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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
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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)
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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

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.

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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]

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