

Hyperthyroidism, or an overactive thyroid gland, affects about 1% of people in the United States and is often caused by autoimmune over-stimulation of the thyroid gland (Graves' disease), or by thyroid tumors. Drugs currently used for treatment of hyperthyroidism inhibit synthesis of thyroid hormones; the TSH receptor antagonist compounds encompassed by this technology have the advantage of directly inhibiting activity of the TSH receptor, rather than inhibiting thyroid hormone synthesis.

Applications:

- Diagnostic tools for evaluation and treatment of thyroid cancer.

- Therapeutics for thyroid cancer, hyperthyroidism, and hypothyroidism.

Market: Approximately 1 in 13 Americans suffers from a thyroid disorder, and 10 million have a thyroid-related condition that requires ongoing immunodiagnostic monitoring.

Development Status: Early stage.

Inventors: Marvin C. Gershengorn *et al.* (NIDDK)

Publications:

1. S Moore, H Jaeschke, G Kleinau, S Neumann, S Costanzi, JK Jiang, J Childress, BM Raaka, A Colson, R Paschke, G Krause, CJ Thomas, MC Gershengorn. Evaluation of small-molecule modulators of the luteinizing hormone/choriogonadotropin and thyroid stimulating hormone receptors: structure-activity relationships and selective binding patterns. *J Med Chem.* 2006 Jun 29;49(13):3888–3896.

2. S Neumann, G Kleinau, S Costanzi, S Moore, BM Raaka, CJ Thomas, G Krause, MC Gershengorn. A low molecular weight antagonist for the human thyrotropin receptor with therapeutic potential for hyperthyroidism. *Endocrinology* 2008 Dec;149(12):5945–5950.

3. Unpublished data are also available for review under a CDA.

Patent Status:

HHS Reference Nos. E–223–2006/0 and E–223–2006/1 —

- International Patent Application No. PCT/US2007/011951 filed 17 May 2007, which published as WO 2007/136776 on 29 Nov 2007.

- National Phase entered in Australia, Canada, Europe, Japan, and the United States.

HHS Reference No. E–284–2008/0 —

- International Patent Application No. PCT/US2008/011958 filed 20 Oct 2008.

Licensing Status: Available for licensing.

Licensing Contact: Tara L. Kirby, PhD; 301–435–4426; tarak@mail.nih.gov.

Collaborative Research Opportunity: The NIDDK, Clinical Endocrinology Branch is seeking statements of

capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize small molecule TSH receptor modulators. Please contact Patricia Mello Lake; 301–451–3636; lakep@mail.nih.gov for more information.

April 3, 2009.

Richard U. Rodriguez,

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

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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.

Selective Killing of Cancer Cells by Inhibition of Geminin

Description of Technology: The current strategy for developing cancer therapeutics is to identify unique differences between cancer cells and normal cells that can serve as specific targets for chemotherapeutic drugs, thereby allowing elimination of cancer cells with minimal toxicity to normal tissues. Geminin, an inhibitor of DNA replication, is typically undetectable in normal cells while rapidly proliferating cancer cells express geminin and hence could be targeted for cancer treatment.

The NIH researchers have discovered that inhibition of geminin expression induced DNA re-replication in most of the tested cancer cell lines, but not in matched non-cancer cell lines from the same tissues. DNA re-replication occurs when DNA synthesis is initiated multiple times from the same replication origin during one cycle of cell division resulting in DNA damage which halts cell proliferation and induces apoptosis in a wide variety of cancer cells. The researchers also analyzed the effect of suppression of geminin expression on apoptosis and cell survival in cancer and non-cancer cell lines. They found that the geminin siRNA induced apoptosis in a colon carcinoma cell line, but not in a normal skin fibroblast cell line. Furthermore, suppression of geminin expression markedly reduced cell survival of several cancer cell lines, but not non-cancer cell lines. Therefore, suppressing the level of geminin expression can be potentially used to selectively kill cancer cells.

Applications: Therapeutic for treating breast, colon and rectal, kidney (renal cell), lung, brain, and bone cancers.

Advantages: Targeted therapeutic; No requirement for use of other cell cycle inhibitors

Market: Cancer continues to be a burden to the public health of Americans. After heart disease, cancer is the most common cause of death in the United States. For 2008, it was estimated that about 565,650 Americans were expected to die of cancer. The incidence of cancer has been dropping over the years but it is estimated that over 1.4 million Americans would be diagnosed with cancer in 2008. Therefore, there is a continued need for the development of new therapies to effectively treat this disease.

Inventors: Wenge Zhu and Melvin L. DePamphilis (NICHD)

Publications: Paper accepted for publication in *Cancer Research*.

Patent Status: U.S. Provisional Application No. 61/106,465 filed 17 Oct 2008 (HHS Reference No. E–324–2008/0–US–01)

Licensing Status: Available for licensing.

Licensing Contact: Betty Tong, PhD; 301–594–6565; tongb@mail.nih.gov.

Transgenic Mice in Which the Gene for MCP–1 Is Deleted

Description of Technology: Dr. Yoshimura has developed a transgenic mouse which does not express the chemokine MCP–1 due to a deletion of the gene for MCP–1. MCP–1 is a CC chemokine which is responsible for recruiting monocytes into sites of

inflammation and cancer. Using a thioglycollate challenge as a measure of the impact of the deletion of MCP-1, MCP-1 deficient mice exhibit a 60% reduction in the number of monocytes/macrophages at 96 hours compared to wild type mice. Unlike previously generated MCP-1 deficient mice in which the expression of the neighboring gene for MCP-3 is down-regulated (our own data), the expression of MCP-3 is up-regulated in this mouse model.

Applications: This mouse may be useful as an in vivo model for evaluating the role of MCP-1 and MCP-3 in cancer or other diseases associated with inflammation due to the accumulation of monocytes.

Inventor: Teizo Yoshimura (NCI)

Patent Status: HHS Reference No. E-241-2005/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Status: Available for licensing under a Biological Materials License Agreement.

Licensing Contact: Betty Tong, PhD; 301-594-6565; tongb@mail.nih.gov.

Collaborative Research Opportunity: The National Cancer Institute, Center for Cancer Research, Laboratory of Molecular Immunoregulation, Cancer and Inflammation Program, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize agents useful to treat patients with inflammation or cancer. Please contact John D. Hewes, PhD at 301-435-3121 or hewesj@mail.nih.gov for more information.

DU145 Camptothecin (CPT)-Resistant Cell Line

Description of Technology: Drug resistance is a major limitation of chemotherapy. Understanding how drug resistance develops may lead to more effective treatments. This invention describes the DU145 Camptothecin (CPT)-resistant prostate cancer cell line that can be used to study mechanisms of drug resistance.

Inventor: Yves G. Pommier (NCI)

Related Publication: Y Urasaki *et al.* Characterization of a novel topoisomerase I mutation from a camptothecin-resistant human prostate cancer cell line. *Cancer Res.* 2001 Mar 1;61(5):1964-1969.

Patent Status: HHS Reference No. E-159-2005/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Status: Available for licensing under a Biological Materials License Agreement.

Licensing Contact: Betty Tong, PhD; 301-594-6565; tongb@mail.nih.gov.

Creation and Characterization of Carcinogen-Altered Mouse Epidermal Cell Lines

Description of Technology: The invention relates to the creation of three (3) cell lines that may be used as models of putative initiated cancer cells. The cell lines can be used in basic research assays and low/high throughput screening assays.

Cell line 308 evolved from a calcium-resistant focus from adult mouse epidermis that was exposed to the carcinogen, 7,12-dimethylbenz[a]anthracene (DMBA). Cell lines F and D were derived by treating primary newborn mouse epidermal cells in culture with *N*-methyl-*N*-nitro-*N*-nitrosoguanidine (MNNG) and DMBA, respectively. These three (3) noncancerous cell lines derived from differentiation-resistant, carcinogen-induced foci may be considered to be putative initiated cells.

Inventor: Stuart H. Yuspa (NCI)

Related Publications:

1. SH Yuspa and DL Morgan. Mouse skin cells resistant to terminal differentiation associated with initiation of carcinogenesis. *Nature* 1981 Sep 3;293(5287):72-74.

2. H Hennings *et al.* Response of carcinogen-altered mouse epidermal cells to phorbol ester tumor promoters and calcium. *J Invest Dermatol.* 1987 Jan;88(1):60-65.

Patent Status: HHS Reference No. E-154-2004/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Status: Available for licensing under a Biological Materials License Agreement.

Licensing Contact: Betty Tong, PhD; 301-594-6565; tongb@mail.nih.gov.

A Mouse Model for Conditional Gene Deletion of c-Met Receptor

Description of Technology: c-Met oncogene has been implicated in a variety of human cancers as well as degenerative diseases. Signaling via the c-Met receptor is essential for survival as evidenced by the embryonal death of mice in which the c-Met has been deleted. Further analysis of the role of the signaling pathway supported by c-Met receptor in the adult organism is hindered by its embryonic lethality. The establishment of a mouse model for the conditional c-Met gene deletion will provide a unique opportunity to explore the function of c-Met in the adult mouse by selectively deleting the receptor gene in various tissues. Such a mouse model is established at the National Institutes of Health and available for licensing.

Applications:

- Animal model to study the physiological role of the c-Met receptor.
- Animal model for testing potential drug targeted to the c-Met signal transduction pathway.

Inventor: Snorri S. Thorgeirsson (NCI)

Patent Status: HHS Reference No. E-048-2003/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Status: c-Met receptor conditional KO mice are available for licensing.

Licensing Contact: Betty Tong, PhD; 301-594-6565; tongb@mail.nih.gov.

Dated: April 6, 2009.

Richard U. Rodriguez,

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

[FR Doc. E9-8212 Filed 4-9-09; 8:45 am]

BILLING CODE 4140-01-P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Amended Notice of Meeting

Notice is hereby given of a change in the meeting of the Center for Scientific Review Special Emphasis Panel, March 26, 2009, 12 p.m. to March 27, 2009, 3 p.m., National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892 which was published in the **Federal Register** on March 12, 2009, 74 FR 10748.

The meeting will be held April 16, 2009, 3 p.m. to April 17, 2009, 6 p.m. The meeting location remains the same. The meeting is closed to the public.

Dated: April 2, 2009.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. E9-8018 Filed 4-9-09; 8:45 am]

BILLING CODE 4140-01-M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Amended Notice of Meeting

Notice is hereby given of a change in the meeting of the Center for Scientific Review Special Emphasis Panel, April 20, 2009, 9 a.m. to April 21, 2009, 3 p.m., National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892 which was published in the **Federal Register** on March 31, 2009, 74 FR 14570-14571.