epithelial cells and blocked adherence of P. aeruginosa on epithelial cells. When the chimeric protein was injected into rabbits, the rabbits produced antibodies that blocked bacterial adherence and neutralized the cell killing activity of native exotoxin A.

A Plasmid for Expression of a More Soluble Form of HIV Integrase Protein in E. coli

Robert Craigie (NIDDK)
DHHS Reference No. E–110–01/0
Licensing Contact: Sally Hu; 301/496–7056 ext. 265; e-mail: hus@od.nih.gov

The invention describes a plasmid that provides a convenient method for producing large quantities of integrase protein. This integrase protein is more soluble because amino acid residue Phe185 is changed to Lsy. This change does not affect the in vitro activity of the protein, but the improved solubility facilitates large-scale purification and handling. Since HIV integrase is a candidate target for antiviral drugs and an assay system or a source of HIV integrase is required to identify lead compounds, this invention could be very useful for an efficient means of producing integrase protein on a large scale. The integrase protein could be used in screening for integrase inhibitors that could be developed as anti-HIV drugs. This invention is available for licensing through a Biological Materials License, as no patent application exists.

Benzoylalkylindolepyridinium Compounds and Pharmaceutical Compositions Comprising Such Compounds

William G. Rice, Mingjun Huang, Robert W. Buckheit, Jr., David G, Covell, Grzegorz Czerwinski, Christopher Michejda, and Vadim Makarov (NCI)
DHHS Reference Nos. E–278–98/0 and E–278–98/1, filed Dec 18, 2000
Licensing Contact: Sally Hu; 301/496–7056 ext. 265; e-mail: hus@od.nih.gov

The present invention provides novel antiviral compounds active against HIV. These compounds, referred to as benzoylalkylindolepyridinium compounds (BAIPs) are effective against HIV isolates that have developed mutations rendering conventional drugs ineffective. BAIPs apparently do not require intracellular phosphorylation nor bind to the reverse transcriptase (RT) active site, which distinguishes their mechanism of action from the dideoxynucleoside (ddN) and acyclic nucleoside phosphonate (ANP) nucleoside analog drugs. ddN and ANP have proven clinically effective against limiting human immunodeficiency virus (HIV) infection, but resistance rapidly emerges due to mutations in and around the RT active site. The BAIPs also may be distinguished from non-nucleoside reverse transcriptase inhibitors (NNRTIs), in part because the BAIPs bind to a different site on the RT enzyme. The usage of NNRTIs is limited by the rapid emergence of resistant strains also. Moreover, unlike the NNRTIs, BAIPs of the present invention have been shown to be effective against HIV–1, HIV–2 and simian immunodeficiency virus (SIV) proliferation. Thus, BAIPs are broadly antiviral, non-nucleoside reverse transcriptase inhibitors (BANNRTIs).

This abstract modifies an abstract for this technology published in the **Federal Register** on Tuesday, February 13, 2001 (66 FR 10027).

Dated: May 17, 2001.

Jack Spiegel,

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

[FR Doc. 01–13345 Filed 5–25–01; 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, DHHS.

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.

Combined Inhibition of Phosphodiesterase-4 (PDE–4) and Phosphodiesterase-3 (PDF–3) as a Therapy for Th1 Mediated Autoimmune Diseases

Dr. Bibiana Bielekova et al. (NINDS)
DHHS Reference No. E–077–00/0, filed Dec 22 2000
Licensing Contact: Marlene Shinn; 301/496–7056 ext. 285; e-mail: shinnm@od.nih.gov

Hyperactive Th1-mediated immune responses are thought to be involved in the pathogenesis of many autoimmune diseases, including rheumatoid arthritis, diabetes, inflammatory bowel disease, vitiligo, and multiple sclerosis among others. Immune cells are known to produce primarily two classes of phosphodiesterases (PDE), the PDE4 and the PDE3 classes. Inhibitors of these PDEs have been shown to down-regulate the expression or production of Th1 cytokines and have either no effect or augment the production of Th2 cytokines, therefore making them good candidates for the treatment of Th1-mediated autoimmune diseases.

The NIH announces a new technology wherein PDE–4 and PDE–3 inhibitors are used in combination and a synergistic enhancement of therapeutic activity is achieved. This results in a more potent immunomodulatory effect on the immune cells and could lead to the administration of lower dose rate of the inhibitors. This new form of treatment will alleviate side effects through the use of a lower dose rate for each and will make for a more effective therapy.

Determination of AM-Binding Proteins and the Association of Adrenomedullin (AM) Therewith

F. Cuttitta et al. (NCI)
DHHS Reference No. E–256–99/1 filed, Sep 08 2000 (Note: This invention is related to E–206–95/3, filed Aug 18 1996, the disclosure of which is incorporated herein.)
Licensing Contact: Matthew Kiser; 301/496–7056 ext. 224; e-mail: kiserm@od.nih.gov

The present invention provides methods for the isolation, identification, and purification of adrenomedullin (AM)-binding proteins. Methods for utilizing the purified AM-binding proteins, or functional portions thereof, to diagnose, treat, and monitor AM-related diseases are described. A second aspect of this technology discloses the identification and isolation of a novel complex between AM and a specific AM-binding protein 1 (AMBP–1), designated factor H (fH). The identification of small molecule

antagonist, which down-regulate the function of AM, factor H, and the AM/fH complex has been achieved. Collectively, the invention provides methods for treating conditions such as cancer or diabetes, via antibodies and small molecule antagonists.

Adrenomedullin (AM) is expressed in human cancer cell lines of diverse origin and functions as a universal autocrine growth factor, driving neoplastic proliferation. Experimental models for use in identifying the role of AM in pancreatic physiology have been validated and are available for licensing. The interesting observations show that AM inhibits insulin secretion in a dose-dependent manner. Further experiments have shown that a neutralizing antibody up-regulates insulin release at least five-fold, an effect that is reversed with the addition of synthetic AM.

Novel Inhibitors of p53 for Treatment of Neurodegenerative Disorders, Myocardial Infarction and Other Tissue Insults

Nigel H. Greig, et al. (NIA)

Serial No. 60/216,388, filed July 6, 2000

Licensing Contact: Norbert Pontzer; 301/496–7736, ext. 284; e-mail: pontzern@nih.gov

The tumor suppressor protein p53 is a key modulator of stress responses, and activation of p53 precedes apoptosis (programmed cell death) in many cell types. Conditions that stress tissue, such as deposition of amyloid b-peptide, may thus cause tissue degeneration through activation or up-regulation of p53. This invention provides novel inhibitors of p53 and methods of using these inhibitors for the prevention or treatment of the stress related tissue degeneration observed in Alzheimer's disease, myocardial infarction and stroke. In vitro and ex vivo studies demonstrated that p53 inhibition protected nerve cells from toxic insults that otherwise induced programmed cell death. In a rat model of stroke, p53 inhibition produced a 50% reduction in stroke volume.

Dated: May 17, 2001.

Jack Spiegel,

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

[FR Doc. 01–13346 Filed 5–25–01; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Heart, Lung, and Blood Institute; 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 Heart, Lung, and Blood Institute Special Emphasis Panel NHLBI, National Research Service Training, SEP (K's).

Date: June 28–29, 2001.

Time: 7 PM to 5 PM.

Agenda: To review and evaluate grant applications.

Place: Chevy Chase Holiday Inn, 5520 Wisconsin Ave., Chevy Chase, MD 20815.

Contact Person: Roy L. White, Phd, Review Branch, NIH, NHLBI, Rockledge Building II, 6701 Rockledge Drive, Room 7196, Bethesda, MD 20892, 301–435–0291.

(Catalogue of Federal Domestic Assistance Program Nos. 93.233, National Center for Sleep Disorders Research; 93.837, Heart and Vascular Diseases Research; 93.838, Lung Diseases Research; 93.839, Blood Diseases and Resources Research, National Institutes of Health, HHS)

Dated: May 22, 2001.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 01–13335 Filed 5–25–01; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Heart, Lung, and Blood Institute; 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 Heart, Lung, and Blood Institute Special Emphasis Panel ACTION–A–CHF trial Investigating Outcomes, of Exercise Training.

Date: June 19, 2001.

Time: 2 to 5.

Agenda: To review and evaluate grant applications.

Place: 5520 Wisconsin Avenue, Chevy Chase, MD 20815.

Contact Person: Joyce A. Hunter, PhD, Review Branch, Room 7194, Division of Extramural Affairs, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, MD 20872.

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. 93.233, National Center for Sleep Disorders Research; 93.837, Heart and Vascular Diseases Research; 93.838, Lung Diseases Research; 93.839, Blood Diseases and Resources Research, National Institutes of Health, HHS)

Dated: May 22, 2001.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 01–13336 Filed 5–25–01; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Heart, Lung, and Blood Institute; 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.