the presentation of another participant. Only the presiding officer and panel members may question any person during or at the conclusion of each presentation.

Public hearings under part 15 are subject to FDA's policy and procedures for electronic media coverage of FDA's public administrative proceedings (part 10, subpart C (21 CFR part 10, subpart C)). Under § 10.205, representatives of the electronic media may be permitted, subject to certain limitations, to videotape, film, or otherwise record FDA's public administrative proceedings, including presentations by participants. The hearing will be transcribed as stipulated in § 15.30(b). The transcript of the hearing will be available on the Internet at *http://www.fda.gov/ohrms/dockets*, and orders for copies of the transcript can be placed at the meeting or through the Dockets Management Branch (*see* **ADDRESSES**).

Any handicapped persons requiring special accommodations to attend the hearing should direct those needs to the contact person (*see* **FOR FURTHER INFORMATION CONTACT**).

To the extent that the conditions for the hearing, as described in this notice, conflict with any provisions set out in part 15, this notice acts as a waiver of those provisions as specified in § 15.30(h).

IV. Request for Comments

Interested persons may submit to the Dockets Management Branch (address above) written or electronic notices of participation and comments for consideration at the hearing by April 23, 2002. To permit time for all interested persons to submit data, information, or views on this subject, the administrative record of the hearing will remain open following the hearing until June 21, 2002. Persons who wish to provide additional materials for consideration should file these materials with the Dockets Management Branch (*see* **ADDRESSES**) by June 21, 2002. Two copies of any written comments are to be submitted, except that individuals may submit one copy. Comments are to be identified with the docket number at the heading of this document. Received comments may be seen in the office above between 9 a.m. and 4 p.m., Monday through Friday.

V. References

The following references have been placed on display in the Dockets Management Branch (address above) and may be seen by interested persons between 9 a.m. and 4 p.m., Monday through Friday.

1. Jones, J. K., D. Fife, S. Curkendall et al., ''Coprescribing and Codispensing of Cisapride and Contraindicated Drugs,'' *Journal of the American Medical Association*, 286:1607–1609, 2001.

2. Graham, D. J., C. R. Drinkhard, D. Shatin et al., ''Liver Enzyme Monitoring in Patients Treated With Troglitazone,'' *Journal of the American Medical Association*, 286:831–833, 2001.

3. Smalley, W., D. Shatin, D. K. Wysowski et al., ''Contraindicated Use of Cisapride: Impact of Food and Drug Administration Regulatory Action,'' *Journal of the American Medical Association*, 284: 3036–3039, 2002.

Dated: April 8, 2002.

Margaret M. Dotzel,

Associate Commissioner for Policy.

[FR Doc. 02–9096 Filed 4–12–02; 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 agencies 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.

Methods for Using Modulators of Extracellular Adenosine or an Adenosine Receptor to Enhance Immune Response and Inflammation

Michail V. Sitkovsky, Akio Ohta (NIAID),
DHHS Reference No. E–051–02/1 filed 19 Dec 2001,
Licensing Contact: Cristina Thalhammer-Reyero; 301/496–7736 ext. 263; e-mail: *thalhamc@od.nih.gov.*

Local inflammation processes are crucially important in the host defense against pathogens and for successful immunization because pro-inflammatory cytokines are necessary for initiation and propagation of an immune response. However, normal inflammatory responses are eventually terminated by physiological termination mechanisms, thereby limiting the strength and duration of immune responses, especially to weak antigens. The inventors have shown that adenosine receptors play a critical and non-redundant role in down-regulation of inflammation in vivo by acting as the physiological termination mechanism that can limit the immune response. The adenosine A2a and A3a receptors have been identified as playing a critical role in down-regulation of the immune response during inflammation.

This invention claims methods for inhibiting signaling through the adenosine receptor to prolong and intensify the immune response. The method involves administering either an adenosine-degrading drug or an adenosine receptor agonist. Also claimed in the invention is use of adenosine receptor agonists or adenosine-degrading drugs as vaccine adjuvants and methods for accomplishing targeted tissue damage such as for tumor destruction. This invention is further described in Ohta A et al., ''Role of G-protein-coupled adenosine receptors in downregulation of inflammation and protection from tissue damage,'' Nature 2001 Dec 20–27;414(6866):916–20.

Novel Spore Wall Proteins and Genes From Microsporidia

J. Russell Hayman, John T. Conrad, Theodore Nash (NIAID),
DHHS Reference No. E–125–01/0 filed 04 Dec 2001,
Licensing Contact: Peter Soukas; 301/496–7056 ext. 268; e-mail: *soukasp@od.nih.gov.*

Microsporidia are obligate intracellular organisms that infect a wide variety of animals ranging from insects and fish to mammals, including humans. Of over 1000 microsporidial species identified, at least thirteen are known to infect humans. The species most commonly identified in humans are members of the families Encephalitozoonidae and Enterocytozoonidae. In humans, microsporidiosis is most often found in HIV/AIDS patients and commonly results in severe diarrhea and wasting. However, microsporidiosis also occurs in immunocompetent individuals and common farm animals. The disease is

transmitted via environmentally resistant spores.

This invention claims two spore wall constituents (SWP1 and SWP2) from the microsporidian *Encephalitozoon intestinalis* and the genes from which these two proteins are derived. Further claimed are methods of diagnosing and treating microsporidiosis in a subject. Also claimed are methods for producing an immunoprotective response in a subject. SWP1 is expressed on the surfaces of developing sporonts and SWP2 is expressed on the surfaces of fully formed sporonts. Therefore, they should be exposed to the host cell environment. Based on this theory, antibody responses to SWP1 and SWP2 were addressed in an in vivo mouse model. Immunoprecipitation and Western blot analyses indicated that SWP1 and SWP2 are immunogenic in mouse infections.

This invention is further described in Hayman et al., ''Developmental expression of two spore wall proteins during maturation of the microsporidian *Encephalitozoon intestinalis,*'' *Infect. Immun.* 2001 Nov;69(11):7057–66.

Activated Dual Specificity Lymphocytes and Their Methods of Use

P. Hwu, M.H. Kershaw, and S.A. Rosenberg (NCI),
U.S. Utility Patent Application 09/803,578 filed 09 Mar 2001,
Licensing Contact: Jonathan Dixon; 301/496–7735 ext. 270; *e-mail: dixonj@od.nih.gov.*

While T-cell therapies can work in some patients, the use of these cells to treat cancer and viral diseases is often limited by the poor survival and proliferation of these cells in vivo. Cancer clinical trials have demonstrated that the transferred lymphocytes can recognize tumors in vitro, but human subjects often do not respond to infusion. Gene marking studies have demonstrated that the transferred cells often survive for only short periods of time in vivo, thus limiting their effectiveness.

The current invention relates to a method that using genetic modification to generate leukocytes with multiple specificities. To improve proliferation and activation of the transduced T cells, cell transfer is combined with stimulation using a second antigen. Thus T cells are stimulated through their native T cell receptor, using a powerful immunogen, which facilitates expansion and activation. In experiments, mice receiving alloantigen stimulated cells rejected tumors while mice receiving the unstimulated cells did not reject the tumor cells.

This technology represents a potential therapy for a wide variety of malignancies, and because of the genetic modification used, this therapy will be applicable to patients of any MHC type.

Effect of COMT Genotype on Frontal Lobe Function

Daniel R. Weinberger (NIMH), Michael F. Egan (NIMH), Terry E. Goldberg (NIMH), David Goldman (NIAAA), Joseph H. Callicott (NIMH),
DHHS Reference No. E–174–00/0 filed 11 May 2001,
Licensing Contact: Norbert Pontzer; 301/496–7736, ext. 284; *e-mail: np59n@nih.gov.*

Abnormalities of prefrontal cortical function are prominent features of schizophrenia and have been associated with genetic risk, suggesting that susceptibility genes for schizophrenia may impact on the molecular mechanisms of prefrontal function. A potential susceptibility mechanism involves regulation of prefrontal dopamine, which modulates the response of prefrontal neurons during working memory. The Catechol-o-methyltranferase (COMT) gene contains a G to A mutation which causes a substitution of methionine for valine at codon 158. The met allele has a four fold reduction in enzyme activity which leads to an increase in prefrontal cortical dopamine levels. NIH investigators observed that the functional polymorphism in the gene encoding COMT is associated with variations in executive function and efficiency of working memory in normal controls and schizophrenic patients.

The invention provides a method of detecting impaired prefrontal cognitive function in a subject individual comprising determining the individual's COMT genotype and associating a high activity val allele with impaired prefrontal cognitive function and a low activity met allele with enhanced prefrontal cognitive function. The COMT genotype can be determined using a relatively simple restriction fragment length polymorphism analysis after PCR amplification of the polymorphic region of exon four since the *met* substitution introduces a NlaIII restriction site into the allele. Clinical medical tests to determine prognosis in schizophrenia and other conditions associated with the polymorphism would thus be possible. The invention also provides for treating patients with COMT inhibitors after tests that predict the response of a patient with schizophrenia, other neurological disorders or aging related declines in cognition to administration of a COMT inhibitor.

Identification of a Transforming Fragment of Herpes Simplex Type 2 and Detection thereof in Clinical Specimen

Joseph A. DiPaolo (NCI), Allegria Dessous-Elbaz, Francois Coutlee,
U.S. Provisional Application SN 60/020,957 filed 01 Jul 1996; PCT Application No. PCT/CA97/00470 filed 30 Jun 1997; U.S. Patent Application SN 09/202,918 filed 23 Dec 1998; Canadian Patent Application SN 2,259,657 filed 23 Dec 1998,
Licensing Contact: Uri Reichman; 301/496–7736 ext. 240; e-mail: *reichmau@od.nih.gov.*

The present invention relates to novel diagnostic and therapeutic methods for Herpes Simplex Virus Type 2 (HSV–2). HSV–2 infects approximately one fifth of adults in the United States and is the most common cause of genital ulceration. The invention relates to the detection of HSV–2 based on a transforming nucleic acid sequence and its protein product. This DNA sequence harbors the potential to induce the tumorigenic transformation of normal cells in in vitro and in vivo assays and thus will be useful as a means of prognostic evaluation in predicting the development of genital or cervical cancer. Current HSV–2 diagnostic tests relying on tedious viral culture and/or immunoassays do not have the sensitivity and the specificity essential for diagnosis. Using PCR, the current invention will provide a superior method for viral detection and subtyping. In addition the in vivo administration of the antisense primers corresponding to the transforming DNA sequence and the use of antibodies against the protein product can be powerful therapeutic treatments against HSV–2.

Mitochondrial Topoisomerase I

Yves Pommier and Hong-Liang Zhang (NCI),
DHHS Reference No. E–099–01/0 filed 16 Feb 2001,
Licensing Contact: Matthew Kiser; 301/496–7056 ext. 223; e-mail: *kiserm@od.nih.gov.*

This invention describes a gene that codes for a human topoisomerase that exclusively acts on mitochondrial DNA, and is the first described mitochondrial topoisomerase. Since a number of diseases are caused by mitochondrial malfunction, this gene could form the basis of a number of different therapies. For instance, mitochondrial malfunctions could lead to disturbances in energy metabolism and programmed cell death (apoptosis). This

mitochondrial gene product could thus lead to new diagnoses and therapies centered on apoptosis, which is a critical event in cancer and autoimmune disorders.

In addition to the gene sequence, the patent application covers the encoded protein, protein fragments, monoclonal and polyclonal antibodies, and methods to alter the level of this gene's expression. Also included in the claims are methods to identify activators or inhibitors of the topoisomerase enzyme. NIH invites commercial partners to apply for either an exclusive or non-exclusive license to this technology. We also invite companies who may be interested in commercializing the topoisomerase or the antibodies for research reagent use.

This abstract replaces one published in the **Federal Register** on January 28, 2002 (67 FR 3905).

Dated: April 3, 2002.

Jack Spiegel,

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

[FR Doc. 02–9094 Filed 4–12–02; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Mental Health; Notice of Closed Meeting

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 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: National Institute of Mental Health Special Emphasis Panel.

Date: April 8, 2002.

Time: 3:30 p.m. to 4 p.m.

Agenda: To review and evaluate grant applications.

Place: Bethesda Holiday Inn, 8120 Wisconsin Avenue, Bethesda, MD 20814.

Contact Person: Michael J. Kozak, PhD, Scientific Review Administrator, Division of Extramural Activities, National Institute of Mental Health, NIH, Neuroscience Center, 6001 Executive Blvd., Room 6138, MSC 9608, Bethesda, MD 20892–9608, 301–443–6471, kozakm@mail.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.

(Catalogue of Federal Domestic Assistance Program Nos. 93.242, Mental Health Research Grants; 93.281, Scientist Development Award, Scientist Development Award for Clinicians, and Research Scientist Award; 93.282, Mental Health National Research Service Awards for Research Training, National Institutes of Health, HHS)

Dated: April 5, 2002.

Anna Snouffer,

Deputy Director, Office of Federal Advisory Committee Policy.

[FR Doc. 02–9090 Filed 4–12–02; 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 Meeting

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 meeting.

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

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

Date: April 11, 2002.

Time: 3 p.m. to 5 p.m.

Agenda: To review and evaluate grant applications and/or proposals.

Place: NIH, Rockledge 2, Bethesda, MD 20892, (Telephone Conference Call).

Contact Person: Robert T. Su, PhD, Scientific Review Administrator, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 4134, MSC 7802, Bethesda, MD 20892, (301) 435–1195.

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.

(Catalogue of Federal Domestic Assistance Program Nos. 93.306, Comparative Medicine, 93.306; 93.333, Clinical Research, 93.333, 93.337, 93.393–93.396, 93.837–93.844, 93.846–93.878, 93.892, 93.893, National Institutes of Health HHS)

Dated: April 8, 2002.

Anna Snouffer,

Deputy Director, Office of Federal Advisory Committee Policy.

[FR Doc. 02–9091 Filed 4–12–02; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Prospective Grant of Exclusive License: Glycoprotein Hormone Superagonists

AGENCY: National Institutes of Health, Public Health Service, HHS.

ACTION: Notice.

SUMMARY: This is notice, in accordance with 35 U.S.C. 209(c)(1) and 37 CFR 404.7(a)(1)(i), that the National Institutes of Health (NIH), Department of Health and Human Services, is contemplating the grant of an exclusive license worldwide to practice the invention embodied in U.S. Patent Application Serial No. 09/185,408 filed May 6, 1996, and U.S. Patent Application Serial No. 10/057,113 filed January 24, 2002, entitled ''Glycoprotein Hormone Superagonists,'' to Trophogen, having a place of business in the state of Maryland. The field of use may be limited to the treatment of human infertility, Graves Disease, thyroid cancer, and contraceptives. The United States of America is the assignee of the patent rights in this invention. This announcement replaces three previous notices to grant an exclusive license to this technology: July 19, 1999 (64 FR 38685), February 7, 2000 (65 FR 5878–5879), and May 15, 2001 (66 FR 26871).

DATES: Only written comments and/or application for a license, which are received by the NIH Office of Technology Transfer on or before June 14, 2002, will be considered.

ADDRESSES: Requests for a copy of the patent applications, inquiries, comments and other materials relating to the contemplated license should be directed to: Marlene Shinn, Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, MD 20852–3821; Telephone: (301) 496–7056, ext. 285; Facsimile: (301) 402–0220; E-mail: *MS482M@NIH.GOV.*

SUPPLEMENTARY INFORMATION: This invention relates generally to modified glycoprotein hormones and specifically to modifications to a human