Type of respondents	Number of respondents	Frequency of response	Average burden hours per response	Annual hour burden
PLCO participants	150	1	5 minutes (0.083)	12.5
Physicians office staff	50	1	20 minutes (0.333)	16.7
Totals	200			29.2

The annualized cost to respondents is estimated at: $487.50. 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.

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 Attention: NIH Desk Officer, Office of Management and Budget, at *OIRA_submission@omb.eop.gov* or by fax to 202–395–6974. To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact: Dr. Maria Pisu, Division of Preventive Medicine, University of Alabama at Birmingham, MT 628, 1530 3rd Avenue South, Birmingham, AL 35294–4410, or call non-toll-free number (205) 975–7366 or e-mail your request, including your address to: *mpisu@uab.edu*

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: February 20, 2008.

Vivian Horovitch-Kelley,

NCI Project Clearance Liaison Office, National Institutes of Health.

[FR Doc. E8–3836 Filed 2–28–08; 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.

A Pharmacophore for Isatin-β-Thiosemicarbazone Compounds With MDR1-Inverse Activity

Description of Technology: One of the major hindrances to successful cancer chemotherapy is multi-drug resistance (MDR), which is frequently caused by the increased expression or activity of ABC transporter proteins. Research has generally been directed to overcoming MDR during cancer therapy by inhibiting the activity of ABC transporters. However, compounds that inhibit ABC transporter activity often elicit strong and undesirable side-effects, restricting their usefulness in therapy.

In an alternative approach to reducing the debilitating effects of MDR in cancer therapy, scientists at the National Cancer Institute identified a family of compounds whose antiproliferative effects were actually enhanced in cells with MDR. These compounds included NSC 73306, a specific compound that increased the chemosensitivity of cells that overexpress ABC transporters without inhibiting ABC transporter activity. This invention concerns new analogs of NSC 73306 with improved selectivity and solubility, and the use of the analogs as therapeutics.

Applications:

Treatment of cancers associated with multi-drug resistance, either alone or in combination with other therapeutics.

Development of a pharmacophore for improved effectiveness in treating cancers associated with multi-drug resistance.

Advantages:

The agents capitalize on one of the most common drawbacks to cancer therapies (MDR) by using it as an advantage to treating cancer.

Increased specificity allows these analogs to be tailored to treating cancers associated with the overexpression and hyperactivity of particular ABC transporters.

Increased solubility allows greater access of the agent to tumor cells, increasing therapeutic effectiveness of the agents.

Benefits: Cancer is the second leading cause of death in United States and it is estimated that there will be approximately 600,000 deaths caused by cancer in 2007. Improving the quality of life and duration of life of cancer patients will depend on chemotherapies with increased effectiveness and reduced toxicity, thus this technology can contribute significantly to a social cause. Furthermore, small molecule cancer therapy technologies have a potential market of more than $2 billion.

Inventors: Matthew D. Hall et al. (NCI).

U.S. Patent Status: Provisional U.S. Application (HHS Reference No. E–017–2008/0–US–01).

Licensing Contact: David A. Lambertson, PhD; 301–435–4632; *lambertsond@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute's Laboratory of Cell Biology is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize for the clinic, compounds that demonstrate MDR1-inverse activity. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

An Improved Non Viral System for Tumor Specific Suicide Gene Therapy

Description of Technology: Numerous tumor specific promoters have been identified and developed for targeted gene therapy. Survivin promoter activity is upregulated in 75% of tumors, however the activity is specific but low, resulting in sub-optimal suicide gene expression. Combination of survivin promoter with Bax, a proapoptotic gene, previously used in such therapy has demonstrated low efficacy.

Scientists at NCI have made a plasmid construct consisting of survivin promoter driven mutant form of bax that is constitutively active. This construct is more potent than the wild type bax, improving its efficacy several-fold, while, retaining specificity for tumors, as determined by in vitro and in vivo studies.

This new technology does not use CMV or SV–40 promoters, alleviating the need for modifications for commercialization.

Advantages:

Can be used with cationic liposomes or other DNA delivery systems.

Can be incorporated into adenoviral and lentiviral vectors.

Excludes viral promoters.

Can be modified easily to use other promoters/suicide genes.

Applications:

Cancer therapeutics

Targeted Gene therapy

Market: In patients with advanced solid tumors or recurrences despite surgery, chemotherapy can provide quality survival. However, responses are usually partial, often disappointingly brief and unpredictable and coupled with side effects. These limitations of traditional cytotoxic chemotherapy make it necessary to explore other therapies such as targeted gene therapy. Viruses, while the carrier of choice in most gene therapy studies, present a variety of potential problems to the patient—toxicity, immune and inflammatory responses, and gene control and targeting issues. In addition, there is a fear that the viral vector may recover its ability to cause disease in the patient. Our new technology addresses some of the above issues making it a suitable agent for cancer and gene therapy.

Development Status: Early.

Inventors: Himanshu Garg and Robert P. Blumenthal (NCI).

Patent Status: HHS Reference No. E–245–2007/0—Research Tool. Patent protection is not being sought for this technology.

Licensing Status: Available for non-exclusive licensing.

Licensing Contact: John Stansberry, PhD; 301–435–5236; *stansbej@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute Center for Cancer Research Nanobiology Program is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize tumor specific suicide gene therapy using survivin promoter driven mutant bax. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Eeyarestatins: Novel Deubiquitination Inhibitors for the Treatment of Drug-Resistant Cancers

Description of Technology: The ubiquitin-proteasome system has recently been recognized to play a central role in tumor biology. Bortezomib, an inhibitor of the chymotrypsin-like activity of the proteasome, has clinical activity in a variety of hematologic malignancies and is FDA approved for use in Multiple Myeloma and Mantle Cell Lymphoma.

The present invention for the first time describes that Eeyarestatins, a new class of small molecules, are potential anti-cancer agents. The compounds inhibit the deubiquitination of proteins by targeting the deubiquitination enzymes in the protein degradation pathway. More specifically, the inventors have demonstrated that the Eeyarestatins successfully kill different leukemia and lymphoma cell lines as well as leukemia cells isolated from patients with chronic lymphocytic leukemia by inducing the expression of Noxa, a pro-apoptotic member of the Bcl-2 protein family. Additionally, Eeyarestatins are active against cells that are resistant to Bortezomib and thus can be effective against drug-resistant tumors.

Applications:

Eeyarestatins can be developed for the treatment of deubiquitination related disorders such as cancers and proliferative disorders.

Eeyarestatins can potentially have broader use against HIV and immune related disorders considering the role of deubiquitination in budding of retroviruses and immune regulation.

Advantages:

Eeyarestatins are active against cells that are resistant to Bortezomib.

In vitro data shows activity of Eeyarestatins against primary cells from patients with chronic lymphocytic leukemia. Clinical trials show that Bortezomib is inactive against patients suffering from chronic lymphocytic leukemia.

Market: The current cancer chemotherapeutic market is valued at $42 billion and expected to grow. Additionally, this compound has potential use in HIV and immune related disorders.

Development Status: In vitro studies are completed and *in vivo* animal model studies are planned.

Inventors: Adrian Wiestner (NHLBI), Yihong Ye (NIDDK), Qiuyan Wang (NIDDK), Helena Mora-Jensen (NHLBI)

Publication: Q Wang, L Li, Y Ye. Inhibition of p97-dependent protein degradation by Eeyarestatin I. J Biol Chem. 2008 Jan 16; Epub ahead of print, doi 10.1074/jbc.M708347200.

Patent Status: U.S. Provisional Application No. 60/961,202 filed 17 Jul 2007 (HHS Reference No. E–208–2007/0–US–01)

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

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

Collaborative Research Opportunity: The National Institutes of Health laboratories of Dr. Adrian Wiestner (NHLBI) and Dr. Yihong Ye (NIDDK) are seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Eeyarestatins. Please contact Dr. Wiestner (301–594–6855, *wiestnera@mail.nih.gov*) or Dr. Ye (301–594–0845, *yihongy@mail.nih.gov*) for more information.

Synergistic Effect of TGF-Beta Blockade and Immunogenic Agents on Tumors

Description of Technology: Overcoming immune suppression in cancer patients is a major challenge for the success of cancer immunotherapy. TGF-β and its receptors are expressed in essentially all tissues, and they have been found to be important in many cellular processes including cell growth inhibition. The inhibition of TGF-β

signaling has been shown to have an inhibitory effect on tumor growth. However, TGF-β also has immunosuppressive properties.

Cancer vaccines are one of many therapies available for treatment and prevention. In particular, vaccines that elicit immune responses have been used to treat or control tumor growth that has evaded immunosurveillance. However, these vaccines have demonstrated limited success.

Available for licensing is a method for synergistically affecting tumor growth involving the administration of an agent that blocks the TGF-β signaling pathway, in combination with an immunogenic agent. The agent that blocks the TGF-β signaling pathway may inhibit the immunosuppressive effects of TGF-β, while the immunogenic agent is believed to enhance an immune response. Surprisingly, the combination of such elements produces a synergistic effect. The administration of the 1D11.16 anti-TGF-β antibody in combination with the human papilloma virus E7(49–57) peptide enhances tumor regression in an animal model. The administration of the 1D11.16 anti-TGF-β antibody in combination with irradiated CT26 cells enhances tumor regression in another mouse model. The investigators found that administering the combination of agents is more effective than the sum of their individual effects.

Applications: A method of cancer combination therapy based on immunotherapeutics.

Development Status: The invention is in the clinical stages of development.

Inventors: Masaki Terabe (NCI) et al.

Publications:

1. PCT patent publication WO 2006/089251, August 24, 2006.

2. M Terabe et al. Transforming growth factor-beta production and myeloid cells are an effector mechanism through which CD1d-restricted T cells block cytotoxic T lymphocyte-mediated tumor immunosurveillance: abrogation prevents tumor recurrence. J Exp Med. 2003 Dec 1;198(11):1741–1752.

Patent Status: U.S. Provisional Application No. 60/654,329 filed 17 Feb 2005 (HHS Reference No. E–019–2005/0–US–01); PCT Application No. PCT/US2006/005888 filed 16 Feb 2006 (HHS Reference No. E–019–2005/0–PCT–02); U.S. Patent Application No. 11/816,410 filed 15 Aug 2007 (HHS Reference No. E–019–2005/0–US–06)

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

Licensing Contact: Jennifer Wong; 301–435–4633; *wongje@mail.nih.gov*.

Biologically Active Macrolides, Compositions and Uses Thereof

Description of Technology: The current invention embodies the identification of a novel class of potent vacuolar-type (H+)-ATPase-inhibitory compounds. Vacuolar-type (H+)-ATPases are present in many tissues and cells of the body and are involved in the maintenance of various physiological functions. The modification of these functions, via inhibition of vacuolar-type (H+)-ATPases, may represent an effective means of treating various disease states, including Alzheimer's disease, glaucoma, and osteoporosis. In addition, these inhibitors may also be of particular value for use against cancer, as vacuolar-type (H+)-ATPases have been implicated in processes relating to cellular proliferation, angiogenesis, tumor cell invasiveness, metastasis, and drug resistance.

Inventors: Michael R. Boyd (NCI), Kirk R. Gustafson (NCI), et al.

Patent Status: U.S. Patent No. 7,144,918 issued 05 Dec 2006 (HHS Reference No. E–203–2000/0–US–04); U.S. Patent Application No. 11/435,189 filed 16 May 2006 (HHS Reference No. E–203–2000/08–US–08)

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

Licensing Contact: Adaku Nwachukwu, J.D.; 301–435–5560; *madua@mail.nih.gov*.

Human p53 Mutations and a Genetic System in Yeast for Functional Identification of Human p53 Mutations

Description of Technology: The tumor suppressor gene p53, a key regulator of cellular mechanisms that maintain genome integrity, is the most commonly inactivated gene target associated with neoplastic transformation. p53 is mutated in about 50% of all human tumors and more than 80% of these mutations are missense, leading to single amino acid changes. This invention relates to human p53 mutants and identification methods using screening assays in the yeast *Saccharomyces cerevisiae* to functionally categorize expressed p53 mutant proteins at varying levels of expression towards several human target response sequences. Additionally, the invention relates to methods of detecting or generating novel human p53 mutations with properties that can include toxicity in yeast and growth suppression in human cells, enhanced or reduced transactivation relative to wildtype p53, altered promoter selectivity, and reactivation by mutation or chemical modification of common tumor mutations for the transactivation function of major p53 downstream genes. In particular, the inventors have discovered a V122A p53 mutation exhibits strong cell proliferation inhibition. This feature suggests that p53 alleles such as V122A might be valuable both for functional studies of p53-regulated cellular responses and possibly for p53 based cancer gene therapy.

Applications:

Cancer therapeutics.

Model to screen for small molecules or peptides that can modify p53 functions.

Pharmaceutical screen for p53 drug modifiers.

Market:

An estimated 1,444,920 new cancer diagnoses in the U.S. in 2007.

Cancer drug market is estimated to be $50 billion a year in 2010.

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

Inventors: Michael A. Resnick and Alberto Inga (NIEHS)

Publications:

1. A Jegga, A Inga, D Menendez, BJ Aronow, MA Resnick. Functional evolution of the p53 regulatory network through its target response elements. Proc Natl Acad Sci. USA. 2008 Jan 22;105(3):944–949.

2. MM Horvath, X Wang, MA Resnick, DA Bell. Divergent evolution of human p53 binding sites: cell cycle versus apoptosis. PLoS Genet. 2007 Jul;3(7):1284–1295.

3. D Menendez, A Inga, J Snipe,O Krysiak, G Schönfelder, MA Resnick. A single-nucleotide polymorphism in a half-binding site creates p53 and estrogen receptor control of vascular endothelial growth factor receptor 1. Mol Cell Biol. 2007 Apr;27(7):2590–2600.

4. P Monti, Y Ciribilli, J Jordan, P Menichini, DM Umbach, MA Resnick, L Luzzato, A Inga, G Fronza. Transcriptional functionality of germ line p53 mutants influences cancer phenotype. Clin Can Res. 2006 Jul 1;13(13):3789–3795.

5. D Menendez, A Inga, J Jordan, MA Resnick. Changing the p53 master regulatory network: *ELEMENTary*, my dear Mr. Watson. Oncogene. 2007 Apr 2;26(15):2191–2201.

6. D Menendez, A Inga, J Jordan, MA Resnick. The biological impact of the human master regulator p53 can be altered by mutations that change the spectrum and expression of its target genes. Mol Cell Biol. 2006 Mar;26(6):2297–2308.

7. DJ Tomso, A Inga, D Menendez, G Pittman, M Campbell, D Bell, MA

Resnick. Functionally distinct polymorphic sequences in the human genome that are targets for p53 transactivation. Proc Natl Acad Sci USA. 2005 May 3;102(18):6431–6436.

8. MA Resnick and A Inga. Functional mutations in the sequence-specific transcription factor p53 and implications for master genes of diversity. Proc Nat Acad Sci USA. 2003 Aug 19;100(17):9934–9939.

9. A Inga, F Storici, TA Darden, MA Resnick. Differential transactivation by the p53 transcription factor is highly dependent on p53 level and promoter target sequence. Mol Cell Biol. 2002 Dec;22(24):8612–8625, 2002.

Patent Status:

U.S. Patent No. 7,256,260 issued 14 Aug 2007 (HHS Reference No. E–183–1999/0–US–07)

U.S. Patent Application No. 11/893,037 filed 14 Aug 2007 (HHS Reference No. E–183–1999/0–US–09)

European Patent Application No. 0094897.0 filed 28 July 2007, recently allowed (HHS Reference No. E–183–1999/0–EP–05)

Australian Patent No. 784293 issued 14 Aug 2007 (HHS Reference No. E–183–1999/0–AU–03)

Australian Patent Application No. 2006202361 filed 2 Jun 2006 (HHS Reference No. E–183–1999/0–AU–08)

Canadian Patent Application No. 2380631 filed 28 July 2000 (HHS Reference No. E–183–1999/0–CA–04)

Japanese Patent Application No. 2001–514117 filed 28 July 2000 (HHS Reference No. E–183–1999/0–JP–03)

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

Licensing Contact: Jennifer Wong; 301–435–4633; *wongje@mail.nih.gov*.

Dated: February 21, 2008.

Steven M. Ferguson,

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

[FR Doc. E8–3837 Filed 2–28–08; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Office of the Director, National Institutes of Health; Notice of Meeting

Pursuant to section 10(a) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the Advisory Committee on Research on Women's Health.

The meeting will be open to the public, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the meeting.

Name of Committee: Advisory Committee on Research on Women's Health.

Date: March 17, 2008.

Time: 8:30 a.m. to 4 p.m.

Agenda: Provide advice to the Office of Research on Women's Health (ORWH) on appropriate research activities with respect to women's health and related studies to be undertaken by the national research institutes; to provide recommendations regarding ORWH activities; to meet the mandates of the office; and for discussion of scientific issues.

Place: National Institutes of Health, Building 31, 31 Center Drive, Conference Room 6, Bethesda, MD 20892.

Contact Person: Joyce Rudick, Director, Programs & Management, Office of Research on Women's Health, Office of the Director, National Institutes of Health, Building 1, Room 201, Bethesda, MD 20892, 301/402–1770.

Any interested person may file written comments with the committee by forwarding the statement to the Contact Person listed on this notice. The statement should include the name, address, telephone number and when applicable, the business or professional affiliation of the interested person.

Information is also available on the Institute's/Center's home page: *http://www.od.nih.gov/orwh/*, where an agenda and any additional information for the meeting will be posted when available.

(Catalogue of Federal Domestic Assistance Program Nos. 93.14, Intramural Research Training Award; 93.22, Clinical Research Loan Repayment Program for Individuals from Disadvantaged Backgrounds; 93.232, Loan Repayment Program for Research Generally; 93.39, Academic Research Enhancement Award; 93.936, NIH Acquired Immunodeficiency Syndrome Research Loan Repayment Program; 93.187, Undergraduate Scholarship Program for Individuals from Disadvantaged Backgrounds, National Institutes of Health, HHS)

Dated: February 21, 2008.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 08–888 Filed 2–28–08; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Substance Abuse and Mental Health Services Administration

Agency Information Collection Activities: Proposed Collection; Comment Request

In compliance with section 3506(c)(2)(A) of the Paperwork Reduction Act of 1995 concerning opportunity for public comment on proposed collections of information, the Substance Abuse and Mental Health Services Administration will publish periodic summaries of proposed projects. To request more information on the proposed projects or to obtain a copy of the information collection plans, call the SAMHSA Reports Clearance Officer on (240) 276–1243.

Comments are invited on: (a) Whether the proposed collections of information are necessary for the proper performance of the functions of the agency, including whether the information shall have practical utility; (b) the accuracy of the agency's estimate of the burden of the proposed collection of information; (c) ways to enhance the quality, utility, and clarity of the information to be collected; and (d) ways to minimize the burden of the collection of information on respondents, including through the use of automated collection techniques or other forms of information technology.

Proposed Project: Data Toolkit Protocol for the Crisis Counseling Assistance and Training Program (CCP) (OMB No. 0930–0270)—Revision

The Substance Abuse and Mental Health Services Administration's (SAMHSA) Center for Mental Health Services (CMHS) will create a toolkit to be used for the purposes of collecting data on the Crisis Counseling Assistance and Training Program (CCP). The CCP provides supplemental funding to states and territories for individual and community crisis intervention services during a federal declared disaster in accordance with section 416, Robert T. Stafford Disaster Relief and Emergency Assistance Act (Pub. L. 93–288, as amended).

The CCP has provided disaster mental health services to millions of disaster survivors since its inception and, as a result of 30 years of accumulated expertise, it has become an important model for Federal response to a variety of catastrophic events. State CCPs, such as Project HOPE (after Hurricane Floyd in North Carolina), Project Heartland (in Oklahoma City after the Murrah Federal Building bombing), Project Liberty (in New York after 9/11), and Project Outreach for Recovery (after the Rhode Island nightclub fire), gulf coast States affected by the 2005 hurricanes, and recent 2007 southern California wildfires have primarily addressed the short-term mental and behavioral health needs of communities through (a) outreach and public education, (b) individual and group counseling, and (c) referral. Disaster victims are