

## ESTIMATED ANNUALIZED BURDEN HOURS

Type of respondents	Data collection	Number of respondents	Number of responses per respondent	Average time per response	Total annual burden hours
DAIDS Staff, ER/ES .....	Survey .....	500	1	30/60	250
	Focus Group-IC Review .....	81	1	10/60	14
	Focus Group .....	81	1	90/60	122

Dated: April 3, 2014.

**Brandie Taylor,**

*Project Clearance Liaison, NIAID, NIH.*

[FR Doc. 2014-07960 Filed 4-8-14; 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, 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. 209 and 37 CFR part 404 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.

#### Monoclonal Antibody Fragments for Targeting Therapeutics to Growth Plate Cartilage

*Description of Technology:* A child's growth is dependent on the proper functioning of the growth plate, a specialized cartilage structure located at the ends of long bones and within the vertebrae. The primary function of the growth plate is to generate new cartilage, which is then converted into bone tissue and results in the lengthening of bones. Current

treatments for severe short stature and skeletal growth disorders are limited. Recombinant human growth hormone (GH) is typically used but the results are less than optimal and have potential adverse effects. The instant invention discloses that monoclonal antibodies that bind to matrilin-3, a protein specifically expressed in cartilage tissue, could be used for treating or inhibiting growth plate disorders, such as a skeletal dysplasia or short stature.

*Potential Commercial Applications:* A new treatment option for growth plate disorders, such as skeletal dysplasia or short stature.

*Competitive Advantages:* Avoidance of the risks associated with systemic treatment using growth hormone, such as increased intracranial pressure, slipped capital femoral epiphysis, insulin resistance, and possibly type II diabetes.

*Development Stage:*

- Early-stage.
- *In vitro* data available.

*Inventors:* Jeffrey Baron (NICHD), Sao Fong (Crystal) Cheung (NICHD), Chun Kin Julian Lui (NICHD), Dimitar S. Dimitrov (NCI), Zhongyu Zhu (NCI).

*Intellectual Property:* HHS Reference No. E-003-2014/0—US Application No. 61/927,904 filed 15 Jan 2014.

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

*Collaborative Research Opportunity:* The Eunice Kennedy Shriver National Institute of Child Health and Human Development and the National Cancer Institute are seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize treatment of skeletal disorders and short stature to increase growth using targeting antibodies. For collaboration opportunities, please contact Joseph Conrad III, Ph.D. at [jmconrad@mail.nih.gov](mailto:jmconrad@mail.nih.gov).

#### Human Antibodies Against Middle East Respiratory Syndrome Coronavirus

*Description of Technology:* No effective therapeutics or vaccines are available against Middle East Respiratory Syndrome Coronavirus (MERS-CoV). This technology is for

human antibodies targeting MERS-CoV. Certain of these antibodies bind with epitopes of the MERS-CoV receptor binding domain (RBD) of MERS-CoV spike (S) protein with high affinity and are capable of neutralized the virus in a pseudovirus assay. The MERS-CoV-S protein is believed to be required for binding and virus entry during MERS-CoV infection. The human to human aspect of transmission and the high mortality rate associated with MERS-CoV infection have raised concerns over the potential for a future MERS-CoV pandemic and emphasized the need for development of effective therapeutics and vaccines. The antibodies of this technology represent candidate antibody-based therapeutics for treatment of MERS-CoV infection.

*Potential Commercial Applications:* Antibody-based therapeutics for treatment of MERS-CoV infection.

*Competitive Advantages:*

- No vaccine or other biologic therapy is available.
- High binding (sub-nanomolar) affinity.
- Relative safety and long half-lives.

*Development Stage:*

- Early-stage.
- *In vitro* data available.

*Inventors:* Dimitar Dimitrov (NCI), Tianlei Ying (NCI), Tina Yu (NCI), Kwok Yuen (University of Hong Kong).

*Publications:*

1. Zaki AM, et al. Isolation of a novel coronavirus from a man with pneumonia in Saudi Arabia. *N Engl J Med.* 2012 Nov 8;367(19):1814-20. [PMID 23075143]
2. Zhu Z, et al. Exceptionally potent cross-reactive neutralization of Nipah and Hendra viruses by a human monoclonal antibody. *J Infect Dis.* 2008 Mar 15;197(6):846-53. [PMID 18271743]
3. Zhu Z, et al. Potent cross-reactive neutralization of SARS coronavirus isolates by human monoclonal antibodies. *Proc Natl Acad Sci U S A.* 2007 Jul 17;104(29):12123-8. [PMID 17620608]

*Intellectual Property:* HHS Reference No. E-002-2014/0—U.S. Patent Application No. 61/892,750 filed 18 Oct 2013.

*Licensing Contact:* Tedd Fenn; 424–297–0336; [Tedd.Fenn@nih.gov](mailto:Tedd.Fenn@nih.gov).

*Collaborative Research Opportunity:* The National Cancer Institute, Cancer and Inflammation Program, Laboratory of Experimental Immunology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize animal studies, cGMP Manufacturing, clinical trials. For collaboration opportunities, please contact John D. Hewes, Ph.D. at [hewesj@mail.nih.gov](mailto:hewesj@mail.nih.gov).

### **Novel Small Molecule Antimalarials for Elimination of Malaria Transmission**

*Description of Technology:* The transmission of malaria begins with injection of sporozoites into a human from the bite of a female anopheles mosquito, which initiates the malarial life cycle in humans. When a mosquito bites an infected human, the ingested male and female malaria gametocytes fuse to form a zygote that eventually becomes an oocyst. Each oocyst produces thousands of sporozoites which migrate to the mosquito salivary glands, ready to infect a new human host.

Currently, the available therapeutics for malaria can effectively eliminate the asexual stages of malarial parasites that cause clinical symptoms in patients. However, their abilities to eliminate the gametocyte (sexual stage of the parasites) as well as the liver stage parasites are limited. The subject technology involves novel compounds, which include Torin 2, that are potently gametocytocidal in *in vitro* assays and in a mouse model of malaria, completely blocked the host-to-mosquito transmission by suppressing oocytes formation in mosquitoes.

*Potential Commercial Applications:* Novel therapeutics for elimination of malaria transmission and treatment of drug resistant malaria patients.

#### *Competitive Advantages:*

- These novel compounds are effective against gametocytes, the sexual stage of malarial parasites, whereas currently available antimalarials have limited effectiveness against this form of the parasite.

- The compounds provide an alternative treatment against malaria for patients with glucose-6-phosphate dehydrogenase deficiency.

- These compounds are active against drug resistant strains of malaria.

#### *Development Stage:*

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

*Inventors:* Wei Sun (NCATS), Wei Zheng (NCATS), Seameen J. Dehdashti

(NCATS), Noel T. Southhall (NCATS), Takeshi Tanaka (NIAID), Wenwei Huang (NCATS), John C. McKew (NCATS).

*Publication:* Sun W, et al. Chemical signatures and new drug targets for gametocytocidal drug development. *Sci Rep.* 2014 Jan 17;4:3743. [PMID 24434750].

*Intellectual Property:* HHS Reference No. E–751–2013/0—U.S. Provisional Patent Application No. 61/904,884 filed 15 Nov 2013.

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

### **Compositions and Methods for Improved Lyme Disease Diagnosis**

*Description of Technology:* This CDC-developed technology entails novel compositions and methods related to the diagnosis of Lyme disease. Lyme disease, caused by the *Borrelia burgdorferi* bacterium, is the most common tick-borne infectious disease in the US and Europe. Diagnosis of Lyme disease is particularly challenging as symptoms often appear long after exposure. At present, the only FDA-approved diagnostic for Lyme disease involves patient blood tests for particular antibodies; these include an ELISA to measure patient antibody levels and a Western blot assay to detect antibodies specific to *B. burgdorferi*. One problem with the current diagnostic approach is that patient antibodies for the bacterium are not detectable until two to five weeks following the initial tick bite, and there is no way to differentiate between antibodies generated by a current infection or by a prior exposure.

This technology hinges on a unique approach that would detect whether a patient has a presently active *B. burgdorferi* infection. A fully developed assay based on these innovations would exploit the detection of the *B. burgdorferi* BbHtrA protease and/or its unique cleavage products to carry out a timely diagnosis of infection. While other direct detection methods, such as culturing, PCR and antigen capture, are often used in research laboratory settings, they have not demonstrated consistent efficacy as clinical diagnostic tools in the first few weeks following tick bite exposure. Further, despite the lack of a rapid and efficient readout for the aforementioned antibody-based Lyme disease diagnostics, there are currently no FDA-approved comparable alternatives. This technology provides a unique opportunity for rapid and accurate identification of *B. burgdorferi* infection, as well as distinguishing current bacterium exposure from prior

exposure, thereby providing critical information to better inform treatment strategy and improve patient outcomes.

#### *Potential Commercial Applications:*

- Lyme disease/*B. burgdorferi* diagnostics.
- Zoonotic/tick-borne disease surveillance.
- Informing clinician strategies and improving patient outcomes.
- Reducing diagnosis time for patients concerned about tick bites.

#### *Competitive Advantages:*

- Present Lyme disease diagnostics cannot distinguish between current bacterium infections and prior exposures; this technology will provide such distinctions.
- Predominant antibody-based diagnostics currently available require weeks before efficacy and may require re-testing at later dates to avoid false negatives; this technology directly addresses this problem.

- Other alternative direct detection methods (e.g., PCR, culturing) have shown limited efficacy as clinical diagnostics.

*Development Stage:* *In vitro* data available.

*Inventors:* Barbara Johnson and Theresa Russell (CDC).

#### *Publications:*

1. Stricker RB, et al. *Borrelia burgdorferi* aggrecanase activity: more evidence for persistent infection in Lyme disease. *Front Cell Infect Microbiol.* 2013 Aug 14;3:40. [PMID 23967405]
2. Russell TM, et al. Lyme disease spirochaetes possess an aggrecan-binding protease with aggrecanase activity. *Mol Microbiol.* 2013 Oct;90(2):228–40. [PMID 23710801]
3. Russell TM, et al. *Borrelia burgdorferi* BbHtrA degrades host ECM proteins and stimulates release of inflammatory cytokines *in vitro*. *Mol Microbiol.* 2013 Oct;90(2):241–51. [PMID 23980719]

*Intellectual Property:* HHS Reference No. E–204–2013/0 –

- U.S. Application No. 61/588,820 filed 20 Jan 2012.
- PCT Application No. PCT/US2013/022379 filed 21 Jan 2013.

*Related Technology:* HHS Reference No. E–573–2013/0.

*Licensing Contact:* Whitney Blair, J.D., M.P.H.; 301–435–4937; [whitney.blair@nih.gov](mailto:whitney.blair@nih.gov).

### **Zirconium-89 PET Imaging Agent for Cancer**

*Description of Technology:* The technology is tetrahydroxamate chelation technology that provides a stable Zr-89 chelated immuno-PET imaging agent for cancer that reduces the amounts of Zr-89 that is released

from the current state of the art chemistry and agent, desferrioxamine B (DFB), that is currently in clinical use. The tetrahydroxamates in either a linear or macrocyclic form exhibit greater stability as chelating agents for Zr-89 as compared to the currently in use siderophore DFB, a trihydroxamate. In imaging agents currently in clinical development, Zr-89 leaks from the DFB chelate which results in radioisotope accumulation in the bone 2–3 days after injection that increases over time. Upon in vitro examination, the tetrahydroxamate chelated Zr-89 remained kinetically inert at 7 or more days while that formed from DFB demonstrated instability.

**Potential Commercial Applications:**

- PET imaging.
- Cancer imaging.
- Immuno-PET imaging.

**Competitive Advantages:**

- High stability.
- Low toxicity.

**Development Status:**

- Prototype.
- In vitro data available.

**Inventors:** Francois Guerard (NCI), Yong Sok Lee (CIT), Martin Brechbiel (NCI).

**Publications:**

1. Zhou Y, et al. Mapping biological behaviors by application of longer-lived positron emitting radionuclides. 2013 Jul;65(8):1098–111. [PMID 23123291]
2. Deri MA, et al. PET imaging with 89Zr: from radiochemistry to the clinic. Nucl Med Biol. 2013 Jan;40(1):3–14. [PMID 22998840]
3. Vosjan MJ, et al. Conjugation and radiolabeling of monoclonal antibodies with zirconium-89 for PET imaging using the bifunctional chelate p-isothiocyanatobenzyl-desferrioxamine. Nat Protoc. 2010 Apr;5(4):739–43. [PMID 20360768]
4. Nayak TK, et al. PET and MRI of metastatic peritoneal and pulmonary colorectal cancer in mice with human epidermal growth factor receptor 1-targeted 89Zr-labeled panitumumab. J Nucl Med. 2012 Jan;53(1):113–20. [PMID 22213822]
5. Evans MJ, et al. Imaging tumor burden in the brain with 89Zr-transferrin. J Nucl Med. 2013 Jan;54(1):90–5. [PMID 23236019]
6. Guerard F, et al. Investigation of Zr(IV) and 89Zr(IV) complexation with hydroxamates: progress towards designing a better chelator than desferrioxamine B for immuno-PET imaging. Chem Commun (Camb). 2013 Feb 1;49(10):1002–4. [PMID 23250287]

7. Guerard F, et al. Rational Design, Synthesis and Evaluation of Tetrahydroxamic Acid Chelators for Stable Complexation of Zr(IV). Chem Eur J. (in press)

**Intellectual Property:** HHS Reference No. E–111–2013/0 –

- U.S. Provisional Patent Application 61/779,016 filed 13 Mar 2013.
- PCT Application PCT/US2014/24048 filed 12 Mar 2014.

**Related Technologies:**

- HHS Reference No. E–194–2007/0.
- HHS Reference No. E–226–2006/0.
- HHS Reference No. E–067–1990/0.

**Licensing Contact:** Michael A. Shmilovich; 301–435–5019; [shmilovm@mail.nih.gov](mailto:shmilovm@mail.nih.gov).

**Collaborative Research Opportunity:**

The Radioimmune & Inorganic Chemistry Section, ROB, CCR, NCI, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize tetrahydroxamate chelation technology for Zirconium-89 PET Imaging. For collaboration opportunities, please contact John D. Hewes, Ph.D. at [hewesj@mail.nih.gov](mailto:hewesj@mail.nih.gov).

**Ex-vivo Production of Regulatory B-Cells (Breg) for Use in Auto-Immune Indications**

**Description of Technology:** Regulatory B-cells (Breg) play an important role in reducing autoimmunity and reduced levels of these cells are implicated in etiology of several auto-inflammatory diseases. Despite their impact in many diseases, their physiological inducers are unknown. Given that Bregs are a very rare B-cell, identifying factors that promote their development would allow in vivo modulation of Breg levels and ex-vivo production of large amounts of antigen-specific Bregs to use in immunotherapy for auto-inflammatory diseases.

The invention herein, is a method of ex-vivo production of Breg. The method of production involves treating isolated primary B cells or B cell lines with IL–35 to induce their conversion into IL–10-producing Breg. Using this method, B-regulatory cells can be produced in large quantity and used in a Breg-based therapy against autoimmune diseases including but not limited to uveitis and sarcoidosis.

**Potential Commercial Applications:**

- In vivo modulation of Breg levels.
- Supplement the low population of Breg in a patient suffering from an autoimmune disease where it is known that B-regulatory cell populations are severely reduced (i.e. uveitis)
- Use in immunotherapy for the treatment of other autoimmune diseases

such as multiple sclerosis, sarcoidosis, colitis, and arthritis.

**Competitive Advantages:**

- There is no known biological or chemical agent that can induce Bregs ex-vivo.
- This method produces large quantities of Bregs and can therefore aid in Breg-based therapy.
- Pre-clinical mouse model data available that uses the Bregs to treat experimental autoimmune uveitis (EAU).

**Development Stage:** In vivo data available (animal).

**Inventors:** Charles E. Egwuagu, Ren-Xi, Wang, Cheng-Rong Yu (all of NEI).

**Relevant Publications:**

1. Shen P, et al. IL–35-producing B cells are critical regulators of immunity during autoimmune and infectious diseases. Nature. 2014 Mar 20;507(7492):366–70. [PMID 24572363]
2. Ding Q, et al. Regulatory B cells are identified by expression of TIM–1 and can be induced through TIM–1 ligation to promote tolerance in mice. J Clin Invest. 2011 Sep;121(9):3645–56. [PMID 21821911]
3. Carter NA, et al. Mice lacking endogenous IL–10-producing regulatory B cells develop exacerbated disease and present with an increased frequency of Th1/Th17 but a decrease in regulatory T cells. J Immunol. 2011 May 15;186(10):5569–79. [PMID 21464089]
4. Collison LW, et al. IL–35-mediated induction of a potent regulatory T cell population. Nat Immunol. 2010 Dec;11(12):1093–101. [PMID 20953201]
5. Kochetkova I, et al. IL–35 stimulation of CD39+ regulatory T cells confers protection against collagen II-induced arthritis via the production of IL–10. J Immunol. 2010 Jun 15;184(12):7144–53. [PMID 20483737]

**Intellectual Property:** HHS Reference No. E–036–2012/0—

- U.S. Patent Application No. 61/637,915 filed 25 Apr 2012.
- PCT Application No. PCT/US2013/036175 filed 11 Apr 2013, which published as WO 2013/162905 on 31 Oct 2013.

**Licensing Contact:** Yolanda Mock-Hawkins, Ph.D., M.B.A.; 301–435–5170; [Yolanda.Hawkins@nih.gov](mailto:Yolanda.Hawkins@nih.gov).

**Collaborative Research Opportunity:** The National Eye Institute, Molecular Immunology Section, is seeking statements of capability or interest from parties interested in collaborative

research to further develop, evaluate or commercialize Ex-vivo Production of Regulatory B-Cells (Breg). For collaboration opportunities, please contact Alan Hubbs, Ph.D. at [hubbsa@mail.nih.gov](mailto:hubbsa@mail.nih.gov).

### SCGB3A2 for Treatment of Cancer

**Description of Technology:** A novel method of treating lung cancer using uteroglobin-related protein 1 (UGRP1), also known as secretoglobin family 3A member 2 (SCGB3A2) is disclosed. SCGB3A2 is a member of the uteroglobin/Clara cell secretory protein or Secretoglobin gene superfamily of secretory proteins that is predominantly expressed in the epithelial cells of the trachea, bronchus, and bronchioles, and is known for its anti-inflammatory activity. The inventors have previously discovered the growth factor and anti-fibrotic activities of SCGB3A2 and proposed the use of SCGB3A2 as a therapeutic to treat neonatal respiratory distress and as an agent to promote lung development, and to inhibit or reduce pulmonary fibrosis caused by an anti-cancer agent. Recently, the inventors have made a surprising discovery that the secretory protein SCGB3A2 also has anti-cancer activity, in addition to its known growth factor, anti-inflammatory, and anti-fibrotic activities. The inventors have used SCGB3A2-induced inhibition of metastasis in the iv- and sc-injected LLC cells lung metastasis model, Scgb3a2-null mice injected with LLC cells with and without SCGB3A2, and Scgb3a2-lung transgenic mice subjected to tobacco carcinogen induced mouse carcinogenesis bioassay to confirm their discovery that SCGB3A2 has anti-cancer activity.

**Potential Commercial Applications:** Therapeutics for treating cancers.

#### Competitive Advantages:

- This technology provides, for the first time, a new mode of treating lung cancer using SCGB3A2.
- Because SCGB3A2 is predominantly expressed in lung airways, low toxicity is anticipated by the use of SCGB3A2 as a therapeutic.
- Unique mode of action (affects both metastasis and growth (proliferation) of cancer cells) makes SCGB3A2 more effective as a therapeutic.

#### Development Stage:

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

**Inventors:** Kimura Shioko, Cai Yan, and Murata Miyuki (NCI).

**Publication:** Cai Y, et al. Preclinical evaluation of human secretoglobin 3A2 in mouse models of lung development and fibrosis. *Am J Physiol Lung Cell*

*Mol Physiol.* 2014 Jan 1;306(1):L10–22. [PMID 24213919].

**Intellectual Property:** HHS Reference No. E-286–2006/3—US Provisional Patent Application No. 61/862,429 filed 05 Aug 2013.

**Related Technologies:** HHS Reference Nos. E-286–2006/0, 1, 2—

- US Patent No. 8,133,859 issued 13 Mar 2012.
- US Patent No. 8,501,688 issued 06 Aug 2013.
- US Patent Application No. 13/959,628 filed 05 Aug 2013.

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

**Collaborative Research Opportunity:** The National Cancer Institute, Laboratory of Metabolism, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize SCGB3A2 as an anti-cancer reagent, which mainly works through the JNK pathway. For collaboration opportunities, please contact John D. Hewes, Ph.D. at [hewesj@mail.nih.gov](mailto:hewesj@mail.nih.gov).

Dated: April 3, 2014.

**Richard U. Rodriguez,**

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

[FR Doc. 2014–07871 Filed 4–8–14; 8:45 am]

**BILLING CODE 4140–01–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

#### National Institute of General Medical Sciences; 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 Institute of General Medical Sciences Special Emphasis Panel; Review of Grant Applications.

**Date:** May 2, 2014.

**Time:** 1:00 p.m. to 5:00 p.m.

**Agenda:** To review and evaluate grant applications.

**Place:** National Institutes of Health, Natcher Building, 45 Center Drive, Room 3An.18B, Bethesda, MD 20892, (Telephone Conference Call).

**Contact Person:** Margaret J. Weidman, Ph.D., Scientific Review Officer, Office of Scientific Review, National Institute of General Medical Sciences, National Institutes of Health, 45 Center Drive, Room 3An.18B, Bethesda, MD 20892, 301–594–2773, [weidmanma@nigms.nih.gov](mailto:weidmanma@nigms.nih.gov).

(Catalogue of Federal Domestic Assistance Program Nos. 93.375, Minority Biomedical Research Support; 93.821, Cell Biology and Biophysics Research; 93.859, Pharmacology, Physiology, and Biological Chemistry Research; 93.862, Genetics and Developmental Biology Research; 93.88, Minority Access to Research Careers; 93.96, Special Minority Initiatives, National Institutes of Health, HHS)

Dated: April 3, 2014.

**David Clary,**

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

[FR Doc. 2014–07863 Filed 4–8–14; 8:45 am]

**BILLING CODE 4140–01–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

#### National Institute of Diabetes and Digestive and Kidney Diseases; 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 Institute of Diabetes and Digestive and Kidney Diseases Special Emphasis Panel; Pragmatic Research and Natural Experiments.

**Date:** June 11, 2014.

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

**Agenda:** To review and evaluate grant applications.

**Place:** National Institutes of Health, Two Democracy Plaza, 6707 Democracy Boulevard, Bethesda, MD 20892, (Telephone Conference Call).

**Contact Person:** Michele L. Barnard, Ph.D., Scientific Review Officer, Review Branch, DEA, NIDDK, National Institutes of Health, Room 753, 6707 Democracy Boulevard, Bethesda, MD 20892–2542, (301) 594–8898, [barnardm@extra.niddk.nih.gov](mailto:barnardm@extra.niddk.nih.gov).