register and existing establishments will update their annual registration using choices on the DRLM menu. Once you choose to register or update your annual registration, the system will prompt you through the entry of information about your establishment and your devices. If you have any problems with this process, email: *reglist@cdrh.fda.gov* or call 301–796–7400 for assistance. (Note: this email address and this telephone number are for assistance with establishment registration only, and not for any other aspects of medical device user fees.) Problems with BERS should be directed to *bloodregis@fda.hhs.gov* or call 301–827–3546.

D. Step Four—Enter Your DFUF Order PIN and PCN

After completing your annual or initial registration and device listing, you will be prompted to enter your DFUF order PIN and PCN, when applicable. This process does not apply to establishments engaged only in the manufacture, preparation, propagation, compounding, or processing of licensed biologic devices. CBER will send invoices for payment of the establishment registration fee to such establishments.

Dated: July 24, 2012.

Leslie Kux,

Assistant Commissioner for Policy.

[FR Doc. 2012–18647 Filed 7–30–12; 8:45 a.m.]

BILLING CODE 4160–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Health Resources and Services Administration

Agency Information Collection Activities: Proposed Collection: Comment Request

In compliance with the requirement for opportunity for public comment on proposed data collection projects (section 3506(c)(2)(A) of Title 44, United States Code, as amended by the Paperwork Reduction Act of 1995, Pub. L. 104–13), the Health Resources and Services Administration (HRSA) publishes periodic summaries of proposed projects being developed for submission to the Office of Management and Budget (OMB) under the Paperwork Reduction Act of 1995. To request more information on the proposed project or to obtain a copy of the data collection plans and draft instruments, email *paperwork@hrsa.gov* or call the HRSA Reports Clearance Officer at (301) 443–1984.

Comments are invited on: (a) The proposed collection of information for the proper performance of the functions of the Agency; (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: Maternal and Child Health Bureau Performance Measures for Discretionary Grants (OMB No. 0915–0298)—[Revision]

The Health Resources and Services Administration's (HRSA) Maternal and Child Health Bureau (MCHB) intends to continue to collect performance data for Special Projects of Regional and National Significance (SPRANS), Community Integrated Service Systems (CISS), and other grant programs administered by MCHB.

HRSA's MCHB proposes to continue using reporting requirements for SPRANS projects, CISS projects, and other grant programs administered by MCHB, including national performance measures, previously approved by OMB, and in accordance with the ''Government Performance and Results Act (GPRA) of 1993'' (Pub. L. 103–62). This Act requires the establishment of measurable goals for Federal Programs that can be reported as part of the budgetary process, thus linking funding decisions with performance. Performance measures for MCHB discretionary grants were initially approved in January 2003. Approval from OMB is being sought to continue the use of these measures. Some of these measures are specific to certain types of programs and will not apply to all grantees. Through the experience of utilizing these measures, we are enhancing them to better reflect program goals. Specifically, additional outcome measures that can be utilized by grantees that predominantly provide infrastructure services are being developed for submission to OMB.

The estimated response burden is as follows:

Form	Number of respondents	Responses per respondent	Total responses	Burden hours per response	Total burden hours
Grant Report	900	1	900	41	36,900
Total	900		900		36,900

Email comments to *paperwork@hrsa.gov* or mail the HRSA Reports Clearance Officer, Room 10–29, Parklawn Building, 5600 Fishers Lane, Rockville, MD 20857. Written comments should be received within 60 days of this notice.

Dated: July 24, 2012.

Jennifer Riggle,

Deputy Director, Office of Management.

[FR Doc. 2012–18637 Filed 7–30–12; 8:45 am]

BILLING CODE 4165–15–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.

Glial Cell Line-Derived Neurotrophic Factor Opposite Strand (GDNFOS) for Treatment of Neurodegenerative Diseases

Description of Technology: Glial cell line-derived neurotrophic factor (GDNF) is a small human protein encoded by the GDNF gene. GDNF has been effective therapy in laboratory animal models of Parkinson's disease and protects several types of neurons in the brain and peripheral nervous system. The NIDA inventors have discovered primate-specific GDNFOS, encoded by the opposite strand of glial cell derived neurotrophic factor (GDNF) gene. The GDNFOS gene encodes for novel peptides that was found to be reduced in human middle temporal gyrus of Alzheimer's disease brains. These secreted growth proteins have potential neurotrophic activity and they might play a synergistic role in neuroprotective effects of GDNF in human brain. The NIDA inventors have also developed antibody against GDNFOS3 and generated compounds that have potential pharmaceutical use. The compounds consist of GDNFOS nucleic acid transcripts, GDNFOS protein or a functional fragment for treatment of human neurodegenerative diseases.

Potential Commercial Applications

- Synergistic role in neuroprotective effects of GDNF.
- Alzheimer's disease, Parkinson's disease, Amyotrophic lateral sclerosis, multiple sclerosis and diseases of peripheral organs such as diabetes mellitus type 1.

Competitive Advantages

- Secreted novel growth peptides.
- An antibody against GDNFOS3 was developed.

Development Stage

- Early-stage.
- Pre-clinical.
- In vitro data available.

Inventors: Qing-Rong Liu, Mikko Airavaara, Barry Hoffer, Brandon K Harvey (all of NIDA).

Publication: Airavaara M, et al. Identification of novel GDNF isoforms and cis-antisense GDNFOS gene and their regulation in human middle temporal gyrus of Alzheimer disease. J Biol Chem. 2011 Dec 30;286(52):45093–102. [PMID 22081608]

Intellectual Property: HHS Reference No. E–044–2012/0—U.S. Provisional Application No. 61/619, 296 filed 02 Apr 2012.

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

Collaborative Research Opportunity: The National Institute on Drug Abuse is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize GDNFOS peptide and non-coding RNAs as therapeutic agents for neurodegenerative diseases. For collaboration opportunities, please contact Vio Conley at *conleyv@mail.nih.gov.*

Increased Therapeutic Effectiveness of Immunotoxins That Use Toxin Domains Lacking Human B-cell Epitopes

Description of Technology: Immunotoxins kill cancer cells while allowing healthy, essential cells to survive. As a result, patients receiving an immunotoxin are less likely to experience the deleterious side-effects associated with non-discriminate therapies such as chemotherapy or radiation therapy. Unfortunately, the continued administration of immunotoxins often leads to a reduced patient response due to the formation of neutralizing antibodies against immunogenic epitopes contained within Pseudomonas exotoxin A (PE). To improve the therapeutic effectiveness of PE-based immunotoxins through multiple rounds of drug administration, NIH inventors have sought to identify and remove the human B-cell epitopes within PE. Previous work demonstrated that the removal of the murine B-cell and T-cell epitopes from PE reduced the immunogenicity of PE and resulted in immunotoxins with improved therapeutic activity. This technology involves the identification and removal of major human B-cell epitopes on PE by mutation or deletion. Considering these immunotoxins will be administered to humans, the removal of human immunogenic epitopes is important. The resulting PE-based immunotoxins have increased resistance to the formation of neutralizing antibodies, and are expected to have improved therapeutic efficacy.

Potential Commercial Applications

- Essential component of immunotoxins.
- Treatment of any disease associated with increased or preferential expression a specific cell surface receptor.
- Specific diseases include hematological cancers, lung cancer, ovarian cancer, breast cancer, and head and neck cancers.

Competitive Advantages

- PE variants now include the removal of human B-cell epitopes, further reducing the formation of neutralizing antibodies against immunotoxins which contain the PE variants.
- Less immunogenic immunotoxins result in improved therapeutic efficacy by permitting multiple rounds of administration in humans.
- Targeted therapy decreases non-specific killing of healthy, essential cells, resulting in fewer non-specific side-effects and healthier patients.

Development Stage: Pre-clinical.

Inventors: Ira H. Pastan et al. (NCI).

Publication: Liu W, et al. Recombinant immunotoxin engineered for low immunogenicity and antigenicity by identifying and silencing human B-cell epitopes. Proc Natl Acad Sci USA. 2012 Jul 17;109(29):11782–7. [PMID 22753489]

Intellectual Property: HHS Reference No. E–263–2011/0—U.S. Provisional Application No. 61/535,668 filed 16 Sep 2011.

Related Technologies

- PCT Patent Publication WO 2011/032022 (HHS Reference No. E–269–2009/0–PCT–02).
- US Patent Publication US 20100215656 A1 (HHS Reference No. E–292–2007/0–US–06).
- US Patent Publication US 20090142341 A1 (HHS Reference No. E–262–2005/0–US–06).
- Multiple additional patent families.

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

Collaborative Research Opportunity: The National Cancer Institute is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize this technology. For collaboration opportunities, please contact John Hewes, Ph.D. at *hewesj@mail.nih.gov.*

Novel Nitroxyl (HNO) Releasing Compounds and Their Use in Treating Diseases

Description of Technology: Nitroxyl (HNO) is a chemical species that exhibits distinct biological properties in comparison to its oxidized product, nitric oxide (NO). Previous investigations have revealed that the

distinct properties of HNO make it a tempting species for wide therapeutic application as it has shown potential in the treatment of heart failure, cancer, and other diseases in various animal and in vitro models. Non-steroidal anti-inflammatory drugs (NSAIDs), such as aspirin and ibuprofen, are compounds that inhibit cycloxygenase (COX)-mediated conversion of arachidonic acids to prostagladins. NSAIDs are known for their analgesic properties and are therapeutically involved in many physiological functions, including the inhibition of chronic pain and inflammation inhibition, prevention of heart disease, renal function, and cancer. Prolonged use of NSAIDs can lead to serious gastrointestinal and renal side effects, including ulcer perforation, upper gastrointestinal bleeding, and death, which has limited NSAID therapies.

The instant invention described HNO-releasing NSAIDs, which combine the potential therapeutic benefits of HNO and NSAIDs without the toxicities associated with chronic NSAID use. These HNO-releasing NSAIDs provide a reliable controlled release of HNO making them desirable HNO prodrugs. The instant invention disclosed various HNO-releasing NSAIDs and methods of treating or preventing various disorders with these compositions, such as cardiovascular disorders, cancers, pain, inflammation, and alcoholism.

Potential Commercial Applications

- Treatment of cancer.
- Treatment of cardiovascular disease.
- Aversion therapy for alcoholism.

Competitive Advantages

- Combination of therapeutic benefits of HNO and NSAIDs.
- Alleviated toxicity associated with chronic NSAID use.
- Controlled release of HNO.

Development Stage

- Early-stage.
- Pre-clinical.

Inventors: David A. Wink and Larry K. Keefer (NCI).

Publication

Miranda KM, et al. Comparison of the NO and HNO donating properties of diazeniumdiolates: primary amine adducts release HNO in vivo. J Med Chem. 2005 Dec 29;48(26):8220–8. [PMID 16366603]

Intellectual Property: HHS Reference No. E–019–2010/2—International Patent Application PCT/US2011/029072 filed 18 Mar 2011.

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

Polyclonal Antibodies for the Specialized Signaling G Protein, Gbeta5

Description of Technology: Researchers at NIDDK have developed polyclonal antibodies against the G protein, Gbeta5. Gbeta5 is a unique and highly specialized G protein that exhibits much less homology than other Gbeta isoforms (~50%) and is preferentially expressed in brain and neuroendocrine tissue. It is expressed prominently in the neuronal cell membrane, as well as in the cytosol and nucleus. Although this distribution pattern suggests that Gbeta5 may shuttle information between classical G protein-signaling elements at the plasma membrane and the cell interior, its function in the brain is largely unknown.

The antibodies were separately generated in rabbits to KLH-conjugates of peptides from the N-terminus of Gbeta5 (antibody ATDG) and the C-terminus of Gbeta5 (antibody SGS). The antibodies can be used for immunoblotting (ATDG, SGS), and immunoprecipitation (ATDG). They can be used to facilitate our understanding of the unique biology and function of Gbeta5 in brain and neurons.

Potential Commercial Applications: These antibodies can be used for research purposes (immunoblotting, immunoprecipitation) by those studying the biology and function of Gbeta5.

Competitive Advantages: Very specific antibodies to study Gbeta5 and G protein signaling.

Development Stage: In vitro data available.

Inventors: William Simonds and Jianhua Zhang (NIDDK).

Publications

1. Zhang JH and Simonds WF. Copurification of brain G-protein beta5 with RGS6 and RGS7. J Neurosci. 2000 Feb 1;20(3):RC59. [PMID 10648734]

2. Zhang JH, et al. Nuclear localization of G protein beta 5 and regulator of G protein signaling 7 in neurons and brain. J Biol Chem. 2001 Mar 30;276(13):10284–9. [PMID 11152459]

3. Zhang S, et al. Selective activation of effector pathways by brain-specific G protein beta5. J Biol Chem. 1996 Dec 27;271(52):33575–9. [PMID 8969224]

4. Zachariou V, et al. An essential role for DeltaFosB in the nucleus accumbens in morphine action. Nat Neurosci. 2006 Feb;9(2):205–11. [PMID 16415864]

Intellectual Property: HHS Reference No. E–192–2006/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Contact: Jaime Greene, M.S.; 301–435–5559; *greenejaime@mail.nih.gov.*

Polyclonal Antibodies for the Gbeta5-Associated Regulator of G Protein Signaling Protein, RGS7

Description of Technology: Researchers at NIDDK have developed polyclonal antibodies against the Regulator of G Protein Signalling (RGS) protein, RGS7. RGS7 binds tightly to Gbeta5, a unique and highly specialized G protein that exhibits much less homology than other Gbeta isoforms (~50%). RGS7 is preferentially expressed in brain and neuroendocrine tissue. Like Gbeta5, RGS7 is expressed prominently in the cell membrane, as well as in the cytosol. Although this distribution pattern suggests that complexes containing Gbeta5 and RGS7 may shuttle information between classical G protein-signaling elements at the plasma membrane and the cell interior, the function of the complex in the brain is largely unknown.

The antibodies were generated in rabbits to a glutathione S-transferase (GST) fusion protein with residues 312–469 of bovine RGS7 (antibody 7RC–1) and react with human and rodent RGS7. The antibodies (7RC–1) can be used for immunoblotting and immunoprecipitation. They can be used to facilitate our understanding of the function of Gbeta5/RGS7 complexes in brain and neurons.

Potential Commercial Applications: These antibodies can be used for research purposes (immunoblotting, immunoprecipitation) by those studying the biology and function of RGS7.

Competitive Advantages: High-titer, multi-epitope antibodies to study RGS7 and RGS7/Gbeta5 complexes and G protein signaling.

Development Stage: In vitro data available.

Inventors: William Simonds and Jianhua Zhang (NIDDK).

Publications

1. Rojkova AM, et al. Ggamma subunit selective G protein beta 5 mutant defines regulators of G protein signaling binding requirement for nuclear localization. J Biol Chem. 2003 Apr 4;278(14):12507–12. [PMID 12551930]

2. Cao Y, et al. Retina Specific GTPase Accelerator RGS11/Gbeta5S/R9AP is a Constitutive Heterotrimer Selectively Targeted to mGluR6 in ON–Bipolar Neurons. J Neurosci 2009 July 22; 29 (29): 9301–13. [PMID 19625520]

3. Anderson GR, et al. Changes in striatal signaling induce remodeling of RGS complexes containing Gbeta5 and R7BP subunits. Mol Cell Biol. 2009 Jun;29(11): 3033–44. [PMID 19332565]

4. Panicker LM, et al. Nuclear localization of the G protein beta5/R7-regulator of G protein signaling protein complex is dependent on R7 binding protein. J

Neurochem. 2010 Jun;113(5):1101–12. [PMID 20100282]

Intellectual Property: HHS Reference No. E–077–2011/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Contact: Jaime Greene, M.S.; 301–435–5559; *greenejaime@mail.nih.gov.*

Dated: July 24, 2012.

Richard U. Rodriguez,

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

[FR Doc. 2012–18651 Filed 7–30–12; 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 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: Center for Scientific Review Special Emphasis Panel Member Conflict: Skeletal Pathobiology and Orthopedics.

Date: August 28, 2012.

Time: 3:00 p.m. to 5:00 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: Yi-Hsin Liu, Ph.D., Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 4214, MSC 7814, Bethesda, MD 20892, 301–435–1781, *liuyh@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: July 25, 2012.

David Clary,

Program Analyst, Office of Federal Advisory Committee Policy.

[FR Doc. 2012–18650 Filed 7–30–12; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HOMELAND SECURITY

U.S. Customs and Border Protection

[Docket No. USCBP–2012–0027]

Advisory Committee on Commercial Operations of Customs and Border Protection (COAC)

AGENCY: U.S. Customs and Border Protection, Department of Homeland Security (DHS).

ACTION: Committee Management; Notice of Federal Advisory Committee Meeting.

SUMMARY: The Advisory Committee on Commercial Operations of Customs and Border Protection (COAC) will meet on August 15, 2012, in Seattle, WA. The meeting will be open to the public.

DATES: COAC will meet on Wednesday, August 15, 2012 from 1:00 p.m. to 5:30 p.m. PST. Please note that the meeting may close early if the committee has completed its business.

Registration: If you plan on attending, please register either online at *https://apps.cbp.gov/te_registration/index.asp?w=80* or by email to *tradeevents@dhs.gov,* or by fax to 202–325–4290 by close-of-business on August 12, 2012.

If you have completed an online on-site registration and wish to cancel your registration, you may do so at *https://apps.cbp.gov/te_registration/cancel.asp?w=80.* Please feel free to share this information with interested members of your organizations or associations.

ADDRESSES: The meeting will be held at Jackson Federal Building, 915 2nd Avenue, Seattle, WA 98174, in the South Auditorium—4th Floor. All visitors report to main lobby of the building. All visitors to the Jackson Federal Building must show a state-issued ID or Passport to proceed through the security checkpoint to be admitted to the building.

For information on facilities or services for individuals with disabilities or to request special assistance at the meeting, contact Ms. Wanda Tate, Office of Trade Relations, U.S. Customs and Border Protection at 202–344–1661 as soon as possible.

To facilitate public participation, we are inviting public comment on the issues to be considered by the committee as listed in the ''Agenda'' section below.

Comments must be submitted in writing no later than August 8, 2012, and must be identified by USCBP–2012–0027 and may be submitted by *one* of the following methods:

- *Federal eRulemaking Portal: http://www.regulations.gov.* Follow the instructions for submitting comments.
- *Email: Tradeevents@dhs.gov.* Include the docket number in the subject line of the message.
- *Fax:* 202–325–4290
- *Mail:* Ms. Wanda Tate, Office of Trade Relations, U.S. Customs and Border Protection, 1300 Pennsylvania Avenue NW., Room 3.5A, Washington, DC 20229.

Instructions: All submissions received must include the words ''Department of Homeland Security'' and the docket number for this action. Comments received will be posted without alteration at *http://www.regulations.gov,* including any personal information provided. Do not submit personal information to this docket.

Docket: For access to the docket to read background documents or comments received by the COAC, go to *http://www.regulations.gov.*

There will be two public comment periods held during the meeting on August 15, 2012. Speakers are requested to limit their comments to two (2) minutes or less to facilitate greater participation. Contact the individual listed below to register as a speaker. Please note that the public comment period for speakers may end before the time indicated on the schedule that is posted on the CBP web page at the time of the meeting.

FOR FURTHER INFORMATION CONTACT: Ms. Wanda Tate, Office of Trade Relations, U.S. Customs and Border Protection, 1300 Pennsylvania Avenue NW., Room 3.5A, Washington, DC 20229; telephone 202–344–1440; facsimile 202–325–4290.

SUPPLEMENTARY INFORMATION: Notice of this meeting is given under the *Federal Advisory Committee Act,* 5 U.S.C. App. (Pub. L. 92–463). The COAC provides advice to the Secretary of Homeland Security, the Secretary of the Treasury, and the Commissioner of U.S. Customs and Border Protection (CBP) on matters pertaining to the commercial operations of CBP and related functions within DHS or the Department of the Treasury.

Agenda

The COAC will hear from the following subcommittees on the topics listed below and then will review, deliberate, and formulate