44231) to describe the Medicare Coverage Advisory Committee (MCAC), which provides advice and recommendations to us about clinical issues. This notice announces the following public meeting of the Diagnostic Imaging Panel (the Panel) of MCAC.

Current Panel Members

Frank Papatheofanis, MD, PhD; Barbara McNeil, MD, PhD; Carole Flamm, MD, MPH; Jeffrey Lerner, PhD; Michael Manyak, MD; Donna Novak, BA; Manuel Cerqueira, MD; Kim Burchiel, MD; Steven Guyton, MD; Sally Hart, JD; Michael Klein, MBA

Meeting Topic

The Panel will hear and discuss presentations from interested persons regarding FDG Positron Emission Tomography imaging for breast cancer diagnosis and staging.

Procedure and Agenda

This meeting is open to the public. The Panel will hear oral presentations from the public for approximately 1.5 hours. The Panel may limit the number and duration of oral presentations to the time available. If you wish to make formal presentations, you must notify the respective Executive Secretary listed in the **FOR FURTHER INFORMATION CONTACT** section of this notice, and submit the following by the Deadline for Presentations and Comments date listed in the **DATES** section of this notice: A brief statement of the general nature of the evidence or arguments you wish to present, and the names and addresses of proposed participants. A written copy of your presentation must be provided to each panel member prior to offering your public comments. We will request that you declare at the meeting whether or not you have any financial involvement with manufacturers of any items or services being discussed (or with their competitors).

After the public and HCFA presentations, the Panel will deliberate openly on the topic. Interested persons may observe the deliberations, but the Panel will not hear further comments during this time except at the request of the chairperson. The Panel will also allow approximately a 30-minute open public session for any attendee to address issues specific to the topic. At the conclusion of the day, the members will vote and the Panel will make its recommendation.

Authority: 5 U.S.C. App. 2, section 10(a)(1) and (a)(2).

(Catalog of Federal Domestic Assistance Program No. 93.774, Medicare—Supplementary Medical Insurance Program)

Dated: April 6, 2001.

Jeffrey L. Kang,

Director, Office of Clinical Standards and Quality, Health Care Financing Administration.

[FR Doc. 01–10638 Filed 4–27–01; 8:45 am]

BILLING CODE 4120–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Proposed Collection; Comment Request; Extended Lung Cancer Incidence Follow-Up for the Mayo Lung Project Participants

SUMMARY: In compliance with the requirement of section 3506(c)(2)(A) of the Paperwork Reduction Act of 1995, for opportunity for public comment on proposed data collection projects, the National Center Institute (NCI), the National Institutes of Health (NIH) will publish periodic summaries of proposed projects to be submitted to the Office of Management and Budget (OMB) for review and approval.

Proposed Collection

Title: Extended Lung Cancer Incidence Follow-Up for the Mayo Lung Project Participants. Type of Information Collection Request: NEW. Need and Use of Information Collection: The Mayo Lung Project (MLP) was an NCI-funded randomized collection trial (RCT) of lung cancer screening conducted among 9,211 male smokers from 1971 to 1983. No reduction in lung cancer mortality was observed in the MLP with an intense regimen of x-ray and sputum cytology screening. Recent analysis of updated mortality and case survival data (through 1996) suggests that lesions with little-to-no clinical relevance (over-diagnosis) may have been detected through screening in the MLP intervention arm. Over-diagnosis leads to unnecessary medical interventions, including diagnostic and treatment procedures that carry with them varying degrees of risk. Consequently, over-diagnosis can result in considerable harm, including premature death, which would not have occurred in the absence of screening. The persistence, after screening ends, of an excess of lung cancer cases in the intervention arm is the strongest evidence in support of over-diagnosis, but this information cannot be adequately obtained with available MLP data. Therefore, we propose to re-contact the MLP participants and/or their next-of-kin to determine the participants who were diagnosed with lung cancer after the formal end of the Project. These data will allow the NCI to either more-convincingly state or perhaps refute the possibility of over-diagnosis in lung cancer screening, and may be used to guide future research agendas and lung cancer screening policies. Frequency of response: Once. Affected public: Individuals. Type of respondents: MLP participants or their next-of-kin. The annual reporting burden is as follows: Maximum number of respondents: 9200; Estimated number of Responses per Respondent: 1. Average Burden Hours Per Response: 0.25; Estimated Maximum Total Annual Burden Hours Requested: 2300. The annualized cost to respondents is estimated at zero. There are no Capital Costs to report. There are no Operating or Maintenance Costs; to report.

Request for Comments

Written comments and/or suggestions from the public and affected agencies should address one or more of the following points: (1) Evaluate whether the proposed collection of information is necessary for the proper performance of the function of the agency, including whether the information will have practical utility; (2) Evaluate the accuracy of the agency's estimate of the burden of the proposed collection of information, including the validity of the methodology and assumptions used; (3) Enhance the quality, utility, and clarity of the information to be collected; and (4) Minimize the burden of the collection of information on those who are to respond, including the use of appropriate automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

FOR FURTHER INFORMATION CONTACT: To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact: Dr. Pamela Marcus, Epidemiologist, Biometry Research Group, Division of Cancer Prevention, National Cancer Institute, Suite 344 EPN, 6130 Executive Blvd, Bethesda, MD 20892–7354; or call non-tool free 301–496–7468; or email *pm145q@nih.gov.*

Comments due date: Comments regarding this information collection are best assured of having their full effect if received on or before June 29, 2001.

Dated: April 20, 2001.

Reesa L. Nichols,

NCI Project Clearance Liaison.

[FR Doc. 01–10582 Filed 4–27–01; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, Public Health Service, DHHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by agencies of the U.S. Government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 to achieve expeditious commercialization of results of federally-funded research and development. Foreign patent applications are filed on selected inventions to extend market coverage for companies and may also be available for licensing.

ADDRESSES: Licensing information and copies of the U.S. patent applications listed below may be obtained by writing to the indicated licensing contact at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7057; fax: 301/402–0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Enhanced Homologous Recombination Mediated by Lambda Recombination Proteins

Drs. E. Lee, N. Copeland, N. Jenkins, and D. Court (NCI)
DHHS Reference No. E–077–01/0 filed Feb 26, 2001
Licensing Contact: John Rambosek; 301/496–7056 ext. 270; e-mail: rambosej@od.nih.gov

The present invention concerns a method to enhance homologous recombination in bacteria using the Red recombination system derived from a defective lambda prophage. This lambda system, like the RecET system, uses homologous recombination proteins to protect and recombine the electroporated linear DNA. However, the lambda system is at least 50 to 100 times more efficient than the RecET system. The high recombination efficiency offered by this system makes it possible to manipulate DNA without drug selection. Point mutations, deletions, or insertions can be engineered into any gene on plasmids or bacterial artificial chromosomes (BACs) for gene functional analysis. This recombination system also can be used to subclone DNA fragments as large as 80 kb from BACs by gap repair. Targeting vectors for embryonic stem cells or transgenic constructs by BAC engineering can now be subcloned with ease, and virtually any region of the engineered BAC may be included in the final subclone. The ability to efficiently and precisely modify genes or regulatory sequences on BACs, combined with the ability to include or exclude them during the subcloning process, should make it possible to dissect the function of these sequences in the whole animal at a high-through-put level not previously possible.

This lambda recombination system has been used to introduce a Cre recombinase gene into the coding region of the mouse neural-specific enolase gene carried on a 250 kb mouse BAC after transfer of the mouse BAC into DY380 E. coli cells which carry the lambda recombination system. Transgenic mice that were subsequently generated which carry this modified BAC specifically expressed Cre in all mature neurons and Cre expression mirrored that of the mouse neural-specific enolase gene.

This abstract modifies an abstract for this technology published in the **Federal Register** on Thursday, April 5, 2001 (66 FR 18098).

Use of Endogenous Vertebrate Phytase to Increase Capacity To Utilize Phytic Acid in Livestock Feed

Stephen Shears (NIEHS), Paul Reynolds, Jim Petitte
DHHS Reference No. E–139–00/0 filed Aug 11, 2000
Licensing Contact: John Rambosek; 301/496–7056 ext. 270; e-mail: rambosej@od.nih.gov

This invention discloses the concept of creating transgenic farm animals that secrete a native phytase enzyme into their digestive tracts. It has long been recognized that monogastric animals (e.g. pigs and chickens) do not utilize dietary phosphorus as efficiently as possible. This is because a high percentage of total phosphorus (70% in cereals, 50% in legume seeds) is present as phytic acid and its salts—phytate. Monogastric animals utilize phytate inefficiently because they lack the enzyme phytase in their digestive systems. Phytase liberates the phosphorus from phytate, thereby making dietary phosphorus available to the animals. This has the dual effect of both promoting more efficient growth of the animals, as well as imposing less of an environmental burden in the form of excess phosphorus in water streams.

Use of phytase as a growth feed supplement is well known. However, in the past the focus has always been on adding exogenous phytase to animal feed, or to increase the level of phytase expression in the seeds making up the feed. The inventors' novel concept is to redirect expression of a naturally occurring phytase gene so that the enzyme will be secreted into the intestinal lumen. This will create farm animals that can more efficiently utilize unsupplemented feeds. Another problem with existing phytases that the present invention overcomes is that phytase tends to be unstable during the heat treatment used to process feed. This invention overcomes this limitation because the phytase does not have to incorporated into feed at all.

Cloning of the Human Nuclear Receptor Co-Repressor Gene

Dr. Johnson M. Liu (NHLBI)
DHHS Reference Number E–088–99/0 filed Aug 3, 1999
Licensing Contact: John Rambosek; 301/496–7056 ext. 270; e-mail: rambosej@od.nih.gov

Alteration in the expression of human genes is critical to the development and progression of many diseases. These include, among others, cancer, inflammation, cardiovascular disease, hypercholesterolemia, blood pressure, and diabetes. The Human Nuclear Receptor Co-Repressor (HuN-Cor) gene represents a technology that may be used to alter the transcription of genes. It provides a general mechanism by which many genes may be modulated throughout the entire range of being turned on to being completely turned off. The HuN-Cor gene encodes for a ubiquitously expressed protein that silences other genes. It does this by specifically recruiting an enzyme complex that causes local folding of chromatin, not allowing other transcription factors to access the DNA. HuN-Cor represents a powerful research tool that can be used to study gene expression and characterization of many different genes. It may ultimately have great utility in controlling gene expression via gene therapy technology, and may also be useful as a target for the isolation of pharmaceutical compounds that enhance or inhibit expression of genes. For example, it may be possible to engineer mutations of the HuN-Cor gene that dominantly inhibit its function; these mutants could then be expressed in appropriate target tissues or cells in order to control gene expression. Finally, the gene product may have utility in the discovery of therapeutic compounds that modulate gene expression via HuN-Cor.

Antibodies That Selectively Detect the Human Nestin Protein

Conrad Messam et al. (NINDS)