starting pre-clinical studies of the conjugates using animal cancer models. In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Maleimide Anti-Tumor Phosphatase Inhibitors

Christopher J. Michejda et al. (NCI).

Some compounds of the current invention are more water soluble compared to normal cells. They exhibit greater specificity for inactivating O6-alkylguanine-DNA alkyltransferase in certain tumor cells, compared to normal cells.

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

Inhibitors of the Protein Kinase Chk2 to Abrogate Apoptosis and Sensitize Cancer Cells to DNA Targeted Therapies

Yves Pommier et al. (NCI).

Chk2 is a protein kinase activated in response to DNA double strand breaks. In normal tissues, Chk2 phosphorylates and thereby activates substrates that induce programmed cell death, or apoptosis, via interactions with p53, E2F1, PML proteins. In cancer tissues, where apoptosis is suppressed, Chk2 phosphorylates and inactivates cell cycle checkpoints (via interactions with Cdc25, phosphatases and Brca1 proteins), which allows cancer cells to repair and tolerate DNA damage. Hence, Chk2 inhibitors would be expected to protect normal tissues by reducing apoptosis, and to sensitize cancer cells to DNA-targeted agents.

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


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

[FR Doc. 05–17457 Filed 9–1–05; 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.
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.

**Method for Inducing T-Cell Proliferation**

Warren J. Leonard et al. (NHBLI).


This technology relates to the use of thymic stromal lymphopoietin (TSLP) or TSLP agonists to induce CD4+ T cell proliferation as well as the use of TSLP antagonists to treat IgE-mediated disorders such as asthma or allergies. The T cell proliferation application of this technology could be of particular relevance for patients in whom this cell population has been significantly reduced by e.g., HIV/AIDS infection or another condition resulting in immunodeficiency. The patent application describes methods of treating individuals afflicted with an immunodeficiency by administering CD4+ T cells that have been isolated and induced to proliferate using TSLP or by direct administration of TSLP or a nucleic acid encoding TSLP. The need for appropriate treatment methods for conditions such as asthma and allergies are well recognized. The patent application describes administration of a TSLP antagonist to an individual suffering from an IgE-mediated disorder to remove or lessen the symptoms. TSLPR knockout mice are also described in the patent application and available for licensing through a biological materials license agreement.

**Vaccines Using Universally Inactivated Viruses, Parasites, and Tumor Cells**

Yossef Raviv et al. (NCI).


This technology relates to improving levels of gene expression using a combination of a constitutive RNA transport element (CTE) with a mutant form of another RNA transport element (RTE). The combination of these elements results in a synergistic effect on stability of mRNA transcripts, which in turn lead to increased expression levels. Using HIV–1 gag as reporter mRNA, one mutated RTE in combination with a CTE was found to improve expression of unstable mRNA by about 500-fold. Similarly this combination of elements lead to synergistically elevated levels of HIV–1 Env expression. The function of CTEs and RTEs is conserved in mammalian cells, so this technology is a simple and useful way of obtaining high levels of expression of otherwise poorly expressed genes and can be used in a number of applications such as but not limited to improvements of gene therapy vectors, expression vectors for mammalian cells.

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

**Recombinant Plasmids Containing HIV Reverse Transcriptase**

Stephen H. Hughes and Paul L. Boyer (NCI).

HHS Reference Nos. E–303–1991/0, /1, /2, /3, and /4—Research Tools. Licensing Contact: Sally Hu; 301/435–5606; hus@mail.nih.gov.

NIH offers below HIV–1 Reverse Transcriptase (RT) Expression plasmids that are available for licensing via biological material licenses (BML). In an appropriate strain of E. coli, these plasmids cause the expression of an HIV–1 RT heterodimer (p66/p51). In the expression plasmid, the RT coding region is flanked by synthetic initiation and termination codons. The amino terminus of the RT made in E. coli has two additional amino acids relative to the viral enzyme (MV); these have no obvious effect on enzymatic activity. The carboxy terminus of p66 carries a 6-histidine tag that facilitates purification. The plasmid also causes the expression of a low level of HIV–1 protease; this leads to the conversion of the approximately half of the p66 synthesized in E. coli to p51. The p66/p51 heterodimer can be easily extracted from the E. coli host and purified by metal-chelate chromatography. Expression constructs for many of the common drug-resistant versions of HIV–1 RT (a partial list is given below) and for a number of other mutants are available. Alternate RT expression plasmids that encode versions of HIV–1 RT that do not have his tags and plasmids that separately encode p51 and p66 (allowing subunit selective mutagenesis) are also available. The HIV–1 RT expression plasmids can be used to generate wild-type and drug resistant RTs that can be used in both biological and clinical research. The RTs are particularly useful in the screening and development of RT
inhibitors in vitro and can be used to test drug candidates for their effectiveness against common drug resistant mutants of HIV–1 RT. Please contact Dr. Hughes directly (hughes@ncifcrf.gov) if you want additional information about RT expression plasmids that are not listed below.

<table>
<thead>
<tr>
<th>Vector</th>
<th>Description</th>
<th>Reference No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wild-type HIV–1 RT</td>
<td>full length, wild type</td>
<td>E–034–1991/0</td>
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<tr>
<td>L100I</td>
<td>NNRTI resistant</td>
<td>E–034–1991/1</td>
</tr>
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<td>K103N</td>
<td>NNRTI resistant</td>
<td>E–034–1991/1</td>
</tr>
<tr>
<td>V106A</td>
<td>NNRTI resistant</td>
<td>E–034–1991/1</td>
</tr>
<tr>
<td>V108I</td>
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<td>E–034–1991/1</td>
</tr>
<tr>
<td>E138K</td>
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<td>E–034–1991/1</td>
</tr>
<tr>
<td>Y181I</td>
<td>NNRTI resistant</td>
<td>E–034–1991/1</td>
</tr>
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<td>Y181C</td>
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<td>E–034–1991/1</td>
</tr>
<tr>
<td>Y188L</td>
<td>NNRTI resistant</td>
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</tr>
<tr>
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<td>NNRTI resistant</td>
<td>E–034–1991/1</td>
</tr>
<tr>
<td>G190A</td>
<td>NNRTI resistant</td>
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</tr>
<tr>
<td>G190S</td>
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</tr>
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</tr>
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<td>NRTI resistant</td>
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<td>T69G</td>
<td>NRTI resistant</td>
<td>E–034–1991/1</td>
</tr>
<tr>
<td>L74V</td>
<td>NRTI resistant</td>
<td>E–034–1991/1</td>
</tr>
<tr>
<td>M184I</td>
<td>Lamivudine (3TC) resistant</td>
<td>E–034–1991/1</td>
</tr>
<tr>
<td>M184I</td>
<td>Lamivudine (3TC) resistant</td>
<td>E–034–1991/1</td>
</tr>
<tr>
<td>AZT–R (6 mutations)</td>
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<td>E–034–1991/1</td>
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<tr>
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<td>Multi-NRTI resistant</td>
<td>E–034–1991/1</td>
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<tr>
<td>Q151M</td>
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<td>E–034–1991/1</td>
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<tr>
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<tr>
<td>SSSR/T215Y</td>
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<td>E–034–1991/1</td>
</tr>
</tbody>
</table>

RTs that carry some combinations of NNRTI mutations, e.g., K103N+Y181I, are also available.

Dated: August 20, 2005.

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

[FR Doc. 05–17517 Filed 9–1–05; 8:45 am]

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Center on Minority Health and Health Disparities; 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 by the National Advisory Council on Minority Health and Health Disparities. The meeting will be open to the public as indicated below, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the 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 Advisory Council on Minority Health and Health Disparities.

Date: September 20, 2005.

Closed: 8:30 a.m. to 10 a.m.

Agenda: To review and evaluate grant applications and/or proposals.

Place: National Institutes of Health, Two Democracy Plaza, 6707 Democracy Boulevard, Suite 800, Bethesda, MD 20892.

Open: 10:30 a.m. to 5:30 p.m.

Contact Person: Donna Brooks, Asst. Director for Administration, National Center on Minority Health and Health Disparities, National Institutes of Health, 6707 Democracy Blvd., Suite 800, Bethesda, MD 20892, 301–435–2135, brooksda@ncmhd.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.


Anthony M. Coelho, Jr.,
Acting Director, Office of Federal Advisory Committee Policy.

[FR Doc. 05–17514 Filed 9–1–05; 8:45 am]

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Eye 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 National Advisory Eye Council.

The meeting will be open to the public as indicated below, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the 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.,