U.S. Patent Application No. 10/439,845 filed 15 May 2003 (DHHS Reference No. E–090–1996/0–US–05);
U.S. Patent Application No. 10/700,313 filed 31 Oct 2003 (DHHS Reference No. E–090–1996/0–US–06);
U.S. Patent Application No. 10/846,185 filed 14 May 2004 (DHHS Reference No. E–090–1996/0–US–07);
PCT Application No. PCT/US97/09586 filed 28 May 1997 (DHHS Reference No. E–090–1996/0–PCT–02);
European Patent Application No. 97929777.7 filed 28 May 1997 (DHHS Reference No. E–090–1996/0–EP–03).

Licensing Contact: Peter Soukas; 301/435–4646; *soukasp@mail.nih.gov.*

Chemokine receptors are expressed by many cells, including lymphoid cells, and function to mediate cell trafficking and localization. CC chemokine receptor 5 (CCR5) is a seven-transmembrane, G protein-coupled receptor (GPCR) which regulates trafficking and effector functions of memory/effector T-lymphocytes, macrophages, and immature dendritic cells. Chemokine binding to CCR5 leads to cellular activation through pertussis toxin-sensitive heterotrimeric G proteins as well as G protein-independent signalling pathways. Like many other GPCR, CCR5 is regulated by agonist-dependent processes which involve G protein coupled receptor kinase (GRK)-dependent phosphorylation, beta-arrestin-mediated desensitization and internalization.

Human CCR5 also functions as the main coreceptor for the fusion and entry of many strains of human immunodeficiency virus (HIV–1, HIV–2). HIV-1 transmission almost invariably involves such CCR5-specific variants (designated R5); individuals lacking functional CCR5 (by virtue of homozygosity for a defective CCR5 allele) are almost completely resistant to HIV–1 infection. Specific blocking of CCR5 (*e.g.* with chemokine ligands, anti-CCR5 antibodies, CCR5-blocking low MW inhibitors, etc.) inhibits entry/infection of target cells by R5 HIV strains. Cells expressing CCR5 and CD4 are useful for screening for agents that inhibit HIV by binding to CCR5. Such agents represent potential new approaches to block HIV transmission and to treat infected people. A small animal expressing both human CCR5 along with human CD4 supports entry of HIV into target cells, a necessary hurdle that must be overcome for development of a small animal model (*e.g.* transgenic mouse, rat, rabbit, mink) to study HIV infection and its inhibition.

The invention embodies the CCR5 genetic sequence, cell lines and transgenic mice, the cells of which coexpress human CD4 and CCR5, and which may represent valuable tools for the study of HIV infection and for screening anti-HIV agents. The invention also embodies anti-CCR5 agents that block HIV env-mediated membrane fusion associated with HIV entry into human CD4-positive target cells or between HIV-infected cells and uninfected human CD4-positive target cells.

This technology was reported in Alkhatib *et al.*, ''CC CKR5: a RANTES, MIP–1alpha, MIP–1beta receptor as a fusion cofactor for macrophage-tropic HIV–1,'' Science 272:1955–1958 (1996). The technology is available for exclusive or nonexclusive licensing.

Dated: March 25, 2005.

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

[FR Doc. 05–6895 Filed 4–6–05; 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.

Identification of Molecular Markers for Endometriosis in Blood Lymphocytes Using DNA Microarrays

Idhaliz Flores (NHGRI), *et al.*

U.S. Provisional Application filed 18 Feb 2005 (DHHS Reference No. E–068–2005/0–US–01).

Licensing Contact: Marlene Shinn-Astor; (301) 435–4426; *shinnm@mail.nih.gov.*

Endometriosis is a common, non-malignant gynecological disease that affects up to 20% of women during their reproductive years. Endometriosis is characterized by the growth of endometrial tissue outside the uterus. This growth of tissue causes recurring severe pain and can lead to infertility. As the current procedure used for diagnosis is invasive and not entirely accurate, there is a need for a fast, accurate, and minimally invasive test to test for endometriosis.

Using DNA microarray analysis of blood lymphocytes, the inventors have identified two gene markers expressed in blood that are able to discriminate between those women who have endometriosis and those that don't. The two gene markers identified are interleukin-2 receptor gamma (IL–2RG, a component of cytokine receptors) and lysyl oxidase-like 1 (LOXL1, which plays an important role in collagen synthesis and has also been implicated as a growth regulatory gene). Other genes identified in the same manner and which also represent potential biomarkers for endometriosis await further validation studies.

The test would be minimally invasive and quick using a blood sample from the patient. Currently, patients must undergo a laparoscopy with the diagnosis dependent upon the expertise of the surgeon performing the procedure.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Increased Protein Production

Drs. Shankar Adhya and Sudeshna Kar (NCI).

U.S. Provisional Application No. 60/571,943 filed 18 May 2004 (DHHS Reference No. E–261–2003/0-US–01).

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

There is a continuing market need to identify biological measures to enhance recombinant protein production for therapeutic inventions for the treatment of diseases. In general, the field of recombinant protein production, including inducement of protein production both by cloning and non-cloning methods and incorporation of antibiotic resistance genes in vectors appeared to be relatively crowded.

However, this invention pertains to the creation of a specific 2.4 kb gene cassette that includes a specific gene that confers resistance to aminoglycoside antibiotics, increases protein levels inside a cell and increases yield of production of recombinatant proteins, when inserted. In particular, the inventors have identified a specific gene *aad*A1 (adenyltransferase gene) that codes for a 28.876 Kd protein that normally confers aminoglycoside resistance to cells. Further, the inventors have found that a ''gene cassette'' carrying the *aad*A1 gene which when transferred to bacterial strains induces enhancement of protein production and accumulation. Additionally, this inducement is not restricted by the nature of the vector, induction system or nature of protein. In short, the invention provides a method of reconstruction of a cell for increased yield of recombinant protein, which involves a ''one-step procedure of induction of a new gene into the cell.'' Therefore, the technology may have a substantial commercial value to the pharmaceutical industry.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Endothelial Protective Actions of Cytochrome P450 Epoxygenase-derived Eicosanoids

Darryl C. Zeldin (NIEHS), *et al.*

U.S. Patent Application No. 09/634,369 filed 09 Aug 2000, notice of allowance issued (DHHS Reference No. E–252–1999/0–US–02).

Licensing Contact: Marlene Shinn-Astor; (301) 435–4426; *shinnm@mail.nih.gov.*

Cytochrome P450s catalyze the NADPH-dependent oxidation of arachidonic acid to various eicosanoids found in several species including humans. The eicosanoids are biosynthesized in numerous tissues including pancreas, intestine, kidney, heart, and lung where they are involved in many different biological activities.

The NIH announces a new therapy wherein epoxyeicosatrienoic acid (EET) compositions have been found to be useful in preventing endothelial cell death due to hypoxia-reoxygenation. Given that endothelial injury is an important early event in the development of the atherosclerotic plaque and is associated with myocardial dysfunction in ischemic heart disease, reduced EET levels are speculated to be involved in the pathogenesis of these cardiovascular disorders.

This research is described in *Yang et al.,* Molecular Pharmacology 60: 310–320, 2001.

T-Cell Receptor Alternate Reading Frame Protein, (TARP) and Uses Thereof

Ira Pastan, Magnus Essand, Byungkook Lee, George Vasmatzis, Ulrich Brinkman, Paul Duray, and Curt Wolfgang (NCI).

U.S. Patent Application No. 10/031,158 filed 11 Jan 2002, and multiple National Stage foreign filings (DHHS Reference No. E–104–1999/2).

Licensing Contact: Brenda Hefti; (301) 435–4632; *heftib@mail.nih.gov.*

This invention relates to a tumor-associated protein, TARP, which is expressed in breast and prostate cancer cells. This antigen target might be a useful tool for the diagnosis and treatment of breast and prostate cancer. TARP has shown efficacy in vivo as a potential therapeutic for the treatment of cancer. TARP has been the subject of several publications, including: J. Biol. Chem. (2004 Jun 4) 279(23):24561–24568, Epub 2004 Mar 29 as doi:10.1074/jbc.M402492200; Cancer Res. (2004 Apr 1) 64(7):2610–2618; Endocrinology (2003 Aug) 144(8):3433–40; Cancer Res. (2001 Nov 15) 61(22):8122–8126; Proc. Natl. Acad. Sci. USA (2000 Aug 15) 97(17):9437–9442.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Method for Reducing the Immunogenicity of Antibody Variable Domains

Eduardo Padlan (NIDDK) *et al.*

U.S. Patent No. 6,797,492 issued 28 Sep 2004 (DHHS Ref. No. E–163–1991/2–US–02)

Licensing Contact: Jeff Walenta; (301) 435–4633; *walentaj@mail.nih.gov.*

The current invention addresses a limitation of monoclonal antibodies used in immunotherapy. Monoclonal antibodies with high selectivity for human antigens are commonly produced in mice. However, when introduced into humans for therapy, the antibodies can be neutralized by the human immune system and their duration and effectiveness limited. Modification of non-human antibodies to avoid the human immune system often produces antibodies with reduced affinity for the antigen and which remain antigenic in humans.

The current invention provides a method for producing ''humanized'' antibodies that retain antigen binding properties but which have eliminated or reduced antigenicity. The method comprises substituting residues in the variable region of the non-human antibody with residues found in the variable region of human antibodies, with particular emphasis on residues that are solvent exposed and that are not adjacent to complementarity determining regions.

When tested in monkeys, the serum longevity of the ''veneered'' antibodies produced by the current invention was significantly greater than that of mouse antibodies or chimeric mouse-human antibodies. Accordingly, the technology could enhance the effectiveness of monoclonal antibodies designed for therapy of cancer or other diseases.

Dated: March 25, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–6896 Filed 4–6–05; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Eye 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 Eye Institute Special Emphasis Panel, Loan Repayment Program Applications.

Date: April 18, 2005.

Time: 8:30 a.m. to 5 p.m.

Agenda: To review and evaluate loan Repayment applications.

Place: Embassy Suites at the Chevy Chase Pavilion, 4300 Military Road, NW., Washington, DC 20015.

Contact Person: Anne Schaffner, PhD, Scientific Review Administrator, Division of Extramural Research, National Eye Institute, 5635 Fishers Lane, Suite 1300, MSC 9300, Bethesda, MD 20892–9300, (301) 451–2020, *aes@nei.nih.gov.*