Type of respondents	Survey instrument	Number of respondents	Frequency of responses	Average time per response (minutes/hour)	Annual burden hours
Smoking Cessation ''Quitline'' Clients [1,2]					
Reactive Service Clients	Smoking Cessation ''Intake'' Questions	4,641	1	5/60	386.75
	Demographic Questions	1,300	1	2/60	43.33
Proactive Callback Service Clients [3]	Follow-Up	928	4	1/60	61.87
LiveHelp Clients [4]					
	Demographic questions	7,014	1	2/60	233.80
Total		97,883			2524.00

[1] Approximately 36% of telephone and quitline clients will be sampled for the demographic questions, and 100% of telephone clients will be sampled for the customer service questions. Estimates based on 77.5% response rate.
[2] 100% of smoking cessation clients will be asked the smoking intake questions. Estimates for quitline callers answering demographic questions are based on 77.8% response rate.
[3] 100% of smoking cessation clients participating in the proactive callback service (about 20% of all smoking callers) will be asked the smoking follow-up question (at up to 4 callbacks).
[4] Approximately 50% of LiveHelp clients will be sampled for the demographic questions.

The annualized cost to the respondents is estimated at $48,752. There are no Capital Costs, Operating Costs, and/or Maintenance Costs to report.

Request for Comments: Written comments and/or suggestions from the public and affected agencies should address one or more of the following points: (1) Evaluate whether the proposed collection of information is necessary for the proper performance of the function of the agency, including whether the information will have practical utility; (2) Evaluate the accuracy of the agency's estimate of the burden of the proposed collection of information, including the validity of the methodology and assumptions used; (3) Enhance the quality, utility, and clarity of the information to be collected; and (4) Minimize the burden of the collection of information on those who are to respond, including the use of appropriate automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

Direct Comments to OMB: Written comments and/or suggestions regarding the item(s) contained in this notice, especially regarding the estimated public burden and associated response time, should be directed to the Attention: NIH Desk Officer, Office of Management and Budget, at *OIRA_submission@omb.eop.gov* or by fax to 202–395–6974. To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact Mary Anne Bright, Office of Public Information and Resource Management, Office of Communications and Education, National Cancer Institute, 6116 Executive Blvd., Room 3049, MSC 8322, Bethesda, MD 20892–8322 or call the non-toll-free number 301–594–9048 or e-mail your request, including your address, to: *brightma@mail.nih.gov.*

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 30 days of the date of this publication.

Dated: June 23, 2009.

Vivian Horovitch-Kelley,

NCI Project Clearance Liaison, National Institutes of Health.

[FR Doc. E9–15583 Filed 6–30–09; 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, 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.

ADDRESSES: 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.

New Inhibitors of Polo-like Kinase 1 (PLK1) as Anti-Cancer Agents

Description of Technology: Tumor formation is the result of uncontrolled cellular growth and invasion. Polo-like kinase 1 (PLK1) is a regulator of cell growth whose overexpression has been associated with several types of cancer (e.g., breast cancer, prostate cancer, ovarian cancer, non-small cell lung carcinoma). It has been shown that inhibition of PLK1 causes cell death (apoptosis) in tumor cells but not normal cells. This suggested that inhibiting PLK1 could be an effective treatment for cancer patients without causing unwanted side-effects.

PLK1 contains a unique protein domain known as the polo box domain (PBD), which is essential for its function. One strategy for inhibiting PLK1 involves preventing the PBD domain from interacting with PLK1 substrates. A synthetic peptide with the ability to selectively bind to the PBD was recently identified. Using this peptide as a platform, NIH inventors have designed peptide mimetics that interact with the PBD with greater affinity than the wild-type peptide. By inhibiting PLK1 and selectively inducing apoptosis in cancer cells, these mimetics could serve as potential anti-cancer therapies.

Applications:

- New anti-cancer therapies that specifically target PLK1
- Platform for the development of further improved PLK1 inhibitors

Advantages:

• The peptide mimetics have an increased affinity for the polo box domain of PLK1 compared to the wild-type peptide, making them superior as inhibitors of PLK1.

• The peptide mimetics provide greater metabolic stability and potential effectiveness over synthetic peptides prepared using coded amino acids.

• Inhibiting PLK1 provides an opportunity for successful treatment of cancer with fewer side effects because only tumor cells are killed.

Development Status: Preclinical stage of development

Inventors: Terrence R. Burke Jr. *et al.* (NCI)

Patent Status: US Provisional Application No. 61/178,593 (HHS Reference No. E–181–2009/0–US–01)

For more information, see:

1. F Liu *et al.* SAR by oxime-containing peptide libraries: application to Tsg101 ligand optimization. Chembiochem. 2008 Aug 11;9(12):2000–2004.

2. F Liu *et al.* Protected aminooxyprolines for expedited library synthesis: Application to Tsg101-directed proline-oxime containing peptides. Bioorg Med Chem Lett. 2008 Feb 1;18(3):1096–1101.

3. PCT Application WO 2004/046317, ''Crystal structure of human Polo-like kinase Plk1, Polo Box domain-binding phosphopeptide core sequences, and their therapeutic uses for cancer.''

Licensing Status: Available for licensing.

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

Increasing the Effectiveness of Cancer Treatment: T Cell Receptors Designed To Release Interleukin-12 Specifically at Cancer Sites

Description of Technology: Many conventional chemotherapy drugs currently utilized to treat cancer also yield harsh side effects in patients. In addition, many patients do not respond to generalized chemotherapy and radiation treatments for cancer. There is an urgent need to develop new therapeutic strategies combining fewer side-effects and more specific anti-tumor activity in individual patients. Adoptive immunotherapy is a promising new approach to cancer treatment that engineers an individual's innate and adaptive immune system to fight against specific diseases, including cancer.

T cell receptors (TCRs) are proteins that recognize antigens in the context of infected or transformed cells and activate T cells to mediate an immune response and destroy abnormal cells. TCRs consist of two domains, one variable domain that recognizes the antigen and one constant region that helps the TCR anchor to the membrane and transmit recognition signals by interacting with other proteins. When a TCR is stimulated by an antigen, such as a tumor antigen, some signaling pathways activated in the cell lead to the production of cytokines, which mediate the immune response.

Scientists at the National Institutes of Health (NIH) have developed T cells genetically engineered to express the human interleukin 12 (IL–12) cytokine only in the tumor environment. Specifically, these T cells have been designed to express a human IL–12 gene under the control of the nuclear factor of activated T cells (NFAT) promoter. When the TCR on these T cells recognizes a tumor antigen, IL–12 expression is induced through activation of the NFAT promoter. Thus, IL–12 is only released at the cancer site and only after the activation of the T cell. This technology makes it possible to control the expression of IL–12 to enhance T cell cytolytic activity while also reducing or eliminating the IL–12 toxicity observed with other IL–12 related therapies. Infusing these IL–12 expressing T cells into patients via adoptive immunotherapy could prove to be powerful new tools for attacking tumors.

Applications:

• Immunotherapeutics to treat and/or prevent the recurrence of a variety of human cancers by adoptively transferring the gene-modified T cells into patients.

• A drug component of a combination immunotherapy regimen aimed at targeting the specific tumor-associated antigens expressed by cancer cells within individual patients.

Advantages: The combination of enhanced T cell activity with reduced IL–12 toxicity: IL–12 has shown remarkable properties as an anti-tumor agent, but its clinical development has been hindered by its toxicity. This current technology delivers IL–12 only when and where it is needed—at the tumor site.

Development Status: Clinical trials utilizing this technology are currently in the planning stage.

Market: Cancer continues to be a medical and financial burden on US public health. According to US estimates, cancer is the second leading cause of death with over 565,000 deaths reported in 2008 and almost 1.5 million new cases were reported (excluding some skin cancers) in 2008. In 2007, the NIH estimated that the overall cost of cancer was $219.2 billion dollars and $89 billion went to direct medical costs. Despite our increasing knowledge of oncology and cancer treatment methods, the fight against cancer will continue to benefit from the development of new therapeutics aimed at treating individual patients.

Inventors: Richard A. Morgan *et al.* (NCI)

Publications:

1. L Zhang *et al.* Improving adoptive T cell therapy using NFAT driven human single chain IL–12 expression vector. 2009 American Society of Gene Therapy, abstract submitted.

2. B Heemskerk *et al.* Adoptive cell therapy for patients with melanoma, using tumor-infiltrating lymphocytes genetically engineered to secrete interleukin-2. Hum Gene Ther. 2008 May;19(5):496–510.

3. RA Morgan *et al.* Cancer regression in patients after transfer of genetically engineered lymphocytes. Science 2006 Oct 6;314(5796):126–129.

Patent Status: U.S. Provisional Application No. 61/174,046 filed 30 Apr 2009 (HHS Reference No. E–170–2009/0–US–01)

Licensing Status: Available for licensing.

Licensing Contact: Samuel E. Bish, Ph.D.; 301–435–5282; *bishse@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute, Surgery Branch, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize adoptive immunotherapies or the development of cancer therapeutics based on the use of T cell receptors. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

A Novel System for Producing Infectious Hepatitis C Virus (HCV) Virions and Development of a Novel Reporter System for Studying HCV Entry

Description of Technology: HCV has infected an estimated 3% of the world population in whom viral infection persists for more than two third of the cases, often resulting in life-threatening complications. The standard of care (pegylated interferon alpha-2 plus ribavirin) is efficient in only 50% of treated patients, costly and has numerous side effects. In addition, viral resistance to newly developed drugs—targeting viral protease or RNA polymerase—has been described, but no vaccine is yet available. The difficulty in developing HCV vaccines is largely due to the broad sequence-diversity

displayed by HCV, the frequent occurrence of viral mutations within immunogenic epitopes in vivo, and the lack of proper standard/definition for viral neutralization.

One alternative strategy in HCV-vaccine or drug development comprises measuring viral entry, the first step in viral infection. Such measurements are limited by the available screening systems, in that, HCV pseudo-typed retroviral particles have a different envelope conformation and contain foreign components that are likely to interfere with the measured HCV entry. Moreover, HCV lab strain requires intensive replication for its in vitro production, resulting in numerous mutations that impede development of convenient screening tools.

The inventors have developed a system for generating infectious HCV particles and HCV-like particles (HCV–LP) suitable for a qualitative single-cycle entry assay, completely independent of HCV replication. To adapt this system as a single assay to study HCV–LP entry, HCV non-structural genes were replaced with a heterologous gene that upon viral-entry triggers firefly luciferase and EGFP expressions in target as well as non-permissive cells. The pretreatment of HCV-replication permissive HuH–7.5 cells with siRNA targeting HCV candidate receptors inhibited viral entry. These new systems enable production of authentic HCV infectious particles as well as HCV–LPs suitable for single-cycle entry assays adaptable to high throughput screening.

Applications:

• Screening a library expressed in non-permissive cells for identifying new HCV candidate receptor(s) or entry molecule(s).

• Testing drugs or compounds inhibiting HCV particle entry or viral genome uncoating, or neutralizing antibodies in target cells.

• Testing drugs or compounds that inhibit virus assembly, maturation and/or egress, or genome packaging, in producer cells.

• Incorporating a 'tag' in the genome of various HCV genotypes to more conveniently study virus spreading and dissemination in an organ, tissue and/or small animal model.

• Enhancing immune response in patients: one way to trigger high level anti-HCV immunity is by isolating antigen-presenting cells from patients and incubating them with HCV particles produced with this system using replication-defective viral genome (with or without an immunogenic tag and/or in combination with other viral epitopes) and eventually re-inject their primed cells to the patients.

Advantages:

• These systems do not use pseudo-typed HCV particles, i.e. no foreign proteins present in the virus particles.

• Particle production in the producing cells is independent of HCV RNA replication, hence avoids the occurrence of adaptive mutations that could be detrimental for virus particle's infectivity or could alter tags or nucleotide sequences incorporated in the viral genome.

• These systems are not specifically dedicated to HCV of a particular genotype, i.e. they can be used to generate HCV particles of various genotypes without requiring the use of chimeras.

Development Status:

• Proof of concept.

• Preliminary tools and techniques for screening strategies.

Inventors: Bertrand Saunier, Miriam Triyatni, Edward A. Berger (all NIAID)

Patent Status: U.S. Provisional Application No. 61/195,088 filed 03 Oct 2008 (HHS Reference No. E–005–2009/0–US–01)

Licensing Status: Available for licensing.

Licensing Contact: RC Tang JD, LLM; 301–435–5031; *tangrc@mail.nih.gov.*

Collaborative Research Opportunity: The NIAID OTD is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize a novel system for producing infectious HCV virions and developing a reporter system for studying HCV entry. Please contact Michael Piziali at 301–496–2644 for more information.

Recombinant Virus-Like Particle (VLP) and DNA Vaccines for Chikungunya Virus (CHIKV) and Other Alphaviruses

Description of Technology: Available for licensing and commercial development are compositions and methods of use as vaccines of virus-like particles (VLPs) expressing one or more Alphavirus capsid and envelope proteins, and in particular Chikungunya virus (CHIKV) core and envelope proteins. The invention also describes DNA, viral or other gene-based vector and VLP vaccines, methods of making and methods of their use in inducing immunity, for example to CHIKV infection.

Alphaviruses are RNA-containing viruses that cause a wide variety of mosquito-transmitted diseases, including equine encephalitis. CHIKV, an Alphavirus in the family Togaviridae, was first isolated in Tanzania in 1952 and is transmitted to humans by mosquitoes. The disease caused by CHIKV resembles infection by dengue virus, characterized by rash, high fever, and severe, sometimes persistent arthritis. By 2007, an estimated 1.4–6.5 million people in India, Southeast Asia, Africa and Europe had been infected. Vaccines or anti-viral therapies against CHIKV are not available, raising concerns about its continued evolution and spread in humans. There has been limited success to date in developing a safe and effective CHIKV vaccine. A live CHIKV vaccine candidate caused transient arthralagia in volunteers. Other efforts to develop a CHIKV vaccine include a live attenuated vaccine, a formalin-killed vaccine, a Venezuelan equine encephalitis/CHIKV chimeric live attenuated vaccine and a consensus-based DNA vaccine, but development of a safe and effective CHIKV vaccine will require additional evaluation in humans.

This invention provides CHIKV vaccines based on plasmid expression vectors encoding structural proteins of the virus, which gave rise to VLPs in transfected cells and also served as DNA vaccines. The VLPs consisted of the core, E1 and E2 proteins and were similar in buoyant density and morphology to replication-competent virus. To evaluate the potency and specificity of neutralizing antibodies, pseudotyped lentiviral vectors bearing the CHIKV glycoproteins E1/E2 were developed that showed pH-dependent entry and antibody inhibition similar to CHIKV. Mice were immunized with VLPs (West African strain, 37997) or with DNA vaccines encoding viral gene products from 37997 as well as the latest outbreak strain, OPY–1. Immunization with VLPs elicited high titer neutralizing antibodies against homologous and heterologous strain envelope at >100 fold higher titers than DNA vaccines. These vaccines also induced CD4 and CD8 T-cell responses by analysis with intracellular cytokine staining (ICS). These VLP vaccines are likely to confer protection against emerging CHIKV outbreaks and represent a strategy that could be applied to other pathogenic viruses to prevent their infection and spread.

Applications:

• Development of vaccines against CHIKV

• Development of vaccines against other Alphavirus

Advantages:

• Immunization of mice with VLPs plus adjuvant results in neutralizing antibodies against both homologous and heterologous strains with titers at least two orders of magnitude greater than immunization with a DNA vaccine.

• VLPs induce innate immunity responses as well as CD8 T-cell responses.

• VLPs closely resemble mature virions but they do not contain viral genomic material. Therefore, VLPs are non-replicative in nature, which make them safe for administration in the form of immunogenic compositions in vaccines.

Development Status: This technology is in the pre-clinical stage of development.

Inventors: Gary J. Nabel and Wataru Akahata (NIAID)

Patent Status: U.S. Provisional Application No. 61/201,118 filed 05 Dec 2008, entitled ''Virus Like Particle Compositions and Methods of Use'' (HHS Reference No. E–004–2009/0–US–01)

Licensing Status: Available for licensing.

Licensing Contact: Cristina Thalhammer-Reyero, PhD, MBA; 301–435–4507; *thalhamc@mail.nih.gov.*

Inflammatory Genes and MicroRNA–21 as Biomarkers for Colon Cancer Prognosis

Description of Technology: Colon adenocarcinoma is the leading cause of cancer mortality world-wide and accounts for approximately 50,000 deaths annually in the United States. Adjuvant therapies improve survival for stage III colon cancer patients; however, it remains controversial if stage II patients should be given these therapies. Some stage II patients will benefit from therapy (such as patients with undetectable micro-metastases where surgery will not be curative); but therapy for others will harm quality of life with little therapeutic benefit (such as patients where surgery removed all cancerous tissue and therefore do not need additional therapy). Thus, there is a need for biomarkers capable of accurately identifying high risk, stage II patients that are suitable for therapeutic intervention.

The investigators have identified an inflammatory gene and microRNA biomarker portfolio that can predict aggressive colon cancer, colon cancer patient survival, and patients that are candidates for adjuvant therapy. These biomarkers provide clinicians with a powerful tool to diagnose colon cancer patients and chose effective treatment methods.

Applications:

• Method to predict aggressive form of colon cancer, especially in stage II cancer patients

• Method to determine appropriate colon cancer patients for adjuvant therapy

• Diagnostic arrays

Advantages:

• Rapid, easy to use arrays to accurately predict colon cancer and patients suitable for adjuvant therapy

• Method to stratify colon cancer patients for adjuvant therapy to minimize negative side effects

• Method to identify stage II patients that are likely to have undetectable micro-metastases

Development Status: The technology is currently in the pre-clinical stage of development.

Market:

• Global cancer market is worth more than eight percent of total global pharmaceutical sales

• Cancer industry is predicted to expand to $85.3 billion by 2010

Inventors: Curtis C. Harris and Aaron J. Schetter (NCI)

Relevant Publication: AJ Schetter *et al.* MicroRNA expression profiles associated with prognosis and therapeutic outcome in colon adenocarcinoma. JAMA. 2008 Jan 30;299(4):425–436.

Patent Status: U.S. Provisional Application No. 61/194,340 filed 25 Sep 2008 (HHS Reference No. E–314–2008/0–US–01)

Licensing Status: Available for licensing.

Licensing Contact: Jennifer Wong; 301–435–4633; *wongje@mail.nih.gov.*

Collaborative Research Opportunity: The NCI Laboratory of Human Carcinogenesis is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize cancer biomarkers and therapeutic targets. Please contact *Curtis_Harris@nih.gov* for more information.

Differentiation of Human Embryonic Stem Cells Into Dopaminergic Nerve Cells

Description of Technology: The invention described here is a novel method of differentiating human embryonic stem cells (hESCs) into dopaminergic nerve cells, which is preferable to the currently available dopaminergic differentiation techniques.

This invention potentially provides a source of sufficient dopaminergic cells not only for the clinical transplantation of dopaminergic tissue but also for in vitro studies of human cells useful for pharmaceutical screens related to neurodegenerative disorders and substance abuse.

Neurodegenerative disorders encompass a range of debilitating conditions including Parkinson's disease, Alzheimer's disease, and Huntington's disease. The primary cause of cognitive dysfunction for these three disorders has been directly linked to neuron degeneration, usually in specific areas of the brain. Transplantation of fetal dopaminergic neurons in affected areas of the brain in late stage Parkinson's disease has demonstrated clinical utility in human patients. However, fetal transplantation therapy generally requires human tissue from at least 3–5 embryos to obtain a clinically reliable improvement in the patient, thus demonstrating a need for a larger and more reliable source of dopaminergic cells. HESCs are a promising alternative source of cells because they can grow in culture indefinitely and have the ability to differentiate into a variety of cell types. One of the most efficient methods for conversion of hESCs to dopaminergic neurons requires the presence of mouse stromal cells which have an undefined dopaminergic inducing activity. However, the major disadvantage of this method is the exposure of hESC to mouse cells, which hinders any downstream clinical application due to possible transfer of animal cells and pathogens. This invention has unveiled the molecular nature of the activity of the mouse cells and established an efficient alternative approach for dopamine neuron generation, which is more suitable for clinical application. This innovative approach potentially provides a large and reliable source of dopaminergic cells sufficient for clinically relevant transplantation of dopaminergic tissue as well as in vitro pharmacologic studies of human dopaminergic cells.

Applications:

• Human dopaminergic cell source for neuronal transplantation, with potential clinical application to Parkinson's disease and possibly other neurodegenerative disorders.

• Human dopaminergic cell source for in vitro models for pharmaceutical screens relevant to neurodegenerative disorders and substance abuse.

Market: Parkinson's disease, the second most common neurological disorder, affects approximately 4.1 million people worldwide. In 2006, global sales of Parkinson's disease therapeutics were $3.1 billion, with sales expected to exceed $4.6 billion by 2012.

Development Status: Early stage.

Inventors: William Freed and Tandis Vazin (NIDA).

Publication: In preparation.

Patent Status: U.S. Provisional Application No. 61/199,652 filed 18

Nov 2008 (HHS Reference No. E–176–2008/0–US–01).

Licensing Status: Available for licensing.

Licensing Contact: Norbert Pontzer, J.D., PhD; 301–435–5502; *pontzern@mail.nih.gov.*

Collaborative Research Opportunity: The National Institute on Drug Abuse, Development and Plasticity Section, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact Vio Conley, M.S. at 301–496–0477 or *conleyv@mail.nih.gov* for more information.

Dated: June 22, 2009.

Richard U. Rodriguez,

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

[FR Doc. E9–15578 Filed 6–30–09; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Amended Notice of Meeting

Notice is hereby given of a change in the meeting of the Center for Scientific Review Special Emphasis Panel, July 20, 2009, 8 a.m. to July 21, 2009, 7:30 p.m., Hyatt Regency Bethesda, One Bethesda Metro Center, 7400 Wisconsin Avenue, Bethesda, MD 20814 which was published in the **Federal Register** on June 15, 2009, 74 FR 28260–28262.

The meeting will be held at the Doubletree Hotel Washington, 1515 Rhode Island Avenue, NW., Washington, DC 20005. The meeting dates and time remain the same. The meeting is closed to the public.

Dated: June 22, 2009.

Anna Snouffer,

Deputy Director, Office of Federal Advisory Committee Policy.

[FR Doc. E9–15580 Filed 6–30–09; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Amended Notice of Meeting

Notice is hereby given of a change in the meeting of the Center for Scientific Review Special Emphasis Panel, July 20, 2009, 8 a.m. to July 21, 2009, 4 p.m., DoubleTree Hotel Bethesda, 8120 Wisconsin Avenue, Bethesda, MD, 20814 which was published in the **Federal Register** on June 22, 2009, 74 FR 29500–29502.

The meeting will be held at the Hyatt Regency Bethesda, One Bethesda Metro Center, 7400 Wisconsin Avenue, Bethesda, MD 20814. The meeting date and time remain the same. The meeting is closed to the public.

Dated: June 22, 2009.

Anna Snouffer,

Deputy Director, Office of Federal Advisory Committee Policy.

[FR Doc. E9–15582 Filed 6–30–09; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Amended Notice of Meeting

Notice is hereby given of a change in the meeting of the Center for Scientific Review Special Emphasis Panel, July 20, 2009, 8 a.m. to July 21, 2009, 6 p.m., Ritz Carlton Hotel, 1150 22nd Street, NW., Washington, DC, 20037 which was published in the **Federal Register** on June 15, 2009, 74 FR 28260–28262.

The meeting will be held at the Embassy Suites at the Chevy Chase Pavilion, 4300 Military Road, NW., Washington, DC 20015. The meeting dates and time remain the same. The meeting is closed to the public.

Dated: June 22, 2009.

Anna Snouffer,

Deputy Director, Office of Federal Advisory Committee Policy.

[FR Doc. E9–15588 Filed 6–30–09; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Eye 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.

Name of Committee: National Eye Institute Special Emphasis Panel; NEI Research Project Grant, Cooperative Agreement and Competitive Supplement Applications.

Date: August 3, 2009.

Time: 8:30 a.m. to 5 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: Anne E. Schaffner, PhD, Scientific Review Officer, Division of Extramural Research, National Eye Institute, 5635 Fishers Lane, Suite 1300, MSC 9300, Bethesda, MD 20892–9300, (301) 451–2020, *aes@nei.nih.gov.*

Name of Committee: National Eye Institute Special Emphasis Panel NEI Translational Research Program on Therapy for Visual Disorders.

Date: August 4, 2009.

Time: 8:30 a.m. to 2 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: Anne E. Schaffner, PhD, Scientific Review Officer, Division of Extramural Research, National Eye Institute, 5635 Fishers Lane, Suite 1300, MSC 9300, Bethesda, Md 20892–9300, (301) 451–2020, *aes@nei.nih.gov.*

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

Dated: June 25, 2009.

Anna Snouffer,

Deputy Director, Office of Federal Advisory Committee Policy.

[FR Doc. E9–15592 Filed 6–30–09; 8:45 am]

BILLING CODE 4140–01–M

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