Paperwork Reduction Act of 1995: Data collection requirements have been approved by the Office of Management and Budget (OMB) and are in effect, as required under the Paperwork Reduction Act of 1995 (OMB No. 0915–0272).

Executive Order 12372: The MCH Federal Set-Aside program has been determined to not be a program which is subject to the provisions of Executive Order 12372 concerning intergovernmental review of Federal programs.

Dated: November 28, 2003.

Elizabeth M. Duke,

Administrator.

[FR Doc. 03–30597 Filed 12–5–03; 12:05 pm]

BILLING CODE 4165–15–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Proposed Collection; Comment Request; Submission for OMB Review; Comment Request; Request for Generic Clearance To Collect Medical Outcome and Risk Factor Data From a Cohort of U.S. Radiologic Technologists

SUMMARY: Under the provisions of section 3507(a)(1(D) of the Paperwork Reduction Act of 1995, the National Cancer Institute, the National Institutes of Health (NIH) has submitted to the Office of Management and Budget (OMB) a request to review and approve the information collection listed below. This proposed information collection was previously published in the **Federal Register** on August 26, 2003, pages 51275–51276 and allowed 60 days for public comment. No public comments were received. 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 a currently valid OMB control number.

Proposed Collection: *Title:* Request for Generic Clearance to Collect Medical Outcome and Risk Factor Data from a Cohort of U.S. Radiologic Technologists. *Type of Information Collection Request:* Reinstatement with change of a previously approved collection (OMB) No. 0925–0405 expired 09/30/1999). Need and Use of Information Collection. The primary aim of this project is to substantially increase knowledge about the long-term health affects associated with protracted low- to moderate-dose radiation exposures. With this submission, the NIH, Office of Communications and Public Liaison, seeks to obtain OMB's generic approval to conduct occasional surveys of a cohort of U.S. radiologic technologist to ascertain incident cancers, benign conditions associated with high risk of cancer, and selected other health outcomes, as well as demographic, lifestyle, reproductive, employment, and other characteristics that may influence health risks. Researchers an the National Cancer Institute and the University of Minnesota have followed a nationwide cohort of 146,000 radiologic technologist since 1982, of whom 110,000 completed at least one of two prior questionnaire surveys and 17,000 are deceased. This cohort is unique because estimates of cumulative radiation dose to specific organs (*e.g.,* breast) are available and the cohort is largely female, offering a rare opportunity to study effects of low-dose radiation exposure on breast and thyroid cancers, the two most sensitive organ sites for radiation carcinogenesis in women. Primary objectives are to quantify radiation dose-response for: (1) Cancers of the breast, thyroid, other radiogenic sites or histologies, and other cancers; (2) benign breast disease, thyroid nodules, and other benign conditions associated with increased cancer risk; and (3) other selected health outcomes that may be related to radiation exposure (*e.g.,* cardiovascular disease). Findings from this study will address an important gap in the scientific understanding of radiation dose-rate affects, *i.e.,* whether cumulative exposures of the same magnitude have the same health effects when received in single or a few doses over a very short period of time (as in atomic bomb or therapeutic exposures) or in many small doses over a protracted period of time (as in medical or nuclear occupational settings). The first survey will be mailed in 2004 to approximately 100,000 living cohort members who completed at least one prior survey and will collect information on: (1) Medical outcomes (as described above) to assess radiation-related risks; (2) detailed job-specific frequency of performing high-dose procedures (*e.g.,* handling isotopes), use of protective measures (*e.g.,* using lead aprons or standing behind shields), and other work practices (*e.g.,* holding patients for x-rays) to refine the organ dose estimates and associated uncertainty distributions; and (3) behavioral, susceptibility, and residential histories for refining estimates of lifetime ultraviolet (UV) radiation exposure to assess in greater detail the risks of melanoma and non-melanoma skin cancer associated with UV and ionizing radiation exposures, separately and jointly. Subsequent surveys will collect updated information on medical outcomes and risk factors of interest at that time. All surveys will be in optical-read format for computerized data capture. *Frequency of Response:* On occasion. *Affected Public:* U.S. radiologic technologists who have willingly participated in earlier investigations to quantify the carcinogenic risks of protracted low- to moderate-dose occupational radiation exposures. The annual reporting burden is as follows: *Estimated Number of Respondents:* 59,200. *Estimated Number of Responses Per Respondent:* 1. *Average Burden Hours per Response:* 0.4983. *Annual Burden Hours Requested:* 29,500. There are no capital costs, operating costs and/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.

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 information on the proposed project or to obtain a copy of the date collection plans and instruments, contact: Michele M. Doody, Radiation Epidemiology Branch, National Cancer Institute, Executive Plaza South, Room 7040, Bethesda, MD 20892–7238, or call non-toll-free at (301) 594–7203 or e-mail

your request, including your address to: *doodym@exchange.nih.gov*.

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 30 days of the date of this publication.

Dated: December 1, 2003.

Reesa Nichols,

NCI Project Clearance Liaison.

[FR Doc. 03–30495 Filed 12–8–03; 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 an agency 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.

A Mouse Model for Systemic Inflammation in Glucocerebrosidase-Deficient Mice With Minimal Glucosylceramide Storage

Richard L. Proia (NIDDK). DHHS Reference No. E–256–2003/0—Research Tool. *Licensing Contact:* Susan Carson; (301) 435–5020; *carsonsu@mail.nih.gov.*

Gaucher disease, the most common lysosomal storage disease, is an inherited metabolic disorder in which harmful quantities of the lipid glucocerebroside accumulate in the spleen, liver, lungs, bone marrow and in rare cases in the brain, due to a deficiency of the enzyme glucocerebrosidase (Gba) that catalyses the first step in the biodegradation of glucocerebrosides. Type 1 Gaucher disease is the most common and is distinguished from the other forms of the disease, types 2 and 3, by the lack of neurologic involvement. The clinical features of Type 1 are heterogeneous, vary broadly in clinical severity and affect many organ systems. The major disease manifestations include enlarged spleen and liver, bone lesions, hematologic abnormalities and lung involvement. The disease has also been associated with a sustained inflammatory reaction. Gaucher disease is most prevalent in the Ashkenazi Jewish population with an incidence of approximately 1 in 450 persons while in the general public the incidence is 1 in 100,000. There are an estimated 30,000 Gaucher disease patients world-wide with approximately 3000 patients currently receiving enzyme replacement therapy which has been shown to be highly effective in treatment of the disease. The cost of therapy is approximately $100,000-$300,000 annually and is a life-long treatment, which makes the case for affordable new therapies urgent.

The etiology of the disease has been difficult to study due to the absence of viable mouse models for the disease, as a complete disruption of the glucocerebrosidase (Gba) gene results in rapid neonatal death. In an attempt to produce a viable model scientists at the NIDDK introduced a human Gaucher disease point mutation, L444P, into the mouse Gba gene in order to cause a partial enzyme deficiency (J. Clin. Invest (2002) 109, 1215–1221; Proc. Natl. Acad. Sci. USA (1998) 95, 2503–2508).

The mice exhibit a partial glucocerebrosidase deficiency (15–20% of normal activity), without bulk accumulation of glucosylceramide or the presence of Gaucher cells. The mice demonstrate other clinical features of Gaucher disease, including multisystem inflammation, B cell hyperproliferation, skin abnormalities, anemia and lymphadenopathy. These mice provide a useful model for studying certain aspects of Gaucher disease pathology and in evaluating new therapeutic treatments.

Tec Kinase Deficient Mice

Pamela L. Schwartzberg (NHGRI), Michael J. Lenardo (NIAID), Harold Varmus (EM), Dan Littman (EM).

DHHS Reference No. E–178–2003/0 and DHHS Reference No. E–178–2003/1—Research Tools.

Licensing Contact: Susan Carson; (301) 435–5020; *carsonsu@mail.nih.gov.*

Stimulation of T lymphocytes through the T Cell Receptor (TCR) elicits broad responses required for proper immune function, including cell proliferation, cytokine production and apoptosis. Activation of distinct families of tyrosine kinases (Zap-70, Src) are important in TCR signalling, while the role of other tyrosine kinases, such as the Tec Kinases Rlk and Itk is less clear. However, evidence suggests that these kinases play a role in CD4+ T helper (Th) cell differentiation. Responses to infection are regulated in part by two distinct types of T helper cells, type 1 (Th1) and Th2 subclasses which produce different cytokines and have discrete effector functions. Th1 cells produce interferon-gamma (IFN-gamma), which is a key mediator of cellular immunity. In contrast Th2 cells produce interleukin 4 (IL–4), Il–5, Il–10, and Il–13 which assist humoral immunity and dominate immune responses to both helminths and allergens. Regulation of these subclasses is important not only for normal immune response, but also for abnormal disease processes, including autoimmunity and hypersensitivity. Generation of type 1 and type 2 Th cells is influenced by multiple factors including cytokines, costimulation and TCR-based signals. Understanding the mechanisms and signals important in T cell signalling is important for identifying new therapeutics that target Th1 and Th2-mediated pathologies (for example autoimmune disorders and asthma, respectively).

The Tec family of tyrosine kinases have been implicated as important mediators of polarized cytokine production and Th2 cell differentiation. Rlk is preferentially expressed in Th1 cells and Itk is important in Th2 response. Numerous studies have implicated alterations in the strength of TCR-mediated signals as playing important roles in Th cell differentiation. Researchers at the NIH have developed transgenic mouse models in order to address these issues. Rlk-deficient mice and Rlk/Itk double-deficient mice were generated and have been shown to have defects in TCR responses including proliferation, cytokine production and apoptosis in vitro and adaptive immune response to infectious agents in vivo (Science (1999) 284, 638–641; Nature Immunol (2001) 2: 1183–188). Molecular analyses of cells from these mice indicate that these kinases are critical for proper regulation of phospholipase C, calcium mobilisation and ERK activation as well as activation of downstream transcription factors in response to T cell receptor stimulation. Defects are minor in Rlk-deficient animals and most severe in Rlk/Itk double-deficient mice.