

completed. Together these studies, which were done in my laboratory at MIT, at CalTech, and at Berkeley, revealed the pathway of action of Ras. Now cancer biologists and drug companies alike are using this knowledge of the Ras pathway both for further studies of how Ras causes cancer in people and for the development of drugs, drugs that can block the various steps in the Ras pathway.

VII. PROGRAMMED CELL DEATH,
NEURODEGENERATIVE DISEASE AND CANCER

The third example I'll offer from worms relates both the cancer and to neurodegenerative diseases, which include AD. This example again is one in which studies of a basic biological phenomenon in the roundworm have had a major impact on our understanding of and approach to human disease. The biology in this case involves a phenomenon called "programmed cell death." For many years, biologists assumed that cells died because they were unhappy, i.e. because somehow they had been injured. However, a variety of studies revealed that many cells die during the normal course of development. For example, as our brains form, as many as 85 percent of the nerve cells made at certain times and certain parts of our brains die. Such death is a natural phenomenon and for this reason is often referred to as "Programmed Cell Death."

Given that cell death is a natural aspect of development, some years ago my colleagues and I reasoned that like other aspects of development, PCD should be controlled by genes. We sought such defined a 15-gene genetic pathway that controls programmed cell death in the worm. It now appears that a least some of these genes correspond to human genes that caused disease. For example, we talked earlier about neurodegenerative diseases, such as AD, Huntington's Disease, Lou Gehrig's Disease and Parkinson's Disease. Many researchers believe that these diseases, which are characterized by the death of nerve cells, are diseases in which the normal process of PCD has gone amok. Specifically, the normal pathway that causes cells to die by PCD during development for some reason may be unleashed in nerve cells that are not meant to die.

How might we stop such deaths? By blocking the killer genes responsible! And what are the killer genes? We have ID'd two such genes in the worm, genes we call CED-3 and CED-4, for "cell-death abnormal." Given these worm genes, others have gone on to find similar genes in humans that also act to cause cell death. These genes have now become major drug targets: many companies in the pharmaceutical industry are attempting to block the action of these killer genes, with the goal of preventing such neurodegenerative diseases.

It turns out the genetic pathway for PCD we have defined is relevant not only to neurodegenerative disease but also to cancer.

Let me explain. What is cancer? In brief, cancer reflects an uncontrolled increase in cell number. How can you get such an increase? One way is to make too many cells. This is precisely what happens when the Ras gene, which we just discussed, is mutated. However, it turns out there is another way to make too many cells. The number of cells in our bodies is really an equilibrium number. Cells are always being added to our bodies, by the process of cell division, but cells are also always being taken away, by the process of programmed cell death. So, we can generate too many cells—as in cancer—not only by too much cell division but also by too little cell loss.

How can we bet too little cell loss? One of the genes we identified as controlling cell

death in the worm is not a killer gene but rather a protector gene—it protects cells from dying by PCD. If a gene like this is too active, too many cells would survive, and cancer would result. In fact, there is a human cancer gene that is very similar to this worm protector gene, so similar that the human gene can work in worms to protect against worm cell death and to substitute for the worm gene. Given such protector genes, how might one prevent? Again, this is precisely the approach that is now being taken in the pharmaceutical industry, and there is great hope that by learning to control such protector genes it will be possible to control certain cancers.

VIII. CONCLUSIONS

Let me conclude very briefly by summarizing what I've said. First, a gene is a gene is a gene. Genes in humans are fundamentally no different from genes in other organisms and are so similar in many ceases that a human gene can be put into another organism and work just fine. Second, genes are much easier to analyze in experimental organisms than in people. In few years, the Human Genome Project, sponsored by the NIH, will tell us what all of our genes look like. But what do they do? To find out, we must study experimentally tractable organisms. Third, time and time again truly basic studies of genes in experimental organisms have proved directly relevant to human diseases and disease genes, once we knew what those human genes looked like. An investment in such basic studies is an effective investment indeed, as it means that knowledge will proceed at an enormous pace once a human disease gene is identified. Finally, knowledge of what the counterparts of human disease genes do in an experimental organism can be directly used both in the understanding of what that gene does in people and also in the application of that knowledge to the development of a treatment of cure. I thank you for your time.

EXTENDING CERTAIN PROGRAMS
UNDER THE ENERGY POLICY
AND CONSERVATION ACT

SPEECH OF

HON. HENRY J. HYDE

OF ILLINOIS

IN THE HOUSE OF REPRESENTATIVES

Sunday, November 9, 1997

Mr. HYDE. Mr. Speaker, I ask that this exchange of letters between me and Chairman BLILEY be placed in the RECORD following debate on H.R. 2472.

HOUSE OF REPRESENTATIVES,
COMMITTEE ON COMMERCE,

Washington, DC, November 8, 1997.

Hon. HENRY J. HYDE,
Chairman, Committee on the Judiciary, U.S. House of Representatives, Washington, DC.

DEAR HENRY: Thank you for your letter regarding H.R. 2472, a bill to extend provisions of the Energy Policy and Conservation Act (EPCA) through September 1, 1998.

EPCA is one of the legislative cornerstones of our national energy security policy. Among other things, it authorizes the operation and maintenance of the Strategic Petroleum Reserve and provides limited immunity to American oil companies to participate in activities pursuant to the International Energy Agreement. In light of current actions in the Middle East and the important activities authorized by this Act, prompt passage of this EPCA extension is necessary.

I appreciate your interest in H.R. 2472 and I acknowledge that I will bring it to the

House Floor in the form of a simple extension through September 1, 1998 without any substantive change to the antitrust provisions. I also acknowledge that your action in allowing this legislation to go forward does not affect any future rights of the Committee on the Judiciary. Consistent with the Judiciary Committee's jurisdiction over antitrust issues under Rule X and with the Commerce Committee's jurisdiction over energy issues under Rule X, I would be pleased to work with you to develop legislation which ensures an effective national energy security policy.

In keeping with your request, I will place your letter and this response in the record of the debate on H.R. 2472.

Sincerely,

TOM BLILEY,
Chairman.

HOUSE OF REPRESENTATIVES,
COMMITTEE ON THE JUDICIARY,
Washington, DC, November 8, 1997.

Hon. TOM BLILEY,
Chairman, Committee on Commerce, U.S. House of Representatives, Washington, DC.

DEAR TOM: I understand that today or tomorrow you intend to move to suspend the rules and concur in the Senate amendment to H.R. 2472 with an amendment.

The version of H.R. 2472 you plan to bring up would extend through September 1, 1998 certain provisions of the Energy Policy and conservation Act, 42 U.S.C. §6201 *et seq.* Under Rule X, the Committee on the Judiciary has jurisdiction over provisions of the Act: the antitrust defense provided in Section 252, 42 U.S.C. §6272, the participation of the antitrust enforcement agencies in activities under that section, and any amendment, extension, or expansion of these provisions or any other antitrust immunity provided in the Act.

Because of the urgency of passing this important national security legislation, I am willing to waive this Committee's right to a sequential referral of H.R. 2472. I will allow this legislation to go forward so long as it remains a simple extension through September 1, 1998 without any substantive change to the existing antitrust defense or the participation of the antitrust agencies. However, my doing so does not constitute any waiver of the Committee's jurisdiction over these provisions and does not prejudice its rights in any future legislation relating to these provisions or any other antitrust immunity provided in the Act. I will, of course, insist that Members of this Committee be named as conferees on these provisions or any other antitrust immunity provided in the Act should the bill go to conference.

If the foregoing meets with your understanding of the matter, I would appreciate your placing this letter and your response in the record during the debate on H.R. 2472. Thank you for your cooperation in this matter.

Sincerely,

HENRY J. HYDE,
Chairman.

INSTITUTE FOR COMMUNITY
LIVING

HON. NYDIA M. VELÁZQUEZ

OF NEW YORK

IN THE HOUSE OF REPRESENTATIVES

Wednesday, November 12, 1997

Mr. VELÁZQUEZ. Mr. Speaker, I rise today to pay tribute to the marvelous work of the Institute for Community Living, on the occasion