listed below. This proposed information collection was previously published in the **Federal Register** on December 30, 2008, page 79889–79890 and allowed 60-days for public comment. One comment was received and appropriate response was made. 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 current valid OMB control number.

Proposed Collection: Title: Women's Health Initiative (WHI) Observational Study. *Type of Information Collection Request:* REVISION: OMB No. 0925–0414, Expiration date: 05/31/2009. *Need and Use of Information Collection:* This study will be used by the NIH to evaluate risk factors for chronic disease among older women by developing and following a large cohort of postmenopausal women and relating subsequent disease development to baseline assessments of historical, physical, psychosocial, and physiologic characteristics. In addition, the observational study will complement the clinical trial (which has received clinical exemption) and provide additional information on the common causes of frailty, disability and death for postmenopausal women, namely, coronary heart disease, breast and colorectal cancer, and osteoporotic fractures. Continuation of follow-up years for ascertainment of medical history update forms will provide essential data for outcomes assessment for this population of aging women. *Frequency of Response:* Annually. *Affected Public:* Individuals and physicians. *Type of Respondents:* Women, next-of-kin, and physician's office staff. The annual reporting burden is as follows:

ESTIMATE OF ANNUAL HOUR BURDEN

Type of response	Number of respondents	Frequency of response	Average hours per response	Annual hour burden
Observational Study Participants	63,230	1.1	.3383	23,509
Next of Kin[1]	1,163	1	.083	97
Health Care Providers[1]	9	1	.083	.77
Total	64,402	.....	.....	23,607

[1] Annual burden is placed on health care providers and respondent relatives/informants through requests for information which will help in the compilation of the number and nature of new fatal and nonfatal events.

The annualized cost burden to respondents is estimated at $377,725. 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 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, *OIRA_submission@omb.eop.gov* or by fax to 202–395–6974, Attention: Desk Officer for NIH. To request more information on the proposed project or to obtain a copy of the data collection plan and instruments, contact: Shari Eason Ludlam, Project Officer, Women's Health Initiative Program Office, 6701 Rockledge Drive, 2 Rockledge Centre, Suite 10018, MSC 7936, Bethesda, MD 20892–7936, or call (301) 402–2900 or E-mail your request, including your address to: *ludlams@mail.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: March 2, 2009.

Michael S. Lauer,

Director, Division of Prevention and Population Sciences, NHLBI, National Institutes of Health.

Dated: March 3, 2009.

Suzanne Freeman,

Chief, FOIA, NHLBI, National Institutes of Health.

[FR Doc. E9–5521 Filed 3–12–09; 8:45 am]

BILLING CODE 4140–01–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.

Gene Signature for Predicting Hepatocellular Carcinoma Patient Prognosis

Description of Technology: A progressive sequence of somatic mutations and epigenetic changes of oncogenes or tumor suppressor genes are believed to cause tumor development. However, high genomic instability in tumors causes the accumulation of genomic aberrations that do not contribute to tumor progression. Therefore it is important to distinguish between ''driver'' mutations which are functionally important and ''passenger'' mutations which do not provide a selective advantage to the tumor cells.

The current invention describes a driver gene signature for predicting survival in patients with hepatocellular carcinoma (HCC). The gene signature includes ten HCC-associated genes, and the NIH researchers further discovered that a decrease in DNA copy number or mRNA expression of some genes is associated with a poor prognosis in HCC tumors, while a decrease in DNA copy number or mRNA expression of a few other genes is associated with a good prognosis.

Available for licensing is a method of predicting the prognosis of a patient diagnosed with HCC by detecting expression of one of more HCC-associated genes, and a method of identifying an agent for use in treating HCC.

Applications: Prognosis for hepatocellular carcinoma (HCC) patient survival; Potential new method to identify therapeutic treatment for HCC patients.

Market: Hepatocellular carcinoma (HCC) is the most frequent malignant tumor in the liver and the third leading cause of cancer death worldwide. Systemic chemotherapy has been shown to be ineffective and tumor recurrence rate after surgical resection is high due to relapse and metastasis. Therefore, the development of new drugs will be crucial to prevent relapse and to prolong patient survival.

Development Status: Early-stage development.

Inventors: Xin Wei Wang and Stephanie Roessler (NCI).

Patent Status: U.S. Provisional Application No. 61/198,813 filed 10 Nov 2008 (HHS Reference No. E–024–2009/0–US–01).

Licensing Status: Available for licensing.

Licensing Contact: Betty Tong, Ph.D.; 301–594–6565; *tongb@mail.nih.gov*.

Collaborative Research Opportunity: The National Cancer Institute, Center for Cancer Research, Laboratory of Human Carcinogenesis, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Gene Signature for Predicting Hepatocellular Carcinoma Patient Prognosis. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Lasonolide Compounds as Reagents for Inducing Premature Chromosome Condensation and Methods of Treating Cancer

Description of Technology: Lasonolide A is a natural product initially isolated from an extract of the shallow water Caribbean marine sponge. The chemical structure of lasonolide A was identified in 2002, and it was chemically synthesized in 2007. The current invention discloses the discovery that lasonolide A may be used as a new reagent for inducing premature chromosome condensation in non-dividing cells; and a novel anti-proliferative and anti-metastatic agent for cancer treatment. Currently, it is difficult to analyze the cytogenetic composition of the genome of non-dividing cells because the chromosomes are loosely distributed in the nucleus, lasonolide A may be useful for performing cytogenetic studies in cells by inducing premature chromosome condensation without inducing mitosis. In addition, the invention also reveals that lasonolide A inhibits cancer cell motility. As such, lasonolide A may be used as an anti-cancer agent by itself or in combination with other anti-cancer agents such as inhibitors of topoisomerases.

Applications: A new reagent for inducing premature chromosome condensation in non-dividing cells; a novel anti-cancer agent.

Market: Cancer continues to be a burden to the public health of Americans. After heart disease, cancer is the most common cause of death in the United States. For 2008, it was estimated that about 565,650 Americans were expected to die of cancer. The incidence of cancer has been dropping over the years but it is estimated that over 1.4 million Americans would be diagnosed with cancer in 2008. Therefore, there is a continued need for the development of new therapies to effectively treat this disease.

Development Status: Early-stage development.

Inventors: Yves G. Pommier (NCI) *et al.*

Patent Status: U.S. Provisional Application No. 61/137,193 filed 28 Jul 2008 (HHS Reference No. E–247–2008/0–US–01).

Licensing Status: Available for licensing.

Licensing Contact: Betty Tong, Ph.D.; 301–594–6565; *tongb@mail.nih.gov*.

Collaborative Research Opportunity: The National Cancer Institute, Center for Cancer Research, Laboratory of Molecular Pharmacology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Lasonolide Compounds as Reagents for Inducing Premature Chromosome Condensation and Methods of Treating Cancer. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Increased Image Quality for Early Colon Polyp Detection

Description of Technology: The invention relates to a method for improving the specificity and sensitivity of computer tomographic colonoscopy (CTC) computer aided detection (CAD). Currently CTC CAD programs are capable of delivering high sensitivity and low false positive results when used to detect large polyps of 1 cm or greater in diameter. However, CTC CAD is not as effective at detecting medium-sized polyps (6–9 mm in diameter) as it demonstrates lower sensitivities and higher false positives in this range. Since early polyp detection is critical to the survival of patients with colon cancer, the ability to accurately detect medium size polyps could be advantageous to the outcome of colon cancer treatment.

The invention uses a wavelet-based analysis to distinguish true polyps from false positives in CTC images. The steps involved include generating a 2D projection image, computing features of the 2D images from their Haar wavelet coefficients, applying the feature selection algorithm, and training a classifier using the selected features to classify CTC CAD.

Using this technology, it will be possible to create high quality images for viewing the colon surface in 3D with reduced false positives in the medium-sized range for colon polyps. The technology can also be used to locate anomalies in both medical and non-medically related image applications such as endoscopy, microscopy, and photography.

Applications: High quality images for early colon polyp detection; Sensitive and efficient colon cancer diagnosis; Locating anomalies in several different image applications.

Development Status: Early stage.

Inventors: Ronald M. Summers *et al.* (CC)

Publications:

1. J Li, R Van Uitert, J Yao, N Petrick, M Franaszek, A Huang, RM Summers. Wavelet method for CT colonography computer-aided polyp detection. Med Phys. 2008 Aug;35(8):3527–3538.

2. S Greenblum, J Li, A Huang, RM Summers. Wavelet analysis in virtual colonoscopy. Proc. SPIE, Vol. 6143, 614336 (March 13, 2006); doi:10.1117/12.655680.

Patent Status: U.S. Patent Application No. 11/685,127 filed 12 Mar 2007 (HHS Reference No. E–314–2006/0–US–02); No foreign rights available.

Licensing Status: Available for licensing.

Licensing Contact: Jeffrey A. James, Ph.D.; 301–435–5474; *jeffreyja@mail.nih.gov*.

Microdissection and High-Throughput Analysis of Biological Samples

Description of Technology: A variety of techniques have been used to microdissect specific cells or cell populations from a histological sample under direct microscopic visualization. Original microdissection techniques involved painstaking (and sometimes clumsy) manual dissection using needles or other micro-manipulation devices to isolate individual cells based on visible, histological characteristics.

The subject technology is a method of performing specific target activated transfer from a biological sample (*i.e.*, tissue) for analysis using a device system that can be automated for high throughput analysis. The method employs a localized reagent, such as an absorbative stain, that specifically determines the microadhesion of desired cellular material in a tissue sample to a transfer surface such as a thermoplastic polymer film. The energy from a light or heat source causes the microadhesion of the target cells or cell populations to the thermoplastic transfer surface. Subsequent separation of the film from the tissue section selectively removes the adhered target from the tissue section. The transfer surface is activated from within the target to adhere the target to the transfer surface, for example by heating the target to adhere or to a thermoplastic transfer surface. Such *in situ* activation can be achieved by exposing the biological sample to an immunoreagent that specifically binds to the target (or a component of the target). The immunoreagent can alter the transfer surface directly (for example with a heat generating enzyme carried by the immunoreagent), or indirectly (for example by changing a characteristic of the target). In some embodiments, the immunoreagent deposits a precipitate in the target that increases its light absorption relative to surrounding tissue, such that the biological specimen can be exposed to light to selectively heat the target. Alternatively, the immunoreagent is an immunofluorescent agent that carries a fluorophore that absorbs light and emits heat.

Applications: Microdissection of specific cells or cell populations from a histological sample; High throughput analysis of biological samples.

Advantages: Automated system for high throughput microdissection and analysis; Does not require a visual detection step.

Development Status: In vitro data can be provided upon request.

Inventors: Michael R. Emmert-Buck (NCI), Robert F. Bonner (NICHD), Michael A. Tangrea (NCI), Thomas J. Pohida (CIT), Rodrigo F. Chuaqui (NCI).

Patent Status:

International Patent Application No. PCT/US03/23317 filed 23 July 2003, which published as WO 2004/068104 on 12 Aug 2004 (HHS Reference No. E–113–2003/0–PCT–02),

U.S. Patent Application No. 10/543,218 filed 22 Jul 2005 (HHS Reference No. E–113–2003/0–US–03),

Canadian Patent Application No. 2513646 filed 23 Jul 2003 (HHS Reference No. E–113–2003/0–CA–05),

Australian Patent Application No. 2003256803 filed 23 Jul 2003 (HHS Reference No. E–113–2003/0–AU–04),

U.S. Patent Application No. 11/202,848 filed 12 Aug 2005 (HHS Reference No. E–113–2003/1–US–01).

Licensing Status: Available for licensing,

Licensing Contact: Kevin W. Chang, Ph.D.; 301–435–5018, *changke@mail.nih.gov*,

Collaborative Research Opportunity: The National Cancer Institute, Center for Cancer Research, Laboratory of Pathology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Target Activated Microtransfer—Expression Microdissection (xMD). Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Use of Anthrax Lethal Factor To Treat Cancer and Screening Methods for MAPK Kinase Protease Activity

Description of Technology: Anthrax toxin, produced by *Bacillus anthracis,* is composed of three proteins: Protective antigen (PA), edema factor (EF), and lethal factor (LF). PA by itself has little or no toxic effect upon cells, but serves to bind cell surface receptors and mediate the entry of EF and LF into the cell. EF has been identified as an adenylate cyclase and together with PA forms a toxin (edema toxin; EdTx) which can induce edema formation when injected subcutaneously. LF and PA together form a toxin (lethal toxin; LeTx) which can cause rapid lysis of certain macrophage-derived cell lines *in vitro* as well as death when injected intravenously.

Indirect evidence had suggested that LF was a metalloprotease. However, the intracellular target of LF remained unknown until recently when NIH scientists discovered that LF proteolytically inactivates mitogen activated protein kinase kinase 1 and 2 (MAPKK1, 2). Using oocytes of the frog *Xenopus laevis* as well as tumor derived NIH3T3 (490) cells expressing an effector domain mutant form of the human V12HaRas oncogene these scientists demonstrated that LF induced proteolysis of MAPKK 1 and 2, resulting in their irreversible inactivation. MAPKK 1 and 2 are components of the mitogen activated protein kinase (MAPK) signal transduction pathway, an evolutionarily conserved pathway that controls cell proliferation and differentiation in response to extracellular signals and also plays a crucial role in regulating oocyte meiotic maturation. Further, the MAPK pathway has been shown to be constitutively activated in many primary human as well as in tumor-derived cell lines. Consistent with this, treatment of V12Ha-Ras transformed NIH 3T3 cells with LeTx inhibits cell proliferation and causes their reversion to a non-transformed phenotype.

This invention specifically relates to *in vitro* and *ex vivo* methods of screening for modulators, homologues, and mimetics of LF mitogen activated protein kinase kinase (MAPKK) protease activity. Applications for this technology could be:

- A novel tool (LF) for the study of the cellular role of the MAPK pathway in normal or tumor cells.
- Investigation of LF for developing inhibitors for cancer therapy. By analyzing structural-functional relationships, additional compounds with improved specificity, increased potency, and reduced toxicity can be generated. Mimetics which block MAPKK activity or the determination of mechanisms of regulation of proteases that target MAPKK at or near the same site targeted by LF could be developed.
- A protease-based assay for LF by using a peptide to test for LF cleavage.

There is no commercial test for anthrax. This assay could be used for testing soldiers for anthrax exposure. Characterization of the interaction between LF and MAPKK at the amino acid level may lead to the generation of inhibitors which may prove useful in treating anthrax.

Inventors: Nicholas S. Duesbery (NCI), Craig Webb (NCI), Stephen H. Leppla (NIDCR), George F. Vande Woude (NCI).

Patent Status:

U.S. Patent 6,485,925 issued 26 Nov 2002 (HHS Reference No. E–068–1998/0–US–06).

U.S. Patent 6,893,835 issued 17 May 2005 (HHS Reference No. E–068–1998/0–US–07).

U.S. Patent 6,911,203 issued 28 Jun 2005 (HHS Reference No. E–068–1998/0–US–08).

U.S. Patent 7,056,693 issued 06 Jun 2006 (HHS Reference No. E–068–1998/0–US–10).

U.S. Patent 7,183,071 issued 27 Feb 2007 (HHS Reference No. E–068–1998/0–US–11).

International rights available.

Licensing Status: Available for licensing.

Licensing Contact: Surekha Vathyam, Ph.D.; 301–435–4076; *vathyams@mail.nih.gov*.

Dated: March 5, 2009.

Richard U. Rodriguez,

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

[FR Doc. E9–5418 Filed 3–12–09; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the following meetings.

The meetings will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: Center for Scientific Review Special Emphasis Panel, Small Business: Orthopaedics and Skeletal Biology.

Date: March 20, 2009.

Time: 8 a.m. to 5 p.m.

Agenda: To review and evaluate grant applications.

Place: Beacon Hotel and Corporate Quarters, 1615 Rhode Island Avenue, NW., Washington, DC 20036.

Contact Person: Daniel F. McDonald, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 4110, MSC 7814, Bethesda, MD 20892, (301) 435–1215, *mcdonald@csr.nih.gov.*

This notice is being published less than 15 days prior to the meeting due to the timing limitations imposed by the review and funding cycle.

Name of Committee: Center for Scientific Review Special Emphasis Panel, Ligament/Tendon Repair and Replacement.

Date: March 26, 2009.

Time: 10 a.m. to 12:30 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892, (Telephone Conference Call).

Contact Person: John P. Holden, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 4211, MSC 7814, Bethesda, MD 20892, 301–496–8551, *holdenjo@csr.nih.gov.*

This notice is being published less than 15 days prior to the meeting due to the timing limitations imposed by the review and funding cycle.

Name of Committee: Center for Scientific Review Special Emphasis Panel, Review of HIV/AIDS Related SBIR/STTR Applications.

Date: April 1, 2009.

Time: 10 a.m. to 2 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892 (Virtual Meeting).

Contact Person: Mark P. Rubert, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 5218, MSC 7852, Bethesda, MD 20892, 301–435–1775, *rubertm@csr.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel, BMIT/MEDI Member Conflict—Imaging.

Date: April 2, 2009.

Time: 11 a.m. to 3 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892 (Virtual Meeting).

Contact Person: Dharam S. Dhindsa, DVM, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 5110, MSC 7854, Bethesda, MD 20892, (301) 435–1174, *dhindsad@csr.nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.306, Comparative Medicine; 93.333, Clinical Research, 93.306, 93.333, 93.337, 93.393–93.396, 93.837–93.844, 93.846–93.878, 93.892, 93.893, National Institutes of Health, HHS)

Dated: March 4, 2009.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. E9–5139 Filed 3–12–09; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Amended Notice of Meeting

Notice is hereby given of a change in the meeting of the Center for Scientific Review Special Emphasis Panel, February 26, 2009, 8 a.m. to February 28, 2009, 5 p.m., National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD, 20892 which was published in the **Federal Register** on February 6, 2009, 74 FR 6292–6294.

The meeting will be held March 25, 2009 to March 27, 2009. The meeting time and location remain the same. The meeting is closed to the public.

Dated: March 5, 2009.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. E9–5344 Filed 3–12–09; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Eunice Kennedy Shriver National Institute of Child Health & Human Development; Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the following meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.