the information will have practical utility; (2) the accuracy of FDA s estimate of the burden of the proposed collection of information, including the validity of the methodology and assumptions used; (3) ways to enhance the quality, utility, and clarity of the information to be collected; and (4) ways to minimize the burden of the collection of information on respondents, including through the use of automated collection techniques, when appropriate, and other forms of information technology.

**Veterinary Feed Directive (OMB Control Number 0910–0363)—Extension**

With the passage of Animal Drug Availability Act (ADAA), the Congress enacted legislation establishing a new class of restricted feed use drugs, VFD drugs, which may be distributed without involving State pharmacy laws. Although controls on the distribution and use of VFD drugs are similar to those for prescription drugs regulated under section 503(f) of the Federal Food, Drug, and Cosmetic Act (21 U.S.C. 353(f)), the implementing VFD regulation (21 CFR 558.6), is tailored to the unique circumstances relating to the distribution of medicated feeds. The content of the VFD is spelled out in the regulation. All distributors of medicated feed containing VFD drugs must notify FDA of their intent to distribute, and records must be maintained of the distribution of all medicated feed containing VFD drugs. The VFD regulation ensures the protection of public health while enabling animal producers to obtain and use needed drugs as efficiently and cost-effectively as possible.

FDA estimates the burden for this collection of information as follows:

TABLE 1.—ESTIMATED ANNUAL REPORTING BURDEN[1]

| 21 CFR Section | No. of Respondents | Annual Frequency per Response | Total Annual Responses | Hours per Response | Total Hours |
|---|---|---|---|---|---|
| 558.6(a)(3) through (a)(5) | 15,000 | 25 | 375,000 | 0.25 | 93,750 |
| 558.6(d)(1)(i) through (d)(1)(iii) | 500 | 1 | 500 | 0.25 | 125 |
| 558.6(d)(1)(iv) | 20 | 1 | 20 | 0.25 | 5 |
| 558.6(d)(2) | 1,000 | 5 | 5,000 | 0.25 | 1,250 |
| 514.1(b)(9) | 1 | 1 | 1 | 3.00 | 3 |
| Total Hours | 16,521 | | | | 95,133 |

[1] There are no capital costs or operating and maintenance costs associated with this collection of information.

TABLE 2.—ESTIMATED ANNUAL RECORDKEEPING BURDEN[1]

| 21 CFR Section | No. of Recordkeepers | Annual Frequency per Recordkeeper | Total Annual Records | Hours per Record | Total Hours |
|---|---|---|---|---|---|
| 558.6(c)(1) through (c)(4) | 112,500 | 10 | 1,125,000 | .0167 | 18,788 |
| 558.6(e)(1) through (e)(4) | 5,000 | 75 | 375,000 | .0167 | 6,263 |
| Total Hours | | | | | 25,051 |

[1] There are no capital costs or operating and maintenance costs associated with this collection of information.

The estimate of the times required for record preparation and maintenance is based on agency communication with industry and agency records and experience.

Dated: June 6, 2005.

**Jeffrey Shuren,**

*Assistant Commissioner for Policy.*

[FR Doc. 05–11581 Filed 6–9–05; 8:45 am]

**BILLING CODE 4160–01–S**

## 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.

Identification of H2–Db and HLA–A2 Specific CD8 Epitopes From Human KDR/VEGFR–2 That Inhibit Angiogenesis by Vaccination

Drs. Samir Khleif and Yujun Dong (NCI).

U.S. Provisional Application No. 60/671,867 filed 15 Apr 2005 (DHHS Reference No. E–158–2005/0–US–01).

*Licensing Contact:* John Stansberry; (301) 435–5236; *stansbej@mail.nih.gov.*

Vascular Endothelial Growth Factor Receptor 2 (VEGFR–2/KDR) is a promising target for cancer therapy due to its critical role in tumor associated angiogenesis and vascularization. This invention describes the amino acid sequences of seven short peptides based upon epitopes of human Vascular Endothelial Growth Factor Receptor-2

(VEGFR–2) that bind human Histocompatibility Leukocyte Antigen A2 (HLA-A2). These peptides can potentially induce Cytotoxic T Lymphocyte (CTL)-mediated lysis of tumor vascularization and inhibit tumor growth. The inventors have demonstrated the principles described in this invention in vivo in mice for VEGFR–2, using murine H2-Db specific peptides instead of HLA-A2. This invention has the potential to inhibit angiogenesis and may be applicable to tumor and autoimmune disease therapy.

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

**Novel Anti-CD30 Antibodies and Recombinant Immunotoxins Containing Disulfide-Stabilized Fv Fragments**

Ira H. Pastan *et al.* (NCI).
U.S. Provisional Application No. 60/387,293 filed 07 Jun 2002 (DHHS Reference No. E–135–2002/0–US–01);
PCT Application No. PCT/US03/18373 filed 07 Jun 2003, which published as WO 03/104432 on 18 Dec 2003 (DHHS Reference No. E–135–2002/1–PCT–01);
U.S. Patent Application filed 03 Dec 2004 (DHHS Reference No. E–135–2002/1–US–02).
*Licensing Contact:* Jesse S. Kindra; (301) 435–5559; *kindraj@mail.nih.gov.*

The present invention discloses the creation of new anti-CD30 stalk antibodies and anti-CD30 dsFv-immunotoxins, which have shown good cytotoxic activity.

CD30 is a member of the tumor necrosis factor receptor super family. It is an excellent target due to its high expression in malignant Reed Sternberg cells of Hodgkin's Lymphoma (HL) and in anaplastic large cell lymphomas (ALCL), and due to its expression in only a small subset of normal lymphocytes. Previous attempts to target CD30 include the scFv immunotoxin Ki-4 that has shown specific binding to CD30-positive lymphoma cell lines and killed target cells.

As claimed in this patent application, some of the antibodies do not bind or bind very weakly CD30 released from cells, although they do bind strongly to cell associated CD30. This enhancement further increases the ability of immunotoxins and other immunoconjugates to target and treat lymphomas expressing CD30.

The immunotoxins of the present invention are more stable and have higher affinity for CD30 then their predecessors. Research thus far has shown that the dsFv-immunotoxins are able to kill a variety of CD30-positive lymphoma cell lines in vitro as well as CD30-transfected A431 cells via specific binding to CD30.

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

**Compositions and Methods for Inhibiting Vascular Channels and Methods of Inhibiting Proliferation**

Myung Hee Park, Paul M.J. Clement, Hartmut M. Hanauske-Abel, Edith C. Wolff, Hynda K. Kleinman, Bernadette M. Cracchiolo (NIDCR).
U.S. Provisional Application No. 60/314,561 filed 23 Aug 2001 (DHHS Reference No. E–320–2001/0–US–01);
PCT Application No. PCT/US02/26909 filed 23 Aug 2002, which published as WO 03/018014A2 on 06 Mar 2003 (DHHS Reference No. E–320–2001/0–PCT–02);
U.S. Patent Application No. 10/486,671 filed 11 May 2004 (DHHS Reference No. E–320–2001/0–US–03).
*Licensing Contact:* John Stansberry; (301) 435–5236; *stansbej@mail.nih.gov.*

Angiogenesis, the recruitment of new blood vessels, is recognized as an important factor in tumor proliferation in many types of cancer. It is generally accepted that therapeutic approaches that inhibit angiogenesis effectively limit, or even prevent, the formation of solid tumors. It has also been shown that anti-angiogenic therapeutics allow conventional radiation therapy and chemotherapy to be more effective.

This invention pertains to certain compounds that inhibit angiogenesis in a previously unrecognized way. These compounds also inhibit the proliferation of cells within intraepithelial neoplasias (clusters of abnormally proliferating epithelial cells that are the origin of cancers). The subject compounds specifically block the formation of the amino acids hypusine and hydroxyproline. The former is the critical residue of eukaryotic translation initiation factor 5A (eIF5A), which is important in cell cycle progression, and hydroxyproline constitutes the critical residue of the collagens. The targeted enzymes are deoxyhypusine hydroxylase and prolyl 4-hydroxylase, respectively.

This invention provides evidence for an important role of eIF–5A in angiogenesis, and discloses a family of compounds with useful clinical properties. Specifically, these compounds include the core structures and potential derivatives of ciclopirox olamine, deferiprone, deferoxamine, and 2,2'-dipyridyl.

Ciclopirox olamine has potential for treatment of oral-pharyngeal cancer, and chemoprevention and treatment of cervical and vulvar cancer. Notably, this drug is FDA-approved in the USA as a topical medication against fungal infections while, in Europe, it is also approved for the treatment of yeast infections of the genital tract. The compound has a known clinical profile and lacks teratogenicity, potentially expediting clinical trials for new cancer treatment indications.

Dated: June 3, 2005.

**Steven M. Ferguson,**

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

[FR Doc. 05–11575 Filed 6–9–05; 8:45 am]

**BILLING CODE 4140–01–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

### Government-Owned Inventions; Availability for Licensing: 3-Hydroxy-3-Methylglutaryl Coenzyme A Reductase Inhibitors as a Modality in Cancer Therapy

**AGENCY:** National Institutes of Health, Public Health Service, DHHS.

**ACTION:** Notice.

**SUMMARY:** The invention described below is owned by an agency of the U.S. Government and is 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 may be obtained by contacting George G. Pipia, Ph.D., at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: (301) 435–5560; fax: (301) 402–0220; e-mail: *PipiaG@mail.nih.gov*.

**Use of Inhibitors of 3-Hydroxy-3-Methylglutaryl Coenzyme A Reductase as a Modality in Cancer Therapy**

Charles Myers, Jane Trepel, Won Ki Kang, Luke Whitsell, Leonard Neckers (NCI). U.S. Patent No. 6,040,334 issued 21 Mar 2000 (DHHS Reference No. E–146–1992/0–US–23).