(FDA). The meeting will be open to the public.

*Name of Committee*: Advisory Committee for Pharmaceutical Science.

*General Function of the Committee*: To provide advice and recommendations to the agency on FDA's regulatory issues.

*Date and Time*: The meeting will be held on April 13 and 14, 2004, from 8:30 a.m. to 5 p.m.

*Location*: Center for Drug Evaluation and Research Advisory Committee Conference Room, rm. 1066, 5630 Fishers Lane, Rockville, MD.

*Contact Person*: Hilda Scharen or Kimberly Topper, Center for Drug Evaluation and Research (HFD–21), Food and Drug Administration, 5600 Fishers Lane (for express delivery, 5630 Fishers Lane, rm. 1093), Rockville, MD 20857, 301–827–7001, e-mail: *SCHARENH@cder.fda.gov* or *TOPPERK@cder.fda.gov*, or FDA Advisory Committee Information Line, 1–800–741–8138 (301–443–0572 in the Washington, DC area), code 3014512539. Please call the Information Line for up-to-date information on this meeting.

*Agenda*: On April 13, 2004, the committee will receive an update from the Clinical Pharmacology Subcommittee. The committee will also discuss and provide comments on the following topics: (1) A proposal for resolving the issues related to the parametric tolerance interval test for dose content uniformity for inhalation products, (2) the Process Analytical Technologies progress and next steps, and (3) process analytical technology for products in the Office of Biotechnology Products, Center for Drug Evaluation and Research and in the Center for Biologics Evaluation and Research. On April 14, 2004, the committee will discuss and provide comments on the following topics: (1) Bioequivalence testing/methods strategy for products exhibiting high variability and (2) bioinequivalence concepts and definition.

*Procedure*: Interested persons may present data, information, or views, orally or in writing, on issues pending before the committee. Written submissions may be made to the contact person by April 6, 2004. Oral presentations from the public will be scheduled between approximately 1 p.m. and 2 p.m. on April 13, 2004, and 1 p.m. and 2 p.m. on April 14, 2004. Time allotted for each presentation may be limited. Those desiring to make formal oral presentations should notify the contact person before April 6, 2004, and submit a brief statement of the general nature of the evidence or arguments they wish to present, the names and addresses of proposed participants, and an indication of the approximate time requested to make their presentation.

Persons attending FDA's advisory committee meetings are advised that the agency is not responsible for providing access to electrical outlets.

FDA welcomes the attendance of the public at its advisory committee meetings and will make every effort to accommodate persons with physical disabilities or special needs. If you require special accommodations due to a disability, please contact Hilda Scharen or Kimberly Topper at least 7 days in advance of the meeting.

Notice of this meeting is given under the Federal Advisory Committee Act (5 U.S.C. app. 2).

Dated: March 17, 2004.

**Peter J. Pitts,**

*Associate Commissioner for External Relations.*

[FR Doc. 04–6484 Filed 3–23–04; 8:45 am]

**BILLING CODE 4160–01–S**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

### Government-Owned Inventions; Availability for Licensing

**AGENCY:** National Institutes of Health, Public Health Service, HHS.

**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.

**Cell-Based Assay of I-kappaB Kinase Activity**

Richard Eric Davis *et al.* (NCI).

DHHS Reference No. E–109–2004/0—Research Tool.

Licensing Contact: Mojdeh Bahar; (301) 435–2950; *baharm@mail.nih.gov.*

The present invention is directed to a cell-based assay of I-kappaB Kinase (IKK) Activity, providing a facile probe for cellular assays of activity/activation and drug screening. IKK activation is essential to diseases including certain types of cancer and undesired immune responsiveness such as autoimmunity and transplant rejection. In the assay, cells of interest are engineered to express an easily-measured exogenous form of IkB, driven by a promoter that is relatively independent of Nuclear factor of Kappa-B (NFkB) activity. Since the rate of synthesis of the ''reporter'' from the IkB is stable, its level is then principally determined by its rate of degradation, and therefore correlates inversely with IKK activity.

The assay has been used to screen molecules for their IKK inhibitor activity, determine whether the toxicity of agents towards NFkB-dependent lines occurs via the inhibition of IKK, and to determine whether genetic manipulations such as overexpression, exogenous mutants, or RNA interference affect constitutive or inducible IKK activity or activation.

Available materials include retroviral plasmids encoding the reporter or controls, and cell lines stably infected with these plasmids and validated in the assay. These reporter cell lines have IKK activity that is either constitutively high or easily inducible through various pathways (TNF-alpha, CD40L, IL–1beta, etc.).

**Specific Antibodies to the Lymphatic Endothelial Cell Specific Hyaluronic Acid Receptor, LYVE–1**

Sam T. Hwang, Adela R. Cardones (NCI).

DHHS Reference No. E–107–2004/0—Research Tool.

Licensing Contact: Mojdeh Bahar; 301/435–2950; *baharm@mail.nih.gov.*

This invention is drawn to specific anti-sera to human LYVE–1. LYVE–1 is a lymphatic endothelial cell-specific transmembrane form of CD44 that may have a role in recruitment of immune cells and cancer cells to lymphatics. Following skin-immunization of 10 mice with cDNA encoding human LYVE–1, the inventors have developed specific anti-sera to human LYVE–1. The anti-sera work well for flow cytometry and fluorescence microscopy and recognize native LYVE–1 epitopes. The antibody would be useful to scientists studying the role of lymphatic vessels in immune diseases and cancer. It may also be useful for clinical

pathologists. Commercially available LYVE–1 antibodies have drawbacks as they do not work well for flow cytometry analysis or tissue staining.

**Peptide Inhibitors of HIV–1 Integrase Useful for the Treatment of Retroviral Infection and HIV**

Drs. Peter Roller, Christophe Marchand, Krysztof Krajewski, Vinay K. Pathak, Yijun Zhang, and Yves Pommier (NCI).

U.S. Provisional Application No. 60/534,378 filed 06 Jan 2004 (DHHS Reference No. E–039–2004/0–US–01).

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

The invention describes the discovery of short peptides, derived from the natural peptide named indolicidin that have an ability to inhibit HIV–1 integrase and exhibit antiviral activity. In particular, this invention shows that synthesized derivatives of the indolicidin peptides named RIN–25 exhibit a significant higher anti-viral and anti-integrase activity when compared to the parent compound named RIN–42. HIV–1 integrase has a good potential of being the next therapeutic target since HIV–1 integrase is essential for viral replication and there is no cellular equivalent. Thus, subject invention may be used in the development of therapeutics for the treatment of retroviral infections, such as AIDS, or other retroviral-related diseases (*i.e.*, cancer, immune disorders). In addition, these novel peptides described in this invention may also have particular value when used in combination treatments with other antiviral therapies directed at other viral targets, such as protease and reverse transcriptase.

**Multipotent Postnatal Stem Cells From Human Periodontal Ligament and Uses Thereof**

Dr. Songtao Shi *et al.* (NIDCR).

U.S. Provisional Application filed 20 Nov 2003 (DHHS Reference No. E–033–2004/0–US–01).

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

It is estimated that over 40 percent of the adult population in the United States has periodontal disease in one form or another. Periodontal Disease is a chronic infection of the periodontal ligament (PDL) and the adjacent bone and cementum. The effects of Periodontal Disease range from simple gum inflammation to, in extreme cases, tooth loss.

The NIH announces a new technology wherein stem cells from the PDL have been isolated from adult human PDL. These cells are capable of forming cementum and PDL in immunocompromised mice. In cell culture, PDL stem cells differentiate into collagen fiber forming cells (fibroblasts), cementoblasts, and adipocytes. It is anticipated that these PDL stem cells will be useful for periodontal tissue regeneration to treat periodontal disease.

**A Novel Interferon-Gamma-Inducible Secretoglobin**

Anil B. Mukherjee *et al.* (NICHD).

U.S. Provisional Application No. 60/534,381 filed 06 Jan 2004 (DHHS Reference No. E–028–2004/0–US–01).

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

Interferons (IFNs) are a family of cytokines that are paramount in protecting the host from viral infections. The effects of the IFNs is mediated through interactions with specific cellular receptors, activation of second messenger systems effecting the expression of several antiviral and immunomodulatory proteins.

This invention describes a novel gene that is induced by IFN-gamma treatment of lymphoblast cells. This gene, termed IIS (IFN-inducible Secretoglobin), is a member of the uteroglobin (UG) superfamily and shares 30% amino acid identity with uteroglobin (UG), the founding member of the secretoglobin family of proteins. Data shows that IIS is expressed in virtually all tissues with highest levels found in lymph nodes, tonsils, lymphoblasts and ovary. IIS levels are also highly elevated in $CD8^+$ and $CD19^+$ cells. In further experiments, antisense-s-oligonucleotides to IIS are shown to prevent chemotactic migration and invasion of immune cells. Taken together, these data give insight into the immunological function of this novel IIS gene.

**Method Evolved for Recognition of Thrombophilia (MERT)**

Cigdem F. Dogulu *et al.* (NICHD).

DHHS Reference No. E–282–2003/0–US–01 filed 15 Jan 2004.

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

Venous thrombosis affects 1 per 1000 individuals annually and is one of the leading causes of mortality and morbidity resulting in approximately 300,000 hospitalizations and 50,000 fatalities per year in the United States alone.

Although venous thrombosis is one of the leading causes of morbidity and mortality in developed countries, it is an avoidable disease by the use of prophylactic treatment that is currently available. To avoid the development of venous thromboembolism, it is beneficial to estimate the individual thrombotic risk to develop stratification protocols for an individual risk-adapted prophylaxis.

This invention proposes methods to predict an individual's genetic susceptibility to venous thrombosis, as well as arrays and kits that can be used to practice such methods. The method includes screening for combinations of mutations and polymorphisms in venous thrombosis-related molecules such as factor V, prothrombin (factor II), fibrinogen, protein C, protein S, antithrombin III, angiotensin I-converting enzyme (ACE) and methylenetetrahydrofolate reductase (MTHFR) that allow one to predict the genetic susceptibility of an individual to developing venous thrombosis with high accuracy in several ethnic populations.

**Stimulation or Inhibition of Gamma Delta T-Cells To Promote or Inhibit Bone Growth**

Dr. Nona T. Colburn (NIAMS).

U.S. Provisional Application filed 07 Nov 2003 (DHHS Reference No. E–277–2003/0–US–01).

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

Bone injury is a common occurrence that disables people from working and otherwise carrying on their daily lives and is an important health concern. When fracture healing does not go as intended, the ability of a bone to heal without a scar is lost. This can result in disfigurement, pain, and loss or impairment of bodily function. It has been shown that within the segment of the clinical population afflicted with non-healing fractures, a large number of these non-unions are a direct result of the patient's immune system status. Bone repair involves a series of phases, the first of which is the initial inflammatory phase controlled by the body's innate immune response. Of those first responder cells are the gamma delta T-cells that are capable of recognizing products of cell damage. This technology relates to the stimulation or inhibition of the gamma delta T-cells to modulate bone growth. It is anticipated that the manipulation of the gamma delta T-cell population will reduce the need to perform operations such as bone grafting which are currently used to treat non-unions.

**Methods for Inhibiting HIV and Other Viral Infections by Modulating Ceramide Metabolism**

Robert Blumenthal, Catherine M. Finnegan (NCI).

U.S. Provisional Application No. 60/528,411 filed 09 Dec 2003 (DHHS Reference No. E–265–2003/0–US–01).

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

This invention provides methods of inhibiting or preventing HIV–1 infections by inducing either the de novo biosynthesis of ceramide, or by activating enzymes (e.g., sphingomyelinase) involved in the generation of ceramide at the plasma membrane, or by direct incorporation of exogenous ceramide into target cell membranes. The invention describes methods for administration of a retinamide compound, particularly an N–(aryl) retinamide compound such as N–(4–hydroxyphenyl) retinamide (4–HPR) resulting in increased plasma membrane ceramide levels, which results in the inhibition of HIV–1 infection in monocyte/macrophages by perturbing membrane organization. In addition, because of its low toxicity in non-tumor cells, 4–HPR and related compounds are particularly suitable for long-term preventative or therapeutic administration to subjects suffering from an HIV infection or who are at risk of contracting an HIV infection. Thus, this invention provides a novel means of treating or inhibiting HIV and other viral infections by administering a retinamide compound to a patient suffering from or susceptible to such a viral infection.

**ELISA Assay of Serum Soluble CD22 To Assess Tumor Burden/Relapse in Subjects With Leukemia and Lymphoma**

Robert Kreitman *et al.* (NCI).

PCT Application No. PCT/US03/16298 filed 20 May 2003 (DHHS Reference No. E–065–2002/0–PCT–02), with priority to 20 May 2002.

Licensing Contact: Jesse Kindra; (301) 435–5559; *kindraj@mail.nih.gov.*

Disclosed are methods of using previously unknown soluble forms of CD22 (sCD22) present in the serum of subjects with B-cell leukemias and lymphomas to assess tumor burden in the subjects. Also disclosed are methods of diagnosing or prognosing development or progression of a B-cell lymphoma or leukemia in a subject, including detecting sCD22 in a body fluid sample taken or derived from the subject, for instance, serum. In some embodiments, soluble CD22 levels are quantified. By way of example, the B-cell lymphoma or leukemia can be hairy cell leukemia, chronic lymphocytic leukemia, or non-Hodgkin's lymphoma. Soluble CD22 in some embodiments is detected by a specific binding agent, and optionally, the specific binding agent can be detectably labeled.

Also disclosed are methods of selecting a B-cell lymphoma or leukemia therapy that include detecting an increase or decrease in sCD22 levels in a subject compared to a control, and, if such increase or decrease is identified, selecting a treatment to prevent or reduce B-cell lymphoma or leukemia or to delay the onset of B-cell lymphoma or leukemia.

Other embodiments are kits for measuring a soluble CD22 level, which kits include a specific binding molecule that selectively binds to the CD22, e.g. an antibody or antibody fragment that selectively binds CD22.

Further disclosed methods are methods for screening for a compound useful in treating, reducing, or preventing B-cell lymphomas or leukemias, or development or progression of B-cell lymphomas or leukemias, which methods include determining if application of a test compound lowers soluble CD22 levels in a subject, and selecting a compound that so lowers sCD22 levels.

**C–C Chemokines That Inhibit Retrovirus Infection**

Paolo Lusso, Robert C. Gallo, Fiorenza Cocchi, Anthony L. De Vico, Alfredo Garzino-Demo (NCI).

PCT Application No. PCT/US96/18993 filed 27 Nov 1996 (DHHS Reference No. E–008–1996/0–PCT–02); U.S. Patent Application No. 09/077,614 filed 29 May 1998 (DHHS Reference No. E–008–1996/0–US–04) (with priority to 30 Nov 1995).

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

This invention concerns three members of the human C–C chemokine family, RANTES, macrophage inflammatory protein 1alpha (MIP–1alpha) and macrophage inflammatory protein 1beta (MIP–1beta), which are produced and secreted by several cell types, including CD8-positive T lymphocytes, and which act in vitro as HIV suppressive factors. These factors and their respective genes may be used in the diagnosis, prognosis, treatment and prevention of AIDS and other retrovirus-induced diseases. The invention provides a therapeutic preparation, methods for therapeutic and prophylactic treatment of retroviral infection, and a method of prognosis for retroviral infection. The technology was reported in Science 270(8):1560–1561 (December 8, 1995).

Dated: March 18, 2004.

**Steven M. Ferguson,**

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

[FR Doc. 04–6608 Filed 3–23–04; 8:45 am]

**BILLING CODE 4140–01–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

### National Institute of Diabetes and Digestive and Kidney Diseases; 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 Institute of Diabetes and Digestive and Kidney Diseases Special Emphasis Panel, Extramural Loan Repayment Program.

*Date:* April 13, 2004.

*Time:* 2 p.m. to 4 p.m.

*Agenda:* To review and evaluate grant applications.

*Place:* National Institutes of Health, Two Democracy Plaza, 6707 Democracy Boulevard, Bethesda, MD 20892 (Telephone Conference Call).

*Contact Person:* D.G. Patel, PhD, Scientific Review Administrator, Review Branch, DEA, NIDDK, National Institutes of Health, Room 747, 6707 Democracy Boulevard, Bethesda, MD 20892, (301) 594–7682, *pateldg@extra.niddk.nih.gov.*

This notice is being published less than 15 days prior to the meeting due to the timing limitations imposed by the review and funding cycle.

(Catalogue of Federal Domestic Assistance Program Nos. 93.847, Diabetes, Endocrinology and Metabolic Research; 93.848, Digestive Diseases and Nutrition Research; 93.849, Kidney Diseases, Urology and Hematology Research, National Institutes of Health, HHS)