Market

Cancer Imaging

Development Status

• Early-stage technology with preclinical mouse models as of 18 July 2006

Inventors

- Hisataka Kobayashi (NCI)
- Peter Choyke (NCI)
- Urano Yasuteru (University of Tokyo)

Patent Status

• U.S. Provisional Patent Application filed June 30, 2006 (serial number not assigned); closely related to HHS Ref. No. E–335–2005; U.S. Provisional Patent Application No. 60/751,429 filed December 16, 2005.

Availability

• Available for exclusive, nonexclusive licensing or collaborative opportunity.

Licensing Contact

Chekesha S. Clingman, PhD., Technology Licensing Specialist, Office of Technology Transfer, The National Institutes of Health, 6011 Executive Blvd., Suite 325, Rockville, MD 20852, phone: (301) 435–5018, fax: (301) 402– 0220, clingmac@mail.nih.gov.

Collaborative Research Opportunity

The NCI Molecular Imaging Program is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize target specific activatable optical probes. Please contact Hisataka Kobayashi or Peter Choyke at 301–451–4220 *pchoyke@nih.gov* for more information.

Dated: July 31, 2006.

Steven M. Ferguson,

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

[FR Doc. 05–6881 Filed 8–11–06; 8:45 am]

BILLING CODE 4140-01-M

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 incontext patterns

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.

Amyloid Beta Is a Ligand for FPR Class Receptors

Description of Technology: Alzheimer's disease is the most important dementing illness in the United States because of its high prevalence. Five to ten percent of the United States population 65 years and older are afflicted with the disease. In 1990 there were approximately 4 million individuals with Alzheimer's, and this number is expected to reach 14 million by the year 2050. It is the fourth leading cause of death for adults. resulting in more than 100,000 deaths annually. Amyloid beta has been identified as playing an important role in the neurodegeneration of Alzheimer's disease. However, the mechanism by which this occurred was unknown, but has been postulated to be either direct or indirect through an induction of inflammatory responses.

The NIH announces the identification of the 7-transmembrane, G-proteincoupled receptor, FPRL-1, in the cellular uptake and fibrillar aggregation of amyloid $\beta\beta(A\beta\beta)$ peptides. The $A\beta\beta$ peptides use the FPRL-1 receptor to attract and activate human monocytes and mouse microglial cells (publications referenced below), and have been identified as a principal component of the amyloid plaques associated with Alzheimer's disease. In addition, the known anti-inflammatory drug, Colchicine, has been shown to inhibit the FPRL1 activation by amyloid ββ** and the internalization of FPRL1/ amyloid beta complexes.

Inventors: Ji Ming Wang et al. (NCI). *Publications:*

1. Y Le, W Gong, L Tiffany, A Tumanov, S Nedospasov, W Shen, NM Dunlop, J-L Gao, PM Murphy, JJ Oppenheim, and JM Wang, "Amyloid (beta)42 activates a G-protein-coupled chemoattractant receptor, FPR-like-1," J. Neuroscience 2001 Jan 15; 21(2):RC123.

2. HL Tiffany, MC Lavigne, YH Cui, JM Wang, TL Leto, JL Gao, and PM Murphy, "Amyloid-beta induces chemotaxis and oxidant stress by acting at formylpeptide receptor 2, a G proteincoupled receptor expressed in phagocytes and brain," J Biol Chem. 2001 Jun 29;276(26):23645–52.

3. YH Cui, Y Le, W Gong, P Proost, J Van Damme, WJ Murphy, and JM Wang, "Bacterial lipopolysaccharide selectively up-regulates the function of the chemotactic peptide receptor formyl peptide receptor 2 in murine microglial cells," J Immunol. 2002 Jan 1;168(1):434–42.

4. H Yazawa., Z-X Yu, K Takeda, Y Le, W Gong, VJ Ferrans, JJ Oppenheim, CC Li, and JM Wang, "Beta amyloid peptide (Ab42) is internalized via the Gprotein coupled receptor FPRL1 and forms fibrillar aggregates in macrophages," FASEB J. 2001 Nov; 15(13):2454–2642.

5. P Iribarren, K Chen, J Hu, G Gong, EH Cho, S Lockett, B Uranchimeg, and JM Wang, "CpG-containing oligodeoxynucleotide promotes microglial the up-take of amyloid beta 1-42 by up-regulating the expression of the G-protein coupled receptor mFPR2,".FASEB J. 2005 Dec;19(14):2032–4.

6. K Chen, P Iribarren, J Hu, J Chen, G Gong, EH Cho, S Lockett, NM Dunlop, and JM Wang, "Activation of Toll-like receptor 2 on microglia promotes cell uptake of Alzheimer disease-associated amyloid beta peptide," J Biol Chem. 2006 Feb 10;281(6):3651–9.

Patent Status: U.S. Patent Application No. 10/831,524 filed 23 Apr 2004 (HHS Reference No. E–336–01/0–US–02), claiming priority to 26 Oct 2001.

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

Licensing Contact: 301/496–7057; nihott@mail.nih.gov

Collaborative Research Opportunity: The National Cancer Institute, Laboratory of Molecular Immunoregulation, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialized siRNA delivery development. Please contact Diana Bialozor at 301/846–5465 or *bialozod@mail.nih.gov* for more information. Dated: August 1, 2006. **Steven M. Ferguson**, Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health. [FR Doc. E6–13191 Filed 8–11–06; 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.

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Adoptive T-Cell Transfer After Lymphodepletion Promotes Tumor Regression

Description of Technology: Available for licensing is a method of adoptive cell transfer (ACT) immunotherapy. Since its first description, ACT is now being developed for the supportive treatment of a variety of infectious diseases and cancer.

Current ACT methods to treat cancer are based on the ex vivo selection of lymphocytes with high avidity for recognition of tumor antigens, and their activation and numerical expansion before re-infusion to the autologous tumor-bearing host. The current invention improves ACT by including a pre-treatment regimen to ensure permissive conditions in the host for in vivo proliferation of the transferred cells. Specifically, the immune system is suppressed by pre-treatment with lymphodepleting chemotherapy. Two separate clinical trials have demonstrated that using this approach, ACT can induce lasting tumor shrinkage.

Lymphodepleting chemotherapy followed by ACT resulted in tumor shrinkage of at least 50 percent in 6 out of 13 treated patients suffering from refractory melanoma. Several patients remained cancer free for more than a year after treatment. The usefulness of combined ACT and lymphodepleting therapy for cancer treatment was confirmed when this study was extended to include 35 melanoma patients. Eighteen of the 35 patients (51%) responded to the treatment, including 3 patients who experienced ongoing complete disappearance of cancer and 15 patients had tumor shrinkage of at least 50 percent with a mean duration of almost a year after treatment. In a recent clinical trial that is not yet published, using a modified protocol to treat 23 patients, a similar response rate (56%) was seen.

This approach to ACT offers a potentially significant improvement in the treatment of many types of cancer. In addition, this method might be applicable in treating other diseases such as AIDS, immunodeficiency, or other autoimmunity for which immune effector cells can impact the clinical outcome.

Inventors: Mark E. Dudley, Steven A. Rosenberg, John R. Wunderlich (NCI) Publications:

1 . Dudley ME, et al. "Adoptive cell transfer therapy following nonmyeloablative but lymphodepleting chemotherapy for the treatment of patients with refractory metastatic melanoma." J Clin Oncol. 2005 Apr 1;23(10):2346–2357.

2 . Dudley ME, et al. "Cancer regression and autoimmunity in patients after clonal repopulation with antitumor lymphocytes." Science. 2002 Oct 25;298(5594):850–854.

Patent Status: U.S. Provisional Application No. 60/408,681 filed 06 Sep 2002 (HHS Reference No. E–275–2002/ 0–US–01) PCT Application No. PCT/ US03/27873 filed 05 Sep 2003, which published as WO 2004/021995 on 18 Mar 2004 (HHS Reference No. E–275– 2002/1–PCT–01)

U.S. Patent Application No. 10/ 526,697 filed 05 May 2005 (HHS Reference No. E–275–2002/1–US–02) *Licensing Status:* Available for

exclusive and non-exclusive licensing. Licensing Contact: Michelle A.

Booden, Ph.D.; 301/451–7337; boodenm@mail.nih.gov

Collaborative Research Opportunity: The NCI Surgery Branch is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize ACT therapy. Please contact Steven A. Rosenberg, M.D., Ph.D. at 301–496–4164 for more information.

Dated: August 3, 2006.

Steven M. Ferguson,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health. [FR Doc. E6–13193 Filed 8–11–06; 8:45 am]

BILLING CODE 4140-01-P

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, Cardiovascular Cell Therapy Research Network Review.

Date: August 14-15, 2006.

Time: 8 a.m. to 5:30 p.m.

Agenda: To review and evaluate grant applications.

Place: Bethesda Marriott, 5151 Pooks Hill Road, Bethesda, MD 20814.

Contact Person: David A. Wilson, PhD, Scientific Review Administrator, Review Branch, Division of Extramural Affairs, National Heart, Lung, and Blood Institute, National Institutes of Health, 6701 Rockledge Drive, Room 7204, MSC 7924, Bethesda, MD 20892, 301/435–0929, *wilsond@nhlbi.nih.gov.*

This notice is being published less than 15 days prior to the meeting due to the timing limitations imposed by the review and funding cycle.

Name of Committee: National Heart, Lung, and Blood Institute, Special Emphasis Panel, Minority Undergraduation Biomedical Education.

Date: August 16, 2006.

Time: 9 a.m. to 12 p.m.

Agenda: To review and evaluate grant applications.