question if the protocol was met with me, considering the number of days spent and the testing schedule. I also question the tests done on me which I've recieved few results. One test in particular I'm concerned about is the M.R.I. which I was told by the lab technition something was found. WHAT? I'm also concerned about different things discussed with me by docters, things such as skin cancer, Leismaniasis a disease which effects the immune system, and a brain tumor. When I asked if this is apart of whats wrong with me , the replys were, we are not saying this is your problems, we're discussing these items with you because there is a possibility of these. When docters would approach me , they would address me as a Gulf War Syndrome Patient, yet when I would ask if I were diagnosed with the Gulf War Syndrome I would be told " No, No-one has that diagnosis.

** See attached list of Testing Schedule from Washington D.C. V.A. Hospital

CURRENT LIST OF COMPLAINTS

1. Headaches/Migraines
2. Light Sensitivity
   - Hypertropia
   - Large Vertical Muscle Imbalance
   - Esophoria at Near
   - Accommodative Deficiency
3. Ringing in the ears
4. Sore Throat
5. Tightness/Chest Pain
6. Shortness of Breath
7. Heart palpitations
8. Extreme gas and bloating
9. Constant battle between diarrhea or being constipated
10. Poor appetite
11. Painful urination
12. Rash around groin area
13. Memory losses
14. Pain in muscles/muscle twitches and spasms
15. Pain in joints
    - all my joints crack as if you were cracking your knuckles
    - I have severe pain in my left hip which causes a limp in my walk
16. Sleep disorders
17. Numbness and tingly feelings in all extremities
18. Hot and cold flashes including cold sweats
19. Constant feeling of fatigue

R=95%

PERSIAN GULF REFERRAL PROGRAM
TESTING SCHEDULE

The following tests have been scheduled for Mr. Canterbury

Wednesday	**5/7/97**	**Neuropsychology Testing 9:00AM, room 2A154**
Thursday	**5/8/97**	**Colonoscopy in AM. Needs to be NPO in computer**
Friday	**5/9/97**	**M.R.I. 9:00AM, in basement, room BH200**
	5/9/97	**E.E.G. 1:00pm, ROOM 3A112**
Sunday	**5/11/97**	**Sleep study test will sleep there night of 5/11/97**
Monday	**5/12/97**	**Needs to follow up with MSLT q 2 hours during day at room 3A112**

Theresa King, PA will assist you in coordinating his care, beeper #28443 Thank-You

R=95%

Mr. SHAYS. Thank you, Mr. Canterbury. Mr. Roman.

Col. ROMAN. Sir, I am ready.

Mr. SHAYS. OK. And I am going to emphasize again——

Col. ROMAN. Yes, sir.

Mr. SHAYS [continuing]. The only time restraint we have now would be self-imposed by you.

Col. ROMAN. OK.

Mr. SHAYS. So I want you to really take it slower.

Col. ROMAN. Thank you, sir. One of the reasons I started talking a little fast was because I felt the blood starting to come, and I was trying to get it over with before I start—well, anyway, thank you very much. I will continue. If I could pick up right from where I left off, I would appreciate it.

Mr. SHAYS. I am going to ask you just to slow down a second and tell me where were you. Do we have the same document you have? What page are you on?

Col. ROMAN. I am on page 3——

Mr. SHAYS. OK.

Col. ROMAN [continuing]. And I am down at the last paragraph, and I am not giving it all; I have cutoff some of it.

Mr. SHAYS. You cutoff some of the better parts, frankly.

Col. ROMAN. I was afraid, trying——

Mr. SHAYS. OK.

Col. ROMAN. OK. I think what I will do, sir, because I think I was bleeding all over myself at the time that I was talking earlier, is pick up just at the second paragraph on page 3, if I may.

Mr. SHAYS. That is fine.

Col. ROMAN. And say to you that my first time to Riyadh, I became ill, was treated for nausea and headaches and vomiting, diarrhea, and a high temperature.

My commander, who was a physician, was with me, and he treated me for the symptoms which appear to be like food poisoning. This nausea, headaches, and vomiting-like symptoms continued throughout the time I was in the Persian Gulf, and I continued to treat it like food poisoning, with Immodium and 800-milligram Motrin. As I indicated, it really is the Army's blessed answer to all pain because it works, at least for pain.

Rashes, I had over my body while I was in the Gulf, I thought they were normal and expected, since I spent most of my days in the sand and the field, wind and sun, with all the attendant fleas, flies, and other desert parasites. I used a calamine lotion-like substance which served to sooth but did not relieve or get rid of the severe rashes that I experienced.

Life in the theater of operations was a constant adrenalin rush, with 3 to 4 hours' sleep in between. Headaches I began to experience, I attributed to fatigue and the lack of sleep. Upon returning to the States and my discharge from active duty, I returned home like thousands of other United States soldiers, and the symptoms I had experienced in the Persian Gulf continued after I returned home and got progressively worse, as a matter of fact.

In 1993, I registered myself into the Washington, DC Veterans Hospital after receiving an invitation from the VA to come in for an examination if I was a Persian Gulf vet. The Washington, DC VA noted a number of problems, including sleep apnea—and short-

term memory loss, hearing loss, and they recorded all the ailments I had indicated to them, including my flu-like symptoms, swelling in my hands, knees, and ankles, respiratory problems, and severe headaches.

No treatment was offered. Rather, the VA Hospital billed me for my supposed free examination and ended up attaching my next year's meager tax return for money I owed them for an examination that I was offered, which I was requested to take by the VA. So I do not know why I was being billed, but I could not fight it enough. They kept fighting it back, and they sent it over to the IRS, and they took the money out of my return.

I went back to Denver in 1994 and registered at the Denver VA Hospital, where instead of requesting my examination files from the Washington, DC VA, I underwent a second complete re-examination, with, I might add, similar results.

Then, in 1995, the United States Army sent me a letter to report to Fitzsimmons Army Medical Center if I was suffering any ill effects from the Persian Gulf war. Once more, I underwent a complete examination, from blood to MRI, and everything in between. The results this time were much clearer. The Army doctors found out again that I had chronic fatigue, precancerous nasal and colon polyps, chronic skin rashes and hives, which have not been tied to a cause yet, sleep apnea—respiratory illness of mysterious origin, short-term memory loss, flu-like symptoms which would come and go, lasted for 6 weeks, and chronic arthritis of the joints.

The young Army doctors tried to treat me and had scheduled me for an operation to remove the polyps from my nose. Had they done that, maybe I would not be bleeding, but the colonel in charge of the Persian Gulf examinations advised me that they could not treat me because it was not determined that I had been injured or had received that particular illness in the Persian Gulf.

To date, although I now have had three official VA and Army examinations since 1993, I still continue to receive requests for more and more information from the VA Claims Office in Phoenix, AZ. Materials I send them are never acknowledged as received, and the telephone numbers that are given are not to any VA-recognized exchange, and the name given for contact is not a true VA employee; at least the number that answers at IRS, by the way, is not the name of the VA office I have tried to reach.

Frustration is a word that does not begin to explain the feeling of being in the system 4 years now with no real contact from a person, just requests for more and more information. It is particularly maddening when I personally sent my records from the VA hospitals and the Army to them for evaluation, yet when I called them in the winter of 1997 in Phoenix and left a message via a third party to advise me of what records they had, they sent me back a written message that said they were requesting my records from the VA hospitals in Washington, DC, and in Denver.

I thought they were evaluating me at that time, but without those records, how could they have been evaluating me?

1996 was not a good year for me. I was hospitalized three times and was treated by my private physician for a respiratory ailment. I could not walk more than 25 steps without having to stop, out of breath and fatigued. This ailment, which was life-threatening,

would not allow me to lie on my back to sleep, as I would begin to drown, or at least it would feel like I was drowning, when my lungs were filling up with fluid.

I was forced to sit up to sleep and was constantly fatigued due to the lack of sleep and no energy. My cardiologist in Denver, Dr. Peter Steele, diagnosed me as having cardiomyopathy with congestive heart failure. The onset of symptoms, he said, "which would suggest that possibility that this was induced by a source in the Middle East during the Gulf war." "What is clear," Dr. Steele stated, is that "he served in the Middle East and that he has a cardiomyopathy." He goes on to say that I would submit that this may well be a part of the Gulf War Syndrome; I attach a letter for your convenience from Dr. Steele.

Last December 1996, I was examined by Dr. William Baumzweiger, and I misspelled the name. For the record, it is B-A-U-M-Z-W-E-I-G-E-R. He is a neurologist at the Los Angeles Veterans Hospital. After a 3-hour examination, Dr. Baumzweiger advised me that I had suffered severe neurological damage while in the Persian Gulf and had, in fact, suffered brain stem damage as well. Dr. Baumzweiger further advised me that my neurological damage was as severe as he had seen and was, in fact, caused by exposure to unknown chemical agents while in the Persian Gulf.

He also advised me that I probably would not live as long as I would have had I not been in the Persian Gulf and that unless I took 1 year off to do nothing but recuperate, I would most likely be a candidate for a heart transplant within 3 to 5 years.

Dr. Baumzweiger also concurred with Dr. Peter Steele's diagnosis of cardiomyopathy caused by my service in the Middle East during the war. He suggested that this cardiomyopathy may well be a part of the Gulf War Syndrome.

Incidentally, while I was in Dr. Baumzweier's office, he was summoned into the chief neurologist's office. Upon his return, he informed me that he was no longer authorized to treat Persian Gulf vets. When I asked him why, he advised me that his findings had not coincided with the VA's on the reasons for Gulf war vets' illnesses; therefore, he was asked to not treat Persian Gulf vets anymore.

Mr. SHAYS. Col. Roman, you are under oath right now, and you are saying that while you were having this examination, this doctor left and then came back, and, to the best of your recollection, this is precisely what he said, almost what he said, or maybe something like that?

Col. ROMAN. He advised me exactly what I just said.

Mr. SHAYS. OK.

Col. ROMAN. And I also saw or happened to see the letter that he had from the chief neurologist where he was asked not to treat Persian Gulf vets anymore. I just glanced at it. They had it on the table there, and I saw it. He was somewhat distressed, by the way, at the time.

The still-too-recent memory of the Vietnam veterans and Agent Orange casts a pall on the ongoing denial by the same bureaucracies who continue to deceive Persian Gulf veterans. Didn't we learn anything from the Agent Orange debacle? Must we be con-

demned to remaking the same mistakes with our Persian Gulf veterans?

Ironically, on November 2, 1994, the President signed a Veterans Benefits Improvements Act of 1994, Public Law 103–446. This law authorized the Department of Veterans' Affairs to pay service-connected compensation to Persian Gulf veterans who are suffering from chronic disabilities resulting from undiagnosed illnesses. And I think "undiagnosed illnesses" here is the key, since the precedent had already been made to Agent Orange victims who, after many, many years a compensatory fund was created for them by the U.S. Congress. This occurred after a study by the Centers for Disease Control failed to establish a link between Dioxin absorption to any serious Vietnam-veteran malady.

Two and a half years after this law went into effect, the information letter I received from Secretary of Veterans' Affairs Jessie Brown still has not borne fruit for most of my fellow Persian Gulf veterans. Lip service and voluminous correspondence from the VA is all that has resulted for most of us.

A bullet from an AK–47, a land mine, a mortar shell, or grenade would all cause trauma to the body or death. How different are these weapons of war to those invisible, but equally devastating, mortar weapons of war in the form of lethal chemicals and biological agents? Answer: There is no difference in the effect; it just takes a little longer to cause the casualty.

I am going to add to you that on the question that you asked me, Mr. Chairman, I did have a retired Major, Denise Nichols, fax a letter that I wrote on that particular issue to Dr. Baumzweiger, and I gave her in writing what had happened on that particular day so that I had it on the record.

I believe that every Gulf war veteran who has suffered the effects of a chemical-biological-warfare weapon should be just as eligible for the Purple Heart as those wounded by conventional weapons. The wounds might look different, but the effect is the same.

Thank you for allowing me to testify today.

[The prepared statement of Col. Roman follows:]

"THE GULF WAR SYNDROME"

Report Stings Pentagon – GAO Study says "Gulf War illness clues overlooked". (The Denver Post (17 June 1997).

Poisoned in the Gulf? : New clues to the veterans' mysterious illnesses (Newsweek, 29 April 1996).

Gulf War Fallout : Colorado's Gulf veterans, who claim toxic agents are wreaking havoc on their bodies, take on government for benefits. (Rocky Mountain News, 2 April 1995). In this story it was reported that biological warfare pathogens also were discovered, according to Congressional documents. The story went on to state how a captured Iraqi gas mask held the residue of two pathogens – Q fever and brucella. Some veterans have since tested positive for antibodies to these pathogens, the story went on to state.

Sick Gulf Vets : Weary wait for treatment. (Army Times/March 28, 1994). "We are committed to the treatment of veterans of the Persian Gulf conflict who are experiencing problems as a result of their service, " said Edwin Dorn, Undersecretary of Defense for Personnel and Readiness. "We are determined to fashion compensation for those who are too sick to work." (Army Times, 28 March 1994).

Dr. Grace Ziem, a toxicology expert in Baltimore, said she has been treating Persian Gulf veterans who share many similar respiratory and neurological problems with her civilian patients with the syndrome. "These veterans on the average are sicker than the civilian patients I see," she said.

The preceding news reports were culled from a file I began collecting in 1994. It shows a non-progressive, seemingly uncaring Department of Defense (DOD) and Veterans' Administration (VA) in denial of the facts being presented by military personnel and the medical community at large. It also shows that in three years there has been virtually no change in the narrow, parochial thinking, or for that matter, policy, of these two federal departments most responsible for the plight of U.S. veterans of Desert Shield/Desert Storm.

I am greatly saddened by these newspaper accounts because I see a continuing pattern of official DOD misinformation and negligence tantamount to malfeasance in office for ignoring testimony and documentation referring to the use or presence of chemicals and other biological agents that reports indicate were found in the Theater of Operations during Desert Shield/Desert Storm.

I arrived in the Theater on my birthday 6 January 1991, after volunteering to serve in the Persian Gulf and being brought on active duty in December, 1990. My primary responsibility as Colonel, Medical Service Corps, was as Deputy Commander of the 311th Evacuation Hospital. I was responsible for Operations, Logistics, and Security. As secondary assignments I was also the Public Affairs Officer and Liaison to the Ministry of Health in Abu Dhabi, United Arab Emirates (UAE) where the 311th was located. We were also near Al Dafra and Al Bateen Air Force Bases where the U.S. Air Force flew daily sorties out to bomb Iraq. Also flying out of Al Bateen were daily air shuttles called the "Star Shuttle" which were either C-130's or C-141's that flew daily shuttles to Riyadh, Dharhan, King Kalid Military City (KKMC) and other points in the Theater of Operations. I often flew

these Star Shuttle flights into Riyadh, Dharhan, or KKMC. During several official visits to these strategic military cities there were frequent SCUD attacks during which I often heard the chemical alarms sound -- when I asked if these alarms meant chemicals had been detected I was told that the chemical alarms had malfunctioned.

My first time to Riyadh I became ill and was treated for nausea, headaches, vomiting, diarrhea and a high temperature. My Commander, a physician, was with me and he treated me for the symptoms which appeared to be food poison. This nausea, headache, vomiting and flu-like symptoms continued throughout the time I was in the Persian Gulf and I continued to treat it like food poisoning with Immodium and 800 mg. Motrin (the Army's blessed answer to all pain).

Rashes I had over my body, while I was in the Gulf, I thought were normal and expected since I spent most of my days in the sand, wind and sun with all the attendant fleas, flies and other desert parasites. A Calamine lotion-like substance served to soothe but not to relieve or get rid of the severe rashes I experienced.

Life in the Theater of Operations was a constant adrenaline rush with 3-4 hours sleep in between. Headaches I began to experience I attributed to fatigue and lack of sleep. A trip to Kuwait to assist in treating Iraqi prisoners of war resulted in my exposure to hundreds of Iraqi prisoners and burning oil wells.

Upon returning to the States and my discharge from active duty, I returned home like thousands of other U.S. soldiers. The symptoms I had experienced in the Persian Gulf continued after I returned home and got progressively worse. In 1993 I registered myself into the Washington, D.C. Veterans' Hospital after receiving an invitation from the VA to come in for an examination if I was a Persian Gulf veteran. The Washington, D.C. VA

noted a number of problems including sleep apnea, short-term memory loss, hearing loss and they recorded all of the ailments I indicated to them including my flu-like symptoms, swelling of my hands, knees and ankles, respiratory problems and severe headaches. No treatment was offered. Rather, the VA hospital billed me for my supposed free examination and they ended up attaching my next year's meager tax return for money I "owed them" for an examination I was offered and which I was requested to take by the VA.

I moved back to Denver in 1994, and registered at the Denver VA Hospital where instead of requesting my examination files from the Washington, D.C. VA, I underwent a complete re-examination.

The U.S. Army then sent me a letter to report to Fitzsimmons Army Medical Center if I was suffering any ill effects from the Persian Gulf War. Once more I underwent a complete examination from blood to MRI and everything in between. The results this time were much clearer. The Army doctors found again that I had chronic fatigue; Pre-cancerous nasal and colon polyps; chronic skin rashes and hives (which have not been tied to a cause); sleep apnea; respiratory ailments of mysterious origin; short term memory loss; flu-like symptoms which would come and go lasting up to six weeks; and chronic arthritis of the joints. Young Army doctors tried to treat me and had scheduled me for an operation to remove the polyps but the Colonel in charge of the Persian Gulf examinations advised them that they could not treat me.

To date, although I have now had three official VA and Army examinations since 1993, I still continue to receive requests for more and more information from the VA Claims Office in Phoenix, Arizona. Materials I send there are never acknowledged as received

R-97%

and telephone numbers that are given are not to any VA recognized exchange and the name given for contact is not a true VA employee or at least the number that answers (at IRS) does not know the name or the VA office I have tried to reach. Frustration is a word that does not begin to explain the feeling of being in the "system" four years with no real contact from a person; just requests for more information. It is particularly maddening when I personally sent my records from the VA hospitals and the Army to them for evaluation. Yet, when I called them in the winter of 1997, and left a message via a third party to advise me of what records they had, they sent me a message that they were requesting my records from the VA Hospitals in Washington, D.C. and Denver. Without those records how could they have evaluated me?

Meanwhile in 1996, I was hospitalized three times and was treated by my private physician for a respiratory ailment. I could not walk more than 25 steps without having to stop; out of breath and fatigued. This ailment, which was life threatening, would not allow me to lie on my back to sleep as I would begin to drown or at least it would feel like it as my lungs filled with fluid. I was forced to sit up for sleep and was constantly fatigued due to lack of sleep and no energy.

My cardiologist in Denver, Dr. Peter Steele, diagnosed me as having . . . "cardiomyopathy with congestive heart failure. The onset of symptoms which would suggest the possibility that this was induced by service in the Middle East during the Gulf War." "What is clear", Dr. Steele stated, "is that he served in the Middle East and that he has a cardiomyopathy." . . . "I would submit that this may well be part of the Gulf War Syndrome." (Letter attached).

1

On 27 December 1996, I was examined by Dr. William Baumschweiger, Neurologist, at the Los Angeles Veterans' Hospital. After a three hour examination Dr. Baumschweiger advised me that I had suffered severe neurological damage while in the Persian Gulf and had in fact suffered brain stem damage as well. Dr. Baumschweiger further advised me that my neurological damage was as severe as he had seen and that it was in fact caused by exposure to unknown chemical agents while in the Persian Gulf. He also advised me that I probably would not live as long as I would have had I not been in the Persian Gulf and that unless I took one year off to do nothing but recuperate that I would most likely be a candidate for a heart transplant within 3-5 years. Dr. Baumschweiger also concurred with Dr. Peter Steele's diagnosis of cardiomyopathy caused by my service in the Middle East during the War and suggested that this (cardiomyopathy) may well be part of the Gulf War Syndrome. Coincidentally, while I was in Dr. Baumschweiger's office, he was summoned into the Chief Neurologist's office. Upon his return he informed me that he was no longer authorized to treat Persian Gulf Vets. When I asked him why he advised me that his findings did not coincide with the VA's on the reasons for Gulf War Vets' illnesses.

I am not certain why the DOD and the VA have chosen the position they have -- I am only certain of the mistrust and alienation this has caused among veterans and their families toward the very federal government that has asked our young men and women to trust them and has promised that they would be well taken care of in the event that they would become casualties.

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The still too recent memory of Vietnam veterans and Agent Orange casts a pall on the ongoing denial by the same bureaucracies who continue to deceive Persian Gulf veterans. Didn't we learn anything from the Agent Orange debacle? Must we be condemned to remaking the same mistakes with our veterans?

Ironically, on November 2, 1994, the President signed the "Veterans' Benefits Improvements Act of 1994", Public Law 103-446. This law authorized the Department of Veterans' Affairs to pay service connected compensation to Persian Gulf War veterans who are suffering from chronic disabilities resulting from undiagnosed illnesses. Undiagnosed illnesses is the key here since a precedent had already been made to Agent Orange victims, who after many, many years a compensatory fund was created for them by the U.S. Congress. This occurred after a study by the Center for Disease Control failed to establish a link between Dioxin absorption to any serious Vietnam Veteran malady.

Two and one-half years after this law went into effect the information letter I received from Secretary of Veterans' Affairs, Jesse Brown still has not borne fruit for most of my fellow Persian Gulf veterans. Lip service and voluminous correspondence from the VA is all that has resulted for most of us.

A bullet from an AK-47, a land mine, a mortar shell or grenade would all cause trauma to the body or death . . . how different are these weapons of war to those invisible but equally devastating and mortal weapons of war in the form of lethal chemicals and biological agents? Answer: There is no difference in the effect; it just takes a little longer to cause the casualty.

R=97%

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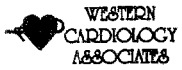
I believe every Persian Gulf veteran who has suffered the effects of a Chemical Biological Warfare (CBW) weapon should be just as eligible for the Purple Heart as those wounded by conventional weapons. The wounds might look different but the effect is the same.

Thank you for allowing me to testify today.

Gilbert D. Roman
Colonel, U.S. Army
(Retired-Reserve)

R=97%



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November 8, 1996

Gilbert D. Roman
1770 South Williams Street, No. 1004N
Denver, Colorado 80210

Dear Mr. Roman:

As our conversation, you may use the following in respect to your dealings with the Veterans' Affairs.

Mr. Roman has a cardiomyopathy with congestive heart failure. The onset of symptoms would suggest the possibility that this was induced by service in the Middle East during the Gulf War.

What is clear is that he served in the Middle East and that he has a cardiomyopathy. Whether or not there is a relation is difficult to be certain of. Cardiomyopathies usually do not have a clear-cut cause that can be developed with certainty. I would submit that this may well be part of the Gulf War Syndrome.

Sincerely,

Peter P. Steele, M.D.

PPS/bve

Mr. SHAYS. Thank you, Colonel. Colonel, we are going to be asking questions as soon as we hear from Mr. Stacy, who is next, and Staff Sgt. Zeller.

Mr. Stacy.

Mr. STACY. Thank you. I would like to say it is an honor for me to be here today. My name is Michael J. Stacy. I was a loader on an M1A1 main battle tank. I was exposed to depleted uranium and various other toxins, including possible chemical and biological agents. First, I would like to thank God; my wife; daughters, Haskell and Suzanne Dixon; Dan Fahey; the Military Toxins Project; and all of our family and friends.

Before deploying, I was in prime physical condition. I weighed 185 pounds, served with the 2nd Armored Company. I served with Alpha Company, 2nd Armored Division, Forward. I served in the Gulf from December 30, 1990 to May 6, 1991. I did get the anthrax shot, as well as others. I did take the PB pills three times a day. We kept the same chemical suits, even though it was opened 1 month before the war started. We took our protective gear off before we crossed the border into Iraq. We had the RAD meters. They looked like a wrist watch, but only key personnel were issued these.

In a report from the U.S. Army Ballistics Research Lab, dated December 1989, test results showed that soldiers who came into contact with contaminated vehicles could inhale resuspended depleted uranium dust or ingest depleted uranium via food intake, cigarette smoking, et cetera, prior to not washing hands and face. It was a very unclean environment over there.

Also, I have a letter here from the Office of the Secretary of Defense. It is dated May 30, 1997 and signed by Bernard Rostker. Prior to fielding the M1A1 tank and the munitions containing depleted uranium, controlled-burn tests were conducted in the United States to determine the hazards of depleted uranium burning at high temperatures. Such a high temperature would have to be sufficient to melt steel.

In the event of such a fire, a small fraction of the material may be dispersed into the atmosphere as the depleted uranium oxide fume or smoke and hence could be inhaled by the persons situated immediately down wind of an accidental fire or explosion involving depleted uranium ammunition. We saw tanks that were melted, that burned hot enough to melt steel.

I was involved in more than one friendly fire incident while I served in the Gulf. Our tanks had depleted uranium armor. I slept on the tank, over the blowout panels. We spent 90 percent of our time on the tanks. We were never warned of any health risks of depleted uranium. I climbed on and in tanks, trucks, and bunkers after they were hit with depleted uranium to inspect damage. We were never warned of the health risks. We knew we were shooting depleted uranium, but we were never warned of the health risks.

We went back through the battlefields after the war. I first got sick while in the Gulf, with headaches, nausea, chest pains, stomach cramps, and diarrhea. We assumed that it was from the water that we were drinking. We were told to go on a 48-hour fast, but under the operating conditions, we were unable to do that, so we just dealt with our condition.

My wife miscarried soon after returning to the Gulf. At that time, we did not know who to go to. She was 1 month pregnant. We did not report this incident, to my knowledge.

We returned to the States. My wife's health got worse. My health got worse. I have been diagnosed with multiple—I have multiple, undiagnosed illnesses: chronic fatigue, chest pain, joint pain, swelling of the joints, upper respiratory problems, sinus problems, and severe memory loss.

The VA has denied me for testing of depleted uranium. The VA has denied me for further testing. The VA still said all of my problems are from PTSD. We gave the Iraqi POWs when we captured them better treatment than the VA provides for myself. I believe my declining health is due to the shots taken before and in country, the PB pills, depleted uranium, and possible chemical and biological agents.

This has been a disgrace to me, my family, my unit, and the soldiers who died over there. Something needs to be done before my wife dies, before I die, or any other Gulf war vets die. I would also like to say, my wife weighed 127 pounds before I deployed to the Gulf. She was an ornery, mean, Oklahoma girl. Since my return, she has weighed under 100 pounds. She has dropped under 80 pounds. We were told by the doctor at the Indian Hospital in Claremore, OK, it would be in my best interest to have her committed to an insane asylum. They said they cannot find any reason why she is sick.

My daughter was born before the Gulf. She is displaying some symptoms. She has got aching bones and sinus problems. So was everybody I was around when I got back. I watched my wife's grandfather. His health severely declined. I believe it was because I spent lots of time with him. He passed away this spring—cancer. It ate up his whole body. His immune system failed.

My wife's mother-in-law; we lived with them when we soon returned from the Gulf. They started developing upper respiratory problems, other ailments since. They have moved to Nashville now. We are no longer around them. Her health has seemed to improve.

I feel abandoned. I feel mistreated. My wife has suffered the brunt of this illness. My wife sits behind me. She has lost all pride, all dignity—but supports and believes in me. I have been told for too long that it is all related to stress, and I will not take that any longer.

Thank you.

[The prepared statement of Mr. Stacy follows:]

Michael Joseph Stacy
Private 1st Class
445 72 4293

I address Representatives of the 105th Congress of the United States of America with great pride and gratitude for the invitation to speak before you concerning the Gulf War Syndrome and exposure to Depleted Uranium (DU). Thank you for the interest you have shown in not only me, but all those veterans who are suffering from these devestating unknown factors, which have stricken a lot of those who served under that command.

I would like to begin by saying that my greatest concern is my wife and child and their health and happiness. If I would have known then, what I know now, I would have taken the necessary precautions and possibly prevented my wife from having the health problems she has presently. Because without them, I would not be sitting before you today. They have given me the support, strength and encouragement to hold my head high with dignity and pride and to wear the uniform that I so dearly respected and to tell you my story.

Before I even considered the military as my life long ambition, I had desires of fathering a fairly large family of five (5) boys. My wife Shawna and I were married in August of 1988, with that exact goal in mind. After our daughter, Kinder Suzanne was born, I decided that it was time to be a man and make a better way of life for the beginnings of my future family, so I joined the U.S. Army in April, 1990. I went to boot camp and was assigned to the 2nd Armored Division Forward (Hell on Wheels) and was a M1A1 Tank Crewman. My orders sent me to Germany and I became an outstanding soldier, Excellence in Armor Private First Class. I ran a 12 minute 2 miles, 110 pushups in 2 minutes and 90 situps 2 minutes. I am trained in weapons, demolitions, hand-to-hand combat and am a Certified Combat Life Saver. I was the youngest gunner in my entire Battalion, but one of the few to not volunteer for service in the Gulf.

We (the Battalion) didn't think we would ever go to the Persian Gulf, because why would they want a tank unit this far North in Germany? And besides, I couldn't wait for my wife and 17 month old child to arrive and begin our new life together. So finally, after many months (before the war broke out), I had achieved my goal of getting my family back together and we settled into off-base housing and I prepared (in October of 1990) for a 2 week field trip to Bergen, Germany. I kissed my wife good-bye and off we went, and boy was it cold. During our field trip, one tank had run into another and ruptured a ballistic skirt, which contained Depleted Uranium. The tank commander ordered the hole to be covered up and we were told General Dynamics was informed of the incident, the soldiers who saw the inside were then de-briefed. This very instance fueled the curiosity about Depleted Uranium.

We came back from the Field, and Lt. Skavdahl came to notify us that we were going to be deploying to the Gulf. We landed In Country, December 30. We set up at the Port of Jubal and then moved to a forward camp waiting on our tanks. There were Marines, Sailors, Airborne, you name it...We were so proud. We were the newest and baddest, I felt no Marine had anything on me...I was a loader on a brand new M1A1 Heavy Main Battle Tank. I remember when we were at our forward camp we dug bunkers, at the time I thought, what a waste of time...But we did hear

alarms and had to get to MOPP 4 (full MOPP gear) and take cover in those bunkers. The hard work it took to dig those holes was such a comfort at that time.

On January 21, 1991, also a day I'll never forget, my 22nd birthday, we rolled toward Iraq. We prepared for war and waited and waited and waited and waited and waited...Then it started February 16, 1991. A Company from our Brigade made contact. They shot a truck and burm with tow missiles and had a couple of small gun fights. February 17th, war hit home and we lost a Bradley to enemy fire. A rocket propelled grenade. Two killed and 4 badly wounded. The bombing continued...so heavy at times, you couldn't sleep for days. We crossed the Border for good on February 24, 1991. When we crossed, we were in MOPP 4, after we crossed we went to MOPP level 0 and then repackaged our MOPP suits. The same ones we opened at the base camp back in early January of 1991, and had worn on numerous training expeditions and had repackaged each time...Unaware that they were useless after their initial opening. On the same day, February 24, 1991, we came under attack from enemy artillery, some of which exploded in the sky over us and created a white cloud, which then disintegrated over us. We had thought it was a marker, so we moved...This continued all day until our artillery knocked them out for good.

February 25, 1991, we started making contact as we were moving towards Iran. February 26th, at 18:30 we rolled out into a small battlefield, a place I'll never forget...There were trucks, tanks, BMP's and troops. We freely engaged the enemy until 04:15. I heard cease-fire, cease-fire, cease-fire, there's friendlies to the front. Before the 3rd cease-fire, we had already engaged them, shooting an American M1A1 from 3rd Armored Division. We shot them 6 times, we provided rescue efforts, but their ammo was cooking off (exploding) so we abandoned any further rescue, backed up 100 meters and set a perimeter and waited until dawn. That night we lost an M1A1 tank and a Bradley Fighting Vehicle to friendly fire. Eight buddies from my Battalion were dead and we were responsible for killing them.

The first DU penetrator we fired was on January 27, 1991, we fired Sabot and Heat. The Sabot is a DU penetrator and the Heat is high explosive. We fired 3 of each to battle sight our main gun. After we fired our first Sabot round, we knew then the DU penetrator was the round of choice. I think a full combat load for an M1A1 Heavy is 56 rounds. There's a certain load plan you follow, so many Sabot and Heat in your main ammo storage area and so many of each in your secondary storage area. I was the loader on my tank and after we fired our first DU penetrator, our platoon switched the ammo. Put all DU penetrators in the main storage area and put the Heat rounds in secondary storage. We were told, I quote, "Shoot 'em while you got 'em." The DU penetrators were so devastating that we used them for everything. Tanks, trucks, light armored vehicles, bunkers, everything but the troops. We found out very fast that the DU penetrators were 1000 times more devastating than we expected. When a bunker is shot with a DU penetrator, just the percussion from the round will kill any troops in the area. We wanted to shoot the good stuff and as much as possible.

We were at war with the best equipment out of all the Coalition Forces. No law, no rules, engage at will. My Platoon alone fired approximately 120 DU penetrators, with 4 tanks per Platoon. Thirty penetrators fired per tank, plus another 3 penetrators to battle sight our main gun, on January 27th. Plus another 3 penetrators on January 28, 1991. That's approximately 36 DU

penetrators each tank fired. There are 12 tanks per Company, 4 Companies per Battalion, we had 2 Battalions on line, 2nd Armored and 3rd Armored, that's a total of 96 tanks. Plus we were attached to 1st Infantry Division. They are a mechanized Battalion with an unknown number of Bradleys which fired 30 mm DU rounds. So as you can well expect, we were constantly in contact with this ammo.

After the war, we got a chance to see the tanks we shot up. Remember when I told you about going to the field in Bergen, Germany, where our curiosity about the DU in the Ballistic Skirts was first started? We wanted to look at this tank. We saw it sitting on the back of a heavy equipment transport, just outside of Kuwait. It was the talk of the Company, knowing we shot that tank and SGT Applegate had died inside. We were all over that tank. Inside and out. Curious about the perfect hole the penetrator made. All the ammo went off and the blowout panels, made to protect the crew if the ammo ignites, they are made so the explosion will go out the top. We poked around and talked about how the blowout panels saved those who were alive inside. We sat in the oilfields waiting for orders and in the meantime, we went on clearing missions...Blowing up ammo equipment and scavenger hunting. We climbed in and out of blown up equipment. Found discarding fins from the DU penetrators and one soldier found the bottom of a DU penetrator, which he proudly showed and handed to everyone for their own inspection. We spent several days looking for shrapnel, checking out blown up vehicles, tanks and bunkers. We were like kids, curious and excited, everyday was an adventure. Soon, we were in competition to find a complete DU penetrator, a highly valued prize to a tanker. We kept the ASFAB Caps (the rounds are combustible) and all that is left was an ashtray-sized primer and that's what we kept them for--ashtrays! The ASFAB Cap is sand stone compared to a DU penetrator from a tanks. We sat in the oilfields, went on clearing missions and destroyed equipment and ordnance until March 15th.

We got orders to go back to Iraq, I thought what! I thought the war was over. We crossed the border March 16, 1991, and went into a small town in Iraq as a show of force. We drove past an ammo dump on our way. It was huge, already blown up. All the battlefields were covered with blown up equipment, dead bodies and unexploded bombs. On March 18, 1991, we went to a firing range to shoot up our extra ammunition. I personally was able to fire my first service round, I don't recall how many total. March 19, 1991, we headed back into Iraq, supposedly going home. March 20, 1991, the U.S. shot down another Iraqi warplane, Saddam has been using mustard gas on his own people, we were at MOPP level 3 most of that day. I don't know exactly where we were in Iraq, but March 21st we headed 100 miles deeper into Iraq. April 1, 1991, we went back to the firing range, fired tables 6 and 7. Again I sat in the gunner's seat. April 8th, we were getting ready to go on a screening mission back through the area where the ammo dump was. April 10, 1991, found out we're headed to Basra, somewhere near the Euphrates River. April 12, 1991, things sure changed fast, we were headed out of Basra and back through Iraq, the same way we came.

April 14, 1991, after many friends were sick and avoiding it for a couple of weeks, I finally got sick, diarrhea, nausea, stomach cramps, tightness in my chest and headaches. The medic said it was a virus going around. So we believed that. We had quit testing for chemicals in February of '91. But we were still carrying our souvenirs, shrapnel, DU bullets from the A-10's and a bottom of a DU penetrator. Plus we all were using our ASFAB Cap ashtrays. April 16, 1991, we finally

crossed the border into Saudi Arabia...There were Saudi soldiers there to greet us, they treated us like we were heroes. That was the first and last time I was treated that way. No parade back in Germany.

Somewhere between April 12, 1991, and April 16, 1991, we went back through the same area where the night battle took place and where our brothers had been mistaken for Enemy and were killed. We spent the day there looking around, wondering what went wrong. Walking around looking at blown up vehicles, looking in bunkers, then and there we realized how major that night was. It was untouched from the first time we were there, except our friendly vehicles that were destroyed had been removed. We found partial DU penetrators, discarding fins and ASFAB caps everywhere. I had originally estimated 36 DU penetrators fired from each tank. But we were on a movement to contact, for 36 hours. Non-stop, no sleep...just refuel, reload ammo and move. We had been on a 200 kilometer attack through Iraq. Total kills for my Company, with 12 tanks to a Company, were over 60 tanks, 40 trucks, 20 BMP's and unknown numbers of troops and bunkers that were destroyed with DU penetrators. These numbers come from my daily diary, Sunday, March 3rd, 1991... By regulations, we were supposed to engage trucks and BMP's with Heat rounds or small arms. But we didn't, we shot DU penetrators 90% of the time and we also used them to shoot bunkers.

May 8, 1991, after being in the Syrian desert, Iraq, Iran, and Kuwait, all the way to the ocean shore and back. No baths, no water to brush your teeth with, millions of flies, treated water out of the river and diarrhea forever (it seemed), we were alive and finally at the King Khalid Military City. One hot shower before we go home. We had to turn in all bullets, discarding fins, ASFAB Caps and a couple of partial DU penetrators, which we threw in a trash dumpster at King Khalid Military City. We washed our tanks and totally cleaned them inside and out, then prepared to head home.

May 8, 1991, after an eternity (it seemed like) I finally stepped off the bus and saw my wife and daughter. Her first response was "I love you" and mine was, "how about those 5 boys you promised me"... My health was soon going straight down hill, I was feeling bad, teeth rotting and my wife was having numerous infections... Bladder, yeast, urinary tract. Her stomach was hurting every day and I went from 185 to 150 and from a 12 minute 2 mile, to barely making standard. My wife went from 125 to 90 lbs. The end of July, 1991, we finally had a glimmer of good fortune...my wife was pregnant with our second child. She miscarried a month later. I didn't know what to do, or who to turn to... The whole post was falling apart... 3rd Armored and 1st Infantry hated 2nd Armored, because we had shot and killed some of their men. Everyone was eaten up with guilt and about 30% of my Company was sick. There was no leadership or counseling, we were all fighting with each other, drinking too much and getting sicker as time passed. My wife Shawna was sick and hurting so bad, she was totally homebound.

I was discharged out of the Army on December 31, 1991. My records, medical and personal, were lost. My accomplishments had included Excellence in Armor, a Letter of Achievement from LTC Frank J. Gehrki, an Army Commendation Medal with V for Valor, an Army Service Ribbon Liberation of Kuwait Medal (from Kuwait), an Army Service Ribbon Liberation of Kuwait Medal (from Saudi Arabia), National Defense, Southwest Asia Service and a Valitorious Unit Citation.

My highest rank was Specialist E-4, promotable and held Corporal stripes for a short time. This was where I had planned and hoped for a future, I had become a model soldier before and during the war. But after the war was a different matter. I was discharged.

Summer of '92... We were glad to be back in Oklahoma. Shawna was hanging in pretty tough, but her health was going downhill fast. She had dizzy spells, irregular weight loss, but her main problem was her stomach. She couldn't go a day without feeling sick to her stomach. In September '92, my wife had to have 5 porcelain caps put in her mouth, because her teeth were (and still are) falling out. Along with Shawna's problems, I was dealing with chronic fatigue, weight loss, aching joints, respiratory and sinus problems. I went to the VA and saw the doctors. They would do some tests, give me some pills and send me home. Pretty soon, I had a very impressive collection of pills to take. At age 24, just out of the Army...an ex-12 minute 2 miler...I thought this was odd, but trusted the doctors, after all, they're the experts. I told them about my exposure to hazards in the Gulf, including DU and possible chemicals. I explained to the Doctor that I might be one of those guys with the so-called Gulf War Syndrome. His response was, you're working too hard, try to slow down. I worked an average 8 hour a day job, just like everyone else. But for me, after 8 hours, I could hardly walk because of swollen joints. So the doctor told me to slow down and I did, but this did not help me and meanwhile, my wife was getting sicker. But because we were told that they couldn't find anything wrong, we decided to start trying to have kids again... but nothing would work, so we figured that one of us must have a fertility problem.

In the summer of '93, Shawna and I were having severe health problems, at this point Shawna had been in and out of many emergency rooms. They would do tests and tell me she's not sick... Take her home. I was still being told by the Tulsa VA and Muskogee VA that there was no Gulf War Illness. I didn't know in 1993, that there was a Gulf War Exam... I still believed the VA and the doctors that said there was nothing wrong with me. I trusted their explanation. As for my wife, it was in 1993 that doctors started telling me that my wife must have some sort of mental problem, because she was so sick, without any sign of a problem. They did find out that she had chronic yeast infections, bladder and urinary tract infections. She then started telling me that my semen burned her. Shawna said at that point, "There's something very wrong, do you think something might have happened to you?" My reaction was, "No way! If anything was wrong, don't you think the VA doctors, whom I've put all my trust in, would tell me if there were any problems?"

In the summer of '94, I felt things were going better. I was promoted to a full time letter carrier with the U.S. Post Office. I thought OK, now we'll buy a house, be able to pay bills, forget our past problems and get healthy. We started going to a specialist for Shawna. This was new compared to all the emergency room trips we were having to make and felt that finally someone would be able to either give us an explanation and/or find the cause of her problems.

In the summer of '95, her weight was down under 90 lbs. At this point, she has 85% of the symptoms on the enclosed list. The VA has almost convinced me that my health problems are mental instead of physical. My medical records, which I have copies of, state, "I believe the patient has been coached and is trying to get increased evaluation." I'd pretty much given up on trying to get any medical care, because where do you go after they slam every door in your face?

Even non-service connected vets are supposed to get care for Gulf War ailments, but what are we going to do? How do we get any tests done, if they don't acknowledge any Gulf War Syndrome? My frustration was and still is that I've told them my ailments and where I hurt. I know what the doctor needs to check for, because I was there and I know what I handled and was exposed to and I'm sure there were a lot of unknown agents from the enemy that I also came in contact with, but he's got the education, he's the doctor and instead of saying this is wrong and this is what needs to be done...and that is to test for DU exposure and chemical and biological exposure, etc....but instead they do nothing but try and blame it on Post Traumatic Stress. They need to try and help these Veterans and their families. If they can transplant a heart, do brain surgery and find cures for all sorts of ailments and diseases, then why don't they run enough tests until they find out what we have? But the VA will not do tests, will not try to help, they act like they don't even care.

At this point in time, my performance at the Post Office is failing, but I'm able to put on a great show outside of the home. I was fearful of losing my job, because we had just bought a home and for once I thought that just maybe I could be man enough to overlook how I was feeling physically and try to get on with our lives. But by then, I was displaying ½ of the symptoms on the enclosed list, swelling joints, fatigue and memory loss, were the main ailments affecting my performance at work. I was skipping lunch breaks to keep up my deliveries, because speed and accuracy were an intricate part of a walking postman's route. There were times I couldn't remember what I was forgetting. I would get lost, put wrong mail in people's boxes and I would panic. Things got worse, more sick days, when before I never missed work. Before the Gulf War, Shawna and I were never sick. So I had no choice but to leave my $30,000 a year job, plus 2 retirements, plus insurance, to live on $462.00 a month. And that $462.00 is what we have lived off of for the past year. Our house payment is $500.00 a month, so we're already in the hole. Our friends and Shawna's family have paid all of our utilities. Any other bills go unpaid. We have no car insurance, no house insurance, creditors threatening to sue us and sometimes no gas to go to town or to the doctor. I now spend all my time taking care of my wife and my medical needs. My wife is totally homebound, can't drive and will pass out in heat and from strong scents.

The VA continues to deny any benefits (other than Post Traumatic Stress), any medical care (pertaining to my exposure) or any Gulf War Syndrome. I asked to go to a Gulf War Hospital for better testing, the VA doctor said OK then left the room and that was it! I asked later when I was going for further testing, the VA said your doctor feels you do not need it. I've volunteered myself and my wife for any special testing,

example: Radiation Exposure... My claim for exposure to DU was cancelled at the Tulsa VA.
example: Burning Semen Syndrome... I called Dr. Todd at the Muskogee VA. He gives the Gulf War Exams. He said, "I've never heard of that." I told hime it was VA funded. He said, "Sorry, I can't help."

I've been misdiagnosed, mistreated, abused, used, I've been lied to and cheated. If the VA thinks I'm in it for just the money, well I've got to tell you that there is no amount of money, not even a billion dollars, that could take back the mental and physical suffering my wife has been throught these last 6 years. If it wasn't for the Indian Program of the Cherokee Nation, we would be eating

out of the trash. Can the Government resolve this matter quickly and start helping us, or are we going to be forgotten?

Up until now, I trusted their doctors with my life, because during the war they would have been my lifeline, should I have been wounded. But the attitude at the VA now is that the only reason I'm trying to seek help is to get money. Well, I am not in a hand to hand combat war now, but I have been wounded, my body is crying out for help, it's deteriorating. This same organization who would have risked saving me during the war, should stand behind me and give me, my family, and the other Veterans suffering from Gulf War Syndrome, the same medical attention as they would have on the battlefield. Many have died since returning from the Gulf and we are fighting for our lives. Just as Vietnam Veterans exposed to Agent Orange fought for help, please don't wait until it's too late.

Thank you for your time.

Sincerely,
Michael Joseph Stacy

Attachments: Two pages
(List of Symptoms)

1. Heart palpitations
2. Tight Chest/Pain
3. Irritability/Flashes of rage
4. Problems with teeth/gums
5. Abnormal Headaches
6. Hard to swallow
7. Thick saliva/Phlegm
8. Chronic Fatigue
9. Short of breath
10. Don't participate w/ others
11. Difficulty concentrating
12. Hard to pay attention
13. Short term memory loss
14. Aching joints
15. Imbalance, dizziness
16. Ringing/roaring in ears
17. Tingling/Numb hands, toes
18. Metallic, yeasty taste
19. Stuffed in limbs
20. Reoccuring rashes/warts
21. Recurring flu/coughs
22. Cuts slower to heal
23. Abdominal pain, diarrhea
24. Hair loss, change of color
25. Swollen lymph glands
26. Red, watery eyes
27. Blurred/double vision
28. Eyelids twitching
29. Stuttering mom
30. Inability to finish sentences
31. Swelling of limbs
32. Weight swings up & down
33. Mushy/bleeding gums
34. Whitish coating on tongue
35. Difficulty finding words
36. Sleeplessness/insomnia

37. Hard time waking up ___ ___ _____
38. Nausea or vomiting ___ ___ _____
39. Increased thirst or hunger ___ ___ _____
40. Intolerant of exhaust/smoke ___ ___ _____
41. Intolerant of paint/solvents ___ ___ _____
42. Intolerant of cleaners ___ ___ _____
43. Intolerant of perfumes ___ ___ _____
44. Less capacity for alcohol ___ ___ _____
45. More sensitive to light ___ ___ _____
46. Night sweats ___ ___ _____
47. Crawling sensations ___ ___ _____
48. Teeth more easily chilled ___ ___ _____
49. Sensitivity to cold ___ ___ _____
50. Mouth/lip sores ___ ___ _____
51. Breathless when exercising ___ ___ _____
52. Suicidal thoughts ___ ___ _____
53. More easily spooked ___ ___ _____
54. Spouse with symptoms ___ ___ _____
55. Numbness/drooling ___ ___ _____
56. Mind on "fast-forward" ___ ___ _____
57. Weak and unsteady ___ ___ _____
58. No energy/motivation ___ ___ _____
59. Feeling of confusion ___ ___ _____
60. Hazy thinking ___ ___ _____
61. Frequent clearing of throat ___ ___ _____
62. Deteriorated night vision ___ ___ _____

Mr. SHAYS. Mr. Stacy, thank you. I would like to ask you. You did not read much of your testimony. On page 2 of the testimony I have, I have a typed sheet. Do you have that typed testimony?

Mr. STACY. No, sir. Sir, I am 40 percent disabled. I receive $467 a month. I left the Post Office after 3 years. My house payment is $500 a month. I do not even have money to drive or put gas in my car. We are literally starving to death. We receive no help from nobody. I was unable to get this typed. I was lucky to be able to get my written testimony typed and sent to you whenever I did.

Mr. SHAYS. So the testimony we are looking at, we retyped ourselves.

Mr. STACY. I believe so, sir.

Mr. SHAYS. Well, I just feel inclined to ask you to read what we have on page 2, the typed sheet. Do you have it? It starts on January 21.

Mr. STACY. Talking about on January 21?

Mr. SHAYS. I would like you to read that page.

Mr. STACY. On January 21, 1991, which, sir, I would like to say every bit of this came from my diary, is true, to the best of my knowledge. Some information may be incorrect, but everything written in my diary was written at that date.

"On January 21, 1991"—also a day I will never forget, my 22nd birthday—"we rolled toward Iraq. We prepared for war and waited and waited and waited and waited and waited. Then it started, February 16, 1991. A company from our brigade made contact. They shot a truck and a berm with TOW missiles and had a couple of small gun fights. February 17, war hit home, and we lost a Bradley to enemy fire, a rocket-propelled grenade. Two killed, four badly wounded. The bombing continued, so heavy at times, you could not sleep for days.

"We crossed the border for good on February 24, 1991. When we crossed, we were in MOPP–4; after we crossed, we went to MOPP Level 0, and then repackaged our MOPP suits, the same ones we opened in base camp back in early January 1991, and had worn on numerous training expeditions and had repackaged each time, unaware that they were useless after their initial opening. On the same day, February 24, 1991, we came under attack from enemy artillery, some of which exploded in the sky over us and created a white cloud, which then disintegrated over us.

"We had thought it was a marker, so we moved. This continued all day until our artillery knocked them out for good."

Sir, would you like for me to continue?

Mr. SHAYS. Yes, I would like you to continue.

Mr. STACY. "February 25, 1991, we started making contact as we were moving toward Iran. February 26, 1830 hours, we rolled out onto a small battlefield, a place I will never forget. There were trucks, tanks, BMPs, and troops. We freely engaged the enemy until 4:15 a.m. I heard 'Cease fire, cease-fire, cease-fire. There are friendlies to the front.' Before the third 'cease-fire,' we had already engaged them, shooting an American M1A1 from the 3rd Armored Division. We shot them six times. We provided rescue efforts, but their ammo was cooking off, exploding, so we abandoned any further rescue, backed up 100 meters, set up a perimeter, and waited until dawn.

"That night, we lost an M1A1 and a Bradley Fighting Vehicle tofriendly fire. Eight buddies from my battalion were dead, and wewere responsible for killing them."

Would you like me to continue, sir?

Mr. SHAYS. If you do not mind. This is your testimony, and I just hink it is important.

Mr. STACY. "The first depleted uranium penetrator we fired was on January 27, 1991. We fired Sabot and Heat." "Sabot" is a depleted uranium penetrator, and the "Heat" is high explosive. "We fired three of each to battle sight our main gun. After we fired our first Sabot round, we knew then the DU penetrator was the round of choice. I think a full combat load for an M1A1 Heavy is 56 rounds. There is a certain load plan you follow, so many Sabot and Heat in your main ammo storage area and so many of each in your secondary storage area. I was the loader on my tank, and after we fired our first DU penetrator, our platoon switched the ammo. Put all DU penetrators in the main storage area, put the heat rounds in secondary storage. We were told, I quote: 'Shoot 'em while you got 'em.' The DU penetrators were so devastating that we used them for everything, tanks, trucks, light-armored vehicles, bunkers—everything but the troops. We found out very fast that the depleted uranium penetrators were 1,000 times more devastating than we expected. When a bunker was shot with the DU penetrator, just the percussion from the round will kill any troops in the area. We wanted to shoot the good stuff and as much as possible.

"We were at war, with the best equipment out of all the Coalition Forces. No law, no rules, engage at will. My Platoon alone," these numbers are all estimated; no numbers are exact. I was just a private in the Gulf; I only knew what I was told. "Fired approximately 120 DU penetrators, with 4 tanks per Platoon. Thirty penetrators fired per tank, plus another 3 penetrators to battle sight our main gun, on January 27. Plus another 3 penetrators on January 28, 1991. That is approximately 36 depleted uranium penetrators each tank fired. There are 12 tanks per Company, 4 Companies per Battalion, we had two battalions on line, the 2nd Armored Division and 3rd, that is a total of 96 tanks. Plus we were attached to the 1st Infantry Division. They are a mechanized battalion with unknown numbers of Bradleys, which fired 30-millimeter DU rounds. So as you can well expect, we were constantly in contact with this ammo."

Mr. SHAYS. Thank you, Mr. Stacy.

Mr. STACY. Thank you, sir.

Mr. SHAYS. Is there anything else of your testimony you want to read us, or shall we go to Staff Sgt. Zeller?

Mr. STACY. No, sir. Thank you.

Mr. SHAYS. Thank you. Sgt. Zeller.

Staff SGT. ZELLER. Yes, sir. Gentlepersons, this is about a grunt's life, blood, sweat, and tears. Some of you up here will not understand this. I am not a good speaker, but was blessed with the gift of gab. I thank you for the opportunity to speak, and I hope the money spent for me coming here will change my present position.

Possible causation: Service in the Gulf war due to combat in the theater. Nuclear radioactive weapons and atomic energy plants. Depleted uranium, microwave technology kill zone creating electric

storms. We had electric storms in the desert, sir, that the lightning went this way—OK—and that is explainable.

Destroyed power plant at Quasyr Hammid and Al Anbar Atomic Research Space Center. OK? And that is documented. Biological weapons: cholera, anthrax, botuluum microtoxin. Chemical weapons: cyclo sarin/sarin, soman, tabun, mustard, blister blood. Prophylactic drugs, serums, and vaccines, malarian drugs. PB (pyridostigmine bromide); it causes damage to number 6 chromosome. My children are susceptible at third generation to come out being deformed.

Specific oil-based adjuvants: NTTTP, squalene, Vax Syn, Vacinae, Type C Retrovirus, Canary Pox Virus, Glycoprotein 120 and 160 Antigens; oil fire environmental factors.

They are now recognizing Exxon Valdez Syndrome, leshamenasis, ultraviolet-light overexposure, malaria; Mohammed's Revenge, which is a rare bacterium that is very pathogenic.

Questions: Plausible denial, true or false? I am it, sir. I have been in SOCOM, Special Operations Command. Something is wrong when all the resources are spent on history, how we got sick, instead of cures for it, if any. Diagnoses have been made, but no treatments are being utilized or considered.

I would like to point out that Congress is being wrongfully briefed by the leadership of the investigation. Could agencies in the Government utilize national security titles to develop and research without notice? Could this national security title provide them protection from within to continue the facade?

From the registration data base in California to the investigation team in Virginia, what is their purpose and command? At the end of the command, why is there a logistics expert and not a doctor of medicine? Is the data base to cure or to count, sir? Why are they not using subject matter experts like us Gulf war sick and wounded to get the most eyewitness accounts? I volunteer for the investigation team.

Can it be that the truth or the cure is intentionally being ignored for the purpose of protocol protection? Are the studies conducted for a possible cure to our disease process or for reinventing the intelligence already available? Does research take time, and is this a delay so we never find a possible cure or remedy? Can the cure be purposely hidden as not to expose the cause of the effect? The cure is the effect and will inevitably expose the cause to this Gulf War Syndrome.

Is the cause reflective of some unethical decision or practice? We have come back on our word many times in the investigation process. Why not consider the possibilities? Cause: If your word is changed several times, either you are lacking knowledge or intentionally diverting the information. Can we hold supposed national security in such high degree as to allow our brave soldiers denied causation? National security is to protect the Nation. It seems as though we are taking individuals' protection to a new height.

Will this protection continue until every last vet is dead and gone, or will the priorities be recognized? Human life is too precious a resource to be sacrificed for the good of a few men's securities. What gives the person not in harm's way the power to make

these decisions? Considerations: If they had done this, it might have been better.

This is brief, due to time limit on testimony. Most Gulf war veterans are not actually interested in cause of illness, just being cured or at least being acknowledged as having the physical illness. The below items may make the search for a cure more readily available. We know a lot of these things about this illness, but most of it is considered not conclusive or left at that. We need to take restrictions off valuable information and consider it for cause.

All I want to do is to see my grandchildren grow up, because this illness is not over with me. And my five boys at least deserve this. Koch's Postulate: Define the pathology. This is doctor talk, sir, that I looked up. Define the pathology. Isolate suspect ideology agent. Reintroduce that agent into host. Reproduce the pathology. Naturally, we cannot do this with humans; however, we can back track and use information which is out there to build our case.

Cardinal rule in science: "Occam's Razor." Entities should not be multiplied unnecessarily, or, more succinctly, the simplest of competing theories be preferred to the more complex. Definitions: What is the disease process? Systemic autoimmune disease process; neurological disease process. Environmental illness infectious disease. Chemical-characterized disease process. Biological-characterized disease process. Radiological characterized disease process.

What do we know? All forms of immune disease are being recognized.

Next one. From the central nervous system two peripheral neuropathies are being recognized: cholinergic crisis due to PB tablets. That also happens with nerve agent. Oil well smoke and spill Exxon-Valdez Sickness is being recognized. The chemicals——

Mr. SHAYS. Sergeant, let me just interrupt a second.

Staff SGT. ZELLER. Sir, yes, sir.

Mr. SHAYS. I want to make sure that I learn from what you are saying. I am having a hard time knowing—I got the first part of your testimony. I am in left field now in this part of your testimony. Tell me your point in going through this list.

Staff SGT. ZELLER. The point is, sir, on the definitions, for example, like, I went all the way up to Walter Reed——

Mr. SHAYS. Right.

Staff SGT. ZELLER [continuing]. And they said, well, this sounds like lupus, but they did not do anything for it. Well, I went home and did my homework, sir, and I read lupus; and what I have wrong with me, sir, is to a "T" lupus. And there are ways to test for lupus and find out for sure, and why haven't they done it, sir?

Mr. SHAYS. OK. It is important for me that you be allowed to continue and do what you want to do.

Staff SGT. ZELLER. Sir, yes, sir.

Mr. SHAYS. And then I just need to tell you what I think it will be helpful for the committee afterwards, but maybe I could tell that to you now as well. We need to know a little bit about your experience in the Gulf because we have others who testify, we are trying to see where there are similarities.

I mean, for instance, the first two witnesses talked about the alarms going off. The military has consistently told us the hundreds, if not thousands of times, the alarms went off, they were

false; and yet we have people who will come to this committee, sometimes off the record, and say that is simply garbage, that those alarms were not false; they detected low-level chemicals. And we are building a case, and we need to make sure we are doing that.

It is important for me that you continue because this is important to you, and, therefore, it is important to me. Afterwards, I am going to have you come back and describe a little of your experience. OK?

Staff SGT. ZELLER. Sir, yes, sir.

Mr. SHAYS. Feel free to continue.

Staff SGT. ZELLER. Radiological characterized disease process. What do we know? All forms of autoimmune disease are being recognized, from central nervous system to peripheral neuropathies are being recognized. Oil well smoke, chemical chiasma, as well as isolated reports in theater not considered official are being recognized. Rad Haz Al Anbar, Quasyr Hammid Atomic Power Plant, and depleted uranium 235 are now being recognized.

Quasyr Hamid, sir, was an atomic energy plant that was bombed by the Coalition. Cholinergic crisis due to pyridostigmine bromide also nerve agent. And it talks about the receptors and how they work in motors, causing contractions in the muscles, secretion-causing glands to secrete. I have got all these things, sir. Inhibitory causing most organs to become quiescent. Well, sir, this diarrhea and stuff that we have that is really gross, well, the intestinal tract becomes quiescent, and that is why we have this happening to us.

And it goes on and goes on, and then final on this, before I get to who I am, the chain of command to the Commander-in-Chief, I have solicited them, sir, and I thank you so much for letting me come here because I have not gotten anything but a wall that I cannot climb, sir.

Another thing, sir, is it talks about this cholinergic crisis, and it talks about the body, and I have got military reports, the cover sheets for them, and it says that if you take pyridostigmine bromide for more than 8 days constantly, and I took over six packs of 22 or 24 in a pack of those pyridostigmine bromides because they told us to take them and never told us to stop, so——

Mr. SHAYS. Sergeant, they did not tell you; they ordered you to take them.

Staff SGT. ZELLER. Yes, sir. Yes, sir. They said, you do it, or you die, that type of order. The 101st is expecting no mercy, sir. But it says here, body will dysfunction; it causes permanent injury through the blood-brain barrier, and it says it in the reports.

Sir, I just want to finish this, and I want to say that this Iraqi protection prisoner program, taxpayers' money, I regret to be treated like the enemy. I watched C–141's, and I watched Iraqis get on the aircraft and come home, here in the United States, sir. OK.

This is only the beginning of the information out in the field. God help us and our families. P.S.: We are like Teenage Mutant Ninja Turtles, if you are familiar. I have five boys that teach me about them.

Sir, my name is Mark James Zeller, and I went through basic training in 1986, and I got my Aircraft Armament School in 1987. I went from there, in February 1988, to Fort Bragg, NC, and was

assigned to 1st SOCOM. I participated in Operation Prime Chance I, Prime Chance II, Praying Mantis, and Earnest Will, Persian Gulf-Kuwaiti Oil Tanker Security Force. April 1989 to present day, Fort Rucker, Delta Company, 2/2nd 229th. I participated in Operations Desert Shield, Desert Storm, Desert Calm.

Sir, these are all in the same geographic area. My involvement in this geographic area dates back to the late 1980's. While assigned at Fort Rucker, NC, we assisted Kuwait and Iraq with their oil flow to the rest of the world, because the common enemy was Iran. The common enemy, Iran, discontinued their actions after we built Iraq up with military might, but Iraq changed their minds after defeating Iran. The operations I list above are continuations to the present day to include this committee meeting today.

The Persian Gulf war is not officially over, sir. I do not know if you realize that. My tour of duties at Fort Bragg were temporary duty missions with 90-day intervals. During this time, I lived off the economy and only got sick from Rift Valley Fever from eating uncooked lamb meat in a souk, or ancient, mall-like area, downtown.

I was honestly protected less from the environment during this mission because it was all stuff provided by host nations. What I mean is USDA and grading of health is not as controlled, and green tracers were a fact from the Iranian Government.

So why do I get only Rift Valley Fever and come home well as to be expected? In addition, PTSDs should have surfaced then under stress of Special Ops. Why am I now sick as can be with Gulf War Syndrome?

Let me try to explain my maneuvers and experiences now during Desert Shield and Desert Storm. Duty: Aircraft Armament Technician, AH–64 Apaches. Assigned, 2/229th Attack Helicopter Regiment, Fort Rucker, AL, June 1990, aka 2/101st AHR after deployed. OPCON to the commander of 101st Air Assault Division, Fort Campbell, KY, August 1990, via deployed to Kingdom of Saudi Arabia. Arrived, Dhahran International Airport, August 1990. Moves—"moves," sir, meaning going from one FOB to another of forward operating base.

Moves, FOB Eagle I or King Fahd International Airport between August 1990 to December 1990. Explored the FOB Tranquility Area, readied all aircraft for deep attack mission. Key personnel got wrist watches with no face on them and strange, smoke-screen operation was conducted at King Fahd International Airport.

Sir, that wrist watch, I do not know this gentleman. I did not know him until we got in the hotel, and we just started gabbing. A long story short: He has the same claims I have. This wrist watch, on the back of it, they are told not to take them off. I peeled it back on one of the females that was in our unit, a medic, and it said "PRC Radiac 27" on the back. OK? So I know it is a Radiac meter.

And there were people that came along, and they had this, like, board thing they put over it, and they were able to register whatever it was, but they never told us what the registrations were, what was going on, and so forth and so on. It was the circle with the "A" in the middle, Third Army. It was a test-activity group.

Smokescreen operations was conducted at King Fahd International Airport. The wrist watches were Radiac monitors labeled on back, and why we needed them, I do not know. They were picked up from key personnel, March 1991, and the smoke screen is still a mystery to all of us to this day. We were only told it was something they were testing. I got really sick after being at FOB Tranquility, so for the first time I came back to the airport with a 104 fever, sweats, chills, and loose stools, if I may say that, sir. It is pretty disgusting. I got a real bad reaction to medicine given by a flight surgeon after being seen.

FOB Tranquility or West Nariya, North AxZil-Fi-Frontier, December 1990. I started taking pyridostigmine pills for the prevention of nerve agent poisoning in the event Iraq chose to use it. I was only told it would help me survive, not that it could have side effects or kill you and that it was experimental. I took more than 60 of these pills.

Sir, I must also add about those pyridostigmine situations, there were people dying, dropping dead on Taplin Road, and if you cannot find records of it, I cannot either, but I remember the National Guard unit in Florida that had the dolphins on their Blackhawks, and whatever that regiment is, you will be able to figure it out, and they will be able to tell you about these people.

Mr. SHAYS. Let me just say, that is now part of your testimony.

Staff SGT. ZELLER. Roger that, sir. A radio message——

Mr. SHAYS. Sergeant?

Staff SGT. ZELLER. Sir?

Mr. SHAYS. I do not want you to be casual.

Staff SGT. ZELLER. Sir, yes, sir.

Mr. SHAYS. You were saying "dropping dead." You just mean collapsing out of fatigue?

Staff SGT. ZELLER. Well, cholinergic crisis, sir, causes the nerves——

Mr. SHAYS. No, no. That is not what I asked. You made a statement that people walking on the street——

Staff SGT. ZELLER. Their hearts stopped, sir. That is what they said. They said their heart stopped, is the way they died.

Mr. SHAYS. OK. And you are saying that you know for a fact that they were dead?

Staff SGT. ZELLER. Yes, sir.

Mr. SHAYS. How many soldiers are you talking about?

Staff SGT. ZELLER. What I was told, down the Taplin was, like, 125 people had adverse effects from these PB tablets.

Mr. SHAYS. What I am going to ask you to do is we are going to have a vote, and we are going to have a second vote after. I am going to ask you to finish your testimony in the next 3 or 4 minutes, and then we are going to go vote, and then we are going to ask questions.

Staff SGT. ZELLER. Sir, yes, sir. A radio message came down, go to MOPP Level 4, full protective gear; SCUD has been fired in your vicinity and is down wind, contaminant to your vicinity. That night, a few M–8 alarms went off, but it was told to be all clear. I began to leak blood out of my ears on a pillow every morning, just spots; nosebleeds; lip began to split. Sir, my lip split all the way up to my nose, and the center of my tongue started splitting. I do

not know what it was, sir, but it happened. They tried to say it was dehydration, but I do not see how it was dehydration for that to be happening.

Headaches, pounding in the ears, eyes sore. My hair felt like something was pulling it out, and I urinated a lot. I hope I am allowed to say that. FOB Eagle II, or King Khalid Military City, Al Qaysumah/Hafar Al Batin-Frontier, January 1991. On guard duty around midnight to the west of our position SCUD missile is shot down.

Next morning, before stand-to, some more M–8 alarms go off, but it was ignored. Also received these injections said to be benefit of my health. One was called gamma globulin, and the other two were coined "NUC Juice" and "Bot Tox/Anthrax Vaccine." I stepped up to receive my shots with shot records in hand, and only GG was annotated, so I objected to the other two shots. The flight surgeon took me immediately to the commanding officer, who showed me the blue book and told me, "Take them or be court-martialed."

I felt really sick after the shots, became really tired, and could sleep through anything, including allied bombing over the border. FOB G–Day, or North Samah/South Al Julaydak-Frontier, January 1991. This is a rally point for ground convoys to assemble for insertion into Iraq.

MSR Dakota to Virginia. Many practices were held prior to the actual day. We were instructed to go to MOPP Level 4 because we will be entering a known contaminated area. A man was seen on a fence who died instantly, and animal herds, no flies around, alive. SCUD missile found next to road south of Samah. M–8 alarms connected to vehicles went off and paper was changing colors. Drive on, was the order. I began to get lesions all over my body. I told by medical personnel it is because of the lack of hygiene under these conditions.

FOB Cobra, or North Tukayyid/North Quiban Layyah/South Al Busayyah.

Mr. SHAYS. Sergeant, I am going to ask you to pick the best, most important part of what remains because we do need to——

Staff SGT. ZELLER. Yes, sir. Upon arriving in Cobra, we received artillery that produced proofs of off-white dust. Air Force A–10 aircraft, 10 foot off deck, flew overhead after calling rear for support and leveled a flat-top mountain north of Quiban Layyah. I began to have pinkish conjunctivitis, blurred vision, thick spit, tasted metal, no hunger, ears, fingernails bled. I slept a lot when not working, and hands and feet felt like ants crawling on them.

I had an incident where I was driving, could see, hear, et cetera, but could not move all of a sudden. I ran into a sand ditch, and hitting the steering wheel shook me out of it. The prisoners that I was able to talk to said they were sick and tired. I was starting to worry at this point because it sounded like they had what I had. Alarms and paper were turning colors during the whole time.

Fire Base Viper——

Mr. SHAYS. Sergeant, you are not going to be able to read the whole thing through, so just pick the part that you think in the end——

Staff SGT. ZELLER. OK. This is Dhahran/Kasmeeyah, and there were more dead animals, POWs, very sick ones, I may add, MPs

holding them, with inoperable radio and no water or food, left behind by their unit because movement; MPs told to stay and guard them by their commander. We gave them what food and water we had and called to the rear National Guard Fills—that means people that were put in their unit were helping out convoy police. On the road past that, Tallil, were bunkers with SCUD trucks next to them on the left.

When we were coming back out of the country, sir, at Fire Base Cobra, where we came back to the same fire base we came from——

Mr. SHAYS. Excuse me. We are going to have to go. We have 3 minutes before——

Staff SGT. ZELLER. OK, sir.

Mr. SHAYS. I am going to interrupt you and just say one last point before we go. During Watergate, when Martha Mitchell was describing absurd things that were happening, everyone thought she was crazy, and everyone around her was sane. When she talked about all these crazy things that were happening around her, she happened to have been right, and I just say that to some of our audience who may hear some things that sound a little strange, but may in the end it may be very right. We are going to recess.

[The prepared statement of Staff Sgt. Zeller follows:]

OFFICIAL TESTIMONY

MARK JAMES ZELLER - 263-69-9623
MILITARY HISTORY - Nov. 1986 - Feb. 1987 Basic Training
Feb. 1987 - Feb. 1988 AIT 68J 10 X1 AIRCRAFT ARMAMENT
Feb. 1988 - APR 1989 FT BRAGG, NC Task Force 118 SOCOM
OPERATIONS: Prime Chance I & II, Preying Mantis and Earnest Will (Persian Gulf Kuwaiti Oil Tanker Security Force)
APR 1989 - Present FT RUCKER, AL D Co 2/229th AHR
OPERATIONS: Desert Shield, Storm & Calm (Liberation of Kuwait)

GENTLE PERSONS,

PRIME CHANCE I & II, PREYING MANTIS, EARNEST WILL (Oil Tanker Escort)

My involvement in this geographical area dates back to late 1980's. While assigned at Ft Bragg, NC we assisted Kuwait and Iraq with their oil flow to the rest of the world because of the common enemy, Iran. The common enemy, Iran, discontinued their actions after we built Iraq up with military might but Iraq changed their minds after defeating Iran. The operations I list above are a continuation to present day to include this committee meeting today. My tour of duties in FT Bragg, NC were of a temporary duty mission with 90 day intervals. During this time I lived off the economy and only got sick from Rift Valley Fever, from eating uncooked lamb meat in a Souk or ancient mall like area (downtown). I was honestly protected less from the environment during this mission because it was all stuff provided by host nations. What I mean is USDA and grading of health is not as controlled and green tracers were a fact from the Iranian Govt. So, why do I get only Rift Valley Fever and come home well as to be expected. In addition, PTSD should have surfaced then under the stresses of Special Ops. Why am I now sick as can be with "GULF WAR SYNDROME" ??? Let me try and explain my maneuvers and experiences now during Desert Shield, Storm and Calm.

DESERT SHIELD, STORM, and CALM (Liberation of Kuwait)

DUTY- Aircraft Armament Technician AH-64 APACHES
ASSIGNED- 2/229th Attack Helicopter Regiment FT Rucker, AL JUN 1990 A.K.A.- 2/101 AHR after deployed
OPCON TO- CDR, 101st AASLT DIV FT Campbell, KY AUG 1990 via DEPLOYED to Kingdom Saudi Arabia
ARRIVAL- DHAHRAN INTERNATIONAL AIRPORT / AUG 1990
MOVES: FOB EAGLE I or KING FAHD INTERNATIONAL AIRPORT / AUG 1990 - DEC 1990

Explored the FOB Tranquility area readied all aircraft for deep attack mission, key personnel got wrist watches with no face on them and strange smoking screen operation was conducted at King Fahd International Airport? The wrist watches were radiac monitors labeled on back and why we needed them I do not know. They were picked up from key personnel MAR 1991 and the smoke screen is still a mystery to all of us to this day. We were only told it was something they were testing . I got really sick after being at FOB Tranquility so for the first time I came back to Airport with 104° fever, sweats, chills and loose stools. I got a real bad reaction to medicine given by flight surgeon after being seen.

FOB TRANQUILITY or W. NARIYA / N.AzZIR.FI- Frontier / DEC 1990

Started taking Pyridostigmine Bromide (PB) Pills for the prevention of nerve agent poisoning in the event that Iraq chose to use it. I was only told it would help me survive not that it could have side affects or kill you and that it was experimental (took more than 60 of the pills). A radio message came down, "Go to MOPP 4 full protective gear SCUD has been fired in our vicinity and is downwind. Contaminant to the vicinity". That night a few M-8 alarms went off but it was told to be all clear. I began to leak blood out my ears on pillow every morning, just spots, nose bleed, lip began to split, head aches, pounding in ears, eyes sore, hair felt like something was pulling it and urinated a lot.

FOB EAGLE II or KING KHALID MILITARY CITY / AJ QAYSUMAH / HAFAR AL BATIN- Frontier / JAN 1991

On guard duty around midnight to the west of our position a SCUD missile is shot down and next morning before "stand to", some more M-8 alarms go off but it was ignored. Also, received these injections said to be for the benefit of my health. One was called Gamma Globulin. The other two were coined "NUC Juice" and "Bot Tox / Anthrax Vaccines". I stepped up to receive my shots with shot records in hand and only G.G. was administered so I objected to the other two shots. The flight surgeon took me immediately to the commanding officer, who showed me the blue book and told me take them or be Court-Marshaled. I felt really sick after shots became really tired and could sleep through anything including allied bombing over the border.

FOB G DAY or N. SAMAH / S. AI JULAYDAK- Frontier / JAN 1991

This is a rally point for ground convoys to assemble for the insertion into Iraq MSR Dakota to Virginia. Many practices were held prior to the actual day. We were instructed to go to MOPP 4 because we will be entering a known contaminated area. A man was seen on a fence who had died instantly and animal herds (no flies around alive), SCUD Missile found next to road south of SULMAN. M-8 Alarms connected to vehicles went off and paper was changing color, drive on was the order. Began, to get lesions all over body and told by medical personnel its because of the lack of hygiene under these conditions.

FOB COBRA or N. TUKAYYID / N. QUIBAN LAYYAH / S. AI BUSAYYAH- Frontier / JAN 1991 - FEB 1991

Upon arriving at Cobra we received artillery that produced poofs of off-white dust. Air Force A-10 aircraft ten feet off deck flew over head after calling rear for support and leveled a flat top mountain north of QUIBAN LAYYAH. Began, to have pinkish conjunctivitis, blurred vision, thick spit, taste metal, no hunger, ears fingernails bleed, slept a lot when not working and hands / feet felt like ants crawling on them. Had an incident where I was driving; could see, hear, etc., but could not move all of a sudden. I ran into a sand ditch and hitting the steering wheel shook me out of it? The prisoners that I was able to talk to said they were sick and tired. I was starting to worry at that point because it sounded like they had what I had! Alarms and paper were turning color during the whole time there.

FOB VIPER or E. TALLIL / S. Al HAMMAR / W. SAFWAN- Frontier / FEB 1991 - MAR 1991 FOB COBRA

On our way to Viper up MSR Virginia just before getting to Al BUSAYYAH seen dead animals and POW's (very sick ones I may add). MP's holding them had inoperable radio and no water or food left. Left behind by their unit because movement, MP's told to stay and guard them by their commander. We gave them what food and water we had and called to the rear (National Guard Fills-Convoy Police). On, the road past Al BUSAYYAH before TALLIL there were bunkers with SCUD trucks next to them on the left. TALLIL was pothole bombed down Hwy 8 to cut off point in Al BASRA more dead animals were found. At FOB Viper minutes before cease fire was made to the NW of our position two missiles were launched and we called it in. After the cease fire we spent a couple of weeks at FOB Viper then to fall back to FOB Cobra to protect the Kurdish people of Iraq.

FOB COBRA or N. TUKAYYID / N. QUIBAN LAYYAH / S. Al BUSAYYAH- Frontier / MAR 1991

Spent a couple of weeks in place to have a show of force to support the Kurds. We began to see civilians and it wasn't a pretty sight. Women and children mostly sick and thin looking, looked crippled most of them. Still, had all these weird medical problems with no reason ourselves.

FOB EAGLE II / FOB TRANQUILITY / PASS and REVIEW RHIYADH / DHAHRAN / MAR 1991 - APR 1991

A few days in each place on our way to Dhahran. Spent a few weeks there for decontamination. I was on the cleaning crew of a few vehicles, and I asked why their being so picky about cracks and crevices they're only trucks and helicopters? The officer told me, we have to get all the spores out of those vehicles so they don't go home with us. Back home continued to be sick but found it better to weigh it out rather than get ridiculed or maybe worse.

EXPERIENCES FROM MEDICAL SERVICES

For the next year after being back home sick with very bad cold it would seem. Doctors make statements about me riding sick call. So, I become more aware of my problem:

APR 1991 - JUN 1991 VERY SICK WITH "FLU"
-Very sick and given Tetracycline and told I'm coming here to Lyster Hospital too office
-I watch buddies begin to receive MEB's and kicked out of military for sickness with no diagnosis or care.
-Begin to duck head and suffer with my illness for fear of being kicked out for it.

JUL 1991 FILL OUT DA 4700: Southwest Asia Demobilization/Redeployment Medical Evaluation
-Filled this form out to the best of my knowledge medical personnel stated its only for database no significant record
-First clue to the importance of my sickness.

AUG 1991 - AUG 1995 LEARN TO LIVE WITH MY SICKNESS
- given Aspirin, Tylenol, Motrin and cold and cough medicine to pacify symptoms.

SEP 1995 BETHESDA NAVAL RESEARCH CENTER CALLS TO FORCE ME IN CCEP PROGRAM
-Was forced to come forward or be held accountable for with holding vital information.

OCT 1995 - NOV 1995 I CALLED CCEP REGISTRATION TO ENROLL 1(800) 796-9699
-Was enrolled and awaited my letters in the mail for registration verification.

DEC 1995 GET PHASE I FIRST APPOINTMENT, SURVEY
-Begun to get bad attitude from chain of command due to news media reports of "No such thing as Gulf War syndrome"
-Appointment was a lame excuse for symptom gathering survey.

JAN 1996 GULF WAR REPORTING LINE CALLS GIVE MY RECOLLECTIONS. ENROLL FAMILY
-Gave my statement of what I encountered in the desert.
-Gave my family up to the registry for survey.

FEB 1996 - MAR 1996 SEE CLINIC DOCTORS (But only for what the non-specialist refer to the specialists)?
-Internal Medicine Doctor calling the shots for specialized clinic doctors.
-If the specialized doctor sees something, he should be able to investigate without further permission.

APR 1996 I CALL CCEP REGISTRATION SUPERVISOR TO TELL OF DR. PROHIBITED CARE
-Doctor stands in the way of progressing through Phase II.
-Doctor states there are no appointments available at this time.
-I check with Keesler, AFB and find that their needing more participants.
-Called Supervisor and told her the problem, she called doctor and Keesler, AFB I was found to be truthful

MAY 1996 FINALLY GET PHASE II FIRST APPOINTMENT (Still non-specialist referring)?
-My Doctor still calling shots for the specialized fields of medicine in Keesler, AFB.
-Doctors at Keesler, AFB admit things are wrong but your doctor referred you for this and this only.

JUN 1996 - JUL 1996 GOING TO APPOINTMENTS BUT NOT REFERRALS FOR SYMPTOMS
-The referrals were for bogus non-related illness or things that are not manifested.
-One trip to Keesler, AFB was for one week and only one hour a day was occupied by hospital.

AUG 1996 PAPERWORK IS INITIATED FOR PHASE III, WALTER REED HOSPITAL
-Certain suggestions were annotated by my Lyster doctor that the need to go to Walter Reed was unnecessary.

SEP 1996 - OCT 1996 ATTENDED PHASE III, SPECIALIZED CARE CENTER WALTER REED HOSPITAL
-Arrived and received psycho evaluation without commanders knowledge or my permission.
-Phase I and II psycho evaluations were completed and were diagnosed as normal.
-Phase III was supposed to ONLY be further physical symptom specialized medicine.
-Told I would not gain any assistance physically without psycho evaluation completed.
-Misdiagnosed Post Trauma Stress Disorder PTSD, Psychosomatic Disorders and Munchausen Disease
-Found positive porphyria after three weeks of complaining about no physical testing.(Porphyria test results positive)
-Recreation, Art, Coping, Counselling Therapies were over emphasized...
-I ask to see Gen. Blanck, but was denied.
-Doctors all committed gross acts of criminal negligence and violation of Hippocratic oath vs. institution.

NOV 1996 PENSACOLA NAS HOSPITAL TO NUEROLOGY FOR SYMPTOMS OF PORPHYRIA
-Checks Electro Motor Cirum sees decrease in sensory nervous system and discusses symptoms as signs of Porphyria.
-Doctor at Lyster said Pensacola sent you home with "suggestive" diagnosis not confirmed.
-At this point I already had two Porphyria tests confirmed positive.
DEC 1996 - JAN 1997 SPEAK WITH DOCTOR IN INFECTIOUS DESEASE, GAIN SUGGESTIONS
-Suggests that I go see Endocrine doctor at Eisenhower hospital.
FEB 1997 - MAR 1997 GO TO EISENHOWER HOSPITAL TO ENDOCRINOLOGY AS PER INFECTIOUS DESEASE
-Checks pituitary gland MRI and sees Empty Sella Syndrome.
-States it possibly happened from gland tumor infarction or something 'punched' on my brain.
APR 1997 DOCTOR AT LYSTER, DISCUSSED MEB FINAL EVAL. SUMMARY I QUESTIONED IT
-When he stated his findings, I said what about the other diagnosis listed in my records.
-He said, they're not text book perfect to a T. The best thing for me to do is find good doctors on outside and fight this.
-I replied, I can't and won't. The U.S.Army is obligated to provide me the best care not pawn me off to private sector.
-He said he was done and was not willing to discuss anymore after stating he was just a measly Internal Medicine Doctor and, I was never going to be satisfied with the findings because I'm frustrated.
-I told him I was and am betrayed by the Army's lack of commitment to it sick and wounded.
MAY 1997 RETURNED TO PENSACOLA NAS HOSPITAL FOR ACQUIRED PORPHYRIA DIAGNOSIS
-The Doctor said I acquired Porphyria due to Gulf War, but other things are still challenging.
JUN 1997 RECEIVE SUMMARY DRAFT FOR MEB
-Medical summary draft for MEB was a lame piece of work for what I have to experience in my life.

I pray this meeting will shed some light on the situation Gulf War Vets and their families have had to deal with. Thank you for your time and consideration Gentle ladies and men.

[Recess.]

Mr. SHAYS. My hope is that we can have a candid dialog, in which we will learn more about your experiences in the hope of coming to this ultimate goal of properly diagnosing, effectively treating, and fairly you and our other veterans. That is the motive of this committee.

Now, we want to properly diagnose, effectively treat, and fairly compensate. Now, some of what I have heard today, I understood; some of what I heard, I did not quite understand. One thing, though, I will say to you: It wrenches my heart to think that you have so little faith in the DOD, the Department of Veterans' Administration, in the doctors that have looked at you, that you then start the process of trying to figure out what is wrong with you because you do not think the doctors are, and that wrenches my heart.

And we have had other veterans who have come and testified. When I was going to vote—I have come to this conclusion. I am tired of the DOD telling us that the alarms were false alarms. I have just come to conclude that I cannot accept that anymore.

And the reason is that those alarms did not go off basically before the war; they went off during the war. They did not go off when they were exposed to all other things, but when the war started, these alarms started to go off. I cannot reconcile the loss of data, and so your testimony of the alarms going off and so on are not unimportant to me. I am not going to accept anymore the DOD position that these are false alarms. I just do not accept it.

Col. ROMAN. Thank you.

Mr. SHAYS. They are going to have to prove to me that they are false before I accept them. I obviously discounted a long time ago the DOD's position that held us back for 4 years, that if you did not basically show acute signs of chemical exposure, that if you were exposed to low-level exposure that was not, therefore, acute, in the end there is no harmful result from that. We know in this country, working with chemicals, low-level exposure leads to illness and death. So I do not accept that and I did not accept a long time ago.

Now, all of you are heroes, all of you, and you served our country with distinction, and I believe your testimony will help us get the answers you want. Now, one of the lines of questions that I want is, I want to know—again, some of you have said on this record, if you were in any area of the operation where the alarms went off, and I want to know those experiences, and I want to know how you reacted to it. I want to know what you did and how you reacted to it. Col. Roman, I will start with you.

Col. ROMAN. I was in SCUD Alley there in Riyadh the first time I saw a SCUD. They trained us to put on our gas mask equipment in 14 seconds, from the time you pop it out of your container until you put it over your head and tie it real quick. I think the first time I saw a SCUD, I had it down to about 7 seconds, maybe shorter than that, and at that point in time the alarm did, in fact, go off. After the Patriot struck the SCUD, the chemical alarm started going off, and five or six times subsequent to that or after that, about the same things happened.

Mr. SHAYS. Was that the first time the alarm went off, after the war had begun?

Col. ROMAN. After the war had begun.

Mr. SHAYS. So you did not hear alarms before the war.

Col. ROMAN. Oh, no, sir, only when the SCUDs were in the air.

Mr. SHAYS. Now, that is conceivable that the alarm went off, though, because of the SCUD and not because of a detector. Is that correct?

Col. ROMAN. Yes, sir.

Mr. SHAYS. OK.

Col. ROMAN. I did not hear them go off at any other time personally unless there was a SCUD attack. And on one occasion—no, two occasions, I heard the alarms go off while I was in the Dhahran, but also it was after the SCUD attack.

Mr. SHAYS. Did you have confidence in the gear that you wore? Did you feel that if you were, in fact, exposed to chemicals, that the gear would do the job it was required to do?

Col. ROMAN. That the what, sir?

Mr. SHAYS. Did you have confidence that the protective gear, when you went into MOPP–4, that when you put this protective gear on, that it would do the job?

Col. ROMAN. We were not given that MOPP gear, sir. We had our chemical masks with us at all times, but we did not have access to the MOPP gear when I was——

Mr. SHAYS. So you just had the masks.

Col. ROMAN. Yes, sir.

Mr. SHAYS. No other equipment?

Col. ROMAN. No, sir.

Mr. SHAYS. OK. And did you take PB?

Col. ROMAN. Yes, sir.

Mr. SHAYS. For how many days?

Col. ROMAN. The required number of days and the required number of dosage.

Mr. SHAYS. Were you warned that it was an experimental drug for that purpose?

Col. ROMAN. No, sir.

Mr. SHAYS. Were you asked to take it, requested to take it, or what?

Col. ROMAN. No. It was given to us. In the Army you are given something to take like that as part of your equipment or as part of your dosage, and you take it without question.

Mr. SHAYS. OK. Thank you.

Mr. CANTERBURY. Sir, I remember the first time I experienced chemical alarms sounding and SCUD alerts was when just days prior to the air campaign starting. I was at King Khalid Military City when this occurred, and I remember during those alarms we immediately donned our masks, got into protective gear, MOPP–4.

Mr. SHAYS. So you had protective gear.

Mr. CANTERBURY. That is correct, sir.

Mr. SHAYS. And you put it on. Let me first go back to you, Col. Roman. How many times did you end up putting the mask on during the operation before?

Col. ROMAN. I remember having the mask on only on two occasions. Yes, sir.

Mr. SHAYS. How many times, Mr. Canterbury?

Mr. CANTERBURY. I am going to guesstimate probably about eight, total.

Mr. SHAYS. So it happened enough times that you are not quite sure of the count.

Mr. CANTERBURY. That is correct, sir.

Mr. SHAYS. In any of those experiences of the first two of you did you feel, taste, or react to those alarms? Did you see anything, did you feel anything, and did you have any effect? Colonel.

Col. ROMAN. No, sir. I could not say that I felt anything different or saw anything different.

Mr. SHAYS. OK.

Mr. CANTERBURY. The same, sir.

Mr. SHAYS. OK.

Mr. CANTERBURY. I cannot really honestly say.

Mr. SHAYS. Thank you. Mr. Stacy.

Mr. STACY. Sir, to my knowledge, we at one time——

Mr. SHAYS. First off, how many times did you hear an alarm?

Mr. STACY. Sir, I did not hear an alarm. At one time, the only people I saw the whole time I served in the Gulf was the 16 men in my company. I am unaware of anybody that was testing for chemicals. We did not test for chemicals. We did not have any fear of chemicals. As soon as we crossed the border was the last time we had our protective gear on.

Mr. SHAYS. But you had the protective gear with you.

Mr. STACY. Yes, sir.

Mr. SHAYS. And you never heard any alarms?

Mr. STACY. No, sir.

Mr. SHAYS. And in terms of you took the pill, the PB, Mr. Canterbury, you took the prescribed doses?

Mr. CANTERBURY. Yes, I did, sir. I took it for probably a period of 8 to 9 days.

Mr. SHAYS. Did you take it voluntarily, under order?

Mr. CANTERBURY. Under order and in the presence of a noncommissioned officer.

Mr. SHAYS. Now, the purpose of the noncommissioned officer was to what?

Mr. CANTERBURY. To ensure that the younger enlisted were taking those pills.

Mr. SHAYS. It was not to just help you figure out how to take it; it was to make sure you took it.

Mr. CANTERBURY. That is correct, sir.

Mr. SHAYS. Were you warned by anyone that it was an experimental drug?

Mr. CANTERBURY. No, sir, not that drug.

Mr. SHAYS. Mr. Stacy.

Mr. STACY. Sir, I would like to add, we did go past a blown-up ammunition dump, and we did find rounds that were suspected to be chemical. We took the pills. I never asked any questions because I believed in my chain of command, I believed we were doing the right thing, and I wanted to survive in case there was chemicals used on us.

Mr. SHAYS. So you were not warned that it was an experimental drug.

Mr. STACY. No, sir.

Mr. SHAYS. OK. And since you did not hear any alarm in the course of the operations that you were involved in, did you come into areas where others had the masks on?

Mr. STACY. No, sir. We had very little contact with any other people. We were in the tanks, so we would not be able to hear any alarms if they did go off. The NBC NCO from my unit, I never saw him until the war was over and we were ready to deploy back to Germany.

I do not know if they were testing. I know that we did not test. There is one tank out of our platoon that is designated to test for chemicals and they did not test. I was under the assumption that we were trying to either save the materials for testing or that we were not in the fear of any chemicals.

Mr. SHAYS. I know others are going to question you on depleted uranium. I am going to come back on that issue. As someone who was involved in sending you to Kuwait, we rejoiced in the fact that there were so few who lost their lives; and, frankly, much of what we heard was the battle from the air and what occurred there.

You have had very vivid description of the battlefield as it took place, and it was something that I do not think enough Americans have an appreciation of because there were some units that were never really involved in the battle directly to the intensity you were.

Mr. STACY. Sir, that was the only skirmish that we had encountered that was of any significance to myself or any of the other soldiers. To my knowledge, now this engagement, the night engagement I am talking about, first of all, we were on a 200-kilometer attack into Iraq, 36 hours nonstop, no sleep. It was at night. We were already tired. To my knowledge, we did not even move 5 miles. We rolled out there at 6:30 p.m., and it was 4 a.m., when the friendly fire incident did occur, and we stopped and sat in the battlefield until daylight and moved from there.

Mr. SHAYS. Well, we are going to come back, just to talk to you about depleted uranium. So you did take the pill. You were not warned that it was an experimental drug.

Mr. STACY. Yes, sir.

Mr. SHAYS. OK.

Mr. STACY. Sir, I would like to also say, we did take another pill. I did not know what it was. I have been told by another soldier in my unit they were malaria pills. I am not sure of that, though.

Mr. SHAYS. Let me go to Sgt. Zeller. Sgt. Zeller, did you hear any alarms go off?

Staff SGT. ZELLER. Sir, the ones I admitted to in this testimony, and then, like I said before, the ones that were connected to the vehicles who were convoying, they just went off all the time. And before we did the convoy, we were told to go to MOPP Level 4, and do not take it off until you go to the river, and we literally lived in them. And, sir, I just want to let you know something, that I was a chemical NCO, not fully schooled trained to have the MOS but enough trained to be a battalion NCO.

The long story short is, sir, those cry-backs with the suits in them with all the charcoal all over them, that we looked black as night after we wore them, those things only last 12 hours, sir, and

the one I testified to when I was up at Eagle, OK, and I was on guard duty—no, not guard duty, but in Tranquility, and they said SCUD is in the vicinity, we ripped the bags open and wore the suits. Twelve hours later, we should have gotten new suits, sir. We wore those suits all the way to Basra. We went all the way up to Basra and then back, cutting off the Republican Guard.

Mr. SHAYS. How many days was that?

Staff SGT. ZELLER. Sir, that had to be at least 2 months. We worked that long, and we sweat our tail feathers off in them. There was no more charcoal left with them, but we still had nothing else. We had no other skin.

Mr. SHAYS. I know you have it in your testimony, but I want it part of this questioning when we refer back to the record. How many times did the alarm go off that you recall?

Staff SGT. ZELLER. While driving all the time, it would just like go off, and whoever's alarm went off, they would stop, and then they would go and they would reset it and then we would drive along and then keep going.

Mr. SHAYS. Well, let me ask you this. Did you have the alarm before when you were driving, and did it go off?

Staff SGT. ZELLER. No, sir. It did not go off while we were on the other side of the border.

Mr. SHAYS. So when you were on the other side of the border driving——

Staff SGT. ZELLER. When we crossed——

Mr. SHAYS. Hear my question. Hear my question.

Staff SGT. ZELLER. Sir.

Mr. SHAYS. You had the alarm in this vehicle for a while.

Staff SGT. ZELLER. No, sir. We did not put them on until we were entering.

Mr. SHAYS. You did not turn on the alarm.

Staff SGT. ZELLER. We did not hook them to the vehicles until we were entering Iraq——

Mr. SHAYS. OK.

Staff SGT. ZELLER [continuing]. Because they had knowledge of contamination.

Mr. SHAYS. So it would really be pointless for me to make an assumption that the alarm did not go off before because you did not have them hooked up.

Staff SGT. ZELLER. Well, they were hooked up to a ground point. On our perimeter we would set up a camp, and we would have them on the outskirts of our perimeter, and each company was at the outskirts of the perimeter, and they had responsibility for theirs, and they had responsibility for theirs, and it was set up strategically so if the wind blew this way and took something, that one would go off, and all the message——

Mr. SHAYS. So those alarms did not go off, but when you mounted them on the vehicle——

Staff SGT. ZELLER. They were going off all the time when we were driving, sir.

Mr. SHAYS. OK. Well, that would lend argument to the fact that they would have been false alarms on the vehicles. It was not like you had been driving with them on the vehicle before and not going off.

Staff SGT. ZELLER. Sir, I would challenge that for the fact that we saw dead animals and so forth.

Mr. SHAYS. OK.

Staff SGT. ZELLER. And they did not have a bullet in them.

Mr. SHAYS. OK. I am going to just end with that, and we are going to go. We have plenty of time here. The dead animals; I am wondering if this is becoming folklore here. I would like to have any of you who saw dead animals, did you see dead animals sometimes with flies and sometimes without? Did you see humans dead with flies, without? Can any of you respond? I will start with you Sgt. Zeller.

Staff SGT. ZELLER. Sir, the flies were like epidemic.

Mr. SHAYS. They were everywhere.

Staff SGT. ZELLER. Everywhere, but when we found these herds, they were nowhere to be found, or they would be laying on the animal dead, deader than a door nail.

Mr. SHAYS. What would be laying on the animal?

Staff SGT. ZELLER. The fly.

Mr. SHAYS. So if you saw flies on the animal, they were dead flies?

Staff SGT. ZELLER. Right, sir, on the animal. I mean, why would the flies not be there, and they are everywhere else——

Mr. SHAYS. OK.

Staff SGT. ZELLER [continuing]. That might be clean possibly?

Mr. SHAYS. And then when you saw them, they were dead. That is something you saw. Correct? If you saw——

Staff SGT. ZELLER. They were on the side of the road, sir. I mean, you would be jogging, and then all of a sudden you would see a flock of animals deader than a door nail.

Mr. SHAYS. OK. And you did not see gunshot wounds.

Staff SGT. ZELLER. No, sir. No bombs dropped, no nothing. The area we went through, sir, was like the 82nd, 24th, and 101st. We all went through an area that was called, like, a spearhead move. We were not supposed to even be known that we were there. We jumped over the top of Taplin Road. We moved up Taplin Road, and supposedly Saddam did not know we were going to be there because he moved all his men down toward Kuwait.

Mr. SHAYS. Yes. OK.

Staff SGT. ZELLER. So it was one of those, like, blitzkrieg situations, and we were a part of that. So——

Mr. SHAYS. I hear you. Mr. Stacy, any dead animals without flies or dead flies?

Mr. STACY. Sir, the dead that we saw, we were mounted on the tanks. We did not stop and investigate. We just assumed everything that we saw dead as a casualty of the war.

Mr. SHAYS. And you do not have any story about flies not being on them. That was not something——

Mr. STACY. We did not investigate, sir.

Mr. SHAYS. I understand.

Mr. STACY. Until the war was over with, that is when we started going on clearing missions, but at that time we were in a different location, and I did not witness any of that.

Mr. SHAYS. OK. And when you did the clearing mission at the end, you did not witness any of that?

Mr. STACY. Not to my knowledge, sir. I have memory loss, and it is hard to recollect some things.

Mr. SHAYS. Listen, I do not expect you are going to always have an answer that you are going to know or that I am going to like; I just want it on this record. OK?

Mr. STACY. Yes, sir.

Mr. SHAYS. Let me just ask the two of you gentlemen, and then we will get to Mr. Sanders, did you see dead animals? Did you see humans? Were there flies? Weren't there flies?

Col. ROMAN. I have no experience, Mr. Chairman, with the dead animals. The only flies I saw were on myself usually.

Mr. SHAYS. OK.

Mr. CANTERBURY. Sir, I experienced both dead sheep and human bodies. I, like the sergeant down here, was up near the Taplin, and that is where I experienced the sheep, and I did not see any flies on those sheep.

Mr. SHAYS. And at the time did you notice it? Was it that interesting, or was it later on that people—I am just wondering if we are reconstructing this later.

Mr. CANTERBURY. No, sir.

Mr. SHAYS. Did you notice it then?

Mr. CANTERBURY. I remember discussing it with fellow soldiers at that point in time. We were just right across the street from the Taplin that the sergeant speaks of, and there were a lot of sheep herding around that area, and just days prior to ground war is when I saw, I personally saw the dead sheep with no flies. As far as humans, I saw a lot, but I was in a convoy, and I did not have time to look to see. OK? Sir?

Mr. SHAYS. Yes, sir.

Mr. CANTERBURY. There is something that I want to re-emphasize about the chemical alarms.

Mr. SHAYS. Yes, sir.

Mr. CANTERBURY. You asked, what did you do when chemical alarms would go off? We would get into our MOPP–4 gear. Something that bothers me a lot is the fact that I was a private over there, and I was expendable. I was forced to take my mask off to see if it was all clear. It bothers me when this French detection team is 2 miles up the road that could come down and check out the area to see if it is all clear, but instead, because my lieutenant and my platoon sergeant were uncomfortable in their protective gear at 100-some-degree temperatures, they would grab a private, take off your mask.

Mr. SHAYS. When they said that, did they keep their mask on?

Mr. CANTERBURY. Yes. They kept their mask on, and there were times when a couple of us privates——

Mr. SHAYS. Yes, sir.

Mr. CANTERBURY [continuing]. Basically said, we are not taking our masks off. And they threatened with court martials and threatened to have NCOs come over and take the masks off of you. I had my mask removed, and I am going to tell you right now, I took a sergeant's mask off with me.

Mr. SHAYS. So your testimony before this committee is that you were ordered to take off your mask and you did not and then a sergeant attempted to take it off and you took his mask off with you.

Mr. CANTERBURY. Along with it, as he was taking mine off, sir. I am sorry.

Mr. SHAYS. That needs to be part of the record.

Mr. CANTERBURY. There is no regard for human life there, and this is my life he was playing with.

Mr. SHAYS. I would like to think that that was an unusual experience in the war. We have never had anyone else testify to that, but I think it is very important that you made it part of the record.

Col. ROMAN. I would like to think, sir, that that would have been an isolated incident as well because as a member of the officer corps and having been a former enlisted person myself, an NCO, the men always came first.

Mr. CANTERBURY. Not a private.

Mr. SHAYS. Pardon me?

Mr. CANTERBURY. Not a private.

Mr. SHAYS. Mr. Sanders, you have as much time as you would like.

Mr. SANDERS. Thank you, Mr. Chairman, and I will be reasonably brief. I would just like to ask all of you if you could just respond.

As you know, or may know, the official position of the Presidential Advisory Committee in trying to understand Persian Gulf war illness is that they did not believe that chemicals played a role in the illness and believed that the primary cause was stress, that stress is the cause of Persian Gulf illness. Could you give me your observation on their conclusion? Just, Colonel, if you would start and just go down the line. Do you agree with that conclusion?

Col. ROMAN. I do not. I am not certain how trained medical personnel could come to that conclusion, much less a committee or a commission such as has been appointed by the White House to investigate this. I think that everyone who goes to war or who is in combat has some form of a trauma or a stress; however, to put the blame on trauma or stress like that is ridiculous.

It goes way beyond, I think, a conclusion that most normal people would have, and it particularly concerns me because it also obliterates the obvious, and that is that, in fact, chemicals and biological warfare weapons were found in the Gulf after the war, and that has become a matter of congressional testimony in the Congressional Record, so I cannot understand how they come to that conclusion.

Mr. SANDERS. Thank you.

Mr. CANTERBURY. Sir, my opinion about stress is that I do believe that stress can have a reaction on the human body, but as far as it having a reaction the way it is having on me, I doubt it, sir. I doubt it very much. There has got to be something more to my ailments, my illness, whatever, however you want to put it, sir. There has got to be.

Mr. STACY. Sir, I agree with him also. I have had some anger problems, but I would like to say——

Mr. SANDERS. So have we all.

Mr. STACY. Well, sir, to be honest and to be blunt, we are trained killers, defenders of this country. You cannot expect a soldier to take his training and to use that training and to come back the peaceful man that he once was. The things that you do in combat,

I can only say that it is like hell. That is what hell is going to be like.

Now, I agree, my health is not because of stress. I have tried the counseling and everything else. It has not helped any at all.

Mr. SANDERS. Sergeant.

Staff SGT. ZELLER. Sir, yes, sir. Sir, I was in SOCOM before this mission. OK? And you are pedigreed. You are taken care of. Your every need you ever needed was done. You never had to do anything but get in an aircraft and deploy. When you would come back, you would get that same old brief. It is called a "down brief." You are told about zoning. You are told about PTSD, so forth and so on. So, sir, I was somewhat educated. OK? And that is why I have been so argumentative, all the way up to Walter Reed about them trying to say that I have frustration.

No, I have aggravation, and the aggravation is caused by the U.S. military using plausible denial on me to cover the protocol.

Mr. SANDERS. The bottom line is you do not believe that stress is the source of it.

Staff SGT. ZELLER. No, sir, because I was trained on every deployment prior to that.

Mr. SANDERS. OK. Let me ask my second question, and that is—and the chairman has already gone over this a little bit—there have been a number of studies, including one from the DOD actually, which suggest that pyridostigmine bromide, in combination with other chemicals, can cause perhaps problems. Could you just very briefly tell us in your own personal observations with PB reactions that you may have had and what you have heard from your comrades about that. Colonel, did you want to start on that?

Col. ROMAN. Sir, I would love to answer the question if I had enough detail or information. I am not qualified to make that.

Mr. SANDERS. Thank you. If that is the case, that is the answer that we want. Private?

Mr. CANTERBURY. Sir, I did not know about bromide tablets until I got into the Persian Gulf region, and I do not know enough about it to form an opinion also.

Mr. SANDERS. OK. Your own personal observations of what people may or may not have—if you do not know, then that is the answer that we want. We do not want you to say what you do not know. Private?

Mr. STACY. That is the same here, sir. I took the pills, but I cannot recollect any effects I had from them. The shots, I know I had gotten sick from one of the shots. There was too many things going on to be concerned about the effects of any pills. We were under a lot of stress. We were tired, et cetera.

Mr. SANDERS. OK. Sergeant?

Staff SGT. ZELLER. Sir, I will be happy to give you what I have. I knew nothing about them. I will be honest with you. I did not get interested in them until some people were talking about them having adverse effects. So I studied them and I asked all the right people and they gave me this cholinergic crisis. So I can tell you now I know all about them, and every one of my symptoms that I have can be given to that. I have endocrine problems. I have nerve problems.

Mr. SANDERS. But in terms of your observations when you were over there in the midst of all this stuff——

Staff SGT. ZELLER. Sir, I did not think nothing of it; I was thinking of my job. I am the gun bunny on helicopters, and it is a tough job.

Mr. SANDERS. OK. My next question actually is for Private Stacy; and, Private, you indicated to us that right now your family is in serious financial straits. Why are you not receiving compensation that might be due to you because of your wartime condition?

Mr. STACY. I have been denied, sir. I have been denied undiagnosed illnesses. I have a claim pending for chronic fatigue. It has been pending for 2 years. My records are being shuffled back and forth from Nashville, TN, to Muskogee. They believe that all of my complaints are due to stress. I have a copy of my medical records, which I do not have on me now. But the doctor does say in my records, I believe the patient is exaggerating symptoms, I believe the patient has been coached, and I believe he is here to try to get increased disability.

Mr. SANDERS. OK. My last question, maybe I will start with you, Private, again, is you mention memory loss. In my own State of Vermont, we have run into folks who are suffering the same problem. Can you talk a little bit about what that is like and how that compares to——

Mr. CANTERBURY. Are you addressing me, sir?

Mr. SANDERS. I was talking to Private Stacy. I want anybody who feels comfortable to answer that. Talk to me a little bit about——

Mr. STACY. I will answer that, sir. I was unaware I had memory loss because I could not remember that I was forgetting. Whenever questions are asked me, I can recall incidents. Certain details, I did not focus on because I was more concerned about my wife and my daughter and coming home. My diary is real sketchy. I was not concerned about any of those things.

I did not realize I had memory loss until my wife started putting little notes up and stuff. And it is just little stuff, sir, just little things, going back and forth, trying to get something done around the house. I go back and forth, forget what I was doing, just little things that I am not even aware of.

Mr. SANDERS. OK. And is this different than before you went over?

Mr. STACY. Yes, sir. I have changed. My friends, nobody wants to be around me. They think I am crazy. They think I am talking about this Gulf war illness, that there is no such illness. It would be easier to convince people that I was abducted by aliens than I got sick in the Gulf.

Mr. SANDERS. OK. Would anybody else like to comment on memory loss?

Col. ROMAN. Yes, sir, I would.

Mr. SANDERS. Colonel.

Col. ROMAN. In my two VA evaluations and my one U.S. Army evaluation of myself at Fitzsimmons, two out of the three doctors who examined me who were certified to examine me for that particular question indicated in their notes that I have short-term memory loss, and I am not sure, quite frankly, whether it is be-

cause of my advancing age or because of something that occurred in the Gulf, but the neurologist, Dr. Baumzweiger, as I said earlier, did indicate that my short-term memory loss, at least a good portion of it, is due to neurological damage, perhaps brain-stem damage.

Mr. SANDERS. Anybody else want to comment on the issue of memory loss? Sergeant.

Staff SGT. ZELLER. Sir, I have got, like, I will give you an example that happened here. I forgot to pack my head gear coming here or I forget my keys or my wife asks me to do something, and I will go through the entire day, get home, and she said, Well, where is this? or What did you do with this? or How come you did not do that? And I am, like, I forgot. And it is very aggravating, sir.

Mr. SANDERS. Colonel, let me go back to one point that you just made. You indicated that you visited a physician in L.A. at the Veterans——

Col. ROMAN. At the VA, yes, sir.

Mr. SANDERS [continuing]. Who examined you and concluded that you were suffering from nerve damage as a result of chemical exposure?

Col. ROMAN. His exact words, sir, to be for the record would be that I had severe, neurological damage as a result of my service in the Gulf war. Perhaps chemicals or whatever you contracted over there was responsible for your nerve damage, but something occurred over there while you were there.

Mr. SANDERS. He determined that you had nerve damage as a result of your service.

Col. ROMAN. Yes, sir. Yes, sir.

Mr. SANDERS. And he later indicated to you that the VA no longer wanted him to be treating——

Col. ROMAN. At that particular time, he had finished his examination at 12 o'clock. It started at 9. He was summoned by the chief neurologist. He came back half an hour later somewhat in distress. He indicated that he was no longer authorized or allowed to treat Gulf war veterans because—he felt it was because that his conclusions or his findings that he was coming up with were not the same or expected findings that the VA expected him to find.

Mr. SANDERS. Unfortunately, Mr. Chairman, we have heard that tale once or twice before, I think, as well. Mr. Chairman, that is the extent of my questions at this time. Thank you.

Mr. SHAYS. Thank you. Just a few more questions, not many. I would like to know if each of you are registered in either the VA Health Registry and/or the Comprehensive Clinical Evaluation Program.

Col. ROMAN. Yes, sir. I am registered with both the VA in DC and the VA in Denver and the U.S. Army's program when they had me on their register as well.

Mr. SHAYS. I am sorry. Mr. Towns, you came back. I am sorry. I am going to go right to Mr. Towns. I am used to your being right here.

Mr. TOWNS. Thank you, Mr. Chairman, and I will try to be brief.

Mr. SHAYS. You can have as much time as you want.

Mr. TOWNS. Thank you. Let me begin by first saying, do any of you know whether other people in your units have experienced the same symptoms that you have? Colonel.

Col. ROMAN. Thank you, sir. Sir, I have been in touch with a number of people in my unit, a small number, because they are scattered throughout the country, and at least three of the people that I have been in contact with have registered with the VA; and, in fact, a couple of them were being treated at the VA—correction—the Fitzsimmons Army Hospital while I was there back in 1995, but I have not made any effort to find out what the rest of them were doing. There was 400 people I was serving with.

Mr. STACY. Sir, I have only been in contact with one other member of my unit since I have been out. He in turn states that he has been in contact with several other members. He states that he is experiencing some of the symptoms that I experienced. He has also stated that there are two other members from my unit who have been very ill for several years now and have been, the way he described it, on their death bed. They have not been able to come out of their bed for a couple of years now.

Mr. TOWNS. Stacy, you were in a pretty small unit.

Mr. STACY. Sir, I have heard rumors, but I will stick to fact. Fifty percent of the soldiers I have contacted, which I have not contacted over 10, are sick. The other 50 percent, which I was in denial for years—I believed the VA, I believed the doctors, and it is too hard of a battle to go and fight the VA. It is hard to accept the fact that this condition is going to ultimately be my end. Denial is a big problem with this.

Mr. TOWNS. Just for the record, let me just make certain. How many were in your unit?

Mr. STACY. Let's see. There are 12 tanks in a company, 4 men per tank.

Mr. TOWNS. Forty-eight?

Mr. STACY. Yes, sir. I have only contacted less than 10. A few are sick. One soldier lost a kidney. He had his liver patched and his spleen due to an accident playing softball. When he goes to the VA, they are able to tell him that these problems are because of these losses or organs, and he accepts that. Another soldier from my unit had a healthy child, to the best of my knowledge, and does not want to even speak about anything in the Gulf. He is in denial about it. He does not want to hear it. We had two soldiers that left the Gulf before combat even started, complaining of health problems.

Another soldier, I have got another friend that is not sick; still he is in the reserves, but he left over a month before we did. Another soldier is sick, and that is about all that I know.

Mr. TOWNS. OK. Thank you very much, Mr. Stacy. Sgt. Zeller.

Staff SGT. ZELLER. Sir, I know of an officer that I rode everywhere around the desert with, and I am here to tell you that he came down with something where they pumped him full of steroids, they pumped him full of this paquenele or whatever, and then the last-ditch effort was they finally got off their dukeses after he went after the hospital commander to send him to Walter Reed. And he had this very strange situation where they did plasma parises on

him and stuck him with several liters of sandaglobulin, which is like giving him a brand-new immune system.

And I have come to some information most recently about autoimmune disorders. That is why I have come up with that as one of the things. This guy was suffering from autoimmune symptoms, for some unknown reason. He was diagnosed with poly—CDIP—something poly something-neuropathy, chronic inflammatory polyneuropathy or something like that.

He was diagnosed with Guillaume-Barre. OK? Guillaume-Barre; I looked it up, and the only way you can get that is vaccines. OK? He was told at Walter Reed not to ever say anything about his problem because it cost beaucoup dollars for him to have anything like this done to him and that it would be way too much to do for everybody that is in the Army.

Sir, I am here to tell you that we were everywhere together. We drank out of the same water holes. We ate from the same logistics points and so forth and so on, and I think I am a little more heartier than he is, but he was sick with this, and I will be more than happy to give you his telephone number and let you call him and let him tell you his horror story, and it was about money. Walter Reed said it was about money. So I can give you a for-real.

And then the other situation, sir, that is really grave, I was drawn out. OK? In September 1995, I was working on a helicopter accident that happened at Fort Rucker, and I was investigating it, and Bethesda Naval Research Center called me and said I had to come forward or else, and I had no choice. I mean, I have been in where I was hiding, because I had seen it off in 1991. I had seen it when I filled out a DA–4700, demobilize/remobilize work sheet for Southwest Asia, and I put down all my problems then. OK? That was July 1991.

The long story short is this form was produced April 1991. Now, tell me if someone does not know something is going on. I have this form here. I will be happy to show you this form that I filled out. It has an NCO that signed me off, so it is official, and if you call him up, I am sure he will say, Yes, I did sign this guy off, and I did tell him it was only for a data base. We are not interested in taking care of you right now.

So when I started going to sick call and I had doctors telling me that you are riding sick call that you are trying to do something, so forth and so on, giving me all these ridiculing-type remarks, which I am not into, I started hiding. And there are soldiers hiding. There are people hiding because they do not want ridicule. They do not want to be treated like a second-class citizen for this ailment that they have, whatever it might be. It could be several things.

Mr. TOWNS. Right. Thank you.

Staff SGT. ZELLER. Sir, yes, sir.

Mr. TOWNS. Colonel, do you want to add something?

Mr. SHAYS. Could we put on the record, if I might?

Mr. TOWNS. Sure.

Mr. SHAYS. The people you suggest who were hiring are active military personnel who do not want to have to deal with this within the military system. Is that correct? You are not talking——

Staff SGT. ZELLER. Well, they have seen everybody——

Mr. SHAYS. Listen to my question.

Staff SGT. ZELLER. Sir, yes, sir.

Mr. SHAYS. What I am asking is, just for the record to make sure we know the difference, you are not suggesting that soldiers who have left active duty are necessarily hiding. You are talking right now, for the purposes of responding to Mr. Towns, you are responding to the fact that active military personnel, some in particular, chose to hide rather than come forward within the military system.

Staff SGT. ZELLER. Because I do not want to get kicked out, sir. I needed my job.

Mr. SHAYS. I just want the answer.

Staff SGT. ZELLER. Sir, yes, sir.

Mr. SHAYS. And I understand that.

Staff SGT. ZELLER. Sir, yes, sir.

Mr. SHAYS. And I am not critical of it; I just want to understand it.

Col. ROMAN. Fine. Mr. Towns.

Mr. TOWNS. Good point, Mr. Chairman. Yes.

Col. ROMAN. Thank you. I think I have been very conservative in my responses, and I am very careful in what I say, and so I would like to add for the record that to answer your question about the former people that I worked with or do I know about anybody else who was ill, I would like to answer into the record that Dr. Stuart Hiatt, H-I-A-T-T, who is an M.D. of his position, a surgeon, who was in top shape and could run 1 mile in about 4½ minutes, 5 minutes at the age of 50, went to the Gulf war was a volunteer.

He was my former commander, and he got there, was there 3 or 4 days, and they had to air lift him out of there. He did not see combat. They had to air lift him out of the Gulf. He went back into Fitzsimmons for examination, and they kept him 3 or 4 months after the war for unknown symptoms, unknown ailments in San Antonio.

I believe if you got his records, as a matter of fact this particular committee hearings, that you would be able to find out a whole lot more than I know about it at this point in time, but when you get a man who is in the prime of his life, and he gets to the Gulf and 3 days later he is air lifted out for unknown ailments and it is nothing to do with his physical fitness, because he is fit, then that is a problem.

Mr. TOWNS. Right. Let me ask you another question. How many of you have children? Do all of you have children?

Col. ROMAN. Yes, sir.

Mr. CANTERBURY. Yes, sir.

Staff SGT. ZELLER. Yes, sir.

Mr. TOWNS. Have you seen any problems with your children?

Col. ROMAN. I am not, sir. My children are adults, and they have children.

Mr. STACY. Sir, I would like to say, my daughter was born before I deployed, and she is having some problems. And I would also like to say, my wife and daughter did have a Gulf War Registry Exam in Jackson, MS.

Mr. CANTERBURY. Sir, I have three children, one boy, two girls. My youngest was conceived after I came home from the Persian Gulf. All three of my children complain about their arms and their legs and different muscles in their body hurting. My youngest

daughter has respiratory problems, and that is about the extent that I am aware of at this point in time, but for them to come to me basically every time I see them and complain that their arms and their legs hurt, I mean, I know children have growing pains, but they should not be complaining about them, almost on a daily basis.

Mr. TOWNS. Yes. Do you want to add?

Mr. STACY. Sir, these veterans and children, they are casualties of this war. Something should be done for them also.

Mr. TOWNS. Thank you very much, all of you, for your testimony. Mr. Chairman, I yield back.

Mr. SHAYS. I thank the gentleman, and I just want to go through—some of you responded, but I want all of you to respond in this part of the record.

I started out asking you, Col. Roman, and you told me you were registered in both the VA Health Registry and the DOD Comprehensive Clinical Evaluation Program; you were in both.

Col. ROMAN. Yes, sir, both with the VA and with the U.S. Army sent out a letter and asked me to be registered. I am. I believe the CDC also sent out something to me to fill out as a questionnaire type, and I filled that out as well.

Mr. SHAYS. Very good. Mr. Canterbury.

Mr. CANTERBURY. Sir, I have been registered on the Persian Gulf Registry Examination twice. I am registered on the DOD Health Registry.

Mr. SHAYS. Private Stacy.

Mr. STACY. Sir, I am also on the Gulf War Exam Registry. I did an updated data sheet. My doctor told me that they do not know what is wrong. They have run all the tests. I did request to go to the hospital there in Houston, TX for further tests, and they denied me that. As a matter of fact, it is not even in my records that I requested that.

Mr. SHAYS. Sgt. Zeller.

Staff SGT. ZELLER. Sir, I have been on it because Bethesda Naval Research called me September 1995. Since that time, I have had two different incidence, at Seaside, CA, where the registration is, where they told me that I was not registered. Somehow the data base, they switched computers or something like that, so I have had to, like, reapply twice.

And one of the situations was when I was calling the investigation team and, like, research team, the one you call and you tell where you were and what you did and so forth, which is a lot like the one I told here, they said in Washington that we have no registry of you. That was the second time this happened, and then I called California, and they told me that is crazy; we have you here.

So, in other words, California sort of lost me once, and then Washington was not in the groove with California. And, furthermore, my children and my wife have been on that since November or December 1995, when the guys that said tell your story, they said it is significant enough to put your whole family on this because there could be a possibility that they might have something wrong with them, too.

Well, since 1995, sir, not one of my relatives, until I went off on them most recently, was being seen, and the one they took was, like, superficially looked at, sir.

Mr. SHAYS. Now, all four of you had symptoms during the war. Is that correct?

Staff SGT. ZELLER. Sir, yes, sir.

Mr. STACY. Yes, sir.

Col. ROMAN. Yes, sir.

Mr. CANTERBURY. Yes, sir.

Mr. SHAYS. All four of you had it.

Col. ROMAN. Yes, sir.

Mr. STACY. That is correct, sir.

Mr. SHAYS. I want to know, in simple terms, what your diagnosis was and if any treatment was provided.

Col. ROMAN. For myself, sir, it occurred when I was in Riyadh and I was traveling with my commander at the time, who happened to be a physician. He did not happen to be; he is a good physician. And I got real sick, started vomiting, and——

Mr. SHAYS. Right now, I just want to know what your ultimate diagnosis was and what your treatment was.

Col. ROMAN. He diagnosed it at that point in time as food poisoning; not knowing anything else, that is what it was.

Mr. SHAYS. But that was onsite.

Col. ROMAN. Onsite. That is right.

Mr. SHAYS. But once you went to the VA, what was the diagnosis that you were given?

Col. ROMAN. I have given the VA and Fitzsimmons stool samples, when I am in the process of flu-like symptoms, and they have never found any kind of parasite or anything else that would cause me to have that kind of an illness.

Mr. SHAYS. You went to the VA. Correct?

Col. ROMAN. Yes, sir. After the war.

Mr. SHAYS. And after the war, what was your diagnosis?

Col. ROMAN. They have not diagnosed it.

Mr. SHAYS. OK. Private Canterbury.

Mr. CANTERBURY. Sir, are you asking what the diagnosis was in the Gulf or at the VA?

Mr. SHAYS. At the VA.

Mr. CANTERBURY. At the VA, I have been diagnosed, as far as I know of, with fibromyalgia and migraines. I am not service-connected, and that is it and I have gone through three different hospitals. I cannot get results from tests, sir.

Mr. SHAYS. Private Stacy.

Mr. STACY. Sir, I was undiagnosed also. I would also like to say, for the past year I have been pushed and pushed toward mental health. I am 30 percent service-connected for PTSD. I would not pursue that any further until just here recently because we were just starving to death. I would not accept the fact that it was PTSD, but all of my symptoms are undiagnosed.

Mr. SHAYS. So, in other words, for you to get some kind of compensation, that is the one you have to accept.

Mr. STACY. Yes, sir, and I would not do it. We have been starving for 1 year. Our family and friends, if it was not for them and God, we would not have made it.

Mr. SHAYS. Sgt. Zeller.

Staff SGT. ZELLER. Sir, if I may, I need to say it this way. They are trying to do my MEB now because I have caused so much of a ruckus. The long story short of it is it all happened when I tried to solicit the President, tried to call him and talk to him possibly.

Mr. SHAYS. Well, I do not think you are going to accomplish much doing that, and that is no disrespect to the President.

Staff SGT. ZELLER. Right, sir.

Mr. SHAYS. So I want you to answer my question, then.

Staff SGT. ZELLER. Well, the question is, they asked me——

Mr. SHAYS. I am going to interrupt you. I am sorry.

Staff SGT. ZELLER. Sir, yes, sir.

Mr. SHAYS. I am going to give you a chance to make your point, but I want the answer first, the answer to: what is your diagnosis?

Staff SGT. ZELLER. They did not give me one. They asked me what my most significant illnesses are, and that is all they want to focus on, sir. I cannot say it any other way. I am confused. I am not a doctor.

Mr. SHAYS. No. I just want to know if they had given you——

Staff SGT. ZELLER. Sir, yes, sir.

Mr. SHAYS. OK.

Staff SGT. ZELLER. They have not given me one.

Mr. SHAYS. I did say I would let you make your point. What is the point you want to make?

Staff SGT. ZELLER. The point is that, I mean, they focus on the significant illnesses and make them insignificant, and then I wind up just like this man here, living in Appalachia with five sons, starving to death, sir.

Mr. SHAYS. Because you are not being allowed to re-enlist. Your worst fear has come true. Your worst fear was that you came forward, you came forward, and you are not being allowed to re-enlist, so you are out. You are presently an active member of the Armed Services.

Staff SGT. ZELLER. Sir, yes, sir.

Mr. SHAYS. But you will be inactive when?

Staff SGT. ZELLER. As soon as they get the MEB together because I am fussing because there is nothing on there like what is happening to me.

Mr. SHAYS. Let me just conclude by asking, is there any question that I should have asked you that you wanted on the record? I will start with you, Colonel Roman. Is there any question that you wish we had asked or any one that you were prepared to answer that we should have asked?

Col. ROMAN. No, sir. I think you have been quite thorough.

Mr. CANTERBURY. I cannot think of any at this time, unless you could ask me—give me some time to think about that.

Mr. SHAYS. Well, the record will be open, and so you will be allowed to submit additional.

Mr. STACY. Not at this time also, sir. Thank you very much.

Mr. SHAYS. OK.

Staff SGT. ZELLER. Sir, this book right here I think has a lot to do with the situation, and I do not know how you can get a copy, because I could not get it in any book store, but I think you might really want to take a peek at this. I do not know if that is a ques-

tion, but maybe, Staff Sgt. Zeller, can I look at your book? I would be happy to show it to you, sir.

I am sorry, sir, but this book right here dates back to my original——

Mr. SHAYS. If that is the question you wanted me to ask, Sergeant, may I look at your book?

Staff SGT. ZELLER. Certainly, sir.

Mr. SHAYS. Thank you. I would like to.

Staff SGT. ZELLER. Sir, yes, sir.

Mr. SHAYS. One of the things that we think we can learn is to learn what has happened with the civilian population in Iraq. When we blew up the 21 to 36 potential sites—that number is classified, but when we blew up whatever number it was, the document said that the plumes would go away from the troops, not toward them. We know that some of the plumes went toward our troops, not away. But we then ask the question, well, if they went away from our troops, where did they go? And we suspect some went to civilian populations in Iraq.

The problem the Iraqi people have is they have a leader who is not about to admit that his stockpiling—think of this extraordinarily potentially wealthy country that instead of going toward war could have gone toward peace and used its resources. They had one of the highest educated communities. Women were given tremendous rights, these Arab women, and yet you have a country that has many sites that were blown up and had stores of chemicals, potentially biological agents, and we have reason to believe that many Iraqi citizens have been impacted.

And if we could learn what has happened to them, we might learn a little bit more about what has happened to all of you.

I thank all four of you for being here, and your testimony was extraordinarily valuable, and I know that there have been one or two references to not feeling a sense of pride and the love for your country, and you have the greatest country in the world, and you are going to see that to be true, if you do not feel it now; but you should also feel pride in your service to your country. And I hope our paths cross often in the future.

Col. ROMAN. Sir, I thought for the record I would like to interrupt and say that I did serve in the Gulf, and I served in Honduras during the Contras-Nicaragua situation, and I have a lot of pride, and I would do it again. I would not hesitate at all. I do not have any problem with the Army or the VA, except that we are not being treated. Treat us.

Mr. SHAYS. OK. A good way to end. Thank you. Thank you, gentlemen.

Mr. STACY. Thank you.

Mr. CANTERBURY. Thank you.

Staff SGT. ZELLER. Thank you, sir.

Mr. SHAYS. We will take our next panel. You are free to go.

Col. ROMAN. Thank you, sir.

Mr. SHAYS. Now, my understanding is that panel 2, that part of panel 2 is not available right now. Dr. Rostker has to go somewhere. OK. Dr. Rostker, why don't you just tell me what guidance you want to provide?

Mr. ROSTKER. I need to be at the Pentagon for about 45 minutes, starting at 1:30.

Mr. SHAYS. I think what we will do is go with panel 3 and then come to Panel 2.

Mr. ROSTKER. And I will come right back as soon as I can, sir.

Mr. SHAYS. Doctor, you have been very cooperative with this committee, and so we are happy to accommodate you.

Mr. ROSTKER. Thank you very much.

Mr. SHAYS. Thank you. And I appreciate the VA for accommodating Dr. Rostker. We will go with panel 3, and that is Dr. Garth Nicolson, the chief scientific officer, Institute of Molecular Medicine; Mr. Leonard Dietz, a physicist and research scientist, retired; and Dr. Durakovic—am I saying that correctly?

Dr. DURAKOVIC. Correct.

Mr. SHAYS. OK. Well, my assistant said it correctly, and I copied him. Dr. Durakovic, chief nuclear medicine science (former), Wilmington, DE.

We are going to ask all four of you to stand up, and we will swear you all in. Would you raise your right hands, please?

[Witnesses sworn.]

Mr. SHAYS. Please be seated. For the record, all four have responded in the affirmative. Can we go in the order in which I called you? Basically, we will start with you, Dr. Nicolson, and we will work our way down.

STATEMENTS OF GARTH NICOLSON, CHIEF SCIENTIFIC OFFICER, INSTITUTE FOR MOLECULAR MEDICINE, ACCOMPANIED BY NANCY NICOLSON, CHIEF EXECUTIVE OFFICER, INSTITUTE FOR MOLECULAR MEDICINE; LEONARD DIETZ, PHYSICIST AND RESEARCH SCIENTIST; AND ASAF DURAKOVIC, FORMER CHIEF, NUCLEAR MEDICINE SERVICE, WILMINGTON, DE

Mr. NICOLSON. I am Garth Nicolson, the chief scientific officer of the Institute for Molecular Medicine, a nonprofit, private institute in Irvine, CA. I am also a professor of internal medicine and a professor of pathology and laboratory medicine. I am joined here by my wife, Dr. Nancy Nicolson, who is the chief executive officer of the Institute for Molecular Medicine. She has degrees in physics and molecular biophysics.

We got involved in this issue when our stepdaughter returned from her service in the Gulf. She was a crew chief on a Blackhawk helicopter in the 101st Airborne, and she developed the unusual signs and symptoms that we know as Gulf War Syndrome, or we prefer, Gulf war illnesses, illnesses because we think there are a variety of different illnesses that make up this syndrome. In my first figure—those of you that have written testimony can follow it; the panel, I think, can follow it as well and hopefully they can see it—our hypothesis has been all along that our soldiers were exposed to combinations of chemical, radiological, and biological agents during their service in the Gulf.

We are particularly interested in the combinations of multiple chemical and biological agents. The reason we are very interested in the biological agents, particularly those that cause chronic illnesses, is that this is the only way that you can adequately explain

the illnesses passing to immediate family members, spouses, and children. We will come back to this.

Mr. SHAYS. Doctor, I am going to have you just slow down when you talk just a little bit.

Mr. NICOLSON. All right.

Mr. SHAYS. I am going to put the clock on. I will let you go another round.

Mr. NICOLSON. OK. In this figure are shown the signs and symptoms of Gulf war illnesses. You might notice that it is very complex. It involves 20 to 40 different signs and symptoms, and this, I think, has confused the diagnosis of this particular group of illnesses for some time; and as you have heard, many of the soldiers that testified before you were given the category of "undiagnosable illnesses," or they were put in the category of "stress-related illnesses" or Post-Traumatic Stress Disorder.

We do not feel that Post-Traumatic Stress Disorder is a major cause of the Gulf war illnesses. We think that it is caused by combinations of chemical and biological agents that produce these very complex signs and symptoms. We do not see how it could be produced any other way.

Now, in this figure we have compared the 650 soldiers that we examined or received information on with civilians who had Chronic Fatigue Syndrome or Fibromyalgia; and as you can see, the signs and symptoms shown here in the red bars compared to the light-blue bars are almost identical, meaning that these veterans probably did not have unidentifiable illnesses; they probably had Chronic Fatigue Syndrome—Fibromyalgia-like illnesses.

Now, these illnesses can be caused by a combination of different types of exposures, and we found recently with Chronic Fatigue Syndrome that biologic agents, such as chronic microorganisms, can cause these same illnesses. In fact, of the three candidates of microorganisms that are most likely to cause illness like this, viruses, bacteria, and bacteria-like microorganisms called mycoplasmas, we were attracted to the fact that mycoplasmas might be underlying at least some of the signs and symptoms of Gulf war illness.

Now, the reason is this type of microorganism can cause virtually all of the different signs and symptoms that I showed in the previous figure. In addition, the species of mycoplasma that we found predominantly in the Gulf war illness patients is a very unusual species of mycoplasma called Mycoplasma fermentans. This particular mycoplasma has the property that it can actually enter cells, and when it enters cells, it can cause havoc with the metabolism of the cell and can cause unusual signs and symptoms because it can colonize or go into virtually any tissue or organ.

When it gets in certain locations, like the synovial cells of the joints, it can cause an arthritis-like condition. In fact, aching joints and joint problems, or arthritic conditions, are very common, probably one of the most common signs and symptoms of Gulf war illness. And the reason that may happen is that as these microorganisms leave the cell, they take a piece of the plasma membrane with it, and in doing so they can stimulate an immune response against the host antigens that were carried on the mycoplasma as

it left the cell. Thus, some of the autoimmune signs and symptoms can be explained by this type of microorganism.

We have developed new diagnostic techniques based on the techniques of molecular biology, and we have been able to diagnose Gulf war illness in several hundred patients as due to this type of microorganism plus other potential infectious agents as well. We have found in our study 45 percent of the veterans that we tested, and in some cases their immediate family members were symptomatic, for this type of mycoplasmal infection.

We have looked now at nondeployed forces, and we find it in less than 4 percent of subjects, so there is a significant difference depending on whether they were deployed to the Persian Gulf and have the illness.

Now, the important thing is that this type of illness can be treated. It can be treated with multiple cycles of antibiotics; and, in fact, we found five different antibiotics that are effective, and these different antibiotics can be used in different combinations and different sequences of 6 week therapeutic treatments. The whole therapy can take over a year. We are dealing with very slow-growing microorganisms that are only moderately sensitive to antibiotics, and this is why it takes so long.

There is some information that I have listed here, nutritional requirements, and other recommendations that I will not go into now. We have been working with Dr. Bill Rae in Dallas and Dr. Charles Hinshaw in Wichita, and Dr. Jim Privatra in California on the nutritional requirements that are important.

This is what happened when we looked at 170 soldiers with Gulf war illness. Seventy-six of these proved to be positive for mycoplasmal infections. Seventy-three of them underwent the antibiotic therapy, and as you can see, after the first 6-weeks of therapy none of them recovered. They all relapsed with the usual signs and symptoms, but after subsequent therapy some of them recovered so that after five or six cycles of therapy, most of them had recovered from the illness.

Now, when I mean "recovery," that does not necessarily mean they are "cured," but they could return to active duty and undergo the physical requirements of their service. Now, that is with 73 patients, and that represents patients from every service in our armed forces except the Coast Guard.

And, finally, in the last figure that I am going to show and discuss briefly are what are the potential or possible origins of these chronic microorganisms. The first source that we have heard already is they could have been in the vaccines as contaminants, for example. It is not uncommon that these small, bacteria-like microorganisms like mycoplasmas can contaminate vaccines.

First, vaccines in the Gulf were given, multiple vaccinations were given simultaneously, and this is not the effective way to vaccinate someone. By giving all these multiple vaccines at once, you tend to immunosuppress an individual, and that could have made them susceptible to endogenous agents. Second, agents in the environment that were in the sand or in the water or so on, now mycoplasma can survive for some time in the sand, and Professor Luce Montagnier in Paris has indicated that these types of agents can persist in the environment.

The third point, which has been brought up, is that the plumes from the destruction of chemical-biological-warfare factories and bunkers that were destroyed during and after the war could contain these infectious agents, and they could have blown back across our lines. I think that this is also very likely. For the SCUDs. Some of the units that we have looked at were under repeated SCUD attack, and they now have health problems, and some of these SCUDS may have been equipped with CBW warheads or chemical or biological warheads to deliver these agents.

The Iraqis were operating under Soviet War Doctrine. We know that. That has been admitted by our intelligence. They would tend to mix agents, chemical plus biological together in an offensive attack; and if they did this, then this could explain the complex signs and symptoms that we see in Gulf war illnesses.

I thank you for the chance to address the panel and will be willing to answer any questions.

[The prepared statement of Mr. Nicolson follows:]

WRITTEN TESTIMONY OF

Dr. Garth L. Nicolson and Dr. Nancy L. Nicolson

COMMITTEE ON GOVERNMENT REFORM AND OVERSIGHT

Subcommittee on Human Resource and Intergovernmental Relations

UNITED STATES HOUSE OF REPRESENTATIVES

June 26, 1997

Dr. Garth Nicolson is currently the Chief Scientific Officer and Research Professor at the Institute for Molecular Medicine in Irvine, California. He was formally the David Bruton Jr. Chair in Cancer Research and Professor at the University of Texas M. D. Anderson Cancer Center in Houston, and he remains Professor of Internal Medicine and Professor of Pathology and Laboratory Medicine at the University of Texas Medical School at Houston. He is also Adjunct Professor of Comparative Medicine at Texas A & M University. Among the most cited scientists in the world, having published over 450 medical and scientific papers, edited 13 books, served on the Editorial Boards of 12 medical and scientific journals and currently serving as Editor of two (*Clinical & Experimental Metastasis* and the *Journal of Cellular Biochemistry*), Professor Nicolson has active peer-reviewed research grants from the U.S. Army, National Cancer Institute, National Institutes of Health, American Cancer Society and the National Foundation for Cancer Research. Dr. Nancy Nicolson is trained in molecular biophysics and is the Chief Executive Officer of the Institute for Molecular Medicine and President of the Rhodon Foundation for Biomedical Research. She has published over 30 medical and scientific papers and has delivered over 60 international and national scientific presentations. She is the Who's Who in the World International Woman of the Year for 1996-97.

We are here today as medical researchers who have been engaged in studying Gulf War Illnesses (GWI) but also as a family that has suffered from GWI. Our step-daughter returned from service in Desert Storm in 1991 as a Staff Sergeant and Crew Chief of a Blackhawk helicopter in the U.S. Army's 101st Airborne Division (Air Assault) and developed the unusual, multiple signs and symptoms of GWI that prevented her from finishing pilot training. She eventually left the Army, and we have been involved since that time in a research effort to identify some of the possible causes of GWI and develop treatments for GWI patients. Our hypothesis (Figure 1) is that GWI is not caused by stress, it is caused by multiple exposures to chemical, environmental, radiological and/or biological agents that cause chronic multisystem signs and symptoms that for the most part can be diagnosed as existing diseases [1]. We have been particularly interested in veterans with GWI whose family members are now also sick with similar signs and symptoms, suggesting that many GWI patients suffer from biological, not chemical or radiological origins for their illnesses. Illnesses caused by chemical or radiological exposures should not be transmitted to family members. GWI in immediate family members is officially denied by the Departments of Defense (DoD) and Veterans' Affairs (DVA). Although some family members could

have developed their illnesses by contact with war souvenirs, packs or uniforms, only biological causes of GWI can account for the overwhelming fraction of family members contracting the same illness in this important subset of GWI patients. Our research into GWI and the laboratory tests for GWI-associated pathogens that we developed have been done completely without compensation or funding from the U.S. Government. Since the Institute for Molecular Medicine is a not-for-profit private research institute dedicated to discovering new diagnostic and therapeutic solutions for chronic human diseases, we do not charge veterans or their family members for our assistance and services. In fact, we are assisting without compensation Desert Storm veterans from other Coalition countries that also have GWI casualties.

In addition to an unknown number of immediate family members with GWI, over 100,000 Desert Storm veterans are experiencing a variety of chronic signs and symptoms characterized by disabling fatigue, intermittent fever, joint and muscle pain, impairments in short-term memory, headaches, skin rashes, diarrhea, vision and gastrointestinal problems and a collection of additional signs and symptoms that has defied a clinical case definition (Figure 2) [2], but has been called Mucocutaneous-Intestinal-Rheumatic Desert Syndrome by Murray-Leisure et al. [3] of the DVA. These chronic signs and symptoms usually do not progress to cause death [4], but there are now thousands of U.S. Desert Storm veterans dead from a variety of illnesses [5]. Part of the confusion in diagnosing GWI is that somewhat similar or overlapping signs and symptoms can be caused by quite different types of exposures (chemical, radiological or biological or more likely combinations of these). The diagnosis and successful treatment of GWI are dependent on identifying the underlying exposures involved, because these illnesses are treated differently if their origins are chemical, radiological or biological. For the most part, GWI signs and symptoms began to present between 6 months to one year or more after the end of Operation Desert Storm, and when immediate family members present with the same illness, their onset usually occurred from 6 months to one year or more after the onset of the veterans' illness, and not every family member always develops GWI. Because of the apparent slow rate of transmission of GWI to immediate family members, we do not feel that the general public is at high risk for contracting GWI from casual contact with GWI patients.

The DoD has claimed from their clinical evaluation program that Gulf War veterans do not show higher rates of health problems than the U.S. population as a whole. They fail to mention, however, that all personnel that served in the Gulf received health clearances before they were deployed, and yet many returned with illnesses or later developed illnesses that cannot be explained. National Guard and Air Force Reserve units were studied by the Center for Disease Control (CDC) in Atlanta for evidence of chronic health problems associated with deployment to the Persian Gulf, and it is clear from this CDC study that the Persian Gulf deployed soldiers have much higher frequencies (from 2.5-times to 13.5-times higher) of chronic health problems (> 6 months duration) than those who were not deployed to the Persian Gulf Theater of Operations [6].

A major problem for Gulf War veterans with GWI is obtaining adequate care for their illnesses. Unfortunately, the signs and symptoms of GWI are not well established as criteria for particular diseases treated by the DoD or DVA [3]. Indeed, most GWI patients do not readily fit into DoD or VA diagnosis categories, resulting in many veterans receiving unknown diagnoses or psychological diagnoses, such as Post Traumatic Stress Disorder (PTSD). Although stress can exacerbate clinical conditions, we felt it unlikely that the complex signs and symptoms of GWI that veterans displayed (and especially those where immediate family members have similar signs and symptoms) were due to PTSD [5]. When we studied 650 veterans of Operation Desert Storm and their immediate family members who suffer from GWI, we found that their multiple chronic signs and symptoms were very similar to patients with Chronic Fatigue Syndrome (CFS) (often called Chronic Fatigue-Immune Dysfunction Syndrome or CFIDS) or Fibromyalgia (FM) (Figure 2) [7, 8]. These chronic conditions can have stress as an exacerbating factor, but they are unlikely to be solely caused by stress or psychiatric problems. In addition, the fact that many immediate family members have also presented with similar signs and symptoms indicates that diagnoses biased on PTSD may be a gross oversimplification of GWI [7]. The variable incubation time of GWI, ranging from months to years after presumed exposure, the cyclic nature of the relapsing fevers and other signs and symptoms, and the types of signs and symptoms are consistent with diseases caused by combinations of biological and/or chemical or radiological agent(s) (Figure 1) [7-10].

We suggested that GWI/CFS/FM can be explained in many patients by exposure of veterans to various biological agents (chronic pathogenic infections) in combination with chemical exposures and in veterans' family members to biological agents transported back home by the veterans (Figure 1). To confirm or eliminate the possibility that chronic infections were an important factor in GWI, and especially in immediate family members with GWI, we began by examining a variety of biological agents (bacteria, viruses, etc.) that can cause the chronic, overlapping, system-wide signs and symptoms seen in GWI. We could eliminate most of the acute or fast acting bacteria (listed in Figure 1), because of the chronic nature of GWI and the slow appearance and nature of signs and symptoms. After examining GWI patients' blood for the presence of chronic biological agents, the most common infection found was an unusual microorganism, *Mycoplasma fermentans* (incognitus strain), a slow-growing mycoplasma located deep inside blood leukocytes (white blood cells) of slightly under one-half of GWI patients studied [11, 12]. This microorganism is similar to a bacterium without a cell wall, and although mycoplasmas are often found at superficial sites in humans, such as in the oral cavity, they are rarely found in the blood. When they are in the blood, similar to other bacteria, they can cause a dangerous system-wide or systemic infection [13]. In addition, cell-penetrating mycoplasmas, such as *Mycoplasma fermentans*, may produce unusual autoimmune-like signs and symptoms when they escape from nerve and other cell types and stimulate host immune responses to host cell antigens carried on the mycoplasma surface (Figure 3). Our detection of mycoplasmal infections in the blood leukocytes of ~45% of the

GWI patients examined (76 out of 170 patients), including 2 out of 2 British Desert Storm veterans with GWI, indicate that systemic infections may be a major contributor to GWI [11, 12].

In response to our published studies [7-12] and formal lectures at the DoD (in 1994 and 1996) and DVA (in 1995), Dr. Steven Joseph, then Assistant Secretary of Defense for Health Affairs, and Dr. Kenneth Kizer, Undersecretary for Health, DVA, have stated in letters to the press and various members of Congress that this type of infection is commonly found, not dangerous and not even a human pathogen, and our results have not been duplicated by other laboratories. These statements could not be further from the truth. The Uniformed Services University of the Health Sciences, the U.S. military's medical school, has been teaching its medical students for years that this type of infection, although rare in the U.S. population, is very dangerous and can progress to system-wide organ failure and death (see attachments). In addition, the Armed Forces Institute of Pathology (AFIP) has been publishing for years that this type of infection can result in death in nonhuman primates [14] and in man [15]. The AFIP has also suggested treating patients with this type of infection with doxycycline [16], which is one of the types of treatment that we have recommended [9-12]. Then why has the DVA issued guidelines stating that GWI patients should not be treated with antibiotics like doxycycline, even though in a significant number of patients it has been shown to be beneficial [9-12]? In response to the comments that our tests have not been duplicated, a certified diagnostic clinical laboratory, Immunosciences Laboratories of Beverly Hills, CA, has been conducting diagnostic tests on mycoplasmal infections in blood of GWI and CFIDS patients, and they are finding essentially the same results as we have found. Thus our results have been replicated by a certified commercial laboratory. The DoD and DVA have also stated that we have not cooperated with them or the CDC in studying this problem. This is not true. We have done everything possible to cooperate with the DoD, DVA and CDC on this problem, and we even published a letter in the Washington Post on 25 January 1997 indicating that we have done everything possible to cooperate with government agencies on GWI issues. We formally invited DoD and DVA scientists and physicians to the Institute for Molecular Medicine to learn our diagnostic procedures on 23 December 1996 at a meeting convened at Walter Reed Army Medical Center by Major General Leslie Burger at the request of Congressman Norman Dicks (D-WA). We have been and are fully prepared to share our data and procedures with government scientists and physicians. Although government laboratories can test for mycoplasmal infections and have been conducting their own examination of mycoplasmal infections in GWI patients, they are using relatively insensitive, outdated antibody tests or conventional molecular biological tests, and we would not expect them to detect the infection by these procedures.

In GWI patients that tested positive for mycoplasmal infections in their blood, we have found that this type of infection can be successfully treated with multiple courses of specific antibiotics, such as doxycycline (200 mg/day for 6 weeks per cycle), ciprofloxacin (or Cipro, 1500 mg/day for 6 weeks per cycle), azithromycin (or Zithromax, 500 mg/day for 6 weeks per cycle), clarithromycin (or Biaxin, 500-1000 mg/day per 6 week cycle) or minocycline (200 mg/day for 6 weeks per cycle) [9, 11, 12], along

with other nutritional recommendations (Figure 4). Multiple treatment cycles are required, and patients relapse often after the first few cycles, but subsequent relapses are milder and patients eventually recover (Figure 5). Using the techniques of Nucleoprotein Gene Tracking [17] and forensic Polymerase Chain Reaction, slightly under one-half (~45%) of the Desert Storm veterans and their immediate family members who had GWI/CFS/FM signs and symptoms in our studies showed evidence of mycoplasmal infections in their blood leukocytes [11, 12]. In contrast, in nondeployed, healthy adults the incidence of mycoplasma-positive tests were <5% [12]. We need to stress that these studies do not involve controlled patient populations, such as all veterans that served in a single unit compared to similar numbers of nondeployed personnel from the same unit; therefore, the percentage of mycoplasma-positive patients overall is likely to be lower than found in our studies. This is reasonable, since GWI patients that have come to us for assistance are probably more advanced patients (with more progressed disease) than the average GWI patient. We found that patients on antibiotic therapy (n=73) relapsed within weeks after the first 6-week cycle of therapy, but 58/73 recovered after up to six cycles of therapy and 14/73 are still undergoing therapy (Figure 5). GWI patients who recovered from their illness after several (3-7) 6-week cycles of antibiotic therapy were retested for mycoplasmal infection and were found to have reverted to a mycoplasma-negative phenotype [11, 12]. We hypothesize that the therapy takes a long time because of the microorganism(s) involved (a mycoplasma) is slow-growing and is localized deep inside cells in tissues, which are more difficult locations to achieve proper antibiotic therapeutic concentrations. As stated above, multiple cycles of therapy result in eventual recovery in a high percentage of mycoplasma-positive GWI patients. Although anti-inflammatory drugs can alleviate some of the signs and symptoms of GWI, the signs and symptoms appear to quickly return after discontinuing drug use. If the effect was due to an anti-inflammatory action of the antibiotics, then the antibiotics would have to be continuously applied and they would be expected to eliminate only some of the signs and symptoms of GWI. In addition, not all antibiotics, even those that have anti-inflammatory effects, appear to work. Only the types of antibiotics that are known to be effective against mycoplasmas are effective; most have no effect at all on the signs and symptoms of GWI/CFS/FM, and some antibiotics make the condition worse. Thus the antibiotic therapy does not appear to be a placebo effect, because only a few types of antibiotics are effective and some, like penicillin, make the condition worse. We also believe that this type of infection is immune-suppressing and can lead to other opportunistic infections by viruses and other microorganisms or increases in endogenous virus titers. Although we have been criticized for not conducting double-blinded, controlled clinical studies on large numbers of patients, such studies are quite labor intensive and expensive, and all of our studies were conducted without any government support or help whatsoever. We have designed a doubled-blinded, cross-over clinical trial that includes placebo and two antibiotics, and we would like to obtain government support for such a trial (Figure 6).

We consider it quite likely that many of the Desert Storm veterans suffering from the GWI/CFS/FM signs and symptoms may have been exposed to chemical/biological cocktails (or endogenous sources of these agents) containing slowly proliferating microorganisms, including pathogenic mycoplasmas and

quite possibly other bacteria and viruses, and such infections, although not usually fatal, can produce various chronic signs and symptoms long after exposure. The DoD has maintained that Iraqi offensive Chemical and Biological Weapons were not released during or after the Gulf War, but we did not have detection equipment deployed to be able to determine whether Biological Weapons were present. The Iraqi armed forces were operating under Soviet War Doctrine, which stresses offensive use of combinations of Chemical and Biological Weapons together with conventional weapons. Evidence presented to this subcommittee indicates that it was extremely likely that Chemical Weapons were released during and certainly after the conflict when bunkers containing Chemical and Biological Weapons were destroyed. We indicate again that chemical and/or radiological exposure(s) can result in somewhat similar signs and symptoms but this does not explain the apparent contagious nature of GWI and the delayed appearance of similar GWI/CFS/FM signs and symptoms in immediate family members. Fortunately, the types of slow-growing, chronic infections in GWI can be diagnosed and successfully treated with multiple cycles of specific antibiotics.

There were several potential sources of chronic biological agents in the Persian Gulf Theater of Operations (Figure 7) [18]. First, deployed soldiers were given multiple inoculations of experimental vaccines in unproven immunization schemes, such as vaccines that were given all at once instead of using an appropriate schedule of inoculations. Multiple vaccinations given simultaneously can result in immunosuppression and leave an individual susceptible to opportunistic infections. Some of these experimental vaccines could also have been contaminated with small amounts of slow-growing microorganisms. Next, the Iraqis were known to have extensive stockpiles of Biological Weapons and the potential to deliver these weapons offensively, at short range in modified biological sprayers that deliver Biological Weapons onto the sand to create exclusionary zones or 'biological minefields' and at long range in modified SCUD-B (SS-1) missiles with 'skyburst' warheads. As mentioned above, many of the storage and factory facilities where Chemical and Biological Weapons were stored were destroyed immediately up to, during and after the Desert Storm ground offensive, releasing plumes containing these agents high in the atmosphere where they could be carried downwind ('blow-back' exposures) to our lines. These and other possible mechanisms of potential exposure must be carefully examined, not categorically dismissed by DoD personnel in Washington with little first-hand knowledge of the conditions on the ground [18]. There are a number of possible reasons why the DoD and DVA deny that our forces were exposed to Chemical and Biological agents during the Gulf War, and several possibilities are listed in Figure 8.

Finally, can GWI be completely explained by chronic bacterial and/or other infections? The answer to this is no. GWI is not one disease; it appears to be a collection of various disorders and illnesses that produce complex chronic signs and symptoms. Desert Storm veterans were exposed to a variety of toxic agents in the Persian Gulf, including oil well fires, battlefield smoke, anti-nerve agents, insecticides, or multiple chemical agents, and in some cases radiological agents (DU), and many GWI patients now have

multiple sensitivities to various chemicals because of these exposures. Chemical exposures can cause toxicological effects and produce many but not all of the signs and symptoms of GWI [18, 19]. In addition, chemical exposures can result in immunosuppression and leave an individual susceptible to infections. Future efforts should be directed at determining the types of exposures that occurred in the Persian Gulf region, including chemical, radiological and biological exposures and how combinations of these could be involved in causing chronic illnesses. In the case of biological agents or infections where treatments exist, controlled clinical trials will have to be designed and initiated, and the necessary resources to conduct and evaluate these trials will have to be allocated. There is, however, a bonus from our efforts at understanding the role of chronic infections in GWI. We and other laboratories have now found similar chronic infections in a rather large subset of civilian cases of CFS and FM, patients with these illnesses have been diagnosed and successfully treated, and these patients are recovering after years of unexplained illness. Since there are over one million CFS/FM patients in the U.S. alone, this means that hundreds of thousands of Americans may be able to regain their health using the diagnostic tests and treatment suggestions developed for GWI.

We believe that Congress holds the key to solving the problem of GWI. This and associated disorders (CFS and FM) must be studied and solutions found using the peer-reviewed grant award system, such as that used by NIH. Efforts to direct funding away from or rebudget allocated funds for CFS and FM research, such as done over the last several years by NIH, should be stopped. GWI research and treatment cannot be left to the DoD and DVA, because they have not shown themselves to be especially effective or responsive to the health problems of afflicted Gulf War veterans and their family members. We consider it appropriate that civilian scientists and physicians collaborate closely with their counterparts in government to study and solve this problem in as objective manner as possible. We thank you for the opportunity to address this important issue.

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Under penalty of perjury, we swear that the statements above are true and correct to the best of our knowledge, information and belief.

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The Institute for Molecular Medicine
and
Professor of Internal Medicine
Professor of Pathology and Laboratory Medicine

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National Institutes of Health, National Cancer Institute, 5RO1-CA63045-03 - "Breast Cancer Metastasis-Associated Genes", Garth L. Nicolson, Principal Investigator, 5/1/94-8/30/97

U.S. Army Medical Research and Development Command Grant DAMD 17-94-J-4513 - "Breast Cancer Metastases: Prognosis and Monitoring" Garth L. Nicolson, Principal Investigator, 10/1/94-9/30/98

Figure 1

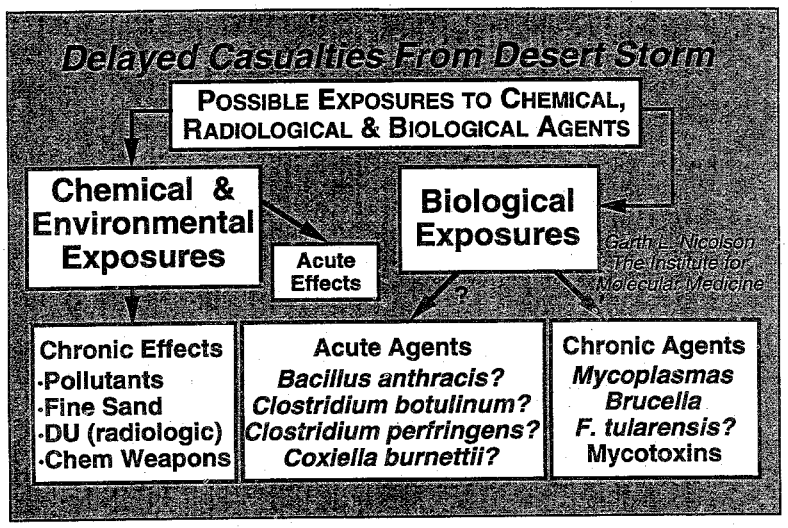

Delayed Casualties From Desert Storm
Possible Exposures to Chemical, Radiological & Biological Agents
Chemical & Environmental Exposures
Acute Effects
Biological Exposures
Garth L. Nicolson
The Institute for Molecular Medicine
?
Chronic Effects
·Pollutants
·Fine Sand
·DU (radiologic)
·Chem Weapons
Acute Agents
Bacillus anthracis?
Clostridium botulinum?
Clostridium perfringens?
Coxiella burnettii?
Chronic Agents
Mycoplasmas
Brucella
F. tularensis?
Mycotoxins

Figure 2

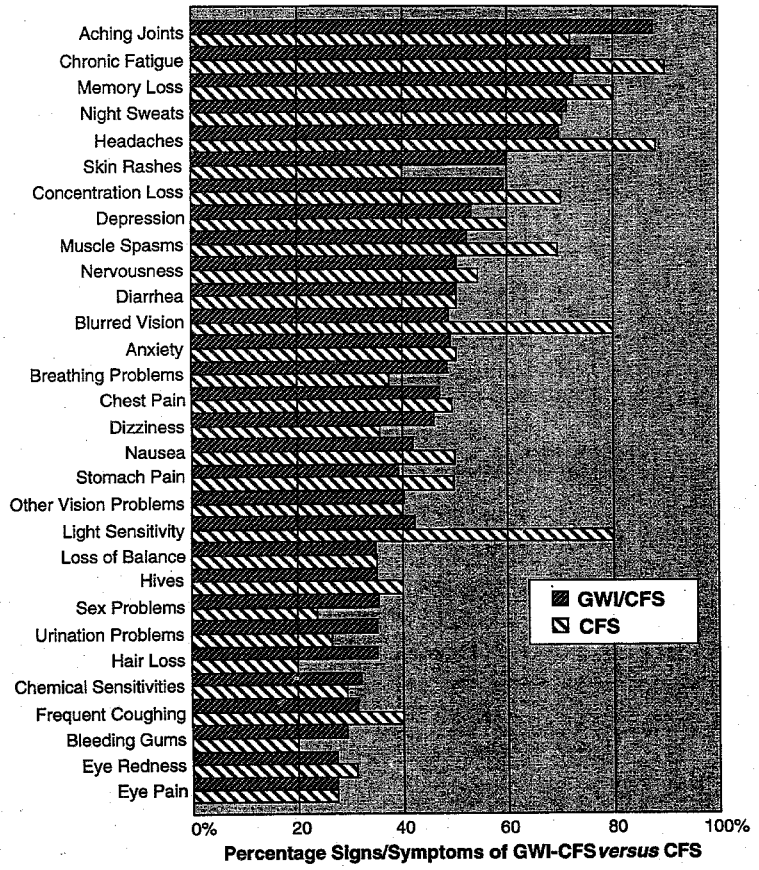
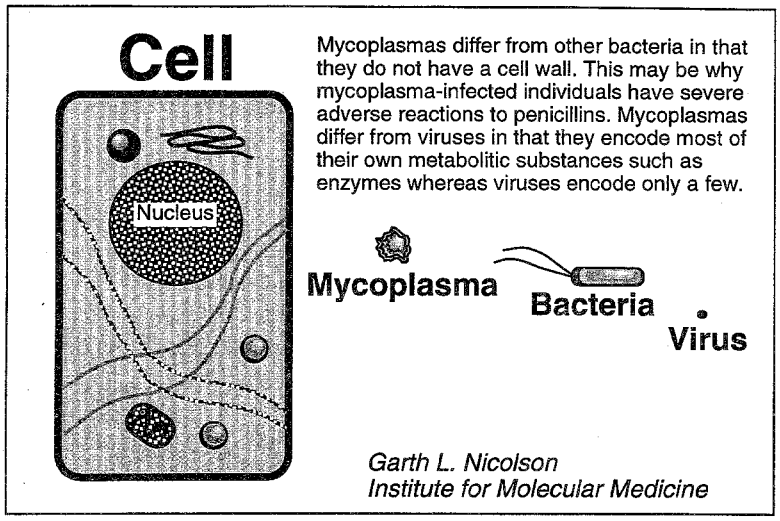


Figure 2. The signs and symptoms of GWI/CFS in 650 Desert Storm veterans.

Figure 3


Cell
Nucleus
Mycoplasmas differ from other bacteria in that they do not have a cell wall. This may be why mycoplasma-infected individuals have severe adverse reactions to penicillins. Mycoplasmas differ from viruses in that they encode most of their own metabolitic substances such as enzymes whereas viruses encode only a few.
Mycoplasma
Bacteria
Virus
Garth L. Nicolson
Institute for Molecular Medicine

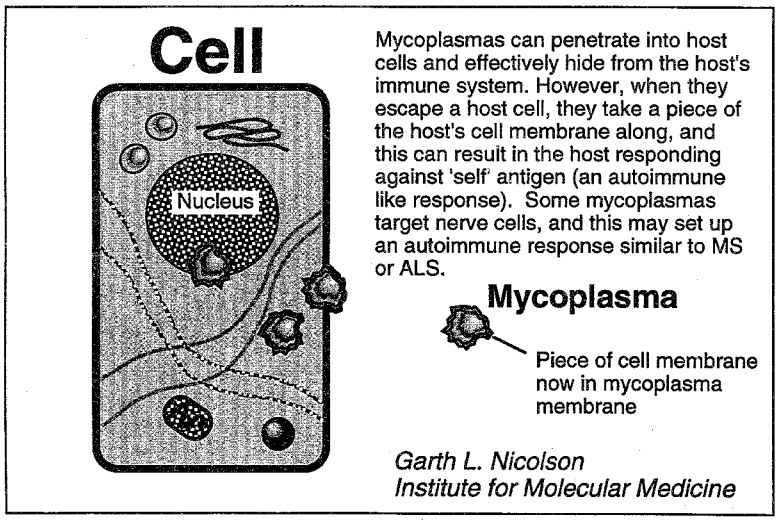

Cell
Nucleus
Mycoplasmas can penetrate into host cells and effectively hide from the host's immune system. However, when they escape a host cell, they take a piece of the host's cell membrane along, and this can result in the host responding against 'self' antigen (an autoimmune like response). Some mycoplasmas target nerve cells, and this may set up an autoimmune response similar to MS or ALS.
Mycoplasma
Piece of cell membrane now in mycoplasma membrane
Garth L. Nicolson
Institute for Molecular Medicine

Figure 4

Diagnostic and Treatment Recommendations

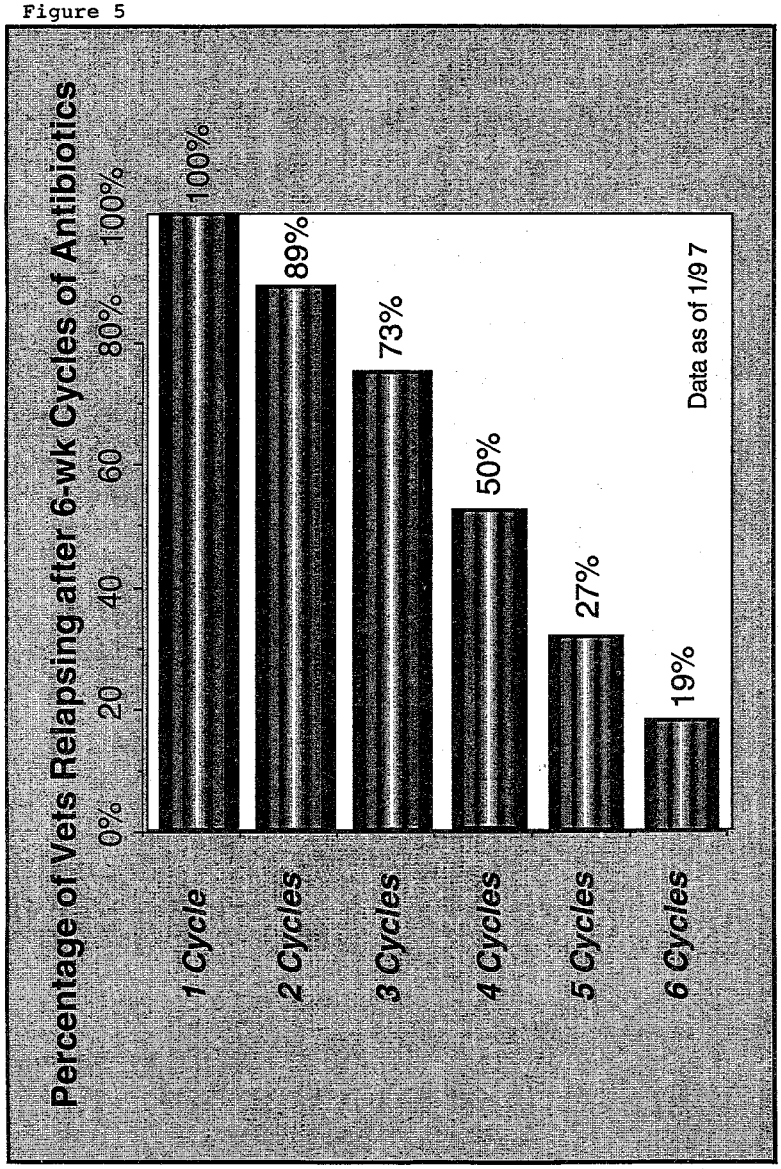
DIAGNOSTIC FINDINGS:
45% of GWI soldiers and symptomatic family members have systemic mycoplasmal infections (n > 200) found in their white blood cells.

TREATMENT RECOMMENDATIONS:
Several 6 week cycles of these antibiotics:
- Doxycycline (200 mg/d)
- Ciprofloxacin (1,000-1,500 mg/d)
- Azithromycin (500 mg/d)
- Clarithromycin (500 mg/d)
- Minocycline (200 mg/d)

NUTRITIONAL RECOMMENDATIONS:
Vitamins: sublingual B complex, C, E, CoQ_{10}
Minerals: Zinc, Chromium, Magnesium, Selenium.

OTHER RECOMMENDATIONS:
Reduce refined sugar, caffeine, alcohol, fats
Increase natural foods, vegitables, fruits
Replace depleted gut flora with *Lactobaccillus*
Control fungal/yeast, bacterial infections
Some moderate physical activity, saunas

Figure 5



Source of Data: Nicolson et al., Intern. J. Medicine (1997)

Figure 6

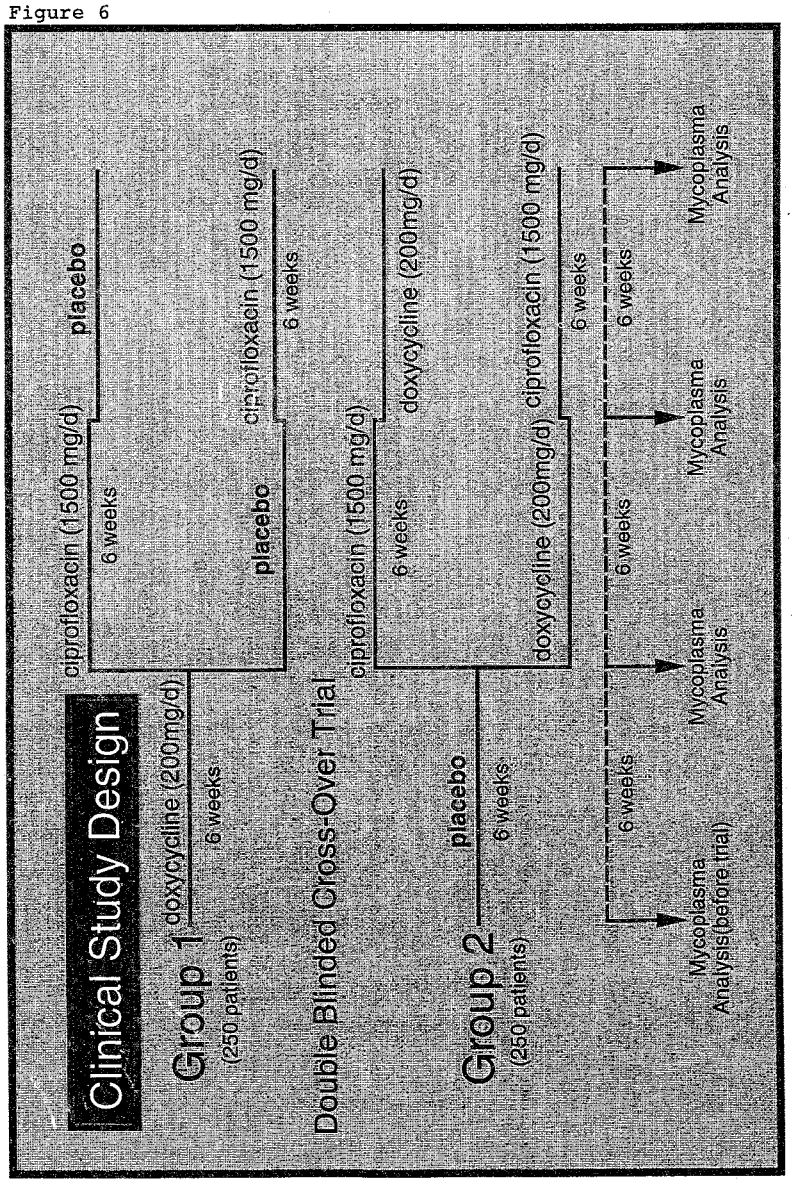

Clinical Study Design
Double Blinded Cross-Over Trial
Group 1
(250 patients)
doxycycline (200mg/d)
6 weeks
ciprofloxacin (1500 mg/d)
6 weeks
placebo
ciprofloxacin (1500 mg/d)
6 weeks
placebo
Group 2
(250 patients)
placebo
6 weeks
ciprofloxacin (1500 mg/d)
6 weeks
doxycycline (200mg/d)
6 weeks
doxycycline (200mg/d)
6 weeks
ciprofloxacin (1500 mg/d)
6 weeks
Mycoplasma Analysis (before trial)
Mycoplasma Analysis
Mycoplasma Analysis
Mycoplasma Analysis

Figure 7

Hypotheses: Mycoplasma/GWI Origin

Testimony to the US Congress (26 June 97)
House Committee on Government Reform/Oversight

1. Vaccines (mycoplasma contamination?)
   Multiple vaccination immunosuppression?
2. Endogenous Sources (Sand, Water, etc.)?
3. Plumes from bombing of CBW Factories and Demolition of Bunkers (Blow-Back)
4. Purposeful seeding of exclusionary regions (>50 Italian-made Biological Sprayers)
5. SCUD B (SS-1) CBW skyburst warheads

Figure 8

Possible Reasons: Denial of CBW Use

1. Future Credibility? U.S. threat to use Nuclear or CBW if Iraq used CBW.
2. False Alarms? Lack of immediate large scale battlefield casualties suggested many CW alarms might be false alarms?
3. Origins of CBW Agents? Some CBW Agents were U.S. made and sent to Iraq.
4. Troop Morale? Denial to prevent any potential panic among Coalition Forces?
5. Compensation? Acknowledgment of CBW use will result in increased claims.
6. Military Preparedness? Lack of proper preparation, equipment and training would be obvious if CBW acknowledged.
7. Chemical Weapons Convention? Did the Government deny CBW use to prevent pressure for CW deterrent and vote to kill the CWC ratification in Congress?

DEPARTMENT OF VETERANS AFFAIRS
UNDER SECRETARY FOR HEALTH
WASHINGTON DC 20420

Dear Editor:

I will appreciate your assistance in sharing the following message of critical importance to your readers -- veterans and non-veterans alike. Thank you for the opportunity to set the record straight.

You may have read or heard news reports in recent days that indicate that the illnesses suffered by some Gulf War veterans are caused by bacteria and are contagious. As the head of the Veterans Health Administration, I would like to assure the public that there is no evidence that Gulf War veterans are suffering from a transmissible disease. These reports, based on an unsubstantiated theory of one non-physician researcher, have unduly alarmed the public that sick and disabled Gulf War veterans can transmit contagious illnesses by casual contact. Based on all we know, and all science has shown to date, this is inaccurate and has served only to needlessly frighten people.

The best medical evidence available finds no basis to claim that the illnesses which some veterans attribute to their service in the Gulf are contagious or that, as some reports have stated, the nation's blood supply is somehow endangered by Gulf War veterans who give blood. The Department of Veterans Affairs (VA) employs tens of thousands of health care workers and, in contrast to what has been reported, not one physician or nurse in our medical facilities has become ill as a result of caring for Gulf War veterans. Likewise, in 1994, VA asked the Centers for Disease Control and Prevention to investigate concerns that a group of Gulf War veterans in Pennsylvania was suffering from an infectious disease. CDC carefully studied those veterans and found no evidence of a contagious illness.

Our Gulf War veterans and their families -- and the general public -- deserve facts, not harmful, unfounded hypotheses. We are reaching out to Gulf War veterans who are sick or have health concerns, urging them to contact VA through our around-the-clock toll-free helpline --1-800-PGW-VETS. VA can provide the medical care and scientific information that Gulf War veterans need.

Sincerely,

Kenneth W. Kizer, M.D., M.P.H.

THE ASSISTANT SECRETARY OF DEFENSE
WASHINGTON, D. C. 20301-1200

HEALTH AFFAIRS

Honorable William V. Roth
United States Senate
Washington, DC 20510

Dear Senator Roth:

This is in response to your letter requesting a reply to Mr. Aubrey J. Leager who expressed concerns about the risk of mycoplasma infection resulting from service in the Persian Gulf. The Department is acutely aware of how important it is to protect our troops from potential hazards while deployed. As a result, we are sponsoring several large epidemiological research studies designed to evaluate a number of possible health consequences of the Persian Gulf deployment, including infectious diseases like mycoplasma. Results from some of these studies are expected soon.

Mr. Aubrey J. Leager also mentioned concern for the possible use of chemical/biological weapons during Operation Desert Storm. We have asked several independent groups of scientific experts to review information concerning health issues of the Persian Gulf War. Some of these groups have specifically investigated the possible use of chemical/biological warfare during Operations Desert Shield/Storm. Among them was the Defense Science Board which found "There is no scientific or medical evidence that either chemical or biological warfare was deployed at any level against us, nor that there were any exposures of US service members to chemical or biological warfare agents in Kuwait or Saudi Arabia." (June, 1994). Additionally, the Institute of Medicine, in its initial report, Health Consequences of Service During the Persian Gulf War: Initial Findings and Recommendations for Immediate Action (January, 1995), stated "The committee could find absolutely no reliable intelligence, and no medical or biological justification for any of these purported claims (chemical/biological warfare). Furthermore, analysis of the attacks indicated that the alarms were false positives generated by dust particulates. When analysis of the alarms was followed by more sophisticated tests, the results were confirmed to be negative." In light of these and other reports, the Department has found no evidence of chemical/ biological warfare usage. We remain open to any new evidence that may be presented.

The Department of Defense is fully committed to providing the very best medical care available to its veterans. Currently, we are providing extensive medical examinations through the Comprehensive Clinical Evaluation Program (CCEP) to eligible beneficiaries who are experiencing illnesses which they believe are related to the Persian Gulf War. I have enclosed, for your information, the third report on the CCEP, incorporating the

results of approximately 10,000 examinations. The CCEP experience, to date, is consistent with the conclusions of the National Institutes of Health Technical Assessment Workshop that the unexplained illnesses being reported by many Persian Gulf War veterans are not a single, apparent disease, but rather a "range of illnesses with overlapping symptoms."

An expanded Persian Gulf Veterans Research Working Group of DoD, VA, and Centers for Disease Control physicians and scientists met with Professor Nicolson on August 4, 1995, to discuss his theory regarding mycoplasma infection. I have enclosed an information paper detailing the meeting. Professor Nicolson has been provided the necessary information to submit a formal research proposal. There are two possible avenues we have offered Professor Nicolson. First, there is a specific Persian Gulf health research program. A formal call for proposals was published in the Commerce Business Daily in May 1995. Secondly, and in addition to the very specific Persian Gulf research program, the U.S. Army Medical Research and Materiel Command (USAMRMC) administers numerous other research programs that may have application. Some USAMRMC research programs include infectious diseases, human immunodeficiency virus, combat casualty care, field medical/dental equipment/material, combat dental care, army systems hazards, medical biological defense, medical chemical defense, and special programs. Researchers are encouraged to submit proposals which address major research thrusts, technical approaches, research goals and military relevancy. Additional information can be obtained by writing the Commander of the USAMRMC. The address and telephone number are as follows:

Commander
U.S. Army Medical Research & Materiel Command
ATTN: SGRD-ACQ-BA
Fort Detrick, Frederick, MD 21702-5012

Telephone Number: (301) 619-7216

I trust you will find this information helpful.

Sincerely,

Stephen C. Joseph, M.D., M.P.H.

Enclosures
As Stated

Interferon-alpha 2b-helpful for chronic HBV, HDV, and HCV.[21, 22]

Recombinant Vaccinia -can control the level of viraemia after HDV superinfection.[26]

G. Other Viruses

HTLV-1 is sexually transmitted and blood-borne.[27] Most infected persons remain healthy carriers. There is some association with adult T-cell leukemia/lymphoma (ATL), immune dysfunction, and chronic progressive myelopathy. The only clinical and pathologic manifestations occur in individuals with the associated diseases.

Clinical manifestations of ATL: Lymphadenopathy, skin lesions, hepatosplanomegaly, hypercalcemia, anemia, thrombocytopenia, bone lesions. Propensity for pyogenic and/or opportunistic infections.

Pathologic manifestations of ATL: Skin, lymph node, and bone marrow biopsy reveals large cell, mixed, or unclassifiable non-Hodgkin's lymphoma cells with anti-Tac + (IL-2 receptor) and usually T4 phenotype. Lymphocytes with convoluted nuclei.

Treatment of HTLV-1

Azidothymidin2-will inhibit the in vitro infectivity of HTLV-1.

III. Bacterial

A. Mycoplasma infections

Mycoplasmas belong to the class Mollicutes. Mollicutes are the smallest organisms capable of self-replication in cell free media. Unlike viruses, Mollicutes have both DNA and RNA. They are tiny bacteria that stain gram-negative because they lack a ridged cell wall.

Mycoplasma fermentans, *M. penetrans*, *M. genitalium*, *M. hominis* and *Ureaplasma urealyticum* are the mycoplasma organisms that are known to be transmitted sexually.

The incidence rate of genital mycoplasmas is significantly affected by the type of contraception, the number of sexual partners, socioeconomic states, degree of cultural traditional, as well as hormonal status.

Results obtained in vitro suggest that mycoplasmas act as cofactors with the human immunodeficiency virus (HIV) in the development of AIDS and mycoplasmas, including *M. fermentans*, *M. pirum*, and *M. penetrans* have been isolated from HIV-infected individuals. These mycoplasmas have the capacity to invade cells and to be potent immunomodulators. They can produce systemic infections in HIV-infected individuals, but their pathogenic role in association with HIV remains to be determined.[28]

Clinical and Pathologic Changes:

A. *Mycoplasma fermentans*

The most serious presentation of *M. fermentans* infection is that of a fulminant systemic disease that begins as a flu-like illness.[28] Patients rapidly deteriorate developing severe complications including adult respiratory distress syndrome, disseminated intravascular coagulation, and/or multiple organ failure.

The organs of patients with fulminant *M. fermentans* infection exhibit extensive necroses. Necrosis is most pronounced in lung, liver, spleen, lymph nodes, adrenal glands, heart, and brain. *M. fermentans* is identified in areas of necrosis, particularly in the advancing margin of necrosis, by the immunohistochemistry using specific anti-*M. fermentans var* and *incognitus* antibody and/or by in situ hybridization assays using cloned incognitus strain DNA (psb-2.2). Mycoplasma-like particles are found intracellularly and extracellularly by electron microscopy.

Lo and associates demonstrated *M. fermentans* infection in the tissues of 70% of AIDS patients with clinical manifestations of functional organ deficits. No other microorganisms were present in these lesions of these AIDS patients. *M. fermentans* occurred in tissues with only mild histopathological changes, and in areas with degenerating cells with patchy necrosis. Generally there was no significant tissue reactive process or acute inflammatory reaction. Again, identification of *M. fermentans* utilized electron microscopy, immunohistochemistry with specific anti-*M. fermentans var* and *incognitus* antibody, and in situ hybridization assays using cloned incognitus strain DNA (psb-2.2).

Kidney infection with *M. fermentans* produces 1) focal segmental and global glomerulosclerosis, frequently characterized by visceral epithelial cell hypertrophy and vacuolization; 2) microcystic dilation of tubules that contain large proteinaceous casts; 3) tubular cell degeneration with necrosis; and 4) variable degrees of interstitial edema and inflammation. These changes have been termed "AIDS associated nephropathy" in HIV-1 infected patients with AIDS. Bauer and colleagues tested renal tissue from 15 AIDS patients with AIDS associated nephropathy and 15 AIDS patients without AIDS associated nephropathy for the presence of *M. fermentans* by immunohistochemistry and electron microscopy. All 15 AIDS patients with AIDS associated nephropathy had *M. fermentans* within glomerular endothelial and epithelial cells, glomerular basement membrane, tubular epithelial cells, tubular casts, and mononuclear interstitial cells. All 15 AIDS patients without AIDS associated nephropathy showed no evidence of *M. fermentans* infection in renal parenchymal cells.[30]

B. *Mycoplasma penetrans*

Mycoplasma penetrans is uniquely associated with HIV-1 infection and AIDS disease. Enzyme-linked immunosorbent assay (ELIZA) and Western blot analysis reveals that 40% of AIDS patients infected with HIV-1 have antibodies against *M. penetrans* but only 0.3% of HIV-1 negative control subjects have antibodies to *M. penetrans*. Only 0.9% of HIV-1 negative individuals attending sexually transmitted disease clinics have antibodies to *M. penetrans*.[31,32] We do not yet understand the role of this mollicute in AIDS disease nor do we know what symptoms it produces, if any, independent of HIV-1 infection, but Lo has evidence that it could be very important in the development of Kaposi's sarcoma. He has found a greater than 60% incidence of *M. penetrans* infection in those HIV positive individuals (in the male homosexual group) who have Kaposi's sarcoma.

Extensive invasion by *M. penetrans* produces cytopathic effects and can cause cell death. Lo and associates have demonstrated this organism as the sole agent in human organs with demonstrable tissue damage. EM studies of urine with *M. penetrans* show the tip-like structure of the organism buried deep in the cytoplasm of urothelial cells.

C. *Mycoplasma pirum*

Montagnier and colleagues have isolated *M. pirum* from mononuclear cells in the blood of AIDS patients.[33] Chirgwin and colleagues have isolated *M. pirum* from urine samples from HIV-1 infected patients. The role of this organism in clinical disease, if any, is unknown.

No data is available regarding any histopathologic alterations produced, if any, by *M. pirum*.

D. *Mycoplasma genitalium*

M. genitalium was originally described in 1981 by Tully and colleagues.[34] They recovered it from the urethra of two male homosexuals with nongonococcal urethritis (NGU).

Non-gonococcal urethritis (NGU), is a poorly understood disorder linked to *Chlamydia trachomatis*. In 1993 Horner et al and colleagues[35] detected *M. genitalium* in urethral samples from 24 (23%) of 103 men with symptoms, signs, or both, of acute NGU, but from only 3 (6%) of 53 men without NGU ($p < 0.006$) using specific polymerase chain reaction (PCR). The association was independent of the presence of *C. trachomatis* and could not be explained by differences in age, ethnic origin, lifetime number of sexual partners or a change in sexual partner during the previous 3 months. The mycoplasma-positive men responded well to doxycycline treatment, a response that was at least as satisfactory as that of the chlamydia-positive men. Their findings suggest that the *M. genitalium* is probably a cause of NGU in humans. This correlates well with the known virulence characteristics of *M. genitalium* and its ability to cause urethritis in male sub-human primates.

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M. genitalium is also implicated in pelvic inflammatory disease (PID). Approximately one quarter of infertile women have antibody to *M. genitalium*, although there are no associated changes on the hysterosalpingogram.

M. genitalium has also been recovered from the respiratory tract, along with *M. pneumoniae*[36]

E. *Mycoplasma hominis*

M. hominis is also associated with PID. *M. hominis* infections place pregnant women at increased risk for having a preterm delivery. This is particularly true if the woman has frequent intercourse.[37] *M. hominis* can be isolated from the bloodstream of women with postpartum fever and endometritis, and can cause septic arthritis post-partum. *M. hominis* is also causes some cases of acute pyelonephritis.

F. *Ureaplasma urealyticum*

Ureaplasma urealyticum can also produce NGU and PID independent of *M. genitalium* and of *C. trachomatis*. Chronic prostatitis is also linked to *U. urealyticum*.[38] *U. urealyticum* is associated with altered motility of sperm, but its role in infertility remains unclear.[39]

Patients with acute anterior uveitis (AAU) had a high incidence of asymptomatic infection of the urethra and/or cervix with *U. urealyticum*, *Chlamydia trachomatis* and *M. hominis* in a study by Graninger et al. They found that infections with *U. urealyticum* were significantly more frequent in patients with AAU when compared with a sex- and age-matched control group.[40]

U. urealyticum is one cause of chorioamnionitis.[41] Ureaplasmal infection of the chorioamnion is significantly associated with premature spontaneous labor and delivery. Congenital infection with *U. urealyticum* is linked to central nervous system damage[42] Case reports have irrefutably demonstrated the ability of *U. urealyticum* to cause neonatal bacteremia, pneumonia, and meningitis. *U. urealyticum* is also implicated as a cause of chronic lung disease of prematurity and in particular of bronchopulmonary dysplasia (BPD).[43 44] *U. urealyticum* infection should also be considered in the differential diagnosis of hydrops fetalis.[45]

Treatment of mycoplasm infections:

Mycoplasmas are susceptible to antimicrobial agents that affect DNA, RNA, protein synthesis, or the integrity of the cell membrane. Mollicutes, as a group, are innately resistant to antibiotics that act on the cell wall (such as penicillin and cephalosporin) because they lack cell walls and the peptidoglycan that largely make up cell walls. They are also resistant to agents that interfere with synthesis of folic acid (eg. sulfa drugs).

Tetracycline and related compounds (eg. minocycline and doxycycline) are among the few antibiotics that are effective against virtually all species of mollicutes. These compounds are effective against *M. fermentans*.[46] Tetracycline antibiotics are bacteriostatic, they bind to the S7 protein of the 30s subunit of the bacterial ribosome and prevent binding of aminoacyl-tRNA to the ribosome acceptor site, thus blocking protein production.

Quinolone antibiotics, (especially ciprofloxacin, but also ofloxacin and temafloxacin) seem to be the most effective against *M. fermentans*. These are useful for other mycoplasmas especially for those that have become resistant to tetracycline or erythromycin. But, quinolones do not have reliable in vitro activity against *U. urealyticum*.

Erythromycin is often used to treat *Mycoplasma pneumonia* but most mollicutes, with the notable exceptions of *M. pneumonia* and *Ureaplasma urealyticum* are resistant to erythromycin. Erythromycin resistance by *M. pneumonia* and *U. urealyticum* has arisen in recent years. Erythromycin belongs to the macrolide-lincosamide-streptogramin B (MLS) family of antibiotics. MLS antibiotics inhibit protein synthesis, probably by binding to the 50S ribosomal subunit or perhaps, by breaking up the mRNA-bound 70S ribosomal complex leading to degradation of the 50S subunit.

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Note: The efficacy of antimicrobial treatments of mycoplasma infection of immunocompromised patients is uncertain. Concerns include: 1) lack of an intact immune system may require treatment with these bacteriostatic antibiotics for a prolonged (even indefinite) time and at high dosages. 2) prolonged treatment can lead to the development of resistant strains, without complete eradication of the organism, 3) the presence of the IS-like element in *M. fermentans* can accelerate the development of resistant strains.

B. Chlamydial infections

Chlamydia are 0.2-1.0 μm coccoid, obligate intracellular bacteria found within cytoplasmic vacuoles. *Chlamydia trachomatis* of serotype A, B, Ba, and C are associated with a hyperendemic chronic blinding conjunctivitis called Trachoma. Serologically distinct, more invasive serotypes of *C. trachomatis* (L-1,L-2, and L-3) cause Lymphogranuloma venereum. Another serogroup of *C. trachomatis* (serotypes D-K) are a major cause of non-gonococcal urethritis (NGU), pelvic inflammatory disease (PID), proctitis, epididymitis, and inclusion conjunctivitis (adult and newborn). In addition, *C. trachomatis* (serotypes D-K) are associated with an abnormal hysterosalpingogram and with female infertility.

The developmental cycle of chlamydia is distinctive. The small (350 nm) infectious particle, the elementary body (EB) attaches to the host cell and enters a phagosome; the EB becomes metabolically active and forms a large (1 μm) reticulate particle (RB-also known as initial body). The RB divides producing EB's. The cell ruptures in 2-3 days releasing the new EB's.

Lymphogranuloma venereum

Clinical & Pathologic Features

LGV is virtually always sexually transmitted.

1. Both men and women: 5-21 day incubation period. Original lesion is a herpetiform vesicle (rarely seen in women). Vesicle ruptures producing a painless, punched out, rapidly self-healing ulcer. The chlamydia enter the blood stream and produce a systemic disease with lymphadenopathy, headache, chills, sweats, migratory polyarthritis and splenomegaly. After 1-2 weeks nodes become tender, fluctuant, and ulcerate, releasing pus.

Skin is ulcerated. Microscopically there is suppurative granuloma formation in the area of the ulcer. Lymph nodes also have multiple suppurative granulomas. Distinctively, LGV produces vacuoles that are most notable at the periphery of the ulcers. Vacuoles contain the chlamydia. The organisms are best seen with Warthin-Starry (and other silver stains). They are also visible on Brown-Hopps tissue stain, and with difficulty, can be visualized on H&E.

2. Men: Buboes are common (rare in women). In male homosexuals, chalmydial proctitis is common.

3. Women: Involvement of lymph nodes of perineum can lead to rectal strictures. Scarring of labia, vagina and urethra also occur.

4. Complications:

If infective genital tract discharges are inoculated into the eye either during sexual activity or by hand to eye contact, conjunctivitis may develop. This is an acute follicular conjunctivitis often with keratitis and micropannus.

Extensive scarring can cause blockage of lymphatic drainage and elephantis (especially in women).

Reiter's syndrome -a poorly understood reactive arthritis with associated inflammatory mucocutaneous lesions and conjunctivitis/uveitis. *C. trachomatis* has been documented in about 50% of men with sexually acquired Reiter's syndrome. It is suspected that *C. trachomatis* and other infectious agents (*Shigella felxneri*, *Salmonella* spp. *Yersinia enterocolitica*, and *Campylobacter* spp. and perhaps *Neisseria gonorrhoea*.) serve as a trigger in a genetically predisposed host. Postdysenteric Reiter's syndrome occurs primarily in children.

Other complications of LGV include erythema nodosum, erythema multiforme, scarlatiniform skin eruption.

6. Laboratory

INFORMATION PAPER: MYCOPLASMA INCOGNITUS AND PERSIAN GULF VETERANS

Mycoplasmas are the smallest, free-living infectious agents. They are distinct from viruses because of their ability to grow in a cell free media and from bacteria because they lack a cell wall and are able to synthesize cell wall precursors. The Mycoplasmas are ubiquitous in nature. A variety of plant and animal diseases are caused by the Mycoplasmas. However, only Mycoplasma pneumoniae, Ureaplasma urealyticum, and Mycoplasma hominis have been clearly shown to cause disease in man.

Garth Nicolson, PhD., Chairman of Tumor Biology at MD Anderson Cancer Center, Houston, Texas, has reported in numerous medical interviews and a letter to the editor (JAMA 273:618-619, 1995) that Persian Gulf veterans are suffering from a chronic fatigue illness caused by infection with Mycoplasma.

Physicians and scientists from the Department of Veterans Affairs, Defense, and Health and Human Services met with Dr. Garth Nicolson on August 4, 1995, at VA Headquarters in Washington, D.C. Dr. Nicolson presented this group with his investigation of 73 Persian Gulf veterans and his survey results on 650 veterans. To summarize, survey participants have reported multisystem symptoms, including: aching joints, chronic fatigue, cognitive problems, sleep difficulties, headaches, skin rash, depression, muscle spasms, nervousness, diarrhea, etc. Dr. Nicolson reported that 73 symptomatic Persian Gulf have been tested for Mycoplasma using novel forensic polymerase chain reaction and gene tracking techniques. Of the veterans testing positive, 35% M. Fermentans, 10% M. Genitalium, and 55% Mycoplasma species responses were found. Dr. Nicolson also reported that the HIV-1 envelope gene is being isolated in the same subfraction layer as the Mycoplasma cytoplasmic isolates.

Furthermore, he reports that some veterans received empiric antibiotic therapy for this putative infection with Mycoplasma. The antibiotic therapy (doxycycline 100-200 mg/day or macrolide) was administered in an uncontrolled and unblinded trial. Fifty-five of the seventy-three veterans reported good responses to antibiotic therapy and some

eventually "returned to normal." Dr. Nicolson has not examined or reviewed the medical records on the veterans tested or treated to date; results are based solely on self-reported symptoms.

Dr. Nicolson poses a testable hypothesis regarding the nature of unexplained illness in Persian Gulf veterans. It should be noted however, that the researcher himself states that the results are preliminary and not definitive. No causal association can be made between the illnesses reported by these veterans and the test results obtained. These findings require more extensive investigation prior to scientific conclusions being drawn. Such studies would involve blinded-testing of a case-control population, including full medical evaluations of the study participants. In addition, the test procedures used to confirm the presence of Mycoplasma infection in Nicolson's lab are experimental tests with unknown predictive value, sensitivity or specificity. No validated, readily available, diagnostic test for M. Incognitus is currently available for routine diagnostic use.

The Departments of Veterans Affairs, Defense, and Centers for Disease Control and Prevention are exploring means to pursue the questions raised by the work of Dr. Nicolson. We agree with the statement of Dr. Gareth Green (JAMA 273:619, 1995) that until these results can be validated with accepted scientific research methods and are subject to a peer review process, we do not recommend treatment of any individual with doxycycline or other antibiotics.

Mr. SHAYS. Thank you. Mr. Dietz.

Mr. DIETZ. Mr. Chairman, members of the committee, thank you for inviting me to share with you my concerns about depleted uranium and its possible connection to Gulf War Syndrome.

I first became concerned about the health consequences of depleted uranium in the fall of 1979, when I worked at the Knolls Atomic Power Laboratory in Schenectady, NY. The laboratory was operated by the General Electric Co. for the Department of Energy. While troubleshooting a radiological problem, my colleagues and I accidentally discovered depleted uranium aerosols collected in environmental air filters exposed at the Knolls site.

The source of the uranium contamination was the National Lead Industries Plant in Colonie, 10 miles east of the Knolls site, near Albany, NY. National Lead was fabricating depleted uranium penetrators for 30-millimeter cannon rounds. We also discovered depleted uranium in air filters exposed at the Kesselring site in West Milton, NY, where crews for the nuclear Navy are trained, 26 miles northwest of the National Lead plant.

This is by no means the maximum fallout distance for uranium aerosols. The 26-mile radius surrounding the city of Albany corresponds to more than 2,000 square miles where this fallout was occurring.

In January 1980, I wrote an unclassified report documenting the mass spectrometer measurements we made, and it was recently obtained under the Freedom of Information Act, and a photocopy has been given to this committee.

Totally unrelated to the discovery of depleted uranium in Knolls-site air filters, in February 1980, a court order by New York State, citing public health reasons, shut down National Lead for exceeding a New York State Department of Environmental Conservation monthly radioactivity limit of 150 microcuries for airborne emissions. This corresponds to less than 1 pound of depleted uranium metal, equivalent to 1.4 of the small penetrators used in aircraft 30-millimeter cannon rounds.

New York State health officials understood that exposure of its citizens to even small amounts of depleted uranium was harmful; therefore, they stopped it.

Consider what happened in the Gulf war. Uranium metal is pyrophoric, and when a high velocity depleted uranium penetrator hits a tank, its leading end ignites and burns explosively, forming aerosol particles of uranium oxide that are mostly 5 micrometers or less in size. By the way, five micrometers equals two-ten-thousandths of an inch.

These particles become airborne and, like dust, can be spread far and wide by wind action. Their fallout range is virtually unlimited. Uranium microparticles can be inhaled and ingested easily, and that makes them dangerous to human health. Radioactive contamination from depleted uranium is permanent for friend or foe; it does not diminish with time. All three uranium isotopes in depleted uranium are radioactive and produce alpha particles. Prolonged bombardment of lung tissue by alpha particles is known to cause cancer.

During 4 days of ground fighting, at least 300 tons of depleted uranium munitions were fired. An army report describing research

and hard-target testing states that up to 70 percent of a depleted uranium penetrator can become aerosolized when it hits a tank. Even if only 2 percent of the uranium burned up, then at least 6 tons of depleted uranium aerosol particles were generated. This is a huge amount, much of which would have become airborne over the battlefields. This amount in 4 days is more than 10,000 times greater than the maximum airborne emissions of depleted uranium allowed in the air over Albany in 1 month.

In a given region of a battlefield, hundreds of kilograms of micrometer-sized depleted uranium particles were generated suddenly by cannon fire from United States airplanes and tanks at formations of Iraqi armor. Thermocolumns from burning tanks and vehicles carried aloft these localized plumes of uranium particles and dispersed them far and wide by wind action over the battlefield.

Then unprotected U.S. service personnel inhaled and ingested quantities of depleted uranium particles into their lungs and bodies. They were never told about the health dangers of uranium particles. They were given no means to protect themselves.

Unprotected medical and other personnel were exposed to inhaling uranium dust from the uniforms of wounded allied and Iraqi soldiers. This massive exposure to depleted uranium aerosol particles on the battlefield raises many questions about depleted uranium and how it might have caused at least some of the health problems now being experienced by Gulf war veterans.

"Uranium and all its compounds are highly toxic, both from a chemical and a radiological standpoint." This quotation is from the Handbook of Chemistry and Physics, which has been a widely used reference text for generations of scientists and engineers: Chronic exposure to small concentrations of uranium is known to cause kidney failure. Depleted uranium is more than 99 percent Uranium–238, just a single isotope, and is always accompanied by two decay daughters that emit penetrating particles and gamma rays.

As gamma rays and energetic beta particles become absorbed in body tissue, they will traverse hundreds of body cells, potentially causing damage to genetic material in the nuclei of living cells. A biokinetic model developed by the International Commission on Radiation Protection explains how uranium microparticles can enter the body and spread to vital organs. This model shows that an acute intake of uranium particles can result in urinary excretions of uranium for several years afterwards.

After the war, many thousands of service personnel entered Iraqi tanks and armored vehicles that had been destroyed by depleted uranium penetrators, looking for souvenirs. They became contaminated. Others collected spent penetrators and made amulets from the dense, heavy-uranium metal. Wearing these amulets about their bodies, they unwittingly subjected themselves to penetrating gamma radiations from the uranium isotopes and the two decay daughters of Uranium–238.

They were not told that uranium is dangerous to health. After the war, 27 soldiers in the 144th Army National Guard and Supply Company worked on and in 29 U.S. combat vehicles that had been hit by friendly fire and become contaminated with depleted uranium. They worked for 3 weeks without any protective gear before being informed that the vehicles were contaminated.

In July 1991, the ammunition storage area at the United States Army base in Doha, Kuwait caught fire and burned. Four M1A1 tanks with depleted uranium armor were destroyed, along with 660 tank rounds and 9,720 35-millimeter, depleted uranium rounds. More than 9,000 pounds of depleted uranium burned up in the fire. U.S. troops were exposed to depleted uranium during the fire and subsequent cleanup operations. They wore no protective clothing or masks during or after the fire.

Approximately 3,500 soldiers were based here. Some of the soldiers reported cleanup consisted of using brooms and their bare hands. This is something that would make a qualified radiological worker shudder.

Twenty-two veterans still retain depleted uranium shrapnel in their bodies as a result of friendly fire incidents. They have become subjects for the first medical studies to assess health risks related to depleted uranium.

The promotion and sale of depleted uranium munitions to the armies and air forces of many nations guarantees that in future conflicts thousands of soldiers on both sides will inhale and ingest acute doses of uranium aerosols, and many in tanks or armored vehicles struck by depleted uranium penetrators will receive dangerous amounts of nonremovable uranium shrapnel in their bodies.

It has been reported in The Nation that the Department of Veterans' Affairs conducted a Statewide survey of 251 Gulf war veterans' families in Mississippi. Of their children conceived and born since the war, an astonishing 67 percent have illnesses rated severe or have missing eyes, missing ears, blood infections, respiratory problems, and fused fingers. The causes of these birth defects should be investigated.

The human cost of using depleted uranium munitions in conflicts is not worth any short-term advantage if it permanently contaminates the environment and results in irreparable damage to our service personnel and causes genetic defects in their offspring.

Speaking as a World War II veteran, I am troubled about the health of Gulf war veterans and the seeming lack of concern shown by the Department of Veterans' Affairs and the Army. They have refused to investigate the role of depleted uranium as a possible cause of Gulf War Syndrome.

In concluding, I urge this committee to make it possible for a truly independent investigation of depleted uranium to occur, because it was a major chemical and radiological poison that troops were exposed to during the Gulf war. Investigations should be undertaken by scientists and medical doctors not associated with the Department of Defense and who are knowledgeable about heavy metal and radiological poisons and their effects on human health. Gulf war veterans must also have a voice in organizing this effort. Thank you.

[The prepared statement of Mr. Dietz follows:]

Contamination of Persian Gulf War Veterans and Others by Depleted Uranium

Leonard A. Dietz
July 19, 1996

(Available at WISE Uranium Project home page on World-Wide Web at address http://antenna.nl/~wise/wuphome.html)

Abstract

We develop background information about depleted uranium (DU) and use it to describe a physical model of how on the battlefields in Kuwait and Iraq a large number of unprotected Gulf War veterans could easily have acquired dangerous quantities of DU in their bodies.

We examine how U-238, which comprises more than 99% of DU, decays radioactively, producing two decay progeny that are always present with it and add significantly to its radioactivity. The pyrophoric nature of uranium metal causes it to burn (oxidize rapidly) when heated by impact or in fires to form invisible aerosol particles that become airborne.

We refer to scientific measurements that have been made of the atmospheric wind-borne transport of uranium aerosols over distances up to 25 miles (42 km) from their sources. Stokes' well-known physical law helps to explain how airborne transport of DU particles can occur over large distances.

We describe how gamma rays and energetic beta particles become absorbed in body tissue and can traverse large numbers of body cells, potentially causing damage to genetic material in the nuclei of living cells.

We describe a biokinetic model developed by the International Commission on Radiation Protection that explains how uranium microparticles can enter the body and spread to vital organs. The model predicts that an acute intake of uranium particles can result in urinary excretions of uranium for years afterward.

We review estimates of the tonnage of DU munitions fired during the Gulf War. Even if only one or two percent of a low estimate of 300 metric tons of DU fired burned up, this would have produced 3000-6000 kg of DU aerosols.

This background information allows us to propose a plausible contamination model at a battle site. It consists of three steps:

1. A source of hundreds of kilograms of DU aerosols generated suddenly against concentrated Iraqi armor;
2. Widespread rapid dispersal of DU aerosol particles by wind action;
3. Inhalation and ingestion of DU particles by unprotected U.S. service personnel on the battlefield.

The U.S. military and its representatives claim that DU munitions are safe, but they have not publicly addressed health and safety issues that apply after DU munitions have been fired. Apparently the official view is that in a combat situation it is acceptable for unprotected personnel to be exposed to the combustion products of fired DU munitions and assume any health risks involved.

We mention that 22 U.S. service personnel have been reported to have suffered imbedded fragments of DU in their bodies from "friendly fire." More than 5 years after the Gulf War, few of these fragments have been removed and the long-term health situation for these veterans has not yet been determined. We note the astonishingly high incidence of serious birth defects in families of Gulf War veterans in the State of Mississippi.

Finally, we mention how commonly used DU flight control counterweights in aircraft and DU munitions can burn in intense fires and produce dangerous concentrations of airborne DU aerosol particles that can be inhaled and ingested.

Introduction

It has been reported widely in the press that numerous Persian Gulf War veterans have become ill with with Gulf War Syndrome. During the war they were exposed to toxic chemicals, experimental drugs, insect repellents and depleted uranium or DU (Ref. 1). Uranium is known to be highly toxic both chemically and radiologically (Ref. 2).

It has not yet been determined to what degree DU may have caused their illnesses and genetic defects in their children conceived and born after the war. Few veterans were aware that DU munitions were used until after they were exposed to uranium and became ill. Some were told about the gamma emission from DU but no one was told about the health dangers of inhaling fine particles of uranium oxide dust generated when a DU penetrator hits armor (Ref. 3). Eight days after the shooting stopped, a directive from Army Headquarters gave the first instructions to troops on how to treat radioactively contaminated vehicles (Ref. 4).

The main purpose of this paper is to develop a physical model of how easily many Gulf War veterans could have acquired dangerous quantities of DU in their bodies. To accomplish this we review the pyrophoric nature of uranium metal and its radioactivity. We show how readily uranium aerosol dust can be transported great distances by wind action in the atmosphere, pathways that DU aerosol particles can take into the body and become absorbed, and the tonnage of DU munitions fired during the Gulf War.

This information is used to construct a contamination model that explains how large numbers of soldiers very likely became contaminated on the battlefields in Kuwait and Iraq. We show how the U.S. military views the safety of DU munitions, and we close by mentioning some of the known exposures of U.S. soldiers to DU and noting the high percentage of severe birth defects in children conceived and born in many families of Gulf War veterans.

The Pyrophoric Nature of Uranium Metal

The pyrophoric nature of uranium metal is well known (Re. 2, Ref. 5). An estimate used by U.S. Army field commanders is that when a DU penetrator in a cannon round is fired at high velocity against armor, about 10% of it burns up and forms micrometer-size uranium oxide particles that can be inhaled or ingested (Ref. 6). However, a report by the Army Environmental Policy Institute (AEPI) describing research on hard target testing states "As much as 70 percent of a DU penetrator can be aerosolized when it strikes a tank..." (Ref. 7).

Uranium can burn in other ways to generate aerosol particles of uranium oxide. Because elemental uranium is pyrophoric, when DU metal is heated in air at a temperature of 500 deg. C it can oxidize rapidly and sustain slow combustion (Ref. 5). For example, the effects of fires at storage sites for DU munitions have been studied (Ref. 8). The burning of DU metal flight control counterweights at airplane crash sites and the possibility of exposing large numbers of people to kidney poisoning (nephrotoxicity) by uranium oxide particles has been studied by Parker (Ref. 9).

In 1992 an El Al Boeing-747 crashed into an apartment building in Amsterdam, Holland and burned intensely. Approximately 273 kg of DU in the tail of the 747 is unaccounted for; it burned and contaminated the surrounding area (Ref. 10).

Radioactive Decay of Uranium

We look briefly at the uranium decay series. Table I summarizes the isotopic composition of natural and depleted uranium. The isotopic compositions were measured in highly sensitive and accurate mass

spectrometers at the Knolls Atomic Power Laboratory (Ref. 11).

Table I. Isotopic composition of natural and depleted uranium in atom percent.

	U-234	U-235	U-236	U-238
Natural Uranium	0.0055	0.7196	0.0000	99.2749
Depleted Uranium	0.0008	0.2015	0.0030	99.7947

A trace of U-236 from reprocessed nuclear fuel may be present in some of the DU stockpile. The alpha activity in DU is about 43% less than it is in natural uranium because there is less U-234 and U-235, but DU always occurs in highly concentrated form and this more than makes up for its lower alpha activity. In contrast, natural uranium occurs in concentrations of 1-3 parts per million by weight in soils, where it is locked up in non-metallic form in minerals and is relatively inert to chemical action there.

Only the first three isotopes in the uranium decay series or chain headed by U-238 are important in determining the radioactivity of DU (Ref. 12). Uranium–238 decays into thorium-234 (Th-234), which decays into protactinium-234 (Pa-234), which decays into U–234, etc. down the decay chain. The 246,000 year half life of U–234 is too long for it to decay much during our lifetimes and produce significant numbers of decay progeny.

The U-238 decay chain is broken during the chemical reduction of uranium hexafluoride into DU metal and is broken again during the melting and processing of the metal into a penetrator. To determine the maximum time it takes to regain equilibrium in the partial decay chain, we assume a solid sample of uranium that initially contains only the U–238 isotope, i.e. no decay progeny. Using Bateman's equations, (Ref. 13). we calculate the growth of Th–234 and Pa-234 activities as a function of elapsed time in weeks. The results are given in Table II.

Table II. Radioactivity (disintegrations/second) in 1 gram of U-238 with no decay progeny initially present. Half lives used: U-238 = 4.47e9 years; Th-234 = 24.10 days; Pa–234 =1.17 minutes. 6.69 hours (two decay states); U-234 = 2.46e5 years (Ref. 14). Scientific notation is used, i.e. 2.46e5 = 246000.

Weeks	U-238	--> Th-234	--> Pa-234	--> U-234
0	12,430	0	0	0.000
1	12,430	2,270	2,150	0.000
5	12,430	7,890	7,840	0.001
10	12,430	10,770	10,750	0.004
15	12,430	11,830	1,820	0.007
20	12,430	12,210	12,210	0.010
25	12,430	12,350	12,350	0.013
30	12,430	12,400	12,400	0.017

After 25 weeks, Th-234 and Pa-234 have reached 99.4% of the decay rate of U-238 and for practical purposes have reached secular equilibrium with U–238, their parent isotope. Secular equilibrium means that the decay progeny of U-238 are being replaced at the same rate they are decaying; after 25 weeks all three isotopes are decaying at approximately the same rate. This is a maximum time; in reality, equilibrium will be

reached much faster, since these two isotopes can never be separated totally from U-238. The isotope U-238 emits alpha particles and also emits some gamma rays. Its decay progeny Th-234 and Pa-234 each emit beta particles and gamma rays. An alpha particle is a fast helium atom with its two electrons removed, a beta particle is a high-speed electron and a gamma ray is like an X-ray.

From this analysis we conclude that in a solid sample of DU, six months at most after manufacture of a DU penetrator, or DU armor for a tank, or DU particles in a person's body, substantial additional radiation in the form of beta particles and gamma rays always will be present. In fact, most of the penetrating gamma radiation and all of the penetrating beta radiation from DU comes, not from uranium, but from the decay progeny of U-238 (Ref. 15). In a year, only one-thousandth of a gram (1 milligram or mg) of DU generates more than a billion alpha particles, beta particles and gamma rays.

The U.S. Army has investigated the generation of DU aerosols in armored vehicles hit by DU cannon rounds. Their investigators report "...that personnel inside DU struck vehicles could receive a dose in the 'tens of milligrams' range due to inhalation" (Ref. 16). This exposure results in an acute dose of uranium.

Gamma rays become absorbed in body tissue as follows. If their energy exceeds 40 keV, part of the gamma-ray energy is transferred to an atomic electron, setting it in high-speed motion (1 keV = 1000 electron volts energy). The remaining energy is carried off by a new gamma ray. This process, called the Compton effect, repeats until the gamma ray has an energy below about 40 keV where the photoelectric effect dominates and the remaining energy can be transferred to a photoelectron.

For example, using Gofman's method,(Ref. 17) one can calculate that an 850 keV gamma ray absorbed in body tissue will produce a packet of high-speed Compton electrons and a fast photoelectron that on average can traverse 137 body cells. By contrast, according to Gofman, X-rays commonly used in medical diagnosis have a peak energy of 90 keV and an average energy of 30 keV (Ref. 17) A 30 keV X-ray in body tissue can be converted into a photoelectron of this energy, which on average can traverse only 1.7 cells. Ionization along the tracks of high-speed electrons in tissue can cause damage to genetic material in the nuclei of cells.

Thus, a high energy gamma ray from Pa-234 is much more penetrating than a typical medical X-ray and can damage far more living cells. The many 2.29 MeV beta particles emitted by Pa-234 are extremely penetrating in body tissue (1 MeV = 1 million electron volts energy). Referring to the experimental data given by Gofman (Ref. 17), each one of these beta particles can traverse more than 500 body cells.

Alpha, beta and gamma radiations produce the same biological effects on cells and organs, and much of their radiation damage to body tissue can accumulate over the time of exposure (Ref. 18). Therefore, it seems reasonable that not only the continuous radiation of body tissue by alpha particles from U–238, but the energetic beta particles and gamma rays from its decay progeny Th-234 and Pa-234 must also be considered when assessing possible cancer risk and genetic damage.

Airborne Transport of Uranium Particles

The fallout range of airborne DU aerosol dust is virtually unlimited. These micro-particles can be inhaled and ingested easily and that makes them dangerous to human health. Environmental assessments for sites which process DU or test fire DU munitions typically downplay the potential for widespread fallout of DU particles.

For example, one such environmental impact study in 1992 by the U.S. Army Ballistics Research Laboratory (Ref. 19) states, "Because of the mass and density of the DU particle, it only travels short distances when airborne. These two factors alone preclude the off-site release of DU." This is not true for micrometer-size particles of uranium metal or its oxides. In fact, the transport of airborne DU aerosol particles was well

known long before the Army Ballistics Research Laboratory environmental impact study was written, since in 1976 it had been measured up to a distance of 8 km (Ref. 20). What may not have been fully appreciated in 1976 was that DU aerosol particles could be transported by wind action over much greater distances.

In 1979 the author worked at the Knolls Atomic Power Laboratory (KAPL) in Schenectady, New York. While trouble shooting a radiological problem, he and his colleagues in the mass spectrometer component accidentally discovered DU aerosols collected in environmental air filters exposed at the Knolls site (Ref. 21). The origin of the DU contamination proved to be the National Lead Industries plant in Colonie, 10 miles (16 km) east of the Knolls site, on the western boundary of the city of Albany, NY. A local newspaper reported that NL was fabricating DU penetrators for 30–mm cannon rounds and airplane counterweights made of DU metal (Ref. 22).

A total of 16 air filters at three different locations covering 25 weeks of exposure from May through October of 1979 were analyzed; all contained trace amounts of DU. Three of these air filters were exposed for four weeks each at a site 26 miles (42 km) northwest of the NL plant. This is by no means the maximum fallout distance for DU aerosol particles.

Totally unrelated to the discovery of DU in KAPL air filters, in February 1980, a court order by NY State forced NL to cease production, because they exceeded a NY State radioactivity limit of 150 microcuries for airborne emissions in a given month (Ref. 22). The plant closed in 1983 and is now being decontaminated and dismantled. The 150 microcuries corresponds to 387 g of DU metal. For comparison, one GAU-8/A penetrator in an aircraft 30-mm cannon round contains 272 g of DU metal (Ref. 5).

Using a special fission track analysis technique, 26 uranium-bearing particles were extracted from several air filters exposed at KAPL and were analyzed separately for their uranium isotopic content (Ref. 11) Four particles contained pure DU. They were approximately 4-6 micrometers in size, three were irregularly shaped and the fourth was a 3.8 micrometer diameter sphere. Probably it solidified from a molten state as uranium dioxide. The other 22 particles were enriched uranium associated with the radiological trouble-shooting problem.

This widespread trace contamination of DU in the atmosphere was less than one percent of allowable limits. Its presence in the air filters did not concern us nearly as much as the sizes of the DU particles that were born ten miles by the wind from Albany to KAPL. The four DU particles were near the upper end of the respirable size range, which is about 5 micrometers. Respirable means that particles will pass through the upper respiratory airway to the lung and become deposited in various interior regions of the lung, where many will remain for many years. A 5 micrometer uranium dioxide particle can cause a high, localized yearly radiation dose from energetic alpha particles to lung tissue; it is a radioactive hot spot in the lung (Ref. 23).

The density of uranium metal is 19 grams per cubic centimeter; for uranium dioxide it is 11 grams per cubic centimeter, equal to the density of lead. How can a uranium dioxide particle with this density, or a uranium metal particle with a density 1.7 times that of lead remain airborne long enough to be transported by wind 26 miles (42 km)? It might seem a daunting challenge to answer this question, but a complicated physical theory is unnecessary.

Just as a parachute jumper in a free fall through the lower atmosphere quickly reaches a constant terminal velocity of approximately 120 mph, so too a micrometer-size uranium particle falling under gravitational attraction through still air will reach a constant terminal velocity that is determined by its size, density, geometrical shape and air viscosity.

Stokes' law provides an accurate and convincing scientific explanation of how micrometer-size DU particles can remain airborne for many hours. This physical law is well known to scientists and engineers who study fluid dynamics. It was published in 1846 and 1851 by Sir George Stokes, and is described in introductory textbooks on fluid flow (Ref. 24). It is given by the expression

$$V = \frac{2G\,R^2\,(S - A)}{9C}$$, where

R^2 means R squared,

G = 980.4 centimeters per second squared is the acceleration of gravity,

R = the radius of the sphere in centimeters,

S = the density of the sphere in grams per cubic centimeter,

A = 1.213e-3 grams per cubic centimeter is the density of air at one atmosphere and 18 deg. C,

C = 1.827e-4 poise is the viscosity of air at one atmosphere and 18 deg. C.

The terminal velocity V is in centimeters per second if G, R, S, A and C are in the units shown. Stokes' law allows one to calculate the terminal velocity of a microsphere of uranium metal or uranium oxide of known radius and density falling through still air.

Stokes' law is valid for fluid flow described by a Reynolds number of 0.1 or less (Ref. 24). Experiments confirm this upper limit (Ref. 25) The dimensionless Reynolds number Re for a sphere is given by

$$Re = \frac{2\,RAV}{C},$$

where the terms are defined above. A 10 micrometer diameter uranium metal sphere falls at 5.7 cm/sec in still air and Re = 0.038, which is much less than 0.1. Therefore, Stokes' law is accurate for all respirable spherical uranium metal or oxide particles 10 micrometers or less in diameter falling through air. Table III lists the fall rates for a range of particle sizes.

Table III. Terminal (constant) velocities for uranium dioxide spherical particles in still air. Diameters are in micrometers.

dia.	cm/sec.	ft./hr.
5.0	0.82	97
4.0	0.52	62
3.0	0.30	35
2.0	0.13	15
1.0	0.033	4
0.5	0.0082	1

Irregularly-shaped microparticles will fall more slowly than a sphere of the same density and weight. Depleted uranium particles one micrometer or smaller are virtually floating in air and can remain airborne for a very long time. The 3.8 micrometer dia. spherical uranium dioxide particle analyzed at KAPL had a fall rate of 56 ft./hr. It had to reach a height of only 200 ft. in the warm exhaust plume from the National Lead plant for a gentle breeze averaging 3 mph to carry it 10 miles (16 km) to KAPL.

Fallout range can be increased greatly by two more natural phenomena. First, frictional forces in the air or emission of an alpha particle from a uranium atom will electrostatically charge a DU particle. For example, it is well known that a high velocity ion striking a metal oxide surface will dislodge a pulse of secondary electrons from the surface (Ref. 26).

An alpha particle is a high velocity helium ion, and it will generate a large number of secondary electrons below the surface of an uranium oxide particle as it passes through the surface. Many of the momentarily-free electrons just below the surface will escape from an airborne uranium oxide particle, leaving it in a positively-charged state.

Like an electrostatic precipitator collecting dust in a room, an electrically-charged uranium dioxide particle and an oppositely-charged dust particle will attract each other and join together. The average density of the two particles together will be substantially less than 11 grams per cubic centimeter and the fallout range will be greatly increased.

Fallout particles of DU also can become attached to sand or dust particles on the ground and then become resuspended in the air by wind or vehicle action and transported to new locations (Ref. 27). Desert sand in the Persian Gulf region is extremely fine (Ref. 28). Second, random motions of the atmosphere of a few cm/sec are of the same order of magnitude as the terminal velocities of micrometer particles of DU oxide or metal falling through air.

Pathways of DU and Its Radiations into the Body

Routes of intake or pathways of uranium particles into the body include the respiratory tract, the gastro-intestinal tract and the skin, through abrasions or wounds. The International Commission on Radiation Protection (ICRP) has developed a biokinetic model that describes the behavior of uranium within the human body (Ref. 29). The model takes into account aerosol particle size, chemical form, and the excretion rates of absorbed uranium from individual vital organs and bones. Radioactive particles reach the gastrointestinal tract by ingestion and by transfer from the respiratory tract. The model shows that for an acute intake of uranium aerosol particles of uranium dioxide or U3O8, urinary excretion of the inhaled uranium can continue for years.

Exposure to gamma rays emitted from DU is another pathway into the body. Crews are exposed to the equivalent of one chest X-ray for every 20-30 hours they spend in an Abrams tank armed with DU ammunition (Ref. 30). The U.S. Army measured a gamma dose rate of 250 millirems per hour at the surface of a penetrator (Ref. 31). This dose rate is consistent with the 233 millirads per hour dose rate for an unspecified mass of DU listed on a U.S. Department of Labor Material Safety Data Sheet issued to Nuclear Metals, Inc. (Ref. 32). For gamma rays, the rad and rem dose units are equal. At body contact, the 250 millirems per hour is equivalent to a dose rate of up to approximately 50 chest X-rays per hour. Whole penetrators or large fragments of penetrators fired from tank cannon and left on a battlefield have this amount of surface radioactivity.

Estimates of Tonnage of DU Munitions Fired

The actual tonnage of DU munitions fired during the Gulf War is difficult to ascertain. During the war all battlefield news was censored and the expenditure of DU by A-10 attack aircraft was classified (Ref. 33). It has been estimated that these aircraft fired about 95% of the DU munitions used during Desert Shield and Desert Storm (Ref. 34). The U.S. Army now claims (Ref. 35) that "More than 14,000 large caliber DU rounds were consumed during Operations Desert Shield/Desert Storm. As many as 7,000 of these rounds may have been fired in practice. Approximately 4,000 rounds were reportedly fired in combat. The remaining 3,000 rounds are losses that include a substantial loss in a fire at Doha, Kuwait.

The 14,000 rounds contained about 60 metric tons of DU. William Arkin estimates from documents released under the Freedom of Information Act that approximately 300 metric tons of DU littered the battlefields of Kuwait and Iraq after the war (Ref. 34). The LAKA Foundation estimates the total as 800 tons (Ref. 36). Allowing for DU projectiles missing their targets, even if only one or two percent of the lower estimate of 300 metric tons burned up, then 3,000,000-6,000,000 grams of DU aerosol particles could have

become airborne over the battlefields—a huge amount.

Contamination Model

We can now propose a plausible model of how veterans became contaminated with DU during the Gulf War. It consists of a sequence of three steps:

1. **Source**—in a local area of a battlefield, hundreds of kilograms of micrometer-size DU particles were generated suddenly by cannon fire from U.S. airplanes and tanks at concentrated formations of Iraqi armor. Thermal columns from burning tanks and vehicles then carried aloft these localized plumes of DU aerosol particles.

2. **Dispersal**—Clouds of DU aerosol particles were dispersed far and wide by wind action over the battlefield and, based on the KAPL measurements, the fallout range of these uranium micro-particles could be up to 25 miles (42 km) or more (Ref. 11).

3. **Inhalation and Ingestion**—Unprotected U.S. service personnel could inhale and ingest huge numbers of DU particles into their lungs and bodies, where much of the DU could become absorbed in vital organs and bones. The ICRP biokinetic model explains how uranium aerosol particles can enter the body and become absorbed (Ref. 29).

The U.S. Army and the Department of Veterans Affairs have shown an unwillingness to investigate health issues associated with the toxicity and radioactivity of inhaled and ingested DU aerosol particles that have become absorbed in the body. Both have refused to test large numbers of veterans for the presence of DU in their bodies; so far only a handful have been tested. According to Laura Flanders, as of January, 1995, at least 45,000 soldiers deployed to the Persian Gulf during the war are suffering from symptoms connected with their service (Ref. 37).

Workers in DU industrial processing plants and people living in communities surrounding these plants also have been contaminated by fallout of DU particles (Ref. 22). How rapidly contamination takes place depends on the magnitude of the airborne concentration and particle size of the uranium dust. The smaller the particle, the easier it can enter the body.

In written testimony prepared for a 1982 New York State hearing on NL Industries, Dr. Carl Johnson, a principal investigator of the National Cancer Institute Project, stated that some of the workers at the NL plant had concentrations of uranium in their urine as high as 30 picocuries/liter (77 micrograms of uranium/liter). He said this concentration level indicated a very heavy body burden of uranium (Ref. 38).

How the U.S. Military Views the Safety of DU Munitions

In a letter to Senator Sam Nunn, a representative of the U.S. Air Force stated, "...these projectiles are no more hazardous to store, transport, or employ than those composed of lead or copper" (Ref. 39). This view is echoed in the U.S. Army report to Congress that states, "The health risks associated with using DU in peacetime are minimal. This includes risks associated with transporting, storing and handling intact DU munitions and armor during peacetime" (Ref. 40).

Neither the Air Force nor the Army has publicly presented an analysis of the health risks to soldiers and to others who inhale or ingest radioactive fallout particles of DU, or the health risks of living in an environment contaminated with DU after these munitions have been fired—these are the real safety issues they ignore. Furthermore, a General Accounting Office report to Congress states, "...Army officials believe that DU

protective methods can be ignored during battle and other life-threatening situations because DU-related health risks are greatly outweighed by the risks of combat" (Ref. 41).

The Army must know that it would be extremely difficult to provide breathing masks that can efficiently remove all of the respirable DU particles from air breathed by soldiers. Even if highly efficient air filters are used by troops, their surroundings will still be contaminated. The surface of the ground, vegetation, equipment, uniforms and other garments contaminated with DU particles will become secondary sources of airborne DU aerosols whenever they are disturbed or moved, thereby presenting an insurmountable radiological containment and decontamination problem on the battlefield.

In the AEPI report, (Ref. 42) the Army judges it an acceptable risk if its personnel become exposed in an unprotected fashion to the combustion products of fired DU munitions on the battlefield or elsewhere. This report contains much technical information about DU, but many of the assertions and conclusions in the report are not supported by the technical and scientific data presented. A rebuttal to the AEPI report pointing out some major inconsistencies in the Army report has been published by the Military Toxics Project (Ref. 43).

Exposure of U.S. Soldiers and Illnesses in Their Families

Thirty-six U.S. soldiers, including 22 with embedded fragments of DU in their bodies, have sought or reported for medical treatment (Ref. 44). They were in vehicles hit by DU munitions. Another report states there were 35 casualties and 72 wounded in crews of U.S. tanks and Bradley Fighting vehicles in so-called "friendly fire" incidents (Ref. 45). This includes the 36 above and is the total number of service personnel officially admitted to have been exposed to significant quantities of DU aerosol dust and DU fragments during the fighting.

On an NBC Dateline program, (Ref. 6) Sgt. Daryll Clark describes how he and twelve others were in an advanced position in the desert when someone radioed them that 20 Iraqi tanks were approaching his forward radar unit. He called for air support, and shortly a flight of A-10 Warthogs arrived and destroyed all of the tanks with DU-tipped 30-mm cannon rounds.

Clark describes how he and the men with him were coughing and choking on smoke from the burning tanks, but mixed with it was DU aerosol dust, which he and the others breathed. He has had chronic respiratory problems since the war and his daughter Kennedy was born in September 1992 with purple welts called hemangioma covering not only her face and body, but some internal organs as well. Kennedy has serious breathing problems and was born without a thyroid. Clark stated that a geneticist told him that he could have ingested some radiation and that it could affect sperm cells. Almost three years after his exposure to DU, Clark's urine tested positive for uranium.

Army nurse 1st. Sgt. Carol Picou also is featured in the NBC documentary. She and seven other women in her medical team were in a forward position, ahead of the main U.S. forces and surrounded by burning Iraqi tanks and vehicles when they stopped and became exposed to DU from the burning destroyed Iraqi armor. Doctor Thomas Callender of Lafayette, Louisiana has examined Picou and said on the program that her outcome bears a striking similarity to other individuals who had exposures to ingested radioactive elements. Picou has been given a medical discharge.

The 7 medical personnel with Picou and the 12 soldiers with Clark probably became contaminated with DU. These 21 soldiers are not included in the official list of those recognized by the U.S. government as having been exposed to DU. Given the large tonnage of uranium penetrators in cannon rounds that were fired on the battlefields in Iraq and Kuwait, it is likely that many thousands of other soldiers also became contaminated with DU. The U.S. Army and the Department of Veterans Affairs balk at giving urinalysis tests and "in vivo" tests (whole-body counting of gamma rays) to measure the amount of DU in the lungs and other

body organs of Gulf War veterans.

An astonishingly high rate of birth defects in the families of Gulf War veterans is especially troubling. For example, Laura Flanders reports that the Veterans Administration conducted a state-wide survey of 251 Gulf War veterans families in Mississippi (Ref. 46). Of their children conceived and born since the war, 67% have illnesses rated severe or have missing eyes, missing ears, blood infections, respiratory problems and fused fingers. Flanders goes on to say that the birth defects are consistent with the effects of radiation from DU and infection from sand fly bites. Others blame experimental vaccines, chemical warfare pills, the insect repellent DEET and smoke from oil well fires for causing birth defects.

Conclusion

We have shown how easily micrometer particles of DU can spread over a large region and poison many people both radiologically and chemically. The promotion and sale of DU munitions by U.S. arms manufacturers (with U.S. government approval) and by other arms manufacturers to the armies and air forces of many nations will guarantee that in future conflicts thousands of soldiers on both sides will inhale and ingest acute doses of DU aerosols, and many in armored vehicles struck by DU penetrators will receive dangerous doses of non-removable uranium shrapnel in their bodies.

The human cost of using DU munitions in conflicts is not worth the perceived short-term advantages, especially if it results in U.S. veterans and others becoming ill and in genetic defects in their offspring. A comprehensive epidemiological study should be made of all Gulf War veterans and their families, searching for evidence of residual DU in their bodies and for causes of genetic defects in their children. The health issues associated with DU munitions should be investigated and evaluated by independent medical and scientific experts separated completely from the Department of Defense, Department of Veterans Affairs, National Laboratories, U.S. military services and their contractors.

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Mr. SHAYS. Thank you very much. Dr. Durakovic, you have the floor.

Dr. DURAKOVIC. Mr. Chairman, I welcome and am grateful for this opportunity to testify today.

I am a doctor of medicine with a specialty in nuclear medicine, and I also have a doctorate, Ph.D., in nuclear biophysics. My entire scientific and professional life has been dedicated to radioisotope toxicology, in which capacity I served the Government of the United States for over 17 years, being the head of the Nuclear Medicine Department at Walter Reed Hospital, working for the Defense Nuclear Agency, and later for the VA system.

In 1991, 24 veterans were referred to my clinic in Wilmington, DE from the Ventnor Clinic in New Jersey because they were contaminated with uranium in the Persian Gulf. If it was not for my clinic that encountered that population of 24 patients, perhaps we would not be sitting here discussing medical effects of uranium in the Persian Gulf because that was the first referral of the veterans who qualified for the Nuclear Medicine Clinic. Most of them in different parts of the country were seen by general practitioners who have hardly any expertise in handling internal contamination.

So due to the lucky circumstances, those patients were seen by me at Wilmington VA Hospital, and I took their story very seriously, indeed, because my exposure to uranium contamination previous to that time was only with experimental animals, because I did lots of research in the experimental animals dealing with transuranium elements, plutonium, americium, and so on. My works had been published 25 years ago on uranium and transuranium elements.

These soldiers presented with a host of clinical symptoms, ranging from respiratory ailments to renal disease. Some of those patients underwent several surgical procedures to handle their kidney problems. Their problems also included hepatic, gastrointestinal, and endocrine disease. Therefore, I simply focused my attention to the probability of symptomatology related to the endogenous incorporation of uranium in those patients, for which reason I took the very simple route of attempting etiological diagnoses in those unfortunate patients.

Out of 24 patients, I dealt directly with 14 of them because 10 did not show up for my follow-ups. My first line of action was to send them for the objective evaluation of the whole-body counting of radioactive uranium. Since our facility did not have the capacity to deal with the whole-body counting of endogenously incorporated radioisotopes, I sent them to the VA Hospital in Boston, where there was a whole-body counter, unfortunately outdated and not sensitive.

Those patients underwent the whole-body counting with inconclusive results. I suggested to the doctors of the VA Hospital in Boston to improve their methodology by buying more sensitive crystal, which they applied to the Department of Defense, and soon after, the work was discontinued under unexplained circumstances.

Since whole-body counting did not yield any information about the etiological cause of my patients' symptomatology, I suggested to the VA system that we go for another line of action about etiological diagnosis of their problems. I suggested that the patients be

sent to Sandia National Lab in New Mexico, where I am very familiar with their work of uranium in the lungs. That has never been done.

Furthermore, I suggested that urine samples be sent to the Radiochemistry Lab in Aberdeen, MD, and samples were collected, but they never reached Maryland, and they never reached Aberdeen Proving Grounds, so urine analysis is nonexistent. In the case of my 24 patients, there was only 1 urine sample that was analyzed, with inconclusive evidence.

So I consider it very mysterious, the disappearance of the samples of the urine, which were very carefully collected and supposedly sent to Aberdeen Proving Grounds by the VA Hospital in Wilmington, DE.

The third action that I proposed to the Veterans' Administration was to do biopsy samples of the bone tissue of those patients because we know that uranium can be easily detected by autoradiography or even by visualization of a single atom of uranium, which is a big atom, about one Angstrom in size. It can be seen by the specialized microscopic analysis.

None of my recommendations was ever followed, and not a single patient referred to me has been analyzed for the etiological cause of their symptoms. Every conceivable road block was put in my line of management of those patients. I was ridiculed. There were road blocks, and there were obstacles throughout 7 years of my attempt to properly analyze the problems of those patients. I have to quote to you, although it is not my vocabulary or my dictionary, that the chief of staff of my hospital said it is "half-assed research." And it was openly and obviously discouraged that any work done with them.

Nevertheless, there was a Uranium Registry in our hospital, which consisted of taking blood pressures, temperatures, and the pulse rates, and perhaps in some cases, of the lung x rays, which really is far away from proper analysis of the patients for the deeply incorporated uranium.

My plan of management has failed because of the total absence and total lack of interest on the part of the Veterans' Administration to do anything for those unfortunate patients, to analyze why they suffer from the host of the symptoms and what might be the role of uranium in the misery of those patients. Why it was done, I do not know; but I do know that I received several telephone calls from the Department of Defense suggesting to me that this work will not yield any meaningful information and should be discontinued. I have telephone numbers of the references if you desire to see them.

Lost records is another thing, because samples of the urine disappeared but also the records of those patients disappeared, and they were found much later when pressure was put on the VA Hospital.

Now, we are facing a big dilemma in the political, scientific, and professional environment of the United States of America. Is uranium responsible or a real objective cause of a part of the disease of the Persian Gulf veterans, or is it not? The question is very simple, and it can be very simply addressed.

The only thing to do is to do a proper, objective, expensive analysis of the samples of those patients, which has to be conducted not by the charlatans which are present in the Veterans' Administration's offices with the big names of the Gulf veterans uranium groups and clinics and so on and which are populated by people who have no basic knowledge of radiation toxicology, nuclear medicine, or internal incorporation of radioactive uranium.

Now, if uranium is analyzed by these objective methods, we will be able to say whether those patients are related to uranium in their symptomatology or they are not. The studies are very expensive, but I think our country owes it to the veterans who served in the Gulf.

I was a commander of the 531st Army Detachment in the time of Desert Shield. At that time, when I was deployed for the Desert Shield operation, everybody knew my qualification as being an international expert for uranium and transuranium elements. Nobody volunteered to me the information that my expertise might be needed in the Gulf because of the possible use of depleted uranium shells.

I am not questioning it, but I am just saying that we can easily answer the question of the relationship between uranium and the Persian Gulf sickness if we take this issue seriously and if we analyze in this country what is the probability of a connection between uranium and the symptoms.

This country has the capacity. It has sophisticated laboratories. It has professional people who are at the highest level of expertise, and there is absolutely no excuse not to proceed with my method of management.

Total lack of etiological diagnosis, in my opinion, is a shame for all of us because I know for a fact that the soldiers of 144th Transportation Company of New Jersey who worked on those tanks in Saudi Arabia, they never have been informed about the probability of radioactive isotopes in their environment. They never wore protective clothing, never wore the masks, and never wore the dosimeters.

Battle-damage assessment team came in the summer of 1991 to Saudi Arabia, and they were dressed like astronauts, having sophisticated detecting instruments and detecting 0.6 to 1 Rad in one single measurement in those tanks, which is a very high dose. But the veterans were not informed that they were in a radioactive environment.

I am going to conclude my statement at this point with an emphasis that oppression has been exercised in the Veterans' Administration system against professionals like myself who wanted to come to the end of the story, and I am going to quote President Thomas Jefferson, who said: "I swear upon the altar of God eternal hostility toward any oppression over the mind of man."

I think we should take heed of the great President Jefferson, and try to eliminate obstacles to the proper diagnostic management of the Gulf veterans who have been exposed to depleted uranium. Thank you, Mr. Chairman.

[The prepared statement of Dr. Durakovic follows:]

Medical Implications

During the Persian Gulf War, Depleted Uranium was used in anti-tank armor piercing projectiles and in tank armor plating to increase, respectively, penetration capability and resistance to penetration. DU is still very radiotoxic and is highly pyrogenic when ignited, which happens when artillery shells are fired. The intense, searing flame caused by ignition of the uranium not only aids in penetration of tank armor, but also liberates the uranium into the environment making it available for internal contamination via inhalation and ingestion. Tanks made of DU armor and hit by DU shells also ignite in this way creating the same effect, i.e. friendly fire.

Depleted Uranium enters the body via inhalation, ingestion, and absorption through open wounds or imbedded shrapnel. Uranium is water soluble and can be transported throughout the body. The alpha particle released by decay of the uranium atom gives up its large amount energy in a distance no larger than a couple of microns. Causing breaks and ionization of molecules, it is capable of destroying proteins, enzymes, RNA, and damaging DNA in many different ways, including double strand breaks. This kind of damage in the reproductive organs can lead to genetic hazards which can be passed on from generation to generation. Soluble uranium compounds cause mainly chemical damage to the proximal convoluted tubules of the kidney. DU is incorporated into bones where it can have hematopoetic effects as well as causing leukemia. In the lung, DU damages the alveoli. Since DU can cross the placenta, it can create massive problems for the radiosensitive tissues of the fetus. Damage to the fetus may lead to somatic malformations including shortened limbs, damage to the CNS, cardiovascular, and muscular problems. Other effects associated with DU poisoning are: emotional and mental deterioration, fatigue, loss of bowel and bladder control, as well as numerous forms of cancer. Such symptoms are increasingly showing up in Iraq's children and among Gulf War veterans and their offspring. Depleted Uranium also has physiological effects associated with its heavy metal properties. Although most of the ingested DU will be excreted through urine or feces shortly after exposure, a significant quantity of DU will remain in the body. Because of the chemical and radiological toxicity of DU, the small number of particles trapped in the lungs, kidneys, and bone greatly increase the risk of cancer and all other illnesses over time. These small amounts of DU left in the body are a constant source of low-level radiation that damages cell structure. Tons of DU were left in the Gulf region in the form of spent munitions and destroyed tanks which allowed DU to enter the air, water, and reside on the ground, thereby becoming part of the food chain. In this way, DU will continue to plague the health of the people both Iraqi and American who remain in the region for many decades.

According to the results of a survey of 10,051 GW veterans, conducted by Vic Sylvester and the Operation Desert Shield/Desert Storm Association between 1991 and 1995, 82 % of GW veterans entered captured Iraqi vehicles. This would suggest that 123,000 soldiers have been directly exposed to DU.

In 1991, 24 soldiers from the 144th Transportation and Supply Co., New Jersey, were referred to me by Ventnor Clinic in my capacity as Chief of Nuclear Medicine, VA Medical Facility, Wilmington, DE. All of the veterans were referred to me for the opinion and diagnostic assessment of their DU body burden. My expertise is in the internal contamination of radioisotopes and I was the only published researcher in the federal VA system with research on transuranic elements at the time these soldiers were referred to me. Although I personally served in Operation Desert Shield as Unit Commander, my expertise of internal contamination was never used because we were never informed of the intended use of DU prior to or during the war.

The research on the effects of transuranic elements in the human system is not well known as prior accidents have dealt with many isotopes (Chernobyl) and the Persian Gulf War deals with only one actinide, i.e., uranium.

From January 1991 until August 1991, these soldiers were on a tour of active duty in Saudi Arabia and after the ground war started were located at the KKMC, King Khalid Military Camp, where it was their duty to unload battle damaged MAIA tanks, Bradleys, and M113 tanks destroyed by DU armor piercing shells from friendly fire of helicopters, airplanes, and other tanks. The soldiers worked on these tanks During this time, soldiers had constant contact with these vehicles. Those that were required to receive the vehicles actually lived very near them, ate lunch on top of them, and cooled themselves inside of them. They had been told not to let anyone photograph or take souvenirs from them so they kept the tanks close at hand. On March 10, 1991, a Battle Damage Assessment Team dressed in full radioprotective clothing arrived, stating that they were from Washington to assess the radioactivity of specific tanks. They reviewed the tanks for four days, fully dressed in the 90 degree temperatures. At the conclusion of the assessment, the soldier in charge of the crew, required to move the equipment, was told that the tanks were "hot", to mark them with the atomic symbol and not to let people go near them. The Assessment Team had detected .26 to 1.0 rad inside the tanks. After that evaluation, the soldiers were told to cover the tanks with tarps and not to photograph them. The Team stated that the tanks were not dangerous to those required to work in their environment. One soldier was given an outdated dosimeter which began to detect radiation right away despite the fact that it was long past its expiration date.

My diagnostic strategy consisted of their referral to the VAMC of Boston to the internationally known expert on low energy detection of internal contamination, Dr. Belton Burroughs who with Dr. David Slingerland performed whole body count of Uranium 238 on several of the referred veterans. It was found by a rather insensitive and outdated whole body counter that 14 of the 24 patients referred contained decay products of radioactive uranium. On the basis of this more sensitive equipment, specifically a Germanium crystal was applied for the project which was then terminated. All work that was conducted on behalf of DU contamination was coordinated through the Persian Gulf Registry of the Wilmington VA hospital. All records were subsequently lost.

Spectroscopic Analysis

The urine samples of these same patients were sent to the US Army Radiochemistry Lab in Aberdeen Maryland. Again, some samples never reached the lab and the results of those that did were supposedly lost.

According to my experimental research on lab animals and extensive review of the literature, uranium can hardly be detected by the external methods including whole body counting and urine analysis. Therefore I recommended that the veterans should be sent to the SANDIA National Labs in Albuquerque, NM which specializes in the pulmonary pathways of contamination with transuranic elements.

Furthermore, an objective analysis in the main site of uranium incorporation which is the skeletal system, should be performed by an autoradiographic analysis of the skeletal deposition of uranium by the bone necropsy specimens.

Neither of the above recommendations were followed because no one took the veterans' illnesses seriously. Two of the 14 soldiers have died since returning from the Persian Gulf. A recommendation for autopsy which should have included autoradiographic analysis of the skeletal deposition of uranium, was ignored.

The 144th Transportation and Supply CO has since been scattered all around the United States, making it impossible for unified testing and analysis.

Due to the current proliferation of DU weaponry, the battlefields of the future will be unlike any battlefields in history. Since the effects of contamination by uranium cannot be directed or contained, uranium's chemical and radiological toxicity will create environments that are hostile not only to the health of enemy forces but of one's own forces as well. When released, DU aerosol particles are carried on the winds, their range as fallout virtually unlimited and as they migrate contaminate air, soil, and water. So released, it is available for uptake by humans via inhalation, ingestion, or absorption. In such a toxic environment, fighting personnel will find themselves victims of their own weapons as well as those of the enemy. Due to the delayed health effects from internal contamination of uranium, injury and death will not always be immediate to the battle, but will remain lingering threats to "survivors" of the battle for years and decades into the future. The battle field will remain a killing zone long after the cessation of hostilities. Environmental contamination will linger for centuries posing an ongoing health threat to the civilians who reclaim the land and subsequent generations.

Asaf Durakovic

Mr. SHAYS. I thank all three of our panelists. We have really not focused in on, and this is the 10th hearing we have had, on biological agents or depleted uranium for a variety of reasons. So this is somewhat new territory for us.

My understanding of depleted uranium in the theater is that we see it in two ways. We see it in the shells, the projectiles. The depleted uranium was almost really the spear on the shell that penetrated the armament and then when it could penetrate through the shell itself, would explode and cause the damage.

And my other understanding is that the depleted uranium is also used on the armament, in particular of the tanks and some of the other vehicles in the theater. So far, am I on target?

Dr. DURAKOVIC. Yes.

Mr. SHAYS. Now, depleted uranium is the term we use. Describe to me where we get the depleted uranium.

Dr. DURAKOVIC. Well, first of all, I really do not deal with terminology like depleted uranium because, as a medical doctor, I deal with terminology of uranium.

Mr. SHAYS. OK.

Dr. DURAKOVIC. All isotopes of uranium, 238, 235——

Mr. SHAYS. I am going to have you slow down.

Dr. DURAKOVIC. I am sorry. All isotopes of uranium, 238, 234, 235, are alpha-emitting, radioactive isotopes. "Depleted" really means a concentration of Uranium 235 and 234 in the entire bulk of uranium, which has to be enriched to the point of utilization in nuclear weapons or nuclear reactors. Uranium exists all over the planet Earth as uranium ore. As we know, we live in a radioactive environment, but that Uranium 238 is not capable of producing and sustaining a reaction that would feed reactors for nuclear weapons. For that reason, it has to be enriched by Uranium 235 and 234.

"Depleted uranium" simply means a concentration of highly fissionable Uranium 235 and 234 is diminished to a certain level in the specific bulk of uranium, if I can be as simple as I have been.

Now, we are talking about radioactive isotopes with a long half-life and alpha-particle radiation. Alpha particles are the heaviest particle produced in nuclear reaction, and in the case of uranium, we deal with an incredible phenomenon that is unique in the history of mankind.

I am very grateful for your question because it leads us to a better understanding of the problem. Uniqueness of uranium incident in the Gulf war is that it is the single, largest mass contamination by a single isotope. Hiroshima and Nagasaki was mass contamination with 440 radioisotopes which are produced in a nuclear explosion. We know that Chernobyl is not a nuclear weapon; it is reactor producing about 440 radioactive isotopes. So these are mass-contamination scenarios in which many isotopes are implicated.

The Gulf war is the first case in the history of mankind where we have one single isotope responsible for mass contamination. What happens in the bodies of the human beings or animals where uranium enters? Whatever——

Mr. SHAYS. I do not want you to go that far yet. I just want to understand. Mr. Dietz, am I saying your name correctly? Is it Dietz?

Mr. DIETZ. Pardon me?

Mr. SHAYS. Am I saying your name correctly when I refer to you as Mr. Dietz?

Mr. DIETZ. Yes.

Mr. SHAYS. I am about as big a generalist as you can get. My daughter knows more than I know on these issues. I guess that should not be surprising; she is a high school student. I just want to first understand kind of the framework I am working in. Maybe depleted uranium just has—my concept of depleted uranium is basically the uranium that comes out of a nuclear plant after it has spent nuclear energy. Now, is that what—I should not be thinking——

Mr. DIETZ. The depleted uranium arises from the gaseous-diffusion plant when the natural uranium, which is also more than 99 percent Uranium 238, is transformed by chemical action into uranium hexafloride and then passed through barriers; and this is a physical process for enriching the U–235.

Mr. SHAYS. But is depleted uranium that basically exists because it was used for something else first?

Mr. DIETZ. That is right.

Mr. SHAYS. OK. It is a waste material, in a sense, of something else.

Mr. DIETZ. I think a way to picture this visually is to think of a stream of uranium hexafloride gas going through the diffusion plant. The enriched goes in one direction, and the tails, what is called the "tails," which is the depleted uranium, goes in the opposite direction.

Mr. SHAYS. That value of depleted uranium, is it is extraordinarily dense?

Mr. DIETZ. It is 1.7 times as dense as lead.

Mr. SHAYS. But not as heavy or——

Mr. DIETZ. Well, the density would be the grams per cubic centimeter.

Mr. SHAYS. So it would be a heavier material. I guess what I am getting to—I do not guess; I am—depleted uranium is relatively inexpensive, expensive?

Mr. DIETZ. We have in storage now I think something like 600,000 metric tons of depleted uranium in the form of hexafloride.

Mr. SHAYS. The bottom line is it is very cheap.

Mr. DIETZ. It is coming out our ears.

Mr. SHAYS. And the military determined that depleted uranium had tremendous strategic value to them in the sense that it was a material that could penetrate most of the armament.

Mr. DIETZ. The uranium is a very dense material. It can be hardened by adding three-quarters-of-a-weight percent of titanium to it to make it superhard, made into a long, thin rod fired at very high velocity so that when it hits a solid object like a tank armor, which is basically mainly iron, it undergoes almost an instant, very high rise in temperature, and because of its pyrophoric nature, it starts to burn or oxidize extremely rapidly, almost explosively, and when that happens, you get these micrometer-sized particles. A 5-micrometer particle can be breathed into your lung and can stay there for many, many years.

Mr. SHAYS. But the Army has it in its shells for penetration, but it also has it on the vehicles themselves for armament. Correct?

Mr. DIETZ. Well, it is the optimum penetrator.

Mr. SHAYS. OK. It is a penetrator, but it also is a stronger material for shield.

Mr. DIETZ. Tungsten is a substitute, but it does not have the pyrophoric nature of uranium. What happens is that "pyrophoric" means that when it burns, it——

Mr. SHAYS. You are telling me something I am not up to yet. I do not want you to get ahead of me here.

Mr. DIETZ. OK.

Mr. SHAYS. I just asked a question. The simple question was—I want to get on to the next one. It is not a big answer.

Mr. DIETZ. OK.

Mr. SHAYS. It is used as a shield on our vehicles as well. Correct?

Mr. DIETZ. Yes.

Mr. SHAYS. OK. So you have it used as a penetrator and as a shield. Now, in the course—this is your area of expertise. When the Army sought to do this, when we moved in this direction and we saw the value of it, certainly the issue of safety was looked at then, what got by us, if, in fact, depleted uranium is the threat that you consider it to be?

What I am trying to just nail down—see, I guess I am looking for motives here, and if I were in the military and I considered this an absolute essential use, I might be a little less inclined to see if there was a negative associated with it; and if there was a negative associated with it, I might want to not just come to grips with it because the implications can be quite significant.

I may be going down a road that I am going to say goes nowhere, but I am willing to just consider this for a second. So all I am asking you now—you are both experts in the field. Correct? You knew depleted uranium was used for military purposes. Was there a group within the scientific community that said this ain't a good idea?

Mr. DIETZ. I do not think so. I do not know who made the decision to use depleted uranium munitions, because all the bad things about uranium from a health standpoint were known long, long before the Gulf war began. I think it is used basically because it——

Mr. SHAYS. I know why it is used. I want to know how it got to be used, and I want to know if we went through a process, and you are not going to be able to answer that question——

Mr. DIETZ. I do not think I can.

Mr. SHAYS [continuing]. That we went through a process of determining that it was totally safe. I just want to determine whether you have the capability on your expertise to answer that question. Are you aware of the process that got us to the point where we used depleted uranium? If you are——

Mr. DIETZ. I am not aware of the historical——

Mr. SHAYS. OK. That is fine.

Dr. DURAKOVIC. I am aware of it, and that is one of the reasons why we have a free United States of America today. In the 1940's German scientists suggested to Hitler to use uranium for the production of nuclear weapons in their research. Hitler used it because he was convinced by his generals that uranium can be used as an

armament in the German tanks, and they, indeed, used uranium from the mines of Yoachimstaal in Czechoslovakia in the German area, and they used that uranium to reinforce the German shielding of their tanks.

So, use of uranium in the shielding of the tanks is not new at all; it goes back 50 years.

Mr. SHAYS. OK. That is good to know. Are our soldiers—hold on 1 second. What I think I am going to do is do a little more research myself on how we got to where we use it. My antenna goes up when I get into an issue like this because the implications of what you gentlemen are saying have tremendous consequence.

If, in fact, our soldiers have been harmed by depleted uranium, that potentially says a lot about what we have to look at, and it says a lot about protocols within the military. One of the protocols we know in the military is that they did not—it is my understanding; I may be corrected later on, but they did not notify our soldiers of the consequence of depleted uranium, and now they are, but they did not then. And some of this boggles my mind, I mean, if that is the case.

So let us just get a little bit more to your expertise here. Have both of you treated or examined Persian Gulf veterans? Mr. Dietz, you have not.

Mr. DIETZ. No.

Dr. DURAKOVIC. I have examined the veterans; yes, they were my patients.

Mr. SHAYS. OK. Mr. Dietz, your point was to show us—in your testimony you gave us other examples of depleted uranium where there was a concentration of it and the consequences of that, and your testimony, as I gather, is to say that was bad. What we have here in the concentration of depleted uranium in the Persian Gulf was even worse. Is that a fair?

Mr. DIETZ. Yes. It is many orders of magnitude worse than the problem at Albany.

Mr. SHAYS. And that leads you to come to what conclusion?

Mr. DIETZ. The only conclusion that I can come to is that this is a truly wonder weapon. The analogy that can be given is that it is as effective against destroying tanks as a machine gun was in World War I against infantry soldiers.

Mr. SHAYS. OK. We know that, but we also know it has a negative side effect.

Mr. DIETZ. I am sorry?

Mr. SHAYS. We also know there is a negative side effect.

Mr. DIETZ. Absolutely. There is a negative side, and I think the military is overlooking the negative side.

Mr. SHAYS. OK. Well, maybe what I will do is Mr. Sanders will get into other areas, and then I will come back.

Mr. SANDERS. Thank you, Mr. Chairman, and I share your concerns about depleted uranium, and that is a whole, huge issue which I think we need to get into, but what I would like to do is just speak to the Nicolsons for a moment.

Dr. Nicolson, one of the interesting aspects of your testimony is that you talk about actual treatment, and we have not heard a whole lot of that discussion here. Now, as I understood from your

testimony, you said that you have treated several hundred Persian Gulf vets. Am I correct in remembering that?

Mr. NICOLSON. Several hundred have been treated. We do not do the treatment ourselves. We are a diagnostic institute. We do the diagnosis. We go to the primary-care physicians who then treat the patients and we do followups with the primary-care physicians.

Mr. SANDERS. Based on your diagnosis?

Mr. NICOLSON. Based on the diagnostic tests that we perform.

Mr. SANDERS. OK. And can you tell us the results of the treatments of the people that you referred to primary physicians, how successful or not successful have those treatments been?

Mr. NICOLSON. I actually showed you some of that data. We have in press in a medical journal the results from 170 patients. Seventy-six of the patients were positive——

Mr. SANDERS. "Positive" meaning production of symptoms?

Mr. NICOLSON. Positive for the infection that we have discovered, the mycoplasmal infection. Seventy-three underwent treatment. Of the 73 that underwent treatment, 58 are now considered to be recovered and are now back on active duty. They may not be cured from this illness, but at least they have recovered to the point where they can perform at their level for their job description.

Mr. SANDERS. So what you are saying is, in terms of the treatment that you have recommended, 58 out of 73 have seen significantly positive results.

Mr. NICOLSON. That is correct.

Mr. SANDERS. Now, given the fact that we have an estimated 70,000 vets who are hurting, that is a pretty interesting and important result. Have those results been confirmed by others? I mean, are people going to argue with me and say, no, that that is not the case?

Mr. NICOLSON. The diagnostic results have been confirmed by a certified diagnostic laboratory, Immunosciences Laboratories, in California. We are in the process or arranging to train DOD scientists to perform the types of tests that we perform.

Mr. SANDERS. OK. Now, given the fact that everybody in the DOD and the VA is concerned about this problem, what has their response been to your approach and the apparent, what you are telling us, very strong, positive success that your diagnosis has had? Is that being replicated elsewhere now?

Mr. NICOLSON. I would say they first ignored us or ridiculed us. Then I think our success, particularly the patients that went to the Walter Reed program and did not recover from their illnesses, but began to recover on these multiple cycles of antibiotic therapy.

They have begun to take a renewed interest, I think, in what we are doing, and it is still, I guess, at that point now that they are very interested in the types of tests that we are running and the types of therapies that are allowing not only the soldiers, the veterans to recover, but their family members who are symptomatic—we have a large frequency of illness in families of Gulf war veterans as well.

Mr. SANDERS. After this panel testifies, we are going to be hearing from the VA and the DOD, and I am going to ask them specifically how they have responded to your work. What are they going to tell me?

Mr. NICOLSON. Well, I think they will tell you that both Nancy and I have addressed the DVA and the DOD in Washington several times over the span of a few years. They have taken an interest in what we are doing. They are making plans to send individuals out to our laboratories to be trained in this, but they have also tried to perform some of their own tests, but unfortunately they are using 1960's technology in their own tests that they are performing, and I do not think they are going to come up with anything. This is not the approach that is necessary.

We want to bring them up to speed to use state-of-the-art, diagnostic procedures for these types of illnesses. They are very difficult to diagnose.

Mr. SANDERS. So am I hearing you say that they are interested in the work that you are doing, but they have not in their own labs been able to replicate what you have done?

Mr. NICOLSON. They have not shown up in our laboratories yet. When they show up, we can train them, and then we can make sure that they replicate the type of data that we are finding routinely. We have trained diagnostic laboratories, and they are replicating our data, so I do not think it could be said that it has not been replicated. It has not been replicated by DVA and DOD scientists—that is true—because they have not come to be trained.

Mr. SANDERS. In your judgment—let me ask you this. Who is treating, in this country today, how successful are we in general in treating Persian Gulf illness? Is the VA and the DOD successful? Do they have any protocol which seems to be working?

Mr. NICOLSON. I think you will have to ask the DOD and the DVA that question. It is my feeling from discussions with various physicians who are now treating Persian Gulf war veterans and their family members, using the protocols that we have established as effective, is that they are gaining ground in this area, but I have to again stress that this is a subset of patients. This is not every patient, because as you have heard, some patients may have radiologic exposure, some may have chemical exposure, some may have biologic exposure, or combinations of them.

And, in fact, some of the veterans who have testified to this committee earlier have come to us, we have tested their blood, and they have turned out to be positive and their spouses who are now ill have turned out to be positive and their children who are now ill have also turned out to be positive.

Mr. SANDERS. In talking to veterans in Vermont, and I think the answer around the country would be the same, what they are saying is that we are hurting, and even if there is not 100-percent guarantee that a new type of treatment might work, if it is not going to hurt us more we would be willing to gamble. Let us see what is going on out there.

So my first question in that regard is, are there side effects? Is your treatment and approach risky? Can it cause additional problems?

Mr. NICOLSON. The approaches that we have proposed are standard medical procedure for the treatment of chronic infections. They are really no different than the treatment of Lyme Disease, for example, and other chronic infection. So I would say that these are pretty standard procedures. The antibiotics that we recommend are

pretty standard antibiotics. Not every antibiotic will work, so it is not a placebo effect.

Mr. SANDERS. I mean, we understand not everything works for everybody, but if somebody were to say, in response to your treatment, "Well, we do not want veterans to be guinea pigs. You know, we do not want vets to be sent there and come back a lot sicker than when they started." How do you respond to that?

Mr. NICOLSON. Well, I think if they are tested and they are found to be positive for these chronic mycoplasmal infections, and they have systemic or system-wide infections, they should be treated. That is standard medical procedure.

Mr. SANDERS. And, in your judgment, they are not going to be, no matter what the result may be, they are not going to be worse off than when they started.

Mr. NICOLSON. Well, from what we have seen, they slowly recover.

Mr. SANDERS. Right. But what I am trying to get at is if somebody argued—I mean, there are treatments out there—if somebody was dying of AIDS, for example, and we tried a radical therapy, it is possible that that might accelerate their death pattern. Correct? But one might say, Well, what is the risk? The person was going to die anyhow. What I am suggesting is that what I am hearing you saying is you do not see that your treatment will make people worse off.

Mr. NICOLSON. No. I mean, the only thing that we see in our treatment is that there is a transient worsening of the signs and symptoms due to the Herxheimer Response, and this is a very common response when an individual who has a chronic infection is on antibiotics, and that usually passes within a few weeks, and then they start to slowly recover. But the whole therapy can take up to a year. There are multiple cycles of antibiotics required.

Mr. SANDERS. Can you give me some examples of people or kinds of treatments, perhaps other than your own approach, which seem to be having some success?

Mr. NICOLSON. Other than the approach that we are taking?

Mr. SANDERS. Yes.

Mr. NICOLSON. Well, for individuals who have their primary problem as chemical exposures, there are a number of treatments to rid these chemicals from the body. There are a number of treatments to block the effects of the chemicals and so on. For those that have biologic exposures, we have to identify what type of agent is involved; otherwise, we really do not know the approach to use. If we identify a particular microorganism that is involved, whether it is virus or a bacteria-like microorganism, then the treatment is really quite different.

If it is, for example, a mycoplasmal infection, or a bacterial infection, then there are certain antibiotics which are fairly standard procedures for use against these types of infection. So we are really not talking about anything that is out of mainstream medicine.

Mr. SANDERS. In your judgment, and I know this may be a little bit askance, a little bit aside from your area of expertise, do you believe in the concept of multiple chemical sensitivity?

Mr. NICOLSON. Yes, I do, and we have seen examples of that actually; but this is not a concept that is well accepted by everyone in the medical profession.

Mr. SANDERS. Right. We are more than aware of that.

Mr. NICOLSON. Nancy also wanted to mention something.

Mr. SANDERS. Nancy, did you want to——

Mrs. NICOLSON. Well, the Multiple Chemical Sensitivity Syndrome does not explain the contagion that affects the families. Now, it is possible, if family members came in contact with gear that was brought back by a veteran and if the family member came in contact with such gear, they could develop multiple chemical sensitivity, but that does not explain the numbers of soldiers becoming sick.

So you would have to look for a biological agent, whether it would be endogenous to the area in the Middle East, because there are probably combinations of agents there, or as a result of some of the weapons that we have been told Iraq possessed. And we have to deal with the fact that given the mindset of the Iraqi Government at the time, they would have used multiple weapons in combination.

So it is a horrible concept to have to deal with. I feel that our Defense Department has been backed into a corner because this is the aftermath of years of cold war policies. What was then the Soviet Union and the other superpowers were engaged in biological weapons research. In fact, in the early 1980's, John Deutch recommended the buildup of biological weapons in the United States.

So what I am saying now is that we need to get past the cold war. We need to acknowledge that there is a strong possibility that many governments were involved in weapons research like this and that no one is going to win this war unless we are bold enough, like the eagle on the flag, to come forward. I believe the United States will lead the way, and other countries will follow suit. I think it is time to stop blaming the Defense Department of this country and other countries, but it is the fear factor, the honor, and the embarrassment, and we still have a problem.

The International Monetary Fund noted last week that there is a 20 percent increase in chronic, infectious disease around the world. This is going to have economic repercussions. So the nitpicking that has gone on in the scientific community has to stop. I think the onus is on the scientific community who went ahead with ill-advised experiments. I am sure the scientists assured the military sector that they could control weapons like the biologicals, but the fact is they cannot. Of all the weapons involved, the biological weapon is the most serious. It is difficult to detect, impossible to contain.

So it is my feeling that we can conquer this problem if the Defense Department would be allowed to tell the truth, and that is the problem. They are in a very difficult position because of outmoded policies and because of embarrassment.

Mr. SANDERS. The bottom line, what you are saying is that you believe that the increase in infectious diseases is related to the work done on biological weapons.

Mrs. NICOLSON. Partly in relation to testing of biological weapons around the world. Those of us in the science community, know who

they are. We know which scientists have done this. They are afraid to come forward because they really thought they were doing the right thing at the time, but the science community needs to be scrutinized. I blame the global science community for this problem because they should never have developed these weapons. It is very simple.

You have what was then the Soviet Union, which was actively engaged in biological weapons research, it forced us to follow this race because no one was thinking. No one was thinking. So I think we need better cooperation between the defense science sector and the civilian science sector, and I think pointing fingers and assessing blame is not the way to go. We have to take care of our soldiers and the people on this planet.

Mr. SANDERS. Mr. Chairman, let me just end my line of questioning just by asking the Nicolsons this question. It would seem to me, given the fact that so many people are hurting, that we would, or that the DOD and the VA would actively be searching out and engaging those people who are involved in a variety of treatments to see if any of those treatments are successful. And we could understand some treatments may not work, but it would seem to me so long as these treatments did not do any more harm to the patient, that we would want to look at as many people and as many ideas as possible.

Now, I have the impression that that has not been the case. I think what I keep hearing from the DOD and the VA is we do not know, that this is not peer reviewed; no, that is not good; no, this is not good; no, that is not good; but we will continue going along the route we are going, even though we do not have any particular understanding, and we do not have any particularly effective treatment.

Am I misstating, do you think, the——

Mr. NICOLSON. No. That is exactly my perception as well. I mean, there has been far too much criticism and not enough cooperation. We need to get beyond that point of simply criticizing those people that come up with preliminary evidence and so on. We were criticized quite extensively initially when we started to get involved in this issue that we did not have extensive data. Well, we had absolutely zero support from the Federal Government, so we used entirely our own funds to collect the research data that we collected. So we had really no financial help whatsoever.

All of the studies we published, including the medical journal articles were done without any Government support whatsoever.

Mr. SANDERS. Have you received up until this day any financial support from the Federal Government?

Mr. NICOLSON. Oh, yes. I currently receive financial support. I have a grant from the U.S. Army, for example, but it is for breast cancer research.

Mr. SANDERS. No. I am talking about not breast cancer. I am talking about this——

Mr. NICOLSON. No, not one nickel. In fact, we put in a proposal in 1995 for this type of study, and they cut the budget by 89 percent, and they did not give it a fundable priority; so even if it were funded, we could not have done the work on 11 percent of the requested budget.

Mr. SANDERS. Are you aware of many researchers who are looking at alternative approaches beyond stress, for example, who are receiving funding? There have been a number of breakthroughs, it seems to me, but are those people receiving the help that they need from the Government, or are they having to do it with private source? Ross Perot, for example.

Mr. NICOLSON. Well, they are having to do it with private sources of funds. For example, James I. Moss, a scientist in Florida, the first one to show that combinations of different chemicals could produce neurologic syndrome——

Mr. SANDERS. He was fired from his job at the Department——

Mr. NICOLSON. No, he was not fired from his job. He received word the other day that his grant that he put in to DOD would not be funded. So they have taken the tactic that they will squeeze us to the point that we cannot do the work that we should be doing.

Mr. SANDERS. Would you be prepared to have your work submitted to significant controls?

Mr. NICOLSON. We have already agreed to do that. I was at a meeting called actually at the behest of Congressman Norman Dicks. Major General Leslie Berger, the commanding officer at Walter Reed Army Medical Center, convened a meeting on December 23rd of last year. I was at that meeting and spoke to the Persian Gulf War Research Group and the rest of the individuals who were interested in this, and at that meeting it was decided that they would send scientists and physicians out to our institute to learn the techniques that we were doing, and we would set up a validation study. Well, we have not heard from them since January.

Mr. SANDERS. Six months have come and gone.

Mr. Chairman, we hear this over and over again. I cannot sit here in judgment and tell you whether the Nicolsons are right or not right. I do not have the background to do that, but it seems to me that if people are treating and claiming to have success, that the DOD and the VA would be falling all over themselves to try to determine whether, in fact, this analysis and proposed treatment is working or not, and that we are doing that for everybody in this country who is coming up with different ideas.

So I would just conclude by thanking, and I am sorry to have ignored you. I do not mean to suggest that your work is not significant, but I did want to focus on this aspect of it. Thank you, Mr. Chairman.

Mr. SHAYS. I think really what we are doing is you are focusing on the biological, and I am just going to be focusing a little bit more on depleted uranium.

I want to know the difference between, say, depleted uranium fragments that might be in a soldier's body versus inhaling, digesting the particles, which I would tend to say would be more dust almost—not gas because they are still particles, but they are almost invisible in some ways. Describe to me the difference in terms of its impact on the health of the soldier. Both of you may do that.

Mr. DIETZ. I am not a medical doctor, so I really cannot comment on that.

Mr. SHAYS. Why don't you start, though, by just prescribing me the scientific difference between the fragment and the particles?

Dr. DURAKOVIC. The difference between inhalation, for instance, ingestion, or embedded particles like shrapnel boils down to the same phenomenon in the body, and that is the release of uranium from the site of incorporation into the bloodstream. In my opinion, it is exceedingly more dangerous to be exposed to uranium in the inhalational pathway than by the shrapnel or the embedded particle for several reasons.

Reason No. 1 is that the embedded particle or shrapnel is protected from the rest of the body fluids by the formation of the fibrous capsule, which is the scar tissue. Scar tissue would contain the particle at the place of its incorporation, and the uranium from the particle would not have early access to the bloodstream. Subsequently, it would not have an early access to the target organs, which are kidneys, liver, and skeleton.

In the event of inhalation, a high amount in percent of uranium is taken to the bloodstream from the lung tissue, and these are really invisible bullets. They are invisible bullets consisting of alpha particles, two protons and neutrons which are bombarding the internal environment of the organism, leading to breakdown of the tissue, necrosis or the death of the tissue, malignant changes like cancer, leukemia, malignant tumors, and genetic malformations in generations to come.

My answer to your question, sir, is this. Regardless of the pathway of contamination, the ultimate fate of uranium is going to be determined by the organ of incorporation. In the case of embedded particles like shrapnel, I believe it is less likely that the henomenology of uranium will be as extensive as the inhalational pathway because simply more radioactive material will have access to the bloodstream through inhalation but not through the ingestion because ingestion is a relatively safe way of being contaminated with uranium, since only a couple of percent of uranium are absorbed in the gastrointestinal tract.

So my conclusion is that the single most important way of adverse effects of uranium would be by the inhalational exposure, which was the case in the Persian Gulf.

Mr. SHAYS. But if the Army were doing studies, and, Mr. Dietz, this question I would ask you as well. Mr. Dietz.

Mr. DIETZ. Yes.

Mr. SHAYS. The question I am asking, I am interested in knowing, if you were doing a study of its impact, it is one thing to say, well, you have this shell, and you have this depleted uranium; here it sits. It strikes me that the kind of study that you need to ultimately do is to determine what happens when this shell is exploded, what happens, what is the effect of the heat on the shell. Is it in fragment form, or is it in particle form?

Are either of you aware of any studies—you may not be—that the DOD has done in regards to—I asked it before; I am asking it again, to be very clear—are either of you aware of any studies that DOD has done on depleted uranium by its use? In other words, not in its form before use but in its form after its use.

Dr. DURAKOVIC. I am aware of that.

Mr. DIETZ. I am not aware of it.

Dr. DURAKOVIC. I am aware of the study that DOD sponsored with the Armed Forces Radiobiological Research Institute in Be-

thesda. There was a study on experimental animals which was presented a couple of months ago at a scientific meeting in the form of an abstract where embedded uranium in the form of the shrapnel was incorporated——

Mr. SHAYS. That is fragments.

Dr. DURAKOVIC. Fragments. That is correct.

Mr. SHAYS. What about particles?

Dr. DURAKOVIC. Inhalation pathway. No, I am not aware of any study by the DOD or the VA.

Mr. SHAYS. And, Dr. Nicolson and Nicolson both, what I am hearing from your testimony, one of the things I am hearing is that the biological agents would be the one way you would explain the potential health problems from one family member to another.

Mr. NICOLSON. We think this is really the only way you can explain it, except for an odd occurrence of someone coming in contact with a souvenir or a pack from Desert Storm or something like that that was contaminated.

Mr. SHAYS. That would be the only way basically. Either they came in contact with something that may have been contaminated by chemicals or by biologic agents.

Mr. NICOLSON. Predominantly biologic agents would explain the illness passing into the family members and health care workers. Nancy wanted me to mention the fact that when we looked at a nonscientific sample of veterans, nonscientific because we have not looked at entire units; a lot of the individuals come to us. But a lot of these individuals served behind the lines, either from the deep insertions into Iraq, such as the Airborne and Special Forces units that we worked extensively with, or the units that were in a support role, command and control, transportation, and so on back behind the lines that were under SCUD attack and other means.

Except for the Marine Corps, we have not seen a lot of patients from the mechanized infantry or armored units. The exception is the Marines, and they were in a very contaminated environment in Kuwait, and so I feel that they had multiple exposures of chemical, radiological, and biological; and, in fact, some of the soldiers I mentioned that testified to this committee previously and those that had very severe neurologic signs and symptoms, we have been able to show that they are infected with one of these biological agents. They are going to be undergoing therapy, and their families are also infected with the same agent.

Mr. SHAYS. Thank you. I am just going to end with you, Dr. Durakovic. I want to be clear on what the symptoms were from the Gulf war veterans that you examined.

Dr. DURAKOVIC. There were multiple symptoms which really cannot be summarized into any logical picture. The symptoms encountered in my patients were primarily respiratory symptoms, including pharyngitis, tracheobronchitis, and in some cases, pneumonia. In endocrine diseases, several patients had thyroid alterations, gastrointestinal symptoms ranging from severe diarrheas to dehydration, vomiting, nausea, hepatic symptoms, and renal symptomatology. Some of my patients underwent several surgical procedures because of kidney problems. Prior to the Gulf war they did not have any kidney problems.

So, my answer to your question is that there is really no simple answer to this question because symptomatology ranged from the respiratory to the renal syndrome in very different organic systems.

Mr. SHAYS. Is there any question that any of you wish we had asked that you would want to answer? We will start with you, Dr. Nicolson.

Mrs. NICOLSON. I really do not have one at the moment.

Mr. SHAYS. Well, that is all right. It just would be one that really was right at the tip of your tongue.

Mr. NICOLSON. It will probably come back. We did touch upon a subject which I think we need to spend a little bit of time on, and that is the family members. This is something that has been avoided and denied officially, that the family members are now actually involved with illness. But it is very hard to deny when young children have the diagnosis of failure to thrive, rashes all over their bodies, and not doing well because of chronic fatigue, fibromyalgia, and other problems.

It is hard to deny the fact that these people are sick, that spouses are sick with this illness and so on. And I think that the biggest tragedy that has happened as a result of our experience is the denial that this type of illness can spread to family members. And, again, there was an official counterattack when we first came out and did our study of the veterans' wives and other family members instead of which we felt would have been the opposite. Here is a problem. It is obviously a problem. Let's try to find the solution to this problem, not just attacking the messengers.

Mrs. NICOLSON. I do have one point. You have asked about the problem in the civilian population of countries like Iraq. We have received communications on this, and, of course, I am not in the intelligence community, so it would be hard for me to provide documentation. But I have many friends in Jordan and in just about every country in the Middle East, and they have contacted us from various clinics, and told us that there is a problem in the civilian populations of Kuwait, where they estimate 15 to 20 percent of the adult population is suffering from a variety of signs and symptoms, and indirectly we have received communications from people in Iraq that there is a major problem there via Jordan, some clinics there.

So that would explain the possible release by a variety of ways that we try to cover of an infectious agent, because it is a civilian problem. It is like a time bomb. It goes off. It is not an acute problem because I believe our soldiers were covered for the acute agents, so there is a problem, and some body, maybe the World Health Organization, needs to address it and release the data so that we can better deal with it.

Mr. NICOLSON. In fact, we are on our way to Europe to do just that. We will be meeting with representatives from the WHO and from several countries that have an interest in seeing this issue resolved.

Mr. SHAYS. Thank you. Mr. Dietz, is there any question you wish we had asked you?

Mr. DIETZ. Any question which I would like——

Mr. SHAYS. Is there any question you wish we had asked you that you would have liked to have responded to?

Mr. DIETZ. I think we have covered everything quite well, and offhand I cannot think of any.

Mr. SHAYS. I appreciate your testimony as well as the Nicolsons'.

Doctor, any question you wish we had asked?

Dr. DURAKOVIC. I only wish to express my thanks for this opportunity.

Mr. SHAYS. Well, it is our opportunity, and we thank all four of you for coming to testify. I know you had to wait through the first panel, and I appreciate you being there. So all of you are free to go, and thank you very much.

We are really now coming to the second panel. I appreciate in particular the Department of Veterans' Affairs for their willingness to have the panels switched.

We have Dr. Thomas Garthwaite, Deputy Under Secretary for Health, Department of Veterans' Affairs, accompanied by Dr. John Fuessner, Chief Research Officer, Department of Veterans' Affairs, accompanied by Dr. Frances Murphy, Director of Environmental Agents Services, Department of Veterans' Affairs. And Dr. Bernard Rostker, Special Assistant for Gulf War Illnesses, Department of Defense, is back. I appreciate you being back, and he is accompanied by Dr. Gary Christopherson. And is there anyone else who might respond to questions, because if so, I am just going to ask them to stand as well.

What I would like all of the panelists to do is, if they would stand, as you know, we swear all our witnesses in, and anyone else who might be that is accompanying you, and we will only introduce them if they then end up testifying; but if whoever else might be potentially responding. Thank you all for your patience. Raise your right hands.

[Witnesses sworn.]

Mr. SHAYS. Thank you. Again, I want to thank all of you. First, I would like to thank again the Department of Veterans' Administration for being here for the first panel, listening to our veterans, being willing to fit into Dr. Rostker's schedule. And, Dr. Rostker, we appreciate you coming back.

Dr. ROSTKER. Thank you, sir.

Mr. SHAYS. What we will do, Dr. Garthwaite, I think we will start with you. And, again, we have a 5-minute timeframe, but we really are more interested in your testimony, and so if you go over, I could care less. In other words, I care more that you give the testimony that you want to give, than about the time.

STATEMENTS OF THOMAS GARTHWAITE, DEPUTY UNDER SECRETARY FOR HEALTH, DEPARTMENT OF VETERANS' AFFAIRS, ACCOMPANIED BY JOHN FUESSNER, CHIEF RESEARCH OFFICER, DEPARTMENT OF VETERANS' AFFAIRS AND FRANCES MURPHY, DIRECTOR OF ENVIRONMENTAL AGENTS SERVICES, DEPARTMENT OF VETERANS' AFFAIRS; BERNARD ROSTKER, SPECIAL ASSISTANT FOR GULF WAR ILLNESSES, DEPARTMENT OF DEFENSE, ACCOMPANIED BY GARY CHRISTOPHERSON, ACTING PRINCIPAL DEPUTY FOR HEALTH AFFAIRS; COL. HERSHELL WOLFE, ASSISTANT FOR OCCUPATIONAL HEALTH, ASSISTANT SECRETARY OF THE ARMY, ASA, ILNC; AND COL. ERIC DAXON, RADIOLOGICAL HYGIENE STAFF OFFICER, AEPI, U.S. ARMY

Dr. GARTHWAITE. Mr. Chairman, I am pleased to have this opportunity to discuss VA programs for Gulf war veterans. Accompanying me today are Dr. Frances Murphy, who heads our Environmental Agents Service, and Dr. John Fuessner, who heads our Research Service.

Mr. SHAYS. Dr. Fuessner, I am sorry I pronounced your name so badly.

Dr. GARTHWAITE. As you requested, my focus today is on our efforts to help Gulf war veterans who may have adverse effects as a result of exposure to chemical warfare agents, depleted uranium, and smoke from oil well fires. While we must learn from the exceptions, it is important to remember the rule as well.

Since 1991, when we developed the VA Registry Program, more than 66,000 Gulf war veterans have completed Registry examinations. We have provided more than 1.8 million ambulatory care visits to about 200,000 unique Gulf war veterans, and more than 20,000 Gulf war veterans have been hospitalized at VA Medical Centers. An additional 400 veterans have been evaluated at our specialized referral centers, and more than 75,000 veterans have been counseled at our vet centers. The majority of veterans have been helped by our efforts.

With regard to chemical warfare agents, we continue to believe that additional research is needed with regard to the effects of low-level exposures to chemical warfare agents on human health. The VA has been working to advance scientific understanding of this area.

Our recent efforts include the following:

First, the Research Working Group has intensified its efforts to fund research related to health effects of low-level exposures to chemical warfare agents. New studies will address exposure to nerve agents alone or in combination with other toxins.

Second, the VA organized and sponsored an international symposium on the health effects of low-level exposure to chemical warfare nerve agents. The conference allowed investigators from around the world to share research findings and to discuss strategies for future research.

Third, VA funded three new toxicology fellowships and five new occupational medicine residency positions. These fellowships begin next week. We anticipate that we will be able to increase this number in future years, although concern has been raised by some program directors concerning the market for trainees after the fellow-

ship. We anticipate that these actions will increase the interest in research on chemical exposures.

Finally, we have altered our research focus to increase the studies which focus on clinical outcome.

With regard to depleted uranium, research on the human health effects of depleted uranium exposure in military occupations is limited, especially regarding depleted uranium's potential chemical toxicity. Two DOD-sponsored research projects currently under way are looking into this. In VA, the VA depleted uranium followup program at the VA Medical Center in Baltimore is a clinical surveillance program for identifying, characterizing, and following individuals who retain depleted uranium fragments from the Gulf war.

With regard to smoke and other toxins released from oil well fires, it is clear the Gulf war troops were exposed to potentially harmful environmental hazards during the Gulf war. The most obvious challenge was smoke from hundreds of oil well fires in eastern Kuwait in January 1991 set by retreating Iraqi forces. Some of the fires lasted until October 1991.

A coordinated, concerted effort has been made by the Department of Defense, Environmental Protection Agency, Department of Health and Human Services, and the National Oceanic and Atmospheric Administration to evaluate the health effects from these fires. Based on data collected from March through December 1991, the concentration of pollutants were within the U.S. air standards except for particulates and occasionally sulfur dioxide. Levels measured were similar to those in U.S. cities such as Houston and Philadelphia.

No cases of illness resembling those observed in Gulf war veterans were seen among firefighters in Kuwait nor among oil well fighters who have spent years experiencing similar exposures. Research efforts investigating the potential health effects of oil well fire exposure are ongoing.

Finally, with regard to enhancing our clinical programs, we continue to aggressively pursue enhancements to our clinical programs for Gulf war veterans. For example, we have implemented service evaluation and action teams in every one of our health care networks. These teams consist of clinicians, patient representatives, and patients who review and act to correct individual and systematic problems for Gulf war veterans.

While these teams are new, I recently reviewed their first submission of meeting minutes, and I believe that these teams will be a positive method to identify and fix many problems as well as an excellent way to identify common problems which can be fixed programmatically.

Second, we have piloted new care models including primary care teams, which develop expertise in caring for Gulf war veterans. This new model facilitates the education of providers about recent developments in Persian Gulf illness, improves the coordination of care, and enhances patient satisfaction.

Third, we have developed a method to oversample Gulf war veterans in our patient satisfaction survey process. This should allow us to have statistically valid assessments of the satisfaction with care of Gulf war veterans.

Fourth, we have had our medical inspector review the adequacy of registry examinations. These results have demonstrated a significant improvement in both accuracy and completeness of those examinations.

And, fifth, we believe that health outcomes are an important measure for all veterans and will be part of all health care in the future. We do not believe that it is done well in the VA or in any health care system that we know of. We have developed and tested a standard, data-gathering instrument that was originally developed by the Health Care Financing Administration. It is called the SF–36. We have tested it already in 32,000 veterans, and we will continue to use that into the future.

As an effort to enhance our understanding of the health of Persian Gulf veterans, we will also oversample Persian Gulf veterans with this instrument to see if we can describe better the current health status of these individuals.

In conclusion, we continue to make progress involving our research and clinical programs regarding Gulf war illness. We remain committed to meeting the challenges of understanding the causes of Gulf war illness and of providing the most effective treatment to Gulf war veterans.

We continue to welcome your feedback and advice on how we might be more responsive to the veterans we serve, and we will be happy to answer any of your questions.

[The prepared statement of Dr. Garthwaite follows:]

Statement of
Thomas L. Garthwaite, M.D
Deputy Under Secretary for Health
Department of Veterans Affairs
before the
House Committee on Government Reform and Oversight
Subcommittee on Human Resources
Hearing on Health Issues
of Gulf War Veterans

June 26, 1997
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Mr. Chairman and members of the Subcommittee, I am pleased to have this opportunity to discuss VA programs for Gulf War veterans. Accompanying me today are Dr. Frances Murphy, Director, VA's Environmental Agents Service and Dr. John Feussner, Chief Research and Development Officer .

As you requested, my focus today is on our efforts to help Gulf War veterans who may have adverse health effects as a result of exposure to chemical warfare agents, depleted uranium, and smoke from oil well fires.

First, I would like to like to briefly describe the VA's overall response to the issues raised by Persian Gulf War veterans and their families. As you may recall, on August 2, 1990, Iraqi forces invaded Kuwait, and United States responded by deploying nearly 700,000 U.S. troops to Southwest Asia in Operations Desert Shield and Desert Storm. After months of tense military build-up in a stark desert environment in which our troops were outnumbered, and under continual threat of chemical and biological warfare attacks, a short air war and a four-day ground war were successfully fought.

However, even after the ceasefire for some Gulf War veterans the pain of war was not over. Shortly after returning from the Gulf War, some veterans began to report a variety of symptoms and illnesses and VA immediately began development of Gulf War veterans programs. The first component of our response was the establishment of health-care program, the VA Registry health examination. The Registry was developed in 1991

and implemented in 1992. Gulf War veterans were given high priority and attention from the start. In 1993, at VA's request, Congress enacted Public Law 103-210, establishing a special eligibility for Gulf War veterans at VA health care facilities.

VA's Gulf War veterans' programs are encompassed in a comprehensive four-pronged approach, addressing relevant medical care, research, compensation, and outreach and educational issues. VA provides Gulf War Registry health examinations and follow-up care at its medical facilities nationwide, specialized evaluations at four regional Referral Centers, and readjustment and sexual trauma counseling to Gulf War veterans. To date, more than 66,000 Gulf War veterans have completed Registry examinations, more than 1.8 million ambulatory care visits have been provided to about 200,000 unique Gulf War veterans, more than 20,000 have been hospitalized at VA medical facilities, nearly 400 Gulf War veterans have received specialized Referral Center evaluations, and more than 75,000 Gulf War veterans have been counseled at VA's Vet Centers.

Gulf War Registry Examinations

Gulf War veterans participating in the Registry examination program have commonly reported that they suffer from a diverse array of symptoms including fatigue, skin rash, headache, muscle and joint pain, memory problems, shortness of breath, sleep disturbances, gastrointestinal symptoms, and chest pain. These symptoms have been treated seriously and they have received appropriate medical evaluations. To date, the diagnoses received by Registry participants do not cluster in one organ system or disease category, but rather span a wide range of illnesses and diagnostic categories. As such, they do not meet the clinical definition of a medical syndrome per se. Having said this, though, in no way diminishes how we view these complaints or what type of evaluation the symptomatic person receives. Also of particular note, 12 percent of the VA Registry examination participants have had no current health complaints but have wished to participate in the examination because they were concerned that their future health might be affected as a consequence of their service in the Gulf War. While 26 percent of the Registry participants rated their health as poor, 73 percent receiving this examination reported their health as all right to good.

With regard to the possible troop exposure to chemical warfare nerve agents, the Department of Defense has provided VA with a list of 21,799 individuals whom they identified likely being present within 50 kilometers of the Khamisiyah Ammunition Storage Area during the demolition period in March 1991. A computer match of the Persian Gulf Health Examination Registry database with this list identified 1,978 veterans, or approximately 9 percent, had completed Registry examinations prior to September 1996. A comparison of the examination results of these individuals with other Registry participants shows that similar types and rates of symptoms, diagnoses, and self-reported health status are reported, but virtually no individual in this group reported no symptoms at the time of their Registry examinations. Furthermore, those Registry participants (n=81) who are identified as part of the on-site Khamisiyah demolition team report symptoms and are diagnosed with musculoskeletal conditions at a higher rate than the 50 kilometer or general Registry group. This health surveillance data is preliminary and is compiled from evaluations of a non-random, self-selected group of the individuals possibly exposed to nerve agents at Khamisiyah. Although this information gives a partial snapshot of the health of some veterans thought to be present at this site in March 1991, the results should not be generalized to the entire Khamisiyah group or considered definitive.

I want to be clear that, contrary to certain reports in the news media, VA has no evidence of a causal link between the illnesses of Registry veterans and exposure to chemical warfare nerve agents. We do not, at present, draw any conclusions about the potential causes for the differences we have identified. They may be due to risk factors other than presence at Khamisiyah. More complete information on the potential exposures, the individuals affected, and research on the possible chronic health effects of low-level chemical warfare nerve agent exposures is needed.

At this point, I should reinforce the points made by past VA witnesses before this Committee and note that the Persian Gulf Registry was never intended or designed to be a scientific research study. It is a health-care program which was established to assist veterans' entry into the continuum of VA care. As such, we encourage all Gulf War veterans, symptomatic or not, to get a Registry examination. Furthermore, if Gulf War veterans have health problems that may be related to an exposure that occurred during their Gulf service, they are eligible for outpatient and inpatient care at VA medical facilities.

No Unique Syndrome

While some symptoms of Gulf War veterans are difficult to diagnose and remain unexplained, there is consensus among government and non-government physicians and scientists that current evidence does not support the existence of a single unique Gulf War Syndrome. The results of recently published research studies also has indicated that Gulf War veterans are not suffering at higher rates from life-threatening medical conditions or being hospitalized at higher rates than their non-deployed counterparts. Does this mean that there is no problem and that Gulf War veterans concerns are being dismissed or ignored by VA? The answer is an emphatic "no."

Statements about the lack of a unique syndrome should not be misconstrued as being equivalent to a denial of the pain, concerns and complaints of Gulf War veterans. Gulf War veterans who seek care from VA are suffering from genuine illnesses and, as indicated already, we are providing treatment for these persons. Likewise, VA provides compensation for many of those who are disabled and is conducting research programs to better understand the nature and causes for their illnesses.

Possible Chemical Warfare Agent Exposure

The record shows that VA has always acknowledged the possibility that Gulf War veterans were exposed to a wide variety of hazardous agents while serving in the Southwest Asia theater of operations, including chemical warfare agents. VA's public statements have always been clear that all exposures, including chemical warfare agents, were being investigated.

In 1993, VA designed its clinical uniform case assessment protocol to detect clinical signs and symptoms related to possible nerve gas and other neurotoxic exposures. Neurologic examinations and cognitive testing were part of the earliest versions of this protocol. As a consequence, VA diagnostic protocols continue to serve as a valid set of clinical guidelines for initial screening examinations (Phase I) and more comprehensive evaluations of difficult-to-diagnose cases (Phase II).

These protocols received positive reviews by highly-respected physicians and scientists in the past. In December 1996, the Presidential Advisory Committee on Gulf War Veterans' Illnesses agreed with a committee constituted by the National Academy of Sciences' Institute of Medicine (IOM) that the clinical evaluation programs set up by VA and the Department of Defense are excellent for evaluation and diagnosis of Gulf War veterans' illnesses. The diagnostic protocol was recently reviewed by another IOM committee contracted by DoD. We are pleased to note that this new IOM committee, in its report released April 22, 1997, concluded that the protocols provide an "appropriate screening approach to the diagnosis of a wide spectrum of neurological diseases and conditions." Moreover, in response to Public Law 103-446, an additional IOM committee will provide VA advice on its examination program, administration and outreach activities.

To date, no valid diagnostic test has been found to identify chemical warfare agent exposures that occurred years ago; therefore, no confirmatory test can be performed for veterans who wish to know whether or not they were exposed to these toxins during their service in the Gulf. In addition, neurologic examinations, neurophysiologic testing and cognitive testing were part of the earliest versions of the protocol for unexplained illnesses. The treatments available are not exclusive of or specific to damage from nerve agent exposures. The best available treatments are specific to symptoms or diagnoses rather than being dependent on cause.

In the wake of information regarding release of nerve agents at Khamisiyah in March 1991, VA has been asked whether we listened to veterans who reported their belief that they had been exposed to chemical warfare agents during their Gulf War service. VA officials did listen to those veterans and did take appropriate action to investigate their concerns. As an example, members of a Navy Reserve Construction Battalion unit from Alabama, Tennessee, North Carolina, and Georgia reported suffering adverse health effects which they attributed to exposure to chemical warfare agents during their Gulf War service. In response, VA established in 1993 a pilot medical program at the Birmingham VA Medical Center to evaluate their health status. As part of this special health-care program, more than 100 veterans were evaluated and treated. Veterans with cognitive symptoms received extensive (7-8 hour) neuropsychological testing and clinical evaluations. In 1995, Birmingham was designated as the fourth

regional Gulf War Referral Center to make this expertise available to a larger number of veterans.

Furthermore, the VA revised Registry examination protocol routinely asks participants to report the exposures they believe occurred during their service in Desert Shield and Desert Storm, including possible exposure to mustard gas or nerve agents. VA's openness to veterans' views and concerns is also evident in the representation of veterans service organizations on the VA Persian Gulf Expert Scientific Committee, whose members are appointed by the Secretary of Veterans Affairs.

In addition to these clinical programs, research studies are in progress to investigate the prevalence of symptoms and medical conditions among Gulf War veterans and to determine whether they are associated with the wide range of reported risk factors, including chemical warfare agents.

Depleted Uranium

Some Gulf War veterans have expressed concern about the possible long-term health consequences of exposure to depleted uranium (DU). We share this concern. As you may know, DU is derived from heavy metal uranium which occurs naturally as mineral deposits which are mined and processed for use in nuclear power plants or nuclear weapons. DU is the natural uranium left over after most of the highly radioactive uranium isotopes used in these plants and weapons are extracted. DU contains about half of the radioactivity of natural uranium. It is considered very low-level radioactive material.

In recent years, the U.S. Armed Forces have used DU in the manufacture of projectiles and armor. It is used in anti-tank munitions because of its highly effective penetrating capabilities and as armor plate due to its extremely dense properties. DU is nearly twice as dense as lead. During the Gulf War, munitions containing DU were used on a large scale for the first time. During the Gulf War, some U.S. tanks and airplanes fired DU munitions, which produced shrapnel and an aerosolized dust on impact with armor or on ignition in accidental munitions fires. A friendly fire incident wounded about three dozen U.S. troops, about two-thirds of these individuals have retained DU fragments. There are an additional 13 U.S. soldiers with potential DU exposures who were wounded and hospitalized but were not specifically identified as having shrapnel.

Other individuals with potential exposure to DU include personnel involved in the assessment, reclamation, decontamination and restoration of damaged vehicles as well as workers involved in the maintenance or modification of armored vehicles.

There are three common routes of absorption: 1) inhalation of DU vapor and fine dust contaminated with DU, 2) dermal exposure as a result of DU dust contamination of skin or a wound, and 3) imbedded, retained shrapnel which may dissolve and also be absorbed and distributed throughout the body. DU dust can be ingested as well, but this is not a likely significant exposure route unless exposure is long-term.

Research on the human health effects of DU exposure in military occupations is limited, especially regarding DU's potential chemical toxicity. However, two DoD-sponsored research projects currently underway are evaluating: (1) the health risks associated with tissue-embedded DU fragments and, (2) the carcinogenic risks associated with long-term exposure to DU-containing shraprel in wounds. These projects are scheduled to be completed in 1998. There are no published epidemiological studies of soldiers exposed to DU dust or vapor in war time environments. Most knowledge about possible effects on humans comes from studies of uranium miners and associated occupations which are somewhat different from Gulf War veterans. For example, these uranium miners were probably exposed to radon and other toxic substances present in the mines, making their experience not directly comparable to Gulf War veterans. Other significant difference relate to exposure intensity and duration.

Acute toxic effects of soluble uranium exposure are chemical in nature and primarily seen in the respiratory system and kidney. Chronic exposure is also thought to primarily affect the kidney. Chronic exposure by inhalation of insoluble uranium presents a potential radiologic hazard to the lung. Uranium miners have a long history of inhaling uranium dust in closed spaces. Although an increased risk of lung cancer has been observed among these miners, researchers think that this is due to simultaneous exposures to radon. Animal data are insufficient to determine whether inhalation of natural uranium causes lung cancer in animals. With acute exposure, if levels are high enough to cause kidney toxicity, but not death,

kidney damage is apparently repaired. There is no evidence of kidney toxicity among veterans either at the time of the Gulf War or now.

The VA DU Follow-up Program at the VA Medical Center, Baltimore, MD, is a clinical surveillance program for identifying, characterizing and following individuals with retained DU fragments from the Gulf War. The specific aims of the project are to provide on-going surveillance of Gulf War veterans with known or suspected DU fragments, and DU contaminated wounds. These individuals may also have significant amounts of inhaled DU. This surveillance will detect health effects, if any, of DU containing shrapnel, and provide recommendations for treatment to participating veterans and physicians caring for them. Thirty-three participants, who had been on or in U.S. Army vehicles when struck by munitions containing DU were evaluated in 1993 and 1994, and are continuing to be followed by the program. All participants underwent a comprehensive medical and psychological evaluation as well as a full body radiologic shrapnel survey. While those individuals with evidence of retained shrapnel showed increased excretion of uranium, no association between such excretion and clinically detectable adverse health effects has been documented. Efforts to improve both the assessment of uranium dose and the detection of toxic effects continue.

The program has facilitated the assignment of primary care providers for the veterans in the group and provides guidance to these participants as needed. A toll-free telephone number has been made available to participants as well as their family members and healthcare providers for consultation and assistance in a variety of clinical and personal issues. Moreover, the Baltimore DU program staff provide consultation to other VA healthcare providers caring for veterans who are concerned about DU exposure during Gulf War service.

While the DU program in Baltimore is a clinical surveillance program, not a research project, the program staff has developed a collaboration of VA and non-VA academic experts in the field of exposure characterization and outcome measurement. A team of specialists in environmental and occupational health, epidemiology, toxicology, radiobiology, physics, psychiatry, neuropsychology, and reproductive health have worked individually and collectively to develop and adapt diagnostic tools to better evaluate, treat, and counsel this unique group of individuals.

Oil Well Fire Smoke

It is clear that Gulf War troops were exposed to a variety of potentially harmful environmental hazards during the Gulf War. The most obvious environmental challenge was smoke from hundreds of oil well fires in Kuwait started in January 1991 by retreating Iraqi forces. Some of these fires lasted until October 1991. Large plumes of billowing smoke typically rose high in the atmosphere. Occasionally, the smoke remained low to the ground, enveloping U.S. military personnel.

A coordinated, concerted effort was made by the Department of Defense, Environmental Protection Agency, Department of Health and Human Services, and National Oceanic and Atmospheric Administration, to evaluate the health effects from these fires. Based on data collected from March through December 1991, the carcinogenic and non-carcinogenic health risks from exposure to oil-well fire smoke were determined to be minimal due to the lofting of the smoke to heights between 1,500 and 15,000 feet above ground level.

Both U.S. Interagency Air Assistance Team (USIAAT) and U.S. Army Environmental Hygiene Agency (USAEHA) found that while there were substantial levels of tetac particulates (sand and soot), concentrations generally were near normal for this region. With the exception of particulates and occasionally sulfur dioxide, concentrations of used criteria air pollutants were within U.S. standards. Furthermore, exposures to volatile organic compounds were similar to levels in Houston and Philadelphia, U.S. cities with major petrochemical industries by the USIAAT. Preliminary monitoring of pollution levels began in early March, with comprehensive air monitoring by USAEHA beginning in early May 1991.

USAEHA relied on modeling for risk assessment of troop exposures to oil well fires and smoke from February through May 1991. The oil well fires apparently did not cause observable acute changes in lung tissue. Researchers at the Armed Forces Institute of Pathology found no significant differences in lung tissue of service members who died after the fires compared to those who died before.

No cases of illnesses resembling those observed in Gulf War veterans were seen among firefighters in Kuwait nor among oil-well firefighters who have spent years experiencing similar exposures. Interviews by U.S. epidemiologists health care professionals in Kuwait in early March 1991, did not reveal any unusual health effects among Kuwaitis aside from the expected acute exacerbations of pre-existing lung diseases such as asthma. Research efforts investigating the potential health effects of oil well fire exposure are ongoing.

Research

To get the best assessment of the health status of the veterans, a carefully designed and well-executed research program is necessary. VA, as lead agent for federally sponsored Gulf War research programs, has already laid the foundation for such a research plan. Under the auspices of the Persian Gulf Veterans Coordinating Board's Research Working Group, VA has developed a structured research portfolio to address the currently recognized, highest priority medical and scientific issues. More than 90 research projects are in progress and others have been completed. We continue to search for answers and to expand our understanding of the complex array of issues related to Persian Gulf War veterans' illnesses. While scientific answers are being sought, VA also continues to provide Persian Gulf War veterans with needed health care and other services to reduce their suffering.

VA's own research programs related to Gulf War veterans' illnesses include more than 30 individual projects being carried out nationwide by VA and University-affiliated investigators.

VA established three Environmental Hazards Research Centers in 1994. All three centers are carrying out projects which address aspects of the potential adverse health outcomes of exposure to a wide variety of hazards, including neurotoxins. In 1996, we established a fourth center at the Louisville VAMC for investigation of adverse reproductive outcomes. In addition, VA's Environmental Epidemiology Service has completed an initial Persian Gulf Veterans Mortality Study and has begun a long-term mortality study.

The VA National Health Survey of Persian Gulf Veterans and Their Families is being carried out by the VA's Environmental Epidemiology

Service. Phase I, a postal survey of 15,000 Gulf War veterans and a comparison group of 15,000 Gulf era veterans, was completed in August 1996. The questions on this survey asked veterans to report health complaints, medical conditions, and possible exposures to a wide variety of possible environmental agents, including oil well fires, DU, pesticides potential nerve gas or mustard gas exposure. Phase II will consist of 8,000 telephone interviews and a review of 4,000 medical records. Phase II will address the potential for non-response bias, provide a more stable estimate of prevalence rates for various health outcomes, and verify self-reported health outcomes in medical records. Planning for the Phase III examination protocol is underway. Oversight of the national survey is provided by a subcommittee of VA's Persian Gulf War Expert Scientific Advisory Committee. Details of these and other government-sponsored research studies are included in the annual reports to Congress, entitled Federally Sponsored Research on Gulf Veterans' Illnesses. The most recent report was issued in April 1997.

Coordinating Board

In January 1994, the President established the Persian Gulf Veterans Coordinating Board, chaired by the Secretaries of VA, DoD, and HHS, to provide interdepartmental coordination and direction of federal programs related to Gulf War veterans. The Coordinating Board provides an interdepartmental means to share information on Gulf War veterans health, to allocate available resources to the apparent highest priorities, and to disseminate new research information. The Coordinating Board has three specific objectives:

- to ensure that Gulf War veterans are provided the complete range of healthcare services necessary to take care of medical problems that may be related to deployment in the Gulf region;

- to develop a research program that will result in the most accurate and complete understanding of the health problems experienced by Gulf War veterans and the factors that have contributed to these problems; and

- to develop clear and consistent guidelines for the evaluation and compensation of disabilities related to Persian Gulf service.

VA plays a central role in the Persian Gulf Veterans Coordinating Board through its participation in the Clinical, Research, and Compensation and Benefits Working Groups. In particular, the Research Working Group provides guidance and coordination for VA, DoD and HHS research activities related to Gulf War veterans' health. It coordinates all studies conducted or sponsored by the three departments to prevent unnecessary duplication and to ensure that important gaps in scientific knowledge are identified and addressed. The working group is actively involved in directing resources toward high priority questions and monitoring the results of federally sponsored research projects. It has produced several reports, including annual reports to Congress and a working plan on research.

One example of the Coordinating Board's proactive role in relevant research administration was its prioritization of the federal government and non-government research proposals submitted for funding pursuant to DoD's 1996 Broad Agency Announcement. The American Institute for Biological Sciences (AIBS) performed peer-review of the 111 proposals submitted. The research working group reviewed those proposals judged scientifically meritorious by AIBS and prioritized them according to relevance and potential to fill research gaps in the existing Gulf War research portfolio. Twelve research projects encompassing the areas of reproductive outcomes, toxicology of pyridostigmine bromide, modeling of respiratory toxicant exposures from tent heaters, neuropsychological outcomes, immune dysfunction, mycoplasma infection, leishmaniasis, chronic fatigue, fibromyalgia, and neuromuscular function were given high priority for funding by the Research Working Group. The Research Working Group is currently engaged in a similar process following solicitation by DOD of $17 million worth of additional research in a number of areas increasing research into the health effects of low-level exposure to chemical warfare agents.

Studies of the potential long-term health effects of low-level (asymptomatic) chemical warfare agent exposure were not given a high priority in the 1995 Coordination Board Research Working Plan because military and intelligence sources had stated that there had been no use, presence, or evidence of exposure to chemical warfare agents in the theater of operations. Based on these repeated assertions, combined with a lack of clear-cut clinical evidence to support a finding of chemical warfare exposure, the Coordinating Board focused its research resources on other

questions. However, while exposures per se were not a focus of research, clearly the health outcomes which could result from such a neurotoxic exposure were given priority. These outcomes included cognitive, neurologic, and neuromuscular effects. This decision was supported by committee reports from the Institute of Medicine, VA Persian Gulf Expert Scientific Committee, the National Institutes of Health Technology Assessment Workshop, the Presidential Advisory Committee, and others.

When DoD made its June 1996 announcement regarding possible exposure of U.S. troops to sarin and cyclosarin as a result of the demolition at Khamisiyah, the Coordinating Board immediately began revision of its action plan. Through the Research Working Group of the Coordinating Board, VA has developed an action plan to address possible long-term health consequences of low-level exposure to chemical warfare nerve toxins and mustard gas.

A recent literature review carried out by independent, non-government and government scientists, suggests that readily-identifiable, long-term adverse health effects due to nerve agent exposures only occur in humans who show signs of acute toxicity or poisoning. However, I should note that the research in this area is sparse and the absence of proof is not proof of absence. In VA's judgment, this information does not mean that clinically important adverse health effects cannot or definitely do not occur in the setting of low-level neurotoxin exposures, especially if combined with other components or environmental stressors. The Coordinating Board has recommended that more research resources be allocated to address this question. I strongly agree with this approach. The Research Working Group has reconsidered the matter and intensified its efforts related to possible effects of low-level exposures to chemical warfare agents.

Based on the Coordinating Board's recommendation, the newest recommendations from the Presidential Advisory Committee regarding investigating of the potential chronic health effects of low-level chemical warfare agent exposures, physiologic effects of non-traumatic and traumatic stress and the effects of pyridostigmine in combination with other exposures, have already been incorporated into the Coordinating Board's latest Working Plan for research, and requests for proposals were published by DoD. The Coordinating Board has reviewed the proposals judged most scientifically meritorious by the AIBS panels and has prioritized the studies

for funding by DoD. Some of these proposals will address the problem of exposures to multiple toxins.

While these efforts represent a good beginning, VA's Office of Research and Development is taking a completely fresh look at these issues in light of the new information provided by DoD. This includes asking them to develop a strategic plan for a research agenda that specifically focuses on low-level exposures to neurotoxins that might result from chemical warfare agents or other military situations. To support this effort, VA sponsored an international conference on the health effects of low-level exposure to chemical warfare nerve agents. This conference was held in conjunction with the Society of Toxicology Annual Meeting in Cincinnati, Ohio, this past March.

Research related to the illnesses of Gulf War veterans is highly complex, and this is especially so for the investigation of concerns related to possible low-level exposure to chemical warfare agents. VA is committed to meeting these challenges and obtaining the most accurate answers we can concerning the health of Gulf War veterans and their families.

New Initiatives and Continuing Education

VA has been proactive in establishing health-care programs for Gulf War veterans. In the immediate post-war period, the medical and scientific community had only limited knowledge of the complexity of Gulf War specific issues and exposures. Through our clinical and research programs, VA has been a leader in the development of health care programs, improvement of understanding concerning Gulf War health issues and dissemination of knowledge on Gulf War-related health issues. We believe that our programs are well-designed and comprehensive, but neither uniformly delivered nor perfect. We also recognize that some Gulf War veterans have not received the kind of reception or care at VA medical facilities that we can be proud of. To those Gulf War veterans, we want to assure you that the Veterans Health Administration (VHA) is working diligently to improve your satisfaction with our services. VHA has established quality monitors and performance standards for the Registry program and is developing a new customer satisfaction survey for Gulf War veterans.

We have established a Service Evaluation and Action Team (SEAT) within each Veterans Integrated Service Network. These programs will allow us to collect data on quality care to veterans.

To keep our health-care providers well-informed on the latest developments, VA has utilized a wide array of communication vehicles including periodic nationwide conference calls, mailings, satellite video-teleconferences and annual on-site continuing medical education (CME) conferences. Our initial efforts were successful in educating a dedicated cadre of well-informed Registry physicians and staff, and we will continue to keep them up-to-date on the latest developments. However, I see an opportunity to improve the understanding of Gulf war-related health issues by other medical personnel. My goal is that all VA health-care providers will have a working understanding of Gulf War exposures and health issues and will be able to discuss with their Gulf War patients how these issues could impact on their current or future health status. To meet this challenge and continue to improve our programs, VHA has developed and will publish a self-study Persian Gulf CME program for every VA physician in this year. We will make this available to non-VA physicians, at cost, as well.

The Presidential Advisory Committee found that our Registry and Referral Center personnel were indeed knowledgeable and well-informed about all aspects of Gulf veterans' health issues. However, education of health care providers not directly involved in the Registry program and VA's risk communication efforts should be enhanced and augmented. VA agrees and these efforts are already under development.

Mr. Chairman, as you may recall from your discussions with Dr. Kizer on January 29, 1997, he agreed with you that VA has not historically had a sufficient reservoir of medical toxicology and occupational medicine expertise. In an effort to increase the number of VA healthcare provide certified in medical toxicology and occupational medicine, Dr. Kizer directed the Office of Academic Affiliations to initiate efforts to fund 12 medical toxicology fellowship positions and 25 occupational medicine residency positions for the 1997-98 academic year. All relevant postgraduate training programs were contacted. The response from medical toxicology programs so far has been disappointing, but not altogether unexpected given the short time between solicitation and the beginning of the academic year. We have identified and finalized arrangements for 3

additional medical toxicology fellowships beginning in July 1997. Efforts to increase this number continue.

A total of 5 new occupational medicine residency positions has been identified so far, and more positions are expected. This brings to 9.25 the number of occupational medicine residents VA will support in the 1997-98 academic year. Both efforts will continue in the future. Based on feedback from several medical toxicology programs, we expect we will have a substantially greater response next year when the training programs have had more time to gear up for additional trainees. Of note, a major reservation expressed by the toxicology programs has been whether there will be a market for their trainees after fellowship.

In conclusion, VA is committed to providing the best available care to Gulf War veterans, to sponsor rigorous scientific investigations which promote better understanding of Gulf War veterans illnesses and prevent similar concerns among future veterans. In the meantime, VA will provide compensation to those eligible veterans who are disabled after their Gulf War service.

President Clinton has made it clear that no effort should be spared in this regard. In the end, long after the headlines fade away and national attention turns elsewhere, VA will still be there to care for Gulf War veterans. It is our mission. It is our nation's irrevocable promise to its veterans.

That concludes my statement. My colleagues and I would be happy to answer any questions you may have.

Mr. SHAYS. Thank you, Doctor. Dr. Rostker. You look like you were in prayer. Has it been a long day?

Mr. ROSTKER. It has been a long week, sir.

Mr. Chairman, members of the committee, it is my pleasure to be here again today and continue our dialog concerning the Department of Defense inquiry into Gulf war illnesses. I have a rather long statement, and I would request that it be placed in the record. I also sent the committee chairman this morning a letter concerning our ongoing interactions with Dr. Garth Nicolson, and I would like to bring that to the Chair's attention.

On December 23, 1996, DOD and the Department of Veterans' Affairs representatives met with Dr. Nicolson to discuss the mycoplasma laboratory test verification project in association with members of the National Institutes of Health's National Institute of Allergy and Infectious Diseases. This meeting was followed by conference calls on January 21 and March 20, 1997 to discuss strawman protocols, several electronic mail communications in a telephone conversation on March 24, 1997 between Dr. Engles and Dr. Nicolson.

At that time, Dr. Nicolson agreed to the project protocol. The final protocol has been written and approved by DOD and HHS scientists and Dr. Nicolson. The protocol will use four laboratories which will test the agreement for various conventional reaction tests and Dr. Nicolson's nucleoprotein gene tracking. Blood from 30 Gulf war veterans with unexplained physical symptoms will be used for the comparative studies. Veterans' blood will be used because of the high mycoplasma detection rates reported by Dr. Nicolsons in the studies.

The result from Dr. Nicolson's laboratories and from the three new labs will be statistically compared. This protocol fits the criteria for establishing the validity of a new diagnostic test and the ability to produce and replicate results.

Currently, contracts are being written for the four study laboratories. This process should be completed within 2 weeks. Once contracts are awarded, we anticipate the timeframe for laboratory data collection and analysis will be another 6 months.

In addition, on our Gulflink home page, there is a solicitation by Walter Reed for volunteers to participate in this program. The reason for the most recent delay was contracting procedures, and since this contract will be a sole-source contract rather than taking the time for a competitive contract, certain stand-off protocols had to be established until the contract can be awarded. But we understand from the contracting organizations that the contract should be awarded within the very near future.

In terms of my prepared remarks, I would like to summarize some major points. As you know, the committee asked me to concentrate my remarks today on three areas of concern: low-level chemical exposure, oil well fires, and depleted uranium.

I am accompanied here today by experts that will be able to augment my testimony if the committee wants to get into further technical details not covered by my remarks, Colonels Wolfe and Daxon from the Army and Dr. Jack Heller from Chipham. In addition, Gary Christopherson, the Acting Principal Deputy Assistant Sec-

retary of Defense for Health Affairs, is also here if the committee wants to discuss the recent GAO report.

All three areas the committee asked me to discuss today are under active and, I might say, continuing investigation by my office. In all areas we are pursuing two lines of inquiry, what does science say and what happened in the Gulf. In answering these questions, we are building on the research base that the DOD has already developed and pushing back the frontiers for our knowledge through new research and analysis.

Potential exposure to low-level, chemical agents continues to be an important area of investigation. One case that has gotten a lot of attention for the potential of lower level chem are the detections by the Czech and French chemical detection equipment. These detections occurred during January 1991 in northern Saudi Arabia. United States technical experts described the principal detection claims by the Czechs as credible, although the source of the chemical is still unknown.

Most importantly, we believe, the Czechs continued to use their sensitive equipment throughout the war, but no further detections were reported. We are continuing to investigate this case. In fact, a team will be visiting France and the Czech Republic this summer to discuss these detections and the issue of low-level chem exposures and the sensitivity of the Czech equipment with the Czechs this summer.

A second area of concern has been the results of coalition bombing during the same period of time, January 1991. The CIA published a study in September 1996 that examined the worst possible case for fallout reaching U.S. troops. The CIA reports said that the analysis and computer models indicate chemical agents released by aerial bombing of chemical warfare facilities did not reach United States troops in Saudia Arabia.

To improve our confidence in the results of the original plume analysis, DOD is working with new models that will further analyze the possible effects of a bombing campaign. The DOD experts in meteorology and modeling from the Navy Research Laboratory and the Defense Special Weapons Agency and the Navy Surface Warfare Center will continue our look at the bombing campaign.

Another claim for possible source of low-level chemical contamination to United States troops is the destruction of the ammunition supply point at Khamasiyah. I think you know that DOD and CIA recently completed a series of small-scale demolition tests designed to assist in developing the models to be used to assess the potential fallout from Khamasiyah. The questions we are most interested in are who may have been exposed to chemical agents in Khamasiyah and to what extent they may have been exposed.

During those tests, we blew up 32 foreign-made, 122-millimeter rockets with warheads filled with simulants. The tests provided fundamentally new information on what may have been vaporized versus what may have been spilled into the ground. Additionally, we have undertaken a series of evaporation tests to determine how nerve agent disperses in the soil and in the woods of the crates that were at the site. This work will be incorporated in our analysis of fallout, which is due in late-July.

Another area of investigation is the Kuwait oil well fires. The setting of these fires first were detected on January 24, 1991, and the number of fires increased until it reached a daily peak of 730 in late February. The emission from these fires had the potential to cause acute-and-chronic health effects. Our soldiers were exposed to heavy smoke and byproducts. Research thus far has not indicated, however, that the exposure to oil well smoke has caused acute health impacts to our troops.

We have also contacted the firefighters that participated in extinguishing the fires, and our conversations with them reveal an absence of symptoms that are reported by our veterans. To date, we have found no apparent health problems or long-term effects from exposure to the oil well fires in Kuwait.

Depleted uranium is another area we are investigating. DU is approximately half as radioactive as natural uranium found in the soil and poses no significant external radiation risk to soldiers. The major toxic problem with DU is from its chemical properties. As a heavy metal, it can concentrate in the liver, bones, and kidney, as does mercury, lead, and tungsten; and tungsten is significant here because it is often spoken of as a replacement for DU in munitions.

The problem basically is DU dust generated when DU burns, and it may be ingested and present a health hazard. Soldiers with the greatest potential for harmful effects of DU are those who are in a vehicle when the vehicle is hit by a DU round. Twenty-nine combat vehicles—15 Bradley, and 14 Abrams tanks—were contaminated in this manner. DU from other Abrams tanks hit all of the Bradleys and eight of the Abrams. Five of the Abrams tanks were contaminated when DU munitions burned in on-board fires. Its on-board DU emissions contaminated the final Abrams after being hit by a Hellfire missile.

In addition, 50 soldiers were injured in the Doha Ammunition Dump incident, and it is unknown how many may have ingested DU dust. The Baltimore Veterans' Affairs Medical Center is conducting health service for individuals who were in U.S. Army vehicles when they were struck by DU rounds. Currently, 33 individuals are being evaluated, including 16 with DU shrapnel in their body. The Health Surveillance Program has shown that those who have retained shrapnel identified radioactively are excreting increased amounts of uranium, indicating that the metal particles are not entirely inert.

So far, analysis of the first round of examinations has shown no evidence of adverse health effects associated with the absorption of uranium.

We recognize that we have been deficient in not properly training all soldiers to the risks of DU armor and munitions. The Army has developed a three-tier training program to meet the needs of every soldier, from the soldier on the battlefield to the technical that works with DU.

There is an axiom that states: On the modern battlefield what can be seen can be hit, what can be hit can be killed. That turns out to be a good axiom for the United States, but was not an appropriate axiom for the Iraqis, largely because of the use of DU both as a penetrator and as a protective shield. U.S. forces using 105-

millimeter and 120–DU Sabot rounds routinely obtained first-round kills of Iraqi T–72 tanks at ranges in excess of 2 miles.

And I think Col. Wolfe has with us a mockup of a Sabot round, and I think he is prepared to just talk about that for a moment.

Mr. SHAYS. Was he sworn in?

Mr. ROSTKER. Yes, he was.

Mr. SHAYS. Thank you, Colonel. I appreciate that. That is the actual size of the——

Mr. WOLFE. Yes, sir. This is the 120-millimeter——

Mr. SHAYS. Let me ask you this. Now, we want to make sure the transcriber can pick you up. OK. That is good. That is good. Identify yourself for the record.

Mr. WOLFE. Sir, I am Col. Wolfe, with the Assistant Secretary of the Army's Office.

Mr. SHAYS. Colonel, it is nice to have you here.

Mr. WOLFE. Thank you, sir.

This is the 120-millimeter Sabot round, the Abrams main battle tank; and the misconception is that this entire round is the depleted uranium. That is not so. It is primarily the penetrator that you see here. We refer to it as the "dart," and this is what we have been talking about all day long, is where depleted uranium goes. There is a similar round that has been developed for the Bradley Fighting Vehicle, again, with a small depleted uranium dart.

Mr. SHAYS. How many of those shells are in a tank? That is not classified, is it?

Mr. WOLFE. I cannot answer that, sir. I am not——

Mr. SHAYS. You do not know if it is classified?

Mr. WOLFE. I am not an armored officer, so I do not know.

Mr. SHAYS. The size of it is quite interesting.

Mr. WOLFE. Yes, sir.

Mr. SHAYS. Thank you.

Mr. WOLFE. Yes, sir.

Mr. ROSTKER. When the round is fired, part of the casing stays. The back part of the casing stays in the tank and is ejected, the front casing falls away, and what flies through the air is simply the dart. Somebody said we have returned to the arrows of our forbearers.

What this dart does effectively is provide a certain, first-shot kill to American gunners, and even in the testimony this morning, there was, again, a recounting of the superb performance of the DU round. That really does protect our troops by making sure that they get that first shot in and that that is an effective first shot.

Moreover, we use DU as protective armament, and the tanks that had the DU presentation, that DU never failed and was always effective against the Iraqi chemical, high-explosive rounds. The only thing that can penetrate a DU armor is another DU penetrator.

Mr. SHAYS. I do not understand when you said "chemical."

Mr. ROSTKER. The normal tank round is a shaped-charge explosive, and it generally went out often as a tandem charge so there would be an explosion to defeat the armor and then a second explosion to burn through and hit the turret. But it was a chemical round; it was an explosive round. The dart in this DU projectile we have talked about is a penetrator. It is known as a "kinetic round,"

meaning it is the force of the projectile, and the round is 1.6 times more dense than lead, and it has such penetrating power, that it often went into the Iraqi tank and out the other side.

It flies true, and so with the superior performance of the Abrams tank, the M1 tank, it was able to engage T72 tanks at ranges that they could not engage, providing a sure, first-short kill. There are numerous accounts of the war, however, where Abrams tanks were ambushed, where the T72's got within 400 meters, firing rounds, and they did not defeat the Abrams tank providing presentation for our troops. There is one account, even in the middle of the summer, where an Iraqi tank hid behind an earthen berm, and the DU penetrator went right through the earthen berm, found the tank on the other side, and blew the turret off the tank.

Increasingly, DU, because of its high effectiveness, has been the recipient of an Iranian-run disinformation campaign. United States intelligence agencies have intercepted message traffic, diplomatic message traffic within Iraq or from Iraq directing their diplomats to engage in a disinformation campaign concerning DU, and that assessment has been declassified and is on Gulflink.

Mr. SHAYS. Your point in this, so I do not have to come back to it, is that it is your sense that the Iraqis want to call into question the environmental safety of the uranium in our shells and in our protective——

Mr. ROSTKER. And the North Koreans are doing the same now also. After the Rico Committee Report, the Iraqi Embassies were told to downplay the conclusions concerning low-level chemical exposure, that there was no danger from chemicals, no fallout, no persistence, but that the real pollutant on the battlefield and the cause for illness was DU.

Mr. SHAYS. We accept that as part of the record, but I hope you understand that this committee will be examining this.

Mr. ROSTKER. Absolutely, and that is why I have asked and they have declassified the assessment, and it is available on Gulflink.

Mr. Chairman, let me just end by saying the Department of Defense remains committed to providing appropriate care for our veterans, to understanding what occurred in the Gulf, and to make the necessary changes to our policies, procedures, equipment, and doctrine to protect our current and future force. Thank you very much, sir.

I believe Mr. Christopherson would like to make a statement.

[The prepared statement of Mr. Rostker follows:]

Statement
of
Dr. Bernard Rostker
Special Assistant to the Deputy Secretary of Defense for Gulf War Illnesses
Before the
Subcommittee on Human Resources
House Committee on Government Reform and Oversight
26 June 1997

Mr. Chairman, I welcome the opportunity to once again appear before the Subcommittee on Human Resources this morning. In previous testimonies, I have outlined the mission of my office, described the full extent of the commitment of the Department of Defense, and explained the case management system we use. In light of the recently released GAO Report: Improved Monitoring of Clinical Progress and Re-examination of Research Emphasis Needed, I feel it is appropriate to readdress my mission. We understand the shortcomings of the past and have owned up to them on many occasions. We have learned from our past and applied these lessons both to caring for our Gulf War veterans and to protecting our troops in the future. We will continue to be open and receptive to constructive criticism and oversight that contributes to our understanding and mission.

My mission as the DoD coordinator for all issues relating to Gulf War illnesses consists of three major elements. First, we must ensure that our Gulf War veterans receive appropriate care. Second, we must do everything possible to understand the events of the Gulf War in order to explain Gulf War illnesses. Finally, we must put into place changes to policy, procedures and doctrine as a result of lessons learned from the Gulf War.

To understand why people are sick, we are pursuing two lines of inquiry -- What does science say and what happened in the Gulf? To obtain answers to those questions, we have implemented a formal structure for our incident investigations. Based on an accepted United Nations methodology for chemical incident investigations, our process consists of five steps. First, substantiate the events by researching operational and intelligence logs/records for records of the event, corroborating evidence, and secondary or confirmatory detections. Next research the medical aspects of the event- deaths, injuries, symptoms, medical records and the science associated with the event. Step three is to interview appropriate people, witnesses, NBC personnel, commanders, medical personnel and subject matter experts in order to get a complete picture of the event. Coordination with appropriate external organizations is the next step. Finally, we publish a case narrative that reports all that we know about the event. After publication, if we receive new information from veterans or other sources, we will reenter the process at the appropriate point and update our narrative and findings accordingly.

We are aggressively investigating the events of the Gulf War. As I have previously testified, requests for proposals were published in the Commerce Business Daily soliciting proposals to investigate the causal relationships between illnesses and symptoms among Gulf War veterans and possible exposures to hazardous material; chemical warfare agents; stress; and combinations of inoculations and investigational new drugs during military service in the Gulf War. Proposals have been received and they are undergoing external scientific review.

To further assist our efforts, we have asked the RAND Corporation to conduct an extensive review of medical, epidemiological, occupational and environmental literature

in several areas including: pesticides, immunizations, chemical warfare agents, pyridostigmine bromide, stress, biological warfare agents, depleted uranium, infectious diseases, and environmental exposure to oil fires. Their process involves identifying the biologic relationship between possible exposures and reported symptoms, identifying new areas of research for further investigation such as the health effects of multiple chemical exposures, and conducting focused reviews of these new hypotheses. RAND plans to complete the literature review by the end of August and have their findings undergo peer review and publication. This effort by RAND will add to the previous investigations by the Institute of Medicine and the Presidential Advisory Committee and will give us a clear picture of the existing knowledge base, identify gaps in the knowledge base and identify future research needs.

Potential exposure to low levels of chemical agents continues to be a very important area for investigation. Currently, over $2.5 million has been allocated to research involving health effects of low-level chemical warfare nerve agents. Four projects are investigating the long-term neurological effects of organophosphate exposure and possible treatment strategies. Two others are investigating the long term effects and detection of nitrogen mustard exposure. All current projects will be completed in the year 2000. In January 1997, two additional calls for research proposals were made. Most of the $12.5 million set aside for this research is allocated for chemical warfare agent medical effects and the effects of combinations of exposures. The Research Working Group has reviewed specific proposals and has made recommendations for award of contracts. Details will be published upon award of the contracts.

I take seriously the concerns expressed by this committee and by our Gulf War veterans about the possible presence of low-level chemical warfare agents and their effects on our troops. In response to that concern, we have several efforts underway to investigate the possibility of that presence and to model the areas of potential contamination from our military operations.

One case that we are investigating deals with reports of chemical warfare agent detection by Czech and French chemical detection equipment. During the first several days of the air war, between January 19 and 24, 1991, Czech and French military units reported possible detections of the presence of nerve and blister agents in the vicinities of Hafar al Batin and King Khalid Military City (KKMC) in Saudi Arabia. After examining Czech procedures and equipment, U.S. technical experts described the principal detections claimed by the Czechs as credible, although the source of the chemicals is still unknown. The Czechs continued to use their sensitive equipment throughout the war, but no further detections were reported to United States Central Command (USCENTCOM) or are formally recognized by the Czech Government. We are currently pursuing more detailed information on the French equipment or procedures.

Although the concentrations of chemicals reported were far below the levels considered by U.S. military standards to be a hazard to troops, the detections do indicate the possible presence of low levels of chemical agents in these areas. We are continuing to investigate this case, and in fact, will be visiting both France and the Czech Republic this summer to discuss these and other issues with their experts.

A second area of concern for me has been the highly-publicized possibility that chemical warfare agents were released as a result of the coalition bombing campaign and

may have subjected U.S. personnel to low-levels of contamination. The CIA published a study in September 1996 that examined the worst case possibility of contamination of US troops due to coalition bombing. It concluded "coalition bombing resulted in damage to filled chemical munitions at only two facilities - Muhammadiyat and Al Muthanna - both located in remote areas west of Baghdad." Muhammadiyat, the closer of the two, is 410km north of where troops were stationed at Rafha, Saudi Arabia and even further from the bulk of where troops were stationed. The CIA report also adds, "...analysis and computer modeling indicate chemical agents released by aerial bombing of chemical warfare facilities did not reach US troops in Saudi Arabia." While we continue to investigate reports of other chemical munitions facilities possibly bombed during the war, we have not been able to confirm any other chemical munitions facilities damage.

The United Nations Special Commission (UNSCOM) reported that Iraq declared 200 DB-2 GB Aerial Bombs, 200 LD-250 Mustard Bombs, and 20,000 CS Mortar shells at Muhammadiyat, and 2,500 122mm GB rockets at Al Muthanna. All were destroyed by coalition bombing. CIA's modeling of Muhammadiyat, the larger and closer release of the two, estimated that 2.9 metric tons of sarin and 15 metric tons of mustard were in that site on all possible bombing dates. Their model shows that, in the worst case, dispersion in the general southerly direction for sarin and mustard would fall below levels dangerous to the general population at about 300 and 130 km, respectively, still over 100km short of U.S. troops. Their model for Al Muthanna used 17 metric tons of sarin and determined that the most southerly dispersal for reaching the general population limit dosage is 160 km, again well short of US troops. To improve our confidence in the results of the original plume analysis, DoD is working to model the extent of potential exposure of our

soldiers after the bombing of these facilities. DoD experts in meteorology and modeling will use multiple models. Within DoD, the Naval Research Laboratory, the Defense Special Weapons Agency and the Naval Surface Warfare Center will be contributing expertise to this effort.

After the war Iraq admitted to production of biological agents at four facilities, none of which showed damage from Coalition bombing when inspected by UNSCOM. UNSCOM has also reported that Iraq has never indicated that its population suffered any casualties from release of chemical or biological agents due to Coalition bombing of their facilities.

Another claim of a possible source of low-level contamination to U.S. troops of which I am concerned is through destruction of ammunition supply depots. Consequently, I have several case investigations underway to determine the facts related to such destructions. One such case is that of Khamisiyah, the destruction of which has been well publicized and for which we published an initial narrative in February. During and after the close of the Gulf War, captured munitions were destroyed throughout the theater of operations by Coalition forces. Included in this massive undertaking was the depot level ammunition supply point (ASP) at Tall al Lahm or Khamisiyah, as it was later known. The United Nations Special Commission (UNSCOM) reported that Iraq declared 2,160 122mm rockets containing a mixture of sarin and cyclosarin nerve agents, and 6,240 155mm mustard rounds were at Khamisiyah. In 1991, the UNSCOM inspectors were driven to Khamisiyah by the Iraqis and shown a destroyed bunker called Bunker 73 that they claimed was supposed to have contained 122mm chemical rockets. During this same inspection, they were also shown an area

called "the Pit" where unfuzed 122mm chemical rockets were found in three bulldozed piles. Later in 1996, Iraq declared this area -- the Pit -- was also destroyed by Coalition forces. Research has confirmed that elements of the XVIII Airborne Corps destroyed the warehouses, bunkers and open storage areas in March and April of 1991. It is vital to point out that the engineer and explosive ordnance disposal units took all due caution in conducting their initial searches of the bunker complex prior to the beginning of demolition operations. Both the EOD teams and the engineer commanders were satisfied that there were no munitions in the bunkers, warehouses or in "the Pit" area that could be identified as chemical weapons. Following the first large demolition of 38 bunkers on March 4, which included Bunker 73, engineers and EOD specialists worked in the ASP and "Pit" area for a period of approximately 6 days preparing for the next demolition. During this time the soldiers were not in protective gear but did have active M-8 alarms deployed in the ASP. No chemical alarms sounded during this time with the exception of one M-8 alarm that occurred about 45 minutes after the first demolition on March 4. It proved to be false through documented, follow-up testing. Interviews of hundreds of soldiers who participated in the destruction of those bunkers as well as the soldiers providing security to the site revealed no instances of health reactions consistent with exposure to nerve agents. Photos of an EOD sergeant without protective gear who participated in "the Pit" demolition show him standing in "the Pit" area a day or two after the stacks of rockets were destroyed. Another photograph of a soldier, taken in the same area several days later by an engineer battalion commander, shows this soldier without protective gear. Recent interviews with most of these individuals indicate they

did not have any physical reactions that could be associated with exposure to nerve agents when they were in the area.

The DoD and CIA recently completed a series of small-scale demolition tests designed to assist in developing a model of the detonation of chemical weapons by U.S. soldiers at Khamisiyah, Iraq in March 1991. The results of the tests are expected to produce data that will assist us in answering two fundamental questions: 1) who may have been exposed to chemical agents at Khamisiyah, and 2) to what extent they may have been exposed. The test used 32 foreign-made 122mm rockets and warheads filled with the simulant triethyl phosphate, a substance which replicates the characteristics of sarin gas. The tests fundamentally examined how the rockets explode and how much material vaporizes or spills onto the ground. The CIA and DOD to include the Naval Research Laboratory, the Defense Special Weapons Agency and the Naval Surface Warfare Center will apply information derived from the tests to multiple models. Additionally, evaporation tests to determine how nerve agent evaporates from soil and wood are now being conducted at Edgewood Research and Development Center, Maryland and Dugway Proving Grounds, Utah. We expect the findings to be published by 21 July1997.

Another major area of investigation is the Kuwaiti oil well fires. The setting on fire of the oil wells was first detected on 24 January 1991. The number of fires increased daily, peaking at 730 between 22-24 February. This coincided with the movement of Coalition forces into position for the ground war, which began on 24 February and ended 28 February. Our soldiers began redeploying in March and most had returned home by the end of April. The first American fire fighters arrived in Kuwait in April and were

part of 10,000 workers from 37 countries ultimately involved in extinguishing the fires. By October 1991, all 730 fires had been extinguished.

Emissions from these fires have the potential to cause acute and chronic health effects. Our soldiers were exposed to heavy smoke and other by-products of the fires. During the early stages of the fires, the smoke was close to the ground and caused minor respiratory problems for some of our soldiers. Later, the smoke lifted and stayed at higher altitude, posing less risk. Sampling of the ambient air and soil began in May of 1991 when 558 oil wells were still burning and continued through December 1991. These samples were analyzed for particulates and metals (sulfates, nitrates, etc.), volatile organic compounds (benzine and toluene), polycyclic aromatic hydrocarbons, acidic gases and criteria pollutant gases and did not reveal any chemicals at levels of concern. Research thus far does not indicate that exposure to oil well fire smoke causes acute health impacts in healthy adults. In addition to other ongoing research, the RAND Corporation is conducting an extensive medical, epidemiological, occupational and environmental literature review to determine future research needs pertaining to the health effects of exposure to oil well fire smoke.

We have contacted several fire fighters that participated in extinguishing the oil well fires. Our conversations with them reveal an absence of any of the symptoms reported by our veterans; none have reported any adverse health effects. Larry Flack, former project manager for all fire fighters in Kuwait states that based on his first hand knowledge of firefighter health screening and his periodic contact with firefighters that "We are not ill." Dr. Gary Friedman, Director of Occupational Medicine in the Pulmonary Division, University of Texas, Houston conducted a health screening study of

40 American firefighters prior to and after their deployment to the Gulf. He found no apparent health problems or long term effects from exposure to oil well fires in Kuwait.

We are currently investigating the events surrounding troop exposure to oil well fire smoke and related by-products. The investigation is focusing on the events leading to the destruction of Kuwaiti oil fields. It will investigate the human health effects associated with exposure to oil well fire smoke, present the results of environmental sampling and monitoring studies conducted in the region, and present the results of human health, exposure and risk assessment studies conducted during this time period. We are also reviewing air quality and dispersion modeling data to determine units exposed. We are interviewing firefighters and members of oil companies to obtain information related to health screening studies and medical examinations of those individuals. And we are reviewing operational logs to identify the impact of oil well fire smoke on military operations. We expect to publish our findings by November 1997.

Depleted Uranium (DU) is another area we are investigating. There are many allegations from various individuals and groups that DU is an unconventional weapon equal to chemical and nuclear weapons; that DU causes genetic damage and childhood cancers; that DU is a greater danger to our soldiers than the enemy; and so on.

Uranium is a natural, chemically toxic and radioactive element. When the uranium isotope is extracted, depleted uranium is the byproduct. DU is approximately half as radioactive as natural uranium found in the soil and poses no significant external radiation risk to soldiers. The major toxicity from DU is from its chemical properties. As a heavy metal, it concentrates in the liver, bones and kidneys, as does mercury or lead,

for example. The DU 'dust' generated when DU burns may be ingested and presents potential health risks.

The soldiers with the greatest potential for harmful effects of DU were those on board vehicles which were hit by friendly fire. Twenty-nine combat vehicles -- fifteen Bradley Fighting Vehicles and fourteen Abrams Tanks -- were contaminated in this manner. DU munitions from other Abrams tanks hit all of the Bradleys and eight of the Abrams. Five of the Abrams were contaminated when DU munitions burned in onboard fires. Its on-board DU munitions contaminated the final Abrams after being hit by a Hellfire missile.

On 11 July 1991 at Doha Ammunition Dump, as many as 3000 DU rounds burned. A fire started on an ammunition carrier and quickly spread to surrounding vehicles and ammunition stored nearby. Fifty soldiers were injured in this incident and it is unknown how many may have ingested DU dust. We are currently gathering information to attempt to determine the level of exposure to personnel in the vicinity of the fire.

The RAND Corporation is conducting an extensive review of existing literature that will evaluate the most current research on this issue. The Baltimore Veteran's Affairs Medical Center is conducting health surveillance of individuals who were in U.S. Army vehicles when they were struck by DU munitions. Currently, thirty-three individuals are being evaluated, including sixteen with DU shrapnel in their bodies. The health surveillance program has shown that "Those who have retained shrapnel identified radiographically are excreting increased amounts of uranium, indicating that these metal particles are not entirely inert. So far, analysis of the first round of examination

participants have shown no evidence of adverse health effects associated with this absorption of uranium." Additionally, twenty-seven soldiers from the 144th Supply and Services Company, have been identified as having potentially inhaled or ingested DU dust while recovering contaminated vehicles. Twelve soldiers have been tested with no indications of radioactivity or renal toxicity.

We recognize that we were deficient in informing all soldiers of the risks associated with DU armor and munitions. Training on DU characteristics and risks was limited to Abrams tank personnel, munitions handlers and explosive ordnance disposal personnel. The Army has developed a three-tiered training program to meet the needs of crewmembers, maintenance, chemical, ordnance and medical personnel who may come into contact with DU materials. Training is scheduled to begin in July of this year.

There is an axiom that states "On the modern battlefield, what can be seen, can be hit. What can be hit, can be killed." Used by the U.S. Army in development of doctrine, tactics and acquisition programs, this axiom proved to be true for U.S. forces and totally false for the Iraqis, largely due to the use of depleted uranium (DU) munitions and armor. U.S. forces, using 105mm and 120mm DU sabot rounds routinely obtained first round kills of Iraqi T-72 tanks at ranges in excess of 3000 meters (approximately 2 miles). Clearly U.S. forces could hit and kill Iraqi targets with DU munitions well outside of the approximate 2000-meter range of the Iraqi T-72 tank, thus enhancing the survivability of our servicemembers. Conversely, the Iraqis could not harm our DU armor protected vehicles. Not one Abrams was destroyed by Iraqi tanks nor was the DU armor compromised. Dan Fahey describes in Metal of Dishonor a vignette of an Abrams that was stuck in the mud. He states "The unit (part of the 24th Infantry Division) had gone

on, leaving this tank to wait for a recovery vehicle. Three T-72's appeared and attacked. The first fired from under 1,000 meters, scoring a hit with a shaped-charge (high explosive) round on the M1A1's frontal armor. The hit did no damage. The M1A1 fired a 120mm armor-piercing (DU) round that penetrated the T-72 turret, causing an explosion that blew the turret into the air. The second T-72 fired another shaped-charge round, hit the frontal armor, and did no damage. The T-72 turned to run, and took a 120mm round in the engine compartment (which) blew the engine into the air. The last T-72 fired a solid shot (sabot) round from 400 meters. This left a groove in the M1A1's frontal armor and bounced off. The T-72 then backed up behind a sand berm and was completely concealed from view. The M1A1 depressed its gun and put a (DU) sabot round through the berm, into the T-72, causing an explosion." Our first responsibility is to protect our soldiers and to provide them the best equipment possible. This is but one example of the effectiveness of DU armor and DU munitions in protecting our soldiers and in ensuring their combat success on the battlefield.

Interestingly, DU munitions were so effective that Iraq ran a disinformation campaign aimed at discrediting the U.S. and at potentially eliminating the munitions from future battlefields. U.S. intelligence assets intercepted several messages wherein Iraqi diplomats were directed to initiate publicity campaign to depict DU as posing a severe health and environmental threat to Iraq. Further, the campaign was to allege that the 40 tons of DU found in Southern Iraq came from radiation weapons used against the Iraqi military and that it was contributing to large increases in diseases such as leukemia. Iraqi diplomats were to relay articles containing this propaganda to parliaments, political parties and movements, peace and solidarity organizations, environmental protection

organizations and friendly political VIPs. We plan to publish our case narrative on DU in September 1997.

I am taking a team of my investigators and staff to Europe to share information with the British, French and Czechs. I am hopeful that we can learn from their experiences with low level chemical detections, pesticide exposure and the health effects their Gulf War veterans are experiencing. In September, I am planning a similar trip to Kuwait, Saudi Arabia, Egypt and Israel.

The Department of Defense remains committed to providing appropriate care for our veterans, to understanding what occurred in the Gulf and to making necessary changes to policy, procedures and doctrine to protect our current and future soldiers. We have the right team in place to conduct the necessary investigations. We are open to oversight, have published all that we know and have a moral obligation to those who served to provide them answers. Clearly, DoD must play a central role in the investigation into Gulf War illnesses.

Mr. SHAYS. Sure. And let me say, before we begin the questions, if those accompanying you just want to make a statement, we are happy to hear them. Yes, sir?

Mr. CHRISTOPHERSON. Mr. Chairman, thank you very much. A lot of the questions you raised earlier with both panels of witnesses had to do more in depth with health. I thought I would just cover a couple of brief points, and then we could come back to more questions.

One is I think it is important to understand, as we have looked back at the Gulf war, it has been a very quick recognition mistakes were made. Things did not go as well as they could have been, and I think it is important for us to understand that that is now clearly the position and that is clearly where we see life being at this point.

The second thing I think, which is important for you, is that a lot of the lessons have been learned. It is learned from the point of view of what we do on the battlefield. It is learned in terms of how we approach research and clinical. I would also argue, we are still learning as we go along.

The third thing is that a lot of changes are being made, and we could talk more about them as you wish. One is I think the idea of the clinical program that both VA and DOD have put into place is a program that we intend to have available for future situations as well; therefore, to be ready to intervene much earlier than in the Gulf war.

The second thing is what you are seeing now in terms of what we deployed in Bosnia and currently in Southwest Asia is again an attempt to take surveillance out much earlier, predeployment, during a deployment, and post-deployment kind of work to learn much more about what is happening out there, give us better exposure data to bring back and better records to bring back.

I think, with respect to the research, we are working very closely with VA have built a better research model for peer review, getting it out there, looking at different kinds of treatments. We have done a number of things now. We have committed about $27 million to research this year, a very multifaceted kind of approach to look at issues, low-level chem, environmental hazards, a number of other areas.

With respect to DU, I think I will defer to Bernie on that, other than the fact that that is an issue that we obviously also have some concern about in terms of what the health consequences may be and how much we still do not know yet and need to learn.

On the low-level chem, we have research in place. We have asked the Institute of Medicine to take a look at our clinical programs to make sure that if there were more than one chemical on the battlefield, whether or not we would have picked it up in our clinical program, and they have given us positive feedback, saying they believe it would have. They have also indicated obviously some things we could refine for the future that would make it even stronger.

On the biological infectious side, as indicated by Dr. Rostker, we are clearly looking at the Nicolsons' work. We will fund that. We have our people ready and trained to do so. We are working with independent laboratories as well to make sure that there is really a good, independent look and not a feeling that we, in quotes, have

done it unfairly in terms of DOD. The area of infectious disease is an area that is of high interest on the part of the Department of Defense, an area where we are launching a rather major initiative, along with the Centers for Disease Control and others as well.

In ending—just again our assurance that our job here is to take care of our troops. We intend to do that. We will do that, both for now and into the future. We have a very key obligation. One of the great learning experiences out of the Gulf war is how we better protect our people in the future and a lot of areas in that as well. For that, I will defer for the questions from you.

Mr. SHAYS. One of the things that we really have not touched on is the GAO report. The inside-the-beltway discussion of this was that some were eager to have the GAO validate the VA and the DOD's work, and much to the surprise of some, was that it did the exact opposite. I guess the question is, one, will you agree with some of the criticisms; and, two, if you agree with them, do you feel you have changed or no longer are deserving of that criticism?

One of the criticisms is that too much of the research that is done on Gulf war illnesses is devoted to stress and Post-Traumatic Stress Disorder, PTSD. Would you explain—my understanding, about a third of all research is, in fact, on this area, and would you explain why; first, if it is true, and, second, why?

Mr. FEUSSNER. There are several—I think that there is a major emphasis on the research in the context of brain and nervous system disorders, that is, along with general health types of research initiatives, that is the major research focus to this date. That includes an array of research that deals with stress and Post-Traumatic Stress Disorder, deals with issues related to cognitive impairment, deals with issues related to Peripheral Neurological Disease, et cetera.

So, in the sense that all of brain and nervous-system disorders are lumped together, that is a major focus. I am not sure that it is correct to categorize most of that as relating to stress; however, there has been interest in the neurobiological aspects of stress and stress as a modulator of various responses to other insults, and that kind of research continues.

Mr. SHAYS. I need to be clear and on the record as to whether the VA rejects Dr. Joseph's point to this committee that there was no acute exposure to chemicals and, in essence, low-leverage exposure is not harmful; and, therefore, chemicals exposure should not, in essence, be considered of major concern. I want to know how the VA basically responds to that.

His quote was: "Current accepted medical knowledge is that chronic symptoms or physical manifestations do not later develop among persons exposed to low levels of chemical nerve agent if they did not first exhibit acute symptoms of toxicity." Now, I need to know if that is—I am going to be asking DOD if that is the operational use still, and I need to know the VA, if they buy into that or if they have finally rejected that.

Dr. GARTHWAITE. I do not think we buy into it. I would think we do not know what the risk is, but we are keeping an open mind. We do not believe there are any reasonable, valid human studies of those kinds of exposures, so to conclude anything, we think, would be premature.

Mr. SHAYS. OK. I am not making my question clear enough. The bottom line to his point was that if you did not see acute manifestations, that you would then not later see chronic effects from chemicals. That was a basis for why the VA did not spend time looking at chemical exposure, because you accepted the DOD's view that there was no acute exposure, and if there was not acute and therefore low level, it would not result in chronic harm later on.

I want to know if we can take Dr. Joseph, who was the Assistant Secretary of Defense for Health Affairs, if we can put that in the trash can and know that that is not a guiding principle of either the DOD or the VA.

Dr. GARTHWAITE. I think that is his opinion. We do not believe that there is any scientific data on which to base an opinion about whether exposure to low levels could lead to a chronic disease or not in humans. There is very little data from studies in animals, either.

Mr. SHAYS. Why don't we forget about any concern of low-level exposure in this country? Get rid of OSHA, say, OSHA, you are not needed anymore because we do not care about low-level exposure to chemicals.

Dr. GARTHWAITE. I am not communicating well. We believe that because there is no data, we need to know whether——

Mr. SHAYS. I am going to come back to you, Doctor.

Mr. ROSTKER. We would not necessarily—that is not our position today, as you stated. We are funding research in low-level chem. We have not ruled it out.

Mr. SHAYS. I would like you to say what is not your——

Mr. CHRISTOPHERSON. Let me elaborate on that.

Mr. SHAYS. I just want to say this to me, is like—before we go out—this is something we should be able to discard quickly.

Mr. CHRISTOPHERSON. We have to agree. Let me go back. It is a need to understand the context of what you said and what it meant, because that is important.

Mr. SHAYS. And I am willing to be clear on this, but I do not want to get into the mind game——

Mr. CHRISTOPHERSON. No, no, no.

Mr. SHAYS [continuing]. Where Mr. Deutch says publicly that our troops were not exposed to offensive use of chemical when he knew our troops were exposed to defensive. Because he used that clever word of "offensive," we made an assumption that, therefore, our troops were not exposed to chemicals. So——

Mr. CHRISTOPHERSON. Right.

Mr. SHAYS. OK.

Mr. CHRISTOPHERSON. What his statement was saying was, based upon the best scientific knowledge which is out there—it actually still is out there at this moment in time—the conclusion you would have is that you do not have chronic without acute in terms of the chemical exposure. Now, the key thing is there, and that was, by the way, still the best knowledge. It is very thin; that is the problem with it.

That is why we have said, while that is essentially true as a current statement of what the information is, you cannot base long-term judgments on that. That is why we said instead two things.

One, the Institute of Medicine said, help us to figure out in our clinical programming in case it is out there, we miss something.

Mr. SHAYS. OK.

Mr. CHRISTOPHERSON. The second thing, we went out there and said, let us go ahead and start to fund some low-level chemical research because we have got to fill in this rather thin body of knowledge. The concern that you are raising back there about and this whole issue of why was not low-level chemical picked up a long time ago, we sort of put in the context of combinations, I think, of things.

It is not that statement of judgment or anyone else. What it is, is a combination of no direct evidence, my understanding is, off the battlefield, combined with the fact that the best knowledge that was available out there was that you generally have to have an acute exposure; and, therefore, people have thought, this does not seem to be the most promising lead, and there may be other more promising leads.

Going back to your EPA point, the germ of the point that is made there is that you are looking at generally longer term exposures at low level as opposed to a short-term exposure. The other assumption is generally that the exposure in the Gulf would be of relatively short duration.

If you think back, for example, to what the witnesses said this morning, they were generally talking about, at the most, there would be eight alarms going off, which is generally indicating, even if there had been some exposure during that time, it would probably have been over a relatively short period of time, maybe 8 days, 2 days, and this kind of thing there, which again is very different than sort of the pesticide issue, which is something the British especially are focusing on.

Mr. SHAYS. I am going to let Mr. Sanders get on this issue before we go on to the next one.

Mr. CHRISTOPHERSON. Sure.

Mr. SHAYS. I do not mean to be—I do not want to strain gnats and swallow camels here, but when you say this is our best knowledge, the word "best" has such a great sense to it. The best knowledge may have been meaningless because your best knowledge may just be absolutely dumb and stupid. And so you can say, "Of the dumb-and-stupid knowledge we have, this is the best, but it is still dumb."

Mr. ROSTKER. You are reading it as in plain English as quite a declarative statement. We would not be happy making that statement as a declarative statement today.

Mr. SHAYS. The problem is the VA used this statement as a basis for a failure to look at low-level exposure.

Mr. ROSTKER. And I think we are talking about history here, not necessarily where we are today.

Mr. SHAYS. And that is why I want to be certain. I just want to make sure that we are not trying to, in a sense, satisfy us, but in your heart of hearts, you still buy into this.

Mr. ROSTKER. It was not the applicable statement today. Today, we are funding research to better understand low-level chem. We are more modest in our statements in terms of our understanding. We have a range of activities going on to better assess what science

is telling us and push back the frontiers of science, so that would not be—it is not an applicable statement today and not a limiting statement for our program today.

Mr. CHRISTOPHERSON. But, again I want to come back. That declarative in nature, which is what you have described, was not the case even back then. In the first place, our moving forward on funding low-level chemical was under the watch of Dr. Joseph. Our movement in that direction was a request to the Institute of Medicine for them to look at our clinical protocol was also to Dr. Joseph. That is why I say——

Mr. SHAYS. What about Dr. Joseph? It was what?

Mr. CHRISTOPHERSON. Under his tenure.

Mr. SHAYS. Well, by then we had Khamasiyah, and you all on a Friday afternoon at 4 o'clock let the world know that maybe we had exposure. So I am just not impressed with that comment.

Let me just go back to the VA, and then I will let you talk. I just want to know where the DOD is. I just need to now know where the VA is. What I hear you saying, so then you correct me, where I start out is may be faulty from your viewpoint.

I start out from the fact that in my life as an American citizen, and as a State legislator, I have been taught to be concerned with low-level exposure, and I have been taught that low-level exposure leads to chronic illness. In my world as an American citizen and as a former State legislator and as a Member of Congress, I pay attention to OSHA, and I empower OSHA not to allow American citizens to be exposed to—low-level exposure to chemicals. That is my world, and what I am hearing you say is, well, that may be true, but if it is low level, it has got to be over an extended period of time.

What I totally reject and am comfortable rejecting is that it has to be acute and if it is acute, it cannot be chronic, because I have never seen anything that would make someone be allowed to make that statement.

Dr. GARTHWAITE. I believe the correct thing to say is we agree with you, and——

Mr. SHAYS. I want you to state it in your own words.

Mr. FEUSSNER. Yes. I think what I would similarly agree and say, that I think it is clear that we have insufficient information to know what the possible long-term sequelae of low-dose exposures are, and I think we need to do additional research to explore that.

I think in some ways we have spoken with our actions when we sponsored the international symposium associated with the Society of Toxicology meeting in Cincinnati in March. We began planning that meeting in September 1996 and invited the international community to help us specifically with the issue of low-level chemical agent exposures, and I think we need additional research to explore the sequelae of possible low-level exposures.

Mr. SHAYS. Mr. Sanders.

Mr. SANDERS. Thank you very much, Mr. Chairman. Thank you all very much for coming, and I apologize for having to miss some of your testimony.

Let me ask for some rather specific responses to my questions. In December 1996, in the final report of the Presidential Advisory Committee on Gulf War Illness, the following statement is made,

and I quote: "Current scientific evidence does not support a causal link between Gulf veterans' illnesses and exposures while in the Gulf region to the following environmental risk factors assessed by the Committee: pesticides, chemical and biological warfare agents, vaccines, pyridostigmine bromide, infectious diseases, depleted uranium, oil well fires, and smoke and petroleum products."

That is from the Presidential Advisory Committee. Today, in late June 1997, do you agree with that finding, or do you find that incomplete and inaccurate? Dr. Rostker, or if anybody else wants to respond.

Mr. ROSTKER. Well, as you know, we have discussed several times my inquiries are looking at what science says, and I have great respect for the PAC and the process they went through. I certainly am considering that, but in my organization I am reserving judgment, final judgment on all of these. I have research going on on every one of the issues that you have raised, and that research continues. I wish it was completed so I could be definitive in my answer. I can only tell you that the research continues in my organization.

Mr. SANDERS. OK. In so many words, what the PAC was saying is that we see no substantial scientific evidence to suggest that there is an environmental factor in Persian Gulf illness. Rather, we believe, bottom line, that it is stress related. That is not the conclusion? Dr. Murphy, I can continue reading, but I believe that that is—but, please, if you disagree with me, I have got the document here.

Dr. MURPHY. Let me try to restate it because I think that the words that I used have a different meaning to scientists than they might to the general public. They said that there was no current evidence of a causal relationship. That is probably the highest scientific standard that we would meet in discussing that, so there is no evidence that those agents at this point caused the illnesses to Persian Gulf veterans.

Mr. SANDERS. That is correct. That is what they said.

Dr. MURPHY. They have not ruled out doing further research.

Mr. SANDERS. I know, but let me ask you, can you respond to that? Do you agree with that? Do you believe that there is no current scientific evidence which sees a causal relationship between environmental——

Dr. MURPHY. There is no rigorous, scientific——

Mr. SANDERS. No rigorous. All right.

Dr. MURPHY [continuing]. Investigation that proves a cause-and-effect relationship between the illnesses of Persian Gulf veterans and those agents. That does not mean that the VA has not given them very serious consideration and does not believe that the investigations need to continue at this point. We are trying to develop the scientific evidence that would allow us to make that scientific, causal link.

Mr. SANDERS. What I have concerns with, Dr. Murphy and everyone else, is when you will finally begin to accept evidence. I am not a scientist. I have other things to do other than research Persian Gulf illness, but I sent a letter out to Dr. Lashoff of the Presidential Advisory Committee, listing a dozen, separate studies which show a link. If you would like, I can list them for you, al-

though I suspect that you are familiar with them, including two studies funded by the DOD itself.

Now, the concern that I have, and let me jump right to the GAO report, and this comes from the summary of it by the New York Times. The GAO report found that the program announced by the Pentagon lacks a coherent approach, and because of flaws in methodology and focus "is not likely to identify the potential causes of the illness."

In other words, what they are saying is there are a dozen different studies here which would respectfully disagree with you, Dr. Murphy. They suggest that there is a causal link. When is enough enough? When do we begin to say, yes, there may be something there; we want to develop treatment based on these studies? I am amazed. Let me give you just two examples, Mr. Chairman, of things which really fascinate me.

The New York Times, April 17, 1996, headline: "Chemical Mix May Be Cause of Illness in Gulf War." What the article primarily deals with is the work that you are familiar with done by Dr. Haley and Dr. Abodonia from Duke, and Haley is from the University of Texas. OK? They describe it, and they say, well, these investigators have suggested that there is a synergistic effect between pyridostigmine bromide, et cetera.

Then they go to a comment from the Department of Defense. The Department of Defense said that the new report raised "some interesting hypotheses," but the Department had "no direct knowledge of the details of the work." Do you know what amazed me? What amazed me is less than a year before, the Department of Defense had done research which came up with exactly the same conclusion at Fort Detrick on rats. Is that true? I hope you know that. That is your own research.

Mr. CHRISTOPHERSON. Yes. What you have got there, there is—and, again, this research, as you know, has been funded—there is research looking right now at the synergistic effects. There are early suggested results that say, in fact, those things do occur. The problem is that what you have seen there, if I may finish for a second here, is it is the first step, and it has to do with how you do sort of the first researchers say, "OK. Could there possibly be under the most severe of circumstances there?" That is Step 1. Step 2 then comes down to initial funding researchers say, "Does it occur under real-life situation?" That is the additional funding and research we need to do.

What you have got then, kind of going back to Dr. Murphy's point there, is there are a number of areas that we are looking at right now which are suggestive of potentials of relationship to Gulf war illnesses. They do not yet stand the rigor of tests yet, so they are suggestive we need to pursue——

Mr. SANDERS. All right, but 1 second. I understand that, but you see, that is always the argument. Let me just pick up, Mr. Chairman, because I found this absolutely fascinating.

New York Times, Wednesday, May 14th, headline: "Study Links Memory Loss to Nerve Gases in Gulf." Do you know who paid for the study? We did. OK? First paragraph: The Defense Department said today the Pentagon-sponsored research have produced "important results" suggesting that exposure to low levels of nerve gas,

Mr. Chairman, and some pesticides can lead to memory loss, a common complaint among veterans of the 1991 Persian Gulf war. This is your study.

Now, what really fascinated me about this article, if you go down three-quarters of the article, and it said: In its statement today, the DOD said, "These initial findings require replication of the species, including nonhuman primates, before it could be possible to draw larger conclusions, the experiments, et cetera, on nonprimates laboratory, et cetera. The Pentagon also questioned whether the experiments in which the rats were injected with the chemicals over a 2-week period offered many clues to the health problems of the veterans. This route of administration and duration of exposure does not parallel any known human exposure to troops."

That is what the DOD said. Do you know what the researchers said? Dr. Pendergast is on your payroll. You know what he said. He said, I do not think it is too early to draw conclusions. "The type of exposure regime that we employed in the animals and the type of exposures that are troops experienced in the Gulf are analogous, and they types of memory deficits that we have seen in the animals and those reported in Gulf war patients are extremely similar."

In other words, you are almost disowning or separating yourself or minimizing the result that your own researchers got.

Here is the point: The GAO says that there is no focus. It would seem to me that if I had a dozen different studies all over the country done by reputable scientists, including some of your own, that suggest that there is a chemical link, I would be jumping on the stuff, I would be funding the stuff, I would be funding the stuff, I would be bringing these people together, and I would be working with a sense of urgency. I would not be going along, da-da-da.

There may have been some major breakthroughs. Am I qualified enough to tell you whether these breakthroughs are substantial? I am not, and I certainly agree with you. But what really upsets me is that I read you a quote where a study done paralleled your own study, and you do not even acknowledge and say, "Yeah, that parallels what we did a year ago, and we are really working frantically hard because we have 70,000 veterans who are hurting, and we are going to leave no stone unturned."

Do you have a sense or urgency? Are you really going after these issues?

Mr. CHRISTOPHERSON. Yes. Mr. Congressman, absolutely yes. Let me be very clear. It is extremely important to us. We have the doctors and nurses and the researchers, as part of what we fund here in Health Affairs, and the rest of the Department take this extremely seriously and have since day one. We have clearly been very active, especially in the last 2 years. Should we have started earlier? That is a different question. Yes, we should have. We already admitted that that is a shortcoming of the whole thing.

It is clear we are funding research as fast as much money as we have to do so——

Mr. SHAYS. Doctor, you are starting to talk as fast as this guy. Because he is a Congressman, I did not want to ask him——

Mr. CHRISTOPHERSON. I can probably outdistance him.

Mr. SHAYS. I did not want to tell him to slow down, though I was tempted, but if you would slow down.

Mr. CHRISTOPHERSON. I will slow down. What we are doing right now is we are pushing—you have got to remember, by the way, there are a lot of different theories out there we are all trying to work through simultaneously. A lot of things have promise, whether it is the plasma kind of issue there, whether it is the issues around the combinations and, therefore, you might go down that road, leave no doubt that there is a serious commitment to try and find the answer.

There are two reasons for this. One is because the Gulf war veterans who are trying to figure out how to take care of them today. I heard the same tragic stories you heard a few hours ago in terms of their—we take these to heart, and leave no doubt about that.

The second thing, we have got to be worried. We have got future deployments to worry about, and we have got to figure out what we are going to do there, and we need to know what we need to change, if anything, to make sure that is better there. What we have got to do now is we also owe it to the troops to do two things: Pursue aggressively and make sure it is good research. What we cannot afford to do is go down wrong paths, start doing treatments that do not make sense. On the other hand, if it makes sense, we cannot afford not to do it, and that is the fine line we keep moving down as we move forward very aggressively.

But no doubt, we are the ones who pushed forward the $27 million and pushed the research out.

Mr. SANDERS. All right. Let me just ask you. Let me quote from Dr. Rostker's prepared statements. Currently over $2.5 million has been allocated to research involving health effects of low-level chemical warfare agents, et cetera. All current projects will be completed in the year 2000.

I mean, you know, is that a sense or urgency, in the year 2000, 3 more years?

Mr. CHRISTOPHERSON. I think what you run into, we unfortunately are living within some of the rules and regulations unfortunately of how you do grants. We are not happy with it either.

Mr. SANDERS. Then break the rules. You know, one of the problems that we have right now—let me finish. All right? And I would like some answers to this question, too. My understanding is that around this time you are releasing about $8 million in grants. Is that correct?

Mr. CHRISTOPHERSON. Correct.

Mr. SANDERS. You are going to announce who is not getting it. I do not know who is getting it. By the time you have announced requests for proposals and you have peer reviewed and you are getting the money out, in my estimate it is going to be a good year. Is that a fair estimate or more than that?

Mr. CHRISTOPHERSON. It is probably in that range, yeah. It takes that time to get it out, unfortunately.

Mr. SANDERS. But why? In other words, the point that I am getting and why I myself no longer believe, in all due respect, that the DOD and the VA should be given this responsibility, is it should not take that long if we are dealing with a sense or urgency.

All right. Let me ask you this question.

Mr. SHAYS. Do you want to just respond, though?

Mr. SANDERS. Why does it take a year when you have 70,000 people who are hurting? Why can't you move it faster?

Mr. CHRISTOPHERSON. The issue—we are caught between two pressure points, and Congress is part of that, where it is part of our own two pressure points. On the one hand, we are told to move forward as fast we can, which we would like to. We are also told to make sure you are doing peer-reviewed research that is going through—we are caught between two things, and then also make sure——

Mr. SANDERS. The chairman is much more polite than I am when he says I should be patient. He is right. I love the word "peer review." You know why I like the word "peer review"? I will tell you why. As you know, and as Ed Towns, I think, appropriately mentioned before, the whole issue of multiple chemical sensitivity is highly controversial. You have honest and good people on both sides of the issue.

Mr. CHRISTOPHERSON. Yes.

Mr. SANDERS. I am not here to denigrate anybody. I happen to believe in it; honest, sincere people do not. Who do you have who is peer reviewing these proposals who believes and knows something about multiple chemical sensitivity? Give me the names of the experts.

Mr. CHRISTOPHERSON. I cannot. In the first place, I do not get down that deep into that part of it there. We use the American Institute of Biological Science as our peer-review organization that what goes out there and does that.

Mr. SANDERS. Well, here is the problem, you see. I do not mean to be facetious about it.

Mr. CHRISTOPHERSON. I understand. I understand. We get along well, and we are working together on this issue. Do you agree? We have disagreements on other issues, Republican, independent, so forth and so on. In the world people look at issues in a different way. I read the response of your folks to one of the proposals that came through, and it was absolutely insulting to the fellow who wrote the proposal.

In other words, if you do not have people on your staff who understand and believe in multiple chemical sensitivity, that every approach that is brought forth will never get peer reviewed, in some cases these researchers will be seen as quacks or frauds. Right? I am arguing and have seen from the beginning, from day one, we do not have people who believe in multiple chemical sensitivity, and I am not even blaming you. There is a whole segment of medicine that does not believe in it.

I think you do not believe in it, and that is fair enough. But there are people who do believe in it who believe that you are way behind the time, who are desperate for solutions, and who want to see some attention given to those folks who do believe in the concept, and I do not think you have the capability of doing it.

I am sure you have wonderful scientists, but tell me the name of one of those scientists who has developed a treatment that is effective for Persian Gulf illness so that he or she can stand in a position of peer reviewing of the research. Who are the people who

have developed the treatment and the understanding? Can you give me the names? You do not have anybody. Is that right?

Mr. CHRISTOPHERSON. Again, this external peer-reviewed stuff. This is not—we are not talking about inside-the-shop kind of thing. The American Institute of Biological Science, which we run this through, is designed to be impartial to a wide range of theories. They are not to be either against or for multiple chemical sensitivity. It really is meant to be a neutral place out there to look at these issues and to be open on the question of what may make good sense, either from researching causes or researching treatments.

The difficulty is, and correct me if I am wrong, that the issue of multiple chemical sensitivity is hotly debated.

Mr. CHRISTOPHERSON. Correct.

Mr. SANDERS. I have spoken before—it must have been 500 doctors in a room in Texas, and you know what? Every one of them believed and works with the concept of multiple chemical sensitivity. And I have met doctors who have said that these people are frauds, that what they are doing is absolutely outrageous, and we have nothing to do with them. Both groups of people, I suspect, are honest.

I think that the VA and the DOD have sided with those groups of people who do not believe in multiple chemical sensitivity, so I am asking you—for example, I would mention that Dr. Claudia Miller, who does believe in multiple chemical sensitivity—I do not want to speak for her. She applied for a grant. She went way up the bureaucratic ladder. The DOD awarded her the grant, and lo and behold, she never got the money; it was called back.

Dr. Mya Shayevetz, who worked for the VA in Northampton, MA, went along the bureaucratic ladder. She treated people based on multiple chemical sensitivity. Suddenly, she did not get any money as well.

Who do you have that is key on your staff who believes in multiple chemical sensitivity? Please answer that.

Mr. Chairman, I do not hear much of a response.

Mr. CHRISTOPHERSON. I cannot point to someone who is a believer in there. What I will indicate to you is that I am being neutral on it. I do not have a strong feeling one way or the other. I do not have an opinion one way or the other in that process there. But if the issue is, as you said, there is a very sharp debate out there——

Mr. SANDERS. Yes.

Mr. CHRISTOPHERSON [continuing]. And that debate continues. It has been part of our discussion. We have talked about the Institute of Medicine. We have added special sessions talking about that because we are open and trying to look at what makes sense, as long as they are good science, and that is the key issue.

Mr. SANDERS. But that is the problem, and you know it, and I know it, that there are many people who say that the whole issue of multiple chemical sensitivity is bad science. Right, Dr. Murphy? Aren't there some people who are saying that?

Dr. MURPHY. There are people who say that.

Mr. SANDERS. I think I have heard people say that. OK? Maybe some people in this room have said that. What about Dr. Haley?

Is his work important? Is he going to work with you? Is he going to get funding from you?

Mr. CHRISTOPHERSON. Dr. Haley is important to us, as both we and Dr. Phil Landrigan, who reviewed his piece there, consider his work important. He has identified a number of key areas to look at. The question is, it has to be taken some next steps to figure out where——

Mr. SANDERS. Is he going to get funding to get research?

Mr. CHRISTOPHERSON. I do not know at this point. Again, the funding part, I cannot speak to.

Mr. SANDERS. Who can speak? Again, when the GAO talks about lack of focus, that is what they are talking. You cannot talk to funding. You are telling us that you are going to do research, but you cannot tell us what line of research is going to get funding. Dr. Rostker, do you want to help us out here?

Mr. ROSTKER. Yes. I think in the process you are talking about specific researchers—in the peer review process——

Mr. SHAYS. Let me say this. One thing is very important. I do not want you to leave without feeling you get to answer a question.

Mr. SANDERS. Absolutely.

Mr. SHAYS. So he is really a nice guy, and you can tell him you want to respond to it.

Mr. CHRISTOPHERSON. In the peer-review process it would really be appropriate as policy and senior people to get down and dictate which researcher is being funded and which is not, and I might say that the intervention that we have done in the case of Dr. Nicolson's research is somewhat unique.

But I look at the focus issue in broader terms, and we went out in this year's allocation of funds through the interagency organization we have with the VA and explicitly went out to fund research in the area of low-level chem because we felt that this had been neglected and that we needed more answers. And so I look at that in terms of the broad focus of the research as distinct from picking the individual research topics.

Mr. SANDERS. Let me respectfully disagree with you. What we are involved in, and please tell me if you disagree with me, is a major controversy over the causation of illness. I happen to believe—I have seen it; I work with people—I believe in the concept of multiple chemical sensitivity. There are many people who do not.

What I am suggesting, and I think there is no question about this, that unless you have scientists and physicians who believe in that concept, that every single time a research grant comes forward based on a diagnosis of multiple chemical sensitivity, the result is going to be, sorry, these people at best do not know very much; at worst, they are frauds.

It cannot be otherwise, and I would say that the evidence indicates up until this point that you have not been sympathetic to the concept of multiple chemical sensitivity. I have asked you if you could tell me—I know the names of some of these people, and I would like you to tell me that they are on board. Is Dr. Miller playing a key role as a peer reviewer? I do not think so.

Dr. MURPHY. Dr. Miller is on the VA Federal Advisory Committee. She is on our Persian Gulf Expert Scientific Committee, and we solicit her advice through that mechanism.

Mr. SANDERS. Yes. Believe me, I do know that, and I do know that her grant was rejected. Can you give me the name of any major researcher who believes in multiple chemical sensitivity who has gotten help from either VA or DOD? Is there any?

Dr. MURPHY. Yes. East Orange Environmental Hazards Research Center has a project ongoing, looking at the issue of multiple chemical sensitivity, and the researchers from East Orange are actively involved in the investigation of MCS in Gulf war veterans at this point.

Mr. SANDERS. Do you know some of the names offhand?

Dr. MURPHY. Howard Kipen. Dr. Howard Kipen is the principal investigator.

Mr. SANDERS. OK. My last question gets down to Dr. Nicolson, and then I am going to get the mic over there. As I heard him—Mr. Chairman, correct me if I misheard him—he indicated he had not been hearing from you guys for a number of months, that originally there was some contact.

Mr. ROSTKER. Yes. I put that in the record. We had a number of interactions through March. In March, the protocols were agreed upon, and because this was going to be a sole-source contract, the DOD rep was advised that what we needed to do was work through the paper work. We have made sure that the money is there——

Mr. SANDERS. Right.

Mr. ROSTKER [continuing]. And that we expect a contract to be awarded to the four laboratories within the next 2 weeks, and those laboratories then will be—and I went over the protocol roughly. Those laboratories then will be trained. Three hundred samples will be drawn. We have already put out a public announcement seeking volunteers to provide blood samples for this research, and we are looking for the research to take about 3 months, which the majority of the time would be training and certifying the labs in the three techniques.

Mr. SANDERS. And what is Nicolson's relationship to this work?

Mr. ROSTKER. He will be contracted with to supervisor the instruction and certification of the labs in his technique.

Mr. CHRISTOPHERSON. And also he will be involved in also training people in his technique——

Mr. SANDERS. OK.

Mr. CHRISTOPHERSON [continuing]. So they will understand what he has got, and then they will go back.

Mr. SANDERS. So he will on day one write off and say these guys are trained, they are doing the work——

Mr. ROSTKER. That is correct.

Mr. SANDERS [continuing]. And we feel good about this, and then we will see the results of that work.

Mr. ROSTKER. And then the independent lab, we are going to have one of our labs and an independent lab both to look at this.

Mr. CHRISTOPHERSON. A given sample will be sent to several labs. In some cases a sample may be sent several times to a lab, and they will see if there is corroboration between the techniques and between different labs using the same technique.

Mr. SANDERS. I yield, Mr. Chairman. Thank you.

Mr. SHAYS. Thank you. Just to give you an idea, I do believe we will get you out of here before 4, just to give you a sense.

I just want to, because there was an interruption—not an interruption, but we went in a slightly different direction, I am going to say that I am not interested when you all appear before me in the future to know what your view is on Dr. Joseph. I am basically accepting on the statement in terms of low-level exposure and what it means and does it ultimately lead to acute symptoms or chronic symptoms—excuse me.

I am going to basically go under the assumption, unless you tell me differently, that you are taking a position of neutrality on that issue. You are basically saying you would either say yes or no, or are you going to say that you believe that low-level exposure can lead to chronic conditions in the future? I would like to have you just tell me where you are on that level, but I at least know you are rejecting that it does not.

Mr. CHRISTOPHERSON. To be clear, we are open on the issue. We are at this point essentially about as neutral as you can get, given sort of the weighing of information, enough so that we are willing to go out and fund research in this area, enough so that we are willing to ask some very tough questions of our clinical program.

Dr. GARTHWAITE. I think the same.

Mr. SHAYS. Dr. Rostker, how many sites were there in Kuwait—excuse me—in Iraq and the Kuwait theater that we suspected had either biological and chemicals in them, be they manufacturing or depots?

Mr. ROSTKER. There were many bombing lists, and targets came on and off the bombing lists based upon the latest information and in some cases the latest fad because things like the shape of a bunker became an indicator to the intelligence analysts of whether or not there may or may not have been chemicals in there. I think the maximum number was something like 34 if you took the intersection of all the lists. About 34 was the maximum number that DIA carried.

Mr. SHAYS. And how many of those were blown up?

Mr. ROSTKER. The manufacturing plants were blown up. The chemical and biological sites were targeted, but it is not clear what was blown up. What we clearly understood after the war was a great deal of the munitions were not in the bunkers but were out on the desert. In fact, the majority of the munitions at Khamasiyah were not at Bunker 73, but were either in the pit or the 6,000 chemical rounds that were simply out in the desert under a tarp, so that what was attacked, whether we hit or did not hit the bunker, was no indication of the amount of chemical munitions we would have detonated.

And, in fact, after the war, when we were able to get into some of these sites because we had occupied that area, like Telio and Ananzarea, the bunkers we thought had chemical munitions did not have chemical munitions.

Mr. SHAYS. Has the U.N. completed site visits of all——

Mr. ROSTKER. The U.N. has done site visits, but the U.N.'s purpose of doing site visits——

Mr. SHAYS. You interrupted me.

Mr. ROSTKER. I am sorry, sir.

Mr. SHAYS [continuing]. Of all these sites?

Mr. ROSTKER. No, sir. The U.N.'s purpose of doing site visits is to investigate the Iraqi claims in their declaration statements. There are a few places where the Iraqis, based upon their own intelligence—excuse me—the UNSCOM, based upon their own intelligence, asked to be taken, and, to the best of my knowledge, they turned out in each case to be a conventional site. And, again, they were looking for S-shaped bunkers or 12-frame bunkers and the like. Khamasiyah and Ananzerea were two of the sites that were declared to UNSCOM——

Mr. SHAYS. Both sites were in the Kuwait theater?

Mr. ROSTKER. Yes. The Kuwaiti theater——

Mr. SHAYS. Our soldiers were in both sites.

Mr. ROSTKER. Yes, but the Kuwaiti theater is sometimes confused with Kuwait, which it is not; it extends past Kuwait, precisely.

Mr. SHAYS. The theater where our troops were?

Mr. ROSTKER. The Kuwaiti theater was a map reference before the war which included southern Iraq, and it had no relationship to where the troops finally went. So some people get hung up on whether it was in or not in the Kuwaiti theater. That is really a technicality. We are talking about Iraq and Kuwait. The area we have absolute knowledge on is Kuwait because that is where we stayed after the war.

Mr. SHAYS. Does the DOD and do you, either one, have knowledge of any sites still being called hot sites that you cannot visit?

Mr. ROSTKER. Not that I know of, no.

Mr. SHAYS. It is your testimony that you have no knowledge of any site being still considered a hot site.

Mr. ROSTKER. No, sir, either by us or by UNSCOM.

Mr. SHAYS. Are you aware of any classified material that either speaks to—let me see how I can ask this question. Are there classified reports about these sites, any of the 34 sites?

Mr. ROSTKER. That are outstanding. No, I do not.

Mr. SHAYS. Have you seen every classified report——

Mr. ROSTKER. I believe so, and there is another check to this, if I might, Mr. Chairman. A totally independent group under the direction of Walt Yako, the Special Assistant to the Secretary of Defense for Intelligence Oversight, has been carrying out a parallel intelligence investigation of Khamasiyah and any other similar sites in Iraq, and I have reviewed their preliminary reports, which had full access to our data and CIA's data, and there were no other sites that were, as you would call, "hot."

Mr. SHAYS. Are there any, to your knowledge, Inspector General reports or reports by the GAO that call into question or review the protective gear that our troops used in Kuwait—excuse me—used in that battle?

Mr. ROSTKER. The protective gear?

Mr. SHAYS. Masks?

Mr. ROSTKER. Say that again, sir.

Mr. SHAYS. Masks?

Mr. ROSTKER. There were concerns about masks fitting, and we have gone to a new, universal mask.

Mr. SHAYS. Have you seen any classified reports that cannot be released to the public that discuss the validity and integrity of either the M–40 or M–17?

Mr. ROSTKER. No, sir.

Mr. SHAYS. Have you seen any reports?

Mr. ROSTKER. No, sir.

Mr. SHAYS. Do you know of any reports existing that discuss them?

Mr. ROSTKER. No. In fact, we just made a report to the President's Advisory Committee on MOPP gear, and those issues, they were not in our data base, and we saw none of that. Now, we were focusing on the war, but, to the best of my knowledge, no, sir.

Mr. SHAYS. Dr. Rostker, it is my sense that you are being given an opportunity to look at that which is classified.

Mr. ROSTKER. Oh, absolutely.

Mr. SHAYS. And it is your testimony before this committee that you have not seen or are not aware of any Inspector General's reports discussing the integrity of the masks used by our soldiers.

Mr. ROSTKER. I am not, but I certainly will poll my staff and provide a clarification of that if I am in error, and that would include anything that we would have seen, either classified or unclassified.

Mr. SHAYS. Let me get into this issue of the GAO report that deals with the health and treatment of our soldiers. I get a sense that basically we are not able to properly diagnose and, therefore, effectively treat our soldiers because we do not really know yet what ails them as far as the VA is concerned and as far as the DOD is concerned. Is that correct? And nodding a head is not going to get in the transcript.

Mr. ROSTKER. Certainly, for the undiagnosed diseases.

Mr. SHAYS. But bottom line is there are tens of thousands of soldiers who have an undiagnosed disease or illness. Is that correct?

Dr. MURPHY. The treatment approach that we have taken is the approach that civilian doctors in VA and DOD doctors would take across the country, there are lots of nonveterans who have undiagnosed symptoms also——

Mr. SHAYS. Lots of what?

Dr. MURPHY. Unexplained symptoms.

Mr. SHAYS. Dr. Murphy, I am going to interrupt you a second, and then I am going to let you answer the question. But I just want to make sure, in the course of you answering the question, I forget what my question was, and my question was, I thought, fairly simple, that is basically is it true that we have—well, I will say it differently now because I forgot how I asked it, but it is my sense that we have tens of thousands of soldiers who have illnesses who the VA and the DOD, in the case of those who are active servicemen, who have no diagnosis. Is this correct? I just want to know the answer to that.

Dr. MURPHY. Yes.

Mr. SHAYS. It is correct. OK. Now, Dr. Murphy, if you want to tell me there are people in the private sector as well who have undiagnosed illnesses, I concede that. Is that your point you want to make?

Dr. MURPHY. The point that I was trying to get to, sir, and I apologize for being so wordy, was that we often treat symptoms,

and we do have very effective treatments for many of the common symptoms of Gulf war veterans. I will admit that there are groups of symptoms, people who have Chronic Fatigue Syndrome and fibromyalgia, where some of our currently used therapies are not as effective as we would like them to be, and one of the approaches that we need to take is to improve some of the therapeutic approaches.

Mr. SHAYS. Hasn't it been the testimony of the VA and maybe the DOD that it is very difficult to diagnose chemical exposure and difficult to——

Dr. MURPHY. Yes.

Mr. CHRISTOPHERSON. Yes, it is.

Mr. SHAYS. OK. So, I mean, we have a lot of soldiers, men and women, who feel that they are sick and are very frustrated that the VA is not treating them, though your testimony, Dr. Murphy, is you are attempting to treat whatever symptoms you see. And this really gets to areas that Representative Sanders was involved in his questioning of you.

I am trying to put myself in the mind of a veteran. It is 6 years after the war, and we are not into treatments, except maybe for some symptoms. We are still into raw, general kinds of research, and we are into research that may not come to fruition until 2002 and beyond. That would scare the hell out of me if I was a veteran, and so I want to get into the concept of how are you treating our soldiers. One way is to try to treat the symptoms.

Do you monitor the health of our veterans? A veteran comes in and they are sick and they have this level, you ask them to come in 6 months later and say, "We wanted to see if you are getting better or worse"?

Dr. GARTHWAITE. Sure. I think that individual physicians and individual care givers monitor the health of the individuals they are treating, and their followup examinations are based on what they think that is. Other than research studies, I know of no systematic approach to studying health outcomes of all patients on a continuous basis as a health care system, per se, other than the research studies. As indicated in my testimony at the beginning——

Mr. SHAYS. Can I interrupt you there? This may be basic to you, but it is not basic to me. If I am a doctor and I am trying to get at what their problem is, and to me it is still a big mystery and to them it is frightening as can be, why would it have to be a research project? Why couldn't it just be the VA saying, "Hey, we want to know how you are doing"?

Dr. GARTHWAITE. We do do that. Each provider does that. If I am your doctor and you come to me for your diabetes, say, and I see you, then I will write your prescription for insulin, educate you how——

Mr. SHAYS. But you have identified——

Dr. GARTHWAITE [continuing]. And you will come back to see me, and I will——

Mr. SHAYS. No, no, no. I am interrupting you only because there you identified an illness and a treatment. I am talking about the people that are ill but you cannot identify quite what the problem is, and I am interested to know, are you saying, well, are you getting sicker, or are you getting better?

Dr. GARTHWAITE. Or identify an illness or not.

Mr. SHAYS. Listen, do I make an assumption that the VA, if they cannot diagnose their problem, says they are not sick?

Dr. GARTHWAITE. I do not believe we do.

Mr. SHAYS. OK. So it is right for me to say, OK, you acknowledge they are sick. You do not quite know what it is. You might think it is, you know, something in their head, but they are sick, and it has had a manifestation on them.

What I am trying to get to is, though, I thought one of the points the GAO was making in their study was that you are really not monitoring the health of the veteran.

Dr. GARTHWAITE. But we cannot go to a computer data base and say, for all Persian Gulf veterans they had X amount of health, whatever the measure is, 4 years ago, and today they have Y health.

Mr. SHAYS. Bernie is a veteran that comes to see you. If he is not well, do you call him in 6 months later? You have told him you do not know what his problem is.

Dr. GARTHWAITE. Sure. Individually, yes, yes.

Mr. SHAYS. Do you have a protocol that does that?

Dr. GARTHWAITE. We—now 75 percent of all of our veterans are enrolled in primary care, which means they have an assigned doctor doing proactive——

Mr. SHAYS. Dr. Garthwaite, do you have a protocol that gets these veterans back in? I just want to know.

Dr. GARTHWAITE. No, no protocol.

Dr. MURPHY. We do not have a protocol, and the reason we do not have a protocol is that the therapy and the followup needs to be tailored to the individual veteran. Clearly, there are some people who need to be seen every couple of weeks or every month. Some might be seen every 3 months, some every 6 months, depending on the severity of their illness and how well they are responding to——

Mr. SHAYS. But the problem is you may not know how——

Dr. MURPHY [continuing]. The treatments they are being given.

Mr. SHAYS. I am sorry. The problem is you may not know how well they are 6 months later because you have not seen them, and they may say, "Why the heck am I going to go back to the VA? They tell me it is in my head, or they say they simply do not know."

What big incentive is there for them to go back unless you proactively—I mean, this is maybe a poor analogy, but when we do case work for someone, we are trying to institute a process where we do not have the answer for Mr. Brown—I am not talking about health—something that is bothering him—it might be the IRS or something else. We then try to just maybe call them up a little later and say, "How are you doing?" and in the process, they say, well, we got a letter from the IRS that we did not get, and we know things have gone along better, or we got three more letters from the IRS, and we say, "Why didn't you call us back?" They said, "Well, we did not know if you could really be helpful" or whatever. But there is not active, proactive protocol that——

Dr. GARTHWAITE. I think that is somewhat individual. We do have it now at every VA Medical Center a call-in line. We have as-

signed, like I say, 70-some percent of our patients, probably more than that because we are about to do another survey, but of our patients in primary care they have teams, they know who their providers are, and they know how they can get in touch with their principle physicians and other health-care providers.

So I think we do that on an individual basis. I think the GAO's criticism was: do we have it on a systematic basis and can we statistically show that to them.

Mr. SHAYS. Dr. Rostker, the GAO's recent report recommends that clinical progress of veterans should be monitored to promote better treatments and provide direction to research agenda. It also recommends that the diagnosis for stress and PTSD be refined. First, I will ask you, Dr. Garthwaite, do you agree with that recommendation?

Mr. ROSTKER. Let me ask my colleague from Health Affairs to respond.

Mr. SHAYS. OK. Why not start with you? I am sorry.

Dr. GARTHWAITE. I missed the last part of it.

Mr. SHAYS. Because I said to Dr. Rostker—I am sorry. I would like both of you to answer. The GAO's report recommends that clinical progress of veterans should be monitored to promote better treatment and provide direction to the research agenda. It also recommends that the diagnoses for stress and PTSD be refined. What do you think of that recommendation?

Mr. FEUSSNER. Yes. I would agree with that recommendation, and last fall the Cooperative Studies Program in VA funded a trial on trauma-related PTSD, a treatment trial involving approximately 350 veterans. Last fall, we funded another study, a multi-site study looking at seeing if we cannot come up with a computerized neurodiagnostic scheme—"protocol" is the word I want—that would allow these diagnostic methods to not only be made efficient, more straightforward, but also make them be useful in a computerized fashion.

Mr. SHAYS. OK. So the recent GAO report recommends that the VA and the DOD monitor the treatment outcomes of sick Gulf war veterans. Are you saying you are doing this, you intend to do this, or you do not know quite how to do it?

Mr. FEUSSNER. I am sorry. I thought your question was about PTSD.

Mr. SHAYS. Well, that was the second part of it.

Mr. FEUSSNER. We plan to monitor——

Mr. SHAYS. Let me just say this to you. If you do not have the resources, that is an issue. There are certain limits that you have.

Mr. FEUSSNER. Yes.

Mr. SHAYS. But in the end, I want to know the answer to the question.

Dr. GARTHWAITE. I would just say, as I have stated in my initial testimony, we believe that we should be monitoring health outcomes for all veterans. We believe all health-care systems should do it. We believe that insurance companies are asking all health-care systems to do it. We do not know of any health-care systems that do it in a systematic fashion. We have surveyed 32,000 veterans, using a form and a questionnaire that we think gets at health status. We plan to implement that——

Mr. SHAYS. Is this an unrealistic recommendation?

Dr. GARTHWAITE. No. We do not know that it is unrealistic or not. We believe that we need the information for all veterans, and we need it especially for Persian Gulf veterans, so we are going to pursue it, irregardless.

We will not get a 100-percent sample because of the large numbers we deal with, but we will get a significant sample, and we will aim to better understand what the functional status of veterans are over time. We have a goal of improving that over time as well, and we are holding our managers accountable.

Mr. SHAYS. The bottom line is that some veterans may be getting sicker, and we do not know it, and they may just choose to not come back to the VA.

Let me just ask you as well, Dr. Christopherson.

Mr. CHRISTOPHERSON. Let me do the latter point first, on the issue of the PTSD. That was a report which was requested and funded by us by the Institute of Medicine to look at our programs there. We do agree with it. We already indicated to ILM we agreed to it and that we are proceeding down that road to fix it.

The second thing, on the issue of the monitoring, it is difficult to do, as Dr. Garthwaite has indicated there. We are committed to trying out some processes. We have already gone out looking for some people to fund to, in quotes, help us to look at a monitoring process. Essentially what you would be looking at is to run some samples down through some particular kinds of—to look at, for example, a particular set of illnesses, for example, some of the undiagnosed or difficult-to-diagnose kind of categories, and see whether we see some progress in those kinds of areas.

It is difficult to do. I think no one should sort of preclude it is not, but we are committed to doing that.

Mr. SHAYS. OK. Let me just do one area, and then Mr. Sanders is going to come back, and that is the issue of depleted uranium. Dr. Rostker, are you aware of any studies that call into question or raise questions about the health consequences of depleted uranium?

Mr. ROSTKER. Let me refer to Dr. Daxon, who is really quite an expert on that.

Mr. SHAYS. OK. And if you get into anything classified——

Mr. ROSTKER. I understand.

Col. DAXON. Between the DOD and the DOE, we have been studying the health effects of uranium since we started the Manhattan Project in 1945. There is a wealth of information, both on inhalation toxicology and general toxicology of acute exposures to uranium.

Mr. SHAYS. So this is not new stuff here.

Col. DAXON. No.

Mr. SHAYS. If that is the case, how come we have not warned our soldiers about the negative consequences of depleted uranium?

Col. DAXON. Sir, I think the GAO report was accurate when it talked about what happened after the Persian Gulf war. We were relatively good about telling the people that actually touched the weapons or touched the tanks. That was relatively good. What we missed was in an actual combat situation, depleted uranium was going to be "touched" by a whole range of different soldiers. That

population, we missed, and that is the population we are trying to train now.

Mr. SHAYS. Well, let me just say this to you. I think you even missed the people who were handling the shells and so on, because they jumped into blown-out tanks and so on. So I guess I call into question whether you have even done that.

What do you have now to notify our soldiers? You have a video of some kind. Do you want to describe that?

Col. DAXON. Yes, sir. I cannot describe it. I am not the person that put it together or saw it, but it is basically a tier-1 training video that describes general procedures and precautions that are required that we are recommending currently for entering vehicles and dealing with vehicles that are contaminated with depleted uranium.

Mr. ROSTKER. We shared the video with your staff earlier, and if I might be so bold, it is a very informative video. I think it is very well put together, and I would encourage you to see it.

Mr. SHAYS. So the bottom line is, though, that is something we are doing now, but we did not do earlier.

Mr. ROSTKER. Yes. In fact, just now, and it needs to be promulgated through the field, and I will take the responsibility to make sure that those recommendations go not only to the Army, but to the Marine Corps and the other services.

The problem here, as I understand it, is not dealing with the shells as we showed them to you; they are quite safe. The issue is when they potentially can vaporize, and then the uranium dust, that dust——

Mr. SHAYS. Well, we know they vaporize. We know that 70 percent of it vaporizes.

Mr. ROSTKER. That is right. And the dust does not travel far because it is so heavy, but as you climb over the vehicles and the like, more precautions should be taken to a wider population than we appreciated.

Mr. SHAYS. That contrasts a little bit with Mr. Dietz, who suggests that it travels quite far, and his testimony was that this was a tremendous, high concentration. You were here for his testimony. Could you respond to it?

Col. DAXON. Yes, sir, I was.

Mr. SHAYS. And, sir, again, I just did not catch your name.

Col. DAXON. It is Col. Eric Daxon.

Mr. SHAYS. Thank you, Colonel.

Col. DAXON. Yes, sir.

Mr. SHAYS. OK. I guess the issue is it would not go for hundreds of miles, but will it last for hundreds of years, the concentration. But let me ask you to respond to Mr. Dietz's comments in particular, his testimony, how you reacted to his testimony?

Col. DAXON. Sir, the key thing with the toxicity of anything, to include radiation, is not only was it there, but how much was there. The Army has done a great deal of studying in determining how much of these aerosols are present at what distance from tank impacts, DU fires, and those sorts of things.

These studies started in the early 1970's. There were two National Materials Board studies that were done that are independent

of DOD. The first was done in the early 1970's that basically gave the green light to using depleted uranium.

Mr. SHAYS. Right.

Col. DAXON. Then there was a second—the DOD conducted a study, and then there was a second National Materials Science Board and two other studies. They all addressed the issue of aerosolization and how far the aerosols go when a tank is struck or when several tanks are struck.

Mr. SHAYS. Colonel, I want to say for the record, we may even decide that it can be quite dangerous, but still decide that we need it, because the alternative is worse.

Col. DAXON. Yes, sir.

Mr. SHAYS. If I am a soldier and I am in a tank, I want a shell that I know is going to do the job, but I just want to know the negative consequences. The more we have gotten into this, the more I have come to realize that if you are a soldier, you have shortened your life, even if you come back. No, I do not mean just on this; I mean on all the challenges that you face in warfare. I believe that in the serving of your country you also put yourself at tremendous health risk, some of it tremendously unavoidable.

But are those studies based on fragments or particles?

Col. DAXON. Sir, the early studies that were done were primarily looking at particulates, and there is a wealth of data on inhalation of uranium particulates. We have done it with the actual uranium-milling industry. There are a lot of studies that have been done on that.

Mr. SHAYS. Would that be available for us to give to others to look at?

Col. DAXON. Yes, sir. Absolutely. This is available in the open literature.

Mr. SHAYS. OK. Then let me just—is there any—because I do not want to get off this—were there any studies that suggested that the depleted uranium could be harmful to our troops?

Col. DAXON. Sir, the key thing is, for all of this stuff, both with radiation and chemical toxicity, the key thing is the amount, the chemical form, and where it came in.

Mr. SHAYS. OK. But I am going to ask my question, unless it is classified information.

Col. DAXON. No, sir. I will answer it directly. In the AEPI report that we put together there is a significant hazard for people that are inside a vehicle while the penetrator is being penetrated, while the tank is being penetrated by a DU penetrator.

Mr. SHAYS. Yes. Well, it is also going to blow up as well.

Col. DAXON. Yes, sir. Yes, sir. But in terms of the——

Mr. SHAYS. That is the least of their problems at that moment.

Col. DAXON. Yes, sir. But we have studied this because we wanted to be careful. In terms of the amount of uranium that would be inhaled, you can get milligram quantities if you are inside the vehicle while it is being penetrated by a DU penetrator.

Mr. SHAYS. Well, I thank you.

Mr. Sanders, thank you for your patience.

Mr. SANDERS. Thank you, Mr. Chairman.

Let me start off with Dr. Rostker, but anyone else can jump in. We had a conference in Vermont last month, and I met with a

number of veterans who are hurting, and if they asked me how they should conclude the performance of the VA and DOD after 6 years in terms of diagnosing the problem and treating the problem, Dr. Rostker, what would we say? And I am sure that you do not have all the money that you want, but you have got a few million bucks there. You have a lot of researchers. What would we say after 6 years? What is the grade that we give the VA and the DOD on this?

Mr. ROSTKER. I think that is a very difficult question to answer in one. To a veteran who has an unknown diagnosis, I can be quite certain, because I saw the same people on my 11-city swing, that they clearly are unhappy and angry and would give us a failing grade.

The question is, what can we do about improving that, particularly for the veteran that we truly do not have a diagnosis? I am reminded of one of the angry veterans on television who said that if he were in charge, he would lock up all the admirals and generals and would not let them out of the room until they gave the answer of why he is sick.

We do not have that answer. I am not sure we will ever get the answer for the individual, but we certainly are trying to understand what happened in the Gulf and to apply and push back the frontiers of science so we can, to the best of our ability, treat them.

Mr. SANDERS. Dr. Rostker, actually I was on a radio show today, and somebody was a little bit harsher. He suggested hanging, but not just putting them in jails.

Let me ask you this. Without for 1 second impugning the sincerity and the hard work and the patriotism and your desire, there is no reason that I can possibly believe that everybody up there in the entire DOD and VA want the answer to this question as much as Chris and I and everybody else on this committee. Right? We all do.

But sometimes we reach a conclusion that for whatever reason—maybe it is the system; maybe it is your bureaucracy; maybe that somebody can move, and it takes you a year to get out a grant—that is the system. We all have to work under systems. Is it possible that you guys are not going to be the agency to do it and that maybe we want to look outside of the VA and the DOD based on 6 years of not particularly effective work, without impugning anyone's sincerity? You know, businesses make these decisions every day, politicians.

Mr. ROSTKER. I think there are a lot of parts to the problem, and one would be the medical. Are we funding the right medical projects? You have raised some concerns. Remember that we put out RFPs, we go through standard practices, et cetera.

The same claims have been made, can we be trusted to assess what happened in the Gulf, and let me address that because that is really the primary concern of my office. We have an absolutely vital stake in that. Moreover, we have the expertise in that in ways that no other organization can possibly have. You cannot put an organization that starts and will have a clean slate on DU and then not have the kind of expertise that I have behind me, if you will.

I think, in terms of the investigations that went on in the Gulf, some of the same questions you asked today of the veterans, that

we are doing a job that is, I hope, credible but certainly expansive. We are not limited by funding. I am not limited largely by the bureaucracy within DOD, and we are truly leaving no stone unturned to try to understand what happened in the Gulf.

Now, that is only part of providing the problem, but it is an important part as seen by even your own methodology here in the way you are approaching the problem. You are asking questions about, as you did today, correctly so, about what the soldiers saw in the Gulf and what happened in the Gulf. Very important, and it is critical for the future; and so in that regard, I think DOD is the only organization that can do that portion of the research.

Mr. SANDERS. I would just suggest, I mean, clearly the function of the Department of Defense is to win wars, and, for example, most people observe and believe that in the Persian Gulf, the Department of Defense functioned very well. They achieved a major victory in a short period of time. Whether that same agency is designed to come up with a solution and treatment for a strange disease, I have my doubts about that. I think those are two separate things. Let me ask you——

Mr. ROSTKER. Congressman, may I respond?

Mr. SANDERS. Sure.

Mr. ROSTKER. A couple of things you have to keep in mind. In the first place is when this is all said and done, when the Gulf war and where maybe history down there, DOD, and I would argue, VA health side, have got to be able to answer the questions.

They have got to do two things. They have got to show they can take care of people. We have 6 million beneficiaries-plus, about 8 million eligibles we take care of every day through our whole system, families, retirees, active duty. This is not just a test around the Gulf war illness issue; this is a test of whether we can take care of people generally.

We have unique situations here with the Gulf war, especially—and, again, you have got to parse this out a little bit. If you are looking for are we doing a good job in dealing with the cancers and the heart and the other kinds of problems there—by the way, a lot of what the illnesses are, by the way, are in those categories. It is the illnesses that are hard to figure out that is the issue here.

I think what we have always said from day one is we have never claimed any exclusive club in terms of trying to find the answer. We believe we have been part of it. I think that is what it is. We have always been welcome to other parties, and that is why we pulled in ILM and a lot of other parties to help us figure some of these things out.

It is also why when we go out in the research side of life—in the early days, we did a lot of research intramural, I mean, inside the building, using our people, because we had to get something started, going fairly quickly. We have now turned on that. We have said, no, let's go outside. Let's poll people from the outside.

Mr. SANDERS. You lead me to my next question.

Mr. FEUSSNER. Before—may I answer your other question?

Mr. SANDERS. Yes.

Mr. FEUSSNER. I would just like to have three points to make. The first is that we have had inputs from the National Institutes of Health, the Institute of Medicine, the international research

community, Federal and non-Federal investigators, so we have asked and involved almost anyone on the planet that can inform the process.

The second issue is that we do have some additional expertise in patient-centered research. A large part of our research is patient centered, not exclusively laboratory based.

And then the third issue is the tradeoff between the time that might be lost by getting up to speed again or making a transition.

Mr. SANDERS. Let me just, actually taking off from both of your responses, you recently made grants—I believe there was a pool of some $8 million. Is my memory correct?

Mr. FEUSSNER. Correct.

Mr. SANDERS. When will that be made public? We have tried to find out who received the grants. I was curious. I did not have success.

Mr. FEUSSNER. What is happening right now, and there are a couple of sets of grants, by the way, that are in process, each on different points. One point is that in the final negotiations with the people who have won the grants to sort of work through the contracting procedures, and that is a close hold until that process is done, so that is coming out. That should be out, I think—Fran, you may know better than I when our research——

Dr. MURPHY. The AIBS has reviewed the proposals to review for scientific merit. They have been prioritized by the Persian Gulf Veterans Coordinating Board, and it is really now in the hands of the people who award the grants.

Mr. SANDERS. When will we know who received the grants?

Dr. MURPHY. Several months.

Mr. FEUSSNER. Yes. The last set is——

Mr. SANDERS. Several months, did you say, Doctor?

Mr. FEUSSNER. Right. Within the next 2 months. The reason is because it does take time to get through there. We have got to sort out—again, if we are going to do this right, 2 months it does take. We are committed to getting this money out this summer, and we will get it out this summer.

Mr. SANDERS. OK. Apparently you have notified some people that they have not received grants.

Mr. SHAYS. I am just going to interrupt the gentleman a second to say that he has as much time as he wants. I just need to say I was a little off on my time before, but I do not have more questions, and if you——

Mr. SANDERS. I will be finished in a few minutes.

Mr. SHAYS. OK. You have as much time as you want, but it will be helpful to——

Mr. SANDERS. So I am hearing that we will not know for sure who received the grants, Dr. Murphy, not until a couple of months. Is that what I am hearing?

Dr. MURPHY. Yes.

Mr. CHRISTOPHERSON. There are a couple of sets of grants coming through. That is why the people you may be referring to may be in one of the earlier grant phases as opposed to the current one we just talked about.

Mr. SANDERS. OK.

Mr. CHRISTOPHERSON. There are two grant sets, and the earlier set, those probably would be knowing by now that they had, but I am talking about the latest set where they would not yet know that.

Mr. SANDERS. OK. Would you be so kind as to send me, for both sets of grants, who the peer reviewers are? Is that public information?

Dr. MURPHY. The peer review is done by the American Institute of Biologic Science [AIBS] under a contract to the DOD, and they would hold those lists.

Mr. SANDERS. They would hold those lists?

Dr. MURPHY. Yes. DOD could request that information from them.

Mr. SANDERS. Come on, I should think that the U.S. Congress and the public has a right to know who reviewed the grants. Am I missing something here? That is very public knowledge. I would like—Mr. Chairman, I think this is an issue here of concern to me, because I want to make sure that the people who are reviewing these grants have an open mind with regard to multiple chemical sensitivity.

Mr. SHAYS. Sure, sure.

Mr. SANDERS. And if I am going to find that they are all hostile, then I think that we have a very bad process. I would like to know who they are.

Mr. CHRISTOPHERSON. My hesitancy was only because this is not—I have not been involved in that part of the process. I am just not sure. I want to make sure I do not give you an incorrect answer. We will get back to you very quickly.

Mr. SHAYS. Yes. If we could have it be part of the record, and you can get——

Mr. CHRISTOPHERSON. One way or the other, we will get back to you. If we can make it available, if there is not some reason, awfully good reason not to, we will get it back to you.

Mr. SANDERS. Yes.

Mr. CHRISTOPHERSON. The answer is yes, if at all possible.

Mr. SHAYS. When would you be getting back to us?

Mr. CHRISTOPHERSON. This is the issue of——

Mr. SHAYS. I said "when." When would you be getting——

Mr. CHRISTOPHERSON. I forget the answer to the question. I just do not know the answer.

Mr. SHAYS. I understand you do not have the answer.

Dr. MURPHY. This should not take a long period of time.

Mr. CHRISTOPHERSON. No, no.

Mr. SHAYS. What is that?

Dr. MURPHY. It should not take a long period of time.

Mr. SHAYS. So by next Wednesday you could get back to us?

Dr. MURPHY. Yes.

Mr. SANDERS. Good. Thank you very much.

Mr. CHRISTOPHERSON. That is reasonable.

[The information referred to follows:]

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WASHINGTON, DC 20301-1200

HEALTH AFFAIRS

Honorable Christopher Shays
Chairman, Subcommittee on Human Resources
House of Representatives
Washington, DC 20515

Dear Mr. Chairman:

Attached is the response to the request for information to be included in the hearing record submitted by Representative Bernard Sanders at the June 26, 1997, hearing "Status of Efforts to Identify Persian Gulf War Syndrome: Multiple Toxic Exposures."

Sincerely,

Gary A. Christopherson
Acting Principal Deputy Assistant Secretary

Attachment:
As stated

cc:
Honorable Bernard Sanders
Honorable Edolphus Towns

Representative Bernard Sanders (I-VT) submitted a request for the following information:

A list of the members of the Department's peer review organization.

Independent, external scientific peer review panel services are provided to the U.S. Army Medical Research and Materiel Command (MRMC) through a contract agreement with the American Institute of Biological Sciences (AIBS). Panel participants are nationally recognized scientists who have entered into a contractual agreement with AIBS, and whose credentials have been reviewed by MRMC to assure appropriateness, high level of credibility and absence of conflict of interest.

Immediately after a peer review is completed, MRMC destroys the list of participant names and only maintains the results of the review. As the MRMC record of names of panel members pertaining to your request was destroyed, we have requested that MRMC obtain a list of panel participants from AIBS. We will provide them to you when released and expect this to be in approximately two weeks.

Since the anonymity of the panel participants is intended to ensure objective, unbiased, critical reviews and to protect the privacy of the reviewers, we respectfully request that this information not be released outside your committee.

Mr. SANDERS. The next question is, Col. Roman, when he was testifying, mentioned a Dr. Baumzweiger at the L.A. VA Hospital, and he indicated that that gentleman was not asked—the physician was not asked to continue treating Gulf war veterans, and that physician had made a diagnosis that Col. Roman suffered nerve damage which may have occurred at the Persian Gulf. Does anybody know anything about that, or can you get us some information on that?

Dr. GARTHWAITE. We can give you more information. Dr. Baumzweiger was a neurology fellow who was working under the supervision of a staff neurologist at a particular medical center within the UCLA program, so there is some confusion in, I think, the patients and so forth, but we can give you lots of detailed information if you would like.

Mr. SANDERS. Can you get that information to this committee?

Dr. GARTHWAITE. Sure.

[The information referred to follows:]

Insert for Page 252, Line #5884

SUBCOMMITTEE ON HUMAN RESOURCES
COMMITTEE ON GOVERNMENT REFORM AND OVERSIGHT
REQUEST FROM REPRESENTATIVE BERNARD SANDERS

QUESTION FOR THOMAS L. GARTHWAITE, M.D.
DEPUTY UNDER SECRETARY FOR HEALTH
DEPARTMENT OF VETERANS AFFAIRS

HEARING ON
"STATUS OF EFFORTS TO IDENTIFY PERSIAN GULF WAR SYNDROME: MULTIPLE TOXIC EXPOSURES"

JUNE 26, 1997

PLEASE PROVIDE WRITTEN RESPONSE(S) FOR INCLUSION IN THE HEARING RECORD:

"Detailed information regarding Dr. William Baumzweiger's tenure at a University of California - Los Angeles medical center, particularly his diagnosis and treatment of Gulf War veterans."

RESPONSE:

- Dr. Baumzweiger was in a training position (neuromuscular fellowship) at the Los Angeles OPC and had no VA Gulf War-related clinical responsibilities; he completed his fellowship as scheduled at the end of the recent academic year, 6/30/97, and subsequently left VA.
- Dr. Baumzweiger has not published his Gulf War theories in a peer-reviewed scientific journal nor does he have any funded research projects.
- The Persian Gulf Veterans Coordinating Board held a special meeting to hear a presentation from Dr. Baumzweiger on his work. The Board concluded that his work represents a case series rather than true research.
- The Coordinating Board encouraged Dr. Baumzweiger to submit his work for peer-reviewed publication and apply for competitive research funding. We continue to encourage him to take these actions.

R=97%

Mr. SANDERS. OK. Let me ask you a question while we are on nerve damage. There are at least two studies that have been done, one by Jamal Hanson, and others, I believe, done in Great Britain, and one by Dr. Haley which suggest that there is actual brain damage—not brain damage, nerve damage for certain Gulf war veterans.

Do you have a thought on that, and have you done any research which suggests that there is actual nerve damage, which then would take us out of the realm of the theoretical, and we would have some very concrete answers to some of the problems our vets are facing?

Dr. MURPHY. The tests that they used were standard, neurologic tests, and if you look at the results of both Dr. Haley's research and Dr. Jamal's research, the results were within the normal range for those individual patients, and only by grouping the results and doing a statistical analysis was there any abnormality found. In fact, very few patients were examined with those techniques by Dr. Haley's group. It was less than a half dozen, and there were total a total number of 14 patients in Dr. Jamal's study.

Individual veterans evaluated, using either the DOD or the VA protocol, have had similar tests, and a small number of them have shown abnormalities, but as a group, that is not a consistent finding.

Mr. SANDERS. Is this an area of research that you are exploring?

Dr. MURPHY. There are currently ongoing research studies looking at both nerve muscle and brain function that are being funded. The GAO report and also our annual report on research lists those for you.

Mr. SANDERS. But what I am hearing you saying is that based on the evidence that you have put together so far, you have not seen any abnormal numbers of people. Is that what I hear you, or did I not hear you say that?

Dr. MURPHY. There has been no consistently found objective abnormality on neurophysiologic testing. In small numbers of patients during clinical evaluations we have found evidence of abnormality.

Mr. SANDERS. I am not exactly sure what that means. You have found something, but you think it is not statistically relevant. Is that——

Dr. MURPHY. We have not been able to tie the abnormal results from our clinical tests to any specific exposure or to their Gulf service, and we do not believe that the research at this point conclusively shows that there are any objective, neurologic tests that are indicative of Gulf war illnesses.

Mr. SANDERS. OK. Mr. Chairman, my last question deals with pyridostigmine bromide. At, I believe, our last hearing, there was a gentleman—what was his name, the pharmacologist from Maryland? Dr. Tom Teidt, who is a pharmacologist from the University of Maryland? OK. Was at the University of Maryland, now lives in Florida, sat exactly where you are sitting now, Dr. Murphy, and gave us a very frightening description of what he believed to be the dangers associated with PB and its use in a hot climate where there is stress and so forth.

I do not know if you are familiar with his testimony. Is he off the wall, is he right, and what work are you doing on that issue?

Mr. CHRISTOPHERSON. The issue of PB and stress, and some of the issues that are raised around there are a concern of us as well. We are looking at that research. We are looking at other research as well that raises a question about whether stress, for example, can exacerbate and create additional problems there. Again, the data is not clear, but, again, it is worth looking at because, again, PB has been very important, obviously especially when you are dealing with exposure to nerve gas or potential exposure there.

What we are doing right now is—so the answer is, yes, we are concerned about it; yes, we are taking a look at it. So far, it is not anything that tells us that for sure we should stop doing it, but it says we need to think about it as part of the total equation.

Here is an important point, I think, when you look at the PB issue. It is true about PB and what we know or do not know yet today on it, and the issue comes down to the following, which is, in the first place, you do not want to do any kind of treatments, pretreatments, vaccinations you do not have to. PB is clearly in that category there.

When you look at PB and how we used it in the Gulf war or how we might use it in the future in terms of there—it will be a very tough test, by the way, should we ever use it again in the future there—it is going to come down to you had better make sure you know which nerve gas you think is going to occur there; and, second, you are going to weigh these "relative risks" between the two things, and it is going to be a very tough discussion the next time we face this issue as to am I more worried about the soman, what is in PB, or am I more worried about the relative risks in terms of that, and we do not yet know all the answers.

Mr. SANDERS. But my point was, and somebody correct me if I am wrong, I think he almost used the expression "poisoning our own," in other words—and I am not saying that he is right or not.

Mr. ROSTKER. What my colleague is saying, in plain terms, is that we are a learning organization and that we are not as sanguine about PB as being as benign as we thought it was 6 years ago.

We are very interested in the research of PB, whoever funds it. We are pulling together a reassessment. That is one of the things that my office is doing. There are very important doctrinal issues. Clearly, in a soman environment, PB has a unique capability. We have to think through the risks here and the warning, and we are gaining knowledge about PB that is not falling on deaf ears.

Mr. SANDERS. In other words, and he was very somber, and, frankly, very scary, and what I am hearing you say is you are not dismissing his statement.

Mr. ROSTKER. No.

Mr. CHRISTOPHERSON. Absolutely not.

Mr. ROSTKER. This is very serious. This is like the DU in terms of there is an advantage, but there is a cost.

Mr. CHRISTOPHERSON. Correct.

Mr. ROSTKER. This is a different kind. DU may be of a smaller magnitude, frankly——

Mr. CHRISTOPHERSON. Correct.

Mr. ROSTKER [continuing]. But this is clearly stuff we want to know a lot more about——

Mr. CHRISTOPHERSON. Correct.

Mr. ROSTKER [continuing]. And we want to make sure we know it now rather than face a decision in another Gulf.

Mr. SANDERS. Well, not only in another Gulf, but in understanding the problem that we have today. Is that correct?

Mr. ROSTKER. Absolutely.

Mr. CHRISTOPHERSON. Correct.

Mr. ROSTKER. Now, some of that is independent on the issue of treatment and diagnosis and the like, but we are very much trying to understand better than we had the issue of pyridostigmine bromide. And I might say we are bringing in data and experiences not only from our country, but from other countries that have done this, particularly the Brits in some earlier testing they did with chemical agents in people.

Mr. SANDERS. So what I am hearing you saying, and I do not want to put words in your mouth—you said it—is that you are very concerned and regard it as a very——

Mr. ROSTKER. We have not drawn a conclusion, but we are actively putting the pieces together to put us in a position to be better informed and draw some conclusions.

Mr. SANDERS. And some of the very serious concerns raised by others——

Mr. ROSTKER. Absolutely.

Mr. SANDERS [continuing]. Are thoughts that you are taking seriously.

Mr. ROSTKER. Absolutely.

Mr. CHRISTOPHERSON. Correct.

Mr. SANDERS. Did anyone else want to comment on PB?

Mr. FEUSSNER. Yes. I would like to echo that. I think one of the intriguing observations that the new research is producing is the effect that stress can have on presentation of——

Mr. ROSTKER [continuing]. The brain area.

Mr. FEUSSNER. Yes. And, again, when some folks think of stress, they think of psychological stress, but stress has neurotransmitter and neuroendocrine sequelae, and this is actually an example of how stress can perhaps create a problem that might not otherwise have occurred.

Mr. SANDERS. If my memory is correct, and somebody up here can correct me if I am wrong, I mean, it was almost like a macabre joke that PB, under stress, and God knows, everybody at war is in stress, and in heat can bring forth a negative reaction; and on top of that, if PB is administered after one is exposed to chemical warfare agents, it could be a very bad effect. Does that make——

Mr. CHRISTOPHERSON. But I think the key thing that has changed in this equation, which is what we have all been referring to here, is the issue of the blood-brain barrier and the question under stress you can cross there. That was a new piece of information. That is what has caused people to go back and take another look at this.

Now, we are still not sure what it means because while it says it can happen, it still does not tell you what the effects might be.

Mr. SANDERS. Right.

Mr. CHRISTOPHERSON. And we have got to figure that out because, again, it is not like the issue is, well, we will just stop using PB and that is the end of the question. You still have this relative-risk issue you have to sort of work through, and we are doing that.

Mr. SANDERS. OK. Mr. Chairman, thank you, and thank all of you.

Mr. SHAYS. I just have one last question, because I looked at my notes and realized that we had information that the Armed Forces Radiological Research Institute, AFRRI, conducted a study in fiscal year 1994 for about $1.7 million, and in fiscal year 1995 less than $1 million, on the hazards of DU. The results were that it was a threat to our troops. The research stopped in fiscal year 1995 and the results were not released.

Colonel, it is a matter of public record, and we would love you just to quash it or sustain it, one or the other. First off, is my information accurate about the study being conducted?

Col. DAXON. Yes, it is.

Mr. SHAYS. OK.

Col. DAXON. I actually put the study together.

Mr. SHAYS. Oh, good. Then you are the man to ask.

Col. DAXON. Yes, sir. I am no longer there, but the study is still ongoing. It is addressing all aspects, and we are focusing on the imbedded fragments because that is where we have some doubts still. The research is being published in the open literature. As we speak, the research is still being continued.

Mr. SHAYS. So there was no result of that it was a threat to our troops.

Col. DAXON. The research is not done yet, sir.

Mr. SHAYS. OK.

Col. DAXON. I hate to draw conclusions when the experiment is not finished.

Mr. SHAYS. There were no conclusions drawn that it was a threat to our troops.

Col. DAXON. At this point, no, sir, none that I am aware of.

Mr. SHAYS. Let me thank all of you because you have been tremendously patient. I guess I should give you the same privileges——

Col. DAXON. Sir, could I?

Mr. SHAYS. Yes, sir. Do you want to say something?

Col. DAXON. Yes, sir, I do. The position of AFRRI and the DOD is not that DU doesn't present a hazard. AFRRI at this point has not found any hazards that were not expected at this juncture. It is not our position that there are no hazards associated with DU.

Mr. SHAYS. No. I think we all agree there are hazards, but you did not come to a preliminary finding that it was a threat to our troops.

Col. DAXON. No, sir.

Mr. SHAYS. OK.

Col. DAXON. I can check that and get with the director of AFRRI to make sure that is still current.

Mr. SHAYS. I think it would be good to have you—in fact, we would like an answer one way or the other, not just no answer. We would like you to either confirm your statement, which is on the record, or disqualify it, and get back to us by Wednesday.

[The information referred to follows:]

OFFICE OF THE SECRETARY OF DEFENSE
1000 DEFENSE PENTAGON
WASHINGTON, DC 20301-1000

Special Assistant
for
Gulf War Illnesses

JUL 0 2 1997

Honorable Christopher Shays
Chairman, Subcommittee on Human Resources
Committee on Government Reform and Oversight
House of Representatives
Washington, D.C. 20515

Dear Mr. Chairman,

Thank you for the opportunity to appear before the Subcommittee on Human Resources on June 24, 1997. Enclosed is the information from the Armed Forces Radiological Research Institute you requested in your letter of June 27, 1997. I look forward to working with you and the Congress in the future.

Sincerely,

for

Bernard Rostker

Enclosure

FEDERAL RECYCLING PROGRAM PRINTED ON RECYCLED PAPER

ARMED FORCES RADIOBIOLOGY RESEARCH INSTITUTE
8901 WISCONSIN AVENUE
BETHESDA, MARYLAND 20889-5603

5023
30 June 1997

MEMORANDUM FOR HEAD, MEDICAL AND HEALTH BENEFIT COLLABORATION
FOR OFFFICE OF SPECIAL ASSISTANT GULF WAR ILLNESS

SUBJECT: Depleted Uranium (DU)

Based on guidance from CAPT Eric Kearsley and phone conversations with you today, I am pleased to provide the following information:

1) A time-line that indicates our research activities on depleted uranium since 1994;

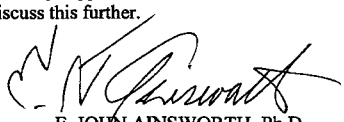
2) An overview of the research program and major accomplishments to date;

3) Selected documents including reports, published abstracts, a manuscript submitted for publication and other documents relevant to our communications of results to the scientific community and various components of DoD. We have also coordinated closely with the Department of Veterans Affairs.

There are a few points I want to highlight. The pilot toxicology studies on rats are proceeding on schedule, but we have not yet completed data collections and analysis. Our initial studies on oncogene activation in cultured cells and the ability of "transformed" cells to produce tumors in immunocompromised mice are completed. These research results have been presented at scientific meetings, a manuscript has been submitted for publication and these documents are enclosed. We are now conducting comparative studies *in vitro* with tantalum. Studies on intact animals will be necessary to estimate any excess cancer risk associated with DU implants; such studies have not yet been initiated. Data generated from future animal studies, together with results from cell culture or other experiments will be made available to appropriate advisory groups that will offer recommendations concerning any excess cancer risks in humans associated with implanted uranium.

Finally, my understanding is there may have been some misunderstanding about termination of ongoing DU studies. This may be based on the August 1995 Program Decision Memorandum #1 that directed closure of AFRRI and shut down of its reactor in FY 1997. This directive was never implemented and there has not been a shut down of DU or other research programs at AFRRI.

I will be happy to provide any supplemental information. Please telephone me at (301) 295-1210 should we need to discuss this further.



E. JOHN AINSWORTH, Ph.D
Scientific Director

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Depleted Uranium 10

Attachment 1

FACT SHEET: STATUS OF AFRRI DEPLETED URANIUM PROGRAM

- There are 15 confirmed Gulf War veterans with retained depleted uranium (DU) fragments. Many more DU-fragment casualties are expected in future conflicts due to the proliferation of DU-containing weapons systems. Current treatment strategies are based on experience with nonuranium, nonradioactive metal fragments, and small arms fire injuries.

- Following the United States experience with friendly-fire casualties in the Gulf War, it became clear that the information concerning depleted uranium (DU) fragment injuries was inadequate. Specifically, information to definitively answer the following questions was not available:

  1. Under what conditions should DU fragments be removed surgically?
  2. What are the long-term health effects of DU embedded in tissues?
  3. What can be done to remove DU by promoting excretion if it cannot be removed surgically?

- In 1994, AFRRI initiated a research program to address these questions.

- AFRRI research strategy:

  1. The behavioral, neural, renal and histiopathological toxicity is being evaluated at 1, 6, 12, and 18 months in rats with DU pellets embedded in the hind limbs.
  2. DU-induced genetic alterations and oncogenesis in human cells is being studied *in vitro*, as well as in the tissues from the rat experiments.
  3. The effect of DU on subsequent generations is being assessed in rats who reproduce after DU implantation.
  4. DU toxicity to the immune system is also being investigated in both the bone marrow of exposed animals as well as isolated immune system cells.

- Major findings:

  A. DU distributes in a time and dose dependent fashion to
     1) Bone
     2) Kidney
     3) Spleen
     4) Liver
     5) Brain

  B. Oncogenes are expressed in both *in vivo and in vitro* studies in a time and dose dependent fashion.

  C. Cells exposed to DU are transformed to tumorigenic cells in immune-compromised mice.

  D. Neuronal function in the hippocampus of the brain is depressed.

  E. DU distributes to the placenta and fetus of pregnant rats.

  F. No evidence of kidney damage is seen despite high levels of DU in the kidney and urine.

- The AFRRI research program is funded in part from grants from the US Army Medical Research and Materiel Command.

- A second research program to assess the possible carcinogenic affect of DU in animals is currently being performed at the Inhalation Toxicology Research Institute (ITRI) which is part of the Lovelace Laboratories.

    1. The project is designed to asses the risk carcinogenesis due to the radiation component of depleted uranium in a rodent model.
    2. The ITRI research program is funded by a grant from the US Army Medical Research and Materiel Command.

- AFRRI has worked with the Department of Veterans Affairs (DVA) in Baltimore MD on the patient followup program from the inception of the program.

    1. The patient followup protocol was written in 1993 by a panel represented by AFRRI, the DVA and the office of the Army Surgeon General.
    2. The DVA is periodically checking the patients injured by depleted uranium for urine uranium levels as well as any signs of adverse health affects related to uranium exposure.
    3. No evidence has been found to date of adverse health affects of DU in these patients.
    4. The DVA is sponsoring a research program at McMaster University that is developing whole body counting and X-ray tools to measure uranium fragments in patients.

- AFRRI has coordinated and hosted two research meetings to date, to review the research progress of AFRRI, ITRI, and the VA on the issue of depleted uranium health effects.

- In summary, the pilot toxicology studies on rats are proceeding on schedule, but AFRRI has not yet completed data collections and analysis. AFRRI's initial studies on oncogene activation in cultured cells and the ability of "transformed" cells to produce tumors in immunocompromised mice are complete. These research results have been presented at scientific meetings, a manuscript has been submitted for publication and these documents are enclosed. AFRRI is now conducting comparative studies *in vitro* with tantalum. Studies on intact animals will be necessary to estimate any excess cancer risk associated with DU implants; such studies have not yet been initiated. Data generated from future animal studies, together with results from cell culture or other experiments will be made available to appropriate advisory groups that will offer recommendations concerning any excess cancer risks in humans associated with implanted uranium.

Attachment 2

In addition to scenarios involving nuclear weapons, the United States must be prepared to deal with radiation dispersal weapons that consist of large amounts of radioactive materials combined with conventional explosives. These combined weapons could be used to heavily contaminate from large to vital areas, exposing large numbers of military or civilian personnel by complicating recovery operations and creating terror in military and civilian populations.

Scenarios involving nuclear weapons accidents, radiation dispersion weapons, damaged nuclear reactors, depleted uranium (DU) munitions, directed energy devices, as well as radiation sources from industrial waste sites, hospital sources and research facilities also must be considered. Preparedness for these nuclear scenarios support both operational requirements, as well as the national military strategy of peacetime engagement through nation assistance and humanitarian operations.

In the context of this strategic plan, the term nuclear encompasses not only traditional nuclear weapons, but also all radiation (ionizing and non-ionizing) hazards identified in this chapter.

CURRENT STATUS:

Military medical planning has historically focused on the treatment of a huge number of casualties expected in a massive nuclear exchange between the US and the former Soviet Union. In the post-Cold War era, current medical planning, doctrine, and training is inappropriate and inadequate to address today's threat. Joint doctrine is based on the nature of the current nuclear threat to include small yield tactical nuclear weapons and other radiation sources which may present, i.e., radiation dispersal, weapons, damaged nuclear reactors, depleted uranium, damaged or bombed nuclear waste sites, etc. Modeling of realistic nuclear scenarios, including scenarios of NW use in civilian, non-conflict areas, combined NW and CW/BW attack, and military operations in low-level radiation environments, should aid in the development of more appropriate doctrine, exercises and training.

CATEGORY: NCB

ACTION ITEM #: 48

SUBJECT: Emerging Radiation Environments

BACKGROUND: New military operational environments in the post-Cold War era demand a review of our medical policies, doctrine, equipment and research requirements. The military health services system must address both immediate and long term health effects associated with operations in nuclear, chemical, and biological (NCB) environments. Current military doctrine for operations in a nuclear environment only provides guidance for health care operations in peacetime and general nuclear war, but does not address military operations other than war (MOOTW). Neither peacetime standards nor general nuclear war procedures apply under MOOTW conditions.

With the involvement of US forces in MOOTW, the greatest radiation exposure risks are from radiation dispersion weapons, damaged nuclear reactors, depleted uranium (DU) munitions, and unknown radiation sources from industrial waste sites, hospital sources and research facilities. The most probable nuclear weapon scenarios are those involving the deployment of relatively low-yield nuclear devices targeted at specific military installations or sensitive political targets. OPLANS have not considered these risks or a combination of these with disease nonbattle injuries (DNBI).

Recent experience in Operation Desert Storm (ODS) and Bosnia highlight the inadequacies of existing doctrine. The application of peacetime standards and DoD infrastructure proved unexpectedly difficult during the Bosnia deployment. The presence of minefields and snipers increased the risk of what would ordinarily be "safe" radiation protection practices.

The draft Defense Medical Program Guidance, dated 14 FEB 96, outlines the requirement to "ensure a robust clinical capability to detect, assess and effectively manage injuries from combat or deployment related medical threats not normally encountered in peacetime health care." Neither peacetime health nor environmental standards are considered in the current framework for medical tactical or deployment operations. We do not have the

combination with multiple insults (e.g. infectious agents and injuries).

(1) Develop data required to make predictions of combined effects.

(2) Incorporate data into casualty prediction models.

b. Develop medical doctrine for internal exposures during military operations (from peacekeeping to high-intensity combat) that is consistent with current and proposed operational exposure guidance and current post-deployment hazard assessment and documentation policies.

(1) Write a policy that provides field criteria for performing assessments of internal doses of radiation.

(2) Identify the force structure (personnel, equipment) required to implement the doctrine for military operations.

(3) Develop guidance for operations in a contaminated environment that will allow commanders to effectively function while accepting risks that are consistent with operational exposure guidance.

c. Establish policy statement regarding the adequacy of operational radiation exposure guidance levels for women in operations other than war.

✔ d. Accomplish research, establish policy, and develop treatment protocols for injuries (immediate and long-term health effects) caused by depleted uranium.

(1) Assess immediate and long-term health effects (for men and women).

(2) Develop treatment protocols and policies for long-term health effects monitoring.

(3) Develop modified equipment sets to execute immediate treatment protocols.

(4) Procure and sustain equipment sets developed to execute immediate treatment protocols.

(5) Develop training programs for medical personnel on the treatment and risks associated with depleted uranium injuries.

* (Pass to R&D Panel) Develop policies that will ensure current and new weapon systems (friendly and opposing forces) are evaluated for development of medical assessment and treatment protocols for personnel wounded or injured by the weapon system. OASD(HA)(CS)

PRIMARY ACTION OFFICES:

a.(1) Tasked to: AFRRI, Army
a.(2) Tasked to: JCS
b.(1) Tasked to: Army
b.(2) Tasked to: Services
b.(3) Tasked to: AFRRI
c. Tasked to: AFRRI, OASD(HA)(CS)
d.(1) Tasked to: AFRRI
d.(2) Tasked to: Army Lead, AFRRI, OASD(HA)(CS)
d.(3) Tasked to: Army
d.(4) Tasked to: Services
d.(5) Tasked to: USUHS

Attachment 3

AFRRI DU Timeline

Year	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
92		Req. from Army SG for evaluation of embedded DU										
93	15Jan93 DoD ltr commenting on GAO draft of Army ability to deal with DRI		AFRRI Tech Reports: 93-1: Risk Assessment, 93-2: GW Vet Monitoring			16Jun93 AFRRI MEMO to ASD(AE) ltr & point paper on DU health effects assessment Passage of House bill H.R. 2481 that funds future DU studies	Initial proposal to DVA for DU toxicology study Formulation of protocol to develop DU animal model					20Dec93 AFRRI SD ltr and research protocol to Cdr,USAMRDC summarizing DU project proposal
94			14Mar94 AFRRI Dir ltr to USArmy requesting funds for DU study	27-29Apr94 NIH tech workshop statement on Persian Gulf exp. and health - Col Daxon, AFRRI, guest speaker Pilot study started to establish DU animal model (AFRRI appropriated funds); funded by U.S. Army PM Maintenance Arms System-Picatiny Arsenal		1Jun94 DU research proposal submitted to USAMRMC Summary Report to Congress		19Aug94 AFRRI DIR ltr to Dep Sec Army (ES&OH) offering update on DU Research Prog.	U.S. Army Environmental Policy Institute and PM-TMAS fund pilot study for fetal exposure to DU	AFRRI/DVA workshop to review pilot study data		U.S. Army awards grant for DU toxicology study U.S. Army awards two grants for DU fetal studies

Year	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
95	5Jan95 Request from USARMC - MCHO-CL-W, for guidance on DU mgm't					PA response to Mr Usdin, Nuclear Remedial Weekly; MFR, 8Jun95		27Aug95 ICRR annual meeting abstract presentation: Transformation of human cells by uranyl chloride		LTC Ryczak, USAMRMC, MCMR-PLC, Ft. Detrick inquiry on behalf of PG Veterans Health Research Project Fetal effects proposal submitted	Annual DU Report to USAMRMC on toxicology study	Request from USAMRMC, MCMR-PLC for copy of DU protocols for ASD(HA)
96	17Jan96 BG Todds's request for summary of DU research results for USAMC	29Feb96 AFRRI Assistance to VA on DU for their annual report to Congress		22Apr96 AACR 96 annual meeting; abstract presentation: Carcinogenesis Risk Assessment and Oncogenic Activation by DU	Presentation to Presidential Advisory Committee on Gulf War Veteran Illnesses - Dr. Pellmar		AFRRI Tech Report 96-3 Establishment of an animal model to eval. DU AIBS peer review of DU program		Coordination with DVA on upcoming DU workshop	1Oct96 Award of Dr. Benson research proposal: Eval. of health risks of DU on pregnancy and offspring development 18Oct96 Invitation to DASD(HA) to attend DU Workshop ISPO annual meeting abstract presentation: Oncogenes as biomarkers for low dose radiation	15Nov96 Du Workshop held at AFRRI; data at 6 month time-point presented for an 18 month ongoing study 18Nov96 Ltr of commendation from VA for DU Workshop Annual DU Report to USAMRMC on toxicology study	

Year	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
97		DU program presented to NEHC - Dr. Livengood Initiated pilot collaboration with DVA for measuring DU in body fluids from GW vets	31Mar97 Dr. Livengood's interview with Nation Magizine The Toxicologist annual meeting abstracts: 874 - Toxicity of Embedded DU Uranium 1308-Uranium levels on the Fetus and Placenta etc. Annual USUHS Research Day abstract presentation: Microdosimetry of alpha particles for *In-vitro* human cell transformation studies etc.	12Apr97 AACR 88th Mtg Publication: Vol 38 Mar97 Biomarkers for Carcinogenesis: Oncogenic Activation by DU	24Mar97 Nation Magizine Article on DU 29May97 Dr. Pellmar's interview with A&E film crew Maj Lucas, OSD request for Update on DU studies and publication list USUHS, PA SSgt Carpenter Request for DU experiments list and costs Presentation to Patrick Williams of The Office of Gulf War Illnesses North American Hypothermia Society annual meeting abstract presentation: P06-135 - Measurements of DU in cell partitions to etc. P22-457 - Transformation of human cells by DU etc.	13Jun97 Manuscript Submitted to Nature: Transformation of human osteoblast cells - Dr. Miller 19Jun97 MEMO and supporting documents to ODDR&E for review and analysis of DU program 30Jun97 Congressman Shays's DU Inquiry The Oxygen Club of Greater Washington, D.C. annual meeting abstract presentation: Transformation of human cells by DU etc.					Projected completion of data collection for first DU toxicology study	

Attachment 4

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workers was estimated to be 1.62 ng/kg/day. This exposure in combination with the NOEL of 50 µg/kg/day from the most sensitive species in toxicology studies, the CF-1 mouse, resulted in a Margin of Exposure (MOE) of 30,900. To correlate actual exposure with that predicted from dislodgeable residues, abamectin dislodgeable foliar residue (DFR) samples were collected and analyzed by HPLC. Abamectin foliar residues ranged from 40.7 to 0.17 ng/cm² at 2 hours and 7 days after the second application, respectively. DFR data at the time of harvest (0.84 ng/cm²) combined with the Zweig transfer factor (5000 cm²/hr) and a dermal penetration of 1% resulted in a worker exposure of only 5.6 ng/kg/day and a corresponding MOE of 8,930. MOEs based upon actual and estimated exposure values were within the same order of magnitude suggesting that DFR data may be used to conservatively predict worker exposure to abamectin.

873 ASSESSMENT OF RISKS AND CAUSES OF ADVERSE HEALTH EFFECTS ASSOCIATED WITH THE GULF WAR.

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As a result of the Iraqi aggression, the population of Kuwait was exposed to diverse causal agents resulting in adverse health effects and risks. The purpose of this study was to identify the causal factors resulting in increased risks and adverse health effects among a population of civilians affected by war. The causal factors evaluated were air pollution, mines and ordnance, stress, lack of sanitation, suspension of immunization programs, lack or denial of health care, poor diet, psychological effects of traumatic injuries and occupation, abandonment of veterinary screening and vector control. A disease etiology model was developed and verified using concordant disease incidence data from Kuwait since 1992. Cause and effect relationships were evaluated based upon temporal relationship of the observed association; consistency of association between exposure to the agent and disease; observable increase in disease incidence/prevalence associated with the agent; dose-response relationship where applicable; specificity of the association between the agent and the effect; biological plausibility; and logical coherence between the cause-and-effect interpretation and knowledge of the natural history of a disease. The results of the evaluation demonstrate projected increased risks resulting from traumatogens and chemical exposures and consistent and significant increases of mortality and morbidity in the exposed population. Loss valuation was also evaluated based upon literature sources for total treatment cost associated with each identified disorder. These costs can be used to project damages associated with invasion, occupation, and liberation. Increased adverse health risks, incidence and prevalence of disease, and health care costs are associated with invasion and liberation and continue for many years after conflict ends. Traumatogens and chemical exposures contribute to these effects.

874 TOXICITY OF EMBEDDED DEPLETED URANIUM (DU) IN THE RAT.

J Hogan, K A Benson, M R Landauer, T C Pellmar. *Radiation Pathophysiology and Toxicology Department, AFRRI, Bethesda, MD.* Sponsor: T J Flynn.

The use of DU munitions during Desert Storm resulted in a unique type of battlefield casualty, DU shrapnel. The toxicity associated with embedded DU may differ significantly from other metals or other routes of uranium administration. This study evaluates kidney, behavioral and neural toxicity associated with embedded DU pellets and assesses tissues for histological changes and for uranium content. Rats were assigned to 5 experimental groups: 1) control (20 1-mmx2-mm chemically inert tantalum (Ta) pellets), 2) high dose (20 1-mmx2-mm DU pellets), 3) medium dose (10 DU and 10 Ta pellets), 4) low dose (4 DU and 16 Ta pellets) and 5) nonsurgical controls. At 30 days following pellet implantation there were no significant changes in creatinine clearance or in urine levels of NAG, LDH, glucose, or protein. Locomotor activity, passive avoidance tests and sciatic nerve conduction velocity were not significantly altered by experimental treatments. Examination of the Ta pellets *in situ* revealed fibrous tissue adhering to the DU but not the Ta pellets, although capsule formation was not yet evident. Uranium levels were high and dose-dependent in kidney, bone, urine and muscle. At the high doses of DU, uranium unexpectedly distributed to brain and to spleen. Urine levels in the medium dose (10 DU pellet) group were approximately 30 µg/l, corresponding to the highest levels measured in the urine of Desert Storm veterans over one year after injury. While these results indicate that toxicity is not evident in the short term with exposure to embedded DU, the high levels accumulated by some body tissues raise concerns about the toxicity of long-term exposure.

This work is supported by funds from US Army Medical Research and Materiel Command.

875 A CASE STUDY IN BIOMATERIALS RISK ASSESSMENT: THE RELEASE OF 4,4'-METHYLENEDIANILINE (MDA) FROM CERTAIN POLYURETHANE MEDICAL DEVICES.

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The degradation of segmented poly(ether urethane), used in medical devices, may result in the release of MDA and exposure of patients to this rodent carcinogen. A quantitative risk assessment was performed for each of two device types (gastric feeding tubes and dialyzers using polyurethane potting glue) in order to assess the potential risks associated with their use. Upper-bound exposure received by patients using these feeding tubes was estimated to be 0.00197 µg MDA/kg body weight/day. Similarly, maximal exposure to MDA from dialyzers using polyurethane potting glue was estimated to be 0.061 µg MDA/kg body weight/day. The toxicity assessment conducted for MDA, due to its positive genotoxicity, was based primarily on rodent carcinogenicity studies. The linearized multistage model was used to provide a q_1 of 8.86×10^{-3} (µg/kg body weight/day)$^{-1}$ for MDA as a genotoxic carcinogen, with a resulting RsD of 0.113 µg MDA/kg body weight/day for a lifetime cancer risk of one-in-one hundred thousand. The overall conclusions of these assessments are that no measurable human health effects from cancer would be expected to occur from exposure to MDA through use of either the feeding tubes or dialyzers.

876 APPLICATION OF METHODOLOGY FOR HAZARDS ANALYSIS OF CHEMICAL MIXTURES.

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Default methodology for analysis of exposures to mixtures of materials accidentally released from DOE facilities has been approved. Additional methodology involving classification of chemicals by toxic consequence was developed for application to specific mixtures of chemicals at a waste facility. Results obtained for one mixture are presented. The objective is to compare exposure consequences with those obtained if chemicals are treated independently and all together. Toxicologic classification by health code number and effect (Patty[1] p 157) was expanded to include target organs. Concentrations and applicable limits are used to calculate hazard indices (HI = concentration/limit, e.g., TEEL-2[2]) for each chemical at each receptor point. The very conservative approach of summing HIs for all chemicals shows that conditions would be unacceptable (i.e., Σ HI $\geq$ 1.00) in three of four cases. Health code numbers can be used to sum HIs only for chemicals that have the same toxic consequence. Health code numbers are extracted from Patty (pp 155–185), or from the "Safety Profile" in SAX[3]. Results are shown for cumulative toxic effects, carcinogenic effects, reproductive effects, narcosis, and respiratory irritation. HIs for irritants were adjusted, depending upon whether irritation was listed as being severe, moderate, or mild. Most unacceptable conditions involve the Σ HIs at 30 meters when TEEL-2 is taken as the applicable limit. The hazard indices are summed for those chemicals having the same toxic consequences.

1 Patty's Industrial Hygiene and Toxicology, Volume 3A, p. 157.
2 Temporary Emergency Exposure Limit, used until Emergency Response Planning Guidelines (ERPGs) are available.
3 SAX's Dangerous Properties of Industrial Materials, 9th Edition, 1996, R. Lewis, Ed.

877 RISK ASSESSMENT FOR AFLATOXIN IN CORN AND PEANUTS IN THE UNITED STATES.

S H Henry, M J Dinovi, J C Bowers, and P M Boiger. *Center for Food Safety and Applied Nutrition, U.S. Food and Drug Administration, Washington, DC.*

Aflatoxins (AF) are a family of metabolites produced by the molds *Aspergillus parasiticus* and *A. flavus*, which infect corn and peanuts, usually associated with severe weather conditions. AFB1 is a potent liver carcinogen in some

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tract, head and body. In contrast to the dam, all fetal tissues examined had equivalent concentrations of 5, 13 or 36 ppg following treatment with 0.05, 0.20 or 1.0 ng/kg, respectively. These low tissue concentrations result from treatments previously shown to produce adverse effects during development. (This abstract does not necessarily represent EPA policy. CHH supported by NIEHS ES07126).

1308 URANIUM LEVELS IN THE FETUS AND PLACENTA OF FEMALE RATS IMPLANTED WITH DEPLETED URANIUM PELLETS PRIOR TO BREEDING.

K A Benson and S A McBride. *Armed Forces Radiobiology Research Institute, Bethesda, MD.* Sponsor: T J Flynn.

Metals such as nickel, lead and uranium have been examined for their toxicological and teratological potential. Acute uranium exposure before or during gestation, via injection or drinking water, has shown teratological effects on the offspring. Constant uranium exposure from implanted uranium metal has not been examined for either toxicological or teratological effects. This research is imperative, due to the increased use of DU munitions by the military, and the potential for shrapnel injury. The initial step in this project was to determine if maternal DU exposure, via surgically implanted pellets, will lead to uranium exposure to the offspring during gestation. Subjects were female Sprague-Dawley (SD) rats, implanted with one of five doses of DU, then bred with male SD rats. On gestational day 20, the dams were euthanized and uranium levels in the placenta, whole fetus, fetal liver and maternal kidney determined. A correlation trend test revealed an increasing trend of uranium levels in maternal kidney, placental tissue, and whole fetus tissue with increasing levels of maternal DU implantation. This effect was not seen in the fetal liver tissues. Measurements of maternal gestational weight gain and food and water intake indicated no adverse maternal effects. Litter parameters such as size of the litter, weight of the pups and male:female proportions were also not affected by the dose of DU. Histology of the maternal kidney indicated no histological damage. Further studies are under way to determine if the uranium exposure during gestation will impact on the neurobehavioral development of the offspring.

1309 ISOTRETINOIN DOSIMETRY IN HAMSTER AND RABBIT.

C Eckhoff[1], A Dubois[1], J Schindler-Horvat[2] and C C Willhite[2]. [1]*Hoffmann-La Roche, Nutley, NJ; and* [2]*SRI International, Menlo Park, CA.*

Isotretinoin (13-c-RA) is teratogenic in all species examined, but identification of the threshold is complicated because its all-*trans* isomer is a normal component of many tissues. Based on the lowest teratogenic oral dose, rabbits are more sensitive than hamsters, but drawing conclusions from the administered dose alone ignores differences in delivered dose to embryo. Distribution and metabolism of 13-c-RA at the NOAEL and LOAEL in rabbit (3 and 15 mg/kg-day) and hamster (7.5 and 37.5 mg/kg-day) found 13-c-4-oxoRA to be the major metabolite. At the NOAEL, Cmax for 13-c-RA, 13-c-4-oxoRA, all-t-RA and all-t-4-oxoRA in hamster plasma was 6 times that in rabbit; at the LOAEL hamster total Cmax was 4 times that in the rabbit. Hamster total absorbed and metabolized dose (plasma AUC) at the NOAEL and LOAEL was 2.3 and 2.6 times that in rabbit. In the embryo, hamster total Cmax was 2.3–2.6 times that in rabbit. Total embryonic delivered dose (total acidic retinoid AUC) at the NOAEL and LOAEL was very similar in both species (1.95 and 5.23 and 2.14 and 5.54 ng.hr.g-1). Embryonic AUC for all-t-RA and all-t-4-oxoRA, metabolites which directly transactivate the retinoid nuclear receptors (RAR), were essentially identical at the LOAEL (1.14 and 1.32 ng.hr.g-1). Therefore, based on embryonic delivered dose, 13-c-RA is an equipotent teratogen in hamster and rabbit.

1310 RESULTS OF A DEVELOPMENTAL SCREEN FOR EFFECTS ON EMBRYO-FETAL DEVELOPMENT IN RABBITS OF CONGENERS OF THALIDOMIDE.

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Thalidomide is a substituted phthalimide which was originally developed for use as a sedative but withdrawn from the market because of its association with fetal abnormalities. Such abnormalities most typically include limb size reduction, polydactyly, ear defects, esophageal or duodenal atresia, Teratology of Fallot and renal agenesis. However, a renewed interest has burgeoned in Thalidomide due to associated immunomodulatory activity. In this study, five different congeners of Thalidomide were evaluated for their effects on embryo-fetal development in rabbits. No abnormalities or clinical signs of toxicity were observed during the course of the study. No dams died, aborted or delivered prematurely. Gravidity for all groups ranged from 72–[illegible]. The results of this study indicate that none of the five congeners had any effect on embryo-fetal development when administered at 250 mg/kg on days 8–10 of gestation.

1311 REPRODUCTIVE AND DEVELOPMENTAL TOXICITY EVALUATION OF LIGHT CATALYTIC CRACKED NAPHTHA DISTILLATE IN RATS.

O Bui[1], R Breslin[1], D Burnett[1], F Koschier[1], H Lapedaite[1], P Podrebarac[1], C Schreiner[1], R White[1], and R Schroeder[2]. [1]*Petroleum Product Stewardship Council, Wash. DC;* [2]*Huntingdon Life Sciences, NJ.*

In an on-going effort to generate toxicity data for various petroleum refinery streams, a modified OECD Guideline 421 reproductive-developmental toxicity study was conducted with light catalytic cracked naphtha distillate (LCCND) in Sprague Dawley rats. LCCND was administered via whole body inhalation as a vapor, 6 hours/day, 7 days/week at target concentrations of 0, 750, 2500, or 7500 ppm. Exposure began 2 weeks prior to mating and continued through gestational day 19. Females were allowed to deliver and nurse their litters until postnatal day 4. Males were treated for 8 consecutive weeks. Systemic toxicity was demonstrated at the 7500 ppm dose level as evidenced by increased kidney weights in parental males and increased spleen weights in parental females. However, inhalation exposure to LCCND had no effect on reproductive performance or fertility up to and including the 7500 ppm dose level. Evaluation of the reproductive organs (ovaries, testes, and epididymides) did not reveal any weight or microscopic changes attributable to LCCND. Neonatal growth and survival to postnatal day 4 was not affected by treatment. No gross anomalies were observed in pups. Under the conditions of this experiment, the no-observed-adverse-effect level (NOAEL) for parental toxicity was 2500 ppm and the NOAEL for reproductive performance and fertility was 7500 ppm. (The Petroleum Product Stewardship Council consists of Amoco, ARCO, BP Oil, Chevron, Mobil, Texaco, and Unocal 76).

1312 LACK OF SELECTIVE DEVELOPMENTAL TOXICITY IN RATS TREATED WITH CEKANOIC C8 ACID.

L H Keller, A L Nikiforov, G W Trimmer, and S B Harris[1]. *Exxon Biomedical Sciences, Inc., East Millstone, NJ; and* [1]*Stephen B. Harris Group, San Diego, CA.*

Cekanoic C8 acid (CAS #25103-52-0) is a branched C8 acid used as a chemical intermediate. This testing was conducted for classification and labeling requirements under the EU 7th Amendment to the Dangerous Substances Directive. Cekanoic C8 acid was administered by oral gavage to 4 groups of CRL:CDBR mated female rats at doses of 0, 200, 400, and 800 mg/kg/day in corn oil on gestation days (GD) 6–15. Clinical observations were made daily during gestation, and body weights and food consumption were measured on GD 0, 6, 9, 12, 15, 18, and 21. On GD 21, cesarean sections and gross necropsies were performed, uterine weights with ovaries measured, uterine contents examined, and uterine implantation data recorded. All live fetuses were weighed, sexed, and examined for gross malformations. Signs of maternal toxicity were seen at 800 mg/kg/day, indicated by significant reductions in body weight (23%) and food consumption (10%) during the treatment period. No malformations or fetal toxicity were seen. Some statistically significant increases in the incidence of common skeletal variations in the treated groups were seen when compared with controls. However, these differences were either within the historical control range of this laboratory or were regarded as alternative normal patterns of development. Therefore, these differences were not considered biologically significant. Under conditions of this study, the maternal no observed adverse effect level (NOAEL) for Cekanoic C8 acid was 400 mg/kg/day. The developmental NOAEL was established at 800 mg/kg/day. This study demonstrates that Cekanoic C8 acid is neither a selective developmental toxicant, embryotoxic nor teratogenic.

Attachment 5

Poster # 1308

URANIUM LEVELS IN THE FETUS AND PLACENTA OF FEMALE RATS IMPLANTED WITH DEPLETED URANIUM PELLETS PRIOR TO BREEDING. K A Benson and S A McBride. Armed Forces Radiobiology Research Institute, Bethesda, MD. Sponsor: T J Flynn

Metals such as nickel, lead and uranium have been examined for their toxicological and teratological potential. Acute uranium exposure before or during gestation, via injection or drinking water, has shown teratological effects on the offspring. Constant uranium exposure from implanted uranium metal has not been examined for either toxicological or teratological effects. This research is imperative, due to the increased use of DU munitions by the military, and the potential for shrapnel injury. The initial step in this project was to determine if maternal DU exposure, via surgically implanted pellets, will lead to uranium exposure to the offspring during gestation. Subjects were female Sprague-Dawley(SD) rats, implanted with one of five doses of DU, then bred with male SD rats. On gestational day 20, the dams were euthanized and uranium levels in the placenta, whole fetus, fetal liver and maternal kidney determined. A correlation trend test revealed an increasing trend of uranium levels in maternal kidney, placental tissue, and whole fetus tissue with increasing levels of maternal DU implantation. This effect was not seen in the fetal liver tissues. Measurements of maternal gestational weight gain and food and water intake indicated no adverse maternal effects. Litter parameters such as size of the litter, weight of the pups and male:female proportions were also not affected by the dose of DU. Histology of the maternal kidney indicated no histological damage. Further studies are under way to determine if the uranium exposure during gestation will impact on the neurobehavioral development of the offspring.

INTRODUCTION

In utero exposure to uranium has recently been shown to produce both fetal and developmental toxicity. For example, administration (s.c.) of uranium in the form of uranyl acetate dihydrate (0.5-2.0 mg/kg/d) to gravid (pregnant) mice from gestational days (GD) 6-15 leads to significant decreases in both maternal weight gain and fetal body weights at GD 18[2]. Soft tissue and skeletal examination of the fetuses also revealed a significant increase in the occurrence of renal hypoplasia in all uranium-treated groups. Skeletal anomalies in these mice included bipartite sternebrae, dorsal hyperkyphosis, and incomplete ossification of several bones. Similar skeletal malformations were also seen following daily oral administration of uranyl acetate dihydrate (5-50 mg/kg/d) in gravid mice during the same period of gestation[4].

While the above results examined the effects of uranium on prenatal development, several studies have been conducted to evaluate the effects of uranium on postnatal development (from birth to age 21 days)[3,6]. Significant decreases in body weight and body length in the offspring of mice treated with 25 mg/kg/d for 14 days prior to mating have been reported[6]. There were also significantly more dead young per litter at this uranium dose at both birth and day 4. Uranyl acetate given orally to gravid mice from GD 13 to 21 days following parturition led to a significant increase in offspring liver weights in all the uranium treated groups (5.0-50.0 mg/kg/d), and decreased mean litter size on day 21 in the highest dose group (50 mg/kg/d). However, developmental parameters such as pinna detachment, incisor eruption and eye opening were unaffected[3].

Unfortunately, uranium levels in the dam, fetus, or placentae were not measured in any of these fetal and developmental toxicity studies. In order to determine the effects of embedded DU on a developing fetus, it is important to know the *in utero* uranium exposure level, though little work has been done to

examine the cross-placental transfer of uranium[1-5]. While there are distinct anatomical differences between the rodent placenta and the human placenta, little correlation has been shown between the anatomic classification of the placenta and the transfer of xenobiotics between mother and fetus[6]. In rodents and primates, the placenta may act as a barrier, limiting or preventing many toxicological insults to the fetus. This does not appear to be the case with uranium. When ^{233}U was administered intravenously to pregnant rats, almost identical levels of uranium were found in the placenta and fetus[7], indicating little discrimination for uranium by the placenta. The soft tissue levels of uranium in 19- to 20-day-old fetuses were equal to or greater than the maternal liver concentrations. Immature bone also exhibited a greater deposition of uranium than did the adult bone[5].

While previous research has demonstrated that the placenta does not act as a barrier to prevent the transfer of uranium from the mother to the fetus[7], the degree of fetal exposure from maternal implanted DU is unknown. The current study was designed to address this question by determining the uranium levels in the placenta and the fetus. This study also determined if the DU pellets impact on the dams ability to become pregnant and carry her litter to term.

MATERIALS AND METHODS

Subjects. Fifty-four female Sprague-Dawley rats(Charles Rivers) weighing 250-300 g were used. Rats were maintained in an AAALAC-accredited facility in accordance with the *Guide for the Care and Use of Laboratory Animals* (NIH Publication No. 86-23). Upon arrival, rats were quarantined and screened for diseases. Except during urine collection, all animals were housed in plastic microisolator rat cages with hardwood chips as bedding. Commercial rodent chow and acidified water (pH 2.5, using concentrated HCl) were provided *ad libitum*. Rats were on a 12-hour light/dark cycle. An additional nine male Sprague-Dawley rats, housed under the same conditions, were used for breeding.

DU and Ta Pellets and Surgical Procedures for Pellet Implantation. DU pellets (1 mm diameter x 2 mm long) were obtained from the Oak Ridge National Laboratories, Oak Ridge, TN. Tantalum (Ta) pellets (1 mm diameter x 2 mm long) were obtained from Alfa Products, Ward Hill, MA and were the heavy metal control. Before the implantation surgery, the DU and Ta pellets were cleaned and sterilized. Anesthesia was induced with ketamine hydrochloride (80 mg/kg) in combination with xylazine hydrochloride (4 mg/kg) and given i.p. in a 0.5-ml bolus, using a 25-gauge needle. The surgical sites were then shaved and cleansed with betadine. Pellets were implanted in each biceps femoris muscle spaced approximately 15 mm apart on the lateral side of each thigh. Implantation was accomplished by placing the pellet in a 16 ga needle, putting a specially designed plunger inside that needle, pushing the needle into the rat muscle, then depressing the plunger. This forced the pellet out of the needle and into the rat muscle.

Dose. Five doses were used in this study: Non-surgery control (N=10), 12 tantalum pellets (12), 4DU pellets and 8 tantalum (11), 8 DU and 4 tantalum (11) and 12 DU pellets (10). At all times, any rat receiving pellets always had a total of 12 pellets implanted in order to keep the size of the implantations approximately equal in all surgery rats.

Prenatal Tissue Collection. Experimental females were housed with non-treated male rats with two females in each male's cage. Gestational Day (GD) 0 was determined by the presence of sperm in the vaginal washing. At this time the females were removed from the males' cages and housed individually.

From GD 0 until GD 20, pregnant rats were monitored daily for weight gain, food intake and water intake. The parameters were used as measures of maternal toxicity of the DU pellets. On GD 20, the dams were euthanized. Dams were immediately cesarean sectioned, and the uterine horns removed. Fetuses were dissected out, and all the placentae for that litter collected. The uterine horns were examined for any resorption sites. Litters were examined, and a record made of (1) total number of fetuses, (2) number of viable fetuses, (3) sex ratio, and (4) any overt signs of teratological effects. All offspring of the litter were analyzed for uranium levels. The placentae from all pups were collected and pooled for uranium analysis for each litter. One male and one female pup separated out and used for analysis of whole fetus. The rest of the litter were used for determining uranium tissue levels. Quickly the liver and kidneys were dissected out of these pups. These tissues were pooled for the entire litter, homogenized, and sent to Quanterra, Inc., Richland WA, for further analysis of uranium content.

RESULTS

Maternal and Litter Effects Tables 1 and 2 present the data on the effects of the DU levels on maternal and litter parameters. From these data, there appears to be no effect of the DU on maternal parameters such as: maternal food and water intake, weight gain during pregnancy, and time-to-pregnancy. Furthermore, the litter parameters such as: number of pups, number of males vs females, and fetal weight were also not affected by the various levels of DU. The DU pellets did not adversely affect the ability of these rats to breed, or for them to maintain the pregnancy until the day of euthanasia. All litters were examined for any overt signs of teratology, and none were noted.

Uranium Distribution Figures 1 and 2 show the placental and whole fetus uranium levels. Comparison of these results by a correlation trend test indicate that uranium accumulates in these tissues in an increasing fashion as the maternal DU dose increases.

Figure 3 shows that a dose response relationship is also evident in the uranium levels found in the dams kidneys. The kidney levels, however, did not achieve the level we had anticipated being necessary for reproducing the effects seen by previous researchers, that being a kidney level of a minimum of 0.7 ug/g. Our highest DU level only averaged approximately 0.5 ug/g U in the maternal kidney.

Figure 4 shows the fetal liver uranium levels. No effect of maternal treatment was seen on the uranium levels detected in the fetal liver tissue.

Figure 5 shows the serum uranium levels obtained in the maternal blood. While the measurable levels are low, the correlation trend test did indicate a trend for increasing uranium levels in the blood as the maternal DU dose increased.

CONCLUSIONS

From the results it would seem that there is a dose response effect on uranium levels in the placenta, whole fetus, maternal kidney and maternal serum. The kidney levels, however, did not achieve the level we had anticipated being necessary for reproducing the teratological effects seen by previous researchers, that being a kidney level of a minimum of 0.7 ug/g. Our highest DU level only averaged approximately 0.5

ug/g U in the maternal kidney. The uranium did not impact on the ability of the rat to breed or to carry the litter to term.

The results of this preliminary study have opened up more questions than were answered. The DU level achieved in the kidneys of our highest dose was not even at the minimum level that is known to be nephrotoxic. Future attempts will be made to achieve and possibly exceed this minimum of level of 0.7 ug/g. This may be done by increasing the DU dose via increased numbers of implanted pellets, or by allowing the pellets to remain longer before the rats are bred. It is possible that a longer time period is needed for the uranium levels to stabilize and our attempts to breed the rats soon after surgery in our preliminary study may have actually hindered our ability to achieve an equilibrium. While previous work in our laboratory indicated that the urine levels of uranium began to stabilize in the 7-14 day period within which we attempted to breed the female rats, we now feel that perhaps a 45 -60 day period is optimal, as this will allow the blood levels to reach a steady state. As it is via the blood that the fetus were exposed to the uranium, it is vital that the blood levels have stabilized prior to impregnation. This will also attempt to mimic a time frame of approximately 1 year in a female soldier potentially wounded with DU fragments.

While these data are preliminary, the fact that uranium was detected in the placenta and whole fetus tissues indicates the potential for developmental toxicity. Fetal exposure to uranium during critical prenatal development may adversely impact on the future behavioral and neurological development of the offspring. Currently, this laboratory is examining this possibility. We are also investigating the effects that the pregnancy state has on the toxicology, distribution and urinary handling of the uranium in the female rat.

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TABLE 1

Effects of Depleted Uranium on Maternal Parameters

Variable	No Surgery	0 DU	4 DU	8 DU	12 DU
# Dams Bred	16	16	13	17	14
Days to Pregnancy (± SEM)	3.9 (±1.76)	2.08 (±0.23)	3.36 (±1.11)	4.36 (±1.42)	4.9 (±1.97)
Mean Weight Gain (g)	133.79 (±8.13)	138.33 (±6.49)	143.26 (±4.69)	138.75 (±4.38)	145.22 (±6.89)
Mean Food Intake (g)	23.44 (±0.67)	24.51 (±0.68)	24.27 (±0.52)	23.67 (±0.79)	24.71 (±0.71)
Mean Water Intake (ml)	43.85 (±2.59)	44.45 (±2.60)	46.33 (±2.10)	48.10 (±1.91)	44.84 (±1.50)

TABLE 2

Effects of Depleted Uranium on Litter Data

Variable	No Surgery	0 DU	4 DU	8 DU	12 DU
Total # Fetuses	13.8 (±.79)	13.5 (±.78)	14.8 (±.54)	15.5 (±.53)	15.0 (±1.09)
# Males	6.6	6.3	8.5	8.7	7.5
# Females	7.2	7.2	6.3	6.8	6.5
# Non-Viable	0	1	1	2	0
Average Pup Weight	3.60 (±.20)	3.16 (±.09)	3.66 (±.27)	3.39 (±.09)	3.38 (±.08)

FIGURE 1

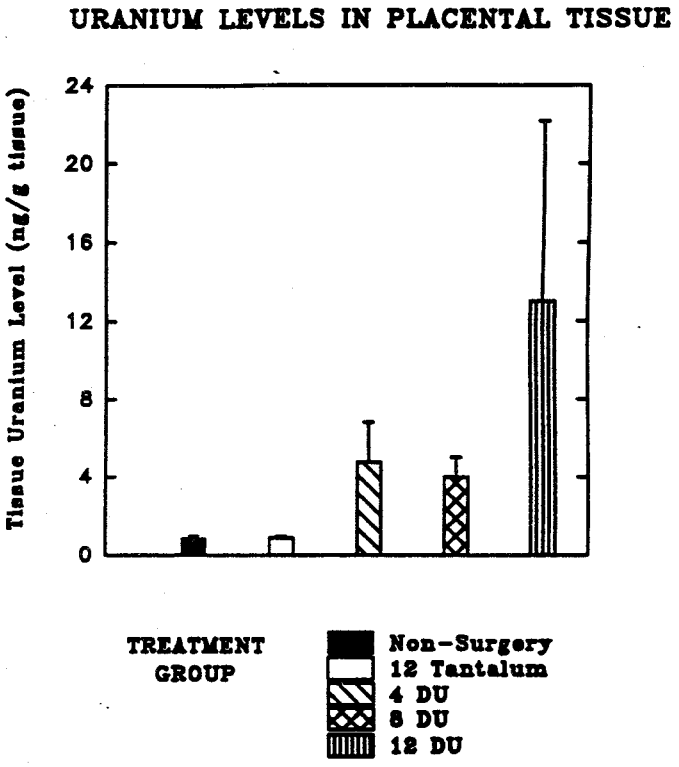

URANIUM LEVELS IN PLACENTAL TISSUE
Tissue Uranium Level (ng/g tissue)
24
20
16
12
8
4
0
TREATMENT GROUP
Non-Surgery
12 Tantalum
4 DU
8 DU
12 DU

FIGURE 2

URANIUM LEVELS IN WHOLE FETUS TISSUE

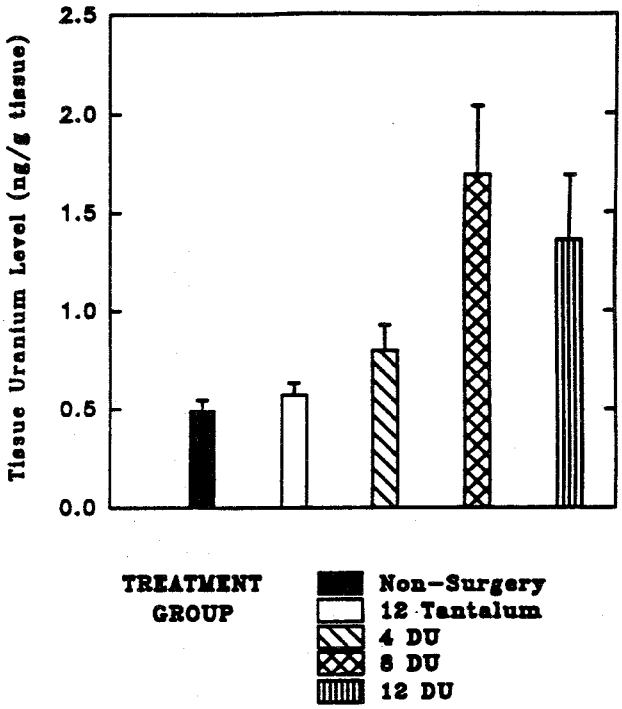


FIGURE 3

URANIUM LEVELS IN MATERNAL KIDNEY TISSUE

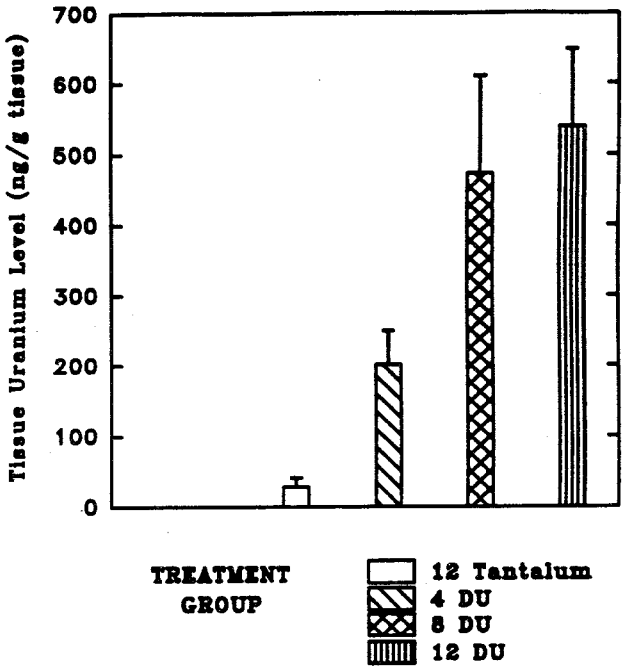


FIGURE 4

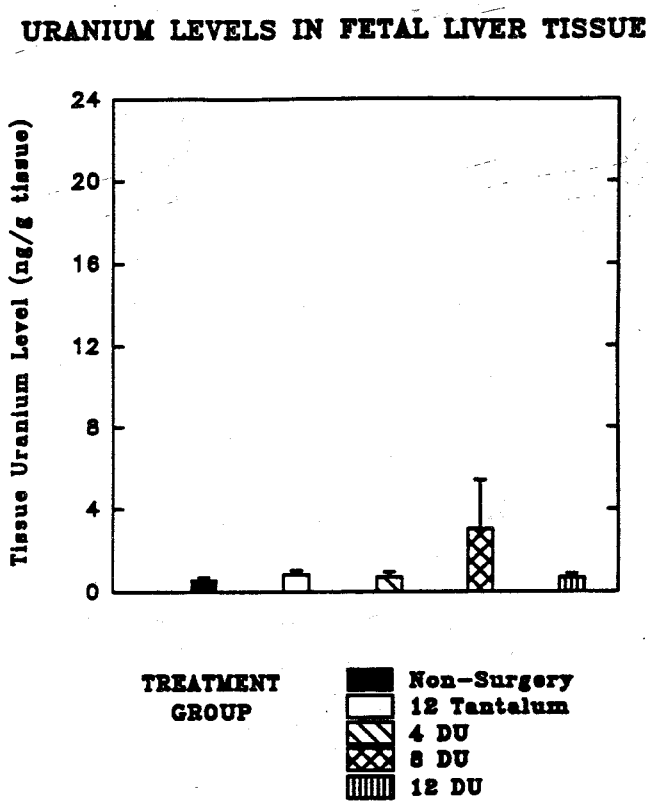
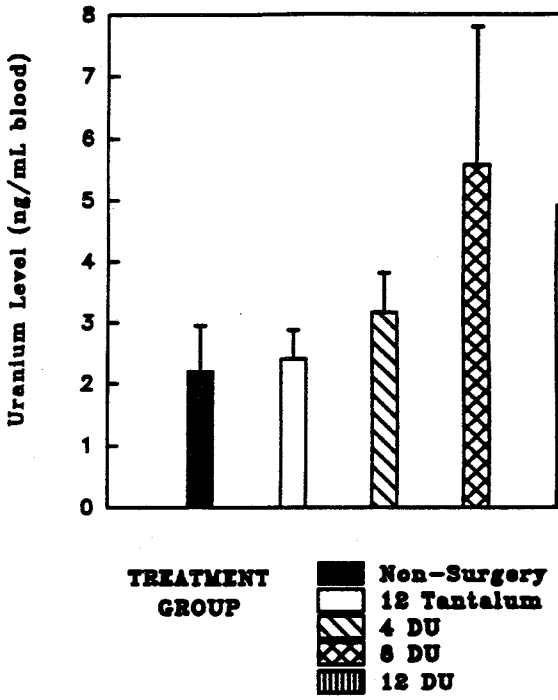

URANIUM LEVELS IN FETAL LIVER TISSUE
Tissue Uranium Level (ng/g tissue)
24
20
16
12
8
4
0
TREATMENT GROUP
Non-Surgery
12 Tantalum
4 DU
8 DU
12 DU

FIGURE 5

URANIUM LEVELS IN MATERNAL BLOOD



Attachment 6

AD_____

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13. ABSTRACT (Maximum 200 words)

This study assesses the health risks associated with embedded depleted uranium (DU) fragments by evaluating the behavioral, physiological and histological consequences of intramuscularly implanted DU pellets in a rodent model. In addition, distribution of uranium is determined and will be used to develop a biokinetic model. In the first year of this study, we established the appropriate doses (5 experimental groups) for subsequent analysis: 1) control (20 1-mmx2-mm chemically inert tantalum (Ta) pellets), 2) high dose (20 1-mmx2-mm DU pellets), 3) medium dose (10 DU and 10 Ta pellets), 4) low dose (4 DU and 16 Ta pellets) and 5) nonsurgical controls. We completed the study of the 30-day time point following pellet implantation, although analysis is still preliminary. Examination of the pellets *in situ* reveals fibrous tissue adhering to the DU but not the Ta pellets. Capsule formation is not yet evident. Uranium levels are high and dose-dependent in kidney, bone and urine and moderately high in muscle, brain (only at the high dose) and spleen. There is no evidence of renal toxicity or behavioral neurotoxicity at this time point. The 6, 12 and 18 month time points will be examined in future experiments.

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Terry Pellmar 11/21/95

PI - Signature Date

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HEALTH RISK ASSESSMENT OF EMBEDDED DEPLETED URANIUM: BEHAVIOR, PHYSIOLOGY, HISTOLOGY AND BIOKINETIC MODELLING

INTRODUCTION

Natural uranium consists of three isotopes: ^{238}U (99.276%), ^{235}U (0.718%) and ^{234}U (0.0056%). During the uranium enrichment process two products are produced, "enriched uranium" and "depleted uranium" (DU), that contain different relative ratios of these three isotopes. Enriched uranium contains the higher amount of the fissionable isotope ^{235}U and is used for nuclear reactor fuel and nuclear weapons. DU has a lower ^{235}U content and is a highly dense material. The DU used by the US in kinetic energy penetrators is alloyed with titanium (0.75% by weight) to retard oxidation. This DU alloy is of concern because the U.S. military currently uses this metal for munitions and armament. During Operation Desert Storm, a number of U.S. military personnel were wounded by shrapnel fragments consisting of DU[6,7]. Since surgical removal can produce excessive tissue damage, these DU fragments were treated as conventional shrapnel and left in place in the wounded soldiers. The radiographs of injured soldiers show multiple embedded fragments ranging in size from 1 mm to over 5 mm in diameter. Fragments as large as 20 mm have been noted in other patients. Uranium bioassays taken over a year after injury indicate that uranium was present in the urine well in excess of natural background, up to 30 μg U/l of urine. DU fragments present a radiologically and toxicologically unique situation with unknown health risks. Congress has mandated the study of these risks.

This study evaluates the consequences of both short-term and long-term exposure to DU fragments in the rat model. Using an interdisciplinary approach, we are assessing neurotoxicity, nephrotoxicity, histopathology of the tissue surrounding the fragment and pathology including evaluation of neoplastic changes in several body tissues. In addition, based on our animal data, we will develop a biokinetic model that describes the distribution of uranium from embedded fragments as a function of time.

Uranium toxicity: Although the toxicity of embedded DU is unknown, numerous studies have ad-

dressed the consequences of inhalation, ingestion and parenteral administration of other forms of uranium[27,38,45,62]. After uranium is absorbed, it circulates in the blood as the uranyl ion forming uranium-carbonate and uranium-albumin complexes[8,26,31]. As the uranium-carbonate complex passes through the kidney, it is filtered rapidly at the glomerulus where 60%-80% of absorbed uranium is excreted in the first 24 hours after acute exposure. The uranium not excreted is reabsorbed by the proximal tubules where it produces acute toxic effects. Uranium also enters the bone where it competes with calcium to form complexes with phosphate ions, thus becoming part of the bone matrix[3,10,16,42]. This bone matrix then serves as a storage site from which uranium is slowly released back into circulation [23,61]. The liver, muscle, and kidney are other major sites of uranium disposition, with a possible long-term storage mechanism in the kidney [19,23,27,51,62]. At low doses, uranium may not readily distribute to the central nervous system (CNS)[45]. With higher doses (8 mg/kg/day orally for 4 weeks), however, brain uranium levels are comparable to those in liver and in bone[45], major sites for uranium accumulation.

Acute morphological and biochemical changes of the kidney result from uranium exposure[8,26,31,42]. The glomerular epithelial architecture is altered[25] and cellular necrosis occurs in the proximal tubules near the corticomedullary junction in the kidney[2,17,18]. In addition, polyuria, enzymuria, glucosuria, and increased excretion of amino acids result[8,9,26,63]. Acute renal failure can be the cause of death with exposure to high doses of either soluble or insoluble forms of uranium[43,57]. Environmental stressors such as restricted diets or changes in housing conditions significantly enhance uranium toxicity[1,4].

Few studies have addressed the chronic toxicity of uranium and the results available are conflicting. Galibin and colleagues[14] reported severe renal toxicity in rats that inhaled the slightly soluble uranium compound, ammonium diuranate (1 or 8 mg/m^3) for 128 days. Urine protein and blood, non-protein nitrogen were elevated. In the proximal tubules, there were sloughed dead cells and abnormal regenerating cells. These animals recovered, although the total number of tubules was reduced, with an accompanying increased proportion of connective tissue in the kidney. In contrast, Leach et al.[29,30] found no renal toxicity in rats repeatedly exposed for a period of 12 months to uranium dioxide dust (5 mg/m^3) (or in dogs or monkeys exposed for 5 years). Yet uranium concentrations in the kidney were as high as 1.1 μg U/g kidney wet weight in the rat (8.3 in the dog and 17.0 in the monkey), levels

reported to cause acute renal toxicity (e.g., [23]). Thus the chronic effects of uranium exposure remain, for the most part, unresolved[8].

The threshold concentration of kidney uranium levels in man that results in kidney chemical toxicity is in dispute[8,26,52]. While the Nuclear Regulatory Commission has set the level at 3 μg/g kidney for renal damage in man, there is evidence from both human and animal reports that this level could be much lower. For example, chronically exposed uranium mill workers, whose kidney uranium levels probably did not exceed 1 μg U/g[54], showed mild renal dysfunction with increased urinary excretion of β_2-microglobulin and various amino acids. In rats exposed subchronically to low doses (cumulative dose: 0.66 or 1.32 mg/kg) of uranyl fluoride, kidney uranium levels as low as 0.7 to 1.4 μg U/g wet kidney produced cellular and tubular necrosis of the proximal tubule, proteinuria, and enzymuria[9]. These changes in rat renal function, however, were temporary, with complete recovery within 35 days after exposure. These studies are important because they indicate that renal injury can occur at kidney uranium levels well below the 3.0 μg U/g limit.

Neurological effects have been reported with uranium exposure. In uranium workers excreting up to 200 μg U/l in their urine, normal mental function was disrupted[24]. One case study linked the handling of a uranium bar and a subsequent increase in stool uranium with foot cramps, leg pain and abnormal gait[15]. With oral and subcutaneous administration of relatively high doses of uranyl acetate (210 mg/kg and 10 mg/kg, respectively), rats exhibited tremors[11]. The uranyl ion has been demonstrated to enhance muscle contraction with acute local concentrations of 200-400 μM[13,32]. At the neuromuscular junction in the mouse, multiple sites of action were identified, including increased duration of the muscle action potential, broadening of the compound nerve action potential, increased amplitude and quantal content of the endplate potential and increased frequency of the miniature endplate potentials[32]. These studies indicate that embedded DU fragments could lead to neural damage, affecting both motor and cognitive function. The CNS effects of uranium toxicity can result from secondary mechanisms since hormonal changes, electrolyte disruption and immune responses can all influence nervous system activity [47].

Local Tissue Response and Capsule Formation: Foreign bodies in tissue elicit an immune

response that can result in encapsulation. Even when encapsulated, DU fragments provide a local, chronic source of α-radiation. Within 10-15 cells of the fragment, the dose rate is expected to be approximately 8.5 Gy/yr. This radiation could result in injury or damage to local muscle or nerve tissue (axonal injury, demyelination)[48,58]. In addition, capsule formation around a DU fragment in close proximity to a nerve could increase the risk of compression injury to those nerves.

Encapsulation could limit the chemical toxicity of the DU fragments by decreasing the rate of release of the metal, as has been observed with lead[35]. Encapsulation can also result in the formation of pseudocysts. Pseudocysts were formed that contained fluid with very high concentrations of soluble lead and insoluble lead dioxide particles[33,35] and with "black pigment...firmly adherent..." to portions of the inner wall of the capsule[33]. If these cysts should rupture, the rapid release of this fluid could cause period spikes in circulating lead levels and result in acute lead toxicity 5 to 40 years after the initial injury[33,35,59]. Similar type lesions may form around DU fragments. Intracapsular fluid may contain high concentrations of both soluble and insoluble DU. Tonry[55] demonstrated that DU disks formed both a soluble fraction and black insoluble particulates when emersed in simulated lung fluid. After a large fragment (approx. 20 mm) was removed from a U.S. soldier 17 months after he was wounded, the surgeon[28] noted that the fragment was encased in a fibrous capsule. When the capsule was breached, approximately 1-2 ml of a black fluid "gushed forth" from the cystic space.

DU can cause both local and systemic toxicity through a variety of mechanisms. Our study defines many of the potential sites of pathology that can result from long-term exposure to DU fragments and will provide a rationale for treatment of our wounded soldiers. The first six months of the study established the doses of DU to be used in future experiments (aim 1). This dose ranging study determined the number of DU pellets required to obtain uranium levels in the range of 0.7 to 1.4 μg/g wet weight of kidney. This level of uranium has been reported to produce early signs of renal damage as measured by both biochemical and histopathological changes[9] and would define the high dose in our toxicological studies. The low dose was chosen to produce no measurable acute toxicity. Subsequent experiments use the established doses to evaluate neurotoxicity, nephrotoxicity and histopathology and determine uranium distribution for biokinetic modelling.

Neurotoxicity is assessed by (a) a battery of behavioral tests to assess functional consequences and (b) conduction velocity studies in motor nerves to uncover any peripheral neuropathies. Behavioral tests have frequently been employed to detect and characterize potential neurotoxic effects in rodents and have been used extensively in animal toxicity studies[44]. The neurobehavioral battery consists of (i) a functional observational battery (FOB), which is a series of tests designed to assess the neuromuscular, autonomic, and sensory integrity of the rat[12,36,37,39,40], (ii) an automated test of locomotor activity and (iii) the passive avoidance test used to evaluate memory. Electrophysiological experiments monitor nerve conduction velocity and integrity of the neuromuscular response. Nerve conduction velocity studies have been used clinically for many years to diagnose peripheral neuropathies and can even detect subclinical neuropathy induced by lead exposure[20,41,49].

Markers of renal function in the urine and plasma are used to assess nephrotoxicity. Altered creatinine clearance and proteinuria can indicate glomerular damage although tubular changes can also contribute. Increased urine content of enzymes such as lactate dehydrogenase (LDH) and N-acetyl-β-glucosaminidase (NAG) have been interpreted to reflect tubular damage[46]. In addition, appearance of glucose in the urine, can indicate alterations in tubule reabsorption. These markers have demonstrated sensitivity with acute uranium nephrotoxicity[8,9,31,63] and should indicate any toxicity that might result from long-term exposure to DU fragments.

Capsule formation and the sporadic release of pseudocyst fluid-contents can significantly influence the time course and concentration of uranium distributed through the body. The encapsulation process and pseudocyst formation is characterized at the time of euthanasia (1, 6, 12, 18 months after implantation), surrounding tissues are histologically examined and any capsular fluid is analyzed for its uranium content. In addition, tissues that are known to accumulate soluble uranium or uranium particulates (liver, bone, kidney, spleen)[19,27,29,30,61,62] are histologically evaluated.

Although the distribution of uranium in the rat has been characterized for a variety of routes of internalization (inhalation, ingestion, and parenteral administration of soluble compounds), this information is not available for embedded fragments. We are measuring uranium in urine, plasma, kidney, bone (tibia and skull), liver, spleen, brain, and skeletal muscle that is proximal and distal from the

embedded pellets. Uranium is transported in plasma and urine and is stored in kidney and bone[19,27,61,62] Uranium has been detected in the liver and spleen of animals[19,28,30] as well as in human subjects[25]. The skeletal muscle is being sampled to determine the local concentrations of uranium. The brain was chosen because of the paucity of data and the need to assess whether any neurological effects observed were due to the direct or indirect interaction of uranium in the body. These data will allow a rat biokinetic model for implanted DU fragments to be developed.

METHODS

Approach: This report describes the data obtained in the first year of a three year study of 325 rats which will provide toxicity data for 3 DU doses (low, medium, high) at 4 time points (1, 6, 12, 18 months). Each rat is thoroughly evaluated for changes in behavior, peripheral nerve function, CNS excitability, renal function and tissue histology including capsule formation. In addition, data on tissue uranium levels from a subgroup of rats are used to develop a biokinetic model to predict uranium distribution.

Rats are randomly assigned to 5 treatment groups: 1) rats implanted with low-dose DU, 2) rats implanted with medium-dose DU, 3) rats implanted with high-dose DU, 4) rats implanted with tantalum (Ta) to control for fragment implantation, and 5) a non-implanted sham-surgical control group. In the low-dose and medium-dose groups, Ta is substituted for a fraction of the DU pellets in order to keep the total number of implanted fragments constant. Half of the total number of pellets are implanted in each thigh.

Based on the variance of control data for neurological effects, a group size of 15 rats is necessary to see significant changes of 20% or greater at the $p \leq 0.05$ level. Additional animals (20 rather than 15) will be implanted for the 18 month time point with the expectation of approximately 25% natural mortality[64-66]. This will provide 15 animals for analysis of neurological and biochemical endpoints in all groups at all 4 time points. Five of the rats in each experimental group provide tissue for uranium quantification. At the time of euthanasia, tissues from 7 animals per group at each time point will be assessed for uranium content and the remainder will be evaluated for histopathology.

Two-way analysis of variance will be used to test statistical significance of any changes. Newman-Keuls test will be used for multiple comparisons. In all analyses, statistical significance is accepted at the $p<0.05$ level.

Since this is a progress report, the total number of animals reported here do not represent the number of animals that will be included in the final analysis. Because of the staggered experimental schedule required for these protocols, the number of subjects for each endpoint will not be identical at the time of this report.

Subjects: Sprague-Dawley rats (8-10 weeks of age) are maintained in an AAALAC-accredited facility in accordance with the *Guide for the Care and Use of Laboratory Animals* (NIH Publication No. 86-23). Upon arrival, rats are quarantined and screened for diseases. Except during urine collection, all animals are housed in plastic microisolator rat cages with hardwood chips as bedding. Commercial rodent chow and water are provided *ad libitum*. Rats are on a 12-hr light/dark cycle.

Fragments: DU fragments, consisting of 99.25% DU and 0.75% titanium by weight, were obtained from Oak Ridge National Laboratories, Oak Ridge, TN. The uranium isotopes present is ^{238}U (99.75%), ^{235}U (0.20%) and trace levels of ^{234}U. This is the same DU alloy used in U.S. military munitions. Tantalum (Ta) fragments were obtained from Alfa Products, Ward Hill, MA. Ta was chosen as the control substance because it is a biologically inert metal[22] with a similar mass to uranium and is frequently used in human prostheses[21,53]. Each fragment (both DU and Ta) is approximately 1 mm diameter x 2 mm long.

Surgery: The DU and Ta pellets are cleaned and chemically sterilized prior to implantation. The pellets are immersed in industrial detergent, rinsed in absolute alcohol, soaked in 50% nitric acid solution for 3 min and then rinsed with water. This procedure completely removes the oxide formation on the surface of the DU pellet[55]. Anesthesia is induced with ketamine hydrochloride (80 mg/kg) in combination with xylazine hydrochloride (4 mg/kg), given i.p.

Fragments are implanted within the gastrocnemius muscle spaced approximately 8-10 mm apart on the lateral side of each leg. The surgical sites are shaved and cleansed with betadine, a topical disinfectant, prior to surgery. Scalpel incisions are made through the skin and pellets are inserted into the

muscle with a trochar (16 gauge needle with plunger). Incisions are closed with absorbable sutures and surgical cement. Rats are closely monitored following surgery until they are ambulatory and an analgesic (Demerol, 10 mg/kg, i.m.) is administered if needed. A veterinarian regularly examines the surgery sites for signs of inflammation, infection and local DU toxicity.

Behavioral neurotoxicity: The functional observational battery (FOB) consists of behavioral evaluations (home-cage, handling and manipulative) and several physiological measures. The parameters to be recorded are listed below and grouped according to the following functional domains: 1) Autonomic: lacrimation, salivation, palpebral closure, piloerection, defecation, urination, 2) Sensorimotor reactivity: tail pinch response, tactile response, click response, approach response; 3) Neuromuscular: gait, foot splay, forelimb and hindlimb grip strength, righting reflex, and 4) CNS Excitability: arousal, posture, ease of removal from cage, handling reactivity, convulsions, and locomotor activity.

The observer is blind as to the identity of each group. The behavioral battery commences with brief home cage observations during which time the observer describes the posture, and the existence of tremors or convulsions, and palpebral closure. The rats are then removed from their cage and rated for ease of removing and handling. While handling the rat, presence of piloerection and the degree of lacrimation and salivation are observed. The animals are then placed in an open-field with a perimeter barrier on clean absorbent white paper for 3 min. The number of rears, the gait, level of alertness, stereotypy (repetitive movements e.g., head weaving), unusual behaviors (e.g., writhing), and the number of fecal boli and urine pools are recorded.

Sensorimotor responses also are determined and include: approach response to a blunt probe, touch on the rump (tactile response), click response (auditory response), and pinch on the tail using forceps. Next, neuromuscular responses are determined and include: righting reflex, forelimb and hindlimb grip strength using digital strain gauges[37], and landing foot splay[12]. The animals are weighed and rectal temperature determined using a digital thermometer. The FOB is conducted during the light portion of the light-dark cycle. Details of the FOB tests can be found in Moser et al.[40] and McDaniel and Moser[36].

Approximately, 1 hr after the FOB, the rats are monitored for horizontal and vertical locomotor

behavior. Motor activity is recorded for 1 hr using automated photocell activity cages (Digiscan Analyzer, Omnitech Electronics, Columbus, OH). On the day following the FOB and motor activity tests, animals are trained on a passive avoidance test. This test is used to determine whether DU affects memory function. The tests are conducted in a passive avoidance apparatus (San Diego Instruments, San Diego, CA) that consists of 2 chambers (1 lighted, 1 darkened) separated by a sliding door. The animal receives a training trial during which time it is initially placed into the lighted chamber. The natural tendency is for the rat to enter the darkened chamber. When it does, it receives a mild foot shock. During this acquisition phase, the rats are tested for eight trials or until criterion is met. The criterion is 2 consecutive trials during which the rat does not cross into the darkened chamber. Each trial is 3 min in duration with a 1 min intertrial interval. Seventy-two hours later the rat is placed into the lighted chamber and retested. A comparison is made with the initial training session to see if memory of the task has been retained.

Conduction velocities: One week following the behavioral testing, the rats are evaluated electrophysiologically. Rats are anesthetized with ketamine (80 mg/kg) with xylazine hydrochloride (4 mg/kg) i.m. (supplemented as necessary). The right sciatic nerve is exposed and bipolar stimulating electrodes are positioned along the nerve in the thigh close to the sciatic notch and in a second location close to the knee. A recording electrode is inserted into the medial gastrocnemius muscle to monitor the compound muscle action potential. Nerve temperature is monitored and maintained near 37° C with a heat lamp. Nerves are stimulated at a frequency of 0.2 Hz. Stimulus intensity is varied between approximately 10 and 100 V (0.1 ms duration) to determine the input-output relationship and the supramaximal stimulation parameters to use. Five muscle responses are averaged and the latency, duration and amplitude of the potentials are measured. Conduction velocities are calculated by dividing the distance between the stimulating electrodes by the average latency difference between the time of onset of the compound muscle action potentials.

Duration of the muscle action potential reflects the synchrony of discharge. In general, the distal stimulating electrode will produce a faster, larger response than the proximal electrode. Greater dispersion and greater decrease in amplitude than normal would suggest nerve damage. For example,

demyelinating disorders cause dispersion of the muscle action potential by slowing the nerve conduction velocities[5,50]. If dispersion occurs over a short segment, compression neuropathy may be indicated[5].

All stimulation and recording are controlled by a 486 PC using standard electrophysiological software (Axon Instruments). Data are analyzed with routines written in AxoBasic (Axon Instruments) and statistical analysis is done with RS/1 (BBN Software Products) routines. Two-way analysis of variance (for time and dose) is used to compare differences among the experimental groups.

Sample collection: Following behavioral testing, blood and urine samples are obtained from all rats for analysis of renal function. To safely collect the blood samples, rats are immobilized by placing them in a Plexiglas restrainer. During each collection, 0.3-0.5 ml of blood is obtained from the tail vein using a 22-gauge needle. The blood is then centrifuged for 5 min at 3,000 X g. The serum is analyzed for uranium levels and/or for biochemical indices of renal function. Serum is stored at -70°C until ready for analysis.

Urine samples are collected by housing the rats in individual metabolism cages (23.5 cm diameter X 12 cm high) where they have continuous access to food and water. However, since these housing procedures have been shown to induce stress and thus increase the toxicity of uranium[4], the rats are acclimated to the metabolic cages for 5 days before the study begins. The metabolic cages are disinfected and decontaminated between each animal use. The 24-hr urine collection sample is obtained from each rat and the volume recorded (10-20 ml). Urine collection at 4°C is unnecessary since enzyme activity has been shown to be stable at room temperature for up to 24 hours[63]. After collection, urine is filtered to remove any debris and stored in plastic containers at 4°C until analyzed (less than 1 wk).

Evaluation of renal function: Measurement of urine volume and osmolarity, urine levels of NAG, LDH, glucose, total protein, creatinine and blood levels of glucose, urea and creatinine are used as indicators of renal function. In addition, since weight loss may be indicative of nephrotoxicity, all the rats are weighed weekly throughout the study. Osmolarity of the urine is measured with a vapor pressure osmometer (Model 5100B, Wescor, Inc.). A Kodak Ektachem 700 Analyzer is used to deter-

mine plasma and urine levels of creatinine, glucose and urea. Total urine protein is measured with a dye-binding assay (Coomassie Blue, BioRad) sensitive down to 1 μg. The activity of NAG is measured by the methods of Tucker et al.[56] using 4-methylumbelliferyl-N-acetyl-β-D-glucosaminide as the fluorescent substrate (excitation wavelength=356 nm; emission wavelength=446 nm). The dilution of the urine for this assay eliminates the effects of any inhibitors present[56]. For LDH measurements, 1 ml of urine is dialyzed for 4 hr at 4°C with 1 liter of deionized water. LDH is quantitated with a colorimetric assay that measures a reaction product which is proportionate to LDH activity (Oxford Biomedical Research Inc). Only 50-100 μl of fluid (urine or plasma) are required for each of these assays.

Although, urine volume and osmolarity can vary greatly with fluid intake, these measures provide physical indicators of renal function. For example, acute kidney failure drastically decreases urine volume, while moderate renal toxicity can increase urine output, as is seen with uranium exposure (e.g., [11]). Osmolarity can reflect the ability of the kidney to concentrate (or dilute) the urine. Plasma urea also changes with renal insufficiency. Since the rate of urea formation is proportionate to the rate of protein metabolism, other factors such as hepatic injury or altered protein intake can affect the measured urea in plasma. A small concentration of protein is normally present in the urine. Increases in total urine protein could result either from glomerular leakage or failure of tubule reabsorption. Urinary enzymes are sensitive, non-invasive markers of toxicity primarily in the kidney tubules[46]. NAG is a lysosomal enzyme found in proximal renal tubule cells. LDH is a cytosolic enzyme of the tubular epithelium.

Creatinine clearance is a commonly used measure of glomerular filtration rate in the rat despite a significant but constant tubular secretion. The use of an intrinsic metabolite has an obvious advantage over inulin or mannitol which (although not secreted) must be infused. Interpretation must be cautious since tubular injury with uranium could cause an underestimate of the glomerular filtration rate regardless of the marker used[8]. Creatinine clearance (C_c) is calculated from the equation: $C_c=U_c*V_u/P_c$ where U_c and P_c are the creatinine concentrations in urine and plasma, respectively, and V_u is the rate of urine production (ml/min).

Appearance of glucose in the urine occurs when the tubule reabsorption maximum from the filtrate is exceeded. This can occur with hyperglycemia or with a decrease in tubular reabsorption capacity. Measurement of both urine and plasma glucose help to distinguish between these two possibilities. Changes in reabsorption is reflected in the calculated fractional excretion (FE): $FE = (U_g/P_g) \div (U_c/P_c)$; where U_g and P_g are the glucose concentrations in urine and plasma, respectively.

The proposed assays provide a broad spectrum of measures of kidney toxicity. Many of these substances have been shown to be very sensitive in acute uranium toxicity[8,31]. Glucose is one of the most sensitive indicators[8,9] showing increased urine glucose, without concurrent increases in plasma. LDH and to a lesser extent NAG increase following uranium exposure[8,31]. A transient increase in urine volume and the appearance of protein in the urine also occur with acute uranium toxicity[31]. These measures are used together as indicators of kidney toxicity and carefully interpreted and correlated with histopathology. Two-way ANOVA is used to test the statistical significance of any changes.

Histopathology. Immediately following euthanasia on the day of electrophysiological analysis, tissue samples from bone (tibia, skull), hippocampus, sciatic nerve, kidney, liver, spleen and fragment capsule with associated skeletal muscle is obtained for histological examination or uranium measurement . Based on the literature, these are the most likely tissues to show increased levels of uranium[19,27,29,30,61,62]. Standard procedures for handling biologic specimens are used in the preparation of the samples. Tissues are perfused, embedded, mounted and stained with hematoxylin and eosin stain (H & E)[34]. Specialized stains are used to demonstrate specific lesions or further delineate lesions not well defined by the H & E stain. For example, silver stains are used on neural tissue to delineate nerve fiber disruption or degeneration[34].

The pathologist evaluating the tissue is blind to the experimental group from which the tissue was obtained. The pathologist generates a 0 to 4 scoring system to evaluate the degree of microscopic changes observed; where 0=no change, 1=minimal change, 2=mild change, 3=moderate change, and 4=marked or severe change. All tissue changes observed in the rats implanted with DU are contrasted and compared to the identical tissues taken from the controls. If there are significant changes noted in a particular system, for example the renal system, a detailed statement of criteria for 0-4 scores is

stated by the pathologist at the time of interpretation.

Uranium measurement Tissue samples are frozen and shipped by overnight courier on dry ice to Battelle, Pacific Northwest Laboratories for analysis of uranium content. The samples are stored at -70 C until the wet ashing procedure. Wet ashing consists of 12 cycles of treatment of the samples (over 3 days) with 2 ml of 16 N nitric acid followed by several hours of heating, brief cooling, addition of 0.5 ml of 30% hydrogen peroxide and reduction of the volume to approximately 0.5 ml. After this, samples are heated to dryness, dissolved in 2 ml of 4 M nitric acid with warming and filtered through 0.45 μm syringe filter units. For analysis, 0.5 ml of sample or identically handled standards are dissolved in 2 ml of Uraplex reagent. The samples are analyzed with a Kinetic Phosphorescence Analyzer (KPA-11, Chemchek Instruments Inc, Richland WA). Background measurements are made using 4 M nitric acid. Calibration curves are established prior to sample analysis. Measurements include analysis of relative standard deviations and correlation coefficients of the luminescence decay curve.

UNPUBLISHED DATA UNPUBLISHED DATA

RESULTS

DOSE RANGING STUDY

The first aim of our study was to determine appropriate doses for the subsequent toxicological analysis. Our pilot studies revealed that 8 DU pellets were well tolerated by the rats despite high urine levels of uranium; biochemical and histopathological damage were not evident. To determine the high dose for the present study we attempted to maximize the implanted number of DU pellets that could be tolerated by the rats and produced kidney uranium levels in the range of 0.7-1.4 μg/g. We implanted 4, 6, 16, 18 and 20 pellets into 4 animals each and evaluated urine, plasma and kidney levels after two weeks. The two-week time point was chosen to allow the urine and kidney levels of uranium to stabilize following implantation. Tantalum pellets were implanted in 4 animals for controls. As illustrated in Figure 1, the uranium (U) levels in urine, plasma and kidney were significantly increased in all DU implanted animals. There was a wide variation in the levels from animal to animal but a dose dependence was evident. Animals implanted with 4 DU pellets averaged 0.66 ± .20 μg U/g while 20 pellets resulted in 1.22 ± .31 μg U/g in the kidney. Urine levels in animals with 4 DU pellets were 83.3 ± 37.2 μg U/l and in animals with 20 pellets were 262.0 ± 99.2 μg U/l. In comparison, tantalum (Ta) implanted animals showed 0.002 μg/g U in kidney and 2.66 μg U/l in urine. None of the implanted or control animals demonstrated any obvious health problems. No significant differences were observed in the biochemical analyses of urine and serum: NAG, LDH, protein, osmolarity, glucose, urea, or creatinine. Based on these data, we chose 20 DU pellets as our high dose and 4 DU pellets as our low dose. The intermediate dose was calculated as the approximate logarithmic mean of the high and low doses; 10 DU pellets. All animals always received a total of 20 pellets, 10 in each hindlimb. For example, the low dose of 4 DU pellets consisted of 2 DU pellets and 8 Ta pellets in each rear leg.

THIRTY-DAY TOXICITY STUDY

Because of the staggered experimental schedule required for complete analysis of the numerous experimental endpoints, all subjects have not been evaluated to date. Sample sizes, therefore, will vary.

Neurotoxicity: Animals implanted with 4, 10 or 20 DU pellets, 20 Ta pellets and non-surgical controls were evaluated for body weight and for changes in the functional observation battery (FOB), locomotor activity, and passive avoidance learning. The rats were weighed weekly and all steadily gained weight. No significant differences in body weight ($P>0.05$) were observed among the 5 experimental groups (N=7-8/group) at any time point (Figure 2).

There were no significant differences among the 5 experimental groups for their performance on the passive avoidance test (N=7-10/group). All animals learned to avoid the mild foot shock within 2-3 trials. The latency to initial crossover (approximately 60 sec) was also not significantly different among groups (Figure 3). In addition, the FOB did not reveal any significant differences among the experimental groups. Grip strength of the hind- and forelimbs was not significantly altered by DU exposure (N=7-10) (Figure 4). Sensorimotor, neuromotor and autonomic responses as well as locomotor activity showed no significant differences across the experimental groups (N=8-9/group). As expected in all groups, the initial locomotor activity was high when the animals were first placed in the activity monitors because of exploratory behavior which subsided over time (Figure 5). Conduction velocity measurements from the nerves of the hindlimb also did not reveal any differences among the experimental groups (data not shown, N=2-3/group).

Nephrotoxicity: The urine and serum samples have been analyzed for biochemical markers of kidney toxicity (N=6/group). Osmolarity, 24-hour volume, pH and urine levels of glucose, protein, NAG, LDH, and urea nitrogen were not significantly altered. The data for urine glucose, protein and NAG are shown in Figure 6. Serum levels of urea nitrogen and glucose were also not significantly affected by experimental procedures (data not shown). Creatinine clearance was not significantly different among the experimental groups: Non-surgical 2.9 ± 0.5; Ta controls 2.6 ± 0.7; 4-DU 2.7 ± 0.6; 10-DU 2.8 ± 0.2; 20-DU 2.6 ± 0.6 (N=8-10/group). Fractional excretion (FE) of glucose (glucose

clearance/creatinine clearance) was similarly not significantly affected by the experimental procedures with all groups showing an FE approximately equal to 0.001 with high variability.

Histopathology: Tissues have been excised and fixed for histopathological analysis. These tissues (bone: tibia and skull, kidney, spleen, liver, brain, and muscle: proximal and distal) have not yet been processed and evaluated. During excision of the pellets it was observed that the depleted uranium pellets but not the tantalum pellets were associated with adherent tissue. In the 30-day animals, a capsule had not fully formed around the pellets and dark fluids were not observed near the fragments.

Uranium distribution: Tissues and fluids from two rats of each of the 5 experimental groups have been analyzed for uranium content by Battelle Pacific Northwest Laboratories and provide some interesting preliminary findings. The remainder of the tissues (N=5 per group) have been shipped recently to the Battelle Laboratories for measurement. In the preliminary analysis uranium in the urine and kidney was dose-dependent, in contrast to serum uranium (Figure 7). Uranium distributed to bone, both skull and tibia, in relation to the number of DU pellets (Figure 8). The levels of uranium in bone were comparable to the levels in kidney. Muscle from the forelimb (distal muscle, Figure 8) also showed a dose-dependent distribution, although at much lower levels (note the change in ordinate scale). Some of the muscle samples that were in close proximity to the DU pellets showed exceptionally high levels of uranium. It is our belief that these high levels resulted directly from fragments of the implanted pellets in the analyzed samples. This "contamination" could have occurred during the removal of the pellets at time of necropsy or might have happened by flaking and redistribution *in vivo*. Further analyses are expected to clarify this issue.

Spleen samples showed a dose-dependent showed accumulation of uranium while liver samples exhibited only background levels (Figure 9). Brain tissue showed low levels of uranium in animals implanted with 4 or 10 DU pellets. However, in animals implanted with 20 DU pellets, brain uranium levels reached concentrations approaching those in the spleen (Figure 9).

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CONCLUSIONS

The dose ranging study provided the data for establishing the appropriate numbers of DU pellets to be used in the toxicological studies. We found that 20 pellets of DU met our criteria for the high dose while 4 pellets was determined to be appropriate for our low dose. Ten pellets was calculated to be the appropriate intermediate dose for the evaluation of the toxicological effects of depleted uranium. The 30-day study was initiated and almost all of the 30-day experimental animals have been now been euthanized. The biochemical analyses have been completed on approximately two-thirds of the samples. Uranium analysis has been completed on only two animals from each experimental group but the remaining samples from an additional 5 animals per group are currently under evaluation. Histological evaluation of the collected tissues will be initiated shortly. This month, we are initiating the surgical implantation of the depleted uranium pellets for all the remaining time points (6 months, 12 months and 18 months). Although our current findings do not demonstrate significant toxicological effects within the first thirty days of exposure, the levels of uranium in various target tissues suggest the potential for measurable toxicity with chronic exposure. Furthermore, continued analyses of the toxicological endpoints and localization of uranium will allow an improved model of the biokinetic distribution of the metal.

The high dose of DU used in our study (20 DU pellets) produced kidney uranium levels of approximately 1.2 μg U/g kidney wet weight within 2 weeks. These levels were found to be sustained for at least 30 days. Although these levels of uranium have been reported by others to cause renal toxicity, our data do not demonstrate any significant signs of nephrotoxicity. Chemical form, route of administration, and the dose of uranium exposure can all affect the toxicological consequences and distribution of uranium. The uranium levels that result in kidney toxicity are a matter of debate in the literature. The Nuclear Regulatory Commission has set 3 μg/g as an lower limit for toxicity. However, several studies reflect damage at lower levels. For example, Diamond et al.[9] observed acute, but reversible, renal toxicity in rats at levels as low as 0.7 μg/g following i.v. injection of uranyl fluoride. In contrast

are the studies of Leach et al.[29,30] demonstrating no renal toxicity in rats following chronic inhalation exposure to uranium dioxide producing kidney levels up to 1.1 μg/g. The absence of effects in our present study does not preclude the possibility that with longer exposures to the uranium, toxicity will develop.

While bone and kidney are well accepted as primary reservoirs of uranium, other organs accumulate the metal to varying degrees. With oral administration of 8 mg/kg/day uranyl acetate for 4 weeks, Ortega et al.[45] found kidney, liver and thyroid as primary sites. A single intravenous injection of sodium uranyl tricarbonate distributed in 24 hours to kidney, liver, spleen and bone but at 30 days was detected predominantly in spleen and bone[60]. As expected, our preliminary data reveal that 30 days after implantation of DU pellets, levels of uranium in bone and kidney are high. Both marrow bones (tibia) and non-marrow bones (skull) accumulated uranium. Concentrations in the liver were not above background while concentrations in the spleen and muscle were significantly higher. Muscle levels raise the possibility that neuromuscular deficits will develop through heavy metal effects. Spleen levels cause concern that immunological consequences could arise. Future studies are planned to address this possibility. In agreement with the literature [45] uranium did not accumulate in the brain at the lower doses of DU. However, at the high dose, the levels were comparable to those in the muscle and spleen. This raises the concern that central nervous system consequences will occur with continued high levels of DU exposure. Our later time points planned for this study will address these concerns with behavioral and electrophysiological analyses. Levels of uranium excreted in the urine remained high throughout the 30 days. This suggests that uranium continues to leech out of the DU pellets, although serum levels are relatively low. This is also reflected in the Desert Storm veterans with embedded DU shrapnel who continue to excrete uranium in their urine even years after injury.

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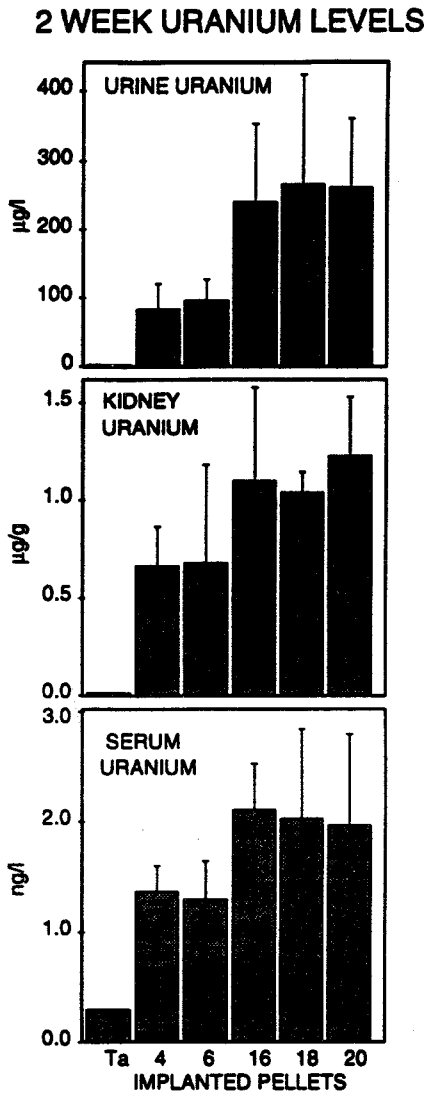
APPENDIX

UNPUBLISHED DATA: FIGURE 1



UNPUBLISHED DATA: FIGURE 2

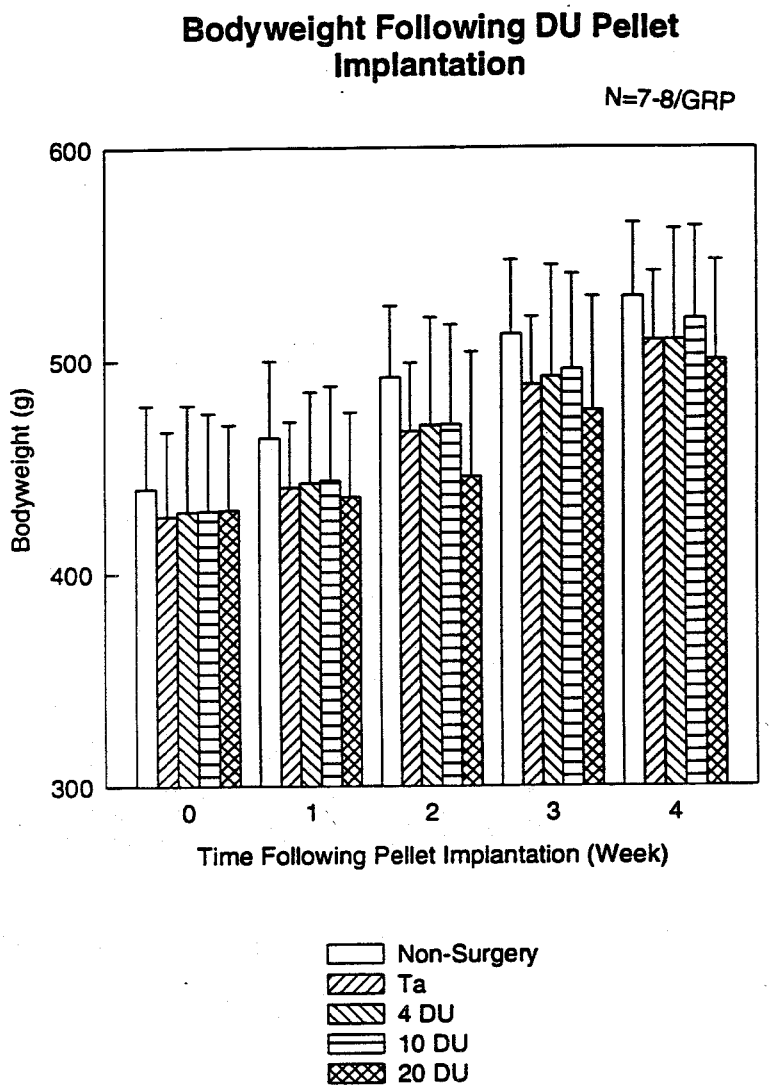
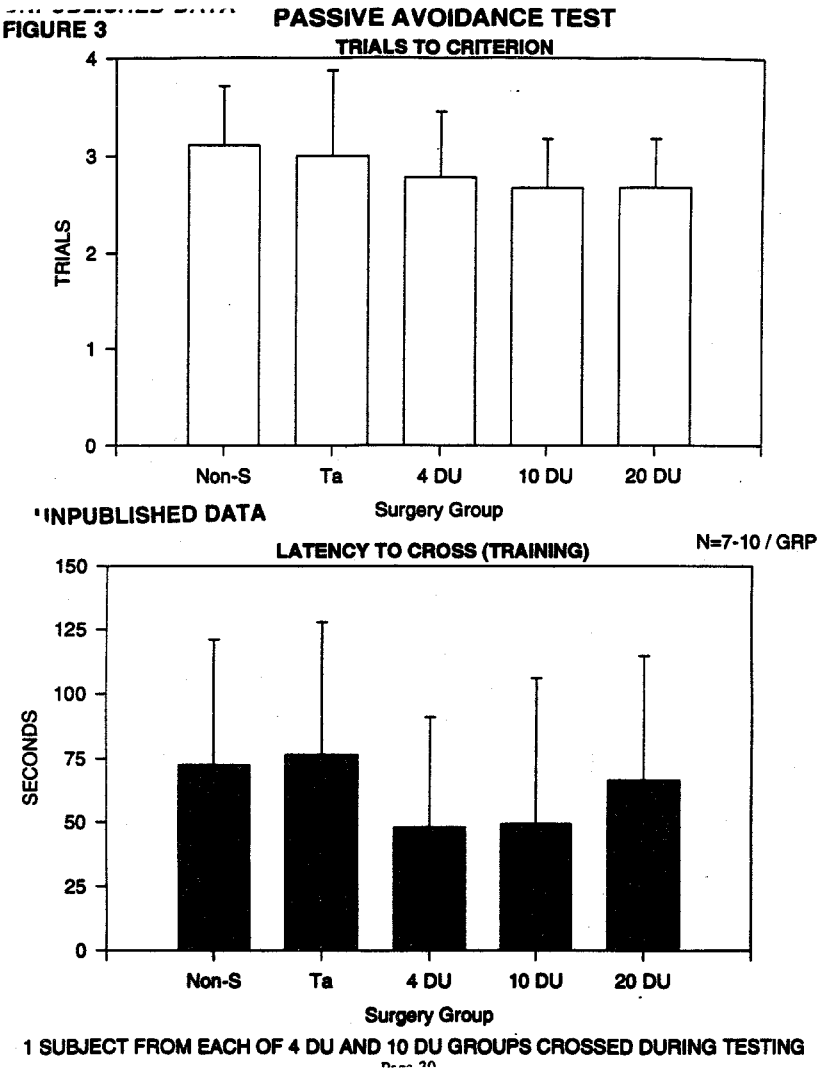
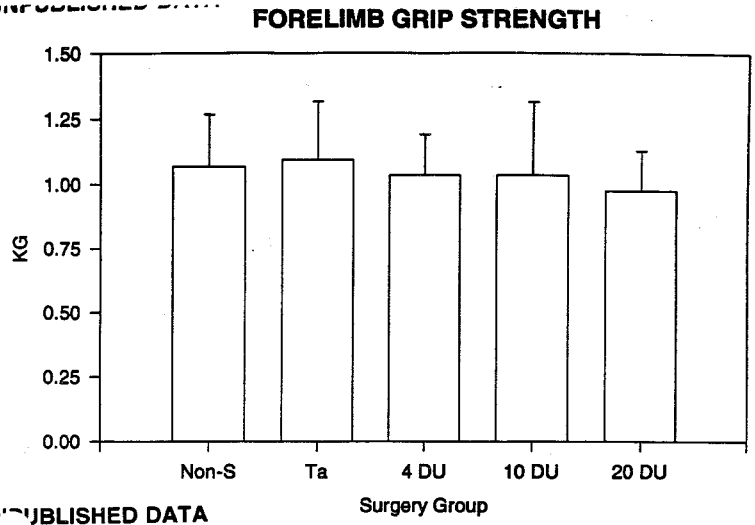


FIGURE 3



1 SUBJECT FROM EACH OF 4 DU AND 10 DU GROUPS CROSSED DURING TESTING

UNPUBLISHED DATA



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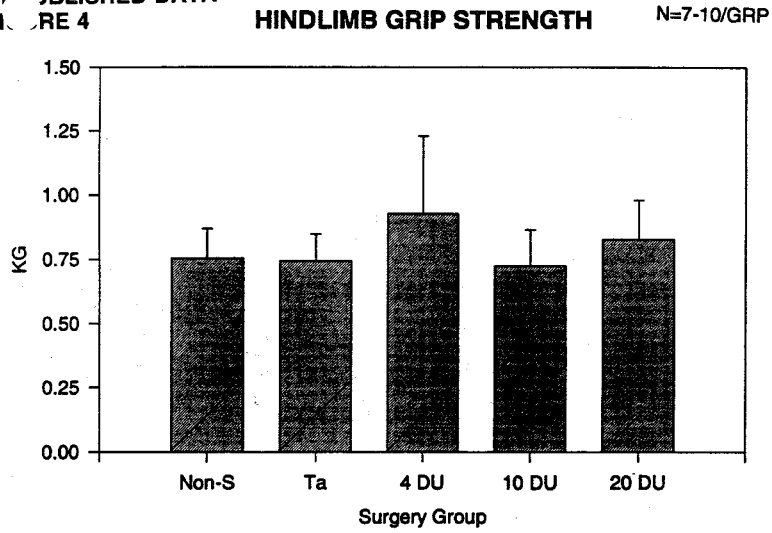

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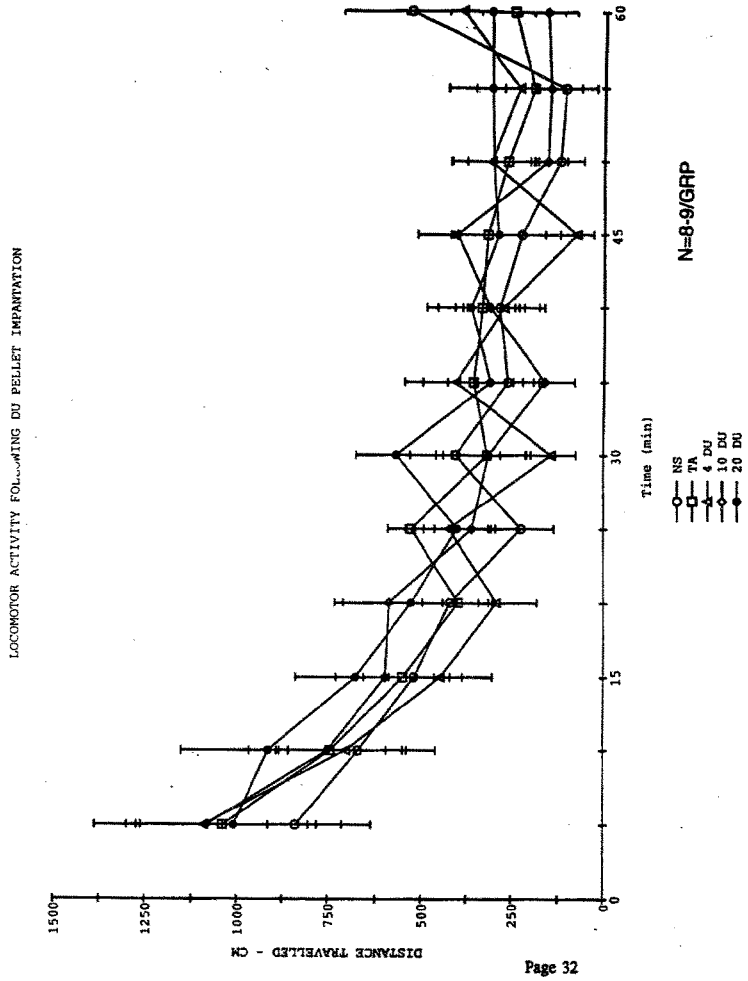

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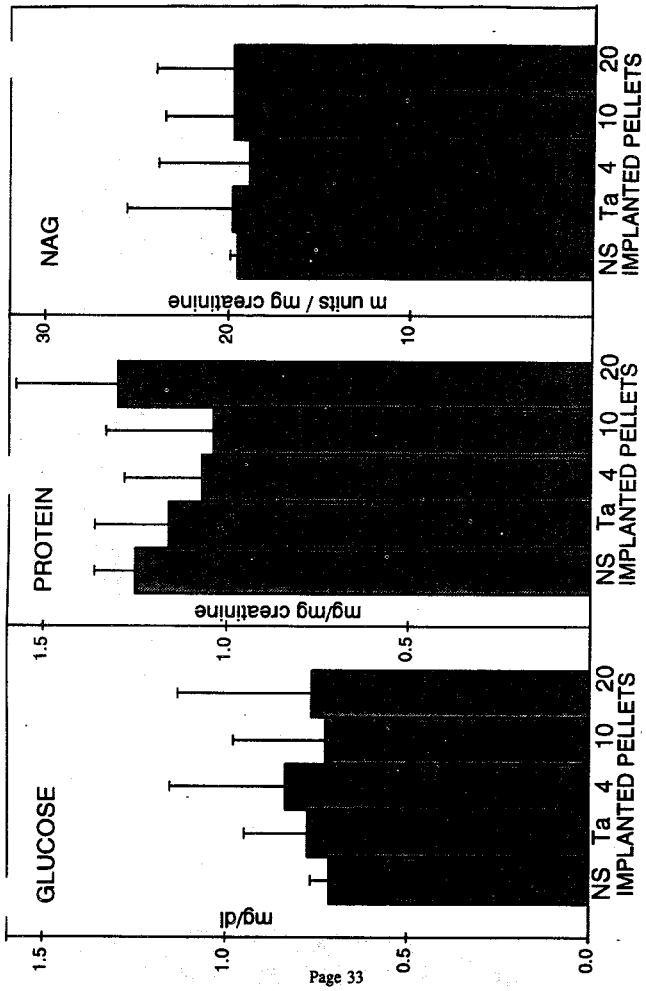

N=6/GRP

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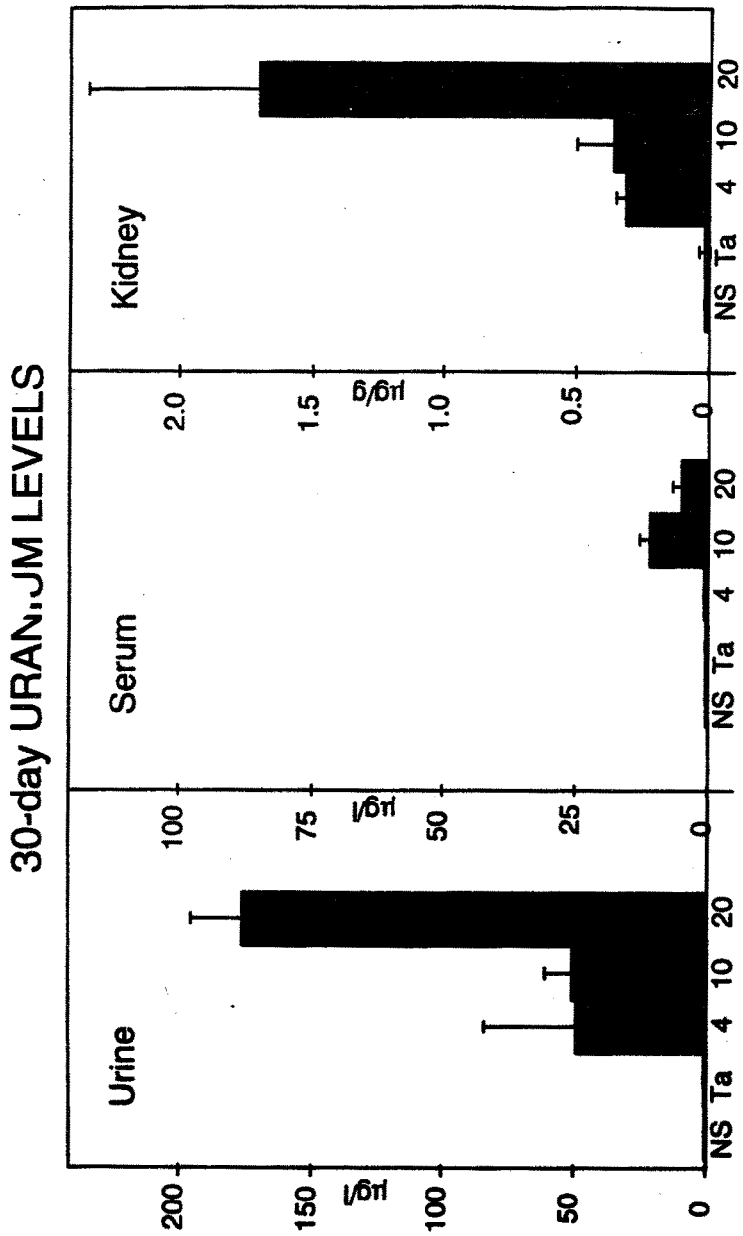

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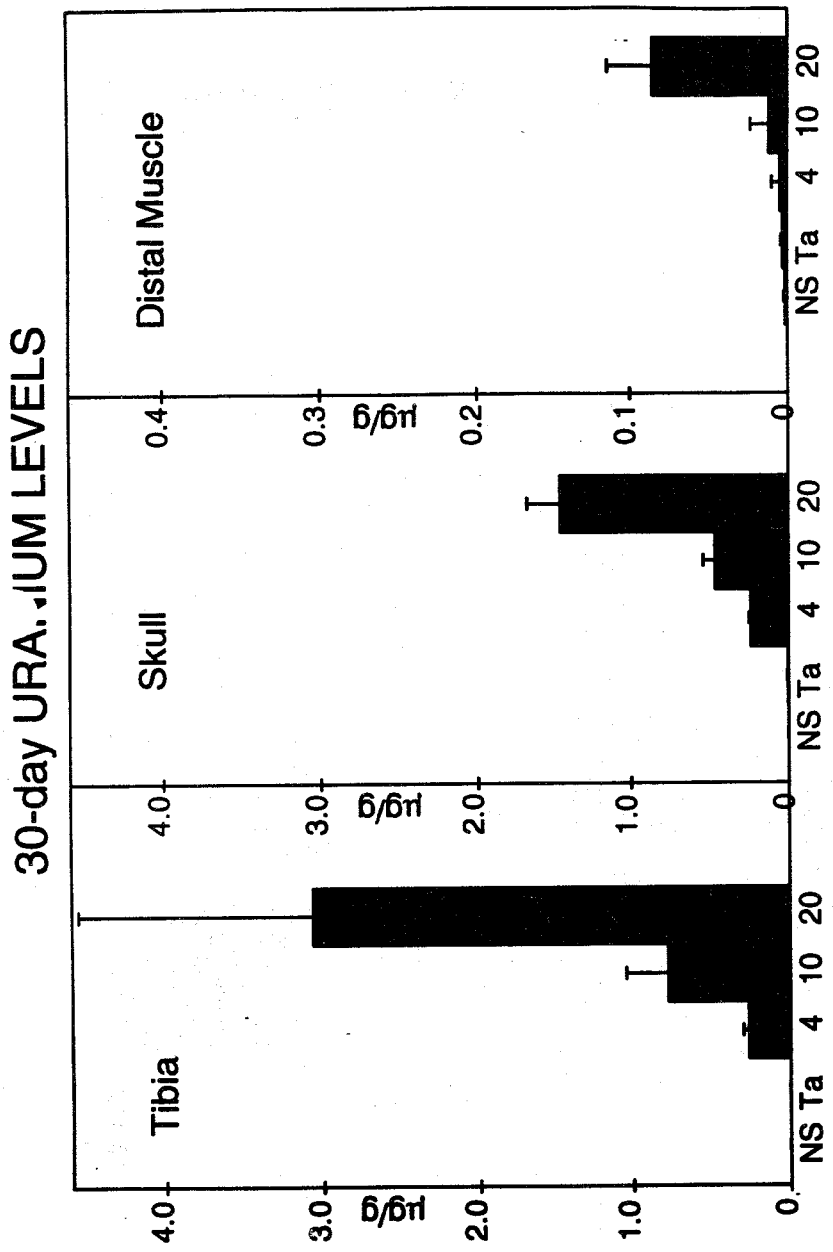

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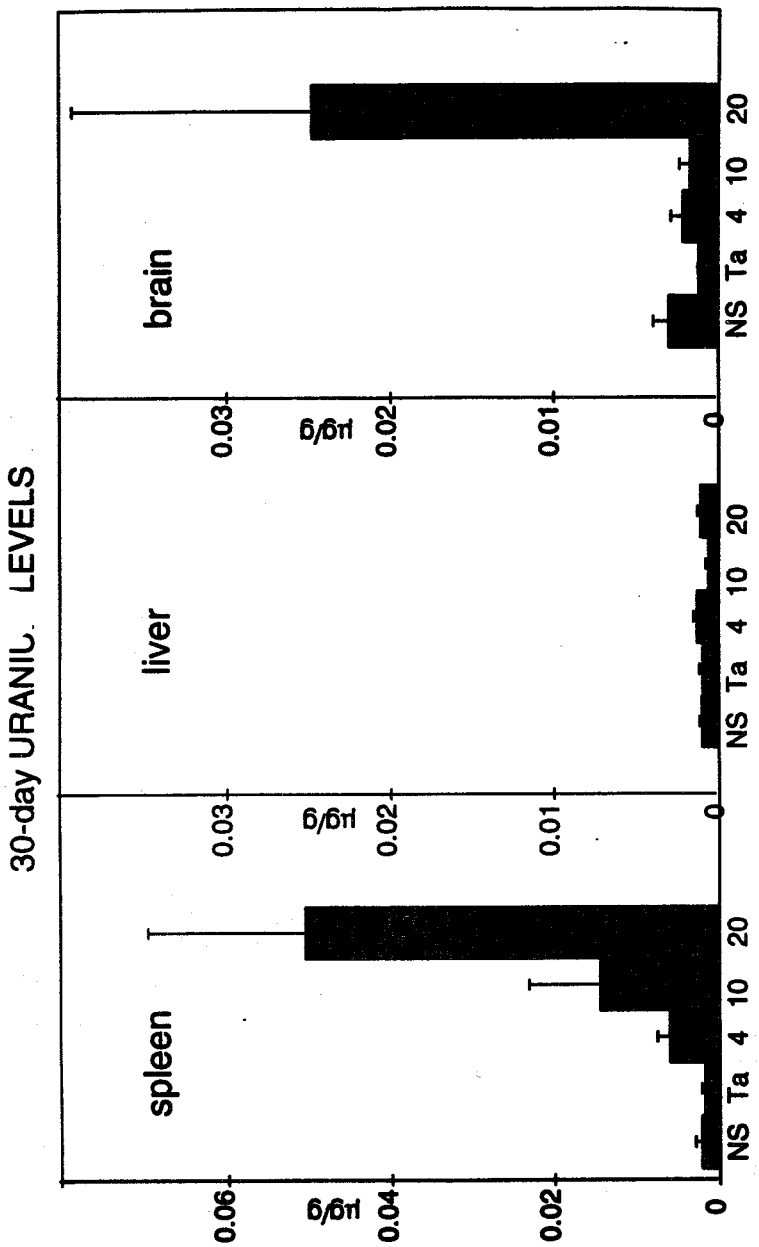

Attachment 7

MICRODOSIMETRY OF ALPHA PARTICLES FOR IN-VITRO HUMAN-CELL TRANSFORMATION STUDIES USING THE MONTE CARLO TECHNIQUE

T.J. St.John, A. C. Miller, R.C. Bhatt, B.A. Torres, and H.M. Gerstenberg
Radiation Sciences Department, Armed Forces Radiobiology Research Institute

Recent studies involving exposure of human osteoblast-like cells (HOS) to a solution of depleted uranium (DU) uranyl chloride (NO_2Cl_2) resulted in genetic alterations associated with an induction of the tumorigenic phenotype [Miller, et al., to be published]. This transformation may result from either heavy-metal induced alterations, exposure to 4.2 MeV alpha particles emitted by ^{238}U in NO_2Cl_2, or a combination of the two. This work describes the modification and application of an existing Monte Carlo calculational code developed by J.L. Humm [Radiation Research 134, 143-150 (1993)], which was designed and tested for use in predicting cell inactivation by alpha-particle emitters, to estimate the specific energy delivered to the HOS cells in-vitro. The relative uptake and partition of 238U onto the cell walls and into the cytoplasm and nuclei were determined and used in the code to calculate the energy deposition stochastics to individual cell nuclei for various exposure times and uranium concentrations. The results of these calculations will be compared with transformation frequency to determine if transformation rate is correlated with radiation dose.

UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES
RESEARCH DAY MARCH 24-25, 1997

P06-135

MEASUREMENT OF DEPLETED URANIUM IN CELL PARTITIONS TO CORRELATE MICRODOSIMETRIC DOSE ESTIMATES AND CARCINOGENESIS STUDIES OF HUMAN OSTEOBLAST-LIKE CELLS (HOS): M.M. Hamilton*, J. W. Ejnik*, A. E. Olufade*, J.N. Sanders*, H.M. Gerstenberg, A.C. Miller, A.J. Carmichael*. Armed Forces Radiobiology Research Institute (AFRRI), Bethesda, MD 20889.

Current efforts to assess possible late health effects of embedded depleted uranium (DU) fragments depend greatly on accurate quantification of uranium in biological samples. Therefore, AFRRI has established an analytical lab to measure uranium by phosphorescence. The phosphorescence instrumentation in use is the Kinetic Phosphorescence Analyzer, KPA-11, with an automatic sampler developed by Chemchek Instruments, Inc. Richland, WA [Analytical Chemistry, 64, 1413-1418 (1992)]. The KPA measures the decaying phosphorescence of the excited uranyl complex with respect to time. The method is sensitive and accurate with a quoted detection limit of 1 ng/L and a standard deviation below 5% for uranium concentrations greater than 100 ng/L. The phosphorescence results will be compared to the DU specific activity by counting alpha and beta particles using gas proportional counters. This work will determine quantities of DU distributed to specific subcellular locations in HOS cells currently used in these transformation studies. The results will be incorporated into the Monte Carlo calculational code for microdosimetric dose-estimates [St. John et al., Abstract, Radiation Research Meeting, May 1997]. Furthermore, the results describing the DU subcellular localization will provide information that is essential to a better understanding of the mutagenic and cytogenetic results [Miller et al., Abstract, Radiation Research Meeting, May 1997] and allow comparisons between the chemical and the radiological toxicity of DU in the carcinogenesis results.

RADIATION RESEARCH SOCIETY
45TH ANNUAL MEETING MAY 3-7, 1997

#787 Monday, April 22, 1996, 8:00–12:00, Poster Section 8

Carcinogenesis risk assessment: Oncogenic activation by depleted uranium compounds *in vitro* and *in vivo*. Miller AC, Whittaker T, Benson K. *Armed Forces Radiobiol. Res. Inst., Bethesda, MD.*

Limited data exists to permit an accurate assessment of risks for carcinogenesis from depleted uranium (DU) embedded fragments or inhaled particulates: DU-risk assessment is complicated by the dual toxicity of imbedded DU fragments, i.e., chemical vs. radiological. Epidemiological studies have indirectly linked exposure to either non-radioactive heavy-metal compounds or radioactive uranium to human carcinogenesis and oncogene activation. At a negligible radiation dose (< 2.75 urad), transformation of human cells to the tumorigenic phenotype was induced using soluble DU-uranyl chloride (UO_2Cl_2). Significant changes were observed in transformation rate, saturation density, growth rate, tumorigenicity *in vivo* and metastatic capability of DU-transformed cells. Genetic alterations included oncogene activation, e.g., *ras*, *bcl2*, and tumor suppressor gene mutation, e.g., p53. Similarly, *in vivo* studies demonstrated expression of *ras*, *fos*, and *jun* in muscle tissue obtained from rats with implanted DU pellets. In contrast, tissues obtained from rats with implanted tantalum pellets did not show oncogene expression. These data suggest that long-term cellular exposure to uranium could potentially be critical to development of neoplastic disease in humans.

AMERICAN ASSOC. FOR CANCER RESEARCH

87TH ANNUAL MEETING APRIL 20-24, 1997

#3092 Biomarkers for carcinogenesis: Oncogenic activation by depleted uranium *in vivo*. Miller, A.C., Whittaker, T., McBride, S., Hogan, J., Benson, K., and Siu, H. *Armed Forces Radiobiology Research Institute, Bethesda, MD 20889*

Limited data exists to permit an accurate assessment of risks for carcinogenesis from depleted uranium (DU) embedded fragments or inhaled particulates; DU-risk assessment is complicated by the dual toxicity of imbedded DU fragments, i.e., chemical vs. radiological. Epidemiological studies have indirectly linked exposure to either non-radioactive heavy-metal compounds or radioactive uranium to human carcinogenesis and oncogene activation. We have shown that DU-uranyl chloride can transform human cells to the tumorigenic phenotype at a negligible radiation dose (0.131 uGy). DU toxicology studies are currently underway using a rat model and tissues are also being analyzed for biomarkers of carcinogenesis, e.g., oncogene activation, p53 alterations, etc. Muscle, liver, and kidney tissues, which showed a 19- to 1000- fold increase in uranium levels six (6) months after pellet implantation, also demonstrated aberrant expression of *ras*, *bcl2*, *fms*, and *cyclin D1*. Mutation of p53 and an increase in p53 content has also been measured. The alteration in oncogene expression and p53 is both time and DU-dose dependent. In contrast, tissues obtained from rats with implanted tantalum pellets did not exhibit activated oncogene expression nor any change in p53. These data suggest that long-term cellular exposure to depleted uranium could potentially be critical to development of neoplastic disease in humans.

AMERICAN ASSOC. FOR CANCER RESEARCH
88TH ANNUAL MEETING APRIL 12-16, 1997

P22-457

TRANSFORMATION OF HUMAN CELLS BY DEPLETED URANIUM-URANYL CHLORIDE: ASSOCIATION WITH MUTAGENIC AND CYTOGENETIC EFFECTS

A.C. Miller[1], T. Whittaker[1], Jiaquan Xu[1], H. Siu[2], N. Page[2] [1]Armed Forces Radiobiology Research Institute and [2]Molecular Pharmacology, Div. Cancer Treatment, NCI, NIH, Bethesda, MD 20889.

Limited data exists to permit an accurate assessment of risks for carcinogenesis from depleted uranium (DU) embedded fragments or inhaled particulates; DU-risk assessment is complicated by the dual toxicity of imbedded DU fragments, i.e., chemical vs. radiological. Epidemiological studies have indirectly linked exposure to either non-radioactive heavy-metal compounds or radioactive uranium to human carcinogenesis and oncogene activation. We have shown that DU-uranyl chloride can transform human cells to the tumorigenic phenotype at a negligible radiation dose (24 hr DU-exposure). Recently, we examined the clastogenic and mutagenic actions of DU-uranyl chloride. DU induced mutations in both the *Salmonella typhimurium* tester strains TA100 and TA102 at concentrations greater than 5 uM. In contrast, nickel sulfate did not induce any mutations in these tester strains. However, both DU-uranyl chloride and nickel sulfate did induce unscheduled DNA synthesis, a measure of DNA repair, in the human cell model. The cytogenetic toxicity of DU-uranyl chloride was also studied. The results demonstrated that DU-uranyl chloride was genotoxic to human cells as revealed by increasing frequencies of micronuclei and sister chromatid exchanges. There was approximately a 2-fold increase in micronuclei and sister-chromatid exchange frequency in DU-treated cells; in contrast, lead acetate treatment caused a slight increase in micronuclei formation but had no significant effect on sister chromatid exchange frequency. These data on the genotoxic and mutagenic behavior of a DU-compound, which concur with our previous results demonstrating the transforming capability of DU-uranyl chloride, continue to suggest that long-term cellular exposure to depleted uranium could potentially be critical to development of neoplastic disease in humans.

THE OXYGEN CLUB OF GREATER WASHINGTON, D.C.
10TH ANNUAL SPRING CONF JUNE 10, 1997

231\203 ONCOGENES AS BIOMARKERS FOR LOW DOSE RADIATION-INDUCED DELAYED HEALTH EFFECTS

AC Miller PhD, T Whittaker, J Hogan, S McBride PhD, K Benson PhD Armed Forces Radiobiology Research Institute, Bethesda, MD 20889, USA

AIM: To determine if sublethal exposures to ionizing radiation relevant to military personnel, e.g. depleted uranium, (60)Co, results in the activation of oncogenes in the carcinogenic pathway and to assess the use of these biomarkers as a molecular epidemiologic approach to study carcinogenesis. METHODS: Two approaches have been developed in our laboratory to investigate the association between oncogene activation and increased incidence of cancer due to exposure to low dose radiation. Rodents were exposed to (60)Co gamma radiation (25 cGy/week/8 weeks, total dose 200 cGy). Animals were serially euthanized and tissues were examined for oncogene activation. Secondly, tissues from rodents implanted with depleted uranium (DU) pellets were similarly analyzed for oncogene activation. RESULTS: Results demonstrated that repeated exposure to (60)Co gamma radiation (25 cGy/week/8 weeks) of B6CF1 mice resulted in the activation of specific oncogenes associated with the initiation of neoplastic growth. Northern analysis of animal tissues demonstrated that *ras*, *myc*, *bcl2*, and *fos* were elevated in both lung and liver tissues 232 days following the radiation regimen. In contrast, lung tissues from animals not exposed to radiation demonstrated only a slight elevation in *myc* expression. Muscle tissues obtained from animals implanted with DU pellets exhibited a similar activation of *ras* and *bcl2* 475 days after pellet implantation. Furthermore, there was a dose-dependent relationship between the level of *ras* expression increase and the number of DU pellets implanted in kidney tissue. Tissues from animals with tantalum implants did not exhibit any significant activation in oncogene expression. CONCLUSIONS: Oncogenes may be effective biomarkers for low dose radiation-induced carcinogenesis.

KEYWORDS: oncogenes, biomarkers, depleted uranium, low dose radiation

INTERNATIONAL SOCIETY FOR PREVENTIVE ONCOLOGY "CANCER DETECTION & PREVENTION"
ISPO 1996 VOLUME 20 ISSUE 5

TRANSFORMATION OF HUMAN CELLS BY URANYL CHLORIDE: GENETIC ALTERATIONS

A.C. Miller[1], T. Whittaker[1], C.R. Woodruff[1], R.C. Bhatt, H.M. Gerstenberg, and S. Shack[2]. [1]Armed Forces Radiobiology Research Institute and [2]Clinical Pharmacology Branch, Div. Cancer Treatment, NCI, NIH, Bethesda, MD 20889.

Recent reports have confirmed heavy metal-induced cellular transformation to the tumorigenic phenotype. Little data exists however, regarding the cellular toxicity of soluble uranium. One component of uranium toxicity may be a metal-induced transformation similar to that observed with nickel and chromium. Immortalized, human osteoblast-like cells (HOS) were exposed to uranyl chloride (UO_2Cl_2), a soluble form of uranium. Cellular exposure to a nontoxic, noncytostatic dose of UO_2Cl_2 (10 nM) for 21 days, giving a radiation dose of approximately 2.75 µrad which is well below background, resulted in transformation of the nontumorigenic phenotype to the tumorigenic phenotype characterized by alteration of morphology, induction of anchorage independence, and activation or mutation of multiple oncogenes and tumor suppressor genes, respectively. These data demonstrate that a nontoxic chemical dose of a soluble uranium compound is capable of promoting human cells to an altered phenotype characteristic of the carcinogenesis cascade and suggest that long-term cellular exposure to uranium could potentially be critical to development of neoplastic disease in humans.

10th INTERNATIONAL CONGRESS OF RADIATION RESEARCH
AUG 27 – SEP 1, 1995

Attachment 8

July 1996

Establishment of an Animal Model to Evaluate the Biological Effects of Intramuscularly Embedded Depleted Uranium Fragments

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Technical Report 96-3

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Introduction

Natural uranium (U) consists of three isotopes: ^{238}U (99.276%), ^{235}U (0.718%), and ^{234}U (0.0056%). During the uranium enrichment process two isotopic mixtures are produced, "enriched uranium" and "depleted uranium" (DU) with different relative ratios of the three isotopes. Enriched uranium contains a higher percentage of the fissionable isotope ^{235}U and is used for nuclear reactor fuel and nuclear weapons. DU has a lower ^{235}U content. The DU used by the U.S. military for kinetic energy penetrators is alloyed with titanium (0.75% by weight) to increase its tensile strength and to retard oxidation. Current U.S. antitank weapons contain DU penetrators, and most of the Abrams tanks are armored with DU. During Operation Desert Storm, DU munitions were fired by the Army and Air Force. Unfortunately, during this conflict, a number of U.S. military personnel were wounded by DU fragments (Daxon, 1993; Daxon and Musk, 1993; GAO Report, 1993). Many of these fragments were not removed because the surgical procedure would produce excessive tissue damage. Radiographs of injured soldiers show multiple embedded fragments ranging in size from 1 mm to over 5 mm in diameter (see figures 1 and

Fig. 1. Radiograph of the leg of a soldier wounded by a DU munition during the Persian Gulf War. This soldier also had DU fragments in the feet and knees of both legs.

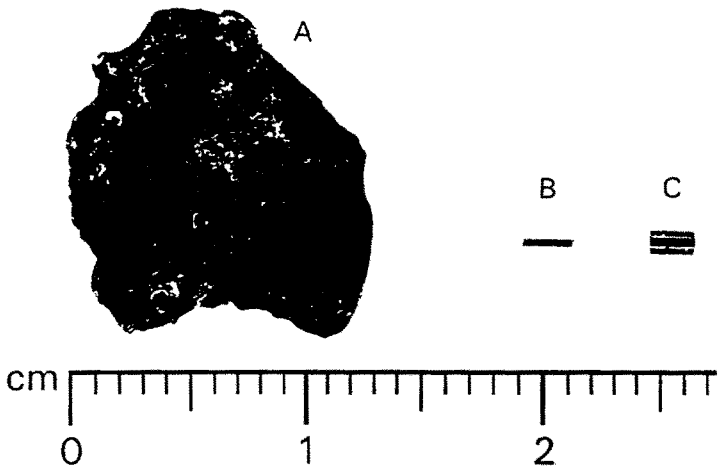

Fig. 2. (A) Photograph of an actual DU fragment removed from a soldier wounded during the Gulf War. (B) Photograph of a Ta pellet implanted in a rat. (C) Photograph of a DU pellet implanted in a rat.

2a). Indeed, fragments as large as 20 mm in diameter have been noted in other patients. Bioassays taken over a year after injury indicate that uranium was present at levels up to 30 µg U/l urine, well in excess of natural background (U.S. Army Environmental Hygiene Agency Memorandum for Office of the Surgeon General, 1994).

Although the toxicity of embedded DU is unknown, numerous studies have addressed the consequences of inhalation, ingestion, and parenteral administration of other forms of uranium (Diamond, 1989; La Touche et al., 1987; Morrow et al., 1982; Ortega et al., 1989a, b; Wrenn et al., 1989). After uranium is absorbed, it circulates in the blood as the uranyl ion, forming uranium-carbonate and uranium-albumin complexes. As the uranium-carbonate complex passes through the kidney, it is filtered rapidly by the glomeruli where 60% to 80% of the absorbed uranium is excreted in the first 24 hours after acute exposure. The uranium that is not excreted is reabsorbed by the proximal tubules where it produces significant toxic effects. Uranium also enters the bone, where it competes with calcium to form complexes with phosphate ions, thus becoming part of the bone matrix (Cabrini et al., 1984; Domingo et al., 1992; Guglielmotti et al., 1989; Neuman, 1950). This bone matrix then serves as both a long- and short-term storage site from which uranium is slowly released back into circulation (Kathren et al., 1989; Wrenn et al., 1985). The liver and muscle are other major sites of uranium deposition, with a possible long-term storage mechanism in the kidney (Kathren et al., 1989; Wrenn et al., 1985).

Acute morphological and biochemical changes of the kidney result from uranium exposure (Diamond, 1989; Kocher, 1989; Leggett, 1989; Neuman, 1950). Changes in the glomerular epithelial architecture (Kobayashi et al., 1984) and cellular necrosis in the proximal tubules near the corticomedullary junction of the kidney have been reported in experimental animals after acute uranium exposure (Brady et al., 1989; Haley et al., 1982; Haley, 1982). In addition,

polyuria, enzymuria, glucosuria, and increased excretion of amino acids have been demonstrated (Diamond, 1989; Diamond et al., 1989; Kocher, 1989; Zalups et al., 1988). Acute renal failure can indeed occur following exposure to high doses of uranium (Neuman, 1950; Ubios et al., 1994). Even acute environmental stressors such as restricted diets or changes in housing conditions have enhanced uranium toxicity significantly (Andrews and Bates, 1987; Damon et al., 1986).

Few studies have addressed the chronic toxicity of uranium, and the results available are conflicting (U.S. Department of Health and Human Services, 1990). Galibin and colleagues (1971) reported severe renal toxicity in rats that inhaled ammonium diuranate (1 or 8 mg/m^3), a slightly soluble uranium compound, for 128 days. Urine protein and blood non-protein nitrogen were elevated. In the proximal tubules, there were sloughed dead cells and abnormal regenerating cells. Although the total number of tubules was reduced and the kidney exhibited an increased amount of connective tissue, all the animals recovered. In contrast, Leach and colleagues (1970; 1973) found no renal toxicity in rats repeatedly exposed to uranium dioxide dust (5 mg/m^3) for a period of 12 months nor in dogs or monkeys exposed for 5 years. Yet uranium concentrations in the kidneys were as high as 1.1 μg U/g kidney wet weight in the rat, 8.3 μg U/g kidney weight in the dog, and 17.0 μg U/g kidney weight in the monkey. Uranium concentrations at these levels have been reported to cause acute renal toxicity (e.g., Kathren et al., 1989). Thus, the chronic effects of uranium exposure remain for the most part unresolved (Diamond, 1989).

The threshold concentration of kidney uranium levels in humans that result in kidney chemical toxicity is in dispute (Diamond, 1989; Kathren and Moore, 1986; Kocher, 1989; Stradling et al., 1988). While the Nuclear Regulatory Commission has set the level at 3.0 μg U/g kidney weight for renal damage in humans, there is evidence from both human and animal reports that this level could be considerably lower. For example, chronically exposed uranium mill workers, whose kidney uranium levels probably did not exceed 1 μg U/g kidney weight (Thun et al., 1985), showed mild renal dysfunction with increased urinary excretion of β_2-microglobulin and various amino acids. In rats exposed subchronically to low doses (cumulative dose: 0.66 or 1.32 mg/kg) of uranyl fluoride, kidney uranium levels as low as 0.7 to 1.4 μg U/g wet weight kidney produced cellular and tubular necrosis of the proximal tubule, proteinuria, and enzymuria (Diamond et al., 1989). These changes in rat renal function, however, were temporary, with complete recovery occurring within 35 days of exposure. These studies are important because they indicate that renal injury can occur at kidney uranium levels well below the 3.0 μg U/g limit.

Currently, no research into the direct toxic effects of embedded DU has been reported. The toxicity data that exist for low-level chronic uranium exposure used other routes of administration, and the results are contradictory. The uranium levels in humans that result in kidney toxicity are in dispute. For these various reasons, it is necessary to determine the health risks to the soldier resulting from long-term exposure to DU fragments. The goal of this pilot study was to establish an animal model that could be used in future research to investigate the biological effects of embedded DU.

Methods

Subjects and Experimental Design

Subjects were 12 naive Sprague-Dawley male rats (8-10 weeks old) obtained from Charles River Breeding Laboratories, Raleigh, N.C. On arrival, rats were quarantined and screened for diseases and were maintained in an AAALAC-accredited facility in accordance with the *Guide for the Care and Use of Laboratory Animals* (NIH Publication No. 86-23). Six rats were implanted with eight DU pellets (four in each biceps femoris muscle of the lateral thigh), and six rats were implanted with eight tantalum (Ta) pellets. Rats were individually housed in plastic Micro-Isolator cages with hardwood chips as bedding; during urine collection, rats were placed in metabolic cages. Commercial rodent chow and acidified water (pH 2.5, using concentrated HCl) were provided *ad libitum*. Rats were on a 12-hour light/dark cycle.

DU and Ta Pellets

DU pellets (1 mm in diameter x 2 mm in length) were obtained from Oak Ridge National Laboratories, Oak Ridge, Tenn. (see figure 2c). The cylindrical shape was chosen because it is the geometrical average of fragments left in soldiers wounded by conventional or DU munitions. The size of the pellets was based on two considerations. First, the total DU implanted was approximately 1% of the total biceps femoris muscle volume and did not seem to cause undue discomfort to the animal. Second, the surface area of 8 DU pellets of this size should result in detectable urinary uranium levels. DU pellets consisted of 99.25% DU and 0.75% titanium by weight. The uranium isotopes in DU were ^{238}U (99.75%), ^{235}U (0.25%), and trace amounts of ^{234}U. This is the same DU alloy used in U.S. military munitions.

Ta pellets (1 mm in diameter x 2 mm in length) were obtained from Alfa Products, Ward Hill, Mass., and served as the heavy metal control (see figure 2b). Ta was selected because its density is similar to DU density, 16.6 g/cm^3 for Ta versus 18.8 g/cm^3 for DU (Radiological Health Handbook, 1970), it is relatively inert in a biological medium (Johansson et al., 1990), and it is commonly used in human orthopedic reconstructive surgery (Hockley et al., 1990).

Surgical Procedures for Pellet Implantation

Before implantation surgery, the DU and Ta pellets were cleaned by immersion in an industrial detergent, rinsed in absolute alcohol, sterilized by immersion in a 50% nitric acid solution for 3 minutes, rinsed with sterile water, and then placed in acetone to inhibit oxidation. These sterilization procedures completely remove the oxide formation from the surface of DU metal (Tonry, 1993), and the results of an abbreviated sterility test of 10 Ta pellets using either a thioglycollate medium or soybean-casein digest medium detected no microorganisms.

Rats were administered atropine (0.05 mg/kg i.m.) before being anesthetized. Anesthesia was induced with ketamine hydrochloride (50 mg/kg) in combination with xylazine hydrochloride (10 mg/kg) given i.p. in a 0.5-ml bolus, using a 25-gauge needle. These injections were administered intraperitoneally to prevent irritating the site of implantation. The surgical sites were then shaved and cleansed with Betadine. Four pellets were implanted approximately 15 mm apart in each biceps femoris muscle on the lateral side of each thigh. Using a scalpel blade, incisions were made through the skin and approximately 10 mm deep into the muscle mass. The proximal incisions were 10 mm distal to the iliac crest and were the implantation sites of the first pellets. Pellets were secured in place with absorbable sutures (Dexon 4-0) to prevent movement. Rats were closely monitored following surgery until they were ambulatory. A veterinarian or a veterinary technician examined the surgical sites for signs of inflammation, infection, and local DU toxicity daily for 2 weeks

following surgery and weekly thereafter throughout the study.

Behavioral Measurements

Locomotor activity and grip strength were assessed on days 3 and 5 before surgical implantation and on days 1, 3, 7, 14, 28, 60, and 120 after surgery. Locomotor activity was quantified using computerized Digiscan activity monitors (Omnitech Electronics, Columbus, Ohio). Each monitor used an array of infrared photodetectors spaced 2.5 cm apart to determine horizontal locomotor activity, which was expressed as total distance traveled. Activity was monitored for 1 h with measurements taken every 5 min (Landauer et al., 1988).

Immediately following locomotor activity testing, the strength of both hindlimb and forelimb grips of each animal was measured using a grip strength apparatus (San Diego Instruments, San Diego, Calif.). In this test, the animal was required to grip a rectangular wire mesh surface (12 x 7 cm) with its forepaws and was then gently pulled back along a platform until its grip was broken. The backward motion was continued until the animal's hindpaw gripped another rectangular wire mesh surface (12 x 10 cm). As with the forelimb grip, the animal was gently pulled back until the hindlimb grip was broken. Readings on three push-pull strain gauges were used to record the maximum strain required to break both forelimb and hindlimb grips. This behavioral test is used in many laboratories to assess muscular weakness (Haggerty, 1989; Meyer et al., 1979).

Urinary Sampling and Collection Procedures

Urine samples were collected following behavioral testing on days 1, 3, 7, 14, 28, 60, and 120 after surgery and analyzed for uranium levels. Sampling at these time points was necessary because signs of nephrotoxicity in laboratory animals exposed to low doses of uranium are frequently not detected until 3 to 5 days after exposure and may subside within 7 days (Diamond, 1989). Urine samples were collected from rats in individual metabolic cages (23.5 cm diameter x 12 cm high) where they had continuous access to food and water. Rats were acclimated to the metabolic cages for 5 days before the study began because naive rats exposed to these housing procedures have shown a stress-induced increase in uranium toxicity (Damon et al., 1986).

A 24-h urine sample was obtained from each rat, and the volume was recorded. In addition, each animal's body weight and food and water consumption were recorded. Care was taken to prevent contaminating the urine with food or feces. After collection, urine was filtered to remove any debris and stored in plastic containers at 4° C until analyzed. The metabolic cages were disinfected and decontaminated between each animal use. During animal-handling periods, overt signs of behavioral toxicity and the overall appearance of the rats were recorded.

Determination of Urinary Uranium Levels

Urinary uranium levels were determined by alpha spectrometric techniques (Martin Marietta Energy Systems, Inc., Oak Ridge, Tenn.). An aliquot of the sample was dissolved in nitric acid (HNO_3) and hydrogen peroxide (H_2O_2). The sample was then wet ashed, and the uranium coprecipitated with calcium oxalate. After dissolving the precipitate in HCl, the uranium was further separated by ion exchange chromatography. The uranium was then eluted from the column with a solution of dilute HCl to which titanous chloride had been added to reduce actinides that may have been in an elevated oxidation state. The final fraction of the eluate was treated first with ascorbic acid to reduce any iron and then with hydrofluoric acid. The uranium isotopes were next coprecipitated on neodymium fluoride. The neodymium was caught on a 0.1-µm filter, which was rinsed, dried, and then mounted on a planchet for alpha spectrometry. The minimum detectable activities (MDA) for uranium in urine using these procedures were 1.4×10^{-6} µg/l for ^{234}U and 0.03 µg/l for ^{238}U.

Results

Surgical Implantation

Two rats assigned to the DU group and one rat assigned to the Ta group did not survive implantation surgery. One of these rats expired during surgery, and the other two within 6 h after surgery. Necropsies indicated asphyxiation, suggesting that the animals received too much anesthetic. The other nine animals were alert and moving in the metabolic cages within 2 h after surgery. Figure 3 is a radiograph of the left rear leg of a rat implanted with four DU pellets; the right rear leg was also implanted with four DU pellets. The cylindrical shape and size of the pellets are similar to DU fragments observed in wounded soldiers (figure 1).

Locomotor Activity and Grip Strength

The locomotor activity of rats implanted with DU pellets was not significantly different from the activity of rats implanted with Ta, $p > 0.05$ (figure 4).

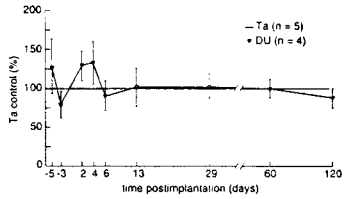

Fig. 4. Locomotor activity of rats surgically implanted with DU pellets expressed as percent of Ta control. Vertical bars represent the SEM (standard error of the mean).

Fig. 3. Radiograph of the left rear leg of a rat surgically implanted with four DU pellets (1 mm in diameter x 2 mm in length).

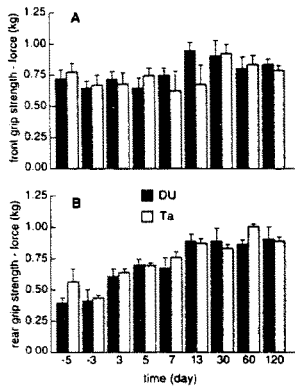

Fig. 5. (A) Forelimb grip strength of rats before and after receiving DU or Ta implants. Vertical bars represent the SEM. (B) Hindlimb grip strength of rats before and after receiving DU or Ta implants. Vertical bars represent the SEM.

Similarly, neither the forelimb nor the hindlimb grip strength of the two groups was different, $p > 0.05$ (figures 5a and 5b).

Body Weights, Food and Water Consumption, and Urinary Output

The body weights of the rats embedded with DU pellets were not different than the body weights of rats embedded with Ta, $p > 0.05$ (figure 6). In fact,

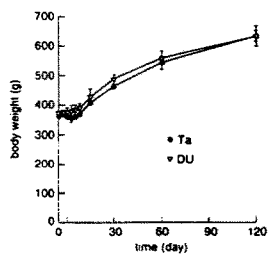

Fig. 6. Body weights of rats before and after receiving DU or Ta implants. Vertical bars represent the SEM.

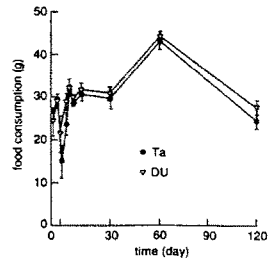

Fig. 7. Food consumption of rats before and after receiving DU or Ta implants. Vertical bars represent the SEM.

the body weights in both groups remained relatively stable for the first week following surgery and, as expected, increased throughout the study as observed in normal rats.

The food and water consumption for the DU- and Ta-implanted rats did not differ, $p > 0.05$ (figures 7 and 8). There was, however, a trend toward a decrease in water consumption for the Ta group and an increase in water consumption for the DU group.

There was a significant difference in the volume of urinary output between the DU and Ta groups. On the day of surgery, urine output for the Ta group decreased but did not change for the DU group, p

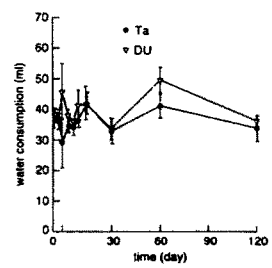

Fig. 8. Water consumption of rats before and after receiving DU or Ta implants. Vertical bars represent the SEM.

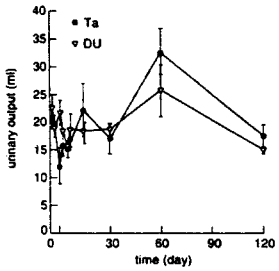

Fig. 9. Urinary output of rats before and after receiving DU or Ta implants. Vertical bars represent the SEM.

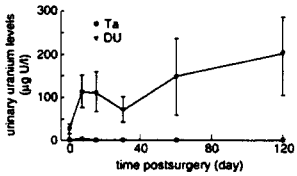

Fig. 10. Time course of uranium levels detected in the urine of rats implanted with either DU or Ta. Uranium concentration detected in the Ta group is at background levels. Vertical bars for the DU group (N = 4) represent the SEM. Urine for the Ta-implanted animals was pooled for uranium analyses.

<0.05 (figure 9). This decrease in the urinary output for the Ta group, however, was temporary and returned to baseline levels by day 3 after surgery.

Urinary Uranium Levels

Figure 10 illustrates mean uranium levels in the urine of DU-implanted animals and the pooled value of the uranium analysis for Ta-implanted animals after implantation surgery. Figure 11 provides the individual urinary uranium levels of the four DU-implanted rats. As expected, only background levels of uranium were detected in the Ta control group. In contrast, significant levels of uranium were detected within 24 h of DU implantation (mean = 28.69 ± 10.00, range = 14.21 to 56.99 µg U/l). By day 7 following surgery, uranium levels had increased nearly fourfold (mean = 111.86 ± 41.05, range = 56.38 to 233.91 µg U/l) and remained elevated at day 120 (mean = 204.56 ± 99.73, range = 35.01 to 458.53 µg U/l).

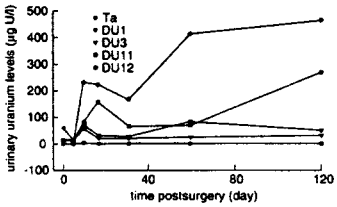

Fig. 11. Individual time courses of uranium levels detected in the urine of each rat implanted with DU. Data on Ta time courses are the same as in figure 10.

Discussion

The purpose of this study was to develop an animal model that could be used in future research to determine the health risks associated with DU fragment injuries. It was especially important to establish procedures in which DU exposure would produce urinary uranium levels comparable to those observed in soldiers wounded by DU munitions during the Persian Gulf War. Measured by these criteria, this initial study was successful. The average urinary uranium level in the rat 24 h after DU implantation was 28.69 μg U/l. This value is very close to the urinary level of 30 μg U/l reported for soldiers wounded during the Persian Gulf War and assayed 1 year after injury. Unfortunately, no bioassays were taken of any of the soldiers within the first year after DU injury so no direct time course comparisons can be made.

It should be emphasized that the urinary uranium levels in the rat did not reach asymptote until day 7 following DU implantation surgery and remained elevated throughout the study (figure 10). Although the data are preliminary, this finding has clinical significance because it indicates that soldiers with suspected DU fragment wounds should be monitored for uranium exposure for at least the first week after injury and perhaps even longer. Certainly a complete pharmacokinetic study should be conducted to definitively address this patient-monitoring issue (Daxon, 1993).

Although numerous studies have assessed the toxic effects of other forms of uranium exposure (Diamond, 1989, and Kocher, 1989, for the latest reviews of the literature), this is the first study that assessed the effects of intramuscularly embedded DU. The rat proved to be an excellent animal model for this purpose. It tolerated the surgical procedures for pellet implantation relatively well, as measured by both locomotor activity and grip strength (figures 4 and 5), both indices of quality of life for humans. Further, the lateral thigh muscle of the adult rat is large enough to implant at least four pellets (1.0 mm diameter x 2 mm length) into each leg (figure 3), with the possibility of as many as ten pellets. Moreover, the rat's lifespan of more than 18 months enables it to be used in chronic toxicity studies (Brady et al., 1989; Lang and White, 1994; Lumley et al., 1992; Lumley and Walker, 1986; Monro, 1993; Nohynek et al., 1993; Rao et al., 1990).

In conclusion, this study was successful in developing a rodent model that can be used to evaluate the biological effects of intramuscularly embedded DU fragments. However, the potential short-term and long-term health risks associated with DU exposure remain to be investigated. Certainly the behavioral, physiological, biochemical, and histological consequences of embedded DU are research areas of immediate concern. Equally important is identification of the health risks to the fetus exposed *in utero* to DU from fragments embedded in the mother before pregnancy (Angleton et al., 1988; Bosque et al., 1993; Domingo et al., 1988a, b, c; Paternain et al., 1989). This latter research area is especially significant considering that the placenta does not prevent cross-placental transfer of uranium (Durbin and Wrenn, 1976; Sikov and Mahlum, 1968). Moreover, fetal toxicity often occurs in the absence of maternal toxicity (e.g., Price et al., 1985). Regardless of the research strategy adopted, a coordinated interdisciplinary health hazard assessment is required to identify the potential medical risks that DU poses to our soldiers wounded by this unconventional munition.

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During the Persian Gulf War, 36 U.S. soldiers were wounded by depleted uranium (DU) munitions. Based on medical guidelines for conventional shrapnel injuries (nonradioactive), many DU fragments were left in soldiers. Unfortunately, health risks associated with embedded DU were unknown, and an animal model to investigate this did not exist. The purpose of this study was to develop an animal model to examine the health risks associated with DU shrapnel injuries. Twelve rats were surgically implanted intramuscularly with 8 DU pellets (1 mm in diameter x 2 mm in length) or 8 chemically inert tantalum (Ta) pellets of similar size. Urinary uranium levels were measured on days 1, 3, 7, 14, 28, 60, and 120 after implantation of DU pellets. Physiological and behavioral parameters, including locomotor activity, forelimb and hindlimb grip strength, food and water consumption, and urinary output, were measured 5 and 3 days before surgery and on days 1, 3, 7, 14, 28, 60, and 120 after surgery. Urinary uranium levels for Ta-implanted rats remained at background levels. In contrast, the average urinary uranium level for DU-implanted rats was significantly elevated on day 1 (28.69 μg U/l) after implantation and remained elevated until day 120 (204.56 μg U/l). There was no significant difference between DU- and Ta-implanted rats in any behavioral or physiological measures. Results indicate that the rat is an appropriate animal model for evaluating biological effects of embedded DU fragments.

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Attachment 9

March 1993

AFRRI

93-1 TECHNICAL REPORT

Assessment of the Risks from Imbedded Fragments of Depleted Uranium

LTC Eric G. Daxon, MS, USA
CPT Jeffery H. Musk, OD, USA

AFRRI TR 93-1

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AFRRI Technical Report 93-1

ASSESSMENT OF THE RISKS FROM IMBEDDED DEPLETED URANIUM FRAGMENTS

Prepared by:

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and
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March 1993

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BRP

27 March 1992

MEMORANDUM FOR DIRECTOR, PROFESSIONAL SERVICES, OFFICE OF THE SURGEON GENERAL (SGPS-PSP)

SUBJECT: Research Request; Health Effects of Depleted Uranium Imbedded in Tissue

Reference: Brigadier General Ronald R. Blanck (SGPS-PSP) letter of 26 February 1992

In response to your letter of 26 February 1992, subject as above, AFRRI has conducted a detailed review of the pertinent scientific literature regarding the health effects of depleted uranium (DU) fragments which are imbedded in tissue. In addition, we have consulted with a wide range of scientists with expertise in this area. A summary of our findings is attached.

It is clear from our analysis that there are several areas in which there is little or no scientific data which would enable more definitive risk assessments to be made. Nevertheless, in order to meet your operational requirements, attachment (1) addresses each of the issues raised in your letter. To address areas in which there remains substantial scientific uncertainty, attachment (1) also identifies specific research needs.

Based on available data, in almost all cases, we recommend that standard medical criteria should be used to determine the advisability of the removal of imbedded DU fragments without regard to the radiological characteristics of the fragment. More specific guidance is provided in attachment (1).

Point of contact is Lieutenant Colonel Eric G. Daxon, Chief, Operational Dosimetry Division, Radiation Biophysics Department, 301-295-2299.

Robert L. Bumgarner

ROBERT L. BUMGARNER
Captain, MC, USN
Director

Attachment:
as stated

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Assessment of the Risks From Imbedded Depleted Uranium Fragments

1. General

a. Authority

Letter, Office of The Army Surgeon General, (SGPS-PSP), Subject: Research Request; Health Effects of Depleted Uranium Imbedded in Tissue, 26 February 1992.

b. Mission

Assess the health risks associated with implanted DU fragments in the body to provide medical guidance for current and future patients with these fragments, and provide recommendations for future research.

2. Background

The primary conclusion of the review of the uranium literature[1-16] and discussions with others[17-26] in the field is that this situation is radiologically and toxicologically unique. The health risks of allowing depleted uranium (DU) fragments or any other radioactive heavy metal to remain imbedded in an organ have not been studied. The uranium literature reviewed is focused on inhaled or ingested uranium compounds. There is only one reported instance of a DU fragment accidentally injected subcutaneously in a patient. This case, as reported by Cole,[22,27] provides little information for long-term effects because the fragment was surgically removed after 8 months.[22,27]

3. Chemical Toxicity

The toxicological effects of uranium are well known. The target organ for uranium heavy metal toxicity is the kidney. The literature concerning the acute effects of uranium heavy-metal poisoning on the kidney is extensive and is summarized in recent articles by Leggett[12], Diamond[13] and Kocher.[14] While the generally accepted threshold level for kidney toxicity is from 1-3 μg of uranium per gram of kidney mass,[1,13,14] there is considerable discussion in recent literature concerning this limit.[12,13,14]

A review of uranium toxicology conducted by USAEHA[11] concluded that, while there was substantial toxicologic data for inhaled and ingested uranium compounds, there was little or no data for the metabolic behavior of implanted DU fragments. Key uncertainties include organ specific solubilities; organ specific retention functions; the metabolic impact of a source term other than the lung or GI tract; the potential for chronic kidney toxicity; the impact of fibrotic encapsulation, if it occurs; and the chemical form of the imbedded fragment.

The potential for wound contamination (the injection of small sub-millimeter fragments) and for spallation of small fragments from large fragments introduces two additional dispersal mechanisms - macrophage transport and the physical movement of intact particles by the blood stream. The impact of both is an increase in the rate at which uranium is deposited in the kidney and other organs. De Rey et al.[2] found insoluble UO_2 particles in the kidney 6 to 48 hours after injection of 4 to 40 micrometer diameter UO_2 micro-spheres into the subcutaneous tissue of the dorsal skin of female rats. This is significantly quicker than predicted by standard metabolic transfer models for insoluble compounds of uranium.

These limitations and uncertainties preclude a definitive assessment of the toxicologic risks of allowing DU fragments to remain in the body for extended periods of time.

4. Radiological Effects

The literature is extensive concerning the deterministic and stochastic effects of acute and chronic exposure to inhaled and ingested uranium compounds.[1,28-32] The lack of data for imbedded uranium fragments precluded a direct determination of the potential long-term radiological effects of these fragments. An estimate of the potential effects was obtained by reviewing the literature available for plutonium, Thorotrast, and hot-particles.

The plutonium (Pu) literature[33-44] reviewed also focused on inhalation and ingestion, but there were several studies that dealt with injected plutonium compounds. Lushbaugh[19,34,42] and Langham et al.[42] summarize the findings of studies of eight patients with injected plutonium. Lagerquist et al.[43] and Carbaugh et al.[44] discuss patients with plutonium contamination of puncture wounds. While these studies are somewhat useful, their usefulness is limited because the exposure duration was relatively short (the longest was 5-8 years), the particle sizes were small, and in each case the wounds were debrided to removed the injected plutonium. For both the animal and human studies, the plutonium injected was in the form of the fine particulates expected from injection wounds caused by contaminated, sharp tools.

The Thorotrast literature is extensive[1,34,47-62] and important because of the radiological similarities with the situation under study. Thorotrast is a colloidal suspension of thorium dioxide (ThO_2) that was used as an intravenously-injected contrast agent for radiographic imaging from the late 1920's until the late 1950's when its long-term radiologic health effects became apparent.[45,46] The Thorotrast literature provides the most definitive evidence that both clinically-significant deterministic and stochastic effects are possible from long-term irradiation of low dose-rate α and β emitting radionuclides.

However, the differences in particle size and chemical properties between Thorotrast and DU are significant enough to preclude a direct application of the data. The ThO_2 particles in Thorotrast were small enough (nanometers in size) to be engulfed by both the mobile and fixed macrophages in the reticuloendothelial (RE) system which led to a time dependent, selective concentration in the liver and spleen. This time dependence makes dose-dependent extrapolations from Thorotrast data to a DU fragment difficult. In addition, the selective retention by the RE system limited the exposure to the organs in this system.

Although directed specifically at the radiation effects on the skin of a highly radioactive, beta-emitting particle, the hot-particle research literature[63-70] provides valuable information concerning the differences between the highly nonuniform irradiation that results from an imbedded fragment and the results of the uniform organ irradiation upon which assessments of radiation risk are based. Specifically, the hot particle research sheds light on the relationship between the fraction of an organ system irradiated and the dose required to produce both deterministic and stochastic effects. The primary conclusion of this work is that the radiation risk of both endpoints is dependent upon dose and the number of cells irradiated.

Based upon this review, the following radiobiological effects are possible from imbedded DU fragments.

a. Granuloma Production

Cole's[22,27] experience and Lushbaugh's[19,34,42] work indicate that granuloma production in the muscle and fatty tissue will probably occur and will occur in all other tissue types that elicit similar cellular responses to foreign bodies. It is still questionable whether this encapsulation is permanent or will undergo the degradation-regeneration cycle suggested by Lushbaugh for the plutonium cases he studied.

The data to date are insufficient to allow a determination of whether Thorotrastoma-like growths are possible. A Thorotrastoma is a large growth that appears at the sites of extravascular Thorotrast with a latent period of from 5-35 years postinjection.[17,47,48,53,60] These granulomas grow to large sizes; in a few cases, clinically significant blood vessels and/or nerves were enveloped, resulting in fatal conditions.[24] While a strictly chemical causation cannot be dismissed, there is sufficient evidence to suggest a radiogenic mechanism.

b. Local Tissue Necrosis

The results of the Thorotrast, lung inhalation studies, and animal studies showed that local tissue necrosis followed by fibrosis was possible from the long-term irradiation of tissues by a low dose-rate, α and β emitting radionuclide.

Dose estimates made at AFRRI based upon published data[71,72,73,74] indicated that the probability of deterministic effects at distances greater than 1-3 mm from the surface of any fragment is negligibly small. Depth-dose calculations indicated that at the distances from the surface of all particle sizes studied (1-4 mm in diameter) the dose-rates were less than the repopulation dose-rate for non-proliferative cells provided by the ICRP[28] (1-5 mGy/d). The assumption in this analysis is that at distances greater than this, deterministic effects will not occur because cell repopulation will compensate for cell death for most tissue types. The most notable exception to this assumption is mature neural tissue, the neurons of which do not usually have a proliferative potential.

The clinical significance of necrosis at distances closer to the fragment is dependent upon the location of the fragment and the body's response to the fragment. Lushbaugh,[34] in his analysis of cases of injected plutonium, found that "...metallic plutonium implanted in the skin in minute amounts elicits a foreign-body reaction of the granulomatous type, which after subsiding in cellular activity becomes fibromatous." As time progressed, the collagen in the vicinity of the fragment liquified.

Lushbaugh speculated that the "pointed" nature of the granulomas he found and the fact that the granulomas became more superficial, suggested that the altered collagen might induce a cycle of inflammatory reaction followed by a reorganization and re-liquefaction of the collagen.

c. Whole-Organ Deterministic Effects

The potential for multiple fragments in a single organ led to the examination of the potential for whole-organ deterministic effects. A whole-organ deterministic effect is defined as one in which there is a clinically significant compromise of organ function due to the ionizing radiations emitted by one or more DU fragments.

The appearance of whole-organ deterministic effects from acute, high dose rate exposure is well documented. Mettler and Mosely,[75] Conklin and Walker,[76] and ICRP 41[28] provide excellent summaries with extensive bibliographies for whole-organ deterministic effects based primarily on examination of the Japanese atomic bomb survivors, radiation accident victims, and radiation therapy patients. Direct extrapolation from high dose rate, acute exposure to low dose rate protracted exposure is difficult because of the dose rate dependence of the threshold dose required to produce a deterministic effect.[28]

The results of inhalation studies with uranium and plutonium summarized in ICRP 31[30] and in other references[6,7,33,39] show that whole organ deterministic effects are possible from inhaled particulates. The Thorotrast studies[1,34,47-62] provide the clearest evidence that deterministic effects are possible from protracted exposures to low dose rate internal alpha emitting isotopes. These studies showed that both fibrosis of the spleen and cirrhosis of the liver could be related to the radiation emitted by the thorium dioxide (ThO_2) in the Thorotrast. The latent period for the onset of clinically significant liver cirrhosis was on the order of 20 years after Thorotrast administration.[57] The latent period for significant spleen fibrosis was not reported but is assumed to be comparable.

A dose calculation, made using similar methodology as described above, showed that the risk of whole-organ stochastic effects do not become significant until the fragment density in the organ exceeds one fragment per cm^3 of organ volume for the fragment sizes considered (1-4 mm diameter). At particle densities greater than this, the average dose rate in the organ will exceed the repopulation dose rate for non-proliferative cells.

d. Stochastic Effects

The standard ICRP stochastic-risk-estimation methodology[77] is directly applicable for systemic DU but can be used only with caution when assessing the risks of imbedded DU fragments. There are several unknowns that could cause this and similar procedures to either overestimate or underestimate the stochastic risks. Included are these specifics:

(1) The hot-particle research indicates that the risk from an imbedded fragment could be significantly less because fewer cells are irradiated. ICRP methodology assumes that the dose is uniformly distributed over all of the cells in the organ while a DU fragment will irradiate only the cells within a finite range of the fragment.

(2) The Thorotrast experience showed evidence that the constant irradiation of the same cell population could increase the risk by adding necrosis-regeneration as an additional cancer induction mechanism. This mechanism is not considered in the ICRP models or cancer risk estimates.

An estimate of the stochastic risk posed by an implanted, insoluble fragment was made by calculating the effective dose equivalent (H_E) for a range of fragments (1-4 mm) for each organ listed in ICRP 60.[31] The actual organ weights were used to calculate the dose as were the actual weighting factors (w_T). The calculation was performed assuming that alpha dose could be ignored because the energy of these particles will be expended producing lethal damage to the cells adjacent to the fragment and thus contribute nothing to the stochastic risk.

For the largest fragment size evaluated (4 mm), the highest H_E is in the thyroid because of its relatively small mass. In this case, H_E is 1 mSv/y (100 mRem/y). Using current risk estimates,[32] these values represent an increase in lifetime risk of fatal cancer of 0.3%. The value for other organs will be substantially lower because of their larger masses.

At this point in the discussion, it is important to recognize that this risk estimate is based upon a single, insoluble fragment imbedded in an organ and does not include the risk from systemic DU.

5. Conclusions

a. Chronic kidney toxicity is a potentially clinically significant health effect from imbedded DU fragments. While the toxicology of uranium in the kidney is well known, little is known about the toxico-kinetic behavior of imbedded uranium. This information is required to make definitive estimates of both the toxicological and radiological risk.

b. Based upon the literature reviewed, the potential exists for both stochastic and deterministic radiation effects from the long-term exposure to imbedded DU fragments.

(1) The most clinically significant, radiogenic effect is the potential for a Thorotrastoma-like growth to form at the site of single or multiple imbedded-fragments. The risk, if any, of this growth formation cannot be estimated. It is still uncertain whether this is a radiation effect or an effect due to the chemical nature of the Thorotrast colloid.

(2) The risks of fragments near neural tissues should be carefully assessed because of the nonproliferative nature of these cells.

(3) The potential does exist for whole-organ deterministic effects but only for organs with a large number of imbedded fragments. The point at which this effect is likely to occur requires a detailed estimate of the dose to the organ from all sources of DU. First order, dose estimates indicate that particle densities greater than one fragment per cm^3 of organ volume are required as long as the fragments are insoluble and there are no other sources of DU in the body. Fragment sizes considered in this calculation range in diameter from 1-4 mm.

(4) Using the best risk estimation procedures available, the estimated increased lifetime risk of fatal cancer from a single, insoluble, DU fragment in any organ is at most 0.3%. Scaling this risk for multiple fragments or fragments with systemic DU is difficult and should be done on a case-by-case basis after assessing the total DU content in the patient.

c. The toxicological and radiological unknowns are significant enough to warrant both follow-up of current patients and research to more clearly define the long-term risks associated with these fragments. This is especially important in light of the latent periods noted for both deterministic and stochastic radiogenic effects.

6. Clinical Recommendations

a. The primary clinical recommendation is to continue to use standard medical criteria for fragment removal. Include consideration of the potential impact of a granuloma or a Thorotrastoma-like growth as a part of the decision making process for fragment removal as well as the potential for tissue necrosis for fragments lodged in or within 1-3 mm of neural tissue.

b. Determine the total amount of DU in the patient and continue to monitor patients with confirmed DU fragments for signs of kidney toxicity and any of the radiological endpoints discussed. Monitoring is required primarily because of the toxicological but also because of the radiological uncertainties.

c. If fragments are excised based upon accepted clinical criteria, save the fragment and surrounding tissue for further analysis.

7. Research Recommendations

a. Epidemiology

Establish a registry that will allow for the efficient acquisition, cataloging, and analysis of the results of patient monitoring. This effort should include

(1) periodic examinations to watch for and catalogue signs of chronic kidney toxicity, granuloma induction, and cancer;

(2) periodic bioassay and whole-body counting to determine the metabolic behavior of the internalized DU and to provide information concerning the solubility of the DU; and

(3) a program for tissue analysis if fragments are subsequently removed for medical reasons.

b. Animal Model Experimentation

The primary objective of animal model experimentation is to allow a detailed observation and study of the pathology of these fragments under controlled conditions. The specific objectives of this experimentation should include the following steps:

(1) Accurately assess the toxico-kinetic properties of the various chemical forms of DU that could be imbedded in patients.

(2) Investigate whether there are DU specific cancer induction mechanisms similar those observed in Thorotrast-specific liver cancers.

(3) Determine whether the radiogenic deterministic effects noted above occur and, if they do, at what fragment densities and latent periods.

(4) Assess the impact of long-term, low-dose-rate irradiation of specific tissues such as those of the nervous system.

(5) Determine the potential for chronic nephrotoxicity as a function of organ in which the DU is implanted

(6) Conduct pathological studies of the tissue surrounding the fragment.

c. Dosimetry

Perform definitive absorbed dose calculations using advanced techniques to determine the significance of particle size and shape.

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Attachment 10

March 1993

AFRRI

93-2 TECHNICAL REPORT

Protocol for Monitoring Gulf War Veterans with Imbedded Fragments of Depleted Uranium

Project Leader
LTC Eric G. Daxon, MS, USA

AFRRI TR 93-2

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AFRRI Technical Report 93-2

PROTOCOL FOR MONITORING GULF WAR VETERANS WITH IMBEDDED DEPLETED URANIUM FRAGMENTS

Project Leader

Eric G. Daxon, LTC, MS, USA

March 1993

Radiation Biophysics Department

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The research requirements of this protocol will be approved by the appropriate DoD or VA Institutional Review Board for the Protection of Human Subjects.

Preface

The Army's Office of The Surgeon General (OTSG) initiated this effort to care for Desert Storm veterans with imbedded depleted uranium (DU) shrapnel. In February 1992, OTSG requested that the Armed Forces Radiobiology Research Institute (AFRRI) conduct a review of the potential health hazards (radiological and toxicological) of allowing DU shrapnel to remain imbedded throughout the lifetime of the soldier. Specifically, OTSG wanted to know if there was any reason to change the current surgical practice for fragment removal. No compelling evidence was found in the literature review[1] to change current surgical criteria for fragment removal. There were, however, significant uncertainties about the impact of DU fragments on the health of these patients that warranted long-term follow-up.

OTSG concurred with this finding and initiated action to implement this follow-up in the Army. The Department of Veterans Affairs (DVA) agreed to perform the follow-up for personnel discharged from the service. Both the DVA and OTSG requested AFRRI's assistance in drafting the protocol to be used in the follow-up effort.

A group of DoD physicians and scientists met at AFRRI to draft the protocol. At a subsequent meeting on 10 September 1992, a panel of experts reviewed and revised the draft protocol; representatives of the DVA and OTSG also attended this meeting. The protocol was once again reviewed and approved by the panel of experts.

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Protocol for Monitoring Gulf War Veterans With Imbedded Depleted Uranium Fragments

1. Objectives

This protocol will implement two separate but complementary efforts. The first is the clinical follow-up of Desert Storm patients with known or suspected imbedded depleted uranium (DU) fragments, DU contaminated wounds or significant amounts of inhaled DU. The second is the conduct of research into the toxicological and radiological effects of this unique exposure modality. Specifically, this protocol will provide the following:

a. Early detection of abnormalities related to the presence of DU so that prompt, efficacious treatment is effected if required. The study will also provide the scientific data required to fairly settle claims for compensation.

b. Treatment recommendations that will provide a firm clinical basis for fragment removal decisions and for decisions concerning the need for efforts to reduce the uranium in the body.

c. Quantification and documentation of the toxicological (heavy metal toxicity) and radiological (cancer and tissue necrosis) risks of imbedded uranium fragments by

(1) measuring and documenting uranium levels in each soldier using *in vivo* and *in vitro* measurement techniques,

(2) determining the parameters and models needed to translate uranium levels in the body into estimates of the increased cancer risk from this exposure,

(3) comparing the clinical course of the body's response to the DU fragments with that for other non-DU fragments to determine whether clinically significant differences exist due to either the chemical or radiological properties of depleted uranium, and

(4) determining the risk of chronic kidney toxicity due to the long-term chronic exposure to elevated levels of uranium.

2. Approach

The comparison of the clinical course of DU fragments with non-DU fragments will be made using a prospective study approach. The data from patients with internalized DU (the exposed population) will be compared to that from two unexposed populations: patients with fragments that are not DU and soldiers who were not wounded and not exposed to DU.

a. Exposed Population

Each crew member of the attacked vehicles is a candidate for inclusion in the exposed group. An initial check has revealed that there are approximately 22 soldiers whose records indicate that they have imbedded fragments that might be DU. There are an additional 13 soldiers who were wounded and hospitalized but were not specifically identified as having shrapnel. The remaining crew members (besides the 35 already discussed) were either not wounded during the incident or had minor wounds that were treated in the field. The latter two sets of soldiers might have inhaled uranium or experienced DU contamination of wounds or minor fragmentation wounds that were either not noticed or did not require extensive treatment.

The small size of the exposed population limits the study's ability to detect differences to only those effects where the differences between DU and non-DU imbedded fragments are large. For example, it is highly unlikely that definitive conclusions concerning cancer induction will be obtainable from the study. However, this approach will allow a direct comparison of differences that may exist in deterministic effects. Examples of such effects include differences in the body's propensity to encapsulate a DU fragment, the onset of local or whole-organ tissue necrosis, thorotrastoma-like growth induction, or the onset of chronic kidney toxicity. In addition, following a nonexposed group will provide information concerning nominal values for each metabolic value studied in protocol (e.g., normal concentrations of uranium in the body and body fluids as well as kidney function variations with age).

There are two criteria for including a soldier in the exposed group. First, the soldier must have been in or on the vehicle when the vehicle was struck by DU munitions. Second, the soldier must have internalized DU at levels that are high enough to cause the uranium in the urine either to exceed background levels of uranium excretion by a factor of four or to be detected by whole-body or partial-body counting. Uranium in urine measurements from the control group will establish the background levels for these two measurements. The exposed group has two subgroups.

(1) The first consists of those soldiers with internalized DU not from imbedded fragments. This group will consist of personnel who have DU from inhalation or through wound contamination. The data from this group will be used in both the metabolic modelling and chronic kidney toxicity studies.

(2) The second consists of soldiers with imbedded DU fragments. The data from this group will be used to compare the clinical course of DU fragments with non-DU shrapnel as well as to provide information for the metabolic modelling and kidney toxicity studies.

Determining the presence of DU shrapnel is not as straightforward as verification of the presence of internalized DU. The analysis is complicated because the penetration of an armored vehicle by a DU penetrator generates DU fragments, non-DU fragments, and fragments that are a mixture of DU and the other components of the vehicle. In addition, the size of these fragments will vary dramatically. In the two cases that have been studied so far, the fragment sizes ranged from just at the resolution limit of film radiography (approximately 0.5 mm) to 15 mm in diameter. Until more experience is gained, a patient is assumed to have DU shrapnel if shrapnel is detected radiographically and internalized DU is detected.

b. Special Study Group

A subset of the exposed group will be selected for inclusion in the special study group. This group will receive the more intensive testing required to determine uranium metabolism accurately, identify early signs of toxicity, monitor fragment dissolution rates, and determine how the uranium is partitioned in the body as a function of time. Evaluation of these variables will provide the information required to construct the metabolic models needed to assess the risks associated with internalized DU.

Criteria for selection include the presence of DU fragments in the body, uranium in urine levels that exceed 14 μg/d (10 μg/l[a]) and the soldier's availability for the intensive monitoring envisioned. Recognizing that participation in the special study group will require a significant commitment, soldiers will be selected who are highly motivated to participate and are located near testing facilities.

---

[a]*All conversions were calculated based on an assumed urinary excretion rate of 1.4 l/d.*

c. Nonexposed Groups

The data from the exposed population will be compared with that from two unexposed populations, which will serve as control groups for the study. The first control group, patients with non-DU fragments, is needed to determine whether the body's response to DU fragments differs from the body's foreign body normal response to shrapnel. The second group, unwounded and nonexposed, is needed to compare normal changes in kidney function with changes that might be due to the presence of uranium. The need for the second control group is based upon the assumption that non-DU fragments might cause changes in the parameters being measured.

Members of the non-DU fragment control population (the first control population) will be selected from veterans wounded in incidents not involving DU munitions. This will eliminate the possibility of a control group member having a small undetected DU fragment. Members of the unwounded and nonexposed control group will be selected from any unwounded population that does not meet the criteria for inclusion in the exposed population. In each case, groups will be appropriately matched (age, sex, smoking habits, similar Desert Storm experiences, etc.) with the exposed population.

d. Study Duration

At this point, it is difficult to determine the study duration, but the long latent periods for some effects[1] require that the study last at least 5 years. The study could extend for the lifetime of the members of the study groups.

3. Program Management

The program management group will supervise the initiation and conduct of the measurements, analysis, and documentation required by this project. The group will exercise oversight of each phase of the study and will control its overall direction. The group will consist of four representatives, at most, from the Department of Veterans Affairs, the Department of the Army and/or the Armed Forces Radiobiology Research Institute (AFRRI). The group has the following responsibilities:

a. Fiscal Management

The group will establish yearly budgetary requirements and maintain the records required to track the expenditure of funds during the fiscal year.

b. Patient Management

The group will identify the patients in each study group and establish mechanisms for patient tracking.

c. Data Gathering

The group will serve as the central repository for the data gathered in all phases of this study, including selecting the laboratories that will perform the required tests and developing and supervising the quality assurance program for these laboratories.

d. Data Analysis

The results of each required test will be submitted to this group for analysis and study. This group will be responsible for calculating and documenting dose estimates for each patient as well as determining if clinically significant changes had occurred.

e. Protocol Changes

The group will direct any changes required to meet the objectives of this study.

f. Treatment Recommendations

The group will be responsible for evaluating the data received to determine if an alteration in treatment is required and will make its recommendations to the attending physician.

g. Research Recommendations

The group will make recommendations for further research based upon their findings as appropriate.

h. Subject Matter Experts

To ensure the availability of the expertise required for this effort, the program management group will be augmented by a panel of subject matter experts. This panel will consist of physicians and scientists with expertise in radiation injury, epidemiology, health physics, uranium toxicology, and the laboratory procedures required by this protocol.

4. Patient Briefing

This briefing will be in sufficient detail to meet the requirements for informed-consent for participation in a human research project. Since long-term patient participation is key to the success of this study, it is recommended that this briefing be given by someone who will be with the project for an extended period of time and who has experience with this type of long-term study. This briefing will include a discussion of

a. the scope of the program and how the data will be used;

b. the tests and the frequency of testing, along with the risks entailed with participation and nonparticipation in the program;

c. the benefits and requirements of participation in the U.S. Uranium Registry;

d. procedures to follow for fragment removal. Standard medical guidelines should be used for decisions concerning fragment removal.[1] Once the removal decision is made, surgeons should use the procedures listed in paragraph 6.d. below for the removal of DU and non-DU fragments.

5. Protocol Test Requirements

a. Tests Required

(1) Table 1 outlines the required tests and test frequencies for each of the study populations. The specifics for each of the tests are explained in paragraph 6.

(2) The increased frequency of testing for soldiers with uranium concentrations in their urine at levels greater than 14 μg/d (10 μg/l) of urine (see Table 2) is based on the clinical need to monitor for signs of long-term kidney toxicity.

b. Modifications of the Test Protocol

(1) The program management group (see paragraph 3) will make modifications to the protocol as a whole or for an individual patient based upon its analysis of the results received. This re-evaluation should take place at least annually.

(2) The presence of symptoms in a patient (e.g., indications of toxicity, unusual growths, or other abnormalities) will trigger an immediate re-evaluation of the required tests and their frequency, and the need for medical/surgical intervention.

***Table 1.** Recommended Tests and Test Frequencies.*

TEST	Exposed Population Urinary Excretion <14 µg/d	≥14 µg/d	Special Study Group	Control Groups
Uranium in Urine	Annually	Table 2	Twice Weekly	Annually
Urine Chemistry	Annually	Table 2	Table 2	Annually
Uranium in Feces[1]			As Needed	As Needed
Tissue Analysis[2]	As Needed	As Needed	As Needed	As Needed
Whole Body and Regional Counting[3]	Initially	Initially	Biennially	Initially
Uranium in the Skeleton			Annually	Annually
Uranium in Blood			Quarterly	Annually
Blood Chemistry	Annually	Table 2	Table 2	Annually
Clinical Evaluation	Annually	Annually	Quarterly	Annually
Diagnostic Imaging[4]	Annually	Annually	Annually	Annually

[1]*Fecal samples will be performed whenever inhalation exposure is suspected. Control group fecal analysis will be used to provide estimates of normal uranium levels in feces.*
[2]*Tissue analysis will be performed on tissue samples taken as a result of a fragment removal procedure for both DU and non-DU fragments.*
[3]*Repeat after fragment removal or as required by the program management group.*
[4]*Radiographs are only required for personnel with imbedded fragments. This is not required for exposed or control group patients who do not have fragments.*

6. Test Specifications

This section describes the purpose and specifications for each of the tests required in the protocol. The specifications are designed to provide minimum test standards required to meet the objectives of the protocol. Selection of the laboratories where these tests are done will be made by the program management group based upon the guidance in this section and an assessment of the site's capabilities. The laboratories must meet the quality assurance requirements in paragraph 7 below. It is highly recommended that the same laboratory be used for each test whenever possible.

References 2 and 3 contain a partial listing of commercial and government laboratories with the capability for whole-body counting and for radiobioassay. While DoD laboratories are not specifically listed, the Army (U.S. Army Environmental Hygiene Agency) and the Air Force (Armstrong Laboratory) have the technology required to perform some of the radiobioassay procedures listed.

Table 2. *Test Frequency for Selected Tests as a Function of Initial Urine Uranium Concentration.*

Uranium Excretion Rate in Urine (μg/d)	*Test Frequency**	*Remarks*
14-50	*Quarterly*	
50-250	*Monthly*	*Potential for the onset of kidney toxicity.*
> 250	*At Least Weekly*	*Potential for kidney toxicity.*

**Tests include uranium in urine, urine chemistry, and blood chemistry.*

a. Uranium Concentration in Urine

(1) Purpose. This test will provide a direct determination of the uranium excretion rate which will be used for metabolic model construction and risk assessment.

(2) Specifications

(a) While a 24-hour urine sample is desirable, timed urine samples are acceptable. For 24-hour urine samples, it is important that all voids be collected. For timed urine samples, accurate accounting of the time period is a requirement and time periods of not less than 12 hours are recommended.

(b) Urine samples must be processed in a laboratory where the uranium measurement methods have a minimum detection limit of 0.4 μg of uranium per liter o urine or better. The laboratory must meet the quality assurance requirements listed in paragraph 7.

(c) Detailed sample collection and preservation procedures will be established by the laboratory performing the analysis.

(d) The nonexposed group will provide urine samples that will be used to establish background urinary excretion levels for uranium.

b. Urine Chemistry

(1) Purpose. The primary purpose of this test is to monitor the urine for signs of kidney toxicity or other abnormal changes in kidney function.

(2) Specifications. Urine chemistry to include a quantitative analysis of gamma-glutamyltransferase, beta-2-microglobulinuria, protein, amino acids, creatinine, phosphorus, and urinalysis (specific gravity, albumin, glucose, and microscopic sediment analysis) is required. Serum creatinine and creatinine clearance studies are needed to assess glomerular function and tubular integrity. It should be noted that these tests might underestimate filtration rate if tubular injury is present.

c. Uranium in Feces

(1) Purpose. This test is designed to give an indirect assessment of the uranium content in the lung and to assist in establishing lung clearance rates for metabolic modeling. This test should be administered only if significant lung contamination is suspected.

(2) Specifications. The specifications for fecal samples will be determined based upon the requirements for each test. As a general rule, the minimum detection limits for laboratories should be less than 3 μg of uranium per sample (less than 1 pCi per sample). Preservation and shipment requirements will be determined by the laboratory doing the analysis.

d. Tissue Analysis

(1) Purpose. This series of tests will be performed on tissues removed from a patient as a result of the patient's decision to have a fragment removed. The purpose of these examinations will be to determine

(a) the uranium content of the tissue (information will be useful in both metabolic modelling and risk assessment) and

(b) whether significant changes have occurred in the tissues surrounding the fragment. Thorotrast data indicate that long-term exposure to low-dose-rate alpha emitters can cause tissue fibrosis and necrosis with latent periods in excess of 5 years.[1]

(2) Specifications

(a) Surgical Removal of the Fragment. In addition to standard procedures, the following steps should be accomplished.

- Photograph the procedure. Of particular interest is evidence of total or partial fibrotic encapsulation; local tissue necrosis; growing granuloma; or if there is evidence of a breakdown, a formed fibrotic capsule.

- If the fragment is encapsulated, remove and save the intact capsule (with the fragment still inside) if possible. If the fragment must be removed from the capsule or if the capsule breaks during removal, document the capsular fluid appearance and volume. The capsule, capsular fluid, and any other tissue removed should be saved for histopathology and radioassay. Take careful note of the physical characteristics of the fragment upon removal. Specifics include color, shape, and any evidence that the fragment is breaking up. Color photographs of the fragment with a means of measuring its size are desirable. Seal the fragment in a plastic bag. Contact the program management group for instructions concerning the disposition of the fragment.

(b) Histopathology. The objective of this series of experiments is to determine if there are any unusual changes in cell structure of the surrounding tissue.

(c) Uranium in Tissue and Fluids. The uranium in retained tissue or fluids should be determined using techniques with the capability of detecting uranium levels on the order of 0.2 μg (0.06) pCi per tissue sample submitted. It should be noted that there are ultrasensitive fission-track counting techniques that can be used to detect 10^{-14} grams of uranium. At this level, the same tissue samples could be used for both histopathology and uranium concentration determinations.

(d) Sample Preservation Techniques. The sample preservation techniques used will depend upon which of the two procedures will be performed. At this point, it is uncertain if the same tissue sample can be used for both uranium concentration and histopathologic procedures. Once notified that a fragment will be removed, the program management group will decide which of the two procedures will be performed.

e. Whole-Body Counting, Regional-Area Counting, and Skeletal Uranium Determination

(1) Purpose. The combination of whole-body and regional-area counting allows for the quantification of the total amount of uranium in the body and the amounts of uranium in key locations in the body, using external measurement techniques. This information will be used in conjunction with urine and feces uranium contents to determine the metabolic models for uranium retention. The *in vivo* skeletal counting is an attempt to track uranium deposition in the skeletal system.

(2) Specifications

(a) The systems used must be capable of performing both whole-body and regional-area counting of uranium. Current systems can provide minimum detectable activities[b] (MDA) for regional-area counting of the lung on the order of at least 2 nCi (6 mg of DU) of DU in the lung by measuring the ^{234}Th progeny of ^{238}U.[3]

(b) Regional-area counting is required for the lungs, kidneys, liver, all wound or burn sites (regardless of how minor the wound), and of all areas with suspected DU fragments.

(c) Radiographs will be used to determine fragment location(s) so that estimates of tissue absorption and self absorption corrections for each of the areas counted.

(d) The *in vivo* skeletal-counting systems used should have an MDA of 10 nCi (30 mg) with adequate procedures for discriminating sources originating in the bone from those originating in the remainder of the body. The skeletal counting system developed at New York University is a good example of an acceptable skeletal-counting system.[3]

f. Uranium Concentration in the Blood

(1) Purpose. The test will measure the concentration of uranium in the blood by measuring the uranium concentration in the serum and cellular components of the blood.

(2) Specifications. Typical minimum detection limits for systems designed to measure the uranium content of the blood are on the order of 1 nano-gram of uranium per ml of blood. The laboratory selected to perform this test should have comparable efficiencies.

g. Blood Chemistry Evaluation

(1) Purpose. These tests are aimed at determining whether or not heavy metal toxicity and/or bone-marrow suppression has occurred.

(2) Specifications. SMA -12/20 or equivalent with complete blood count with differentials and platelet count.

---

[b]*The referenced work defined MDA as 4.65σ where σ is the standard deviation of the background count.*

h. Clinical Evaluation

(1) Purpose. Clinical evaluation will determine the presence of any abnormalities such as nodules or unknown growths in the vicinity of fragmentation wounds or a degradation in the viability of the tissues. Reference 1 contains a discussion of potential abnormalities and estimates of the latent periods associated with each.

(2) Specifications. Emphasis will be placed on organ/structure dysfunction related to the location of the fragment(s) and to the consequences of the potential radiological and chemical effects. Specific tests are determined by the location of the fragment(s). Particular attention will be given to detecting thorotrastoma-like growths at the site of fragment implantation. A thorotrastoma is a growth that appears at the sites of extravascular Thorotrast with a latent period of 5-35 years post injection.[4-8] In some instances, these granulomas grew to enveloped clinically significant blood vessels and nerves and, in some cases, proved fatal.

i. Diagnostic Imaging

(1) Purpose

(a) Determine the composition of the fragments in an attempt to differentiate between solid DU and aluminum DU mixtures.

(b) Determine the approximate size and anatomic location of the fragment(s) in the body with sufficient detail to make absorption and self absorption corrections for whole-body counting data.

(c) Detect or confirm the presence of the formation of the fibrous encapsulation or of a thorotrastoma-like growth.

(d) Determine if there have been any gross changes in the location or size of the fragment.

(2) Specifications

(a) Both magnetic resonance imaging (MRI) and radiographic imaging are required for patients with imbedded fragments. MRI will be used to detect soft tissue abnormalities (granulomas, thorotrastomas) in the tissues surrounding imbedded fragments. MRI will only be performed after determining that there are no ferromagnetic fragments or objects in the patient.

(b) Radiographic imaging will be used to determine the size and position of imbedded fragments with sufficient accuracy to detect changes in the location of the fragment and to make the tissue absorption and self-shielding corrections required for whole-body counting. It is anticipated that at least two projections will be required.

7. Quality Assurance

The long-term nature of this protocol mandates the implementation of a stringent quality assurance program to ensure the accuracy and precision of the data collected. The program management group will develop the details of the quality assurance program. The program must incorporate the following provisions:

a. The use of accredited laboratories when possible. The laboratory performing each of these tests must be accredited by an appropriate accrediting agency to perform the required test. The laboratory must have a viable quality assurance program that is in accordance with the guidance provided in References 9 and 10. The program management group will establish standards based upon the guidance in this protocol when such accreditation is not available.

b. The use of the same laboratory to perform each type of test when possible. Adoption of this strategy will ensure the consistency of the data and enhance the program management group's ability to monitor the quality of the data collected. When the same laboratory cannot be used, the program management group must develop procedures to ensure the comparability of the data generated.

c. The use of intercomparisons by the specific laboratories chosen. The quality assurance program must include either a program of intercomparisons with other laboratories or, ideally, comparisons with a national standard.

Standard records management quality control procedures will be implemented to ensure the accuracy of the records maintained by the program management group.

Glossary of Terms

Exposed population. There are two criteria for including a soldier in the exposed group. First, the soldier must have been in or on the vehicle when the vehicle was struck by DU munitions. Second, the soldier must have internalized DU at levels that are high enough to cause the uranium in the urine either to exceed background levels of uranium excretion by a factor of four or to be detected by whole-body or partial-body counting. Uranium in urine measurements from the control group will establish the background levels for these two measurements.

Nonexposed population. The nonexposed population is composed of two subgroups: The first subgroup includes those soldiers with fragment wounds that are known not to be DU. The second consists of those soldiers who were not wounded and do not have internalized DU. DU is considered not to be present in significant amounts if the uranium concentrations in the urine are less than four times the background level and the results of whole-body or partial-body counting are negative.

Minimum detectable amount (MDA). The smallest amount of a substance that can be detected with a probability β of nondetection (Type II error) while accepting a probability α of erroneously deciding that a positive (non-zero) quantity is present in an appropriate blank sample (Type I error).[9] For this protocol both α and β are set at 0.05.

Program management group. A multi-disciplinary team that will oversee the implementation of the protocol and evaluate the results and direct changes in the protocol as required.

Radiobioassay procedure. For the purposes of this protocol, a radiobioassay procedure is any procedure used to measure the uranium in the body (whole-body counting) or in biologic material excreted or removed from the body for the purposes of estimating the uranium content in the body.[9]

Special study group. A subset of the exposed group that will receive the more intensive testing required to accurately determine uranium metabolism, to identify early signs of toxicity, to monitor fragment dissolution rates, and to determine how the uranium is partitioned in the body as a function of time.

Thorotrastoma. A large growth that appeared at the sites of extravascular thorotrast in patients injected with thorotrast, a thorium containing radiographic contrast agent. The growth appeared with a latent period of 5-35 years postinjection. These granulomas grew to large sizes and some enveloped clinically significant blood vessels and nerves and, in some cases, proved fatal. Thorotrastomas are discussed in the references.

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Col. DAXON. Yes, sir.

Mr. SHAYS. OK. Let me say that is there a question that we should have asked that you would have liked us to have asked? Yes.

Mr. ROSTKER. Two points. On the issue of the MOPP gear, you asked specific questions about specific reports that I do not know of, and I will check; but let me say there was critical assessments of every piece of the MOPP gear and that we have moved substantially over time to a new mask. There was testimony today about the layer of carbon, the newest overgarments following the British have the carbon integrated into the fiber. It lasts longer. The issue today about it lasting only 12 hours was 12 hours in a saturated environment. The garment itself can last for a much longer period of time.

Mr. SHAYS. Dr. Rostker, I need to say on the record that I am aware of two reports that are classified that I would like to talk to you about.

Mr. ROSTKER. OK.

Mr. SHAYS. And, Dr. Murphy.

Mr. ROSTKER. I have one additional thing, if I might. You also raised the question of the alarms.

Mr. SHAYS. Yes, sir.

Mr. ROSTKER. And we just put together a small briefing for the PAC that we gave them earlier this week on the M–8 alarm, and I would like to make that available to you.

Mr. SHAYS. What is the bottom line?

Mr. ROSTKER. The bottom line is that a known interferant that would set off the alarms and provide false positives includes gasoline vapors and diesel-fuel exhaust. So the description of them turning it on on the trucks in a convoy and the alarms going off all the time is absolutely predictable, given the known interference.

Mr. SHAYS. Let me just say, though, Dr. Rostker, that we have testimony that far more of them occurred after the war than before and that there was no noticeable difference in terms of environment.

Mr. ROSTKER. Except for the oil fires and the like, which also were involved. The proper procedure is to, if an M–8 goes off and they MOPP'd as they described, is then to do an all-clear based upon a 256 kit. We are investigating all of the 256-kit positives that we can find.

Mr. SHAYS. I feel that I need to state on the record that individuals have contacted this committee who will—I guess I cannot say that; they have not done it yet. Let's just leave this issue open. OK? We will leave it like that.

Mr. ROSTKER. Yes, sir.

Mr. SHAYS. Dr. Garthwaite.

Dr. GARTHWAITE. Sir, just a couple of things. To the veterans out there, I would urge them to get a Persian Gulf Exam if they have not gotten one; and in relation to their frustration, I will remind all of us that we declared war on cancer, and although we have won some skirmishes, that is an ongoing war, and it has been going on for many years.

The science is very difficult, very complex, and not a simple process, and it is not for lack of trying. And we are all frustrated. I

think many of us in medicine are in medicine because we hope to be able to make a difference and to get some answers for some of these diseases.

Second, I think, we appear before you with a great sense of humility. When I went to medical school, ulcers were definitely caused by too much acid. Today, we can tell you that they are definitely caused by bacteria. So what is very clear today may not be as clear in the future.

And the third thing is about peer review. I would just like to say that it is human beings doing the best they can to judge other human beings, and I think that the point that was made, that there may be somewhat of a systemic bias of peer review for new and more radical ideas is very possible and plausible, and we should take that into account as we think about peer review.

Mr. SHAYS. Thank you. Thank you. Let me just thank our court reporter, Ted Fambro. Also, I would like to thank Denise Nichols for taking care of our veterans, picking them up—the four of them did not live here—and making sure they had a square meal last night.

I would also like to thank my director of this committee, Larry Halloran, and Bob Newman, who staffs and deals with Gulf war illnesses; also, Mr. Sanders' staff, Don Edwards and Cynthia Welgess; and also the minority staff, Cherri Branson; and to say to the witnesses you have been very helpful. You have been extraordinarily patient and tolerant, and it certainly speaks well for your concern about this issue, and we do appreciate that very much.

With that, we will call this hearing adjourned.

[Whereupon, at 4:35 p.m., the subcommittee was adjourned.]

[Additional information submitted for the hearing record follows:]

TESTIMONY FOR

HOUSE SUBCOMMITTEE ON HUMAN RESOURCES
AND INTERGOVERNMENTAL RELATIONS

HEARING: JUNE 26, 1997

OIL FIRES, PETROLEUM AND GULF WAR ILLNESS

TO:
THE HONORABLE
CHRISTOPHER SHAYS
CHAIRMAN
House Subcommittee on
Human Resources &
Intergovernmental Relations
B372 Rayburn H.O.B.
Washington, DC 20515

FROM:
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- From: Presidential Advisory Committee on Gulf War Veterans' Illnesses Final Report (Washington, DC: U.S. Government Printing Office, December, 1996), pp. 120-123.

Exhibit 2 . . . . . page 14

Minutes of Veterans Administration Expert Scientific Panel meeting, 2/27-28/95, p.77.

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The Persian Gulf Veterans Coordinating Board posting to the internet, dated 7/12/95.

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- Institute of Medicine conclusion on Persian Gulf oil well fires and spills.
- From: IOM: Health Consequences of Service During the Persian Gulf War; Recommendations for Research & Information Systems, National Academy Press, Washington, DC, 1996, pp. 44-46.

Exhibit 5 . . . . . page 21

U.S. Congress Office of Technology Assessment, The Department of Defense Kuwait Oil Fire Health Risk

Assessment (The "Persian Gulf Veterans' Registry"), OTA-BP-H-138 (Washington, DC: September 1994).

Exhibit 6 . . . . . page 33

DSB (Defense Science Board). 1994. Final Report: Defense Science Board Task Force on Persian Gulf War Health Effects. Washington, D.C.: Office of the Under Secretary of defense for Acquisition and Technology.

Exhibit 7 . . . . . page 36

- Health advisory, Kuwait Oil Fires.
- From: USAEHA (U.S. Army Environmental Hygiene Agency). 1992. Interim Kuwait Oil Fire Health Risk Assessment, No. 39-26-L192-91, May 5-September 15, 1991. Aberdeen Proving Ground, Md.: USAEHA pp. H-24-H-26.

Exhibit 8 . . . . . page 39

- Testimony of Dr. Jack Heller on air pollution from oil field fires.
- From: Tapes of the NIH Workshop, The Persian Gulf Experience and Health, April 27-29, 1994.

TESTIMONY OF CRAIG F. STEAD

House Subcommittee on Human Resources and Intergovernmental Relations

Hearing of June 26, 1997

OIL FIRES, PETROLEUM, AND GULF WAR ILLNESS

I. INTRODUCTION

For many, the Kuwait oil fires were a strong and lasting impression of the Gulf War. However, the Gulf War was about oil in all aspects. Troops fought in the oil fields, lived in the oil fields, and were constantly exposed to smoke and oil from the oil field fires. Oil and soot rained from the sky. Troops lived in oil soaked uniforms, drank oil contaminated water, showered in oil contaminated water, and had oil in their food.

Since the War, veterans have been ill with multiple symptoms characterized as Gulf War illness. Government investigations into Gulf War illness have dismissed the oil fires and all related petroleum exposures as a cause of Gulf War illness.

Does the government have a scientific basis for dismissing the oil fires and petroleum as a cause of Gulf War illness? I believe not, based upon my 10 years research into petroleum toxicology and 6 years research into the cause of Gulf War illness. A careful reading of the government documents on Gulf War illness and the pertinent scientific papers relating to oil fires and petroleum clearly shows both the oil fires and petroleum must be considered causes of Gulf War illness.

The government studies done on the oil fires, petroleum and Gulf War illness ignore the testimony of veterans on oil fire and petroleum exposure. The government studies ignore a large body of medical and scientific literature showing oil is toxic, causes cancer, respiratory problems, skin rash, fatigue, and other symptoms. The government studies are seriously flawed in methodology, particularly the final Army Kuwait Oil Fire Health Risk Assessment. My testimony will address the connection between the oil fires, petroleum and Gulf War illness and the deficiencies of the government studies.

II. OIL FIRES

A. What the Veterans reported

"When they blew the oil-well fires, it was unlike anything I ever seen in my life. It was like being in a locked closet in the dark. . . . The deal with the oil-well fires is – we are in the middle of 500 oil- well fires. And the only thing that they gave us was a white t-shirt and put it over your face.[1]"

"He was in the center of the oil fires in Kuwait City with no capability of distinguishing the sun from the moon for the first six weeks after the liberation of Kuwait. His body was so oil and soot covered that a black watch band was camouflaged on his wrist.[2]"

"Others report very significant exposures to oil and oil smoke. They report moving through the oil well area and being covered in a thick coating of fuel and of oil.[3]"

B. What the Army found

An early report found "the ground around the sampling site was black and the roof of the clinic was covered by sticky oil droplets and soot from the nearby fires and the odor of oil and petroleum was pervasive[4]."

Yet, the conclusion from the final 1994 report[5] was that in the spring and summer of 1991, Persian Gulf air, despite its appearance, was about as dirty as that of Houston and Philadelphia.[6] Is Houston and Philadelphia air so polluted you cannot see the sun during the day? Does this point to a basic flaw in the Army study of Gulf War air pollution?

---

[1] Testimony of Scott Russell, Presidential Advisory Committee hearing of 8/6/96.

[2] Testimony of Herb Smith, NIH Workshop, The Persian Gulf Experience and Health, August 27-28, 1994.

[3] Minutes of VA Expert Scientific Panel meeting of 2/22-23/94, p. 48.

[4] USAEHA (U.S. Army Environmental Hygiene Agency). 1992. Interim Kuwait Oil Fire Health Risk Assessment, No. 39-26-L192-91, May 5-September 15, 1991. Aberdeen Proving Ground, Md.:USAEHA p. B-44

[5] U.S. Army, Environmental Hygiene Agency, Final Report: Kuwait Oil Fire Health Risk Assessment, 5 May- 3 December, 1991, Report No. 39-26-L192-91, February, 1994.

[6] Presidential Advisory Committee on Gulf War Veterans' Illnesses Final Report (Washington, DC: U.S. Government Printing Office, December, 1996, reference #61B.

III. THE ARMY KUWAIT OIL FIRE HEALTH RISK ASSESSMENT

A. What the Army concluded

In 1994, the Army issued the final Kuwait Oil Fire Health Risk Assessment (HRA). The HRA's purpose was to characterize the cancer and noncancer health risks to Department of Defense (DoD) troops and civilian employees exposed to the Kuwait oil fires during the Gulf War. The HRA concluded the potential for significant long-term adverse health effects from exposure to the oil fires for DoD troops or civilian employees was minimal. The HRA also concluded that the cancer risk from exposure to the oil fires was minimal and well within EPA guidelines for acceptable cancer risk. Finally, the HRA concluded that the ground level pollutant concentrations were lower than anticipated.

B. Who relied on the Army's conclusion

The HRA is a primary document used by government agencies in dismissing the oil fires as a cause of Gulf War illness. The agencies who dismissed the oil fires as a cause and cites from their reports which demonstrate their dismissal are the Presidential Advisory Committee on Gulf War Veterans' Illnesses (Exhibit 1), the Veterans Administration Expert Scientific Panel (Exhibit 2), the Persian Gulf Veterans Coordinating Board (Exhibit 3), the Institute of Medicine Committee to Review the Health Consequences of Service during the Persian Gulf War (Exhibit 4), the U.S. Congress Office of Technology Assessment (Exhibit 5), and the Defense Science Board (Exhibit 6).

C. How the 1994 Health Risk Assessment is flawed and invalid

To be valid, a health risk assessment must include all toxic exposures. Exposure used in the risk assessment must reflect the actual exposure encountered. The HRA has the following three serious flaws.

First, the Health Risk Assessment used Persian Gulf air pollution data gathered May through November, 1991. Air pollution from the oil field fires during this time was much less than during the Gulf War (February and March) for the following reasons. The months of May through November have the Shamal winds blowing from the Northwest causing the smoke plume from the oil field fires to disperse widely and ascend to great heights. During the Gulf War (February and March) low wind speeds and air inversions were common. Under these conditions the smoke plume was on the ground, creating high localized levels of air pollution to which the troops were exposed.

Exhibit 7 is a U. S. Government 1991 winter air pollution health advisory for Kuwait City. Expected high levels of air pollution caused by slow winds and air inversions were the reason for the health advisory. This advisory contains several important points. Severe winter air pollution was expected despite half of the oil fires

having been extinguished. Also, the northern and southern oil fields on fire were expected to have higher levels of air pollution than Kuwait City. Further, for November and December, a third of the days were expected to have stagnant, high pollution weather conditions. Finally, the Kuwait Government did not want to provide public warning of dangerous air pollution conditions.

Air pollution expected in this 1991 health advisory would be much less than the pollution encountered by Gulf War troops because half the oil fires had been extinguished.

The primary author of the 1994 final Health Risk Assessment, Dr. Jack Heller, has testified the Gulf War months had much more ground impact of the oil fire smoke than when the Army air pollution monitoring was done. Dr. Heller did not factor into the HRA the high levels of war time air pollution to which the troops were actually exposed (Exhibit 8).

The second serious flaw is the Health Risk Assessment used EPA methodology for Super Fund Sites. This EPA methodology is **not** appropriate for the Gulf War risk assessment as this methodology assumes **only** exposure to volatile vapors and contaminated soil. Also, this methodology does **not** assess health risk from massive air pollution (oil mist, smoke and soot) as found in the Gulf War.

The third serious flaw in the Health Risk Assessment is liquid petroleum (as crude oil, diesel fuel, or oil mist) was not included as a troop exposure. The HRA neglected troop exposure to the oil rain from the oil field fires. The HRA neglected oil in contaminated drinking and shower water. The HRA neglected oil mist from military smoke generators. And finally, the HRA neglected oil in food from cooking in oil contaminated water.

Liquid petroleum is a known toxin that causes cancer, respiratory problems, skin rash and other symptoms associated with Gulf War illness. For the HRA to be valid, this important toxin exposure must be included in analyzing health risk.

D. Conclusion on the final 1994 Health Risk Assessment

The Health Risk Assessment is seriously flawed in its basic methodology. As a result, the conclusions of the Health Risk Assessment are invalid. The Health Risk Assessment is a **primary** document relied upon by the Department of Defense (DoD), Presidential Advisory Committee (PAC), Veterans Administration (VA), and the Institute of Medicine (IOM) in concluding the oil field fires presented no health hazard to the troops. **Since the Health Risk Assessment's conclusions are invalid, the conclusions of the DoD, PAC, VA and IOM which dismiss the oil fires as a cause of Gulf War illness are also invalid.**

IV. PETROLEUM CAUSE OF GULF WAR ILLNESS

A. Petroleum Exposure

Troops in the Gulf War were continuously exposed to petroleum in a liquid and vapor form. This exposure was unique to the Gulf War experience. Petroleum exposure occurred in several ways. Crude oil rained from the sky from the gushing and burning oil wells[7,8]. Oil contaminated the drinking and shower water[9] and oil contaminated the food. Diesel fuel was sprayed on the roads, campgrounds, and parking areas to suppress dust [10] and diesel refueling spills and splashes occurred[11].

As a result of the oil rain and lack of laundry facilities, troops lived in oil soaked uniforms for days and weeks at a time[12]. The uniforms remained oil contaminated after washing as the wash water was contaminated with oil. Thus, petroleum was inhaled, ingested and absorbed through the skin of the exposed troops, which constitutes a major exposure to this significant toxin..

B. Petroleum toxicity

Petroleum is known to cause cancer[13]. Research has shown Kuwait crude oil is carcinogenic to animals[14]. Skin painting tests on mice are a scientific method for cancer testing. Skin painting tests of Kuwait crude caused tumors in 38% of the mice.

Inhalation of liquid petroleum causes a respiratory disease called lipoid pneumonia. Lipoid pneumonia, caused by petroleum droplets in the lung, is a

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[7] Hobbs PV, Radke LF. Airborne Studies of the Smoke from the Kuwait Oil Fires. Science. 1992;256:987-991.

[8] Presidential Advisory Committee on Gulf War Veterans' Illnesses Final Report (Washington, DC: U.S. Government Printing Office, December, 1996), p. 122.

[9] Veteran testimony to the Presidential Advisory Committee and personal communications by Craig Stead with veterans.

[10] Presidential Advisory Committee Final Report, December, 1996, minutes of VA Expert Scientific Panel.

[11] Minutes of VA Expert Scientific Panel meeting of 2/22-23/94, p. 47.

[12] Testimony of Herb Smith, NIH Workshop, The Persian Gulf Experience and Health, August 27-28, 1994, minutes of VA Expert Scientific Panel meeting of 2/22-23/96, p. 47.

[13] IARC. 1989. IARC monographs on the evaluation of carcinogenic risks to humans. Vol. 45: Occupational exposures in petroleum refining: Crude oil and major petroleum fuels. Lyon, France: World Health Organization, International agency for Research on Cancer.

[14] Coomes RM, Hazer KA. Comparison of the carcinogenic potential of crude oil and shale oil. From: Proceedings of the Symposium, The Toxicology of Petroleum Hydrocarbons, MacFarland HN, Holdsworth CE, et al. editors, American Petroleum Institute, 1982, pp. 208-224.

progressive disease which causes shortness of breath, cough and other symptoms. It is misdiagnosed as: Parkinson's Disease, Cerebral palsy, Hemiplegia, Amyotrophic lateral sclerosis, Degenerative disease of central nervous system, Progressive muscular dystrophy, Rheumatoid arthritis, Hypertrophic arthritis, Arteriosclerotic heart disease, Hypertensive cardiovascular disease, and Generalized arteriosclerosis[15]. Many of the clinical aspects of Gulf War illness are listed above and can be related to petroleum inhalation.

A recent scientific paper describes the cancer causing properties of crude oil[16]. In this paper, the author makes the following statements concerning petroleum toxicity.

> **Crude oil is known to be carcinogenic.** . . . The examination of the carcinogenicity of aromatic and saturated compounds of crude oils . . . demonstrates the high degree of carcinogenicity, with relatively short latency periods, of all fractions tested. **The percentage of animals that developed cancers from the various fractions was between 22% and 98%, a remarkable phenomenon.**
>
> **Crude oil and its vapors emitted into the atmosphere and environment from oil spills and other sources . . . expose humans and the environment to highly toxic and carcinogenic chemicals.**

C. Petroleum cause of Gulf War Illness

Gulf War veterans had a major exposure to petroleum. Petroleum was inhaled, ingested, and absorbed through the skin. A telephone study done on 10,051 sick veterans found:

> **Specific to the oil in the environment there, those breathing or enveloped in oil fire smoke was 96 percent; within clear visual area of the oil fires was 90 percent; worked in, lived in, or made travel through the burning oil fields was 72 percent; washed in water with an oily sheen was 68 percent. Those having oily taste to their food was 66 percent, and those with oily taste to the drinking water was 65 percent[17].**

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[15] Volk BW, Nathanson L, Losner S, Slate WR, Jacobi M. Incidence of lipoid pneumonia in a survey of 389 chronically ill patients. Am J Med. 1951; 10: 316-24.

[16] Mehlman MA. Dangerous and cancer causing properties of products and chemicals in the oil refining and petrochemical industry: Part XI - Carcinogenicity and environmental hazards of crude oil, gasoline, and its components. From: A comprehensive Approach to Problems with Oil Spills in the Marine Environments: The Alaska Story, V. Molak, W. Davis-Hoover, et al. editors. Princeton Scientific Publishing, Princeton, NJ, 1992, pp. 31-55.

[17] Testimony of Debbie Judd, Presidential Advisory Committee Hearing of 11/07/95.

Petroleum in the form of crude oil and refined products (diesel fuel, kerosene) is known to cause cancer, respiratory problems, skin rash, fatigue, and other symptoms. Many symptoms of Gulf War illness are consistent with exposure to petroleum.

Petroleum has been dismissed as a cause of Gulf War illness by all government agencies investigating Gulf War illness. There is no scientific basis for dismissing petroleum as a cause. To the contrary, a large body of scientific information exists which clearly shows petroleum must be considered a cause of Gulf War illness.

At this time, no research is being done on diagnosis and treatment of petroleum illness in Gulf War veterans. It must be emphasized methods **do** exist for diagnosing petroleum illness[18] and therapies **do** exist for attenuating the symptoms of petroleum illness[19].

V. CONCLUSION:

Burning oil fields, oil slicks, oil rain, oil in the water, oil in the food -- that was the combat environment in the Gulf War. Congress was justifiably concerned for the health of the veterans from exposure to the oil fires and smoke. Congress passed Public Law 102-190 in December 5, 1991 requiring the Secretary of Defense to:

a. Establish and maintain a special record relating to members of the Armed Forces who, as determined by the Secretary, were exposed to the fumes of burning oil in the Desert Storm Theater of Operations during the Persian Gulf Conflict, and

b. Submit to the Congress the results of all ongoing studies on the health consequences (short- or long-term) of members of the Armed Forces who were exposed to the fumes of burning oil in the Desert Storm Theater of Operations during the Persian Gulf conflict. This report should also address the need for any additional studies relating to this exposure.

Has the DoD responded to the Congressional directive in this Public Law? The record shows five years have passed and many tax dollars have been spent on meetings, hearings, studies, and data collection. Yet, there is not one study, research project, or report that addresses the **true** troop exposure to the oil fire smoke, and the

[18] For those veterans suffering from petroleum illness, visible droplets of oil should be present in the target organs (lungs, spleen, kidneys, liver). These visible oil droplets can be seen under electron microscope. The oil droplets can be extracted and analyzed using modern laboratory techniques. For those veterans with a Kuwait crude oil exposure, methods exist to match the oil extracted from target organ to Kuwait crude oil. Source, Craig Stead literature search.

[19] Tested therapy for petroleum illness consists of daily oral steroids. Other experimental therapies include washing of the lungs to remove petroleum and use of certain food groups to assist in metabolizing and excreting the petroleum toxins. Source, Craig Stead literature search.

troop exposure to petroleum inhalation, ingestion, and absorption through the skin. No work is being done on diagnosis and treatment of petroleum illness.

The VA Expert Scientific Panel has held 10 meetings generating over 1000 pages of minutes, executive summaries, and recommendations for actions on the part of the DoD, VA, and for research. Within the Scientific Panel minutes are only a few brief comments devoted to the oil fires and no discussion of the massive exposure of the troops to petroleum. Dr. Eula Bingham, Chair of the VA Expert Scientific Panel, has worked many years on petroleum carcinogenicity, sponsored by the American Petroleum Institute. Yet Dr. Bingham has never raised the issue of petroleum exposure, petroleum toxicity, and its possible relationship to Gulf War illness.

The Presidential Advisory Committee heard extensive testimony from Veterans on their exposure to oil fire smoke and petroleum. Yet the PAC has dismissed both the oil fires and petroleum as a cause of Gulf War illness based upon DoD and industry information. The PAC references supporting this dismissal lack a proper scientific and peer reviewed approach.

In conclusion, the government studies and reports have no foundation to dismiss either oil fire smoke or petroleum as a cause of Gulf War illness. This major and common exposure to those who served in the Gulf War must be investigated as a highly likely cause or contributor to Gulf War illness. It is time to initiate the proper studies and research into the connections between the oil fires, petroleum and Gulf War illness.

VI. QUALIFICATIONS OF CRAIG F. STEAD

I have a masters degree in Chemical Engineering from Cornell University and am a Registered Professional Engineer in Vermont and Massachusetts. For 12 years I have researched petroleum toxicology and the health effects of petroleum on animals and humans. My personal library on the subject contains over 1000 medical, technical and scientific papers and books.

I have served as technical advisor to the U. S. EPA, the State of Wisconsin and State of Vermont on petroleum contamination of drinking water. As a result of my technical advice, the State of Wisconsin issued a health advisory on oil leaking submersible well pumps. I also have served as an expert witness to the Vermont Environmental Board on the composition, toxicity and human health effects of diesel exhaust.

Since 1984 I have suffered from petroleum illness from drinking and showering in oil contaminated water. Many of my petroleum illness symptoms are the same as those reported by Desert Storm veterans. My illness initiated my research into petroleum illness, its causes, diagnosis and treatment.

I have provided testimony on petroleum illness and how it relates to Gulf War syndrome to Congressman Joseph Kennedy in 1992, to Senator James Jeffords in 1992, to the Presidential Advisory Committee in 1996, and provided Fort Detrick a proposal on Petroleum Toxicity and Petroleum Induced Illness in 1995.

Thank you for this opportunity to present testimony on oil fires, petroleum and the connection to Gulf War illness. I stand ready to initiate and assist this vital work for the health of the Gulf War veterans.

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and relatively soluble particles are cleared to blood and can affect kidney toxicity.[109] Less soluble particles can remain in the lung longer and in theory could pose a radiological hazard. The U.S. Army has conducted tests to characterize aerosols associated with DU munitions impacts with armor and with accidental DU munitions fires; it concluded a service member's risk exceeds civilian safety standards only when he or she is inside a vehicle when it is penetrated by DU munitions.[110,111] The adequacy of the research supporting this conclusion has been questioned by some reviewers.[112,113]

It is unlikely that health effects reported by Gulf War veterans today are the result of exposure to DU during the Gulf War.

No studies of long-term human health effects of uranium metal implanted in tissue exist. Nevertheless, toxic effects are likely to be similar to the kidney toxicity observed from inhaled or ingested uranium. To date, VA has reported no kidney toxicity among soldiers wounded by DU fragments in friendly fire episodes.[114] VA currently monitors the health of approximately 30 veterans suspected of retaining embedded DU fragments, and the U.S. Army Medical Research and Materiel Command is funding animals studies to investigate the health hazards associated with short- and long-term exposure to DU metal fragments.[115]

What do we conclude about the risks of DU to Gulf War veterans? The Committee concludes it is unlikely that health effects reported by Gulf War veterans today are the result of exposure to DU during the Gulf War. Since uranium is a potential carcinogen, it is possible that exposure to DU during the Gulf War could lead to a slight increase in the risk for lung cancer after decades following the end of the war.

Oil-well Fire Smoke

At the end of the Gulf War, more than 600 Kuwaiti oil wells and several pools of spilled oil were left burning after being ignited by retreating Iraqi troops. Huge, dramatic plumes of billowing smoke from these fires rose high into the atmosphere. Occasionally the smoke remained low to the ground, in some cases enveloping U.S. military personnel.

Some chemicals contained in oil-well fire smoke, such as benzene and PAHs, are human carcinogens. As described earlier in this chapter, the amounts of these pollutants in the air were low. Hence, their contribution to excess cancer risk would be expected to be small and increased rates of cancers likely would not result. The U.S. Army used EPA's standardized methodology to estimate cancer and noncancer risks from the oil-well fire smoke.[116] It concluded "the potential for significant long-term adverse health effects for the exposed DOD troop or civilian employee populations is minimal." Risks from cancers were estimated not to exceed two excess cancers per one million people exposed, a value well within EPA's acceptable range.

Noncancer risks from smoke exposure were calculated as Hazard Indices (HI). When the HI exceeds 1.0, there can be concern about potential noncarcinogenic health effects. In Saudi Arabia, the HI ranged from 0.6 to 2.0, while in Kuwait it ranged from 2.0 to 5.0. Most of this noncancer risk

was contributed by inhalation of VOCs, particularly benzene. The U.S. Army concluded that risk of noncarcinogenic health effects among the U.S. service members was low since HIs are based on EPA toxicity values that are set far below levels thought to cause health effects and that also account for sensitive subpopulations such as children and the elderly. A congressional Office of Technology Assessment analysis of the U.S. Army's risk assessment methods and findings concluded "the risks to health from exposure to the smoke and the background air contaminants in the Persian Gulf are likely to be extremely small."[25]

Oil-well fire smoke appears not to have caused observable changes in lung tissue. Researchers at the Armed Forces Institute of Pathology found no significant differences when they compared lung tissue from autopsies of 33 U.S. service members who died after the start of the oil well fires to lung tissue from autopsies of soldiers who died before the fires.[26]

Information has been gathered from 110 firefighters working for private companies in the Kuwaiti oil fields in 1991. Individuals were deployed for 28-day periods, working daily at the well heads without breathing-protection equipment. Most were over 30 years old and had 10 or more years experience fighting similar well fires, many of them in Kuwait and elsewhere in Southwest Asia. No cases of illnesses resembling those reported by Gulf War veterans were reported, nor have such complaints been observed among thousands of oil-well firefighters who have spent years experiencing similar exposures.[27-31]

It is unlikely exposure to oil-well fire smoke is responsible for symptoms reported today by Gulf War veterans.

Known immediate health effects from inhaling large amounts of smoke and particulates are primarily respiratory, including coughing, wheezing, increased airway resistance, and respiratory infections. Toxic gases that can be found in oil-well fire smoke – such as hydrogen sulfide and sulfur dioxide – can cause eye and nose irritation, decreased pulmonary function, and increased airway reactivity.[312-313] Nevertheless, these toxic gases were not detected at high levels during the fires.[292-293,312-319] High levels of airborne particulates, which sometimes occurred in the Gulf region, are associated with increased rates of asthma and can exacerbate other chronic respiratory conditions. With chronic (months or years) exposure to particulates, there is increased risk of some loss in lung function or chronic bronchitis, especially in cigarette smokers.

What do we conclude about the risks of oil-well fires to Gulf War veterans? Based on research on human and animal health effects of exposure to air pollutants and on currently available exposure data, the Committee concludes it is unlikely exposure to oil-well fire smoke is responsible for symptoms reported today by Gulf War veterans. Although smoke from the oil-well fires did not include levels of carcinogens that would be expected to increase cancer rates among Gulf War participants, VA mortality studies will include cancer surveillance.

Petroleum Products

Diesel, kerosene, gasoline, jet fuel, and other petroleum-based fuels were widely used during the Gulf War for dust suppression, waste incineration, and for fueling vehicles, stoves, heaters and generators. U.S. service members in certain jobs were occupationally exposed to petroleum fuel vapors and combustion products, such as toluene, xylene, benzene, ethyl benzene, carbon monoxide, sulfur dioxide, nitrogen dioxide, particulates, lead, and other pollutants. Additionally, in some areas near the Kuwaiti oil-well fires, unburned crude oil drizzled down, covering the ground and troops below.[112]

Petroleum fuels are a complex mixture of aliphatic hydrocarbons and aromatic hydrocarbons such as benzene and PAHs. These fuels also commonly contain various additives, like lead. When burned, petroleum fuels produce a variety of potentially hazardous combustion products. High-level, short-term exposures to fuel solvents can cause immediate effects. In most cases, however, complete recovery occurs when the exposure ceases.[1,26a]

Exposure to high, nonlethal levels of petroleum fuels usually is followed by complete recovery.

U.S. service members could have been exposed to petroleum fuels by inhalation, ingesting contaminated water or dust, and skin contact. Inhalation exposure could depress the central nervous system (CNS). Symptoms include short-term effects ranging from fatigue, headache, nausea, blurred vision, and dizziness, to convulsions, paralysis, and loss of consciousness depending on the dose.[26,112] Again, exposure to high, nonlethal levels usually is followed by complete recovery, although rare cases of permanent brain damage after massive exposure have been reported.[117,28,202]

Prolonged breathing of diesel fuel vapors can damage kidneys or lower blood clotting ability.[28] Studies of workers occupationally exposed to certain hydrocarbon solvents in petroleum fuels suggest that long-term high-dose exposure over 12 to 14 years can lead to neurotoxic effects.[117,203] For example, psychomotor disturbances, visual memory and perception, and visuomotor learning ability were significantly affected in exposed gasoline-pump workers compared to matched controls, particularly workers exposed for more than a year.[118] Some studies suggest there are neurotoxic effects from long-term exposure, including decrements in memory, cognitive functioning, and sometimes neuromotor functions.[117] Other researchers, however, have challenged the existence of what is sometimes referred to as "chronic toxic encephalopathy," and uncertainty exists about CNS effects from long-term, low-level exposures to solvents.[119]

Benzene makes up about one percent of U.S. gasoline and up to five percent of European formulations. It is a known human carcinogen that is associated with certain types of leukemia. Nevertheless, more than 55 published epidemiologic studies of workers exposed occupationally to hydrocarbons such as gasoline generally do not replicate the carcinogenic effects reported for experimental animals.[117,202] Recent studies of refinery

workers also do not reveal a clear association between gasoline production and leukemia.[29,30] Still, based on the limited evidence from animal studies and the presence of benzene in gasoline, the International Agency for Research on Cancer (IARC) concluded that gasoline is possibly carcinogenic to humans. It is not known if other petroleum products cause cancer in humans. IARC believes there are insufficient data to assess whether light fuel oils or light diesel fuels cause cancer in humans. However, IARC has determined that occupational exposure to fuel oils during petroleum refining is probably carcinogenic to humans.[24]

Although ingesting small amounts of fuel oils is unlikely to cause significant symptoms, ingesting fuel oils in larger quantities can cause vomiting, diarrhea, swelling of the stomach, stomach cramps, coughing, drowsiness, restlessness, irritability, and unconsciousness.[24] Ingestion of fuel oils can be accompanied (during vomiting) by aspiration of some of the material into the lungs, which can produce a chemical pneumonitis.

Skin exposure to large amounts of oil can physically clog pores and hair follicles, compromising body heat loss. Long-term exposure can cause acne and other skin problems. With high concentration or extended exposure, lighter components of crude oil or other fuel oils can defat the skin, leading to redness and itching or dermatitis.[24,32]

Exposure to the normal combustion products of petroleum fuels is also a health concern. Limited epidemiologic evidence indicates daily use of kerosene stoves for cooking or heating does not cause breathing problems for most people.[24] If insufficiently vented, however, carbon monoxide generated from fuel oil combustion can build up, causing drowsiness, nausea, and even asphyxiation. Individuals exposed to unvented combustion of fuels containing lead could experience health effects ranging from subtle biochemical changes in blood to severe CNS effects at high doses. Occupational exposure to inorganic lead is associated with subjective signs of neurotoxicity such as forgetfulness, lethargy, and weakness. These neurological signs and symptoms occur at about the same blood lead levels as other overt signs of lead intoxication, such as gastrointestinal complaints like abdominal pain, nausea, and vomiting.[26]

It is unlikely that health effects reported today by Gulf War veterans are due to exposure to petroleum products during the war.

What do we conclude about the risks of petroleum products to Gulf War veterans? While certain subsets of Gulf War service members could have experienced occupational exposures to petroleum products that would entail increased risks of health effects, it is unlikely that health effects reported today by Gulf War veterans are due to exposure to petroleum products during the war.

Psychological and Physiological Stress

Virtually all Gulf War participants were exposed to a wide range of stressors associated with the war. Throughout human history, observers have noted a correlation between the horrors of war and "mysterious" illnesses in soldiers and veterans.[1] Only recently, however, have the broad range

not been very successful with that. The fact is, we have looked at some of the outcomes that veterans have indicated they have and focused almost entirely on that. I think we are going to be dependent upon the DoD for much of that. I think a year or year and a half ago, DoD was working on some health and exposure assessment that would actually track individuals as to where they were. We could go back then and figure out whether or not they were in fires or they were deloused with this compound, or where they were on February the 10th, or whatever the date. The dates I'm throwing out are not significant dates, so don't take it the wrong way. We still don't have that. I think for many of things that we are interested in, that would be very useful. We have made that recommendation, and I think the DoD understands it, but maybe we ought to, as a committee, make that recommendation to you. Perhaps you could assign some people to do it in conjunction with the DoD. How do we do that? Make a recommendation to you?

DR. KIZER: Well, I think how we do it is a question that becomes one of logistics. Once you take a recommendation, then obviously we have to decide who is best qualified to do it. Who has the resources; if we don't have the resources, how do we get them. You do all of the logistical work. First of all, we need the recommendation, and so I think your question is, should you make that recommendation to me? If you feel that's something that needs to be done, then I think you need to make the recommendation. As I say, having been involved with these, whether they're childhood cancer clusters in the Central Valley of California, or looking at respiratory complaints from trains that have derailed and spilled pesticides in the environment, we usually start by tracking individuals and working through what their complaints were and then trying to assess what types of exposure they had. You start with 1 and 2, and then once you get enough, you start doing the statistical tests and all of that. Unless you have that basic fundamental epidemiologic assessment, it often becomes much harder then to extrapolate down the road when you're seeing people with just complaints, and you don't really know what came before it.

DR. BINGHAM: We'll have to talk about that, but that's really the position we've been in. The only exposure assessment we've had is the Gulf War experience. Is that right? I mean, we haven't narrowed it anymore than that. We do have some data on fires, which doesn't track necessarily with the outcomes that have been described, although it could track in some folks. We don't know whether it does or not, by and large.

COLONEL ERDTMANN: I think our very best data that we have relates to the oil well fires. We have very good data, looking at various pollutants, and the amounts of the pollutants in various parts of the whole area of conflict. OIL FIRES

DR. BINGHAM: Late in the game.

COLONEL ERDTMANN: Yes, but with the ability to, using mathematical modeling, to then extract backwards, to see what the exposures would have been at the time the majority of the troops were in

Persian Gulf Veterans Coordinating Board

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PERSIAN GULF VETERANS COORDINATING BOARD STAFF STATEMENT:
This paper reviews the clinical presentation and potential causes of unexplained illnesses among Persian Gulf war veterans and should serve as a basis for formulating further lines of investigation. No potential causes of illness are being excluded in this review. The Coordinating Board would very much appreciate any new information concerning the health of Persian Gulf War veterans.

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Environmental Hazards

Desert Storm troops were exposed to several potentially harmful environmental hazards in the Persian Gulf, the most spectacular of which was smoke from 605 oil well fires started by the retreating Iraqi army. A concerted effort was made by the DoD, U.S. Environmental Protection Agency (EPA), Department of Health and Human Services (HHS), and the National Oceanic and Atmospheric Administration to evaluate the health effects from these fires. Based on data collected from May through December 1991, the carcinogenic and non-carcinogenic health risks from exposure to oil fire smoke were determined to be minimal due to lofting of the smoke above ground level and nearly complete combustion of most chemical substances [27,28]. In addition, assays of metals and volatile organic compounds (including benzene) among troops indicated extremely low level exposure to harmful substances [27]. It also is notable that there has been no indication of unexplained illnesses among the U.S. civilian fire fighters who were highly exposed to combusted and non-combusted products of damaged oil wells [29].

In addition to smoke, U.S. troops were exposed to low levels of several pesticides, and possible health effects from such exposure are being investigated. The vast majority of pesticides employed in the Gulf were products that have been U.S. EPA registered and have been used without ill effects on numerous prior exercises of U.S. troops in areas like Egypt and Southeast Asia [Table 3]. Also, these pesticides are routinely used in the commercial market and by DoD in the USA. Pet flea collars, which contain organophosphates and carbamates, were used inappropriately by a small number of troops before being prohibited but have not been associated with unexplained illnesses. Herbicides were not used by U.S. forces in this desert environment.

No cases of acute pesticide poisoning are known to have occurred during Operations Desert Shield/Storm. The possibility that pesticides could have increased the acute toxic effects of pyridostigmine bromide is being investigated, but chronic effects are considered unlikely [30]. To further assess the possibility of synergistic effects among various substances that Gulf war troops may have been exposed to, the VA and U.S. Army are conducting studies of potential interactions between pyridostigmine bromide, DEET, and permethrin.

Numerous petrochemical plants are located on the Northeastern coast of Saudi Arabia where many of our troops entered the theater of operations. Most combat troops passed through these port areas rapidly, but large numbers of support personnel were permanently stationed on the coast, a large percentage of whom were reservists. It is possible that exposure to various chemicals in these areas could explain a higher risk of reported illnesses among reservists compared to active duty personnel. However, there have been no accounts of increased health problems among local workers or inhabitants of the cities around these petrochemical plants [5,31]

Several other factors could explain why, at least initially, reservists frequently have been identified with unexplained illnesses: reporting bias is possible because of career concerns among active duty personnel during a period of downsizing; reservists tended to be older and possibly less physically resilient compared to active duty troops; and, reserve personnel may have suffered increased stress because they had to leave civilian jobs and experienced greater disruption of their

personal lives [9,32].

Another unique environmental hazard of this war was exposure to depleted uranium (DU) munitions which are used for their enhanced armor penetrating ability. DU is a heavy metal which is less radioactive than natural uranium and poses minimal health hazard when external to the body, although the impact of DU on armored targets or the involvement of DU munitions in fires can result in localized aerosolization and increased exposure. There were 35 soldiers in vehicles struck by DU during friendly fire incidents (22 who may retain DU fragments). Approximately 32 other soldiers potentially were exposed to DU while fighting a fire in a munitions storage area and from servicing vehicles hit by DU munitions; but, these troops when tested have not had elevated urine uranium levels [9]. Troops directly exposed to DU munitions are being closely followed by VA and DoD and have not had problems with unexplained illnesses. Other ground-based troops are not considered by DoD to have been exposed to excess risk because of the very low levels of radiation involved with DU munitions.

Some troops may have been exposed to a number of other potential environmental hazards, including: microwaves; chemical-agent-resistant-coating (CARC paint) fumes containing isocyanate; various petroleum products like JP4 fuel used in tent heaters and on the ground to keep the sand from blowing; decontamination solution [2], which contains propylene glycol, monomethyl ether, and ethylene glycol; and, airborne allergens and irritants [33]. None of these exposures has been identified as a primary cause of unexplained illnesses, either because they involved small numbers of infrequently affected troops or because they are not known to cause the constellation of chronic multi- system complaints reported by Gulf war veterans [9,27,30]. Nevertheless, all potential environmental exposures are being evaluated extensively to determine their effects on the health of Persian Gulf troops.

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(particularly the reported use of unventilated heaters in living quarters that might have contained mixtures of diesel and jet fuel) could have caused a variety of exposures to combustion products, including lead. Information received in response to a request to DoD on whether leaded fuel was used in tent heaters stated that U.S. Central Command records indicated that approximately 145,000 gallons of gasoline (leaded and from local sources) were consumed per day in the theater between August 1, 1990, and March 30, 1991. These records did not indicate whether the fuel was used for heating tents; it was intended for use in vehicles, cooking, and power generation. The individual services provided information on the use of leaded gasoline in tent heaters as follows: Air Force, electric heat exclusively; Navy, kerosene and diesel fuel only; Marine Corps, diesel only; Army, "has no record of leaded fuel use in tent heaters" (Cusick, 1996).

To investigate further possible effects from this exposure, in combination with other exposures that were prevalent at various times in the Gulf, DoD has funded a laboratory study in rats to evaluate the toxicity of simulated PGW exposures. This research initiative has as its chief aim the investigation of rodent responses to exposure conditions similar to those experienced by PGW veterans. In an effort to construct a rodent model of unexplained illness in PGW veterans, Sprague-Dawley rats will be subjected to controlled experimental exposure to Deet, pyridostigmine, and a mixture of diesel and jet fuel followed by an electrical shock; controls will help to delineate possible effects of chamber exposures alone, compared with exposures with stress-producing electrical shock. Postexposure testing will include examination of central nervous system (CNS) integrity (auditory startle and adaptation to auditory stimulus and photosensitivity), testing of motor skills (grip and total activity), neurotransmitter analysis at sacrifice, two-dimensional gel electrophoresis to investigate whether novel stress-related proteins are produced in stressed animals, clinical chemistry, and measures of immune function.

This ambitious protocol, as reviewed, may produce hypothesis-generating data. However, generalization of any results to veterans will necessarily be problematic. No variation in dosing will be done, and no dose-rate considerations are included. Although these animals are useful models for some known human conditions, the applicability of any adverse (or the opposite) outcomes noted in these studies will inevitably be questioned.

Oil Well Fires and Spills

Many agencies were involved in monitoring and measuring various aspects of the oil well fires and smoke (EPA, 1991; U.S. Gulf Environmental Technical Assistance, 1992; WMO, 1991, 1992). Several efforts have been made to determine whether the oil well fires and spills created by retreating Iraqi forces affected the health of U.S. troops (DoD, 1993; USAEHA, 1992, 1994). One major effort at environmental assessment and health impact was undertaken by the

[Exhibit 4, page 18, Craig Stead Testimony of 6/26/97]

former U.S. Army Environmental Hygiene Agency (USAEHA), currently known as the U.S. Army Center for Health Promotion and Preventive Medicine (USACHPPM). Although exposures began when the first oil well fires were ignited by the Iraqi armed forces during their retreat in February 1991, and some lasted until November 1, 1991, USAEHA could not launch a successful air-sampling effort until the beginning of May, after the more stagnant air conditions of the winter months had passed. Those who undertook the sampling efforts did so with this knowledge. They intended to address the problem as thoroughly as possible by the use of meteorological modeling. A geographical information system (GIS) is being developed to integrate information over space and time on airborne and soil-based exposures, on meteorological conditions throughout the study interval, and on unit troop movements during ODS. Once this model is available, exposure of individual troops can be estimated throughout the region, although further work will still be needed to validate the model and estimate its precision.

There were as many as 10 fixed air-sampling stations in the theater, but 2 of them operated for less than 2 weeks and 2 more operated for only 2 months. Three were in operation through the end of December 1991. These fixed sites were located where troops were concentrated, and soil was sampled from the same areas. The results are to be combined with National Oceanic and Atmospheric Administration (NOAA)-assisted modeling and records of troop movements using the GIS to estimate reasonable maximum individual exposures (RMEs) to the chemical substances sampled. Data available include air and soil pathway analysis and industrial hygiene sampling. Air and soil quality was estimated not to have deteriorated during the sampling interval, and a reference to earlier sampling suggests that air quality at some sites was even higher than before the war. Increases in toxic metals in soil were not found during sampling except for metals unrelated to Kuwaiti crude oil.

Air pollutants expected from the oil fires were classified into four categories: reactants (uncombusted crude oil components), combustion products (e.g., carbon dioxide and water), incomplete combustion products (e.g., carbon monoxide), and products of secondary reactions (photolysis). The substances included short-chain and low to medium molecular weight aliphatics such as butane and heptane (both straight and branched chain) in the range of C_2 to C_{40}, simple and polycyclic aromatic hydrocarbons (PAHs), benzene, heterocyclic compounds including naphthalene and xylene, and substituted compounds such as methylated and halogenated compounds. Air samples were assayed for suspended particulates, both total and less than 10 μm in diameter, and for a series of volatile organics, PAHs, and metals. A subsample was examined for sulfur dioxide, nitrogen dioxide, coal-tar pitch volatiles, and acid aerosols. These agents were chosen as likely to provide a reasonable estimate of the most important particulates of the oil well fires and spills. The sampling was designed so that results could be used to estimate risks of cancer and subchronic noncancer diseases.

[Exhibit 4, page 19, Craig Stead Testimony of 6/26/97]

So far, based on our present knowledge, none of the individual agents sampled or detected seems likely to cause symptoms that would persist for months or years after return from the PG. However, the modeling now in progress may offer some improved understanding of the general environment of troops located in different parts of the war zone or may raise questions about interactive effects or combined exposures.

The USAEHA sampling (after May 1991) documented little deterioration of general air quality during that period of air monitoring. Although substantial increases were noted in particulates, concentrations were still considered "normal" for this area of the Middle East. Exposures to organic compounds were similar to levels observed in Houston and Philadelphia, cities with major petrochemical industries. There were relatively high concentrations of naturally occurring metals, apparently from wind-blown surface soils.

There was some concern about ingestion and dermal absorption of air pollutants that had settled out, and these routes of exposure have been considered (USAEHA, 1994). However, no measurements were taken, so possible exposures through these additional routes can be estimated only by mathematical models.

Further work by USAEHA is expected to provide a model of exposure distributions and to incorporate information from earlier, more limited sampling that might improve estimates of exposures at troop encampments. This work also will examine the frequency and duration of exposures. The model will have to be validated and its precision estimated before evaluating the relevance of the data.

The Armed Forces Institute of Pathology (AFIP) has completed a study of 351 autopsies of U.S. personnel who died between August 1990 and November 1991 in Southwest Asia (SWA) during ODS/S and shortly thereafter. Reviewed were written autopsy records, histopathologic specimens, and toxicologic findings. A group of 149 individuals who died before the oil fires were lit was compared with a group of 202 who died after the fires were lit. No evidence was found to support an association between autopsy, histopathologic, and toxicologic findings and any environmental exposures, including smoke from oil well fires. Analysis for heavy metals from blood and tissue obtained at autopsy did not indicate elevated levels attributable to exposure in the Gulf environment (Peterson and Kalasinsky, 1996). The initial findings (AFIP, 1994) reviewed by this committee indicated that lead levels were elevated in some of the specimens (IOM, 1995a). Since that time, investigators from AFIP have stated that with respect to lead, no valid toxicological conclusions can be drawn because some specimens were reportedly drawn and stored in collection vessels containing lead. The methods and findings of this study are being prepared for submission to a peer-reviewed journal.

[Exhibit 4, page 20, Craig Stead Testimony of 6/26/97]

OTA Review

The Department of Defense Kuwait Oil Fire Health Risk Assessment (The Persian Gulf Veterans' Registry)

INTRODUCTION

Even before the end of Operation Desert Storm, the Department of Defense (DoD) began to assess the likely health impacts of the conflict's most visible icon--billowing smoke from 600 burning Kuwaiti oil wells, ignited by retreating Iraqi troops. Then, just as the last fires were extinguished, the Congress made its own specific demands for information about health risks to each smoke-exposed military participant (in Public Law 102-190 and later expanded on in Public Law 102-585). DoD responded with the Kuwait Oil Fire Health Risk Assessment, the heart of which is a computer-based geographical information system (GIS). The completed part of the risk assessment is based on actual measurements of contaminant levels taken while troops and smoke coexisted in the Persian Gulf. An ongoing part will eventually produce estimates of health risks based on "modeled" contaminant levels where and when no actual measurements were taken.

The Office of Technology Assessment was directed in Public Law 102-585 to assess whether DoD's project "meets the provisions of the law under which it was mandated," to assess its "potential utility . . . for scientific study and assessment of the intermediate and long-term health consequences of military service in the Persian Gulf theater of operations during the Persian Gulf War," and to address some other related questions. A requirement that OTA assess the Department of Veterans Affairs Persian Gulf War Veterans' Health Registry (which was mandated in the same public laws as the DoD effort) was met by a report in October 1993.[1]

This Background Paper describes briefly the work on DoD's Kuwait Oil Fire Health Risk Assessment to date, including the results of a pilot study of health risks, and then answers the questions addressed to OTA in PL 102-585. This review relies heavily on the work of a consultant expert in chemical risk assessment who studied the DoD project for OTA (the consultant's report is available from OTA).[2] His work is based on a variety of contacts with the U.S. Army Environmental Hygiene Agency (EHA; recently renamed the U.S. Army Center for Health Promotion and Preventive Medicine, but referred to in this report as EHA), which has major responsibility for this task within DoD, except for determining troop locations. That latter task falls to the U.S. Army and Joint Services Environmental Support Group (ESG), the Army's experts on military records. OTA's consultant (and OTA staff) gathered information from ESG on their activities relevant to this review.

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[1]U.S. Congress, Office of Technology Assessment, "The Department of Veterans Affairs Persian Gulf Veterans' Health Registry, 1993.

[2]Risks of Radiation and Chemical Compounds, "The DoD Persian Gulf Oil Fire GIS Modeling Project: A Review and Evaluation," submitted to OTA September 1994.

SUMMARY OF OTA'S FINDINGS

DoD have designed a GIS capable of producing individual estimates of risk resulting from exposure to oil fire smoke (and for smoke plus ambient background concentrations of toxic substances using measurements of air samples over a nine-month period) for each person who served in the Persian Gulf region, in response to the Congressional mandate. The system will be fully operational when the exposure information is all placed into the GIS (some time in 1995) and daily location data for each unit stationed in the region during the conflict are completely abstracted from original military records (some time in 1996). The risk assessment framework adopted by DoD is a logical and well-executed response to the mandate, based philosophically on the way the Environmental Protection Agency (EPA) conducts risk assessments under various environmental health laws. This type of risk assessment, because of a desire to protect public health, inherently overestimates risks to health.

In its *Final Report: Kuwait Oil Fire Health Risk Assessment*, EHA reported estimated health risks that were extremely low: an estimated upper limit of lifetime cancer risk of two in a million (possibly rising to three in a million when exposure is extended to the entire period during which the fires burned, and possibly somewhat higher at some places where measurements were not taken), and a low probability of noncancer health risks (not quantified in the same way as cancer risks). These risk levels are similar to estimates for a person spending the same amount of time in a U.S. city, calculated in the same way. Under existing risk assessment scenarios, these risk estimates would be considered so low that, in most cases, they would be dismissed. Scientifically, there is no added value to actually generating (or being able to generate) risks for individuals, all of which would be below these upper limits (or slightly modified upper limits not expected to be much different from these) and would, in any case, not be very accurate. Risk assessment methods have generally been designed to apply to groups of people and not to estimate precise risks for any one individual. Since all estimated risks would be very low, they could not be used to identify any particularly "high risk" cohorts that might benefit from medical surveillance or other intervention.

The GIS may have uses other than generating oil fire health risk estimates. It is a versatile and powerful analytic tool that might be put to use in epidemiologic studies of other exposures in the Persian Gulf, but only if sufficient information on those other exposures were available and could be described accurately in time and place. (OTA is unaware of efforts to systematically catalog exposures in this way.) This type of use will depend on ESG completing its troop location inventory, independent of the needs of the oil fire risk assessment.

The important conclusion that OTA draws from DoD's report on oil fire health risk assessment is that, using state-of-the-art risk assessment methods, the risks to health from exposure to the smoke and the background air contaminants in the Persian Gulf are likely to be extremely small. If aspects of the Persian Gulf experience are causing illness, they are likely to be other than oil fire smoke, according to DoD's risk assessment.

When completed, DoD's GIS and its associated risk assessment system will meet the Congressional mandate for individualized estimates of exposure from Persian Gulf oil fires. The troop location and atmospheric data related to the smoke will be easily accessible indefinitely once they are all entered into the system. The scientific value of the program, however, lies in what already has been accomplished, which establishes that overall risks to health from oil fire smoke are very low. Additional scientific value may come from its use in evaluating other exposures that are suspected of

[Exhibit 5, page 22, Craig Stead Testimony of 6/26/97]

causing health problems among Persian Gulf veterans, but this depends on having detailed information about other potentially harmful exposures. **Actually being able to generate individual exposure estimates, which is required by the law, is of very low value as it relates to learning anything about veterans' health.**

THE OIL FIRES IN KUWAIT

As Iraqi troops withdrew from Kuwait at the end of the Persian Gulf conflict, they destroyed more than 700 oil wells in four major oil fields, and about 600 were burning at the end of February 1991. Other wells were gushing oil and some of the resulting "oil lakes" also burned. When all the fires were burning, perhaps 5 million barrels of oil were consumed each day. Before the last fire was extinguished in early November 1991, 800 million barrels of oil may have burned.

The fires released copious smoke that rose to altitudes of one to four kilometers and moved mostly to the south and west under the influence of prevailing winds. Some fires released predominantly white smoke, indicating large quantities of water vapor; some released very black smoke, indicating high soot content; and some released smoke intermediate in color. Plumes from the individual fires merged as they moved downwind into a "super-plume" that could be tracked from satellite images of the Persian Gulf region. The super-plume was dense enough to block out most sunlight when it was overhead.

The plumes contained both oil combustion products and unburned chemicals originating in the oil, along with minerals associated with soil or water carried aloft by the fires. Significant quantities of the more volatile chemicals in crude oil also were released to the atmosphere by evaporation from gushing oil wells or crude oil pools.

Nearly 700,000 U.S. troops were deployed to the Persian Gulf region and many were in the region while the fires were burning. When a plume was overhead in the vicinity of the troops, there was a potential for exposure, although the densest part of the plume was generally well above the surface.

Soon after the fires began, speculation arose that exposures might cause acute health effects in some people. Measurements of pollutant concentrations and records of health complaints did not reveal a widespread short-term problem, but the possibility remained that smoke exposures could cause diseases, including cancer, later on.

THE CONGRESSIONAL MANDATE TO DOD

The first Congressional mandate came in the National Defense Authorization Act for Fiscal Years 1992 and 1993 (Public Law 102-190, Section 734), passed in December 1991. The law calls for the Secretary of Defense to:

> establish and maintain a special record relating to members of the Armed Forces who, as determined by the Secretary, were exposed to the fumes of burning oil in the Operation Desert Storm theater of operations during the Persian Gulf conflict.

[Exhibit 5, page 23, Craig Stead Testimony of 6/26/97]

This "registry" was to include the name of each exposed individual and "a description of the circumstances of each exposure of that member to the fumes of burning oil . . . including the length of time of the exposure."

About a year later, in the Veterans Health Care Act of 1992 (Public Law 102-585, Section 704), the Congressional mandate for information was expanded to all who served in the Persian Gulf during the conflict, not just those known to be exposed to oil fire smoke. The new mandate called for information (to the extent it is available) on the location and circumstances of each person's service including "atmospheric and other environmental circumstances in such locations." Public Law 102-585 also directs OTA to assess the mandated DoD "registry," as described in the law.

THE KUWAIT OIL FIRE GIS MODELING PROJECT

Status of the Project

As of September 1994, a preliminary version of the GIS had been completed and tested in a pilot project, described in EHA's *Final Report: Kuwait Oil Fire Health Risk Assessment*. EHA's report was made available to OTA in August by the Office of the Assistant Secretary of Defense for Health Affairs for the purpose of completing this Background Paper, but otherwise has not been released by them.

EHA are still working on completing the GIS database, but the basic structure of the system is in place. The main tasks remaining are to define the smoke plume boundary for each day, which is a well-defined activity but one that requires intensive work, and to determine "emission" rates for each toxic substance in the smoke (discussed later). Once this is complete, in early 1995, the system will be ready to generate health risk estimates for each individual who served during the period of the oil fires.

ESG have made considerable progress in computerizing the daily troop locations, but project that it will be another two years before the task is complete. Locations are abstracted on a unit-by-unit basis, so ESG already are able to provide EHA with daily locations for some units and will be able to add units as they progress. In addition, they have once-per-month locations for most of the Army units that served in the Persian Gulf (most units did not move around very much, so these locations probably represent relatively well the dispersion of troops).

Description of the Project

The GIS is a computer-based system designed to capture, maintain, and analyze information about troop exposures to the oil fire smoke and any risks to their health that might result. The information needed for an ideal assessment includes:

- the location of each service person on each day of service in the Desert Shield/Storm theater of operations,
- the location of every smoke plume on each day smoke was in the air,
- the inventory of toxic substances entering each plume,

[Exhibit 5, page 24, Craig Stead Testimony of 6/26/97]

- the concentrations of those toxic substances at the location of each service person on each day,
- the conditions of each person's exposure (e.g., the rate at which each person is inhaling air), and
- the inherent toxicity (including all diseases) of each substance to which each person is exposed.

In spite of its relatively straightforward purpose, the Kuwait Oil Fire GIS is a complex and resource-intensive undertaking. It requires a great many assumptions and procedures that are not demonstrably correct or incorrect. The GIS shares with most other risk assessment systems the need to deal with substantial uncertainties about both toxicity and exposure. As with many such assessments, the GIS copes with uncertainty by using assumptions that overestimate rather than underestimate risk; in the jargon of risk assessment, they are "conservative" assumptions. Both the values for toxicity and the exposure scenario parameters (such as the duration and intensity of exposure) are chosen to minimize the possibility that risks will be underestimated. EHA have generally followed the lead of the EPA in these areas.

For each person, two risk numbers may be produced: the risk of cancer (all cancers combined), which is expressed as a fraction (e.g., one in one million) and the risk of all other chronic diseases (the nature of which are not specified), given as a "hazard index," expressed as a multiple of one, which is set at a level at which no toxicity is likely to occur even after years of exposure (discussed in more detail below).

Information on the toxicity of chemicals found in the smoke comes from various EPA sources and is "generic" (i.e., the same toxicity relationships are applied regardless of the source of the chemical, so the ones used are not specific to these chemicals as constituents of smoke). Information on the levels of those constituents to which troops were exposed is specific to the Persian Gulf experience and comes from a variety of data sources on the oil fires. Troop location data come mainly from written military records. Each component of the system is discussed below.

Troop Identification and Location

The identity of nearly all the 696,000 individuals who served in the Desert Shield/Storm theater of operations during the Persian Gulf conflict have been available since the early development of the GIS. The Defense Manpower Data Center has supplied this information to ESG (including 64 data elements for each person). It is a simple matter to pinpoint the dates of service in the Persian Gulf and the unit with which each person served; these two pieces of information are all that is needed to enter the GIS on an individual basis.

The extent to which military personnel were exposed obviously depends on where people were in relation to the smoke. The whereabouts of each service member at each moment he or she was in the Persian Gulf cannot be known precisely, of course, but accepting a few basic assumptions, locations that in most cases will be reasonably close can be assigned. This aspect of the GIS involves fewer assumptions and less estimating than do others.

For the purposes of the GIS, a location for each company-level unit (most representing about 150 people) will be determined for each day of the Persian Gulf era (not just the period of burning oil fires, following the expanded mandate of PL 102-585). This information for each such unit was recorded on

paper in their daily records. Abstracting these data points requires actually reading through the records for each of these approximately 13,000 units and transcribing each point (by latitude and longitude). The points captured in the records came from automated locators that use satellite contact to calculate position. To the extent the record keeper read the instrument and entered the numbers correctly, the locations should be quite exact.

The most important assumption about the location coordinates is that all members of the unit are assumed to have been at the same place. This clearly is not true. In general, however, it is probably true that troops were relatively near their unit location most of the time. As it turns out, the resolution of other parts of the GIS is not so great as to make locations that might be off by even 15 kilometers a big problem. In addition, it appears that the smoke plumes did not vary a great deal over short distances, so all in all, the troop location data will be sufficiently precise for nearly all individuals. There undoubtedly were times when people were distant from their unit for particular reasons. One can conceive of scenarios in which location away from an individual's unit might be important, but it is unlikely to be a major problem.

Smoke Measurements

The level of risk associated with the oil fire smoke depends on what the various compounds are that made up the smoke and on the levels of each one when and where troops were exposed. One could imagine a map of the smoky region blanketed with numbers that describe the concentrations of each relevant smoke constituent for each day the fires burned (assuming that conditions did not change appreciably over the course of a day). It might then seem a simple matter to describe the level of exposure to each person on the ground in contact with all the various substances. EHA's task in this area is to develop the blanket of numbers from data that are rather limited, in both place and time. They are going about this using two independent methods: first, using measurements from air monitoring in the Gulf while the fires were alight; and second, mathematical modeling of smoke dispersion using meteorological data and information on smoke emissions from the fires. These approaches, which give rise to quite different estimates, are described in the following paragraphs.

EHA sampled air in the Gulf area from early May through December 1991, which includes about a month of sampling after the last fire was extinguished. Using methods recommended by EPA, about 4,000 samples from eight sites (four in Kuwait and four in Saudi Arabia) were taken and analyzed. The sample locations were chosen primarily on the basis of major troop concentrations, and included areas where the smoke was considered heaviest . Samples were analyzed for all toxic substances reasonably anticipated to be in smoke from an oil well fire involving Kuwait crude oil, as well as for other potentially toxic substances. Airborne concentrations can result from the vapors of volatile organic compounds or from less volatile compounds attached to fine particles of smoke or dust. These measurements represent the combined concentration of substances in the smoke and background levels of those substances.

Using the air monitoring data, individuals are assumed to be exposed to the concentrations at the monitoring site closest to their company's location on each day. Estimates will also be made using the HY-SPLIT model (see below) for those locations during the early period of burning oil wells, before monitoring began, from February to April 1991. (These estimates will differ from the monitored levels because they will not include background concentrations.)

[Exhibit 5, page 26, Craig Stead Testimony of 6/26/97]

In the second approach, the National Oceanographic and Atmospheric Administration (NOAA) used a mathematical model, "HY-SPLIT," to estimate for each "grid point" the average daily concentrations that would be caused by burning oil wells. HY-SPLIT is a state-of-the-art "trajectory" model that uses information on wind speed and direction along with particle settling and diffusion rates to simulate the movement of "packets" of particles over time. The model was calibrated using measurements from air samples taken in "flythroughs" of the super-plume by the National Center for Atmospheric Research, the National Aeronautic and Space Administration, university groups, and EHA's ground measurements. A third piece of information on smoke concentrations--satellite imagery of the plumes--is being used as well. By combining this visual "truth" with the boundaries of the HY-SPLIT model plumes, even better location of the smoke with time is possible.

The GIS modeled data from the period when some measurements were available (which has the advantage of calibration with the data from air monitoring) will be used to predict smoke-related concentrations for locations and days when there were no measurements, especially the period before field measurements began. For all periods, the modeled concentrations estimate only the contribution from the smoke itself, and therefore will be lower than the estimates from the ground measurements (which include other sources of pollutants, such as auto emissions). Exposures are assigned based on the nearest grid point to an individual's company location. Because the spacing of the grid points is relatively fine in comparison to the plume dimensions, these modeled exposures are not likely to be misestimated substantially even if a person was not near the grid point all day, as long he or she was closer to that grid point than to any other.

A weak link of the HY-SPLIT procedure is currently in the estimation of "emissions" for each constituent of the oil smoke. Only emissions of sulfur dioxide (SO_2) and soot are known with any certainty because the sulfur and carbon content of the oil can be estimated and because some cross-calibration was possible with measured concentrations in the plumes. For other substances, the estimates are less firm. Even for the metals, which are neither created nor destroyed by the fire, estimates are difficult because of the range of metal content in the crude oil from different wells and uncertainties about the fraction of each metal that reached the plume. Some of the metals would remain in oil pools and never enter the atmosphere, while another portion would attach to large-diameter particles falling out of the smoke early and not reaching the main plume altitude used in the modeling. For volatile and semivolatile organic compounds present in the oil, emission rates are even more speculative. Conversely, the rate of formation of hazardous substances during combustion is very uncertain. EHA are in the process of collecting information on the chemical composition of oil from each oil well field and on the total amount of oil released, to aid in determining appropriate emission rates.

The two types of concentration estimates generated by EHA have one fundamental difference: the <u>measured</u> concentrations provide a way to estimate <u>total</u> exposure for troops located at or near one of the eight sampling sites for any day during which measurements were taken. The <u>modeled</u> concentrations provide a way to estimate <u>smoke-related</u> exposures at any gridded location on any day the fires were burning, but provide no information on the contribution to total exposure from local, non-fire sources (e.g., auto emissions, airborne soil).

[Exhibit 5, page 27, Craig Stead Testimony of 6/26/97]

Personal Exposures

The juxtaposition of people with smoke makes for exposure. EHA included three routes of exposure in their risk analysis: direct inhalation of smoke, incidental ingestion of soil particles with smoke constituents attached to them, and dermal absorption of smoke constituents from soil adhering to the skin.

Inhalation is by far the most important exposure route (accounting for more than 90 percent of the total risk). The airborne concentration estimates described above form the basis for inhaled exposures. To determine how much exposure took place, EHA assumed a breathing rate based on 20 hours of relatively strenuous activity every day and four hours of sleep. Multiplying the total volume of air inhaled daily and the average daily concentration gives an estimate of the total amount of substance inhaled on that day. Dividing by body weight provides an estimate of the inhaled "dose" (milligrams of substance inhaled per unit body weight per day).

Soil is ingested when it sticks to food or fingers and is then eaten. Soil ingestion occurs under very ordinary conditions, and probably occurred to a substantial degree in the dusty Persian Gulf where hand-washing was not always feasible. EHA have assumed that each person ingested 300 mg of soil each day (a relatively high estimate). Soil concentrations have been determined from measurements at each of the same eight sampling sites as for the air concentrations.

Absorption through the skin depends on how much soil adheres to the skin, how much skin is exposed, and the fraction of each constituent that migrates out of the soil and through the skin over a day of exposure (which varies from chemical to chemical). EHA have assumed that one milligram of soil adhered to each square centimeter of exposed skin each day, that on average 4,270 square centimeters of skin were exposed (about 20 to 25 percent of total skin area), and that from 1 to 5 percent of each compound was absorbed.

The exposure assessment, consistent with other aspects of the model, has been deliberately "high-sided," so the exposure numbers coming from these calculations should represent the uppermost plausible level of exposure.

Estimating Risk

Estimates of health risk are calculated by applying generic information about the risks of each toxic substance (toxicity values) to individual quantitative estimates of exposure (discussed above) using some standard formulas. EHA used toxicity values from EPA's Integrated Risk Information System (IRIS), considered to be that agency's most thoroughly reviewed source of toxicity information. Because IRIS has not released toxicity summaries for all the substances of possible interest in the smoke from the Persian Gulf oil fires, other sources of information (mainly from other EPA programs) were used when necessary. Nearly all the toxicity values are based on data from animal experiments. Information on the toxicity of only some metals and a few other compounds is known directly for human beings.

[Exhibit 5, page 28, Craig Stead Testimony of 6/26/97]

OTA EVALUATION

The GIS

The estimation of troop exposures to oil fire smoke might have been handled by any number of computer-based systems, including relational databases or spreadsheet programs. But these types of systems are severely limited in both their analytic and display capabilities. The decision to use a GIS was based on its ability to capture, manage, manipulate, analyze, model, and display spatial data. The key to a GIS, setting it apart from other software (such as computer-aided mapping) is the "topology" that allows the user to query, analyze, and display the data with respect to the connectedness of elements with spatial features (e.g., grid points, lines, or polygons).

The GIS is an essential tool to achieve the objectives of the risk assessment project, cast narrowly to estimate exposures and risks to troops operating in the Persian Gulf. However, the purposes of the project beyond this have not been stated clearly. Neither the types of reports that will be generated for individual veterans nor their formats are well described. It is clear that ESG will use the system to respond to veterans' requests, but no other particular uses (i.e., for research) have been specified. If multiple users are contemplated, there should be more coordination in designing the system to meet their needs.

In principle, the GIS could be used to investigate exposures and risks from any other events or activities involving hazardous substances that could be located by geographic coordinates and date. Some exposures that have been mentioned are spraying of pesticides, use of diesel-fueled heaters in tents, chemical or biological agents deployed by Scud missiles, and depleted uranium used in projectiles and tank walls. OTA learned of no attempts to develop a database for any such exposures. Although it seems feasible to develop a database of location and time for some of these exposures and therefore to identify troops that were nearby, developing quantitative "exposure" estimates would be difficult or impossible. Given the extremely low estimates of risk from the oil smoke itself, using the GIS to aid in investigating other exposures might give added value to the investment that already has been made.

Information on Oil Fire Constituent Levels

EHA has made efficient use of the ground measurements of air during the period that monitoring took place. But there are limits to those data, which will affect EHA's ability to predict health risks over the entire area occupied by troops and over the entire time during which smoke was in the air. The principal limitations of the measured values are:

- **The restriction to eight sampling locations that may not be representative of the distribution of troops.**--Four of the locations are clustered tightly around Kuwait City and the four in Saudi Arabia represent only a portion of the area that was affected by the oil fire plume. Because monitoring sites were chosen on the basis of troop concentration, however, this is probably of little importance.

- **The restriction to the period May through November (and to shorter periods for most of the stations).**--Concentrations of smoke-related substances could have been higher during the period February through April when more fires were burning.

[Exhibit 5, page 29, Craig Stead Testimony of 6/26/97]

- **The inability to separate smoke-related contributions from those related to natural background or other non-smoke sources.**--The dominant source of both cancer and noncancer risk is benzene and it is not clear whether or not this is mainly smoke-related. The variation of chemical concentrations with distance from the fires is often inconsistent with a smoke source; for example, neither chromium nor benzene concentrations were significantly different between the Kuwait and Saudi Arabia stations (although this may be an effect of the super-plume). The degree of overestimation of smoke risk is difficult to evaluate but is probably substantial.

- **The use of one-half the detection limit for the concentration of every chemical reported as below the detection limit.**--Although the inability to detect a chemical in a sample is no assurance that it is not present, it may not be present or may be present a level well below one-half the detection limit. Using one-half the detection limit is appropriate where good reasons exist for assuming the chemical is present, but is questionable when few samples show detectable levels. EHA has followed EPA policy on this point, however.

- **Disposition of "non-target analytes."**--At every sampling location, the total concentration of "non-target analytes" (everything not specifically measured) among the volatile organic compounds was much greater than the total concentration of the target analytes. Some description is needed of the composition of the "non-target analyte" fraction and an explanation of why the detected substances were not target analytes.

- **Lack of consideration of particulate matter.**--Although it includes a section discussing generally the health effects of inhaled particulates, EHA did not formally assess the potential toxicity of particulates (other than "soot," or carbonaceous particles) independent of the chemicals associated with the particles. Respirable particulate matter, which apparently came not from the smoke but mainly from background dust, is considered a health hazard at concentrations lower than some of those measured by EHA.

Exposure estimates based on the HY-SPLIT model are not yet possible because EHA has not fixed on a procedure for estimating emissions of each substance from the various burning wells. Without these emission rates, no modeled exposures can be calculated (but by design, they must be lower than the measured values, which include ambient substances).

Toxicity Information

EPA is widely perceived as the most authoritative source of information on environmental health hazards, so EHA's decision to use EPA toxicity information is well justified. EPA's mandate to protect human health from environmental hazards means, however, that its estimates of the toxic potencies of chemicals are conservative, i.e., tend to err on the side of overestimating risk when the true values are uncertain. This conservatism may or may not be appropriate, depending on how the estimates are to be used.

EHA's use of the RfD to evaluate noncancer risks (in calculating HIs and HQs) may have been unavoidable (no other set of risk relationships is available for a wide range of substances) but it presents significant problems in interpretation. The RfD is intended as an exposure level that is without any risk of toxic effect, even if the exposure continues indefinitely, but somewhat higher doses are not necessarily

risky either. Most RfDs embody uncertainty factors of between 100 and 1,000, so they represent values far above those for which toxic effects may ever have been observed. For nearly all compounds, a true "threshold" for toxic effects in humans is unknown. This great uncertainty even under conditions of continued exposure is compounded by the fact the RfDs cannot be adjusted for assessing the shorter exposures experienced in the Persian Gulf. In order to understand the potential for noncancer effects, the nature of the health conditions that make up the risk should be examined for each substance so that judgments can made about their plausibility.

Interpretation of the Health Risk Assessments

The levels of cancer risk reported by EHA are considered to be below the level of concern under virtually every environmental or occupational regulatory proceeding. If the missing three months of exposure were considered, the highest risk might rise to about three in a million. A risk of one in a million has been considered unacceptable in some risk management decisions, but usually only when large populations are exposed.

The highest noncancer risks suggest that some chronic health effects from these exposures are possible. EHA have pointed out that the estimates contain so many conservative assumptions about both exposure and toxicity that they are likely to be substantial overestimates and may not imply any health risk at all. The addition of the three unmonitored months of exposure might increase the HIs somewhat because more fires were burning then, and the average daily exposure might have been higher. Because calculation of the HIs assumes exposures continuing indefinitely, the extra time itself would not affect the HIs.

The risks reported by EHA were from measurements of the air at ground level, so they represent background plus smoke-related contributions. No risk estimates were reported for modeled concentrations, which would, of course, be lower, because the background would be eliminated. If this risk of total exposure in the Persian Gulf is seen as an appropriate measure of impact on service personnel, then one might also ask what the risk would have been had the person spent eight months in the United States. The lifetime cancer risk of spending that time in the San Francisco Bay area, calculated in the same way as the risks for the Persian Gulf, is in the vicinity of 5 in a million--about twice the calculated risk in the Persian Gulf.

Comparisons such as these may be criticized as trivializing the Persian Gulf experience, and it may be difficult for the troops who were in the Persian Gulf, as well as the public who daily witnessed massive, billowing smoke plumes blocking the sun in Kuwait, to accept the results. But stating them is important to place the risk in some context. It should also be understood that, like the risks predicted from exposure to Persian Gulf oil smoke, risks predicted from monitoring in the United States are overestimates based on conservative assessment methods and are still far too low to be confirmed or disproved by epidemiologic observations in the exposed populations.

Uses of the GIS and Related Information

DoD's mandate from Congress was to describe each individual's exposure to oil fire smoke in the Persian Gulf. EHA has taken this a step further by creating a capability to translate exposures into

[Exhibit 5, page 31, Craig Stead Testimony of 6/26/97]

health risk estimates. EHA's system, relying on the GIS, should be fully functional once the system contains all the relevant atmospheric data (sometime in 1995) and the daily locations for each unit have been entered (sometime in 1996 or possibly sooner). Depending on whether the measured values of substances (from ground monitoring) are used or the modeled concentrations (from HY-SPLIT) are used, the risk will represent either total risk (in the former case) or the incremental risk of exposure to oil fire smoke. Which is preferred is a question of policy rather than science.

Whichever source of risk is chosen, the actual predicted levels will be very low. The figures reported by EHA in their report, which represent risks from both background and smoke (from ground monitoring measurements) ranged only as high as two in a million for lifetime cancer risk. Because of the highly conservative assumptions embodied in the RfDs and their unsuitability for predicting effects of short-term exposure, the highest HI (5.0) is probably of little health consequence. While it is not clear that these estimates represent the absolute worst case, it would be surprising to find exposures much higher anywhere in the Persian Gulf theater of operations. EHA might consider carrying out a "plausible" worse case risk estimate based on existing knowledge of where troops were and the known contaminant levels. Although complete troop location data will not be available for perhaps two years, ESG already has monthly location data for a large number of Army units that served in the Persian Gulf. Using this information now could provide a reasonable estimate of troop dispersion and perhaps identify a confluence of troops and relatively heavy smoke for purposes of estimating the highest of risks.

The question that can't be ignored is whether it is worthwhile to go ahead with a system that will generate extremely low risk numbers for everyone---numbers that are virtually uninterpretable (and not very accurate on an individual basis) in terms of one's health. The numbers generated will undoubtedly change over the years as the best information on risk changes (some upward, some downward), but it would be almost unthinkable that the risks would change by, say, a factor of 10.

Are there other uses for this system? As mentioned earlier, any risk that can be located in place and time is amenable to inclusion in the GIS. Complete information on suspicious exposures of virtually any kind are going to be hard, but not impossible, to come by. Once mapped, they could be matched up with the troops near them spatially and temporally, perhaps as the basis of choosing cohorts for an epidemiologic study. Such studies would require careful planning and cooperation and coordination between the researchers and EHA. (It should be noted that the exposures and resulting risks from the oil fire smoke are, as currently estimated, too low and too small in range to support epidemiologic studies.) If it is decided that the GIS should be available to assist in some of these studies, it would be important to complete the troop location component of the project, and to maintain the GIS as an analytic tool.

If the Congressional mandate to produce individual exposure estimates remains intact, DoD would be compelled to complete the GIS as it is doing, and eventually, the individual information would be available to veterans, whether or not it signifies anything about their future health (or more than would be signified by telling them that no one's risk is above a certain level). Scientifically, this exercise appears to have limited value in terms of the veterans' health if the reported risk numbers are confirmed and it is accepted that the risks from oil fire smoke are negligible. In terms of the veterans themselves, they may want these estimates. According to the Director of ESG, providing individual exposure assessments (along with an explanation of what they mean) may help allay the fears of Persian Gulf veterans concerning their exposures, and there may be value (other than scientific) in that.

[Exhibit 5, page 32, Craig Stead Testimony of 6/26/97]

REPORT

OF THE

DEFENSE SCIENCE BOARD

TASK FORCE

ON

PERSIAN GULF WAR HEALTH EFFECTS

JUNE 1994

94-20766

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DTIC QUALITY INSPECTED 3

Office of the Under Secretary of Defense for Acquisition
and Technology

Washington, D.C. 20301-3140

[Exhibit 6, page 33, Craig Stead Testimony of 6/26/97]

of pesticides, pesticide exposure may come under closer scrutiny as an etiological factor for other participants.

4. Oil Well Fires

On February 23, 1991, Iraqi forces began to destroy and set on fire more than 700 oil wells throughout Kuwait. All the fires were extinguished and the wells were capped by early November, 1991, but there was great concern regarding the potential health risk to personnel in the region as a result of their exposure.[16,17,18,19]

During the 8 month period in which the oil wells were burning, numerous efforts were undertaken to assess the air quality over Kuwait and to determine the health risks posed to the populations living, working, and serving in the military in the region. The U.S. Interagency Air Quality Assessment team arrived in Kuwait in March 1991 to begin to assess the possible health effects of the smoke from the oil fires. This team was composed of scientists from the U.S. Environmental Protection Agency, the National Oceanographic and Atmospheric Administration, and the Department of Health and Human Services.

During the period of the fires, the measured levels of two major air pollutants (sulfur dioxide, nitrogen dioxide) did not reach harmful levels. The level of particulate matter measuring less than 10 microns (PM_{10}), that portion of airborne particulate with the greatest impact on the respiratory system, did exceed the U.S. "alert level" on several occasions. However, Kuwait has frequent sand and dust storms, and the average level of PM_{10} in Kuwait is nearly 600 ug/m^3, the highest in the world.

The hazards to the soldiers posed by the smoke were largely dependent on the concentration of the pollutants in the air near the camps. Fortunately, the plumes resulting from the fires rose up to 10,000 to 12,000 feet, mixing with the air and then being dispersed for several thousand miles downwind over a period of several weeks. As the plume traveled, the particles and gases contained within it became more widely dispersed and also more diluted. The highest concentrations were in the areas nearest the affected oil fields and the areas immediately downwind. Few soldiers were in those areas for long periods of time. Considerable

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[16] Riley JJ, Hicks NG, Thompson TL. Effect of Kuwait oil field fires on human comfort and environment in Jubail, Saudi Arabia. Internat J Biometeorology 1992: 36-38.

[17] Ferek RJ, Hobbs PV, Herring JA, Laursen KK, Weiss RA, Rasmussen RA. Chemical composition of emissions from the Kuwait oil fires. Geophysical Research 1992; 97: 14483-14489.

[18] Hobbs PV, Radke LF. Airborne studies of the smoke from the Kuwait oil fires. Science 1992; 256:987-991.

[19] Laursen KK, Ferek RJ, Hobbs PV, Rasmussen RA. Emission factors for particulates, elemental carbon, and trace gases from the Kuwait oil fires. Geophys Res 1992; 97:14491-14497.

dilution took place over space, such that by the time the plume reached areas of troops in Saudi Arabia, it was far less visible and less concentrated than in Kuwait.

Potential effects on the respiratory system, such as a small loss in lung function or the development of chronic bronchitis, would be of particular concern to those who were exposed for many months to severe particulate pollution. These effects might be more likely to occur in cigarette smokers.

The US Army Environmental Hygiene Agency report of its participation in ODS provides some useful insights regarding industrial hygiene, preventive medicine and the impact of oil fires on health issues. The report cites no incidents regarding exposure to chemical weapons agents. Principal USAEHA efforts were to evaluate the health effects risks due to oil fires. On the basis of air and soil pathway analysis, excess cancer risk resulting from exposure to the Persian Gulf environment ranged from 2 to 5 per 10,000,000 well below the EPA range of concern of 1 per 10,000 through 1 per 1,000,000. The cancer risk assessment was based primarily on the risk from chromium. There was little difference in risk levels found between Saudi permanent monitoring sites and those in Kuwait near the oil fires. These results were based on collection of over 4,000 samples at 10 fixed ground sites over a period of seven months beginning in May 1991.[20]

Additionally, the National Center for Environmental Health, Centers for Disease Control and Prevention, performed surveys of VOC (volatile organic compounds) in the whole blood of two groups; American personnel employed in Kuwait City, about 20 km from the burning wells, and firefighters and medical personnel working at the burning oil wells.[21] Concentrations were compared to those of a random sample of persons in the United States. Median concentrations of the first group were equal or lower than those of the reference group; the firefighters did have elevated levels of some VOCs over those of the reference group. Since US military personnel were not involved directly in the fire fighting operations, their exposures would have been more comparable to those study personnel in Kuwait City, who showed no elevation in VOC level.

5. Sand

Because many US troops trained, executed maneuvers and actually lived out in the desert, there was initial concern for the possible adverse effects of being exposed to high levels of blowing and suspended sand. The sand was often powdery in consistency, and some personnel with respiratory problems did experience aggravated symptoms. An epidemiologic survey conducted among 2598 men stationed in northern Saudi Arabia, however, found that the type of structure in

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[20] Operation Desert Shield/Desert Storm: History of Participation by the US Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD 7 August 1990 - 31 December 1991.

[21] Etzel RA, Ashley DL; Volatile organic compounds in the blood of persons in Kuwait during the oil fires, Int Arch Occup Environ Health, Spring 1994.

SGPS-PSP
16 September 991
(703) 756-0125

INFORMATION PAPER

SUBJECT: Acute Health Effects - Kuwait Oil Fires

1. PURPOSE. To provide updated information concerning potential acute health risks from Kuwait oil fires in the immediate Kuwait City area during predicted meteorological inversions and wind stagnation October 1991 - January 1992.

2. DISCUSSION.

a. The Federal interagency Gulf Task Force working group met 11 September 1991 to examine the National Oceanographic and Atmospheric Administration's (NOAA) model predicting periods of meteorological inversion and wind stagnation for SWA during the period October 1991 - January 1992. NOAA ran the model using recently acquired meteorological data from 1986 through 1990 and determined that sulfur dioxide (SO_2) is predicted to reach alert levels in Kuwait City from southern oil field fires during October, November, and December. The northern oil fields do not appear to be a problem for Kuwait City due to the longer distances.

(1) Additional significant pollutants can come from oil fire smoke, reindustrialization and vehicle traffic. Respirable particulates, nitrogen oxides, sulfuric acid, sulfates, light hydrocarbons, carbon monoxide and miscellaneous acid gases can cause additive or synergistic health effects in combination with SO_2. A conceptual diagram of the inter-relationships of the pollution source and inversion factors is attached (enclosure 1

(2) The U.S. Army Environmental Hygiene Agency is working with NOAA to model estimated levels of the additional pollutants. Predicted amounts of the combined pollutants will be analyzed in relation to the existing military protective masks and shelters. We will make individual and unit protection recommendations based on their results. Although not expected, prediction of high levels of carbon monoxide or nitrogen oxides may require evacuation recommendations.

(3) The models only predict pollution levels in the Kuwait City area. Troop operations, especially in the vicinity of the northern or southern oil fields may have higher levels of pollution exposure. Operational planners must consider this factor during this period.

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[Exhibit 7, page 36, Craig Stead Testimony of 6/26/97]

b. Variables affecting pollution exposure levels include: rate oil fires are extinguished above or below the average 3 day; the amount of nonfire pollution added to the atmosphere; the accuracy of the model predicting a combination of strong low-level inversion and either southern or light and variable winds: e.g., October - 4 days, November - 10 days, December - 11 days; and, the distance to the pollution source which affects dilution of the pollutants.

c. The 1986 to 1990 weather data did not include any period with complete wind stagnation for more than 24-36 hours. This greatly discounts, but doesn't entirely eliminate, the possibility of extremely high pollution concentrations from several days of pollutant buildup in stagnant air at the oil fields.

d. Concentrations of SO_2 are currently not expected to exceed 1 ppm. However, at this level one should expect to see minor eye and respiratory irritation in "normal adults." The potential additive effects of the other pollutants increases the possibility that some soldiers will require hospitalization from respiratory distress. Certainly, the elderly, the very young, persons with pre-existing respiratory problems, and anyone with cardiovascular problems will experience more severe reactions at lower exposures.

(1) DOD personnel in the higher risk categories include any Army Corps of Engineer civilians with the predisposing conditions. Other federal agencies may also have civilian personnel predisposed to health effects in theater.

(2) The local population may experience significant effects since estimates indicate many have cardiovascular or respiratory problems, over 60 percent of adult males smoke and the recent war has had debilitating effect.

e. Enclosure 2 lists initial broad recommendations to prepare for the probable exposures. The AMEDD is already acting to accomplish several of the medical treatment and preventive medicine actions required. Informal contact has been made with DOD (HA), JCS, and CENTCOM.

f. Based on the potential for significant health effects, the EPA, as Gulf Task Force lead, is planning discussions with the Kuwaiti government in late September to ensure Kuwait understands the danger and convince them to take appropriate action.

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(1) Due to the uncertainty inherent in modelling future events, a broad warning statement is being drafted for U.S. release by EPA and PHS. Although its text is not yet available the message will generally state:

> Modeling indicates likelihood of several days of significant pollution occurring during the October to January period. Persons with respiratory disease, cardiovascular problems, the elderly and young may have significant adverse health effects.

(2) Currently the Kuwaiti's plan to have Meteorological forecast and sulfur dioxide monitoring capability available but will not provide public warning of predicted pollution events or real time alert of actual events. They evidently feel that the predictions of acute effects may cause unnecessary panic in the population. They reportedly think the panic is especially unwarranted as a result of an untested model. They do plan to provide the forecast to the government and to hospitals.

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[Exhibit 7, page 38, Craig Stead Testimony of 6/26/97]

TESTIMONY OF DR. JACK HELLER

NIH WORKSHOP

The Persian Gulf Experience and Health, April 27-29, 1994

COMMENT:

Dr. Heller is the principal author of the **Kuwait Oil Fire Health Risk Assessment** prepared by the DoD. This assessment found no increased risk of cancer from exposure to the oil field fire smoke.

Dr. Heller testifies the air pollution he monitored in Kuwait was **substanitally different and less than the air pollution to which the troops were exposed.** Dr. Heller's air pollution data was used in concluding the troops had no increased risk of cancer.

TESTIMONY:

We got in the country (Kuwait) on May 3 and had monitoring operations until all the fires were extinguished and then we carried on for an additional month to gather background data so although we missed the first two months of the fires we were there while there were still over 550 fires burning. **What we did miss was climatic conditions during those first two months. When we got in country in May the Shamal winds had picked up and there was very little ground impact of the pollution. Most of it was being air lofted due to the Shamal winds and thermal loading and formed a plume at 12 to 15 thousand feet. We had obviously a lot of reports from the two months previously when there was a lot more ground impact of plumes** We were not there to measure that. (1)

****

This is our site at the Ahmadi Hospital. You can see how close we were to burning oil fires. We were about a half a mile from active burning fires collecting samples. This is kind of what it looked like as you went into Kuwait. **And as I said earlier as opposed to ground impact a lot of what you had was that lofted thermal effect and the Shamal winds and so you would be under the plume but often would not get a lot of ground impact from the plume.** (2)

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BARRY WALKER: A vet. One of the things you say about the toxic thing Dr. Heller and where you set up your stations in May. If I remember correctly the wind blew off the Gulf and most of your places were close to the Gulf. I remember in March that it took three and four rags to clean windshields so you could drive down the road. It took four or five washings to get the oil out of our clothes. And you're saying indirectly that there is no affect on us.

[Exhibit 8, page 39, Craig Stead Testimony of 6/26/97]

DR. HELLER: No, what I'm saying is **what we measured at the time we were there starting in May when the Shamal winds were strongly blowing and there was a lot of thermal lofting of the pollution. We didn't have those ground level impacts. I've heard you know the accounts of a lot of more ground level and we were not able to get over those first two months. I do not discount that that happens. With those climatological conditions there would have been a lot more ground level impact and we probably would have measured more material during that period We did not, were not able to get over and set up. All I'm saying is from the time we were there when the Shamal winds were blowing we didn't get those.**

In fact the whole time I was there I had one ground level you know impact.

BARRY WALKER: What I'm saying we could have intaked and have plenty in our lungs and be affected by it.

DR. HELLER: **You could of had a a larger impact or intake than we were able to measure due to the climatological conditions not necessarily the number of fires but we were not there to measure it.** We're going to try and do some modeling studies with the National Oceanic and Atmospheric Administration to recreate those conditions and get an idea but we've been working with the VA on the Persian Gulf Registry...

BARRY WALKER: Do you recreate with a fine hose that you state that you spray a vehicle with oil? That is what it was like at times.

DR. HELLER: No. Well we can't do that . As I said, we can only record recreate through modeling efforts. **We know it was a higher level of ground level contamination at that period.**(3)

(1) Testimony from tape 6, side 2, at counter number 207
(2) Testimony from tape 6, side 2, at counter number 232
(3) Testimony from tape 7, side 1, at counter number 385

Transcribed by Craig Stead on 6/3/97. Emphasis by Stead.

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[Exhibit 8, page 40, Craig Stead Testimony of 6/26/97]

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