agenda items are subject to change as priorities dictate.

Time will be provided for public comments. Each public comment is limited to five minutes. Registered attendees for this meeting are encouraged to submit comments prior to the meeting. Comments are to be submitted in writing no later than 5:00 p.m. ET on August 19, 2013.

For Further Information Contact: Individuals who are submitting public comments or who have questions regarding the meeting should contact Keisher Highsmith, Dr.P.H., Director of Special Initiatives and Program Planning and Evaluation, Health Resources and Services Administration, Maternal and Child Health Bureau, telephone: (301) 443–0543; or email: *khighsmith@hrsa.gov*.

Dated: June 14, 2013.

Bahar Niakan,

Director, Division of Policy and Information Coordination.

[FR Doc. 2013–14837 Filed 6–20–13; 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, 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.

GPR116 Knockout and Conditional Knockout Mice

Description of Technology: Pulmonary surfactant plays a critical role in preventing alveolar collapse by decreasing surface tension at the alveolar air-liquid interface. Surfactant deficiency contributes to the pathogenesis of acute lung injury (ALI) and acute respiratory distress syndrome (ARDS), common disorders that can afflict patients of all ages and carry a mortality rate greater than 25%. Excess surfactant leads to pulmonary alveolar proteinosis. The NCI investigators created a G-protein coupled receptor GPR116 mutant mouse model and showed that GPR116 plays a previously unexpected, essential role in maintaining normal surfactant levels in the lung.

The mouse model could aid in the development of drug screens to identify agents that can modulate surfactant levels. Alveolar type II cells have also been isolated from the GPR116 wildtype and knockout mice that could be directly used in such assays. The identification of surfactant modulating agents could be important to a number of lung surfactant disorders.

Potential Commercial Applications: Research materials to study lung surfactant homeostasis and disorders.

Competitive Advantages: Not available elsewhere.

Development Stage:

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

Inventors: Bradley Dean St. Croix and Mi Young Yang (NCI).

Publication: Yang MY, et al. Essential Regulation of Lung Surfactant Homeostasis by the Orphan G Protein-Coupled Receptor GPR116. Cell Rep. 2013 May 30;3(5):1457–64. [PMID 23684610]

Intellectual Property: HHS Reference No. E–269–2012/0—Research Tool. Patent prosecution is not being pursued for this technology.

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

Collaborative Research Opportunity: The Center for Cancer Research Mouse Cancer Genetics Program is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize GPR116 Knockout and Conditional Knockout Mice. For collaboration opportunities, please contact John Hewes, Ph.D. at *hewesj@mail.nih.gov*.

Engineered Anthrax Toxin Variants That Target Cancer

Description of Technology: This technology describes the use of novel mutated anthrax protective antigen (PA) protein variants to target tumor cells and tumor vasculature. NIH scientists have engineered two PA variants that selectively complement one another and combine to form active octamers that target tumor cells. This controlled oligomeric activation of the PA proteins makes the likelihood of toxicity to non-tumor cells very low since non-tumor tissue does not express certain cell-surface proteases required to activate the PA variants. Using proteases that are highly expressed in tumor cells, e.g., matrix metalloproteases (MMP) and urokinase plasminogen activator (uPA), the scientists have shown significant tumor growth suppression with the oligomer in a mouse model. Furthermore, other tumor-specific proteases could also be used to control formation of the targeted octameric anthrax toxin structures. Moreover, the structures can be expanded to include several PA variants. In summary, this technology provides a unique, expandable platform that reduces toxicity to normal tissues compared to other systems and can be used to treat cancers more effectively.

Potential Commercial Applications: Therapeutic treatment for solid tumors, cancers, and infectious diseases.

Competitive Advantages:

- Specificity in targeting tumors while eliminating side effects associated with non-specific targeting of normal cells.
- Method can be expanded to include different proteases and up to eight PA variants.

Development Stage:

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

Inventors: Clinton E. Leysath, Stephen H. Leppla, Damilola D. Phillips (NIAID).

Publication: Phillips DD, et al. Engineering anthrax toxin variants that exclusively form octamers and their application to targeting tumors. J Biol Chem. 2013 Mar 29;288(13):9058–65. [PMID 23393143]

Intellectual Property: HHS Reference No. E–246–2012/0—U.S. Provisional Application No. 61/692,143 filed 22 Aug 2012.

Related Technologies:

- HHS Reference No. E–293–1999—Mutated Anthrax Toxin Protective Antigen Proteins That Specifically Target Cells Containing High Amounts of Cell-Surface Metalloproteinases or Plasminogen Activator Receptors (Leppla/NIAID).

• HHS Reference No. E–070–2007—Human Cancer Therapy Using Engineered Metalloproteinase-Activated Anthrax Lethal Toxin That Target Tumor Vasculature (Leppla/NIAID).
• HHS Reference No. E–059–2004—Multimeric Protein Toxins to Target Cells Having Multiple Identifying Characteristics (Leppla/NIAID).

Licensing Contact: Whitney Hastings; 301–451–7337; *hastingw@mail.nih.gov*.

Intra-Bone Drug Delivery Device and Method

Description of Technology: The invention pertains to devices for directly infusing cellular therapeutics into patient bone. The device monitors intra-bone pressure using pressure sensors disposed at its proximal end and adjusts infusion pressures during infusion such that intra-bone pressure does not exceed levels of systemic blood pressure. Such devices, apparatus and methods are particularly suitable for use in performing bone marrow transplants, particularly transplants that utilize cord blood as a stem cell source.

Potential Commercial Applications: Drug delivery to bones.

Competitive Advantages:
• Therapeutic uptake efficiency.
• Drug delivery efficiency.
• Target specificity.

Development Stage:
• Prototype.
• In vitro data available.

Inventors: Robert Hoyt (NHLBI), Jeremy Pantin (NHLBI), Timothy Hunt (NHLBI), Randall Clevenger (NHLBI), Omer Aras (NIHCC), Richard Childs (NHLBI), Peter Choyke (NCI).

Publication: Pantin JM, et al. ''Optimization of an Intra-Bone Hematopoietic Stem Cell Delivery Technique in a Swine Model.'' Poster abstract presented at the 54th ASH Annual Meeting and Exposition, Atlanta, Georgia, December 8–11, 2012. [*https://ash.confex.com/ash/2012/webprogram/Paper53150.html*]

Intellectual Property: HHS Reference No. E–165–2012/0—U.S. Provisional Patent Application No. 61/771,463 filed 01 Mar 2013.

Related Technology: HHS Reference No. E–196–1998/2—U.S. Patent No. 8,409,166 issued 02 Apr 2013.

Licensing Contact: Michael Shmilovich; 301–435–5019; *shmilovm@mail.nih.gov*.

Collaborative Research Opportunity: The National Heart, Lung, and Blood Institute is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize Intra-bone Drug Delivery Device and Method. For collaboration opportunities, please contact Denise Crooks at *crooksd@nhlbi.nih.gov*.

Method of Inducing Pluripotent or Multipotent Stem Cells by Blocking CD47 Receptor Signaling

Description of Technology: NIH researchers have discovered that blockade of the signaling activity of a single cell-surface receptor, CD47, without transfection or introduction of potentially transforming viral vectors, results in high frequency, spontaneous generation of self-renewing cells with a high proliferative capacity. Induced pluripotent stem cells (iPS cells) are currently produced by transforming cells with viral or other constitutive expression vectors encoding four stem cell transcription factors (c-Myc, Sox2, Klf4, and Oct4), but this method presents challenges such as over-expression of c-Myc, which can result in malignant transformation. The present invention relates to a method of using CD47-modulating compounds to induce multipotent stem cells without the concomitant risk of malignant transformation and without requiring the use of feeder cells. The cellular phenotypes are associated with increased expression of the hallmark stem cell-inducing transcription factors, c-Myc, Sox2, Klf4, and Oct4. The current invention builds on the NIH's previous discoveries of antibodies, antisense morpholino oligonucleotides, peptide compounds and other small molecules that modulate CD47.

Potential Commercial Applications: Regenerative medicine and stem cell therapy.

Competitive Advantages:
• Does not require use of viral vectors.
• Eliminates risk of malignant transformation for clinical applications.
• Eliminates need for feeder cells.
• Allows generation and maintenance of a ready supply of iPS cells using a single defined agent.
• Avoids loss of differentiated phenotype associated with telomerase or T antigen transfection.

Development Stage:
• In vitro data available.
• In vivo data available (animal).

Inventors: David D. Roberts, Sukhbir Kaur, Jeff S. Isenberg (NCI)

Publications:
1. Kaur S, et al. Thrombospondin-1 signaling through CD47 inhibits self-renewal by regulating c-Myc and other stem cell transcription factors. Sci Rep. 2013;3:1673. [PMID 23591719]
2. NCI News Note: A drug target that stimulates development of healthy stem cells. 2013 Apr 17. [*http://www.cancer.gov/newscenter/newsfromnci/2013/cd47stemcell*]

Intellectual Property:
• HHS Reference No. E–086–2012/0—U.S. Application No. 61/621,994 filed 09 Apr 2012.
• HHS Reference No. E–086–2012/1—U.S. Application No. 61/735,701 filed 11 Dec 2012.
• HHS Reference No. E–086–2012/2—International Application PCT/US2013/035838 filed 09 Apr 2013.

Related Technologies: HHS Reference No. E–227–2006/5—
• U.S. Application No. 12/444,364 filed 3 Apr 09.
• CA Application No. 2,665,287 filed 5 Oct 07.
• EP Application No. 07868382.8 filed 27 Mar 09.
• U.S. Application No. 13/546,941 filed 11 Jul 12.

Licensing Contact: Charlene Sydnor, Ph.D.; 301–435–4689; *sydnorc@mail.nih.gov*.

Collaborative Research Opportunity: The National Cancer Institute, Center for Cancer Research, Laboratory of Pathology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate or commercialize CD47 modulators for regenerative medicine and stem cell therapy applications. For collaboration opportunities, please contact John Hewes, Ph.D. at *hewesj@mail.nih.gov*.

Human Monoclonal Antibodies to Glypican-3 Protein and Heparan Sulfate for Treatment of Cancer

Description of Technology: Hepatocellular carcinoma (HCC) is the most common form of liver cancer, and is among the more deadly cancers in the world due to its late detection and poor prognosis. No effective treatment is available for liver cancer therapy.

Glypican-3 (GPC3) is a cell surface protein that is preferentially expressed on HCC cells, making it an attractive potential target for developing a therapeutic. This invention concerns human monoclonal antibodies against GPC3 and their use for the treatment of GPC3-expressing cancers such as HCC.

Specifically, the inventors have generated two distinct human monoclonal antibodies to GPC3. The first antibody (HN3) binds to a conformational epitope on the cell surface domain of GPC3. The second antibody (HS20) binds specifically to heparan sulfate chains on GPC3. These antibodies can inhibit the growth of HCC cells, thereby decreasing the ability of tumors to grow and metastasize. Furthermore, by using the antibodies to target a toxic moiety to only those cells that express GPC3, cancer cells can be eliminated while allowing healthy,

essential cells to remain unharmed. Thus, monoclonal antibodies to GPC3 (and corresponding immunotoxins) represent a novel therapeutic candidate for treatment of HCC, as well as other cancers associated with the differential expression of GPC3.

Potential Commercial Applications:

- Therapeutic antibodies against cancers that overexpress GPC3.
- Therapeutic immunotoxins or antibody-drug conjugates for killing cancer cells that overexpress GPC3.
- Diagnostics for detecting cancers associated with GPC3 overexpression.
- Specific cancers include hepatocellular cancer (HCC), melanoma, ovarian cancer, thyroid cancer, lung squamous cell carcinoma, Wilms' tumor, neuroblastoma, hepatoblastoma, and testicular germ-cell tumors.

Competitive Advantages:

- Monoclonal antibodies create a level of specificity that can reduce deleterious side-effects.
- Multiple treatment strategies available including the killing of cancer cells with a toxic agent or by inhibiting cell signaling.
- Non-invasive and potentially non-liver toxic alternative to current HCC treatment strategies.

Development Stage:

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

Inventors: Mitchell Ho (NCI) et al.

Publications:

1. Feng M, et al. Therapeutically targeting glypican-3 via a conformation-specific single-domain antibody in hepatocellular carcinoma. Proc Natl Acad Sci U S A. 2013 Mar 19;110(12):E1083–91. [PMID 23471984]

2. Feng M, et al. Recombinant soluble glypican 3 protein inhibits the growth of hepatocellular carcinoma in vitro. Int J Cancer 2011 May1;128(9):2246–7. [PMID: 20617511]

3. Zitterman SI, et al. Soluble glypican 3 inhibits the growth of hepatocellular carcinoma in vitro and in vivo. Int J Cancer 2010 Mar 15;126(6):1291–1301. [PMID: 19816934]

Intellectual Property: HHS Reference No. E–130–2011/0—U.S. Provisional Application No. 61/477,020 filed 19 Apr 2011; PCT Application No. PCT/US2012/034186 filed 19 Apr 2012.

Related Technology: HHS Reference No. E–136–2012/0—U.S. Provisional Application No. 61/654,232 filed 01 Jun 2012.

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

Collaborative Research Opportunity: The National Cancer Institute, Center for Cancer Research, Laboratory of Molecular Biology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize novel antibody or antibody-drug conjugate therapies for the treatment of liver cancer. For collaboration opportunities, please contact John Hewes, Ph.D. at *hewesj@mail.nih.gov.*

Dated: June 14, 2013.

Richard U. Rodriguez,

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

[FR Doc. 2013–14821 Filed 6–20–13; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Eye Institute; 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 meetings.

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

Name of Committee: National Eye Institute Special Emphasis Panel; NEI Epidemiology and Genetics.

Date: July 10, 2013.

Time: 11:30 a.m. to 2:30 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, 5635 Fishers Lane, Bethesda, MD 20892 (Telephone Conference Call).

Contact Person: Anne E. Schaffner, Ph.D., Chief, Scientific Review Officer, Division of Extramural Research, National Eye Institute, National Institutes of Health, 5635 Fishers Lane, Suite 1300, MSC 9300, 301–451–2020, *aes@nei.nih.gov.*

Name of Committee: National Eye Institute Special Emphasis Panel; NEI Institutional Training Grants and Conference Grants.

Date: July 30, 2013.

Time: 8:00 a.m. to 5:00 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, Terrace Level Conference Center, 5635 Fishers Lane, Bethesda, MD 20892.

Contact Person: Brian Hoshaw, Ph.D., Scientific Review Branch, Division of Extramural Research, National Eye Institute, National Institutes of Health, 5635 Fishers Lane, Suite 1300, MSC 9300, 301–451–2020, *hoshawb@mail.nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.867, Vision Research, National Institutes of Health, HHS)

Dated: June 17, 2013.

Melanie J. Gray,

Program Analyst, Office of Federal Advisory Committee Policy.

[FR Doc. 2013–14816 Filed 6–20–13; 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; RFA Panel: Molecular and Cellular Substrates of Complex Brain Disorders.

Date: July 19, 2013.

Time: 8:00 a.m. to 5:30 p.m.

Agenda: To review and evaluate grant applications.

Place: Embassy Suites at the Chevy Chase Pavilion, 4300 Military Road NW., Washington, DC 20015.

Contact Person: Deborah L Lewis, Ph.D., Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 4183, MSC 7850, Bethesda, MD 20892, 301–408–9129, *lewisdeb@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: June 14, 2013.

Anna Snouffer,

Deputy Director, Office of Federal Advisory Committee Policy.

[FR Doc. 2013–14815 Filed 6–20–13; 8:45 am]

BILLING CODE 4140–01–P