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.

**Inhibitors of HIV Ribonuclease H Antiviral Properties**

Drs. John Beutler, Stuart LeGrice, Scott Budihas, Antony Wamiru, Roberta Gardella, and Jennifer Wilson (all of NCI); Dr. Michael Parniak (EM)


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

The invention describes a class of compounds that inhibit HIV RNase H and the methods of using these compounds for the treatment of HIV infections. More specifically, these compounds are vinylogous urea derivative containing substituted thiophene core structures and these compounds were part of the 100,000 member library of compounds purchased by NCI from ChemBridge. The selectivity of the antiviral activity was demonstrated in their selective inhibition of HIV–1 and HIV–2 Rnaase H enzymes 1 in the CEM cell line of CD4+ lymphoblast cells. Five members of this class of compounds were able to block the cytopathic effect of the virus at concentrations that did not inhibit cell growth. Thus, these compounds may be used in the development of therapeutics for the treatment of retroviral infections, such as AIDS. In addition, these compounds described in this invention may also have particular value when used in combination treatments with other antiviral therapies directed at the virus itself or when used in combination with other antiviral therapies directed at viral targets, such as protease and integrase.

**Protozoan Derived Antagonist of CCR5**

Drs. Alan Sher, Julio Aliberti, Jose Ribeiro, and John Anderson (all of NIAID); Dr. Hana Golding (FDA)


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

The invention describes the anti-HIV properties of cyclophilin-18, a protein expressed by the protozoan parasite Toxoplasma gondii. The protein was found to bind to the chemokine receptor CCR5 which is also a co-receptor for the HIV virus. Both the native and recombinant molecules display inhibitory activity in HIV–1 fusion (syncitia formation) and infectivity assays with human T cells and macrophages. Thus, Toxoplasma gondii cyclophilin-18 or modified versions of the molecule may be used in the development of treatment for AIDS. In particular, the protein described in this invention may have particular value when used as a microbicide for blocking initial HIV infection. More details of this invention can be found in Golding et al., “Inhibition of HIV–1 Infection by a CCR5 Binding Cyclophilin from Toxoplasma gondii,” Blood 1 Nov 2003 102(9): 3280–3286.

**Treatment of Human Viral Infections (Resveratrol)**

Drs. Steven Zeichner and Vyjayanthi Krishnan (NCI)


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

This application describes the methods for treating or preventing an HIV infection by the administration of an Egr 1 activator called Resveratrol (3, 5, 4′-trihydroxystilbene) and its derivatives. It has been known that HIV, once it infects a cell, integrates into the cellular genome and can (1) rapidly undergo lytic infection, or (2) lay dormant for a period of time (latent infection). The existence of latent infected cells poses a great challenge to HIV therapy because (1) there are no good existing means that can separate the latent infected cells from the uninfected cells; (2) even when antiretroviral drugs are able to completely suppress detectable HIV replication, these latent infected cells will remain and HIV can subsequently complete the viral replication cycle to produce more virus. Since Resveratrol and its derivatives can activate lytic replication from latent infected cells via its effects on Erk1/2 signaling, Resveratrol and its derivatives may lead to therapies in which double control and/or its derivatives is given together with highly active antiretroviral therapy in an...
effort to decrease or eliminate the reservoir of latent infected cells with hope of perhaps eventually curing a patient of HIV infection.

**Treatment of Human Viral Infections (Proteosome Inhibitors)**

Drs. Steven Zeichner and Vyjayanthi Krishnan (NCI)


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

This application describes the methods for treating or preventing an HIV infection by the administration of proteosome inhibitors and their derivatives. It has been known that HIV, once it infects a cell, integrates into the cellular genome and can (1) rapidly undergo lytic infection, or (2) lay dormant for a period of time (latent infection). The existence of latent infected cells poses a great challenge to HIV therapy because (1) there are no good existing means that can separate the latent infected cells from the uninfected cells; (2) even when antiretroviral drugs are able to completely suppress detectable HIV replication, these latent infected cells will remain and HIV can subsequently complete the viral replication cycle to produce more virus. Since imatinib and its derivatives can activate lytic replication from latent infected cells by activating NF–κB, imatinib and its derivatives may lead to therapies in which imatinib and/or its derivatives is given together with highly active antiretroviral therapy in an effort to decrease or eliminate the reservoir of latent infected cells with hope of perhaps eventually curing a patient of HIV infection.

**Treatment of Human Viral Infections (Farnesyl Transferase Inhibitors)**

Drs. Steven Zeichner and Vyjayanthi Krishnan (NCI)


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

This application describes the methods for treating or preventing an HIV infection by the administration of farnesyl transferase inhibitors such as FTI277, L–744832, BMS214662, R115777 and SCH66336. It has been known that HIV, once it infects a cell, integrates into the cellular genome and can (1) rapidly undergo lytic infection, or (2) lay dormant for a period of time (latent infection). The existence of latent infected cells poses a great challenge to HIV therapy because (1) there are no good existing means that can separate the latent infected cells from the uninfected cells; (2) even when antiretroviral drugs are able to completely suppress detectable HIV replication, these latent infected cells will remain and HIV can subsequently complete the viral replication cycle to produce more virus. Since farnesyl transferase inhibitors can activate lytic replication from latent infected cells by modulating membrane-bound Ras–Rho levels, farnesyl transferase inhibitors may lead to therapies in which farnesyl transferase inhibitor is given together with highly active antiretroviral therapy in an effort to decrease or eliminate the reservoir of latent infected cells with hope of perhaps eventually curing a patient of HIV infection.


**Steven M. Ferguson,**

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

[FR Doc. 04–23650 Filed 10–21–04; 8:45 am]

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**DEPARTMENT OF HEALTH AND HUMAN SERVICES**

**National Institutes of Health**

**National Institute on Drug Abuse; 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 on Drug Abuse Special Emphasis Panel. Member Conflict Meeting.

**Date:** November 17, 2004.

**Time:** 6 p.m. to 8 p.m.

**Agenda:** To review and evaluate grant applications.

**Place:** Ritz-Carlton Hotel at Pentagon City, 1250 South Hayes Street, Arlington, VA 22202.

**Contact Person:** Mark Swieter, PhD, Health Scientist Administrator, Office of Extramural Affairs, National Institute on Drug Abuse, National Institutes of Health. DHHS, 6101 Executive Boulevard, Suite 220, Bethesda, MD 20892–8401, (301) 435–1389.


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.

DEPARTMENT OF HEALTH AND HUMAN SERVICES

**National Institutes of Health**

**National Institute of General Medical Sciences; 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.

**LaVerne Y. Stringfield,**

*Director, Office of Federal Advisory Committee Policy.*

[FR Doc. 04–23654 Filed 10–21–04; 8:45 am]

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