October 1, 1995, unless it displays a currently valid OMB control number.

Proposed Collection: Title: A Process/Outcome Evaluation of Parkinson's Disease Research Centers *Type of Information Collection Request:* NEW. *Need and Use of Information Collection:* This study is primarily an outcome evaluation, designed to assess the extent to which the NINDS-funded Morris K. Udall Centers for Excellence in Parkinson's Disease Research have achieved the program's short-term and long-term goals. The study also includes elements of a process evaluation in its examination of the major activities conducted by the Udall Centers, the relationship between Center activities and the achievement of program goals, and the NINDS management of the program. The results of the full-scale evaluation should be very helpful to NINDS in identifying the most relevant measures for tracking the future progress of the Centers, developing strategies to enhance the program's effectiveness, and improving program management. NINDS will also use the findings to inform its National Advisory Neurological Disorders and Stroke Council, and to address inquiries from the public regarding the impact of the Udall Centers Program. Lastly, Udall Center awardees will be able to use the evaluation results to improve the performance of their Centers; and other NIH Institutes and Centers may use the methodology and results of this evaluation to guide their own centers assessments. *Frequency of Response:* Once or twice. *Affected Public:* Researchers, Not-for-profit institutions; Federal Government; individuals or households. *Type of Respondents:* Adult professionals.

The annual reporting burden is provided in the following table:

Type of respondents	Estimated number of respondents	Estimated frequency of response	Estimated average time per response	Estimated annual hour burden
Center Directors	13	2	1.5	39
Project/Core Directors	54	2	1.5	162
Comparison Group	54	1	1.0	54
Totals	121			255

There are no Capital Costs to report. There are no Operating or Maintenance Costs to report.

Request for Comments: Written comments and/or suggestions from the public and affected agencies should address one or more of the following points: (1) Evaluate whether the proposed collection of information is necessary for the proper performance of the function of the agency, including whether the information will have practical utility; (2) Evaluate 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) Enhance the quality, utility, and clarity of the information to be collected; and (4) Minimize the burden of the collection of information on those who are to respond, including the use of appropriated automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

FOR FURTHER INFORMATION CONTACT: Written comments and/or suggestions regarding the item(s) contained in this notice, especially regarding the estimated public burdenand associated response time, should be directed to the: Office of Management and Budget, Office of Regulatory Affairs, New Executive Office Building, Room 10235, Washington, DC 20503, Attention: Desk Officer for NIH. To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact: Dr. Melinda Kelley, Office of Science Policy and Planning, National Institute of Neurological Disorders and Stroke, NIH, Building 31, 31 Center Drive, Room 8A–03, Bethesda, MD 20892; call non-toll-free 301–496–9271; or E-mail your request, including your address to: *ospp@ninds.nih.gov.*

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 30-days of the date of this publication.

Dated: May 11, 2006.

Story C. Landis,

Director, NINDS, National Institutes of Health.

[FR Doc. E6–7626 Filed 5–18–06; 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, 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.

Viral Entry or Replication Inhibition Using siRNA, Small Molecules, or Other Tec Tyrosine Kinase Inhibitors

Julie Readinger and Pamela L. Schwartzberg (NHGRI).

U.S. Provisional Application filed 29 Mar 2006 (HHS Reference No. E–151–2006/0–US–01).

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

The Tec family of tyrosine kinases, consisting of five family members Tec, Btk, Itk, Rlk, and BMX, are key regulators of signaling pathways of T lymphocytes. Many existing antiviral therapies rely on inhibition of viral replication, which leads to emergence or selection of resistant viruses. The current technology provides an alternative target for the prevention or treatment of viral infection through administration of a Tec tyrosine kinase inhibitor. Such inhibitors can be siRNA, small chemical compounds, antisense or antibody. The current technology describes the inhibition of Itk (also

known as Emt and Tsk) and the resulting decrease in HIV infectivity, replication, and transcription for exemplary purposes. Importantly, inhibition of Itk expression does not affect the expression of HIV receptors CCR5, CXCR4, or CD4. The current technology could be used in combination with therapeutics that target multiple stages of the virus life cycle.

This research is described, in part, in the following:

1. D Dombroski, R Houghtling, CM Labno, J Burkhardt, and PL Schwartzberg, ''Kinase-independent functions for Itk in the regulation of Vav and the actin cytoskeleton,'' J. Immunol. 174: 1385–1392, 2005.

2. A Takesono, R Horai, M Mandai, D Dombroski, and PL Schwartzberg, ''Requirement for Tec family kinases in chemokine-induced migration and activation of Cdc42 and Rac,'' Curr. Biol. 10:917–22, 2004.

3. E Schaeffer, G Yap, CM Lewis, M Czar, DW McVicar, AW Cheever, A Sher, and PL Schwartzberg, ''Mutation of Tec family kinases alters T helper cell differentiation,'' Nature Immunol. 2:1183–8, 2001.

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors: The NHGRI, Genetic Disease Research Branch/Cell Signalling Section, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the use of Tec family kinase inhibitors as a therapeutic target for HIV and other viral infections. Please contact Claire Driscoll, Director, NHGRI Technology Transfer Office, at 301/402–2537 or *cdriscol@mail.nih* for more information.

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

Peter P. Roller et al. (NCI)

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

U.S. Provisional Application No. 60/547,067 filed 25 Feb 2004 (HHS Reference No. E–039–2004/1–-US–01).

U.S. Provisional Application No. 60/599,856 filed 10 Aug 2004 (HHS Reference No. E–039–2004/2–US–01).

PCT Application No. PCT/US2004/42726 filed 21 Dec 2004 (HHS Reference No. E–039–2004/3–PCT–01), which published as WO 2005/068492 on 29 Dec 2005.

Licensing Contact: Sally Hu, Ph.D., M.B.A.; 301/435–5606; *hus@mail.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, the 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.

Identification of Candidate Ligands which Modulate Antigen Presenting Cells

Polly Matzinger, John P. Ridge (NIAID).

U.S. Patent No. 6,680,176 issued 20 Jan 2004 (HHS Reference No. E–055–1999/0–US–01).

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

Available for licensing and commercial development are novel biotechnological tools, prophylactics, therapeutics, and methods for modulating the activation state of an antigen presenting cell (APC) and thereby modulating the activation of a killer T cell. The activation of a killer T cell can occur in a two cell complex and two sequential steps: (a) In the first step, an APC stimulates a T helper T cell, which in turn stimulates or ''superactivates'' the APC to differentiate to a state where it can independently stimulate a killer T cell; (b) In the second step, the APC encounters the killer T cell and stimulates it so that killer T cell priming is achieved in a helper independent fashion. The first step can be bypassed altogether by viral infection or an interaction with certain molecules at the cell surface of APCs, such as CD40. More specifically, the invention consists of a method of identifying a ligand as a candidate for incorporation into a pharmaceutical composition, such as a therapeutic or prophylactic product, that modulates antigen presenting cell activity, comprising contacting an APC with a candidate ligand which interacts with the APC, analyzing the activation state of the APC; and selecting ligands that activate a killer T cell in the absence of a helper T cells as the candidates for incorporation into the pharmaceutical. Also claimed are related methods where the ligand interacts with CD40 or where the APC is a dendritic cell. The embodiments have several applications in the field of immunology, and enable to manufacture novel pharmaceuticals and vaccine components for the treatment and prevention of cancer, systemic infection, and autoimmune responses.

The technology is further described in JP Ridge, F Di Rosa, and P Matzinger, ''A conditioned dendritic cell can be a temporal bridge between a CD4+ T-helper and a T-killer cell,'' Nature 1998 Jun 4; 393(6684):474–8.

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

Dated: May 11, 2006.

David R. Sadowski,

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

[FR Doc. E6–7627 Filed 5–18–06; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Amended Notice of Meeting

Notice is hereby given of a change in the meeting of the Center for Scientific Review Special Emphasis Panel, June 13, 2006, 3 p.m. to June 13, 2006, 4:30 p.m., National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892 which was published in the **Federal Register** on May 5, 2006, 71 FR 26550–26552.

The meeting will be held on June 15, 2006. The meeting time and location remain the same. The meeting is closed to the public.

Dated: May 11, 2006.

Anna Snouffer,

Acting Director, Office of Federal Advisory Committee Policy.

[FR Doc. 06–4673 Filed 5–18–06; 8:45 am]

BILLING CODE 4140–01–M