

proposed data collection projects, the National Cancer 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: Health Information National Trends Survey 2007 (HINTS 2007).

Type of Information Collection

Request: New.

Need and Use of Information

Collection: Building on the first two rounds of HINTS data collection, HINTS 2007 will continue to provide NCI with a comprehensive assessment of the American public's current access to, and use of, information about cancer, including cancer prevention, early detection, diagnosis, treatment, and prognosis. The content of the survey

will focus on understanding the degree to which members of the general population understand vital cancer prevention messages. More importantly, this NCI survey will couple knowledge-related questions with inquiries into the communication channels through which understanding is being obtained. HINTS is intended to be the foundation of NCI's effort to build on the opportunities presented by a national shift in communication context, and by so doing, improve the nation's ability to reduce the national cancer burden. Data will be used (1) To understand individuals sources of and access to cancer-related information; (2) to measure progress in improving cancer knowledge and communication to the general public; (3) to develop appropriate messages for the public about cancer prevention, detection, diagnosis, treatment, and survivorship;

and (4) to identify research gaps and guide decisions about NCI's research efforts in health promotion and health communication.

Frequency of Response: One time.

Affected Public: Individuals.

Type of Respondents: U.S. Adults.

The annual reporting burden is as follows:

Estimated Number of Respondents: 10,599.

Estimated Number of Responses per Respondent: 1.

Average Burden Hours per Response: .33.

Estimated Total Annual Burden

Hours Requested: 3,576.

The annualized cost to respondents is estimated at: \$35,760. There are no Capital Costs to report. There are no Operating or Maintenance Costs to report.

| Type of respondent | Estimated number of respondents | Frequency of response | Average hours per response | Annual hour burden |
|--|---------------------------------|-----------------------|----------------------------|--------------------|
| Pilot RDD Screener | 250 | 1 | .0833 | 21 |
| Pilot RDD Interview* | 150 | 1 | .4167 | 63 |
| Pilot Mail Survey | 150 | 1 | .3333 | 50 |
| RDD Screener | 5,833 | 1 | .0833 | 486 |
| RDD Interview* | 3,500 | 1 | .4167 | 1,458 |
| Mail Survey | 3,660 | 1 | .3333 | 1,219 |
| Telephone Screener for Followup of Mail | 956 | 1 | .0833 | 80 |
| Telephone Interview for Follow-up of Mail* | 478 | 1 | .4167 | 199 |
| Totals | | | | 3,576 |

* Pilot survey and HINTS 2007 RDD interview respondents are a subset of the RDD screener respondents. Similarly, the telephone interview respondents in the followup of mail nonrespondents are a subset of the telephone screener respondents in the followup of mail nonrespondents. N = 10,849.

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.

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 Bradford W. Hesse, Ph.D., Project Officer, National Cancer Institute, NIH, EPN 4068, 6130 Executive Boulevard MSC 7365, Bethesda, Maryland 20892-7365, or call non-toll-free number 301-594-9904, or FAX your request to 301-480-2198, or E-mail your request, including your address, to hesseb@mail.nih.gov.

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 60-days of the date of this publication.

Dated: October 18, 2006.

Rachelle Ragland-Greene,

NCI Project Clearance Liaison, National Institutes of Health.

[FR Doc. E6-17964 Filed 10-25-06; 8:45 am]

BILLING CODE 4101-01-P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

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

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.

Manganese Superoxide Dimutase VAL16ALA Polymorphism Predicts Resistance to Doxorubicin Cancer Therapy

Description of Technology: Cancer is the second leading cause of death in the United States and it is estimated that there will be approximately 600,000 deaths caused by cancer in 2006. Major drawbacks of the existing cancer therapies are the interindividual differences in the response and the cytotoxic side-effects that are associated with them. Thus, there is a need to develop new therapeutic approaches to optimize treatment and increase patient survival.

This technology describes the identification of a manganese superoxide dismutase (MnSOD) polymorphism as a novel biomarker for the prognosis of doxorubicin therapeutic response in breast cancer patients, wherein a Val16Ala polymorphism of MnSOD is indicative of patient survival. More specifically, patients undergoing doxorubicin combination therapy with Val/Val, Val/Ala, and Ala/Ala genotypes had 95.2%, 79%, and 45.5% survival rates, respectively, in a case study of 70 unselected breast cancer patients. Carriers of the Ala/Ala genotype had a highly significantly poorer breast cancer-specific survival in a multivariate Cox regression analysis than carriers of the Val/Val genotype. This technology can be developed into an assay to screen for breast cancer patients who will be responsive to doxorubicin treatment. Further, as the MnSOD polymorphism is common in the population (15% to 20% of patients have the Ala/Ala genotype), it is a common risk factor for doxorubicin therapy. This technology can potentially be utilized as a screening tool applicable for all cancer types treated with doxorubicin.

Applications: (1) A novel genetic marker that can predict breast cancer patient survival with doxorubicin treatment; (2) A screening test based on MnSOD Val16Ala genotype that predicts patient response to doxorubicin cancer therapy, wherein treatment can be subsequently individualized according to patient MnSOD genotype.

Development Status: Future studies include determining the mechanism in

which the polymorphism modulates doxorubicin toxicity.

Inventors: Stefan Ambs and Brenda Boersma (NCI).

Patent Status: U.S. Provisional Application No. 60/799,788 filed 11 May 2006 (HHS Reference No. E-137-2006/0-US-01).

Licensing Status: Available for non-exclusive or exclusive licensing.

Licensing Contact: Jennifer Wong; 301/435-4633; wongje@mail.nih.gov.

Collaborative Research Opportunity: The Laboratory of Human Carcinogenesis, Center for Cancer Research, National Cancer Institute, National Institutes of Health, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize MnSOD genotyping assays to assess a patient's response to doxorubicin combination therapy. Please contact Betty Tong, Ph.D. at 301-594-4263 or tongb@mail.nih.gov for more information.

A Novel Magnetic Resonance Radio-Frequency Coil Array that Eliminates Inductive Coupling

Description of Technology: Parallel magnetic resonance imaging (MRI) techniques employ RF coil arrays for faster data acquisition, and have been shown to reduce the overall length of MRI procedures, improve signal-to-noise ratio (SNR) and image quality, thus making MRI more attractive and less costly. Elimination of inductive coupling is an essential step in designing RF coil arrays for parallel MRI. If mutual inductance remains among coils in the RF coil array, the MR signal obtained from one coil may disturb the flux in another coil, making it difficult to match the impedance of each individual element to the input impedance its preamplifier. This non-optimal matching can lead to degradation of MR signal thereby yielding images with low quality. The most common strategy for inductive decoupling involves the use of preamplifiers with very low input impedance and decoupling networks with lumped elements. However, the construction of preamplifiers with low input impedance is not easy to accomplish, and these preamplifiers impose technical restrictions on coil design, requiring the use of overlapping loops to further minimize the amount of mutual inductance between the coils.

The present invention describes a novel RF coil circuitry scheme to remove inductive coupling and to overcome the limitations of having to use overlapping geometries and low-impedance preamplifiers. The coil array

employs a transformer to match the input impedance of the preamplifier. The signal that reaches the preamplifier is coupled in an inductive fashion to the RF coil decoupling network through the transformer's primary coil. Because primary and secondary coils in the transformer are isolated, the preamplifier circuit (and the MRI scanner electronics) is electrically isolated from the MR pickup coil. This arrangement provides a perfect electrical balance and isolation between the array channels, thus making it unnecessary to use traps and balluns in the circuit. At 7T, a 4-channel small animal coil array implementing the novel circuitry provided images with excellent SNR and demonstrated isolation of all individual RF coils and immunity to standing waves and other parasitic signals.

Applications: (1) MR imaging of humans, including imaging of brain; (2) MR imaging of animals, including non-human primates and rodents; (3) Functional imaging of humans and animals.

Advantages: (1) Allows for increased flexibility of coil design including geometries that require array with overlapping receiver coil loops; (2) Can provide high level of mutual inductance decoupling within coils in the array; (3) Isolates the grounds from coil to coil, and cancels all ground loops related to the coil array; (4) Greatly increases the signal to noise ratio in MR imaging.

Development Status: Early stage; Working model made and tested, improved model for animals under testing.

Inventors: George C. Nascimento and Afonso C. Silva (NINDS).

Patent Status: U.S. Provisional Application No. 60/789,934 filed 30 Mar 2006 (HHS Reference No. E-099-2006/0-US-01).

Licensing Status: Available for non-exclusive or exclusive licensing.

Licensing Contact: Chekeshia S. Clingman, Ph.D.; 301/435-5018; clingmac@mail.nih.gov.

PDE11A as a Novel Therapeutic Target for Inherited Form of Cushing Syndrome and Endocrine Tumors

Description of Technology: Cushing Syndrome, a disorder associated with excess production of a steroid hormone, cortisol, affects up to 10 per 15 million people every year. Cushing Syndrome may be caused by several reasons such as cortisol-producing endocrine tumors and can be inherited in some instances. Surgery of the adrenal tumor is the most common method of treatment. New diagnostic and therapeutic approaches

need to be developed for successful management of the disease.

This technology describes the clinical identification of a new disease termed "isolated micronodular adrenocortical disease" (iMAD), as well as the role of PDE11A gene in this disease.

Additionally, the technology also identifies particular sequence variants of the PDE11A gene associated with abnormal or altered function of the gene, PDE11A as a potential novel drug target for the treatment of bilateral adrenal hyperplasia, and possibly other endocrine tumors and malignancies.

Applications and Modality: (1) Identification of PDE11A gene and sequence variants for the diagnosis of "isolated micronodular adrenocortical disease" (iMAD), a form of Cushing Syndrome and endocrine tumors, *i.e.*, as diagnostic tool. (2) Identification of PDE11A as a potential novel drug target for the treatment of bilateral adrenal hyperplasia and other endocrine and non-endocrine tumors and malignancies.

Market: (1) 5 to 10 per 15 million 10 to 15 million new cases of Cushing Syndrome every year; (2) 27,000 new cases of endocrine tumors every year; (3) The technology involving PDE11A genes for the diagnosis and treatment of endocrine tumors including Cushing syndrome; (4) The endocrine drug market is more than 40 billion U.S. dollars.

Development Status: The technology is currently in the pre-clinical stage of development.

Inventor: Dr. Constantine A. Stratakis (NICHD).

Publication: A Horvath *et al.* A genome-wide scan identifies mutations in the gene encoding phosphodiesterase 11A4 (PDE11A) in individuals with adrenocortical hyperplasia. *Nat Genet.* 2006 Jul;38(7):794–800. Epub 2006 Jun 11, doi:10.1038/ng1809. [*PubMed abs*]

Patent Status: U.S. Provisional Application No. 60/761,446 filed 24 Jan 2006 entitled "PDE11A mutations in Adrenal Diseases" (HHS Reference No. E-027-2006/0-US-01).

Licensing Status: Available for exclusive and non-exclusive license.

Licensing Contact: Mojdeh Bahar; 301/435-2950; baharm@mail.nih.gov.

Collaborative Research Opportunity: The NICHD Heritable Disorders Branch is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize testing for PDE11A genetic or functional defects in endocrine disease, and endocrine and other tumors or cancers. Please contact Betty Tong, Ph.D. at 301-

594-4263 or tongb@mail.nih.gov for more information.

2-Amino-*O*⁴-Substituted Pteridines: Improved Chemotherapy Adjuvants

Description of Technology: *O*⁶-Benzylguanine derivatives, some *O*⁶-benzylpyrimidines, and related compounds are known to be inactivators of the human DNA repair protein *O*⁶-alkylguanine-DNA alkyltransferase (alkyltransferase). This repair protein is the primary source of resistance many tumor cells develop when exposed to chemotherapeutic agents that modify the *O*⁶-position of DNA guanine residues. Therefore, inactivation of this protein can bring about a significant improvement in the therapeutic effectiveness of these chemotherapy drugs. The prototype inactivator *O*⁶-benzylguanine is currently in clinical trials in the United States as an adjuvant in combination with the chloroethylating agent 1, 3-bis (2-chloroethyl)-1-nitrosourea (BCNU) and the methylating agent temozolomide. A similar alkyltransferase inactivator, *O*⁶-(4-bromothenyl) guanine is in clinical trials in the UK.

This technology is directed to the discovery of a new class of potent alkyltransferase inactivators, 2-amino-*O*⁴-benzylpteridine derivatives targeted for use in cancer treatment in combination with chemotherapeutic agents such as 1, 3-bis (2-chloroethyl)-1-nitrosourea (BCNU) or temozolomide. The derivatives of the present invention inactivate the *O*⁶-alkylguanine-DNA-alkyltransferase repair protein and thus enhance activity of such chemotherapeutic agents. Some of the derivatives are water soluble and possess tumor cell selectivity in particular by inactivating alkyltransferase in tumor cells that overexpress folic acid receptors. The 2-amino-*O*⁴-benzylpteridine derivatives represent a promising new class of alkyltransferase inactivator with representatives that may be great candidates as chemotherapy adjuvants.

Applications and Modality: (1) New small molecules as alkyltransferase inactivators based on 2-amino-*O*⁴-benzylpteridine compounds; (2) Promising candidates as chemotherapy adjuvants for the treatment of cancer; (3) Therapeutic application for drug resistant tumors where acquired resistance is caused by *O*⁶-alkylguanine-DNA alkyltransferase.

Market: (1) 600,000 deaths from cancer related diseases estimated in 2006; (2) This technology involving small molecule therapeutics for the treatment of several cancers has a

potential market of several billion U.S. dollars.

Development Status: The technology is currently in the pre-clinical stage of development.

Inventors: Robert C. Moschel (NCI) *et al.*

Publication: ME Nelson, NA Loktionova, AE Pegg, RC Moschel. 2-amino-*O*⁴-benzylpteridine derivatives: potent inactivators of *O*⁶-alkylguanine-DNA alkyltransferase. *J Med Chem.* 2004 Jul 15;47(15):3887–3891. Epub 2004 Jun 18, doi 10.1021/jm049758+S0022-2623(04)09758-4.

Patent Status: U.S. Provisional Application No. 60/534,519 filed 06 Jan 2004 (HHS Reference No. E-274-2003/0-US-01); U.S. Patent Application No. 10/585,566 filed 06 Jul 2006 (HHS Reference No. E-274-2003/0-US-03); Foreign equivalents.

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: Adaku Madu, J.D.; 301/435-5560; madua@mail.nih.gov.

Retrovirus-Like Particles as Vaccines and Immunogens

Description of Technology: This technology describes retrovirus-like particles and their production from retroviral constructs in which the gene encoding all but seven amino acids of the nucleocapsid (NC) protein was deleted. NC is critical for both genomic RNA packaging into the virion and viral integration into the host cell. Therefore, this deletion functionally eliminates two essential steps in retrovirus replication, thereby resulting in non-infectious retrovirus-like particles that maintain their full complement of antigenic proteins. Furthermore, efficient formation of these particles requires inhibition of the protease enzymatic activity, either by mutation to the protease gene in the construct or by protease inhibitor thereby ensuring the production of non-infectious retrovirus-like particles by altering two independent targets. These particles can be used in vaccines or immunogenic compositions. Specific examples using HIV-1 constructs are given.

Applications: Retroviral vaccine; Immunogenic compositions.

Development Status: In vitro data available.

Inventor: David E. Ott (NCI).

Publications:

1. DE Ott *et al.* Elimination of protease activity restores efficient virion production to a human immunodeficiency virus type 1 nucleocapsid deletion mutant. *J Virol.* 2003 May;77(10):5547–5556. [*PubMed abs*]

2. DE Ott *et al.* Redundant roles for nucleocapsid and matrix RNA-binding sequences in human immunodeficiency virus type 1 assembly. *J Virol.* 2005 Nov;79(22), 13839–13847. [*PubMed abs*] Patent Status: U.S. Patent Application No. 11/413,614 filed 27 Apr 2006 (HHS Reference No. E–236–2003/0–US–02).

Licensing Status: Available for non-exclusive or exclusive licensing.

Licensing Contact: Susan Ano, Ph.D.; 301/435–5515; anos@mail.nih.gov.

Collaborative Research Opportunity: The NCI, CCR, AIDS Vaccine Program is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize whole retrovirus-like particle vaccines. Please contact Betty Tong, Ph.D. at 301–594–4263 or tongb@mail.nih.gov for more information.

Dated: October 19, 2006.

Steven M. Ferguson,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.

[FR Doc. E6–17966 Filed 10–25–06; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HOMELAND SECURITY

Coast Guard

[USCG–2006–24851]

Draft Environmental Assessment, Draft Finding of No Significant Impact, and Draft Memorandum of Agreement for the Decommissioning and Excessing of the U.S. Coast Guard Cutters STORIS (WMEC–38) and ACUSHNET (WMEC–167)

AGENCY: Coast Guard, DHS.

ACTION: Notice of availability and request for comments.

SUMMARY: The U.S. Coast Guard (USCG) announces the availability of, and seeks comment on, the Environmental Assessment and Draft Finding of No Significant Impact for the proposed decommissioning of the USCG cutters STORIS (WMEC–38) and ACUSHNET (WMEC–167) in Ketchikan and Kodiak, Alaska. The USCG is also announcing the availability and seeking comment on a related Draft Memorandum of Agreement (MOA) with the Alaska State Historic Preservation Office (AK SHPO) and the General Services Administration (GSA).

DATES: Comments and related material must reach Coast Guard Headquarters on or before November 27, 2006.

ADDRESSES: Please submit comments by only one of the following means:

(1) By e-mail to Susan Hathaway at Susan.G.Hathaway@uscg.mil.

(2) By conventional mail delivery to Susan Hathaway, Headquarters, United States Coast Guard, Assistant Commandant for Engineering and Logistics, Environmental Management (CG–443), 2100 Second St., SW., Rm. 6109, Washington, DC 20593.

(3) By fax to Susan Hathaway at (202) 475–5956.

(4) Through the Web Site for the Docket Management System at <http://dms.dot.gov>. The Docket Management Facility maintains the public docket. Comments will become part of this docket and will be available for inspection or copying at the Nassif Building, 400 Seventh Street, SW., Room PL–401, Washington, DC between 9 a.m. and 5 p.m., Monday through Friday, except for Federal holidays. You may also view this docket, including this notice and comments, on the Internet at <http://dms.dot.gov>. Click on Simple Search and enter the docket number (24851).

FOR FURTHER INFORMATION CONTACT: By mail: Susan Hathaway, Headquarters, United States Coast Guard, Assistant Commandant for Engineering and Logistics, Environmental Management (CG–443), 2100 Second St., SW., Rm. 6109, Washington, DC 20593; by telephone: (202) 475–5688; by fax: (202) 475–5956; or by e-mail: Susan.G.Hathaway@uscg.mil.

To view and download the Environmental Assessment (EA), Draft Finding of No Significant Impact (FONSI), and Memorandum of Agreement (MOA), please go to <http://www.uscg.mil/systems/gse/NEPAhot.htm> and scroll to ACUSHNET and STORIS Decommissioning EA for Public Review. The EA, Draft FONSI, and MOA can also be viewed and downloaded from the Docket Management System at <http://dms.dot.gov>. Click on Simple Search and enter the docket number (24851). The Draft FONSI is after the cover sheet at the front of the EA and the MOA is Appendix D of the EA.

SUPPLEMENTARY INFORMATION:

Request for Comments

We encourage you to submit comments on the EA, Draft FONSI, and MOA. If you do so, please include your name and address, identify the docket number for this notice (USCG–2006–24851), and give the reasons for each comment. You may submit your comments by mail, hand delivery, fax, or electronic means to the Docket

Management Facility at the addresses under **ADDRESSES** but please submit your comments by only one means. If you submit them by mail or hand delivery, submit them in an unbound format, no larger than 8½ by 11 inches, suitable for copying and electronic filing. If you submit them by mail and would like to know they reached the Facility, please enclose a stamped, self-addressed postcard or envelope. We will consider all comments received during the comment period.

Proposed Action

After over 60 years of continuous service, the USCGCs STORIS (WMEC–38) and ACUSHNET (WMEC–167) have reached the end of their service lives. The USCG intends to decommission the USCGC STORIS (WMEC–38) in 2007 and the USCGC ACUSHNET (WMEC–167) between 2008 and 2010, and report the vessels as excess personal property to the U.S. General Services Administration (GSA) pursuant to the Federal Property and Administrative Services Act of 1949 and its implementing regulations at Title 41, Code of Federal Regulations (CFR), part 102–36 (41 CFR part 102–36).

Preparation of the EA for the decommissioning of the USCGCs STORIS (WMEC–38) and ACUSHNET (WMEC–167) is being conducted in accordance with the National Environmental Policy Act (NEPA) of 1969 (Section 102[2][c]) and its implementing regulations at 40 CFR Part 1500.

Environmental Assessment

An EA has been prepared that identifies and examines alternatives including a no action alternative and the preferred alternative, the decommissioning and subsequent reporting of the vessels to GSA, as well as a third possible outcome, that is beyond the control of the Coast Guard and entails passage by Congress of specific legislation that directs the vessels' disposition. The EA assesses the potential environmental impacts of these alternatives and the additional possibility of specific legislation.

As the Coast Guard has determined that the vessels are historic for purposes of Section 106 of the National Historic Preservation Act of 1966, the Coast Guard has engaged in Section 106 consultation with the Alaska State Historic Preservation Office (AK SHPO) in developing a MOA on the Coast Guard's intended action of decommissioning of the USCGCs STORIS (WMEC–38) and ACUSHNET (WMEC–167) and then reporting the vessels as excess personal property to