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 Child Health and Human Development Special Emphasis Panel.

Date: May 7, 2015.

Time: 8:00 a.m. to 6:00 p.m.

Agenda: To review and evaluate grant applications.

Place: Residence Inn Bethesda, 7335 Wisconsin Avenue, Bethesda, MD 20814.

Contact Person: Peter Zelazowski, Ph.D., Scientific Review Officer, Scientific Review Branch, Eunice Kennedy Shriver National Institute of Child Health and Human Development, NIH 6100 Executive Boulevard, Room 5B01, Bethesda, MD 20892–9304, (301) 435–6902, *peter.zelazowski@nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.864, Population Research; 93.865, Research for Mothers and Children; 93.929, Center for Medical Rehabilitation Research; 93.209, Contraception and Infertility Loan Repayment Program, National Institutes of Health, HHS)

Dated: April 7, 2015.

David Clary,

Program Analyst, Office of Federal Advisory Committee Policy.

[FR Doc. 2015–08294 Filed 4–9–15; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Human Genome Research 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 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: Date: Center for Inherited Disease Research Access Committee.

Date: April 23, 2015.

Time: 11:30 a.m. to 3:00 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: Camilla E. Day, Ph.D., Scientific Review Officer, CIDR, National Human Genome Research Institute, National Institutes of Health, 5635 Fishers Lane, Suite 4075, Bethesda, MD 20892, 301–402–8837, *camilla.day@nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.172, Human Genome Research, National Institutes of Health, HHS)

Dated: April 6, 2015.

David Clary,

Program Analyst,

Office of Federal Advisory Committee Policy.

[FR Doc. 2015–08213 Filed 4–9–15; 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.

SUPPLEMENTARY INFORMATION: Technology descriptions follow.

Novel Immunotherapy for Cancer Treatment: Chimeric Antigen Receptors Targeting CD70 Antigen

Description of Technology: Scientists at the National Institutes of Health have developed anti-CD70 chimeric antigen receptors (CARs) to treat cancers. CD70 is an antigen that is expressed on a variety of human cancers such as renal cell carcinoma, glioblastoma, non-Hodgkin's lymphoma, and chronic lymphocytic leukemia. The anti-CD70 CARs are hybrid proteins consisting of a receptor portion that recognizes CD70 antigen, and intracellular T cell signaling domains selected to optimally activate the CAR expressing T cells. Genetically engineered T cells that express this CARs will bind to CD70 on the cancer cells and will be activated to induce an immune response that promotes robust tumor cell elimination when infused into cancer patients. This technology can rapidly generate a vigorous T-cell response from the patient's own blood, targeting CD70 expressing cancer cells, and potentially induce tumor rejection.

Potential Commercial Applications:

- Immunotherapeutics to treat cancers that overexpress CD70, such as renal cell carcinoma, glioblastoma, non-Hodgkin's lymphoma, and chronic lymphocytic leukemia.
- A personalized cancer treatment strategy for patients whose tumor cells express CD70 whereby the patient's own T cells are isolated, engineered to express the anti-CD70 CARs, and re-infused into the same patient to attack the tumor(s).

Competitive Advantages:

- CD70-specific CARs expressed on T cells will increase the likelihood of successful targeted therapy.
- CAR–T cells target only CD70 expressing cells and thus may generate fewer side effects than other cancer treatment approaches.
- With the advent of Provenge(R), and Yervoy(R), immunotherapy is now more widely accepted as a viable cancer treatment option.
- T-cell transfer can provide much larger numbers of anti-tumor immune cells compared to other approaches such as vaccines.

Development Stage:

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

Inventors: Qiong J. Wang, Zhiya Yu, James C. Yang (all of NCI).

Publication: Wang QJ, et al. Distinctive features of the differentiated phenotype and infiltration of tumor-reactive lymphocytes in clear cell renal cell carcinoma. Cancer Res. 2012 Dec 1; 72(23):6119–29. [PMID 23071066]

Intellectual Property: HHS Reference No. E–021–2015/0—U.S. Patent Application No. 62/088,882 filed 08 Dec 2014.

Licensing Contact: Whitney A. Hastings, Ph.D.; 301–451–7337; *hastingw@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 chimeric antigen receptors targeting CD70 for cancer treatment. For collaboration opportunities, please contact Steven A. Rosenberg, M.D., Ph.D. at *sar@nih.gov.*

Novel Cancer Immunotherapy: HLA–A11 Restricted T Cell Receptor That Recognizes G12D Variant of Mutated KRAS

Description of Technology: Scientists at the National Institutes of Health have developed T cell receptor (TCR) derived from mouse T cells that recognize mutated Kirsten rat sarcoma viral oncogene homolog (KRAS), in particular the G12D variant. Mutated KRAS, which plays an essential driver role in oncogenesis, is expressed by a variety of human cancers, such as pancreatic, colorectal, lung, endometrial, ovarian, and prostate cancers; but not by normal, noncancerous cells. KRAS is mutated in nearly a third of the most lethal human cancers and could serve as a cancer-specific therapeutic target. Most common mutations occurred at codon 12, as glycine can be substituted with aspartic acid (G12D), valine (G12V), cysteine (G12C), and arginine (G12R), and among these codon 12 substitutions, G12D is the most frequent variant. The TCR is a protein that specifically recognizes the most frequent mutated KRAS G12D variant in the context of major histocompatibility complex (MHC) class I molecule HLA–A11 and activates T-cells. In HLA–A11+ patients, such genetically engineered T cells with TCRs against mutated KRAS are expected to target and kill cancer cells with this mutation while sparing normal tissues after infusion into patients.

Potential Commercial Applications:

• Immunotherapeutics to treat a variety of human cancers that harbor KRAS mutations, in particular, G12D mutation, such as pancreatic, -colorectal, lung, endometrial, ovarian, and prostate cancers.

• T cells expressing mutated KRAS G12D specific TCR may successfully treat or prevent the recurrence of mutated KRAS-positive cancers that do not respond to other types of treatment such as surgery, chemotherapy, and radiation.

Competitive Advantages:

• Genetically engineered T cells with TCRs for HLA–A11-restricted mutated KRAS will increase the likelihood of successful targeted therapy.

• The targeted therapy minimizes side effect. T cells expressing anti-mutated KRAS TCRs target tumor cells expressing mutated KRAS and spare normal tissue. This therapy may have lower tissue toxicities comparing to traditional chemotherapy and radiotherapy.

• With the advent of Provenge(R) and Yervoy(R), immunotherapy is now more widely accepted as a viable cancer treatment option.

Development Stage:

• Early-stage.
• In vitro data available.
• Ex vivo data available.

Inventors: Qiong J. Wang and James C. Yang (NCI).

Intellectual Property: HHS Reference No. E–028–2015/0—US Provisional Patent Application No. 62/084,654 filed 26 Nov 2014.

Related Technologies:

• HHS Reference No. E–106–2006/3.
• HHS Reference No. E–226–2014/0.

Licensing Contact: Whitney A. Hastings, Ph.D.; 301–451–7337; *hastingw@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 anti-mutated KRAS TCRs for cancer treatment. For collaboration opportunities, please contact Steven A. Rosenberg, M.D., Ph.D. at *sar@nih.gov.*

Live Attenuated Japanese Encephalitis Virus Vaccine

Description of Technology: Japanese encephalitis virus (JEV), a member of the genus flavivirus, is maintained in a zoonotic cycle between *Culex* mosquitoes and ardeid birds or domestic swine and is responsible for significant epidemics of viral encephalitis in Asia. Three billion people live in regions with endemic JEV transmission resulting in an estimated 60,000 annual cases, of which 20–40% are fatal and 45–70% of survivors have neurologic sequelae. The live-attenuated JEV SA14–14–2 vaccine, produced in primary hamster kidney cells, is safe and effective. Past attempts to adapt this virus to replicate in cells that are more favorable for vaccine production resulted in mutations that significantly reduced immunogenicity. The inventors have isolated 10 genetically distinct Vero cell-adapted JEV SA14–14–2 variants and a recombinant wild-type JEV clone, modified to contain the JEV SA14–14–2 polyprotein amino acid sequence, was recovered in Vero cells. Mutations were also identified that modulated virus sensitivity to type I interferon-stimulation in Vero cells. A subset of JEV SA14–14–2 variants and the recombinant clone were evaluated in vivo and exhibited levels of attenuation that varied significantly in suckling mice, but were avirulent and highly immunogenic in weanling mice and are promising candidates for the development of a second generation, recombinant vaccine.

Potential Commercial Applications:

• JEV Vaccine.
• JEV Diagnostics.

Competitive Advantages:

• Safe and efficacious vaccine.
• Extremely low production costs.
• Positive preclinical data.
• Vero cell manufacture.

Development Stage:

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

Inventors: Stephen S. Whitehead and Gregory D. Gromowski (NIAID).

Publications:

1. Gromowski G, et al. Genetic and phenotypic properties of vero cell-adapted Japanese encephalitis virus SA14–14–2 vaccine strain variants and a recombinant clone, which demonstrates attenuation and immunogenicity in mice. Am J Trop Med Hyg. 2015 Jan; 92(1)98–107. [PMID 25311701].

2. Gromowski G, et al. Genetic determinants of Japanese encephalitis virus vaccine strain SA14–14–2 that govern attenuation of virulence in mice. J Virol. 2015, in press.

Intellectual Property: HHS Reference No. E–231–2014/0—Research Material. Patent protection is not being pursued for this technology.

Licensing Contact: Peter Soukas; 301–435–4646; *ps193c@nih.gov.*

IFN Gamma for Reducing Adverse Ocular Side Effects of MEK-Inhibitor Therapy in Cancer

Description of Technology: Use of IFN-gamma for treating an adverse side effect in a cancer patient being treated by a MEK-inhibitor (MEKi) is disclosed. MAP kinase/ERK kinase (MEK), an oncogene or signal protein within the P38 mitogen activated protein kinase (MAPK) pathway, is a crucial point of convergence that integrates a variety of protein kinases through Ras. MEKis are currently being tested in monotherapies and combination therapies against a wide variety of cancers. A number of side effects are noticed with treatment of cancer with MEKis, including visual disturbances. The inventors have discovered that MEKis decreases fluid transport from the retina and/or subretinal space of the retinal pigment epithelium (RPE) resulting in the abnormal accumulation of fluid in the retina and subretinal space, which causes retinal detachment and vision loss. Their results also indicate that apical addition of MEKis alters transepithelial resistance in RPE. For the first time, the inventors showed that these effects of MEKis are almost

completely rescued by basolateral addition of IFN-gamma. These results suggest that IFN-gamma can be used to reduce adverse events (retinal edema) associated with the therapeutic use of MEKis.

Potential Commercial Applications: Treatment for or prevention of adverse side effects in cancer patients undergoing MEK inhibitor therapy.

Competitive Advantages: A simple and unique mode of reducing or eliminating ocular side effects in cancer patients undergoing treatments with MEK inhibitors.

Development Stage:

- Early-stage.
- In vitro data available.

Inventors: Sheldon S. Miller (NEI), Arvydas Maminishkis (NEI), Charlotte E. Remé (Merck KGaA).

Intellectual Property: HHS Reference No. E–248–2012/0—

- US Provisional Application No. 61/721,810 filed 02 Nov 2012.
- PCT Patent Application No. PCT/US2013/068056 filed 01 Nov 2013.

Related Technologies: HHS Reference No. E–169–2008/0—

- US Patent No. 8,697,046 issued 15 Apr 2014 (Methods of Administering Interferon Gamma to Absorb Fluid From the Subretinal Space; Li R, et al.).
- US Patent Application No. 14/252,489 filed 14 Apr 2014.

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

Lubiprostone To Treat Retinal Diseases Associated With Fluid Accumulation in Retina & Subretinal Space

Description of Technology: Use of Lubiprostone for treating age-related macular degeneration, chronic macular edema, diabetic retinopathy, retinal detachment, glaucoma, or uveitis by decreasing excess fluid accumulation in the retina and/or subretinal space (SRS) is described. The retinal pigment epithelium (RPE) is a highly pigmented, terminally differentiated monolayer of cells at the back of the eye. The RPE performs numerous processes that are critical for the maintenance of photoreceptor cell health and function. The pathological accumulation of fluid beneath the RPE is a symptom and a contributing factor in the loss of vision in a variety of ocular conditions. Previously, the inventors have shown that human RPE contains apical and basolateral membrane receptors that can be activated to increase cell cAMP or Ca followed by basolateral membrane activation of CFTR or Ca-activated chloride channels resulting in a clinically significant increase in fluid absorption across the RPE. For the first time, using human RPE *in vitro,* the inventors demonstrated that lubiprostone can increase fluid transport from the retinal to the choroidal side of the RPE by activating CLC–2 at the RPE basolateral membrane. Further, they also showed that this increase can be blocked by addition of methadone, a specific CLC–2 channel blocker. Lubiprostone added from either the apical or basolateral side of the epithelium. Methadone also increased transepithelial potential (TEP) and this increase is consistent with a lubiprostone-induced increase in basolateral membrane CLC–2 conductance and subsequent membrane depolarization. These results suggest lubiprostone can be a therapeutic in retinal disease to increase fluid absorption from retina and subretinal space.

Potential Commercial Applications: Treatment for or prevention of age-related macular degeneration, chronic macular edema, diabetic retinopathy, retinal detachment, glaucoma, or uveitis by decreasing the amount of fluid present in the subretinal space (SRS).

Competitive Advantages: A simple and novel therapeutic for retinal diseases characterized by the abnormal fluid accumulation in subretinal space.

Development Stage:

- Early-stage.
- In vitro data available.

Inventors: Sheldon S. Miller, Arvydas Maminishkis, Jeffrey Adijanto, Tina M. Banzon, and Qin Wan (all of NEI).

Intellectual Property: HHS Reference No. E–283–2012/0—

- U.S. Provisional Application No. 61/777,073 filed 12 Mar 2013.
- PCT Patent Application No. PCT/US2014/024724 filed 12 Mar 2014.

Related Technology: HHS Reference No. E–169–2008/0—

- U.S. Patent No. 8,697,046 issued 15 Apr 2014 (Methods of Administering Interferon Gamma to Absorb Fluid From the Subretinal Space; Li R, et al.).
- U.S. Patent Application No. 14/252,489 filed 14 Apr 2014.

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

Dated: March 7, 2015.

Richard U. Rodriguez,

Acting Director, Office of Technology Transfer, National Institutes of Health.

[FR Doc. 2015–08290 Filed 4–9–15; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Substance Abuse and Mental Health Services Administration

Agency Information Collection Activities: Proposed Collection; Comment Request

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

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

Proposed Project: Family Treatment Drug Court Services Evaluation (OMB No. 0930–0330)—REINSTATEMENT

In 2010, the Substance Abuse and Mental Health Services Administration (SAMHSA), Center for Substance Abuse Treatment (CSAT), provided funding to 12 existing Family Treatment Drug Courts (FTDCs) for enhancement and/or expansion of their FTDC's capabilities to provide psycho-social, emotional and mental health services to children (0–17 years) and their families who have methamphetamine use disorders and involvement in child protective services. This program was authorized in House Report 111–220 accompanying HR 3293 in 2010. The Committee language stated that ''these grants will support a collaborative approach, including treatment providers, child welfare specialists, and judges, to provide community-based social services for the children of methamphetamine-addicted parents,'' and were to be awarded to Family Dependency Treatment Drug Courts.