

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.

#### **Antibodies That Specifically Recognize SUMO-Conjugated Proteins**

Dr. Mary Dasso (NICHD).

U.S. Provisional Application Serial No. 60/438,685 filed 08 Jan 2003 (DHHS Reference No. E-066-2002/0-US-01).

Licensing Contact: Marlene Shinn-Astor; 301/435-4426; [shinnm@mail.nih.gov](mailto:shinnm@mail.nih.gov)

SUMO-1 is an ubiquitin-like heat shock protein that can be covalently conjugated to other proteins through an isopeptide linkage. This technology describes polyclonal antibodies that recognize SUMO-1 conjugated proteins, including conjugated RanGAP1. These antibodies could be used as a diagnostic tool to test for diseases that contain SUMO-1 mis-regulation with further development. It is also foreseen that they could be used in large-scale screening of small molecule libraries to find compounds capable of either inhibiting or enhancing the SUMO-1 conjugation pathway.

#### **Modulators of Nuclear Hormone Receptor Activity: Novel Compounds, Diverse Applications for Infectious Diseases, Including Anthrax (*B. anthracis*)**

E. M. Sternberg (NIMH), J. I. Webster (NIMH), L. H. Tonelli (NIMH), S. H. Leppla (NIAID), and M. Maoyeri (NIAID).

DHHS Reference No. E-247-2002/0-US-01 filed 18 October 2002.

Licensing Contact: Peter Soukas; 301/435-4646; [soukasp@mail.nih.gov](mailto:soukasp@mail.nih.gov).

*Technology summary and benefits:* Nuclear hormones such as glucocorticoids dampen inflammatory responses, and thus provide protection to mammals against inflammatory disease and septic shock. The Anthrax lethal factor represses nuclear hormone receptor activity, and thus may contribute to the infectious agent causing even more damage to the host. This observation can be exploited to find new means of studying and interfering with the normal function of nuclear hormone receptors. Scientists at NIH have shown that under the appropriate conditions, these molecules can be used to modulate the activity of various nuclear hormone receptors.

Identifying useful agents that modify these important receptors can provide relief in several human disorders such as inflammation, autoimmune disorders, arthritis, malignancies, shock and hypertension.

*Long-term potential applications:* This invention provides novel agents that can interfere with the action of nuclear hormone receptors. It is well known that malfunction or overdrive of these receptors can lead to a number of diseases such as enhanced inflammation; worse sequelae of infection including shock; diabetes; hypertension and steroid resistance. Hence a means of controlling or fine-tuning the activity of these receptors can be of great benefit. Current means of affecting steroid receptor activity are accompanied by undesirable side-effects. Since the conditions for which these treatments are sought tend to be chronic, there is a critical need for safer drugs that will have manageable side-effects.

*Uniqueness or innovativeness of technology:* The observation that the lethal factor from Anthrax has a striking effect on the activity of nuclear hormone receptors opens up new routes to controlling their activity. The means of action of this repressor is sufficiently different from known modulators of hormone receptors (*i.e.*, the classical antagonists). For instance, the repression of receptor activity is non-competitive, and does not affect hormone binding or DNA binding. Also, the efficacy of nuclear hormone receptor repression by Anthrax lethal factor is sufficiently high that the pharmacological effect of this molecule is seen at vanishingly small concentrations. Taken together, these attributes may satisfy some of the golden rules of drug development such as the uniqueness or novelty of the agent's structure, a low threshold for activity, high level of sophistication and knowledge in the field of enquiry, and the leeway to further refine the molecule by rational means.

*Stage of Development:* In vitro studies have been completed, and a limited number of animal studies have been carried out.

#### **Method for the Treatment of Multiple Sclerosis**

Roland Martin *et al.* (NINDS).

U.S. Provisional Application Serial No. 60/393,021 filed 28 Jun 2002 (DHHS Reference No. E-143-2002/0-US-01), PCT/US02/38290 filed 27 Nov 2002 (DHHS Reference No. E-143-2002/0-PCT-02), U.S. Patent Application filed 27 Jun 2003 (DHHS Reference No. E-143-2002/0-US-03), and PCT/

US03/20428 filed 27 Jun 2003 (DHHS Reference No. E-143-2002/0-PCT-04).

Licensing Contact: Catherine Joyce 301/435-5031; e-mail: [joycec@mail.nih.gov](mailto:joycec@mail.nih.gov).

The invention relates to the discovery that humanized antibodies to the interleukin-2 receptor (IL-2R) such as (daclizumab) are effective in treating multiple sclerosis (MS). In particular, it has been discovered that patients who have failed to respond to therapy with interferon-beta show dramatic improvement when treated with daclizumab, with patients showing both a reduction in the total number of lesions and cessation of appearance of new lesions during the treatment period. Daclizumab is effective both in combination with interferon-beta and alone.

The above-mentioned invention is available for licensing on an exclusive or non-exclusive basis.

Dated: August 4, 2003.

**Steven M. Ferguson,**

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

[FR Doc. 03-20560 Filed 8-12-03; 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.

**ADDRESS:** 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.

**Full-Length cDNA Clone Representing the Consensus Sequence of the RNA Genome of a Human Norovirus (strain MD145-12) that Encodes Biologically Active Proteins**

Gael M. Belliot, Kim Y. Green, Stanislav V. Sosnovtsev (NIAID)  
DHHS Reference No. E-212-2003/0  
Licensing Contact: Sally Hu; 301/435-5606; [hus@mail.nih.gov](mailto:hus@mail.nih.gov)

The invention provides for a full-length cloned cDNA copy of the RNA genome of a predominant norovirus strain designated MD145-12 that was associated with human gastrointestinal illness. The noroviruses, which were formerly known as "Norwalk-like" viruses are estimated to cause 23 million cases of acute gastroenteritis in the USA each year. The virus has been designated into category B of the CDC biodefense-related priority pathogens because it can be used as an agent of bioterrorism. The subject cDNA clone of the virus encodes proteins of the MD145-12 strain that, when expressed in vitro, exhibit properties that would be expected from those produced by the original infectious virus. This cDNA clone is presently the only source to obtain norovirus proteins to facilitate studies aimed at developing control strategies such as vaccines and therapeutic drugs.

It is our intention not to seek patent protection for the above described invention. Instead, the cDNA clone for norovirus strain MD145-12 is available for licensing via biological material license (BML).

**Rapamycin Resistant T Cells and Therapeutic Uses Thereof**

Drs. Daniel Fowler (NCI), Unsu Jung (NCI), Jeannie Hou (NCI), Ronald Gress (NCI), Bruce Levine (U. of Penn.), and Carl June (U. of Penn.)  
U.S. Provisional Application Serial No. 60/478,736 filed 12 Jun 2003 (DHHS Reference No. E-063-2003/0-US-01)  
Licensing Contact: Sally Hu; 301/435-5606; [hus@mail.nih.gov](mailto:hus@mail.nih.gov)

This invention identified T cell culture conditions that use the immune suppression drug rapamycin (sirolimus) to generate rapamycin-resistant cells having Th1, Th2, Tc1 or Tc2 function (Th=T helper lymphocytes; Tc=cytotoxic T lymphocytes). This invention has demonstrated how to generate T cells enriched for Th1, Th2, Tc1 or Tc2 functions as well as how to control these functions *in vivo*. Those methods can make T cell therapies significantly more viable and applicable

for treatment of a variety of diseases states, including cancer, infectious diseases, autoimmune diseases, Graft vs. Host Disease (GVHD) associated with allogeneic hematopoietic stem cell transplantation, and graft rejection. Thus, this invention has many useful purposes that could generate significant interest among groups pursuing immune therapies, particularly T cell-based therapeutic approaches. Diseases in which T cell based therapies would be of major impact include cancer, viral infections such as HIV disease, autoimmunity, transplantation and any other disease in which the T cells participate.

**Computational Prediction Method for T Cell Epitopes Based on Quantitative Properties of MHC Binding Peptides**

Myong-Hee Sung and Richard Simon (NCI)

U.S. Provisional Application Serial No. 60/416,034 filed 03 Oct 2002 (DHHS Reference No. E-110-2002/0-US-01)

Licensing Contact: Cristina Thalhammer-Reyero; 301/435-4507; [thalhamc@mail.nih.gov](mailto:thalhamc@mail.nih.gov)

NIH announces a computational method for the prediction of peptides binding to major histocompatibility complex proteins (MHC), which facilitates the resource-consuming effort required to identify T-cell epitopes. The presentation of such epitopes by the MHC to T-cells can, in conjunction with co-factor interactions, activate the T-cells to initiate the necessary immune response against the epitope source. Consequently, peptides that are predicted to bind to multiple MHC molecules are potentially useful in vaccine design. The invention describes a new method for predicting MHC binding based on peptide property models constructed using biophysical parameters of the constituent amino acids and a training set of known binders. For example, the models can be applied to development of anti-tumor vaccines by scanning proteins over-expressed in cancer cells for peptides that bind to a variety of MHC molecules, as illustrated in the context of identifying candidate T-cell epitopes for melanomas and breast cancers. This computational approach provides an efficient and focused strategy for identifying candidate epitopes for development of vaccines and anti-cancer immunotherapy.

Dated: August 4, 2003.

**Steven M. Ferguson,**

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

[FR Doc. 03-20562 Filed 8-12-03; 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 discussion 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, Hypovolemic Circulatory Collapse: Mechanisms and Opportunities to Improve Resuscitation Outcomes.

*Date:* October 2-3, 2003.

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

*Agenda:* To review and evaluate grant applications.

*Place:* Sheraton Columbia Hotel, 10207 Wincopin Circle, Columbia, MD 21044.

*Contact Person:* Katherine M. Malinda, Ph.D., Scientific Review Administrator, Review Branch, Division of Extramural Affairs, National Heart, Lung, and Blood Institute, National Institutes of Health, 6701 Rockledge Drive, Room 7198, Bethesda, MD 20892, 301/435-0297.

(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: August 5, 2003.

**LaVerne Y. Stringfield,**

*Director, Office of Federal Advisory Committee Policy.*

[FR Doc. 03-20547 Filed 8-12-03; 8:45 am]

**BILLING CODE 4140-01-M**