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.

**Maleiimide Anti-Tumor Phosphatase Inhibitors**

Christopher J. Michejda *et al.* (NCI).
U.S. Provisional Application No. 60/546,841 filed 22 Feb 2004 (HHS Reference No. E–110–2004/0–US–01).
PCT Application No. PCT/US05/05742 filed 22 Feb 2005 (HHS Reference No. E–110–2004/0–PCT–02).

The present invention describes novel phosphatase inhibitors that appear to target the CDC25 family of phosphatases. The new compounds have potent activity against human liver cancer cells in vitro and in vivo against an orthotopic liver cancer in rats. In tumor cells, these new inhibitors appear to target the phosphorylation status of several cell cycle proteins that are important for cell survival and thus could represent a novel class of chemotherapeutic agents targeting cancer cells.

**2-Amino-O4-Substituted Pteridines and Their Use as Inactivators of O6-Alkylguanine-DNA Alkyltransferase**

Robert C. Moschel *et al.* (NCI).
U.S. Provisional Application No. 60/534,519 filed 06 Jan 2004 (HHS Reference No. E–274–2003/0–US–01).
PCT Application No. PCT/US04/41577 filed 10 Dec 2004 (HHS Reference No. E–274–2003/0–PCT–02).

This invention is directed to 2-amino-O4-benzylpteridine derivatives targeted for use in cancer treatment in combination with chemotherapeutic agents such as 1,3-bis(2-chloroethyl)-1-nitrosurea (BCNU) or temozolomide. The derivatives of the present invention inactivate the O6-alkylguanine-DNA-alkyltransferase repair protein and thus enhance activity of such chemotherapeutic agents. Examples of these derivatives have advantages over the earlier O6-benzylguanine compounds from this research group. Some compounds of the current invention are more water soluble compared to O6-benzylguanine and 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.

**Beta-Glucuronidase Cleavable Prodrugs of O6-Alkylguanine-DNA Alkyltransferase Inactivators**

Robert C. Moschel *et al.* (NCI).
U.S. Provisional Application No. 60/608,045 filed 08 Sep 2004 (HHS Reference No. E–307–2004/0–US–01).

The present invention relates to prodrugs of inactivators of O6-alkylguanine-DNA alkyltransferase. The prodrugs are cleaved by the beta-glucuronidase enzyme found in tumor cells or co-administered to the patient, and the drugs are targeted for use in cancer treatment in combination with antineoplastic alkylating agent such as 1,3-bis(2-cloroethyl)-1-nitrosouria or temozolomide.

**Identification of a Tricyclic Amino Amide (NSC–644221) Inhibitor of the Hypoxic Signaling Pathway**

Giovanni Melillo (NCI).
U.S. Provisional Application No. 60/618,279 filed 12 Oct 2004 (HHS Reference No. E–185–2004/0–US–01).
U.S. Provisional Application No. 60/570,615 filed 12 May 2004 (HHS Reference No. E–185–2004/1–US–01).
PCT Application filed 11 May 2005 (HHS Reference No. E–185–2004/2–PCT–01).

This invention describes the identification of a tricyclic (1,4-dioxane) amino amide with confirmed potent activity in inhibiting HIF–1 transcriptional activity.

HIF–1 is a transcription factor and plays an important role in adaptation of cancer cells to an hypoxic environment. HIF–1 significantly increases the ability of cancer cells to survive under strenuous conditions. It contributes to the ability of cancer cells to migrate and invade surrounding tissue, and is important for the formation of new blood vessels that are essential for growth and metastasis of cancer cells. Thus HIF–1 mediates survival and spreading of cancer cells. Previous studies have shown that HIF–1 is also important in human cancers, and therefore, inhibition of HIF–1 activity is contemplated in the field as a therapy for cancer patients.

The inventors, using a cell-based high throughput screen, identified a new compound, NSC–644221, with potent inhibitory activity of the HIF–1 pathway. The compound inhibits expression of HIF–1 and reduces its accumulation in the cell. This compound also inhibits expression of endogenous genes that are under control of HIF–1, such as Vascular Endothelial Growth Factor (VEGF) that is essential for the formation of new blood vessels. Preliminary experiments in xenograft models have indicated that NSC–644221 reaches the tumor tissue when administered intraperitoneally and inhibits HIF–1-dependent luciferase expression in U251–HRE 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).
U.S. Provisional Application filed 29 Jul 2005 (HHS Reference No. E–211–2005/0–US–01).

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.

Dated: August 25, 2005.

**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. 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.* (NHLBI).
U.S. Provisional Application No. 60/555,898 filed 23 Mar 2004 (HHS Reference No. E–104–2004/0–US–01); U.S. Utility Application No. 11/084,408, filed 18 Mar 2005 (HHS Reference No. E–104–2004/0–US–02).
Licensing Contact: Susan Ano; 301/435–5515; *anos@mail.nih.gov.*

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).
U.S. Provisional Application filed 22 Mar 2004 (HHS Reference No. E–303–2003/0–US–01); PCT Application filed 22 Mar 2005 (HHS Reference No. E–303–2003/0–PCT–02).
Licensing Contact: Susan Ano; 301/435–5515; *anos@mail.nih.gov.*

The current technology describes the universal inactivation of viruses, parasites, and tumor cells by hydrophobic, photoactivatable compounds. These non-toxic compounds, such as 1,5-iodoanpthylazide (INA), will selectively accumulate in the innermost regions of biological membrane bilayers, where the compounds will bind to proteins and lipids upon irradiation with light, thus inactivating deeply embedded proteins while maintaining integrity and activity of the proteins on the surface. This inactivation preserves the structural and conformational integrity and therefore immunogenicity of the agent in question, which overcomes a potential problem associated with some other vaccines such as those containing killed pathogens. As representative examples, the patent application describes experimental results obtained using HIV, SIV, and Ebola viruses. The inactivation approach presented in this technology provides for a safe, non-infectious composition for vaccination against the corresponding agent, whereas some vaccines, such as those involving live-attenuated microbial agents, still have a risk of infectivity associated with them.

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

**High Expression Level Vectors Combining of mRNA Transport Elements for Use in Mammalian Cells**

Barbara K. Felber *et al.* (NCI).
U.S. Provisional Application No. 60/471,988 filed 19 May 2003 (HHS Reference No. E–223–2003/0-US–01); U.S. Provisional Application No. 60/472,223, filed 20 May 2003 (HHS Reference No. E–258–2003/0–US–01); PCT Application No. PCT/US04/15776 filed 19 May 2004, which published as WO2004/113547 on 29 Dec 2004 (HHS Reference No. E–223–2003/1–PCT–01).
Licensing Contact: Susan Ano; 301/435–5515; *anos@mail.nih.gov.*

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 leads 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–034–1991/0, /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 medical 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.

| Vector | Description | Reference No. |
|---|---|---|
| Wild-type HIV–1 RT | full length, wild type | E–034–1991/0 |
| L100I | NNRTI resistant | E–034–1991/1 |
| K103N | NNRTI resistant | E–034–1991/1 |
| V106A | NNRTI resistant | E–034–1991/1 |
| V108I | | |
| E138K | NNRTI resistant | E–034–1991/1 |
| Y181I | | |
| Y181C | NNRTI resistant | E–034–1991/1 |
| Y188L | | |
| Y188H | NNRTI resistant | E–034–1991/1 |
| G190A | | |
| G190S | | |
| P236L | NNRTI resistant | E–034–1991/1 |
| RTs that carry some combinations of NNRTI mutations, *e.g.*, K103N+Y181I, are also available. | | |
| K65R | NRTI resistant | E–034–1991/2 |
| T69G | | |
| L74V | NRTI resistant | E–034–1991/1 |
| M184I | Lamivudine (3TC) resistant | |
| M184V | Lamivudine (3TC) resistant | E–034–1991/1 |
| AZT–R (5 mutations) | AZT resistant | E–034–1991/1 |
| Δ67 complex | Multi-NRTI resistant | E–034–1991/4 |
| Q151M | Multi-NRTI resistant | E–034–1991/4 |
| Q151M Complex | Multi-NRTI resistant | E–034–1991/4 |
| SSGR/T215Y | Multi-NRTI resistant | E–034–1991/4 |
| SSSR/T215Y | Multi-NRTI resistant | E–034–1991/4 |

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]

**BILLING CODE 4140–01–P**

## 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.

*Agenda:* The agenda will include Opening Remarks, Administrative Matters, Director's Report, NCMHD, IC Health Disparities Research Report, NCMHD Program Highlights, and other business of the Council.

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

*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, *brooksd@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.

Dated: August 25, 2005.

**Anthony M. Coelho, Jr.,**

*Acting Director, Office of Federal Advisory Committee Policy.*

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

**BILLING CODE 4140–01–M**

## 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.,