whether the information will have practical utility; (2) the accuracy of the agency's estimate of the burden of the proposed collection of information, including the validity of the methodology and assumptions used; (3) ways to enhance the quality, utility, and clarity of the information to be collected; and (4) ways to minimize the burden of the collection of information on those who are to respond, including the use of appropriate automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

FOR FURTHER INFORMATION CONTACT: To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact: Alfred C. Johnson, Ph.D., Deputy Director, Office of Loan Repayment and Scholarship, National Institutes of Health, 2 Center Drive, Room 2E28 (MSC 0230), Bethesda, Maryland 20892–0230. Dr. Johnson may be contacted via e-mail at *ACJohnson@nih.gov* or by telephone at 301–402–6425.

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 60 days of the date of this publication.

Dated: June 3, 2004.

Raynard S. Kington,

Deputy Director, National Institutes of Health.

[FR Doc. 04–13153 Filed 6–9–04; 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.

A Peptide That Elicits Neutralizing Antibodies Targeting the HIV Co-Receptor CCR5

Drs. Hana Golding and Surender Khurana (FDA)

U.S. Provisional Application filed 09 Apr 2004 (DHHS Reference No. E–150–2004/0–US–01)

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

This invention identifies a peptide sequence that closely mimics the conformational epitope in CCR5, recognized by the HIV neutralizing monoclonal antibody targeting the co-receptor, by using a random peptide phage display library. This peptide upon immunization of rabbits generated antibodies that bind to the HIV–1 co-receptor CCR5 resulting in blocking HIV transmission to target cells, including peripheral blood lymphocytes from human and monkeys. Thus, such antibodies could be directly used for preventing mother to child HIV transmission, for therapy of HIV–1 infected individuals, and may also have particular value when used in combination treatments with other antiviral therapies directed at viral targets, such as protease and reverse transcriptase. The peptide sequence can be used for potential vaccine development. This peptide can also be used for screening of human antibody phage display libraries to isolate human monoclonal with HIV entry-blocking potential. In addition, the peptide and antibodies recognizing it can be used as research tools for increasing the understanding of the mechanisms by which HIV, CCR5 and the HIV receptor, CD4, interact, and in general to understand mechanisms of HIV infectivity.

Inhibition of HIV Replication in Resting CD4+ Lymphocytes by Murr1

Gary J. Nabel et al. (NIAID)

U.S. Provisional Application No. 60/523,683 filed 21 Nov 2003 (DHHS Reference No. E–042–2004/0-US–01)

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

This technology describes the inhibition of HIV–1 growth in resting CD4+ T cells by Murr1, a highly conserved protein. This finding therefore could be used to prolong the asymptomatic phase of HIV infection. HIV–1 infects both proliferative and quiescent CD4+ T cells, although the virus replicates poorly in the latter. It has been demonstrated that Murr1 restricts HIV–1 replication by inhibiting basal and cytokine nuclear factor (NF)–κB activity. Short interfering RNAs (siRNAs) experiments that used specific Murr1 siRNAs resulted in lower levels of IκB–A and higher NF–κB activity and HIV–1 replication. These results allude to the potential for a more effective HIV therapeutic that uses Murr1 to regulate viral replication. A Murr1 anti-viral drug that can block viral replication in quiescent lymphocytes and latent cells with provirus might increase the number of patients that remain in the HIV–1 asymptomatic phase and thus lower the number that progress to the AIDS state.

This technology is further described in Ganesh *et al.*, Nature (18/25 December 2003), 426(6968): 853–857.

Mechanisms for Improving the Breadth of the Immune Response to Diverse Strains and Clades of HIV

Gary J. Nabel et al. (NIAID)

U.S. Provisional Application No. 60/503,509 filed 15 Sep 2003 (DHHS Reference No. E–335–2003/0-US–01)

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

This technology describes a multiclade Env vaccine candidate that elicited neutralizing antibodies to a diverse group of primary HIV–1 isolates as compared to antibodies generated from immunization with single clade vaccines. The immunogens of the vaccine included V3 loops from clades A, B, and C and had the cleavage site, fusion peptide, and interhelical regions deleted. Competition studies suggested that the neutralization activity is directed toward shared, conserved epitopes other than the V3 loop. Also described in this technology are immunogens involving deletion of the V3 loop that generated more potent neutralizing antibodies, suggesting that the highly conserved subregions within V3 may be relevant targets to elicit neutralizing antibody responses and increase the immunogenicity of HIV/AIDS vaccines. Such selective deletions in the V3 loop are effective in combination with deletions of other V loops. Immunogens with deletions of the V regions in general (V1—V4), including combinations of deletion immunogens, were also shown to elicit potent neutralizing antibodies. Previous studies of the cell-mediated immune response in mice using the multiclade vaccines of this current technology have shown that they induce Env-specific CD4 and CD8 immune response to

multiple clades. Thus, this technology offers promise in developing a globally effective HIV/AIDS vaccine, which must induce both cellular and humoral immunity to multiple strains from the various clades.

This work is described, in part, in Z. Yang *et al.*, J. Virol. (April 2004) 78(8): 4029–4036.

Methods for Inhibiting Proinflammatory Cytokine Expression Using Ghrelin

Drs. Vishwa D. Dixit, Dennis D. Taub, Eric Schaffer and Dzung Nguyen (NIA)

U.S. Provisional Patent Application filed 11 May 2004 (DHHS Reference No. E–016–2004/0–US–01)

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

Ghrelin, a recently described endogenous ligand for growth hormone secretagogue receptors (GHS–R), is produced from stomach serving as a potent circulating orexigen controlling energy expenditure, adiposity and GH secretion. We have discovered that ghrelin exerts anti-inflammatory effects by inhibiting the secretion of both acute and chronic cytokines including IL–1, IL–6, TNF–α, IFN–γ, IL–12 p40, , chemokines, and CSFs *in vitro* in human cells as well as *in vivo* in mouse model of sepsis and inflammation. We also found that ghrelin directly controls human growth hormone and insulin growth factor expression by human immune cells. This invention is useful for treatment of various inflammatory disorders including inflammatory bowel disease, Crohn's disease, rheumatoid arthritis, multiple sclerosis, atherosclerosis, endotoxemia and graft-versus-host disease.

Stem Cell Factor (SCF) Stimulates Neural Stem Cell Migration to Sites of Brain and Spinal Cord Injury

Howard A. Fine et al. (NCI)

U.S. Provisional Application No. 60/525,760 filed 26 Nov 2003 (DHHS Reference No. E–035–2004/0-US–01) and U.S. Provisional Application filed 19 Apr 2004 (DHHS Reference No. E–035–2004/1–US–01)

Licensing Contact: Fatima Sayyid; 301/435–4521; *ayyidf@mail.nih.gov.*

Endogenous neural stem/progenitor cells (NSPCs) have recently been recognized to hold the promise for therapeutics to combat neurodegenerative diseases, such as Parkinson's and Alzheimer's disease. Endogenous NSPCs have been shown to generate new functional neurons to replace the nerve cells that have been injured, lost, or destroyed in the diseases and recover brain functions. Such therapy, however, is limited due to lack of methods to mobilize endogenous NSPCs to the site of injury.

The present invention relates to methods for recruiting large numbers of NSPC to the specific site of neurological injury through local injection of recombinant or genetic vector-derived Stem Cell Factor (SCF). The inventors have identified that SCF secreted by nerve cells in the site of injury leads to migration of endogenous NSPCs to the site of injury and their proliferation to form neurons. The inventors have shown that local injection of recombinant SCF at the site of brain or spinal cord injury induces increased migration of NSPCs to the site of injury. Therefore, this invention could have significant commercial application in the development of therapeutic interventions including cell-based therapies for neurodegenerative diseases.

Dated: June 4, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–13100 Filed 6–9–04; 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 a meeting of the Board of Scientific Counselors, National Cancer Institute. The meeting will be closed to the public as indicated below in accordance with the provisions set forth in section 552b(c)(6), Title 5 U.S.C., as amended for the review, discussion, and evaluation of individual intramural programs and projects conducted by the National Cancer Institute, including consideration of personal qualifications and performance, and the competence of individual investigators, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: Board of Scientific Counselors, National Cancer Institute; Subcommittee 2—Basic Sciences.

Date: July 12, 2004.

Time: 8:30 a.m. to 4 p.m.

Agenda: To review and evaluate personal qualifications and performance, and competence of individual investigators.

Place: National Institutes of Health; National Cancer Institute; Building 31, Conference Room 6, 9000 Rockville Pike, Bethesda, MD 20892.

Time: 7 p.m. to 9 p.m.

Agenda: To review and evaluate personal qualifications and performance, and competence of individual investigators.

Place: Holiday Inn Bethesda, Versailles IV; 8120 Wisconsin Avenue, Bethesda, MD 20814.

Contact Person: Florence E. Farber, PhD, Health Scientific Administrator, Office of the Director, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, Room 2115, Bethesda, MD 20892, 301–496–7628, *ff6p@nih.gov.*

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

In the interest of security, NIH has instituted stringent procedures for entrance into the building by nongovernment employees. Persons without a government I.D. will need to show a photo I.D. and sign-in at the security desk upon entering the building.

(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: June 4, 2004.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 04–13157 Filed 6–9–04; 8:45 am]

BILLING CODE 4140–01–M

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 a meeting of the Board of Scientific Counselors, National Cancer Institute.

The meeting will be open to the public as indicated below, with attendance limited to space avilable. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should