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.

Influenza DNA Vaccine That Protects Against Lethal H5N1 Challenge

Description of Technology: Concerns about a potential influenza pandemic and its prevention dominate health news, with new cases of bird (avian) influenza (H5N1 strain) cases being reported on a daily basis. Vaccination is one of the most effective ways to minimize suffering and death from influenza. Currently, there is not an effective vaccine to protect against the H5N1 strain, thought to be a leading pandemic candidate. The technology described here relates to a DNA influenza vaccine encoding the matrix 2 (M2) protein, which is highly conserved among different influenza strains. The M2 component can be used either alone or in combination with other influenza components. Specifically, mouse studies showed that the use of M2 from H1N1 strain protected against a lethal challenge with H5N1 strain. The current technology offers several advantages over traditional influenza vaccine approaches, including (a) ease and speed of production without need for eggs, (b) no surveillance to determine dominant strain(s), and (c) no potential for antigenic shift as observed for the components (HA and NA) of current influenza vaccines.

Inventors: Suzanne L. Epstein *et al.* (CBER/FDA).

Patent Status: U.S. Provisional Application No. 60/785,152 filed March 27, 2006 (HHS Reference No. E–076–2006/0–US–01).

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

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

Collaborative Research Opportunity: The Food and Drug Administration's Center for Biologics Evaluation and Research (CBER) is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact Beatrice Droke at 301/827–7008 or *bdroke@oc.fda.gov* for more information.

Methods for Inhibiting HIV and Other Viral Infections by Modulating Ceramide Metabolism

Description of Technology: This invention provides methods of inhibiting or preventing HIV–1 infections by inducing either the de novo biosynthesis of ceramide, or by activating enzymes (*e.g.*, sphingomyelinase) involved in the generation of ceramide at the plasma membrane, or by direct incorporation of exogenous ceramide into target cell membranes. The invention describes methods for administration a retinamide compound particularly an N-(aryl) retinamide compound such as N-(4-hydroxyphenyl) retinamide (4–HPR) resulting in increased plasma membrane ceramide levels, which results in the inhibition of HIV–1 infection in monocyte/macrophages by perturbing membrane organization. In addition, because of its low toxicity in non-tumor cells, 4–HPR and related compounds are particularly suitable for long-term preventative or therapeutic administration to subjects suffering from an HIV infection or who are at risk of contracting an HIV infection. Thus, this invention provides a novel means of treating or inhibiting HIV and other viral infections by administering a retinamide compound to a patient suffering from or susceptible to such a viral infection.

Inventors: Robert P. Blumenthal *et al.* (NCI).

Publications:

1. C.M. Finnegan *et al.*, ''Ceramide, a target for antiretroviral therapy,'' Proc. Natl. Acad. Sci. USA. (2004 Oct 26) 101(43):15452–15457.

2. C.M. Finnegan and R. Blumenthal, ''Fenretinide inhibits HIV infection by promoting viral endocytosis,'' Antiviral Res. (2006 Feb) 69(2):116–123.

Patent Status: U.S. Provisional Application No. 60/528,411 filed December 9, 2003 (HHS Reference No. E–265–2003/0–US–01); PCT Application No. PCT/US2004/41512 filed December 9, 2004, which published as WO 2005/072091 on August 11, 2005 (HHS Reference No. E–265–2003/0–PCT–02).

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

Licensing Contact: Sally Hu, PhD., M.B.A.; 301/435–5606; *hus@mail.nih.gov*

Collaborative Research Opportunity: The National Cancer Institute, Center for Cancer Research Nanobiology Program, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the clinical potential of sphingolipid-based antiviral therapies. Please contact Melissa Maderia at *maderiam@mail.nih.gov* or by phone at 301/846–5465 for more information.

Methods and Compositions for the Inhibition of HIV–1 Replication

Description of Technology: This invention relates to methods and compositions for the attenuation of HIV–1 replication in human cells, and especially in CD4+ human peripheral blood mononuclear cells, such as blood monocyte-derived macrophages by targeting a host cell protein. HIV–1 infected macrophages typically resist cell death, support viral replication, and facilitate HIV–1 transmission. We found that the gene encoding cyclin-dependent kinase inhibitor 1A (CDKN1A) is consistently expressed following virus binding, and re-expressed at the peak of HIV–1 replication. The protein encoded by this gene, also known as p21, is associated with cell cycle regulation, anti-apoptotic response and cell differentiation. Increased levels of p21 may enhance survival and long-term persistence of HIV–1 infected macrophages. Following identification of p21 as a candidate molecule in facilitating viral replication, efforts to curtail its role were investigated as a mode of blunting infection in macrophages. RNA interference (siRNA) represents a tool to regulate gene expression and when siRNA specific for p21 or p21-specific oligonucleotides were transfected into primary macrophages to silence the expression of p21, HIV infection was aborted, thereby validating p21 as a cellular factor essential to productive HIV infection in this population. Extending these observations, a

pharmacologic agent known to influence p21 expression, the synthetic triterpenoid and peroxisome proliferator-activated receptor gamma (PPARg) ligand, 2-cyano-3,12-dioxooleana-1,9-dien-28-oic acid (CDDO) or its derivative di-CDDO, was shown to moderate virally-induced p21 expression and concurrently dampen HIV infection. CDDO is part of a class of synthetic triterpenoids based on natural products resembling steroids in their biogenesis and in their pleiotropic actions. A newly developed CDDO derivative, which is orally bioavailable, also suppresses HIV. These results, coupled with the evidence that macrophage p21 is a requisite macrophage facilitator of viral replication, intensify the interest to further develop these compounds as antiretroviral agents. The anti-retroviral effect of CDDO was evident when peripheral blood mononuclear cells (PBMC) were infected with a T-tropic (X4) or dual tropic viral (R5X4) strain of HIV–1. These studies suggest that these triterpenoids may aid in the control of retroviral replication. Neither p21 oligonucleotides nor CDDO were toxic to the cultured macrophages or peripheral blood mononuclear cells. Thus, p21 inhibitors could be safe and effective anti-HIV therapeutic candidates to be used independently and/or in conjunction with current anti-retroviral therapy. In this regard, CDDO will be entered into human trials for the first time in the near future for its anti-cancer indications, thereby determining its maximally tolerated dose for use in subsequent HIV/AIDS clinical trials. Current anti-retroviral therapy, often characterized by high toxicity and the emergence of drug resistant virus strains, may be augmented through the identification of these and other new anti-viral agents targeting host cellular molecules less prone to mutational events.

Inventors: Sharon M. Wahl, Nancy Vazquez-Maldonado, Teresa Greenwell-Wild (NIDCR).

Publications:

1. S.M. Wahl *et al.*, ''HIV accomplices and adversaries in macrophage infection,'' J. Leukoc. Biol. 2006, in press.

2. N. Vazquez *et al.*, ''Human immunodeficiency virus type 1-induced macrophage gene expression includes the p21 gene, a target for viral regulation,'' J. Virol. (2005 Apr) 79(7):4479–4491.

Patent Status: U.S. Provisional Application No. 60/516,794 filed November 4, 2003 (HHS Reference No. E–114–2003/0–US–01); PCT Application No. PCT/US2004/36492 filed November 3, 2004, which published as WO 2005/046732 on May 26, 2005 (HHS Reference No. E–114–2003/0–PCT–02)

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

Licensing Contact: Sally Hu, PhD., M.B.A.; 301/435–5606; *hus@mail.nih.gov*

Collaborative Research Opportunity: The National Institute of Dental and Craniofacial Research, Oral Infection and Immunity Branch, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact David W. Bradley, PhD., at *bradleyda@nidcr.nih.gov* or by phone at 301/402–0540 for more information.

Dated: May 18, 2006.

David R. Sadowski,

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

[FR Doc. E6–8176 Filed 5–25–06; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Center on Minority Health and Health Disparities; Notice of 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 a meeting of the National Advisory Council on Minority Health and Health Disparities.

The meeting will be open to the public as indicated below, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the 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 Advisory Council on Minority Health and Health Disparities.

Date: June 13, 2006.

Closed: 8:30 a.m. to 10 a.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, Two Democracy Plaza, 6707 Democracy Boulevard, Suite 800, Bethesda, MD 20892.

Open: 10 a.m. to 5 p.m.

Agenda: The agenda will include Opening Remarks, Administrative Matters, Director's Report, NCMHD, IC Strategic Plan Report, NIH Minority Research Training Programs Update, NCMHD Program Highlights, and other business of the Council.

Place: National Institutes of Health, Two Democracy Plaza, 6707 Democracy Boulevard, Suite 800, Bethesda, MD 20892.

Contact Person: Donna Brooks, Asst. Director for Administration, National Center on Minority Health and Health Disparities, National Institutes of Health, 6707 Democracy Blvd., Suite 800, Bethesda, MD 20892. 301–435–2135. *brooksd@ncmhd.nih.gov.*

Any interested person may file written comments with the committee by forwarding the statement to the Contact Person listed on this notice. The statement should include the name, address, telephone number and when applicable, the business or professional affiliation of the interested person.

Dated: May 18, 2006.

Anna Snouffer,

Acting Director, Office of the Federal Advisory Committee Policy.

[FR Doc. 06–4893 Filed 5–25–06; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Allergy and Infectious Diseases; 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: Allergy, Immunology, and Transplantation Research Committee, Allergy, Immunology and Transplantation Research Committee (AITRC).

Date: June 12, 2006.

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