part or all of their indebtedness for professional training time in IHS health care facilities. This program is necessary to augment the critically low health professional staff at IHS health care facilities.

Any health professional wishing to have their health education loans repaid may apply to the IHS Loan Repayment Program. A two-year contract obligation is signed by both parties, and the individual agrees to work at an IHS location and provide health services to Native American and Alaska Native individuals.

The information collected from individuals is analyzed and a score is given to each applicant. This score will determine which applicants will be awarded each fiscal year. The administrative scoring system assigns a score to the geographic location according to vacancy rates for that fiscal year and also considers whether the location is in an isolated area. When an applicant takes employment at a location, they in turn ''pick-up'' the score of that location. *Affected Public:* Individuals and households. *Type of Respondents:* Individuals.

The table below provides: Types of data collection instruments, Estimated number of respondents, Number of responses per respondent, Annual number of responses, Average burden hour per response, and Total annual burden hour(s).

ESTIMATED BURDEN HOURS

Data collection instrument	Estimated number of respondents	Responses per respondent	Average burden hour per response	Total annual burden hours
Section I	510	1	18/60	153.0
Section II	510	1	30/60	255.0
Section III	510	4	15/60	128.0
Contract	510	1	20/60	170.0
Affidavit	510	1	10/60	85.0
Lender's Certification	2,000		15/60	500.0
Total	4,650			1,282.0

There are no Capital Costs, Operating Costs, and/or Maintenance Costs to report.

Request for Comments: Your written comments and/or suggestions are invited on one or more of the following points: (a) Whether the information collection activity is necessary to carry out an agency function; (b) whether the agency processes the information collected in a useful and timely fashion; (c) the accuracy of public burden estimate (the estimated amount of time needed for individual respondents to provide the requested information); (d) whether the methodology and assumptions used to determine the estimates are logical; (e) ways to enhance the quality, utility, and clarity of the information being collected; and (f) ways to minimize the public burden through the use of automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

Send Comments and Requests for Further Information: Send your written comments, requests for more information on the proposed collection, or requests to obtain a copy of the data collection instrument(s) and instructions to: Ms. Chria Rouleau, IHS Reports Clearance Officer, 801 Thompson Avenue, TMP 450, Rockville, MD 20852–1627; call non-toll free (301) 443–5938; send via facsimile to (301) 594–0899; or send your e-mail requests, comments, and return address to: *Christina.Rouleau@ihs.gov.*

Comment Due Date: Your comments regarding this information collection are best assured of having full effect if received within 60 days of the date of this publication.

Dated: May 13, 2008.

Robert G. McSwain,

Director, Indian Health Service.

[FR Doc. E8–11184 Filed 5–20–08; 8:45 am]

BILLING CODE 4165–16–M

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.

Synthetic Analogs of Juxtamembrane Domain of IGF–1 Receptor as Anti-Cancer Agents

Description of Technology: Insulin-like growth factor receptor type one (IGF–1R), part of the receptor tyrosine kinase (RTKs) family, is integral to cancer cell growth and metastasis. Juxtamembrane domains (JM) of RTKs are located in the cytoplasm between the transmembrane and kinase domains. JMs play a crucial role in the inhibition of the regulation of receptor activity. Studies on other small molecules tyrosine kinase inhibitors (TKIs) indicate non-specific binding with the insulin receptor which has high homology with IGF–1R.

The current invention describes synthetic analogs of IGF–1R JM which were found to be potent inhibitors of IGF–1–mediated cell signaling and cancer cell growth. These analogs provide more binding specificity with less likelihood of significant toxic effects.

Applications and Modality:

New inhibitors can be used to treat many types of tumors.

IGR–1R inhibition may be useful as an anti-aging agent.

IGR–1R plays an inhibitory role in regulation of skin development and differentiation. IGF–1R inhibitors may have revitalizing and rejuvenating effect on skin and may stimulate wound healing.

Market:

An estimated 1,444,920 new cancer diagnoses in the U.S. in 2007.

600,000 deaths caused by cancer in the U.S. in 2006.

Cancer is the second leading cause of death in the U.S.

Cancer drug market will likely double to $50 billion in 2010 from $25 billion in 2006.

Development Status: The technology is currently in the preclinical stage of development.

Inventors: Nadya I. Tarasova and Sergey G. Tarasov (NCI).

Patent Status: U.S. Provisional Application No. 61,040,203 filed 28 Mar 2008 (HHS Reference No. E–129–2008/0–US–01).

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

Licensing Contact: Adaku Nwachukwu, J.D.; 301–435–5560; *madua@mail.nih.gov*.

Protein-Tyrosine Phosphotase Inhibitors as Inhibitors of Human Tyrosyl-DNA Phosphodiesterase (Tdp1) and Methods of Treating Disorders

Description of Technology: Tyrosyl-DNA phosphodiesterase (Tdp1) is an enzyme that repairs topoisomerase I (Top1)-mediated DNA damage induced by chemotherapeutic agents (such as camptothecins) and ubiquitous DNA lesions that interfere with transcription and replication. Tdp1 is a relevant target for anticancer therapies due to its role in repairing Top1-mediated DNA damage and DNA damage associated with DNA strand breaks. Tdp1 inhibitors are expected to be effective in cancer treatment when used in combination with Top1 inhibitors.

The current invention is Me-3,4 dephostatin, and more generally protein-tyrosine phosphatase inhibitors, which is a Tdp1 inhibitor. Me-3,4 dephostatin could potentiate the pharmacological action of Top1 inhibitors.

Applications and Modality:

It is anticipated that Tdp1 inhibitors in association with Top1 inhibitors can have selective activity toward tumor tissues.

Tdp1 inhibitors may exhibit antitumor activity by themselves because tumors have excess free radicals.

Market:

An estimated 1,444,920 new cancer diagnoses in the U.S. in 2007.

600,000 deaths caused by cancer in the U.S. in 2006.

Cancer is the second leading cause of death in the U.S.

Cancer drug market will likely double to $50 billion in 2010 from $25 billion in 2006.

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

Inventors: Yves Pommier (NCI) *et al.*

Relevant Publication: S Antony *et al.* Novel high-throughput electrochemiluminescent assay for identification of human tyrosyl-DNA phosphodiesterase (Tdp1) inhibitors and characterization for furamidine (NSC 305831) as an inhibitor of Tdp1. Nucleic Acid Res. 2007;35(13):4474–4484.

Patent Status: U.S. Provisional Application No. 61,040,203 filed 28 Mar 2008 (HHS Ref. No. E–121–2008/0–US–01).

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

Licensing Contact: Adaku Nwachukwu, J.D.; 301–435–5560; *madua@mail.nih.gov*.

Method of Inhibiting ABCG2 and Related Treatments

Description of Technology: The technology is directed to a method of inhibiting ABCG2, which is a multidrug resistance (MDR) protein. It is believed that ABCG2 plays a role in the development of resistance of cancer cells to chemotherapeutics. Therefore, inhibition of ABCG2 would allow chemotherapeutics to be more effective in killing cancer cells, thereby treating cancer. Five compounds were identified in the provisional application that inhibit ABCG2. These compounds are known in the literature and are part of the NCI Developmental Therapeutics Program (DTP).

Applications: Cancer therapeutics; Research tools to study function of ABCG2 proteins.

Advantages: Valuable tools to further developing understanding or normal and cancer cells; Augment efficacy of drugs that are ABCG2 substrates.

Development Status: Early stage.

Market: Cancer is the second leading cause of death in America, after heart disease. Multiple drug resistance is a significant impediment in the treatment of cancers resulting in a poor prognosis. The market for effective cancer treatments is very large.

Inventors: Curtis J. Henrich (SAIC/NCI), Heidi R. Bokesch (SAIC/NCI), Susan E. Bates (NCI), Robert W. Robey (NCI), Suneet Shukla (NCI), Suresh V. Ambudkar (NCI), Michael C. Dean (NCI), and James B. McMahon (NCI).

Patent Status: U.S. Provisional Application No. 60/986,155 filed 07 Nov 2007 (HHS Reference No. E–316–2007/0–US–01).

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

Licensing Contact: John Stansberry, Ph.D.; 301–435–5236; *stansbej@mail.nih.gov*.

Collaborative Research Opportunity: The National Cancer Institute Molecular Targets Development Program is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Method of Inhibiting ABCG2 and Related Treatments. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Method of Inducing Memory B Cell Development and Terminal Differentiation

Description of Technology: Cytokines exert their respective biochemical and physiological effects by binding to specific receptor molecules, which then stimulate signal transduction pathways. Interleukin–21 (IL–21) is a type I cytokine whose receptor is expressed on T, B, and NK cells.

This invention specifically relates to the use of IL–21 to induce differentiation of immature B cells into memory B cells and plasma cells. This invention includes claims of methods for inducing differentiation of a B cell progenitor into memory B cells and/or plasma cells. It also includes claims for enhancing an immune response, treating subjects that lack memory B cells and plasma cells and methods for increasing or decreasing the number of B cells. This invention could conceivably be used in treating or preventing inflammatory disorders, autoimmune diseases, allergies, transplant rejection, cancer, and other immune system disorders.

Inventors: Peter E. Lipsky (NIAMS) *et al.*

Patent Status: U.S. Patent Application No. 11/197,221 filed 03 Aug 2005, allowed (HHS Reference No. E–120–2003/2–US–01).

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

The Use of an Inducible Plasmid Vector Encoding for Active TGF–β for the Treatment of Autoimmune Diseases

Description of Technology: This application describes a composition and method for treating inflammatory bowel disease or other autoimmune diseases.

The composition utilizes a vector which contains a first promoter which controls the expression of a regulatory transcription factor and a second inducible promoter which controls the expression of the gene of interest. The preferred gene of interest encodes an isoform of TGF–β such as TGF–β_1 or TGF–β_3. The isoform of TGF–β does not have to be hTGF–β and can be a latent or active isoform of TGF–β. The preferred inducible promoter is TRE–CMV which can be induced using doxycycline. The usefulness of the composition for treating autoimmune diseases is demonstrated in the application in a murine model of inflammatory bowel disease in which intestinal inflammation was abrogated by the administration of a plasmid vector encoding active TGF–β. The composition may be administered by a variety of delivery systems and intranasal delivery is exemplified.

Inventors: Warren Strober *et al.* (NIAID).

Patent Status: U.S. Patent Application No. 10/258,109 filed 30 Jun 2003 (HHS Reference No. E–096–2000/0–US–03).

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

Inhibition of Cell Motility, Angiogenesis and Metastasis

Description of Technology: The present invention relates to potent, highly selective antagonists of Grb2 Src homology-2 (SH2) domain binding. Grb2, through its SH2 domain, mediates growth factor driven cell motility *in vitro* and angiogenesis *in vivo*. These synthetic, small molecule antagonists have been shown to block cell motility stimulated by hepatocyte growth factor (HGF), fibroblast growth factor (FGF), epidermal growth factor (EGF), and vascular endothelial cell growth factor (VEGF). They also potently inhibit HGF- and VEGF-stimulated morphogenesis and angiogenesis, respectively, in several model systems. HGF stimulates mitogenesis, motogenesis and morphogenesis in a wide range of cellular targets during development and adulthood, and its signaling pathway is frequently over-activated in human cancers, including colon, gastric, breast, lung, thyroid and renal carcinomas, melanoma, several sarcomas as well as glioblastoma. The ability of HGF to initiate a program of cell dissociation and increased cell motility coupled with increased protease production promotes aggressive cellular invasion and is frequently linked to tumor metastasis.

Metastasis, the primary cause of death in most forms of cancer, is a multistep process whereby cells from the primary tumor spread systemically and colonize distant new sites. Blocking critical steps in this process could potentially inhibit tumor metastasis and dramatically improve cancer survival rates. The small, synthetic Grb2 SH2 domain antagonists described in this invention have been shown to inhibit the induced and spontaneous metastasis of melanoma- and prostate cancer-derived tumor cells in mice. These results establish a critical role for Grb2 SH2 domain-mediated interactions in the metastatic process and support the potential efficacy of this class of compound in reducing the metastatic spread of primary solid tumors in humans.

Applications and Modality: Inhibition of cell motility-dependent processes, including angiogenesis and metastasis, in several types of cancer such as prostate, colon, gastric, breast, lung, thyroid and renal carcinomas, melanoma and various sarcomas.

Market:

An estimated 1,444,920 new cancer cases were diagnosed in the U.S. in 2007.

600,000 deaths caused by cancer in the U.S. in 2006.

Cancer is the second leading cause of death in the U.S.

The cancer drug market will likely double to $50 billion in 2010 from $25 billion in 2006.

Development Status: In vivo and *in vitro* studies have been conducted on this technology.

Inventors: Donald P. Bottaro *et al.* (NCI);

Relevant Publications:

1. Atabey N, Breckenridge D, Yao Z-J, Gao Y, Soon L, Soriano JV, Burke TR, Bottaro DP. Potent blockade of Hepatocyte Growth Factor-stimulated cell motility, invasion, and tubulogenesis by antagonists of Grb2-c-Met interaction. J Biol Chem. 2001 Apr 27;276(17):14308–14314.

2. Shi Z-D, Wei C-Q, Wang X, Lee K, Liu H, Zhang M, Vasselli J, Bottaro DP, Linehan WM, Yang D, Burke TR Jr. Macrocyclization in the design of tetra-tetrapeptide mimetics that display potent inhibition of Grb2 SH2 domain binding in whole cell systems. In: Peptide Revolution: Genomics, Proteomics Therapeutics. Chorev, M and Sawyer, TK, Eds. American Peptide Society, pp 515–517, 2003.

3. Soriano JV, Lui N, Gao Y, Yao Z-J, Ishibashi T, Underhill C, Burke TR Jr, Bottaro DP. Grb2 SH2 domain binding antagonists inhibit angiogenesis *in vitro* and *in vivo*. Mol Cancer Ther. 2004 Oct;3(10):1289–1299.

4. Shi Z-D, Karki RG, Worthy KM, Bindu LK, Dharmawardana PG, Nicklaus MC, Bottaro DP, Fisher RJ, Burke TR Jr. Utilization of a nitrobenzoxadiazole (NBD) fluorophore in the design of a Grb2 SH2 domain binding peptide mimetic. Bioorg Med Chem Lett. 2005 Mar 1;15(5):1385–1388.

5. Kang S-U, Shi, Z-D, Karki RG, Worthy KM, Bindu LK, Dharmawardana PG, Choyke SJ, Bottaro DP, Fisher RJ, Burke TR Jr. Examination of phosphoryl-mimicking functionalities within a macrocyclic Grb2 SH2 domain-binding platform. J Med Chem. 2005 Jun 16;48(12):3945–3948.

6. Shi Z-D, Peruzzi B, Dharmawardana PG, Leech T, Appella E, Worthy KM, Bindu LK, Fisher RJ, Bottaro DP, Burke TR Jr. Synthesis and use of C-terminally biotinylated peptidomimetics with high Grb2 SH2 domain-binding affinity. In: Understanding Biology Using Peptides, Blondelle SE (Ed), American Peptide Society, pp 208–209, 2005.

7. Dharmawardana PG, Peruzzi B, Giubellino A, Bottaro DP. Molecular targeting of Grb-2 as an anti-cancer strategy. Anti-Cancer Drugs 2006 Jan;17(1):13–20.

8. Liu F, Worthy KM, Bindu L, Giubellino A, Bottaro DP, Fisher RJ, Burke TR Jr. Utilization of achiral alkenyl amines for the preparation of high affinity Grb2 SH2 domain-binding macrocycles by ring-closing metathesis. Org Biomol Chem. 2007 Jan 21;5(2):367–372.

9. Giubellino A, Gao Y, Lee S, Lee M-J, Vasselli JR, Medepalli S, Trepel JB, Burke TR Jr, Bottaro DP. Inhibition of tumor metastasis by a Grb-2 SH2 domain binding antagonist. Cancer Res. (Priority Report) 2007 Jul 1;67(13):6012–6016.

Patent Status: PCT Patent Application No. PCT/US2007/078494 filed 14 Nov 2007 (HHS Reference No. E–265–1999/2–PCT–02).

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

Licensing Contact: Adaku Nwachukwu, J.D.; 301–435–5560; *madua@mail.nih.gov*.

Collaborative Research Opportunity: The Urologic Oncology Branch of the National Cancer Institute is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize Grb2 SH2 domain antagonsists as anti-cancer drugs. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Dated: May 15, 2008.

Steven M. Ferguson,

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

[FR Doc. E8–11317 Filed 5–20–08; 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 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 Bacterial Pathogenesis.

Date: May 30, 2008.

Time: 3 p.m. to 5 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: Marian Wachtel, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 3208, MSC 7858, Bethesda, MD 20892, 301–435–1148, *wachtelm@csr.nih.gov.*

This notice is being published less than 15 days prior to the meeting due to the timing limitations imposed by the review and funding cycle.

Name of Committee: Genes, Genomes, and Genetics Integrated Review Group: Molecular Genetics A Study Section.

Date: June 5–6, 2008.

Time: 8 a.m. to 2 p.m.

Agenda: To review and evaluate grant applications.

Place: Renaissance M Street Hotel, 1143 New Hampshire Avenue, NW., Washington, DC 20037.

Contact Person: Michael M. Sveda, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 1114, MSC 7890, Bethesda, MD 20892, 301–435–3565, *svedam@csr.nih.gov.*

This notice is being published less than 15 days prior to the meeting due to the timing limitations imposed by the review and funding cycle.

Name of Committee: Genes, Genomes, and Genetics Integrated Review Group: Genetics of Health and Disease Study Section.

Date: June 9–10, 2008.

Time: 8 a.m. to 12:30 p.m.

Agenda: To review and evaluate grant applications.

Place: Holiday Inn Fisherman's Wharf, 1300 Columbus Avenue, San Francisco, CA 94133.

Contact Person: Cheryl M. Corsaro, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 2204, MSC 7890, Bethesda, MD 20892, (301) 435–1045, *corsaroc@csr.nih.gov.*

Name of Committee: Infectious Diseases and Microbiology Integrated Review Group: Virology—A Study Section.

Date: June 12, 2008.

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

Agenda: To review and evaluate grant applications.

Place: Carlyle Suites Hotel, 1731 New Hampshire Avenue, NW., Washington, DC 20009.

Contact Person: Joanna M. Pyper, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 3198, MSC 7808, Bethesda, MD 20892, (301) 435–1151, *pyperj@csr.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel: Fellowships.

Date: June 12–13, 2008.

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

Agenda: To review and evaluate grant applications.

Place: Churchill Hotel, 1914 Connecticut Avenue, NW., Washington, DC 20009.

Contact Person: John Bishop, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 5180, MSC 7844, Bethesda, MD 20892, (301) 435–1250, *bishopj@csr.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel: Member Conflicts: Psychopathology and Health Psychology.

Date: June 12, 2008.

Time: 12 p.m. to 3 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: Estina E. Thompson, MPH, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 3178, MSC 7848, Bethesda, MD 20892, 301–496–5749, *thompsone@mail.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel: Bacterial Pathogenesis.

Date: June 17, 2008.

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

Agenda: To review and evaluate grant applications.

Place: Georgetown Suites, 1000 29th Street, NW., Washington, DC 20007.

Contact Person: Marian R. Wachtel, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 3208, MSC 7858, Bethesda, MD 20892, 301–435–1148, *wachtelm@csr.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel: Eukaryotic Pathogens.

Date: June 19, 2008.

Time: 12 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: Soheyla Saadi PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 3211, MSC 7808, Bethesda, MD 20892, 301–435–0903, *saadisoh@csr.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel: Developmental Disabilities, Communication and Science Education.

Date: June 23, 2008.

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

Agenda: To review and evaluate grant applications.

Place: St. Gregory Hotel, 2033 M Street, NW., Washington, DC 20036.

Contact Person: Dana Jeffrey Plude, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 3176, MSC 7848, Bethesda, MD 20892, 301–435–2309, *pluded@csr.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel: Drug Therapy.

Date: June 25, 2008.

Time: 11 a.m. to 1 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: Manzoor Zarger PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 6208, MSC 7804, Bethesda, MD 20892, (301) 435–2477, *zargerma@csr.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel: Electromagnetic Devices.

Date: June 25, 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, (Virtual Meeting).

Contact Person: Antonio Sastre, PhD, Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 5215, MSC 7412, Bethesda, MD 20892, 301–435–2592, *sastrea@csr.nih.gov.*

Name of Committee: Center for Scientific Review Special Emphasis Panel: F07 Immunology Fellowships and AREA.

Date: June 26, 2008.

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