invention proposes a method to increase GM–CSF levels in a treated subject, and this increase is achieved by inhibiting the degradation of GM–CSF messenger RNA (mRNA). Tristetraprolin (TTP) is one member of a family of cys-cys-cys-his (CCCH) zinc finger proteins, and it is a factor that binds to and causes the instability of GM–CSF mRNA. Methods are provided for the development of screening assays for molecules that inhibit the binding of TTP and its related proteins to GM–CSF mRNA, or otherwise inhibit the effect of TTP to promote breakdown of the mRNA, leading in turn to increased mRNA stability and enhanced production of GM–CSF. Compounds identified by such screens, and their derivatives, could be useful in treating granulocytopenia from whatever cause.

Additional information about this technology may be found in the following research articles:

Carballo, E, Lai, WS and Blackshear, PJ. Evidence that tristetraprolin (TTP) is a physiological regulator of granulocyte-macrophage colony-stimulating factor (GM–CSF) mRNA deadenylation and stability. 2000; Blood 95:1891–1899.

Lai, WS, Carballo, E, Thorn, JM, Kennington, EA and Blackshear, PJ. Interactions of CCCH zinc finger proteins with mRNA. 1. Binding of tristetraprolin-related zinc finger proteins to AU-rich elements and destabilization of mRNA. 2000; J. Biol. Chem., 275:17827–19837.

In addition to licensing, the technology is available for further development through collaborative research with the inventors via a Cooperative Research and Development Agreement (CRADA).

Dated: December 13, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–27782 Filed 12–17–04; 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 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.

The Use of Rabbits With Defined Immunoglobulin Light Chain Genes (C_{kappa} b Allotypes) To Optimize Production of Chimeric and Humanized Monoclonal Antibodies for Therapeutic, Imaging and Diagnostic Applications

Rose G. Mage, Cornelius Alexander (NIAID).

DHHS Reference No. E–332–2004/0—Research Tool.

Licensing Contact: Pradeep Ghosh; (301) 435–5282; *ghoshpr@mail.nih.gov.*

Biological materials are important research tools that can be used for diagnostic as well as therapeutic purposes. Antibodies have become viable drugs in the market today and there is a general market need for systems that may facilitate production of efficient and effective antibodies. In recent years, monoclonal antibodies have gained significant importance in their use, both as diagnostics and therapeutics, to intervene and combat diseases such as cancer, cardiovascular diseases, and infections. The present invention relates to the discovery of rabbits, genetically defined as b9, as the biological vehicle for the isolation of chimeric phage displaying Fab with human constant regions and rabbit immunoglobulin heavy and light chain variable regions for the development of diagnostic antibodies and humanized monoclonal therapeutic antibodies of high affinity and specificity (Popkov et al., J. Molec. Biol. 325: 325–335, 2003; Popkov et al. J. Immunol. Methods 288: 149–164, 2004). Recently, many effective antibodies have been developed as a result of the integration of antibody libraries with phage display technology. The rabbit model described in this invention may be used for production of antibodies that may cross react with both human and mouse antigens. Rabbit monoclonal antibodies that react with both human and mouse antigens are of particular relevance for the preclinical evaluation of therapeutic antibodies in mouse models of human diseases. Therefore, this invention has a broad commercial potential in its use as a source for producing monoclonal antibodies for therapeutic interventions in infectious, autoimmune and neurological diseases, nerve damage and cancer.

Methods for Diagnosis of Atherosclerosis

Paul Hwang et al. (NHLBI).

U.S. Provisional Application No. 60/607,031 filed 03 Sep 2004 (DHHS Reference No. E–276–2004/0–US–01).

Licensing Contact: Fatima Sayyid; 301/435–4521; *sayyidf@mail.nih.gov.*

In industrialized countries coronary heart disease and stroke due to atherosclerosis are the leading causes of morbidity and mortality. Coronary heart disease is the single largest cause of death in the U.S.A. and will cost approximately $133.2 billion according to the 2004 American Heart Association statistics update.

The identification of more sensitive and specific markers of atherosclerosis that are non-invasive and cost-effective may have profound impacts on public health. One such strategy involves the detection of marker genes or their products in blood or serum. Such markers may help identify high-risk patients with subclinical atherosclerosis who may benefit from intensive primary prevention or they may help determine the activity of established disease for monitoring response to treatment, resulting in more targeted secondary prevention.

The present invention relates to methods for detecting atherosclerosis using highly reactive biomarkers (FOS and/or DUSP1) expressed in blood cells or released into serum. Because these markers are also involved in pathogenesis, they may serve as potential targets for drug discovery and for intervention to modify disease progression.

An Improved Method To Separate and Expand Antigen-Specific T Cells

Jonming Li and John Barrett (NHLBI).

U.S. Provisional Application No. 60/606,197 filed 31 Aug 2004 (DHHS Reference No. E–246–2004/0–US–01).

Licensing Contact: Fatima Sayyid; (301) 435–4521; *sayyidf@mail.nih.gov.*

Stem cell transplants can be used to treat patients with leukemia or other disorders. Transplanted donor T cells (lymphocytes) exert strong alloimmune

graft versus leukemia and other anti-tumor effects however they can also cause potentially lethal graft versus host disease (GVHD), requiring post-transplant immunosuppression. Such immunosuppression may place patients at a greater risk of contracting potentially fatal cytomegalovirus infection further reducing their capacity to be cured of their malignant disease.

The transfer of T lymphocytes specific for leukemia cells or micro-organism antigens can be useful since therapeutic immune effects would be enhanced while GVHD reactions would not be induced. Currently available methods for isolating and expanding antigen-specific T cells including selection using HLA tetramers, magnetic beads binding to activation markers or laborious limiting dilution techniques are unreliable, poorly reproducible, expensive and impede clinical progress.

The present invention relates to methods for selecting and expanding antigen specific T-cells that recognize a pre selected target antigen, to purified populations of antigen-specific T cells that recognize a pre selected target antigen and to therapeutic uses of antigen-specific T cells that recognize a pre selected target antigen. Potential applications include treatment of cytomegalovirus, Epstein-Barr virus and adenovirus reactivation following stem cell transplantation or organ transplantation, prevention and treatment of leukemic relapse after transplantation or chemotherapy using autologous expanded T cells, and selective depletion of alloreactive T cells from transplants which may produce GVHD.

Novel Compounds for Selectively Inactivating Pain Pathways

Peter Blumberg, Jeewoo Lee (NCI).
U.S. Provisional Application No. 60/558,003 filed 26 Mar 2004 (DHHS Reference No. E–141–2004/0–US–01).
Licensing Contact: Norbert Pontzer; 301/435–5502; *pontzern@mail.nih.gov.*

Available for licensing are compositions and methods for the long-term control of pain and other pathological conditions caused by the over-activity of pain pathways. Neurons in the dorsal root, trigeminal and nodose ganglia project unmyelinated C-fibers and Aδ-fibers that transmit pain and temperature sensation between the periphery and spinal cord. Along with acute and chronic pain, over activation of those pathways leads to neurogenic and neuropathic inflammation leading to such conditions as post-herpetic neuralgia, diabetic neuropathy, cystitis, and reflex sympathetic dystrophy among many others.

These neurons are activated both centrally and peripherally by a relatively non-selective cation channel initially identified as site of action of capsaicin, the pungent ingredient in chili peppers. That channel is now called VR1 or TRPV1 and is found in high concentration only on C and Aδ neurons. These inventors previously discovered and patented resiniferatoxin (RTX), an ultrapotent agonist of the VR1 receptor. RTX desensitizes C and Aδ-fibers when applied peripherally and may selectively ablate those neurons when applied centrally without causing substantial pain from activation of the neurons. RTX type compounds thus provide a method of controlling pain other conditions caused by C and Aδ-fiber activity. The present invention provides new RTX analogues that may have an improved therapeutic index and metabolic profile.

Dated: December 9, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–27783 Filed 12–17–04; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Human Genome Research 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 Human Genome Research Institute Special Emphasis Panel.

Date: December 16–17, 2004.

Time: December 16, 2004, 7 p.m. to 10 p.m.

Agenda: To review and evaluate grant applications.

Place: Stanford University, Stanford Terrace Inn, 531 Stanford Avenue, Palo Alto, CA 94306.

Time: December 17, 2004, 8 a.m. to 5 p.m.

Agenda: To review and evaluate grant applications.

Place: Stanford University, Stanford Terrace Inn, 531 Stanford Avenue, Palo Alto, CA 94306.

Contact Person: Ken D. Nakamura, PHD, Scientific Review Administrator, Office of Scientific Review, National Human Genome Research Institute, National Institutes of Health, Bethesda, MD 20892, (301) 402–0838.

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 Domstic Assistance Program Nos. 93.172, Human Genome Research, National Institutes of Health, HHS)

Dated: December 14, 2004.

Anna P. Snouffer,

Acting Director, Office of Federal Advisory Committee Policy.

[FR Doc. 04–27779 Filed 12–17–04; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute on Drug Abuse; 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 Drug Abuse.

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 Drug Abuse.

Date: February 15–16, 2005.

Closed: February 15, 2005, 2 p.m. to 4 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, Neuroscience Center, 6001 Executive Boulevard, Rockville, MD 20852.

Open: February 16, 2005, 9 a.m. to 4 p.m.