Ethnic Disparities in Health'' by HHS (*http://raceandhealth.hhs.gov/*) encouraged NIH to help reduce health disparities. In its 1999 reauthorizing legislation, AHRQ was directed to conduct and support research to identify and reduce health care disparities (Pub. L. 106–525). NIH published the ''Strategic Research Plan and Budget to Reduce and Ultimately Eliminate Health Disparities, Fiscal Years 2002–2006'' at (*http://www.ncmhd.nih.gov*). Finally, the ''NIGMS Strategic Plan for Reducing Health Disparities'' (*http://www.nigms.nih.gov/news/reports/health_disparities.html*) presents an NIGMS role in health disparity reduction through its focused programs on research infrastructure to increase the number and capabilities of under-represented minority health researchers. In response to these priorities, the IHS, NIGMS and AHRQ have established a collaboration to support Native American Research Centers for Health. Reducing health disparities among AI/AN communities and individuals may be fostered by greater understanding of how to enhance their strengths and resiliencies. While AI/AN communities have relied on health research and medical science to reduce health disparities, they also have relied on their own psychological, organizational, and cultural assets and strengths to survive major harms and disruptions over the centuries, and to rebound from insults to health. For research about resiliencies, see *http://www.nida.nih.gov/ResilandRiskWG/ResilandRiskWG.html.*

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Dated: February 17, 2004.

Michel E. Lincoln,

Deputy Director, Indian Health Service.

[FR Doc. 04–3867 Filed 2–23–04; 8:45 am]

BILLING CODE 4160–16–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.

SAP/SH2D1A Knockout Mice: A Model for X-linked Lymphoproliferative Disease

Pamela L. Schwartzberg (NHGRI), DHHS Reference No. E–343–2003/0—Research Tool,

Licensing Contact: Cristina Thalhammer-Reyero; 301/435-4507; *thalhamc@mail.nih.gov.*

NIH announces the availability for licensing of SAP/SH2D1A knockout mice, which can be used as a model for X-linked lymphoproliferative disease (XLP), and exploited to design therapeutics or gene-therapy for XLP. These knockout mice can be used as well to study other T cell-mediated diseases, such as asthma and hypersensitivity, involving Th2 cells. This model is also useful for researchers interested in T-cell signaling and cytokine production by T-helper cells.

SAP (SLAM-associated protein) is a small lymphocyte-specific signaling molecule that is defective or absent in patients with XLP. SAP has unusually high affinity for SLAM (also called CD150) and has been suggested to function by blocking binding of SHP–2 or other SH2-containing signaling proteins to SLAM receptors. SAP has also been shown to be required for

recruitment and activation of the Src-family kinase FynT after SLAM ligation, where the SAP SH2 domain binds to the SH3 domain of FynT and directly couples FynT to SLAM.

Mutations in the *SH2D1 A* gene on the Xq24–26 chromosome are known to be responsible for many cases of X-linked Lymphoproliferative syndrome.

Immunoglobulins With Potent and Broad Antiviral (HIV) Activity Based on scFv Joined by Flexible Linker to Fc

Drs. Dimiter Dimitrov (NCI) and Mei-Yun Zhang (SAIC),

U.S. Provisional Patent Application filed 29 Sep 2003 (DHHS Reference No. E–316–2003/0–US–01),

Licensing Contact: Sally Hu; (301) 435–5606; *hus@mail.nih.gov.*

This invention describes methods of inhibiting viral infection (*e.g.*, HIV–1 infection). The method comprises administering a fusion protein comprising a small size, single chain Fv (scFv) antibody binding domain joined to an Fc region by a long flexible linker. In particular, scFv m6 or m9, the single chain variable fragments that were previously identified from a phage display library for binding to $gp140_{89.6}$, $gp120_{JRFL}$, $gp140_{IIIB}$, and their complex with two-domain soluble CD4 is joined to Fc by a long flexible linker to provide a new agent for the inhibition of HIV infection or immunotherapy of HIV-infected individuals. The Fc region provides stability, long half-life, and biological effector functions. The scFv-Fc fragment provides antigen recognition and neutralizing activity. The small size of the scFv-Fc fusion molecule provides easy access to conserved viral epitopes exposed before or during viral entry. In addition, these fusion molecules exhibit neutralization activity that is higher than that of whole IgGs. Thus, this invention may offer a novel approach to treat and prevent HIV–1 infection and/or AIDS.

Potent Combinations of mRNA Transport Elements

Barbara K. Felber et al. (NCI),

U.S. Provisional Application No. 60/471,988 filed 19 May 2003 (DHHS Reference No. E–223–2003/0–US–01);

U.S. Provisional Application No. 60/472,223, filed 20 May 2003 (DHHS Reference No. E–258–2003/0–US–01),

Licensing Contact: Susan Ano; 301/435–5515; *anos@mail.nih.gov.*

This technology relates to improving levels of gene expression using a combination of a constitutive RNA transport element (CTE) with a mutant form of another RNA transport element (RTE). The combination of these elements results in a synergistic effect on stability, and therefore expression levels, of mRNA transcripts. Using HIV–1 gag as reporter mRNA, one mutated RTE in combination with a CTE was found to improve expression of unstable mRNA by about 500-fold. Similarly this combination of elements lead to synergistically elevated levels of HIV–1 env expression. The function of CTEs and RTEs is conserved in mammalian cells, so this technology is a simple and useful way of obtaining high levels of expression of otherwise poorly expressed genes and can be used in a number of applications such as but not limited to improvements of gene therapy vectors, expression vectors for mammalian cells.

Safer Attenuated Virus Vaccines With Missing or Diminished Latency of Infection

Jeffrey Cohen (NIAID), Edward Cox (FDA), Lesley Pesnicak (NIAID),

U.S. Provisional Application No. 60/423,603 filed 05 Nov 2002 (DHHS Reference No. E–250–2002/0–US–01); PCT Application No. PCT/US03/35167 filed 05 Nov 2003 (DHHS Reference No. E–250–2002/0–PCT–02),

Licensing Contact: Susan Ano; (301) 435–5515; *anos@mail.nih.gov.*

This technology describes viruses that have weakened ability to establish and/or maintain latency and their use as live vaccines. The viruses have one or more genetic mutations that allow for continued replication but that inhibit latency. The vaccine materials and methods for their construction are exemplified with the virus that causes chickenpox and whose latent infection results in shingles, a condition that affects up to an estimated 1 million people per year in the United States alone. Specific examples of gene deletion are described. Furthermore, replacement of these deleted genes with other desirable viral antigen encoding sequence(s) and/or cytokine genes in order to enhance a desired immunological response is also described. Aspects of this technology are relevant to other live virus vaccines, thus increasing the safety of such vaccines.

Novel Receptor for Pathogenic Fungi

Victor Jimenez (EM), Victor Ginsburg (NIDDK), Howard Krivan (NIDDK),

U.S. Patent Application No. 07/472,128 filed 30 Jan 1990, which issued as U.S. Patent 5,242,800 on 07 Sep 1993 (DHHS Reference No. E–145–1989/0–US–01),

Licensing Contact: Michael Ambrose; (301) 594–6565; *ambrosem@mail.nih.gov.*

A specific receptor for pathogenic fungi has been isolated and substantially purified for the first time, and a method of using the receptor to prevent adhesion of pathogenic fungi to host cells has been developed. A kit for detecting the presence of certain fungi was also described. These products make possible the detection and removal of two important pathogenic fungi, Candida albicans and Cryptococcus neoformans, and may be useful in preventing yeast diseases.

Dated: February 17, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–3906 Filed 2–23–04; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer 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 552(b)(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 contract proposals, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Cancer Institute Review Group, Subcommittee A—Cancer Centers.

Date: April 15–16, 2004.

Time: 7:30 a.m. to 3 p.m.

Agenda: To review and evaluate grant applications.

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

Contact Person: David E. Maslow, PhD, Scientific Review Administrator, Resources and Training Review Branch, Division of Extramural Activities, National Cancer Institute, 6116 Executive Boulevard, Room 8117, Bethesda, MD 20892–7405, (301) 496–2330.

Any interested person may file written comments with the committee by forwarding