(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).

Licensing Contact: George Pipia; 301/435–5560; *pipiag@mail.nih.gov*.

The invention provides a method for treating mammalian adenocarcinomas and sarcomas comprising administration of an effective amount of an inhibitor of HMG Co-A or homologues of the inhibitor. Adenocarcinoma is known to afflict the prostate, stomach, lung, breast and colon, as well as other sites. Examples of compounds useful in the present invention are lovastatin and simvastatin as well as their homologues. Also included are compounds classified as HMG Co-A inhibitors, as well as their homologues or analogues. Generally, these HMG Co-A inhibitors are known to lower serum cholesterol in humans. However, the present invention is not so limited. That is, an inhibitor of HMG Co-A or one of its homologues may work in the method of the present invention without necessarily lowering serum cholesterol. The invention focuses not on the compound's ability to lower cholesterol, but rather on the compound's ability to treat selected cancers, such as adenocarcinomas of the prostate, stomach, lung, breast and colon and certain sarcomas such as Ewing's sarcoma.

Also provided by the invention is a method of reducing prostate specific antigen (PSA) levels in a patient having prostatic adenocarcinoma comprising administration of an effective amount of a compound which is an inhibitor of HMG Co-A or a homologue of such inhibitor. The invention also includes a method of reducing PSA in conjunction with another treatment modality.

The claims encompassing this technology are directed to the methods of treating certain types of cancer with inhibitors of HMG Co-A reductase, and specifically with lovastatin and simvastatin (see the U.S. issued patent 6,040,334: *http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=/netahtml/srchnum.htm&r=1&f=G&l=50&s1=6,040,334.WKU.&OS=PN/6,040,334&RS=PN/6,040,334).*

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

Dated: June 3, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–11576 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

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.

Proteomic Profiles Associated With Aging

Dr. Shari M. Ling (NIA).
DHHS Reference No. E–354–2004/1—Research Tool.
Licensing Contact: Marlene Shinn-Astor; 301/435–4426; *shinnm@mail.nih.gov*.

This invention relates to proteomic profiles associated with normal aging. Biological markers (Biomarkers) that characterize the state of ''normal aging'' could provide a useful comparison for biomarkers of age-associated diseases (cardiovascular, cancer, arthritis). The profiles could then be used to develop markers linked with other diseases.

The proteins identified could either be included in elisa or multiplex assays, or incorporated into a protein-based chip. These products would be of utility to characterize research subjects for clinical trials. Specific proteins or groups of proteins could be used as potential therapeutic targets to prevent or attenuate disease development or help to improve the normal aging process.

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

AlphaB-Crystallin/HSPBE Gene Knockout Mouse

Dr. Eric F. Wawrousek, et al. (NEI).
DHHS Reference No. E–135–2001/0—Research Tool.
Licensing Contact: Marlene Shinn-Astor; (301) 435–4426; *shinnm@mail.nih.gov*.

The alpha crystallins and other members of the small heat shock family of proteins, have been shown to be very important proteins for preventing the irreversible destruction of other proteins. AlphaA is mostly restricted to the ocular lens, while alphaB is present in almost all cells of the body with the highest levels in ocular lens, heart, and skeletal muscle. The NIH has created lines of mice, which lack the alphaB-crystallin gene (and unintentionally, its neighboring gene HSPB2). These mouse lines could be used to study functions of these proteins in the eye, skeletal muscle, heart, and any other tissue or organ.

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

Three Myelin Basic Protein-Specific T Cell Clones, TL2A6, TL5F6, and TL5G7 That Are Restricted by Multiple Sclerosis-Associated HLA–DR Molecules and Recognize the Immunodominant Myelin Basic Protein (MBP) Peptide MBP (83–99)

Dr. Roland Martin, *et al.* (NINDS).
DHHS Reference No. E–277–1999/0—Research Tool.
Licensing Contact: Marlene Shinn-Astor; (301) 435–4426; *shinnm@mail.nih.gov.*

Autoreactive T cell clones such as TL3A6 and TL5F6 that recognize an autoantigen, which is potentially relevant for an autoimmune disease, for example, multiple sclerosis (MS), offer the potential to examine the disease pathogenesis and develop new treatments. Such treatments aim at disrupting or interfering with the specific interaction between autoreactive T cells, antigen presenting cells and antigenic peptide. Current treatments have immunomodulatory effects and side effects. These T cell lines will be useful for developing novel treatment approaches for multiple sclerosis. The T cell lines can be used to test treatments that block or interfere with surface receptors of these cells.

Mouse Model for Myasthenia Gravis

Dr. Michael J. Lenardo et al. (NIAID).
DHHS Reference No. E–188–1999/0—Research Tool.