Biological Products Advisory Committee were available at this time, the Commissioner of Food and Drugs concluded that it was in the public interest to hold this meeting even if there was not sufficient time for the customary 15-day public notice. Notice of this meeting is given under the Federal Advisory Committee Act (5 U.S.C. app. 2).

Dated: February 17, 2002.

Linda A. Suydam,
Senior Associate Commissioner for Communications and Constituent Relations.

[FR Doc. 02-3476 Filed 2-20-02; 1:27 pm]

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DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Submission for OMB Review; Comment Request; Study of Testicular Germ Cell Cancer in U.S. Military Servicemen: Substudy of Maternal Risk Factors

SUMMARY: Under the provisions of section 3607(a)(1)(D) of the Paperwork Reduction Act of 1995, the National Cancer Institute (NCI), the National Institutes of Health (NIH) has submitted to the Office of Management and Budget (OMB) a request for review and approval of the information collection listed below. This proposed information collection was previously published in the Federal Register on August 23, 2001, page 44362 and allowed 60 days for public comment. One public comment was received that is being addressed in the study. The purpose of this notice is to allow an additional 30 days for public comment. The National Institutes of Health may not conduct or sponsor, and the respondent is not required to respond to, an information collection that has been extended, revised, or implemented on or after October 1, 1995, unless it displays valid OMB control number.

Proposed Collection

Title: Study of Testicular Germ Cell Cancer in U.S. Military Servicemen: Substudy of Maternal Risk Factors. Type of Information Collection Request: NEW. Need and Use of Information Collection: This study will seek to determine the causes of testicular germ cell cancer. The incidence rate of testicular cancer has been increasing for most of the twentieth century. It is the most common tumor among men between the ages of 15 and 35 years, yet its risk factors remain poorly understood. Servicemen are being studied because they are the right age group and testicular cancer is the common cancer among men in the service. The cancer’s relatively young age of onset and its association with several congenital anomalies indicate that events during in-utero life may place men at risk of this tumor. Therefore, this study seeks to interview the mothers of men who developed testicular cancer and mothers of men who did not develop testicular cancer. Mothers will be asked about events surrounding pregnancy with the son and early life events.

Frequency of Response: One interview is requested. Affected Public. Individuals. Type of Respondents: Mothers of servicemen who were diagnosed with testicular cancer and mothers of servicemen who were not diagnosed with testicular cancer. The annual reporting burden is as follows:

Estimated Number of Respondents: 520;

Estimated Number of Responses per Respondent: 1; Average Burden Hours Per Response: 1.0; and

Estimated Total Annual Burden Hours Requests: 520. The annualized cost to respondents is estimated at: $0. 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 are invited on one or more of the following points: (1) 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) 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) Ways to enhance the quality, utility, and clarity of the information to be collected; and (4) Ways to 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.

Direct Comments to OMB

Written comments and/or suggestions regarding the item(s) contained in this notice, especially regarding the estimated public burden and associated response time, should be directed to the:

Office of Management and Budget, Office of Regulatory Affairs, New Executive Office Building, Room 10235, Washington, DC 20503, Attention: Desk Officer for NIH. To request more
Cytochromes P450 catalyze the NADPH-dependent oxidation of arachidonic acid to various eicosanoids found in several species. The eicosanoids are biosynthesized in numerous tissues including pancreas, intestine, kidney, heart, and lung where they are involved in many different biological activities.

The NIH announces cloned cDNAs for several different CYP2J subfamily members and specific peptide-based antibodies to the P450s proteins. The reagents available for licensing include: human CYP2J2 cDNA, rat CYP2J3 cDNA, mouse CYP2J5 cDNA, mouse CYP2J9 cDNA, anti-CYP2J2rec, anti-CYP2J2pep2, anti-CYP2J3pep2, anti-CYP2J5pep, anti-CYP2J6pep, and insect cell microsomes expressing catalytically active CYP2J2. These reagents can be used to examine the expression of the CYP2J subfamily at the RNA and protein level and can be used to screen drugs for possible metabolism by the CYP2J2 subfamily P450s and/or to identify endogenous substrates for the enzyme. The recombinant protein may also be used to investigate cross-reactivity for other antibodies.

Polyclonal Antibody to Detect Human Membrane-Bound Prostaglandin E Synthase
Dr. Thomas Eling et al. (NIEHS)
DHHS Reference No. E–032–02/0–
Research Tool
Licensing Contact: Marlene Shinn; 301/496–7056 ext. 285; e-mail: shinnm@od.nih.gov

Prostaglandin endoperoxide H2 (PGH2) is formed from arachidonic acid by the action of cyclooxygenases (cox)-1 or -2. Human prostaglandin E synthase (PGES) is a member of a protein superfamily consisting of membrane-associated proteins involved in eicosanoid and glutathione metabolism. PGES; a specific prostaglandin, is formed from PGH2 by PGES and is then further metabolized into various eicosanoids. It has been reported that the membrane-bound mPGES is linked to cox-2 protein, which may be induced by proinflammatory cytokines such as IL–β at sites of inflammation.

The NIH announces a polyclonal antibody capable of detecting human mPGES. It is anticipated that the use of this antibody in western analysis, immunostaining and immunoprecipitation studies will aid researchers in understanding prostaglandin creation and could eventually lead to the development of new anti-inflammatory agents.

Amyloid Beta is a Ligand for FPR Class Receptors
Dr. Ji Ming Wang et al. (NCI)
Licensing Contact: Marlene Shinn; 301/496–7056 ext. 285; e-mail: shinnm@od.nih.gov

Alzheimer’s disease is the most important dementing illness in the United States because of its high prevalence. Five to ten percent of the United States population 65 years and older are afflicted with the disease. In 1990 there were approximately 4 million individuals with Alzheimer’s, and this number is expected to reach 14 million by the year 2050. It is the fourth leading cause of death for adults, resulting in more than 100,000 deaths annually. Amyloid beta has been identified as playing an important role in the neurodegeneration of Alzheimer’s disease. However, the mechanism by which this occurred was unknown, but has been postulated to be either direct or indirect through an induction of inflammatory responses.

The NIH announces the identification of the 7-transmembrane, G-protein-coupled receptor, FPR–1, in the cellular uptake and fibrillar aggregation of amyloid ββ (Aββ) peptides. The Aββ peptides use the FPR1–1 receptor to attract and activate human monocytes and mouse microglial cells (publications referenced below), and have been identified as a principal component of the amyloid plaques associated with Alzheimer’s disease. In addition, the known anti-inflammatory drug, Colchicine, has been shown to inhibit the FPR1 activation by amyloid and the internalization of FPR1/amyloid beta complexes.


System for in vivo Site-Directed Mutagenesis Using Oligonucleotides
Dr. Francesca Storici et al. (NIH)
DHHS Reference No. E–204–01/0 filed 27 Jul 2001
Licensing Contact: Marlene Shinn; 301/496–7056 ext. 285; e-mail: shinnm@od.nih.gov

Through the use of molecular techniques to induce mutagenesis, along