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]

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