when the bacteriophage infects a bacterial cell. These bacteriophages are separately contacted with a sample contaminated by a bacterium. Expression of the reporter is then detected, indicating which bacteriophage has infected a bacterial cell and is thus a potential therapeutic phage against the particular bacteria. Also claimed in the application are kits allowing for the rapid identification of potentially therapeutic bacteriophages.

Bacteriophage Having Multiple Host Range

Carl Merril (NIMH), Sankar Adhya (NCI), Dean Scholl (NIMH).

U.S. Provisional Application No. 60/220,987 filed 25 Jul 2000 (HHS Reference No. E–257–2000/0–US–01);

PCT Application No. PCT/US01/22390 filed 25 Jul 2001 (HHS Reference No. E–257–2000/0–PCT–02);

U.S. Patent Application No. 10/350,256 filed 21 Jan 2003 (HHS Reference No. E–257–2000/0–US–03).

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

Recently, there has been a renewed interest in the use of phages to treat bacterial infections. The inventors have discovered FK1–5, a highly lytic, non-lysogenic, stable bacteriophage with the ability to kill bacteria rapidly, making it a good candidate for phage therapy. The designation FK1–5 denotes the phage's ability to infect E. coli strains that contain the K1 polysaccharide in their outer capsule as well as E. coli strains that contain the K5 polysaccharide in their outer capsule. Sequence analysis of the tail proteins of phage FK1–5 by the inventors has shown that they are arranged in a cassette structure, suggesting that the host range of phages can be broadened to other K antigens, and even possibly other species of bacteria by recombinant techniques. FK1–5 has a particular advantage because it recognizes and attaches to the structures that confer virulence to bacteria. The inventors' demonstration that a phage can contain multiple tail proteins that expand its host range is useful for generating phage with broad-spectrum antibacterial properties for the treatment of infectious diseases. The inventors have completed in vitro studies on this phage. Furthermore, because of the possibility of engineering the expression of recombinant tail proteins, gene transfer to organisms that are not normally infected by phages is also contemplated by the invention.

CC Chemokine Receptor 5 DNA, New Animal Models and Therapeutic Agents for HIV Infection

C. Combadiere, Y. Feng, E.A. Berger, G. Alkahatib, P.M. Murphy, C.C. Broder, P.E. Kennedy (NIAID).

U.S. Provisional Application No. 60/018,508 filed 28 May 1996 (HHS Reference No. E–090–1996/0–US–01);

U.S. Patent Application No. 08/864,458 filed 28 May 1997 (HHS Reference No. E–090–1996/0–US–04);

U.S. Patent Application No. 10/439,845 filed 15 May 2003 (HHS Reference No. E–090–1996/0–US–05);

U.S. Patent Application No. 10/700,313 filed 31 Oct 2003 (HHS Reference No. E–090–1996/0–US–06);

U.S. Patent Application No. 10/846,185 filed 14 May 2004 (HHS Reference No. E–090–1996/0–US–07);

PCT Application No. PCT/US97/09586 filed 28 May 1997 (HHS Reference No. E–090–1996/0–PCT–02);

European Patent Application No. 97929777.7 filed 28 May 1997 (HHS 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: July 19, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–15347 Filed 8–2–05; 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.

A32 Monoclonal Antibody Fusion Protein for Use as HIV Inhibitors and Vaccines

Dimiter S. Dimitrov and Mei-yun Zhang (NCI).

U.S. Provisional Application No. 60/618,820 filed 14 Oct 2004 (HHS Reference No. E–302–2004/0–US–01).

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

The invention provides composition claims of a fusion protein, which comprises an antigen binding portion of a human antibody called A32 and one of the following: (a) An antigen-binding portion of a second antibody that binds to an epitope of an envelope protein (*i.e.*, gp120) of a human immunodeficiency virus (HIV) that is exposed upon the HIV binding to a CD4 receptor, (b) an immunogenic portion of an envelope protein of a HIV such as gp120, or (c) a soluble CD4 (sCD4) polypeptide capable of binding to HIV. The invention also provides the method claims to use the above fusion proteins as inhibitors of HIV infection and those containing gp120 such as A32–gp120 as vaccine immunogens for the treatment and prevention of HIV. Further development of the fusion proteins may yield novel therapies and methods in the prevention of mother-to-child transmission of HIV, treatment of accidental exposure to HIV, and chronic infection in patients with resistance to current therapies.

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

Plasmid and Viral Vectors Expressing a Microtubule-Directed Fluorescent Fusion Protein for Cellular Imaging

Dr. Michael J. Iadarola *et al.* (NIDCR).

HHS Reference No. E–153–1999/0—Research Tool.

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

This technology is a fluorescent protein for discrete tracing of intra- and intercellular connections and for sorting and isolation of cells. This recombinantly engineered protein can be expressed from viral vectors for use in living animals and in ex vivo situations involving primary cultured cells or from a plasmid for use in cell lines. The new protein consists of a fusion between the tau protein, which binds to microtubules, and enhanced green fluorescent protein (tau-eGFP). When cloned into adenovirus, the contrast can be used for transducing primary cultures for ex vivo gene therapy and for use as an anterograde tracer in brain circuit analysis. These uses can be a valuable research tool to help scientists find out how the brain works, investigate Alzheimer's disease, and to identify specific cells for treating disease via cell transplantation.

Dated: July 19, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–15348 Filed 8–2–05; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; 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 the meeting of the President's Cancer Panel.

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 section 552b(c)(9)(B), title 5 U.S.C., as amended, because the premature disclosure of information and the discussions would likely to significantly frustrate implementation of recommendations.

Name of Committee: President's Cancer Panel.

Date: August 25, 2005.

Open: August 25, 2005, 8 a.m. to 2:30 p.m.

Agenda: Cancer Survivorship: Treatment Records, Follow-up, and HIPPA.

Place: The Washington Marriott Hotel, 1221 22nd Street, NW., Washington, DC 20037.

Closed: August 25, 2005, 3 p.m. to 5 p.m.

Agenda: The Panel will discuss the treatment records and follow-up care plans.

Place: The Washington Marriott Hotel, 1221 22nd Street, NW., Washington, DC 20037.

Contact Person: Abby Sandler, Ph.D., Executive Secretary, National Cancer Institute, National Institutes of Health, Building 6116, Room 212, 6116 Executive Boulevard, Bethesda, MD 20892. (301) 451–9399.

Any interested person may file written comments with the committee by forwarding the comments to the Contact Person listed on this notice. The comments should include the name, address, telephone number and, when applicable, the business or professional affiliation of the interested person.

Information is also available on the Institute's/Center's Home page: *http://deainfo.nci.nih.gov/advisory/pcp/pcp.htm,* where an agenda and any additional information for the meeting will be posted when available.

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399, Cancer Control, National Institutes of Health, HHS).

Dated: July 26, 2005.

Anthony M. Coelho, Jr.,

Acting Director, Office of Federal Advisory Committee Policy.

[FR Doc. 05–15243 Filed 8–2–04; 8:45 am]

BILLING CODE 4140–01–M

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 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 Heart, Lung, and Blood Institute Special Emphasis Panel, Genetics of Alaska Natives.

Date: August 16, 2005.

Time: 9 a.m. to 11 a.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, 6701 Rockledge Drive, Room 7214, Bethesda, MD 20892. (Telephone conference call.)