Teresa.Watkins@fda.hhs.gov, or FDA Advisory Committee Information Line, 1–800–741–8138 (301–443–0572) in Washington, DC area), codes 3014512529 and 3014512535. Please call the Information Line for up-to-date information on this meeting.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of March 27, 2008, FDA announced that a meeting of the Anesthetic and Life Support Drugs Advisory Committee and the Drug Safety and Risk Management Advisory Committee would be held on May 5 and 6, 2008.

On page 16314, in the third column, the introductory paragraph of the document is amended to read as follows:

This notice announces a forthcoming meeting of a public advisory committee of the Food and Drug Administration (FDA). At least one portion of the meeting will be closed to the public.

On page 16315, the second column of the document is amended to add a portion entitled ''*Closed Committee Deliberations*'' to read as follows:

Closed Committee Deliberations: On May 5, 2008, from 8 a.m. to 9:15 a.m., the meeting will be closed to permit discussion and review of trade secret and/or confidential commercial information (5 U.S.C. 552b(c)(4)). During this session, the committee will discuss the details of a proprietary research report and protocol addressing characteristics of different formulations.

This notice is issued under the Federal Advisory Committee Act (5 U.S.C. app. 2) and 21 CFR part 14, relating to the advisory committees.

Dated: April 16, 2008.

Randall W. Lutter,

Deputy Commissioner for Policy.

[FR Doc. E8–8683 Filed 4–21–08; 8:45 am]

BILLING CODE 4160–01–S

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.

Platform for the High Throughput Screening of Single Nucleotide Polymorphisms and Small Insertions and Deletions

Description of Technology: Available for licensing and commercial development is an oligoarray-based process for gene-specific single nucleotide polymorphism (SNP) genotyping based on comparative hybridization. This process can detect, even in heterozygous conditions, known and potentially flag unknown variants (point mutations, base insertion or deletion) along the complete sequence of a given gene while drastically cutting the time and costs compared to high-throughput direct sequencing without affecting sensitivity and specificity. The accuracy and efficiency of the invention was validated based on the BRCA–1 breast and ovarian cancer predisposing gene. This process can easily be custom designed to include within the same platform a relatively large number of genes relevant to a specific clinical condition and it is particularly useful for the screening of long genomic region with relatively infrequent but clinically relevant variants.

More specifically, the invention is made reliable by the development of two tailored algorithms: the first automatically designs the complete data set of gene-specific probes starting from the genomic sequence according to the user specification (size of the probes, relative position, etc.); and the other is based on an algorithm that flags gene variants in the test sample. This allows detecting unknown variants in the region in which only the reference hybridizes to the probes. These features drastically reduce the amount of sequencing (the gold standard for SNP detection) to small regions in which a discrepancy between test signal and reference signal is found. Moreover, there is no limit, other than the physical area of the slide, to the number of probes that can be added to the array and the number of genes that can be queried simultaneously. Thus, a repertoire of considerable size can be scanned in a single test for each sample with sensitivity and specificity comparable to direct sequencing.

Applications: The immediate clinical applications of this platform is a remarkable improvement of genetic testing by increasing the number of target genes that can be screened in a short time, at a minimal cost using an automated simplified analysis, such as the sequencing-grade screening for BRCA–1 variants and the detection of mutations in cancerous tissues. The method can be also applied to other human genes (coding and non-coding sequences), and other sequences from animals, bacterial and viruses.

Development Status: Method fully developed and validated.

Inventors: Ena Wang (CC), Alessandro Monaco (CC), Francesco M Marincola (CC), et al.

Patent Status: U.S. Provisional Application No. 61/068,182 filed 05 Mar 2008 (HHS Reference No. E–082–2008/0–US–01).

Licensing Status: Available for non-exclusive or exclusive licensing.

Licensing Contact: Cristina Thalhammer-Reyero, Ph.D., M.B.A.; 301–435–4507; *thalhamc@mail.nih.gov*.

Generation of Wild-Type Dengue Viruses for Use in Rhesus Monkey Infection Studies

Description of Technology: Dengue virus is a positive-sense RNA virus belonging to the *Flavivirus* genus of the family Flaviviridae. Dengue virus is widely distributed throughout the tropical and semitropical regions of the world and is transmitted to humans by mosquito vectors. Dengue virus is a leading cause of hospitalization and death in children in at least eight tropical Asian countries. There are four serotypes of dengue virus (DEN–1, DEN–2, DEN–3, and DEN–4) that annually cause an estimated 50–100 million cases of dengue fever and 500,000 cases of the more severe form of dengue virus infection known as dengue hemorrhagic fever/dengue shock syndrome (DHF/DSS). This latter disease is seen predominately in children and adults experiencing a second dengue virus infection with a serotype different than that of their first dengue virus infection and in primary infection of infants who still have circulating dengue-specific maternal antibody. A vaccine is needed to lessen the disease burden caused by dengue virus, but none is licensed.

Because of the association of more severe disease with secondary dengue

virus infection, a successful vaccine must induce immunity to all four serotypes. Immunity is primarily mediated by neutralizing antibody directed against the envelope (E) glycoprotein, a virion structural protein. Infection with one serotype induces long-lived homotypic immunity and a short-lived heterotypic immunity. Therefore, the goal of immunization is to induce a long-lived neutralizing antibody response against DEN–1, DEN–2, DEN–3, and DEN–4, which can best be achieved economically using live attenuated virus vaccines. This is a reasonable goal since a live attenuated vaccine has already been developed for the related yellow fever virus, another mosquito-borne flavivirus present in tropical and semitropical regions of the world.

The evaluation of live attenuated dengue vaccine candidates in rhesus monkeys requires wild type control viruses for each of the four dengue serotypes. These control viruses are used for comparison to the attenuated strains and post-vaccination challenge to assess vaccine efficacy. As such, these viruses need to be well characterized and sufficiently pure to ensure that they will replicate to consistent levels in rhesus monkeys. Characterization generally includes sequence analysis, titration, and evaluation in monkeys. The following viruses have been characterized: (1) DEN1 WP (2) DEN1 Puerto Rico/94 (3) DEN2 NGC prototype (4) DEN2 Tonga/74 (5) DEN3 Sleman/78 and (6) DEN4 Dominica/81.

Application: Dengue/flavivirus vaccine studies, dengue/flavivirus diagnostics, dengue/flavivirus research tools.

Development Status: Materials are available for transfer.

Inventors: Stephen S. Whitehead and Joseph E. Blaney, Jr. (NIAID).

Publications:

1. AP Durbin, RA Karron, W Sun, DW Vaughn, MJ Reynolds, JR Perreault, B Thumar, R Men, C-J Lai, WR Elkins, RM Chanock, BR Murphy, SS Whitehead. A live attenuated dengue virus type 4 vaccine candidate with a 30 nucleotide deletion in the 3′ untranslated region is highly attenuated and immunogenic in humans. Am J Trop Med Hyg. 2001 Nov;65(5):405–413.

2. SS Whitehead, B Falgout, KA Hanley, JE Blaney Jr., L Markoff, BR Murphy. A live, attenuated dengue virus type 1 vaccine candidate with a 30-nucleotide deletion in the 3′ untranslated region is highly attenuated and immunogenic in monkeys. J Virol. 2003 Jan;77(2):1653–1657.

3. SS Whitehead, KA Hanley, JE Blaney Jr., LE Gilmore, WR Elkins, BR Murphy. Substitution of the structural genes of dengue virus type 4 with those of type 2 results in chimeric vaccine candidates which are attenuated for mosquitoes, mice, and rhesus monkeys. Vaccine 2003 Oct 1;21(27–30):4307–4316.

4. JE Blaney Jr., CT Hanson, KA Hanley, BR Murphy, SS Whitehead. Vaccine candidates derived from a novel infectious cDNA clone of an American genotype dengue virus type 2. BMC Infect Dis. 2004 Oct 4;4:39.

5. JE Blaney Jr., CT Hanson, CY Firestone, KA Hanley, BR Murphy, SS Whitehead. Genetically modified, live attenuated dengue virus type 3 vaccine candidates. Am J Trop Med Hyg. 2004 Dec;71(6):811–821.

6. JE Blaney Jr., JM Matro, BR Murphy, SS Whitehead. Recombinant, live-attenuated tetravalent dengue virus vaccine formulations induce a balanced, broad, and protective neutralizing antibody response against each of the four serotypes in rhesus monkeys. J Virol. 2005 May;79(9):5516–5528.

7. JE Blaney Jr., NS Sathe, CT Hanson, CY Firestone, BR Murphy, SS Whitehead. Vaccine candidates for dengue virus type 1 (DEN1) generated by replacement of the structural genes of rDEN4 and rDEN4Delta30 with those of DEN1. Virol J. 2007 Feb 28;4:23.

Patent Status: HHS Reference No. E–042–2008/0—Research Tool. Patent protection is not being sought for this technology.

Licensing Status: Available for nonexclusive biological materials licensing only.

Licensing Contact: Peter A. Soukas, J.D.; 301–435–4646; *soukasp@mail.nih.gov*.

A Rapid Ultrasensitive Assay for Detecting Prions in Samples Based on the Seeded Polymerization of Recombinant Normal Prion Protein (rPrP-sen)

Description of Technology: Prion diseases are infectious neurodegenerative diseases of great public concern. Humans may be infected by eating infected animals (primarily hoofed animals or ungulates). Blood transfusions have also been documented as a cause of human cases of prion infection. Prion diseases include: Creutzfeldt-Jakob disease (CJD) (humans); variant Creutzfeldt-Jakob disease (vCJD) (humans); Scrapie (sheep); Bovine Spongiform Encephalopathy (BSE) (cattle); and Chronic Wasting Disease (deer, elk and moose). Currently available rapid tests for infectious prions, which are routinely used to monitor slaughtered animals, are not sensitive enough to detect prion infections in samples from live animals or humans and must be performed post-mortem. Additionally, these tests cannot be used to detect subinfectious concentrations of infectious prions in humans or animals. An ultrasensitive assay for infectious prions, the protein-misfolding cyclic amplification assay (PMCA), is available for testing live animals or humans; however, this test is expensive because it is difficult to perform, relies on the use of brain homogenates, and can take 2–3 weeks to perform.

This technology enables the rapid detection of extremely low, sub-lethal, concentrations of prions. This assay, like PMCA, is based on the prion-induced polymerization of normal prion protein (PrP-sen). However, this assay, unlike PMCA uses recombinant normal prion protein (rPrP-sen) rather than normal prion protein derived from brain homogenate. The use of rPrP-sen provides major advantages over PMCA. rPrP-sen provides a relatively inexpensive, abundant, and concentrated source of pure PrP-sen as a substrate for the PMCA prion amplification reaction. This permits the detection of PrP-res in 2–3 hours and the ultrasensitive detection of PrP-res in 2 to 3 days. Moreover, relative to PrP-sen in brain tissue, rPrP-sen is much easier to mutate and chemically modify to facilitate detection of prion-induced PMCA amplification products in potentially high-throughput formats. In its current embodiment, the ultrasensitive assay has been used to consistently detect (by western blot) around 50 ag of hamster PrP-Sc (0.003 lethal dose) in cerebral spinal fluid and brain tissue within 2 to 3 days.

Applications:

A diagnostic assay for detecting prion diseases early.

An assay for monitoring the progression of prion disease and the effectiveness of treatments.

A veterinary assay for detecting PrP-res in live animals and assessing the extent of prion disease in live herds.

An assay for the detection of prion in commercial products (e.g., biotechnological or agricultural), blood and blood products, transplantation tissues, medical devices, and environmental samples.

Market:

Currently, there is a need for a rapid, ultrasensitive, veterinary test for prion diseases in live animals used for human consumption and a need for assessing the extent of prion infection in live herds.

Currently, there is a need for a human diagnostic assay to detect prion disease early when treatment is most effective and a need for monitoring the effectiveness of treatments for prion diseases.

Currently, there is a need for a rapid, ultrasensitive test for prions in commercial products (e.g., biotechnological or agricultural), blood and blood products, transplantation tissues, medical devices, and environmental samples in which prion contamination might be a concern.

Inventors: Ryuichiro Atarashi, Roger A. Moore, Suzette A. Priola, and Byron W. Caughey (NIAID).

Related Publication: R Atarashi et al. Ultrasensitive detection of scrapie prion protein using seeded conversion of recombinant prion protein. Nat Methods 2007 Aug;4(8):645–650.

Patent Status: U.S. Provisional Application No.60/961,364 filed 20 Jul 2007 (HHS Reference No. E–109–2007/0-US–01).

Licensing Status: Available for exclusive and non-exclusive licensing.

Licensing Contact: RC Tang, J.D., LL.M.; 301–435–5031; *tangrc@mail.nih.gov*.

Collaborative Research Opportunity: The NIAID Laboratory of Persistent Viral Diseases, TSE/Prion Biochemistry Section, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact Byron Caughey, Ph.D. at (406) 363–9264 or *bcaughey@niaid.nih.gov* for more information.

Identification of a Cell-Surface Co-Receptor That Mediates the Uptake and Immunostimulatory Activity of "D" Type CpG Oligonucleotides

Description of Technology: Unmethylated CpG motifs are present at high frequency in bacterial DNA. They provide a danger signal to the mammalian immune system that triggers a protective immune response characterized by the production of Th1 and proinflammatory cytokines and chemokines. Although the recognition of CpG DNA by B cells and plasmacytoid dendritic cells is mediated by TLR 9, these cell types differ in their ability to bind and respond to structurally distinct classes of CpG oligonucleotides. The inventors' work established that CXCL16, a membrane-bound scavenger receptor, influences the uptake, subcellular localization, and cytokine profile induced by D oligonucleotides.

Knowing that CXCL16 can be used to selectively internalize ODN could be useful for (1) Improving the activity of D type ODN, (2) improving recognition (and side effects) of other types of ODNs by deleting regions that interact with CXCL16 (3) potentially improving the targeting of any drug or biologic to CXCL16 expressing cells, (4) targeting antisense ODNs to immune cells or preventing side effects from antisense therapy, and also applications to (5) DNA vaccines and other agents that require targeting to CXCL16 expressing cells such as dendritic cells and monocytes.

This application claims methods of inducing an immune response that include administering agents that increase the activity and/or expression of CXCL16 and a D ODN. The application also claims methods of decreasing an immune response to a CpG ODN, including administering agents that decrease the activity and/or expression of CXCL16. Compositions including one or more D type ODNs and an agent that modulates the activity and/or expression of CXCL16 are also claimed.

Application: Vaccine adjuvants, production of vaccines, immunotherapeutics.

Developmental Status: Preclinical studies have been performed; oligonucleotides have been synthesized.

Inventors: Dennis Klinman (FDA/CBER; NCI), Ihsan Gursel (FDA/CBER), Mayda Gursel (FDA/CBER).

Publication: M Gursel et al. CXCL16 influences the nature and specificity of CpG-induced immune activation. J Immunol. 2006 Aug 1;177(3):1575–1580.

Patent Status: U.S. Provisional Application No. 60/713,547 filed 31 Aug 2005 (HHS Reference No. E–036–2005/0–US–01); PCT Application No. PCT/US2006/033774 filed 28 Aug 2006 (HHS Reference Number E–036–2005/0–PCT–02); U.S. Patent Application No. 12/065,085 filed 27 Feb 2008 (HHS Reference Number E–036–2005/0–US–03).

Licensing Status: Available for exclusive or nonexclusive licensing.

Licensing Contact: Peter A. Soukas, J.D.; 301–435–4646; *soukasp@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute, Laboratory of Experimental Immunology, Immune Modulation Group, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Use of Suppressive Oligonucleotides To Treat Uveitis

Description of Technology: Uveitis is a major cause of visual loss in industrialized nations. Uveitis refers to an intraocular inflammation of the uveal tract, namely, the iris, choroids, and ciliary body. Uveitis is responsible for about ten percent (10%) of the legal blindness in the United States. Complications associated with uveitis include posterior synechia, cataracts, glaucoma and retinal edema.

Suppressive CpG oligodeoxynucleotides (ODNs) are ODNs capable of reducing an immune response, such as inflammation. Suppressive ODNs are DNA molecules of at least eight nucleotides in length, where the ODN forms a G-tetrad, and has a circular dichroism value greater than 2.9. In a suppressive ODN, the number of guanosines is at least two.

This application claims compositions and methods for the treatment of uveitis. Specifically, the application claims use of suppressive CpG ODNs to treat uveitis. The compositions and methods of the application can be used for the treatment of anterior, posterior and diffuse uveitis.

Application: Vaccine adjuvants, production of vaccines, immunotherapeutics.

Developmental Status: Preclinical studies have been performed; oligonucleotides have been synthesized.

Inventors: Dennis Klinman (FDA/CBER; NCI), Igal Gery (NEI), Chiaki Fujimoto (NEI).

Patent Status: U.S. Provisional Application No. 60/569,276 filed 06 May 2004 (HHS Reference No. E–152–2004/0–US–01); PCT Application No. PCT/US2005/015761 filed 05 May 2005, which published as WO 2005/11539 on 09 Dec 2006 (HHS Reference No. E–152–2004/0–PCT–02); U.S. Patent Application No. 11/579,518 filed 03 Nov 2006 (HHS Reference Number E–152–2004/0–US–03); International filings in Australia, Canada, China, Europe, India, Japan, Mexico.

Licensing Status: Available for exclusive or nonexclusive licensing.

Licensing Contact: Peter A. Soukas, J.D.; 301–435–4646; *soukasp@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute, Laboratory of Experimental Immunology, Immune Modulation Group, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact John D. Hewes, Ph.D. at 301–

435–3121 or *hewesj@mail.nih.gov* for more information.

Mapping Internal and Bulk Motion of an Object With Phase Labeling in Magnetic Resonance Imaging

Description of Technology: Current MRI methods for tracking the motion of an object over a relatively long period of time requires the use of precisely defined grid points that may be inexact because of limited image resolution or the size of the element being tracked. Phase contrast velocity mapping generally provides high spatial resolution and simple data processing. However, it is generally unsuitable for motion tracking and prone to error. This invention is a cutting edge Magnetic Resonance Imaging (MRI) technique that provides a method for mapping the internal and bulk motion of a specimen by labeling the phase of the specimen magnetization with a selected spatial function and measuring changes in the phase of the magnetization. The special function is selectable to provide magnetization phase modulation corresponding to displacements in a selected direction such as Cartesian or radial or azimuthal direction. This method and associated apparatus is capable of producing images based on magnetization phase modulation using data from stimulated echoes and anti-echoes. This invention has important applications in, among other areas, cardiac functional imaging and can be used to compute accurate strain maps of the heart.

Inventors: Anthony H. Aletras and Han Wen (NHLBI).

Patent Status: U.S. Patent No. 7,233,818 issued 19 Jun 2007 (HHS Reference No. E–234–1999/3–US–06); U.S. Patent Application No. 11/800,398 filed 03 May 2007 (HHS Reference No. E–234–1999/3–US–08).

Licensing Status: Available for non-exclusive licensing.

Licensing Contact: Susan Ano, Ph.D.; 301–435–5515; *anos@mail.nih.gov.*

Dated: April 14, 2008.

David Sadowski,

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

[FR Doc. E8–8620 Filed 4–21–08; 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, April 17, 2008, 3 p.m. to April 17, 2008, 5 p.m. National Institutes of Health, 6701 Rockledge Drive, Bethesda MD 20892 which was published in the **Federal Register** on April 9, 2008, 73 FR 19229.

The meeting will be held April 21, 2008. The meeting time and location remain the same. The meeting is closed to the public.

Dated: April 14, 2008.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. E8–8452 Filed 4–21–08; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), 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: Center for Scientific Review Special Emphasis Panel; Multicale Models of Physiome Conflict.

Date: May 21, 2008.

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

Agenda: To review and evaluate grant applications.

Place: Georgetown Suites, 1111 30th Street, NW., Washington, DC 20007.

Contact Person: Ping Fan, PhD, Scientific Review Administrator, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 5154, MSC 7840, Bethesda, MD 20892, 301–435–1740, *fanp@csr.nih.gov.*

Name of Committee: Immunology Integrated Review Group; Transplantation, Tolerance, and Tumor Immunology Study Section.

Date: May 29–30, 2008.

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

Agenda: To review and evaluate grant applications.

Place: Hilton Silver Spring, 8727 Colesville Road, Silver Spring, MD 20910.

Contact Person: Cathleen L. Cooper, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 4208, MSC 7812, Bethesda, MD 20892, 301–435–3566, *cooperc@csr.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel; Member Conflict: Cell Death and Neurodegeneration.

Date: June 2, 2008.

Time: 2 p.m. to 4 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892, (Telephone Conference Call).

Contact Person: Alexander Yakovlev, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 5206, MSC 7846, Bethesda, MD 20892, 301–435–1254, *yakovleva@csr.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel; Chronic Fatigue and Fibromyalgia Syndromes, Temporomandibular Disorders.

Date: June 4, 2008.

Time: 1 p.m. to 4 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892, (Telephone Conference Call).

Contact Person: J. Terrell Hoffeld, DDS, PhD, USPHS Dental Director, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 4116, MSC 7816, Bethesda, MD 20892, 301–435–1781, *th88q@nih.gov.*

Name of Committee: Endocrinology, Metabolism, Nutrition and Reproductive Sciences Integrated Review Group; Cellular Aspects of Diabetes and Obesity Study Section.

Date: June 5–6, 2008.

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

Agenda: To review and evaluate grant applications.

Place: San Francisco Airport Marriott, 1800 Old Bayshore Highway, Burlingame, CA 94010.

Contact Person: Ann A. Jerkins, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 6154, MSC 7892, Bethesda, MD 20892, (301) 435–4514, *jerkinsa@csr.nih.gov.*

Name of Committee: Health of the Population Integrated Review Group; Nursing Science: Children and Families Study Section.

Date: June 5, 2008.

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

Agenda: To review and evaluate grant applications.

Place: Bethesda Marriott, 5151 Pooks Hill Road, Bethesda, MD 20814.