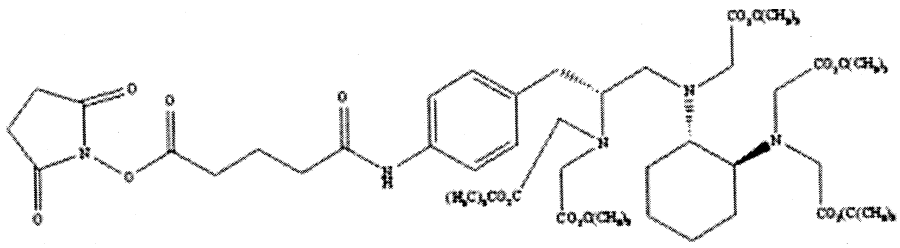
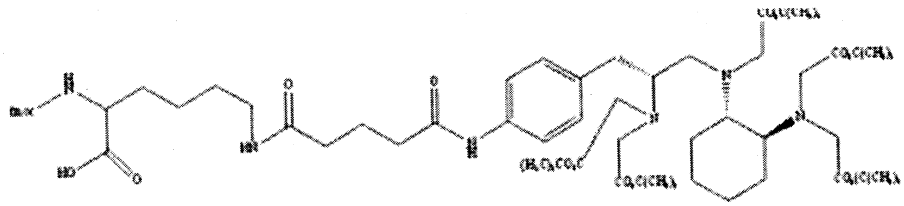


I



II

Inventors: Martin Wade Brechbiel and Thomas Clifford (NCI).

Publications:

1. T Clifford et al. Validation of a novel CHX-A'' derivative suitable for peptide conjugation: small animal PET/CT imaging using yttrium–86–CHX–A''–octreotide. J Med Chem. 2006 Jul 13;49(14):4297–4304.

2. HS Chong et al. Synthesis and evaluation of novel macrocyclic and acyclic ligands as contrast enhancement agents for magnetic resonance imaging. J Med Chem. 2006 Mar 23;49(6):2055–2062.

Licensing Status: Available for exclusive or non-exclusive licensing or collaborative research opportunity.

Patent Status: U.S. Provisional Application No. 60/603,781 filed 23 Aug 2004 (HHS Reference No. E–317–2004/1–US–01); International Patent Application PCT/US2005/028125 filed 09 Aug 2005 (HHS Reference No. E–317–2004/1–PCT–02).

Licensing Contact: Michael A. Shmilovich, Esq.; 301/435–5019; *shmilovm@mail.nih.gov.*

Dated: January 30, 2007.

Steven M. Ferguson,

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

[FR Doc. 07–526 Filed 2–6–07; 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, HHS.

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.

Extended Transgene Expression for a Non-Integrating Adenoviral Vector Containing Retroviral Elements

Description of Technology: Anthrax lethal toxin (LeTx) consists of two components: The protective antigen (PrAg) and the lethal factor (LF). PrAg binds to the cell surface where it is activated by furin protease, followed by the formation of a PrAg heptamer. LF is then translocated into the cytosol of a cell via this heptamer, where it acts as a metalloprotease on all but one mitogen-activated protein kinase kinase (MAPKK). Approximately 70% of human melanomas contain a mutation (B–RAF V600E) that constitutively activates a MAPKK pathway, and LeTx has been shown to have significant toxicity towards cells which have this mutation. This suggested a potential use for LeTx in cancer therapy. Unfortunately, native LeTx is toxic to normal cells, detracting from its *in vivo* applicability.

PrAg has been engineered to be activated by a matrix metalloprotease (MMP), instead of by furin protease. Because MMPs are highly expressed in tumor cells, this modification increases selectivity towards cancer cells. Surprisingly, mouse data shows that the modified LeTx (denoted PrAg-L1/LF) is less cytotoxic to ''normal'' cells *in vivo*, when compared to wild-type LeTx. Significantly, PrAg-L1/LF maintained its high toxicity toward human tumors in mouse xenograft models of human tumors, including melanomas. However, this toxicity applied not only to tumors having mutations that constitutively activate MAPKKs, but also to other tumor types such as lung and colon carcinomas. The absence of toxicity to ''normal'' cells coupled to its effectiveness on a wide range of cancer

cell types suggests that PrAg-L1/LF may represent a novel cancer therapeutic.

Applications: PrAg-L1/LF has applications as a human cancer therapeutic; Applicability extends beyond melanomas, including lung and colon carcinomas.

Market: The worldwide market for melanoma therapeutics is approximately $437M, and is predicted to reach $680M by the year 2009. Approximately 2.4 million people are afflicted with melanoma, with around 150,000 new cases each year. Demonstration of effectiveness *in vivo* for lung and colon carcinomas will increase the market for this technology.

Development Status: The technology is at the preclinical stage.

Inventors: Stephen H. Leppla (NIAID), Shi-hui Liu (NIAID), Thomas H. Bugge (NIDCR), John R. Basile (NIDCR), Brooke Currie (NIDCR).

Related Publications:

1. S Liu *et al.* Intermolecular complementation achieves high-specificity tumor targeting by anthrax toxin. Nat Biotechnol. 2005 Jun;23(6):725–730.

2. RJ Abi-Habib *et al.* A urokinase-activated recombinant anthrax toxin is selectively cytotoxic to many human tumor cell types. Mol Cancer Ther. 2006 Oct;5(10):2556–2562.

Patent Status: U.S. Provisional Application No. 60/870,050 filed 14 Dec 2006 (HHS Reference E–070–2007/0–US–01).

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: David A. Lambertson, Ph.D.; 301/435–4632; *lambertsond@od.nih.gov.*

Collaborative Research Opportunity: The NIAID Laboratory of Bacterial Diseases is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize PrAg-L1/LF as a novel cancer therapeutic. Please contact Stephen H. Leppla, Ph.D. at 301/594–2865 and/or *sleppla@niaid.nih.gov* for more information.

A Novel Combination of CXCR–4 Antagonist T22 With Conventional Immunotherapy Improves Treatment Efficacy in Established Tumors

Description of Technology: Immunotherapy for cancer rarely results in complete responses, possibly due to chemokine receptor mediated activation of prosurvival pathways in cancer cells. CXCR4, is one such receptor that is expressed in a variety of cancers, including melanoma. Inhibiting these chemokine receptors may circumvent the ability of cancer to protect themselves from immunological attack.

This invention provides a method of treating cancers that expresses the chemokine receptor CXCR4 by a novel combination therapeutic approach. More specifically, the invention claims methods and compositions for the improved treatment of metastatic tumors by using a CXCR4 antagonist in conjunction with conventional monoclonal antibody based immunotherapy (*e.g.*, anti-CTLA4 mAb) or immunostimulatory chemotherapeutics (*e.g.*, cyclophosphamide). The invention clearly demonstrates that treatment of *in vivo* experimental lung cancer models with T22, a CXCR4 antagonist, followed by anti-Cytotoxic lymphocyte antigen (CTLA)-4 monoclonal antibody (or cyclophosphamide) treatment synergistically reduced the total tumor burden compared with the reduction of tumor burden when either agent is used alone. T22 treatment alone is not cytotoxic and has no demonstrated ability to increase non-specific host autoimmunity when used in combination with anti-CTLA4 mAb or cyclophosphamide. This invention has significant potential as a new, effective combination immunotherapy.

Applications and Modality: (1) A new method of combination therapy for cancer based on immunotherapeutics, including adoptive transfer of anti-tumor lymphocytes and treatment with immunostimulatory agents (monoclonal antibodies or chemotherapy); (2) A new therapeutic method for the treatment of CXCR4 chemokine receptor expressing cancers; (3) A new therapeutic method exploiting the role of chemokine receptor CXCR4 that potentially renders immunotherapy more effective without further increasing risks of patient autoimmunity.

Market: Chemokine receptor CXCR4 has a proven role in cancer metastasis in several cancers. The anti-cancer market is projected to reach sales of $60 billion by 2010.

Development Status: The technology is currently in the pre-clinical stage of development. Animal data is available.

Inventor: Sam T. Hwang (NCI).

Publications:

1. CH Lee *et al.* Sensitization of B16 tumor cells with a CXCR4 antagonist increases the efficacy of immunotherapy for established lung metastases. Mol Cancer Ther. 2006 Oct;5(10):2592–2599.

2. T Kakinuma and ST Hwang. Chemokines, chemokine receptors, and cancer metastasis. J Leukoc Biol. 2006 Apr;79(4): 639–651.

3. T Murakami *et al.* Expression of CXC chemokine receptor-4 enhances the pulmonary metastatic potential of murine B16 melanoma cells. Cancer Res. 2002 Dec 15;62(24):7328–7334.

Patent Status: U.S. Provisional Application No. 60/840,216 filed 25 Aug 2006, entitled ''Combination Therapy for the Treatment of Cancer'' (HHS Reference No. E–267–2006/0–US–01).

Licensing Status: Available for exclusive and non-exclusive licensing.

Licensing Contact: John Stansberry, Ph.D.; 301/435–5236; *stansbej@mail.nih.gov.*

Lentivirus Based Vector System for Gene Therapy Delivery

Description of Technology: Gene therapy is a technique based on the idea that a genetic disorder can be treated by replacing a dysfunctional gene with a functional copy of that gene. Currently, retroviral vectors and adenoviral vectors are most frequently used for gene therapy clinical trials. Retroviral vectors provide long term gene expression and are capable of transferring genes into non-dividing cells, unlike their adenoviral counterparts. However, retroviral vectors often suffer from weak viral titers and inefficient encapsidation of the therapeutic gene, detracting from their therapeutic value. Thus, there is a need in the art for improved retroviral gene therapy vectors.

This technology family is directed to a retroviral vector system comprising a packaging vector and a transfer vector, and a method of using the vectors for gene therapy. The packaging vector is the result of an HIV-2 lentiviral vector containing mutations in sequences surrounding a splice donor site within the packaging signal. The transfer vector comprises mutations that render a splice donor site non-functional. These mutations increase the viral titer and expression/encapsidation of the transgene, but without a corresponding increase in the packaging of viral RNA. Thus, these vectors may address some of the pressing concerns with current gene therapy vectors systems.

Applications: Improved lentivirus based vector system with practical application in gene therapy/gene transfer; Two vector system minimizes possibility of HIV infection; Packaging vector is a result of HIV-2 Lentivirus vector; Improved packaging and expression ability addresses current low viral titer problem.

Market: The only gene therapy product currently in the market was approved in China in 2004; The R&D market of gene therapy is projected to grow to several billion dollars in the next 5 years.

Development Status: The technology is currently in the pre-clinical stage of development.

Inventors: Suresh K. Arya (NCI).

Publication: SK Arya *et al.* Human immunodeficiency virus type 2 lentivirus vectors for gene transfer: expression and potential for helper virus-free packaging. Hum Gene Ther. 1998 Jun 10;9(9):1371–1380.

Patent Status: U.S. Patent No. 6,790,657 issued 14 Sep 2004, entitled ''Lentivirus Vector System'' (HHS Reference No. E–231–1998/0-US–03); U.S. Patent Application No. 10/731,988 filed 09 Dec 2003, now allowed, entitled ''Lentivirus Vector System'' (HHS Reference No. E–231–1998/0-US–04).

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: David Lambertson, Ph.D.; 301/435–4632; *lambertsond@od.nih.gov*.

Methods and Compositions of Chemokine-Tumor Antigen Fusion Proteins as Cancer Vaccines

Description of Technology: Tumor cells are known to express tumor specific antigens on the cell surface. These antigens are believed to be poorly immunogenic, largely because they represent gene products of oncogenes or other cellular genes which are normally present in the host. As a result, poor immunogenicity of relevant cancer antigens has proven to be a major obstacle to successful immunotherapy with tumor vaccines. Thus, there is a need for a more potent vaccine to elicit an immune response effective in the treatment or prevention of cancer.

The current invention embodies a fusion protein comprising of a chemokine and tumor antigen. The inventors reported in several peer-reviewed manuscripts that these fusion proteins represent potential vaccines for use against cancer. More specifically, the inventors have developed a vaccine construct that expresses fusion protein comprising human monocyte chemotactic protein-3 fused with tumor antigens, such as lymphoma-derived Id or breast cancer Muc-1. Administration of the fusion protein, or a nucleic acid encoding the fusion protein, elicits a specific immune response directed against the tumor antigen or protein, thereby inhibiting the growth of cells expressing this antigen or protein.

Applications and Modality: Potential immunotherapy for cancer.

Market: 600,000 deaths from cancer related diseases estimated in 2006.

Development Status: This technology is currently in the pre-clinical stage of development.

Inventors: Larry Kwak (NCI) and Arya Biragyn (NIA).

Patent Status: U.S. Patent No. 6,562,347 issued 13 May 2003 (HHS Reference No. E–107–1998–0-US–03).

Licensing Status: Available for exclusive and non-exclusive licensing.

Licensing Contact: Jennifer Wong; 301/435–4633; *wongje@mail.nih.gov.*

Dated: January 31, 2007.

Steven M. Ferguson,

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

[FR Doc. E7–1931 Filed 2–6–07; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; Notice of Meeting

Pursuant to section 10(a) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the National Cancer Institute Board of Scientific Advisors.

The meeting will be open to the public, 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.

Name of Committee: National Cancer Institute Board of Scientific Advisors.

Date: March 5–6, 2007.

Time: March 5, 2007, 8 a.m. to 6 p.m.

Agenda: Director's Report: Ongoing and New Business; Reports of Program Review Group(s); and Budget Presentation; Reports of Special Initiatives; RFA and RFP Concept Reviews; and Scientific Presentations.

Place: National Institutes of Health, Building 31, 31 Center Drive, Conference Room 10, Bethesda, MD 20892.

Time: March 6, 2007, 8 a.m. to 1 p.m.

Agenda: Reports of Special Initiatives; RFA and RFP Concept Reviews; and Scientific Presentations.

Place: National Institutes of Health, Building 31, 31 Center Drive, Conference Room 10, Bethesda, MD 20892.

Contact Person: Paulette S. Gray, PhD, Executive Secretary, Director, Division of Extramural Activities, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, 8th Floor, Rm. 8001, Bethesda, MD 20892, 301–496–5147, *grayp@mail.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.

In the interest of security, NIH as instituted stringent procedures for entrance onto the NIH campus. All visitor vehicles, including taxicabs, hotel, and airport shuttles will be inspected before being allowed on campus. Visitors will be asked to show one form of identification (for example, a government-issued photo ID, driver's license, or passport) and to state the purpose of their visit.

Information is also available on the Institute's/Center's home page: *deainfo.nci.nih.gov/advisory/bsa.htm,* where an agenda and any additional information for the meeting will be posted when available.

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399, Cancer Control, National Institutes of Health, HHS)

Dated: January 31, 2007.

Anna Snouffer,

Acting Director, Office of Federal Advisory Committee Policy.

[FR Doc. 07–519 Filed 2–6–07; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Heart, Lung, and Blood Institute; Notice of Closed Meetings

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 meetings.

The meetings 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 Heart, Lung, and Blood Institute Special Emphasis Panel, Research Project in Cardiothoracic Surgery.

Date: March 7–8, 2007.

Time: 8 a.m. to 5 p.m.

Agenda: To review and evaluate grant applications.

Place: Holiday Inn Georgetown, 2101 Wisconsin Avenue, NW., Washington, DC 20007.

Contact Person: Shelly S. Sehnert, PhD., Scientific Review Administrator, Review