appears particularly important to host response in parasitic infection.

The present invention provides for the prevention and treatment of infectious diseases through modification of immune response(s), in particular, to the involvement of GTPase molecule(s) in such immune responses to infectious disease (such as parasitic (*e.g.,* protozoan) disease).

Method of Treating a Viral Infection Using Antagonists or Macrophage Colony Stimulating Factor (M–CSF)

Clouse-Strebel *et al.* (FDA)
DHHS Reference No. E–255–99/0 filed 08 Nov 1999
Licensing Contact: J.P. Kim; 301/496–7056 ext. 264; e-mail: kimj@od.nih.gov

Colony stimulating factors (CSF's) are a class of proteins that stimulate growth and development of bone marrow progenitor cells into mature cells, such as granulocytes, macrophages, megakaryocytes, erythrocytes, lymphocytes, and mast cells. One of these factors is macrophage colony stimulating factor (M-CSF), a homodimeric glycoprotein with subunits linked by disulfide bonds. M-CSF is also known as CSF–1, CSF–69, LSF, MGF, and CSF-HU.

The present invention provides for a method for treating a viral infection, such as HIV–1 and HIV–2, using an amount of an antagonist of M-CSF sufficient to inhibit replication of the virus, either administered alone or in combination with another anti-viral agent.

S-Nitrosoglutathione as a Protease Inhibitor for the Treatment of AIDS and Neurodegenerative Disorders

Chuang C. Chiueh (NIMH), Sang Y. Lee (NIMH), David A. Davis (NCI), Robert Yarchoan (NCI)
DHHS Reference No. E–008–00/0 filed 01 Nov 1999
Licensing Contact: J.P. Kim; 301/496–7056 ext. 264; e-mail: kimj@od.nih.gov

The human immunodeficiency virus (HIV) is the causative agent of acquired immunodeficiency syndrome (AIDS). Over the years, drug-resistance has been a critical factor contributing to the gradual loss of clinical benefit to treatments for HIV infection. There has been great concern regarding this apparent growing resistance of HIV strains to current therapies. Accordingly, there is a great need for new effective HIV therapeutics.

The present invention provides for the use of nitrosylating compounds, such as S-Nitrosoglutathione and derivatives thereof (for example, as an HIV–1 protease inhibitor) for the treatment of AIDS and neurodegenerative disorders.

Enhancement of Hematopoietic Cells

William J. Murphy (NCI), Susan M. Richards (NCI), Dan L. Longo (NIA)
DHHS Reference Nos. E–247–99/0 filed 21 Jan 1997 and E–247-99/1 filed 20 Jan 1998 (PCT/US98/00887)
Licensing Contact: J.P. Kim; 301/496–7056 ext. 264; e-mail: kimj@od.nih.gov

The present invention provides a method for enhancing hematopoiesis by contacting hematopoietic stem or progenitor cells with a composition containing prolactin, preferably recombinant prolactin. Stimulation of hematopoiesis can serve to replace hematopoietic cells. The invention further provides a method for treating an animal to improve hematopoiesis or prevent hematopoietic-suppression by administering a pharmaceutically acceptable composition containing prolactin. The invention further relates to a composition comprising a cytokine that can enhance hematopoiesis and prolactin, and a composition comprising a therapeutic that can cause hematopoietic-suppression and a prolactin.

Dated: July 19, 2000.

Jack Spiegel,

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

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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.

Discovery of a Novel Human Aminopeptidase Which May Regulate Cleavage and Shedding of the Human Type-I Tumor Necrosis Factor Receptor

Stewart J. Levine (NHLBI)
DHHS Reference No. E–003–00/0 filed 28 Feb 2000
Licensing Contact: Richard U. Rodriguez; 301/496–7056 ext. 287; e-mail: rodrigur@od.nih.gov

Cytokines are a large and diverse group of molecules which mediate interactions between cells. Aberrant regulation of cytokine signaling results in a wide variety of hyper-inflammatory, autoimmune and immune-deficiency pathological conditions. Tumor necrosis factor-α (TNF-α) is a multifunctional cytokine mediating pleiotropic biological functions in both healthy and disease states. TNF-α has been shown to have a role in the following activities: Destroying tumors, mediating responses to tissue injury, protecting hosts from infections by various microorganisms and activating numerous genes, including NF-κB and AP–1. TNF-α has also been implicated in the pathogenesis of a variety of diseases and disorders. The present invention provides compositions and methods related to regulation of cytokine signaling through the TNF-α pathway. Specifically, the invention provides a novel gene, polypeptide and related compositions and methods for the regulation of TNF Type-I receptor ectodomain shedding. It is contemplated that the compositions and methods of this invention will find use in therapeutics for the treatment of diseases and disorders of the immune system.

Amplification and Overexpression of Multiple Genes at 17q23 in Breast Cancer

Anne H Kallioniemi, Olli P Kallioniemi, Juha T Kononen, Maarit Barlund (NHGRI)
DHHS Reference No. E–051–00/0 filed 28 Jan 2000
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This invention pertains to gene amplification and its role in the progression and initiation of many solid

tumors, including breast cancer. Amplification is a common mechanism for upregulation of critical genes involved in cancer development and progression. Discovery of HER–2 oncogene amplification in breast cancer has led to a specific therapy for breast cancer patients with an activated HER–2 gene. Chromosomal region 17q23 is frequently amplified in breast cancer but the genes involved in this amplification are not yet known. Amplification of four previously known genes, S6K, TBX2, PAT1, RAD51C, has been identified in breast cancer cell lines and primary breast tumors. The amplification in cell lines leads to overexpression at the mRNA level. Thus, these genes represent putative targets for the 17q23 amplification and their upregulation may contribute to the genesis and progression of breast cancer.

Inhibition of Cell Motility

Donald P. Bottaro, Terrence R. Burke, Jr., Zhu-Jun Yao, Nese S. Atabey, Diane E. Breckenbridge, Yang Gao (NCI)

DHHS Reference No. E–265–99/0 filed 22 Oct 1999

Licensing Contact: Richard U. Rodriguez; 301/496–7056 ext. 287; e-mail: rodrigur@od.nih.gov

The present invention relates to a method of inhibiting cell motility induced by hepatocyte growth factor (HGF) and treating various diseases in a mammal. HGF stimulates mitogenesis, motogenesis and morphogenesis in a wide range of cellular targets including epithelial and endothelial cells, hematopoietic cells, neurons, melanoytes, and hepatocytes. These pleiotropic effects play important roles during development and tissue regeneration, but they are also implicated in several human cancers, including colon, breast, lung, thyroid and renal carcinomas, several sarcomas and gliolastomas. 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 linked to tumor metastasis. The methods of the present invention employ compounds, *e.g.,* phosphotyrosine mimetics, to inhibit cell motility. A key advantage of this invention is that the peptides are free of cytotoxicity. Further development and use of this invention could serve a serious public need.

Fibroblast Growth Factor-5 (FGF–5) Is a Tumor Associated T-cell Antigen for Human Renal Cell Cancer and Other Adenocarcinomas

Ken-ichi Hanada and James C. Yang (NCI)

DHHS Reference No. E–243–99/0 filed 02 Oct 1999

Licensing Contact: Elaine Gese; 301/496–7056 ext. 282; e-mail: gesee@od.nih.gov

Renal cell carcinoma (RCC) is a form of kidney cancer caused when cells in the lining of the renal tubule undergo cancerous changes. The inventors have shown that fibroblast growth factor-5 (FGF–5) is a tumor associated antigen (TAA) for RCC and cancers of the breast and prostate. TAAs can be used to stimulate cytotoxic T-lymphocytes (CTL) which can be directed against specific tumor cells. This can be accomplished in at least two ways: (1) Activating a patient's immune system by administering a vaccine containing the TAA, or (2) by removing a patient's lymphoid cells, activating these cells ex vivo and then reintroducing these activated cells back into the patient to attack the tumor cells. The invention provides for methods of treating RCC and other adenocarcinomas with FGF–5 using the aforementioned approaches.

Genetic System in Yeast for Functional Identification of Human p53 Mutations

Michael A. Resnick, Alberto Inga (NIEHS)

DHHS Reference No. E–183–99/0 filed 30 Jul 1999

Licensing Specialist: Vasant Gandhi; 301/406–7056 ext. 224; e-mail: gandhiv@od.nih.gov

The tumor suppressor gene p53, a key regulator of cellular mechanisms that maintain genome integrity, is the most commonly inactivated gene target associated with neoplastic transformation. About 50% of all human tumors express a mutated form of p53 and more than 80% of these mutations are missense, leading to single amino acid changes. This invention relates to human p53 mutants and identification methods using screening assays in the yeast Saccharomyces cerevisiae to functionally categorize expressed p53 mutant proteins. Additionally, the invention relates to methods of detecting or generating novel human p53 mutations with properties that can include toxicity in yeast and growth suppression in human cells, enhanced or reduced transactivation relative to wild type p53, altered promoter selectivity, and reactivation of common tumor mutations for the transactivation function of major p53 downstream genes. The invention also provides for screening of genetic factors, peptides and chemicals that mimic the toxic or supertransactivating mutations or inhibit p53 function.

Dated: July 19, 2000.

Jack Spiegel,

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

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DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Consensus Development Conference on Antenatal Corticosteroids Revisited: Repeat Courses

Notice is hereby given of the National Institutes of Health (NIH) Consensus Development Conference on ''Antenatal Corticosteroids Revisited: Repeat Courses,'' which will be held August 17–18, 2000, in Masur Auditorium of the NIH Clinical Center, 9000 Rockville Pike, Bethesda, Maryland, 20892. The conference begins at 8 a.m. on August 17, and at 8:30 a.m. on August 18 and is open to the public.

Preterm delivery is a major cause of death and illness in infants. Corticosteroid treatment of pregnant women delivering prematurely was first introduced in 1972 to enhance fetal lung maturity. Subsequent research has focused on the ability of glucocorticoids to reduce mortality and brain injury in preterm neonates.

In 1994 the National Institutes of Health sponsored a Consensus Development Conference on the Effect of Corticosteroids for Fetal Maturation on Perinatal Outcomes to assess the effectiveness of antenatal glucocorticoid therapy. The Consensus Panel concluded, in part, that giving corticosticoids to pregnant women at risk for preterm delivery reduces the risk of death, respiratory distress syndrome, and intraventricular hemorrhage in preterm infants.

The 1994 panel noted that optimal benefit of antenatal corticosteroid therapy last 7 days. The panel also noted that the potential benefits and risk of repeated administration of antenatal corticosteroids 7 days after the initial course are unknown and called for additional research on this issue.

The NIH is organizing this 1½ day conference to present research on repeat courses of antenatal corticosteroid therapy. After a day of presentations and audience discussion, an