

Data collection instrument(s)	Estimated number of respondents	Responses per respondent	Average burden hour per response *	Total annual burden hours
IHS 843-1A .....	7,977	52	3/60	20,740
Total .....				20,740

\*For ease of understanding, burden hours are also provided in actual minutes.

The total estimated burden for this collection is 20,740 hours.

There are no Capital Costs, Operating Costs, and/or Maintenance Costs to report.

*Request for Comments:* Your written comments and/or suggestions are invited on one or more of the following points: (a) Whether the information collection activity is necessary to carry out an agency function; (b) whether the IHS processes the information collected in a useful and timely fashion; (c) the accuracy of the public burden estimate (this is the amount of time needed for individual respondents to provide the requested information); (d) whether the methodology and assumptions used to determine the estimate are logical; (e) ways to enhance the quality, utility, and clarity of the information being collected; and (f) ways to minimize the public burden through the use of automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

*Send Comments and Requests for Further Information:* Send your written comments and requests for more information on the proposed collection or requests to obtain a copy of the data collection instrument(s) and instructions to: Tamara Clay, IHS Reports Clearance Officer, 801 Thompson Avenue, TMP, Suite 450, Rockville, MD 20852; call non-toll free (301) 443-1611; send via facsimile to (301) 443-2316, or send your email requests, comments, and return address to [tamara.clay@ihs.gov](mailto:tamara.clay@ihs.gov).

*Comment Due Date:* Your 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: November 9, 2012.

**Yvette Roubideaux,**

Director, Indian Health Service.

[FR Doc. 2012-28236 Filed 11-20-12; 8:45 am]

**BILLING CODE 4165-16-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.

**FOR FURTHER INFORMATION CONTACT:** 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 New Class, Now in Session: the First HLA Class II Restricted T Cell Receptor That Recognizes the Cancer Testis Antigen, MAGE-A3, Developed for Cancer Immunotherapy**

*Description of Technology:* NIH scientists have developed T cell receptors (TCRs) against the melanoma antigen family A3 (MAGE-A3) tumor antigen in the context of major histocompatibility complex (MHC) class II molecule HLA-DP-beta1\*04. They are the first HLA class II restricted MAGE-A3 TCRs developed for use in adoptive immunotherapy. Previously developed MAGE-A3 TCRs are HLA class I restricted and generate CD8+ T cell responses to mediate tumor regression in some patients with MAGE-A3+ tumors. Other patients may not respond due to a lack of CD4+ T cells participation. Cancer immunotherapy

with these new HLA class II TCRs could yield a robust and effective CD4+ T cell immune response that selectively targets MAGE-A3 expressing tumors without generating toxicity against healthy cells.

MAGE-A3 is a cancer testis antigen expressed on many types of cancer cells that blocks the functions of tumor suppressor proteins to mediate tumor growth and spreading. MAGE-A3 is not expressed on normal cells other than non-MHC expressing germ cells of the testis, which do not generate an immune response. Thus, MAGE-A3 represents an ideal target for cancer immunotherapies that are predicted to generate fewer toxic side effects than current standard cancer treatments.

*Potential Commercial Applications:*

- A personalized immunotherapy to mediate regression of many types of cancers using human T cells expressing a HLA class II TCR.
- An adoptive immunotherapy combining T cells engineered to express a HLA class I restricted TCR with HLA class II TCR-expressing T cells to enhance the antitumor response by eliciting CD8+ and CD4+ T cell immune responses in patients.
- A research tool to investigate signaling pathways in MAGE-A3 antigen expressing cancer cells.
- An in vitro diagnostic tool to screen for cells expressing the MAGE-A3 tumor antigen.

*Competitive Advantages:*

- Class I restricted TCRs can only treat a subset of patients, but since ~80% of patients express the HLA-DP-beta1\*04 class II HLA allele, this TCR expands the population pool treatable with MAGE-A3 TCRs to include the majority of patients.
  - MAGE-A3 is a highly expressed tumor target on many cancer cells, so MAGE-A3 TCR therapy should be a viable treatment option for many cancer cases.
  - MAGE-A3 is only expressed on tumor cells and non-MHC expressing cells so these TCRs should target MAGE-A3 expressing tumor cells with little or no side effects/toxicity to normal cells.
- Development Stage:*
- Early-stage.
  - Pre-clinical.
  - In vitro data available.

*Inventors:* Paul Robbins, Xin Yao, Steven Rosenberg (NCI).

*Intellectual Property:* HHS Reference No. E-230-2012/0 — U.S. Patent Application No. 61/701,056 filed 14 Sep 2012.

*Related Technologies:*

- HHS Reference No. E-236-2010/0—PCT Patent Application No. PCT/US2011/057272.
- HHS Reference No. E-266-2011/0—PCT Patent Application No. PCT/US2012/054623.

*Licensing Contact:* Samuel E. Bish, Ph.D.; 301-435-5282; [bishse@mail.nih.gov](mailto:bishse@mail.nih.gov).

**Novel Small Molecule Agonists of the Relaxin Receptor as Potential Therapy for Heart Failure and Fibrosis**

*Description of Technology:* The present invention is directed to novel small molecule agonists of the mammalian relaxin family receptor 1 (RXFP1), including human RXFP1. Activation of RXFP1 induces: (1) Vasodilation due to up-regulation of the endothelin system; (2) extracellular matrix remodeling; (3) moderation of inflammation by reducing levels of inflammatory cytokines; and (4) angiogenesis. Small molecule agonists of RXFP1 may be useful in treating acute heart failure (AHF), scleroderma, fibrosis, other conditions associated with the biology of relaxin, and in improving reproductive health and wound healing. These compounds are the first and only small molecule agonists of RXFP1.

*Potential Commercial Applications:* Therapeutics for:

- Cardiovascular diseases.
- Ischemia.
- Fibrosis.
- Inflammation.
- Acute heart failure.
- Human and animal reproductive health.

*Competitive Advantages:*

- First and only small molecule agonists of RXFP1.
- Potent and highly selective.
- Bioavailable with excellent exposure.

- Easy to synthesize and scale-up.

*Development Stage:*

- Early-stage.
- In vitro data available.
- In vivo data available (animal).

*Inventors:* Juan J. Marugan (NCATS), et al.

*Publications:*

1. Chen ZC, et al. Identification of small-molecule agonists of human relaxin family receptor 1 (RXFP1) by utilizing a homogeneous cell-based cAMP assay. *J. Biomol. Screen.* 2012, accepted.

2. Additional manuscript is under revision.

*Intellectual Property:* HHS Reference No. E-072-2012/0—U.S. Provisional Application No. 61/642,986 filed 04 May 2012.

*Licensing Contact:* Lauren Nguyen-Antczak, Ph.D., J.D.; 301-435-4074; [nguyenantczakla@mail.nih.gov](mailto:nguyenantczakla@mail.nih.gov).

*Collaborative Research Opportunity:* The National Center for Advancing Translational Sciences is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize small molecule agonists of RXFP1. For collaboration opportunities, please contact Krishna (Balki) Balakrishnan, Ph.D. at 301-217-2336 or [balki@nih.gov](mailto:balki@nih.gov).

**Treatment of Tuberculosis—Adjuvant Therapies To Increase the Efficiency of Antibiotic Treatments**

*Description of Technology:* There is growing evidence that resistance to *Mycobacterium tuberculosis* infection is governed in large part by the regulation of host cell death. Lipid mediators called eicosanoids are thought to play a central role in this process. The subject invention is a novel method of enhancing the efficacy of antibiotic treatments for *Mycobacterium tuberculosis* infection by co-administering an inhibitor of 5-lipoxygenase and a COX-2 dependent prostaglandin. Inhibition of 5-lipoxygenase and treatment with prostaglandin E2 results in alteration of the eicosanoid balance. The synergistic effects of altering the eicosanoid balance and treatment with antibiotics is believed to result in more efficient reduction of the bacterial burden and thus, the period of antibiotic administration and antibiotic dosage could potentially be reduced. In vivo data from mouse models can be provided upon request.

*Potential Commercial Applications:* The subject invention can be used as an adjuvant therapy for existing antibiotic treatment regimens against tuberculosis.

*Competitive Advantages:* The disclosed method can be applied to increase the efficacy of existing antibiotic treatments for tuberculosis, potentially reducing both the duration and dosage of the antibiotic treatment.

*Development State:*

- Early-stage.
- Pre-clinical.
- In vitro data available.
- In vivo data available (animal).

*Inventors:* Katrin D. Mayer, Bruno Bezerril D. Andrade, F. Alan Sher, and Daniel L. Barber (NIAID).

*Intellectual Property:*

- HHS Reference No. E-189-2011/0—U.S. Provisional Patent Application No. 61/515,229 filed 04 Aug 2011.

- HHS Reference No. E-189-2011/1—U.S. Provisional Patent Application No. 61/515,237 filed 04 Aug 2011.

- HHS Reference No. E-189-2011/2—International Application No. PCT/US2012/049280 filed 02 Aug 2012.

*Licensing Contact:* Kevin W. Chang, Ph.D.; 301-435-5018; [changke@mail.nih.gov](mailto:changke@mail.nih.gov).

*Collaborative Research Opportunity:* The National Institute of Allergy and Infectious Diseases is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize adjuvant therapy for antibiotic treatment regimens against tuberculosis. For collaboration opportunities, please contact Katrin Mayer, Ph.D. at [mayerk@niaid.nih.gov](mailto:mayerk@niaid.nih.gov) or 301-594-8061.

**Adeno-Associated Virus Gene Therapy for Diabetes and Obesity**

*Description of Technology:* This invention is directed to adeno-associated virus (AAV) vector delivery of exendin-4 (Ex-4) to salivary glands as treatment for diabetes and obesity. Ex-4 is a potent and long-acting agonist of the receptor for glucagon-like peptide 1 (GLP-1). Scientists at NIDCR have shown that AAV-mediated delivery of Ex-4 resulted in improved glucose homeostasis and weight profile in two rat models of obesity and type 2 diabetes. Further, AAV-mediated delivery of Ex-4 to rat salivary glands resulted in localized and sustained expression of Ex-4 that was biologically active and well tolerated.

AAV-mediated delivery of Ex-4 is superior to administering GLP-1 analogs in that AAV-Ex-4 expression is more stable and longer acting. Like GLP-1 analogs, Ex-4 expression also potentially provides beneficial effects like reduced hypoglycemia, appetite suppression, and potential weight loss.

*Potential Commercial Applications:* Therapy for diabetes or obesity.

*Competitive Advantages:*

- Potential for potent glucose homeostasis therapy with longer duration than current drugs.
- More convenient than daily or weekly injections.

*Development Stage:*

- Early-stage.
- In vitro data available.
- In vivo data available (animal).

*Inventors:* John A. Chiorini (NIDCR), Giovanni DiPasquale (NIDCR), Edoardo Mannucci (Careggi Teaching Hospital).

*Publication:* Di Pasquale G, et al. Sustained exendin-4 secretion through

gene therapy targeting salivary glands in two different rodent models of obesity/type 2 diabetes. PLoS One. 2012;7(7):e40074. [PMID 22808093]

*Intellectual Property:* HHS Reference No. E-142-2011/0—

- U.S. Application No. 61/477,523 filed 20 May 2011.

- PCT Application No. PCT/US2012/34268 filed 19 Apr 2012.

*Licensing Contact:* Lauren Nguyen-Antczak, Ph.D., J.D.; 301-435-4074; [nguyenantczakla@mail.nih.gov](mailto:nguyenantczakla@mail.nih.gov).

*Collaborative Research Opportunity:* The NIDCR is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize treatment of diabetes by expression NGF-extendin 4 protein. For collaboration opportunities, please contact David Bradley, Ph.D. at 301-402-9242 or [bradleyda@nidcr.nih.gov](mailto:bradleyda@nidcr.nih.gov).

### Small Molecule MRS5474 With Anticonvulsant Activity for Treatment of Epilepsy

*Description of Technology:* Adenosine modulates many physiological processes by activating specific adenosine receptors. These adenosine receptors play a critical role in the regulation of cellular signaling and are broadly distributed throughout the body. Thus, the ability to modulate adenosine receptor-mediated signaling is an attractive therapeutic strategy for a broad range of diseases. This technology relates to a group of compounds that display high affinity and specificity for the A1 adenosine receptor subtype.

One of the compounds, MRS5474, displays anticonvulsant activity in the 6 Hz animal model of clonic seizures. In the minimal behavioral toxicity test using the rotarod, no toxicity (zero out of eight mice) was observed at all doses tested up to 30 mg/kg, the highest dose tested, which was nearly completely protective (seven out of eight animals) in the 6 Hz model. MRS 5474 also tested well in the corneal kindled mouse model to examine its effect on focal seizures.

*Potential Commercial Applications:*

- Oral anticonvulsant drug.
- Provides a means to mimic A1AR mediated signaling in vitro and in vivo.

*Competitive Advantages:*

- These small molecules display increased specificity for the A1 type of adenosine receptors, which may reduce unwanted side effects previously seen in A1AR agonist therapies.

- The physical properties of these molecules are drug-like, which makes them attractive for pre-clinical development.

*Development Stage:*

- Early-stage.
- In vitro data available.
- In vivo data available (animal).

*Inventor:* Kenneth A. Jacobson (NIDDK).

*Publication:* Tosh DK, et al. Truncated (N)-Methanocarba Nucleosides as A1 Adenosine Receptor Agonists and Partial Agonists: Receptor Docking and Potent Anticonvulsant Activity. In preparation.

*Intellectual Property:*

- HHS Reference No. E-285-2008/0—International Application No. PCT/US2009/52439 filed 31 Jul 2009.

- HHS Reference No. E-285-2008/1—U.S. Patent Application No. 13/479,973 filed 24 May 2012.

*Licensing Contact:* Betty B. Tong, Ph.D.; 301-594-6565; [tongb@mail.nih.gov](mailto:tongb@mail.nih.gov).

*Collaborative Research Opportunity:* The NIDDK is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize MRS5474, A1 adenosine receptor agonist for treatment of seizures. For collaboration opportunities, please contact Marguerite Miller at [miller marg@mail.nih.gov](mailto:miller marg@mail.nih.gov).

### Glucocorticoid-Induced TNFR Family-Related Receptor Ligand (GITRL) Antibodies for Diagnosis and Treatment of Immune System Disorders

*Description of Technology:* This technology provides novel antibodies and methods for diagnostics and treatment of disorders arising from dysregulation of the immune system using antibodies directed against glucocorticoid-induced tumor necrosis factor receptor family-related receptor ligand (GITRL). Also available are hybridomas producing anti-mouse GITRL monoclonal antibodies (clone 5F1).

Glucocorticoid-induced TNFR family-related receptor (GITR, also known as TNFRSF18) is expressed on the surface of responder T cells (CD4+CD25- or CD8+CD25- T cells). Upon activation of the immune response, GITR is up-regulated and binds to its ligand, GITRL (also known as TNFSF18), which enhances the immune response. The inventors have developed anti-GITRL monoclonal antibodies that block the interaction between GITR and GITRL, and have demonstrated in vitro experiments that administration of these blocking antibodies can suppress the immune response. These antibodies may be useful for treatment of immune system disorders such as multiple sclerosis, rheumatoid arthritis, and other inflammatory diseases.

*Potential Commercial Applications:*

- Development of therapeutic agents for autoimmune diseases, including autoimmune and inflammatory diseases, allergy and transplant rejection.

- Tool for investigating the role of GITRL in enhancement of the T-cell mediated immune response.

*Competitive Advantages:* The GITR/GITRL pathway is a novel target for the treatment of autoimmune diseases.

*Development Stage:*

- In vitro data available.
- In vivo data available (animal).

*Inventors:* Ethan Shevach et al. (NIAID).

*Publication:* Stephens GL, et al. Engagement of glucocorticoid-induced TNFR family-related receptor on effector T cells by its ligand mediates resistance to suppression by CD4+CD25+ T cells. J Immunol. 2004 Oct 15;173(8):5008-20. [PMID 15470044].

*Intellectual Property:* HHS Reference No. E-229-2003/2—

- U.S. Patent No. 7,618,632 issued 17 Nov 2009.

- JP Patent No. 4638876 issued 03 Dec 2010.

*Licensing Contact:* Tara L. Kirby, Ph.D.; 301-435-4426; [tarak@mail.nih.gov](mailto:tarak@mail.nih.gov).

### Treatment for Ichthyosiform Skin Diseases

*Description of Technology:* A synthetic composition that contains the transglutaminase 1 (TGase I) enzyme and a lipid vesicle, which can be used to provide ameliorative therapy for inherited autosomal recessive ichthyoses (ARI). Ichthyoses are rare inherited skin disorders that result in extensive scaling of the skin. Because this abnormality can affect heat and fluid transfer through the skin, individuals with this disease may have an increased risk for dehydration and skin infections. Each year, more than 16,000 babies are born with some form of ichthyosis. Ichthyosis affects people of all ages, races and gender. Currently, there is no cure for this disease and the only treatments available alleviate symptoms without affecting the disease itself. ARI are often caused by defects in lipid barrier function in the skin and are the result of genetic errors of either protein or lipid synthesis. One such disease, termed lamellar ichthyosis, is caused by genetic inactivation of the (TGase I) gene. The TGase I enzyme is essential for maintaining proper skin cornification, which protects skin cells against water loss and infection. Rather than simply treating the disease symptoms superficially, this technology provides a platform for treating the

underlying cause of disease, namely the absence of TGase I function.

*Potential Commercial Applications:*

- Treatment for ichthyosiform skin diseases.

- Method for correcting defects in skin cell cornification.

*Competitive Advantages:* Targets underlying cause of skin disorder rather than just treating the resulting symptoms.

*Development Stage:*

- Early-stage.
- In vitro data available.

*Inventors:* Peter M Steinert, Nemes Zoltan, Lyuben N Marckov (NIAMS).

*Publications:*

1. Candi E, et al. Transglutaminase 1 mutations in lamellar ichthyosis. Loss of activity due to failure of activation by proteolytic processing. *J Biol Chem.* 1998 May 29;273(22):13693–702. [PMID 9593710]

2. Yang YM, et al. Novel mutations of the transglutaminase 1 gene in lamellar ichthyosis. *J Invest Dermatol.* 2001 Aug;117(2):214–8. [PMID 11511296]

*Intellectual Property:* HHS Reference No. E–149–1999/0—U.S. Patent No. 6,852,686 issued 08 Feb 2005.

*Licensing Contact:* Suryanarayana Vepa, Ph.D., J.D.; 301–435–5020; [vepas@mail.nih.gov](mailto:vepas@mail.nih.gov).

Dated: November 16, 2012.

**Richard U. Rodriguez,**

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

[FR Doc. 2012–28276 Filed 11–20–12; 8:45 am]

**BILLING CODE 4140–01–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

#### National Center for Complementary and Alternative Medicine; Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. App.), 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.

*Name of Committee:* National Center for Complementary and Alternative Medicine

Special Emphasis Panel, PAR 12–151: Centers of Excellence for Research on Complementary Alternative Medicine (CAM).  
Date: January 16–18, 2013.

*Time:* 5 p.m. to 5 p.m..

*Agenda:* To review and evaluate grant applications.

*Place:* Bethesda North Marriott Hotel & Conference Center, 5701 Marinelli Road, Bethesda, MD 20852.

*Contact Person:* Martina Schmidt, Ph.D. Scientific Review Officer, Office of Scientific Review, National Center for Complementary, & Alternative Medicine, NIH, 6707 Democracy Blvd., Suite 401, Bethesda, MD 20892, 301–594–3456, [schmidma@mail.nih.gov](mailto:schmidma@mail.nih.gov).

(Catalogue of Federal Domestic Assistance Program Nos. 93.213, Research and Training in Complementary and Alternative Medicine, National Institutes of Health, HHS)

Dated: November 15, 2012.

**Michelle Trout,**

*Program Analyst, Office of Federal Advisory Committee Policy.*

[FR Doc. 2012–28278 Filed 11–20–12; 8:45 am]

**BILLING CODE 4140–01–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

#### National Advisory Council on Alcohol Abuse and Alcoholism, National Advisory Council on Drug Abuse, and National Cancer Advisory Board; Notice of Joint Meeting

Pursuant to section 10(a) of the Federal Advisory Committee Act, as amended (5 U.S.C. App.), notice is hereby given of a joint teleconference and Web cast meeting of the National Advisory Council on Alcohol Abuse and Alcoholism, National Advisory Council on Drug Abuse, and National Cancer Advisory Board. The meeting will be open to the public as indicated below.

*Name of Committee:* National Advisory Council on Alcohol Abuse and Alcoholism, National Advisory Council on Drug Abuse, and National Cancer Advisory Board.

*Date:* December 13, 2012.

*Time:* 11:30 a.m. to 1:00 p.m. EST.

*Agenda:* NIH report on the functional integration of substance use, abuse and addiction-related research and discussion with the NIH Principal Deputy Director and Members of NIAAA and NIDA Councils, and the NCAB.

*Teleconference Line:* 1–888–324–8014 (toll-free); Passcode: 4418818.

*Webcast Site:* <https://webmeeting.nih.gov/suaa/>.

Contact Persons: Abraham Bautista, Ph.D., Office of Extramural Activities, National Institute on Alcohol Abuse and Alcoholism, National Institutes of Health, 5635 Fishers Lane, RM 2085, Rockville, MD 20892, 301–443–9737, [bautista@mail.nih.gov](mailto:bautista@mail.nih.gov).

Teresa Levitin, Ph.D., Office of Extramural Affairs, National Institute on Drug Abuse, National Institutes of Health, 6001 Executive Blvd., RM 4243, Rockville, MD 20852, 301–443–2755, [tlevitin@nida.nih.gov](mailto:tlevitin@nida.nih.gov). Paulette S. Gray, Ph.D., Division of Extramural Activities, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, 8th Floor, Rm. 8001, Bethesda, MD 20892, 301–496–5147, [grayp@mail.nih.gov](mailto:grayp@mail.nih.gov).

Any interested person may file written comments with the committee(s) by forwarding the statement to a 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 will also be available on the Institute's/Center's home pages: <http://www.niaaa.nih.gov/AboutNIAAA/AdvisoryCouncil/Pages/default.aspx>, <http://www.drugabuse.gov/about/organization/nacda/NACDAHome.html>, and <http://deainfo.nci.nih.gov/advisory/ncab/ncab.htm>, as well as NIH's Feedback page: <http://feedback.nih.gov>, where an agenda and any additional information for the meeting will be posted when available.

(Catalogue of Federal Domestic Assistance Program Nos.: 93.271, Alcohol Research Career Development Awards for Scientists and Clinicians; 93.272, Alcohol National Research Service Awards for Research Training; 93.273, Alcohol Research Programs; 93.891, Alcohol Research Center Grants; 93.701, ARRA Related Biomedical Research and Research Support Awards, National Institutes of Health, HHS)

Dated: November 16, 2012.

**Jennifer S. Spaeth,**

*Director, Office of Federal Advisory Committee Policy.*

[FR Doc. 2012–28277 Filed 11–20–12; 8:45 am]

**BILLING CODE 4140–01–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

#### National Heart, Lung, and Blood Institute; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. App.), 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.