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, MD 20852–3804; telephone: 301/496–0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Complement Regulatory Gene Variants as Predictive Tests for Age-related Macular Degeneration (AMD)

Description of Technology

Age-related macular degeneration (AMD) is complex multigenic disorder that affects the central region of the retina (macula) and is the leading cause of legal blindness in developed countries. Age, lifestyle (e.g., smoking, diet) and genetic predisposition are major risk factors for AMD and 1.75 million adults over 40 are affected by advanced AMD in the United States with a further 7 million considered to be at risk (defined by the presence of large retinal deposits or drusen, which are the hallmark of this disease). A variety of immune-associated molecules including immunoglobulins, complement components, activators and regulators, etc. are associated with drusen and evidence suggests that AMD, like other age-related diseases such as Alzheimer's disease and atherosclerosis, involves a major inflammatory component. Several disease-susceptibility genes have been identified in family studies of macular degeneration and in patient cohorts by several groups including NIH researchers and their collaborators, and variants in the factor H gene (CFH)), a major inhibitor of the alternative complement pathway, have been associated with the risk for developing AMD.

NIH researchers and their collaborators have now extended this work to two other regulatory genes of this pathway, Factor B (BF) and complement component 2 (C2). These genes were screened for genetic variation in two independent cohorts comprised of ~900 AMD cases and ~400 matched controls. Haplotype analyses revealed a significant common risk haplotype (H1) and two protective haplotypes (H7 and H10). Combined analysis of the C2/BF haplotypes and CFH variants shows that variation in the two loci can predict the clinical outcome in 74% of the cases and 56% of the controls (Nature Genetics (2006) 38, 458). This suggests that these variants can be used as predictive genetic tests in combination with other potential risk factors.

Available for licensing are methods for identifying a subject at increased risk for developing AMD by determining the presence of protective genotypes at either the BF/C2 locus and at the CFH locus. Microarrays and kits are also provided. The complex and polygenic nature of AMD suggests that the protective and risk haplotypes claimed here can be of great value not only to companies targeting Macular Degeneration but perhaps more broadly to those involved in complement-mediated inflammatory disorders.

Inventors

Michael Dean (NCI), Bert Gold (NCI) et al.

Patent Status

U.S. Provisional Patent Application No. 60/772,989, filed 13 February 2006 (HHS Reference No. E–042–2006/0–US–01).

Licensing Status

Available for non-exclusive or exclusive licensing.

Licensing Contract

Susan Carson, D.Phil.; 301–435–5020; mail to: *carsonsu@mail.nih.gov.*

Collaborative Research Opportunity

The NCI Laboratory of Genomic Diversity is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize functional or genetic tests on complement genes and proteins. Please contact Kathleen Higinbotham at 301–846–5465 for more information.

Dated: July 28, 2006.

Steven M. Ferguson,

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

[FR Doc. 06–6879 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 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.

Licensing Opportunity

From the National Institutes of Health

Target-Specific Activatable Optical Probes for In Vivo Imaging

Description of Technology

Available for licensing and commercial development is an optical imaging method capable of detecting living cancer cells in vivo. The method increases sensitivity and reduces the background signal to extremely low levels. In contrast to conventional fluorescent imaging, the strategy activates the probe after it binds to and is internalized within cancer cells. Using antibodies, reagent-receptor systems, or cytokines to target the agent to the cancer, the agent is internalized by the normal cellular process of endocytosis which in turn, leads to molecular changes within the probe itself; fluorophores are activated only in the living targeted cells.

An activatable fluorophore is one that is normally self-quenched by attachment to a peptide backbone but which can be activated by specific proteases which degrade the peptide resulting in ''de-quenching.'' For example, self-quenching avidin-rhodaminex, which has affinity for lectin on cancer cells, is activated after endocytosis and degradation within the lysosome. Cellular internalization of receptor-ligand pairs with subsequent activation of fluorescence via ''de-quenching'' provides a generalizable and highly sensitive method of detecting cancer microfoci in vivo and has practical implications for assisting surgical and endoscopic procedures.

Application(s)

2. Optical detection of tumor cells and metastatic nodules

4. Photodynamic treatment of tumors

Market

• Cancer Imaging

Development Status

• Early-stage technology with pre-clinical 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, non-exclusive 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 inventions to extend market coverage for companies and may also be available for licensing.

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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-protein-coupled receptor, FPRL–1, in the cellular uptake and fibrillar aggregation of amyloid ββ(Aββ) peptides. The Aββ 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 protein-coupled 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 G-protein 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.