

involved. Present treatments for UC include anti-inflammatory therapy using aminosalicylates or corticosteroids, as well as immunomodulators and diet. However, 25–40 percent of ulcerative colitis patients must eventually have their colons removed due to massive bleeding, severe illness, rupture of the colon, risk of cancer or due to side effects of corticosteroids and novel treatments are still actively being sought. NIH scientists and their collaborators have used a mouse model of experimental colitis (OC) to show that IL-13, a Th2 cytokine, is a significant pathologic factor in OC and that neutralizing IL-13 in these animals effectively prevents colitis (Immunity (2002) 17, 629–638).

OC is a colitis induced by intrarectal administration of a relatively low dose of the haptening agent oxazolone subsequent to skin sensitization with oxazolone. A highly reproducible and chronic colonic inflammation is obtained that is histologically similar to human ulcerative colitis. Studies show that NKT cells rather than conventional CD4+T cells mediate oxazolone colitis and that NKT cells are the source of IL-13, and are activated by CD1 expressing intestinal epithelial cells. Tissue removed from UC patients were also shown to contain increased numbers of nonclassical NKT cells that produce markedly increased amounts of IL-13 and that in keeping with epithelial damage being a key factor in UC, these NKT cells are cytotoxic for epithelial cells (J. Clin. Investigation (2004) 113, 1490–1497).

With obvious implications for the treatment of human Ulcerative Colitis, inflammation in this mouse model has been shown to be effectively blocked by neutralizing IL-13 or by inhibiting the activation of NK-T cells through CD1. Available for licensing are broad claims covering treatments preventing the inflammatory response of colitis by modulating IL-13 and NKT cell activity and methods for screening for therapeutic compounds effective for colitis.

In addition to licensing, the technology is available for further development through collaborative research with the inventors via a Cooperative Research and Development Agreement (CRADA).

Dated: January 18, 2005.

**Steven M. Ferguson,**

*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.

**Null Mutation of the CCAAT/Enhancer Binding Protein Delta (Cebpd) Gene in Mice**

G. Esta Sterneck *et al.* (NCI); DHHS Reference No. E–032–2005/0—Research Tool; Licensing Contact: John Stansberry; 301/435–5236; [stansbej@mail.nih.gov](mailto:stansbej@mail.nih.gov).

The invention describes mice with a deletion of the C/EBPdelta gene and cell lines derived from such mice. C/EBPdelta (CCAAT/enhancer binding protein delta) is implicated in the acute phase inflammatory response, long-term memory, fat cell and osteoblast differentiation, ovarian hormone responses, mammary gland involution and cell death. C/EBPdelta may also be a tumor suppressor. Fibroblasts lacking C/EBPdelta exhibit transformed features such as impaired contact inhibition, reduced serum dependence and chromosomal instability. The mice and cell lines of the invention could be useful for the study of the function of C/EBPdelta such as its potential role in cancer, and to investigate how drug responses are modified in the absence of C/EBPdelta.

In addition to licensing, the technology is available for further development through collaborative

research with the inventors via a Cooperative Research and Development Agreement (CRADA).

**Active Chromatin Domains Are Defined by Acetylation Islands Revealed by Genome-Wide Mapping**

Drs. Keji Zhao and Tae-Young Roh (NHLBI);

U.S. Provisional Application No. 60/619,430 filed 15 Oct 2004 (DHHS Reference No. E–008–2005/0–US–01); Licensing Contact: John Stansberry; 301/435–5236; [stansbej@mail.nih.gov](mailto:stansbej@mail.nih.gov).

Epigenetics play a critical role in cellular development and cellular transformation in many pathogenic processes. For example, many cancers are correlated with changes of their chromatin structure and are sensitive to drugs that modulate the levels of histone acetylation. Epigenetic regulation refers to the modification of histones, which does not involve changes of DNA sequences of target genes. The present technology maps the genome-wide distribution of histone H3 acetylation in human T cells and describes over 40,000 acetylation islands. These acetylation islands are epigenetic markers for transcriptional regulatory elements and chromatin-controlling elements. Changes in acetylation islands may be correlated with early development of T cell lymphoma or leukemia. Specifically, diseases characterized by aberrant transcriptional regulation could be diagnosed earlier with the application of this technology.

**Method of Detecting Cancer Based on Immune Reaction to BORIS**

Victor Lobanekov *et al.* (NIAID); U.S. Provisional Application No. 60/611,798 filed 21 Sep 2004 (DHHS Reference No. E–241–2004/0–US–01); Licensing Contact: Mojdeh Bahar; 301/435–2950; [baharm@mail.nih.gov](mailto:baharm@mail.nih.gov).

The invention provides a method of detecting autoantibodies to BORIS (brother of the regulator of imprinted sites) as a possible screen for cancer and a kit comprising BORIS peptides and epitopes. BORIS is a protein that is expressed in many cancers but not in normal tissues (except testis) and thus is a potential target for a cancer therapeutic or diagnostic.

Importantly, BORIS is a cancer-testis (CT) antigen, which despite that it is intracellular protein upon abnormal expression in cancer it appears to be immunogenic in humans. Thus, BORIS could be employed in cancer diagnosis using serum from patients. In fact, the inventors detected BORIS-specific antibodies in serum from patients with gliomas, lung, breast and prostate

cancer, but not in serum from normal controls.

Few other serum markers are currently in use for cancer diagnosis and they have limited predictive power. Thus, the detection of tumor related anti-BORIS antibodies suggests that the invention has great potential for detection and treatment of a wide variety of cancers.

In addition, the background of the current invention is found in DHHS Reference No. E-227-2001.

### Primer and Probe Sequences for Use in a Diagnostic Tool for Diagnosing Benign Versus Malignant Thyroid Lesions

Steven K. Libutti *et al.* (NCI); U.S. Provisional Application No. 60/622,643 filed 26 Oct 2004 (DHHS Reference No. E-124-2004/1); Licensing Contact: Mojdeh Bahar; 301/435-2950; [baharm@mail.nih.gov](mailto:baharm@mail.nih.gov).

The present invention discloses primer and probe sequences that can be used for distinguishing between benign and malignant thyroid lesions. Analysis of thyroid lesions by traditional means, such as fine needle biopsy, can result in indeterminate results. Thus, there is a need for methods that increase the precision of diagnosis. The primers and probes represent a 6 gene or 10 gene model for diagnosing benign from malignant thyroid cancer. Analysis of these genes in thyroid lesions taken from patients could be used for molecular classification of the lesions.

In addition to licensing, the technology is available for further development through collaborative research with the inventors via a Cooperative Research and Development Agreement (CRADA).

### New Gene Encoding a Membrane Protein Highly Expressed in Many Breast Cancers and Not in Normal Tissues

B. Lee, K. Egland, and I. Pastan (NCI); U.S. Provisional Application No. 60/493,522 filed 08 Aug 2003 (DHHS Reference No. E-292-2003/0-US-01); U.S. Patent Application No. 10/913,196 filed 05 Aug 2004 (DHHS Reference No. E-292-2003/0-US-02); PCT Application No. PCT/US04/25448 filed 06 Aug 2004 (DHHS Reference No. E-292-2003/0-PCT-03); Licensing Contact: Brenda Hefti; 301/435-4632; [heftib@mail.nih.gov](mailto:heftib@mail.nih.gov).

The current invention relates to a new polypeptide (termed 68h05) that is specifically detected in breast cancer and prostate cancer cells, and not in normal tissue. In addition, 16 out of 21 breast tumors and three out of three prostate tumors expressed 68h05. This

invention might have utility as a vaccine therapeutic, antibody-based therapeutic, immunoconjugate therapeutic, or as a diagnostic for the diagnosis or treatment of breast or prostate cancer.

Dated: January 19, 2005.

**Steven M. Ferguson,**

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

[FR Doc. 05-1419 Filed 1-25-05; 8:45 am]

BILLING CODE 4140-01-P

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

#### Fogarty International Center; Notice of Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the Fogarty International Center Advisory Board.

The meeting will be open to the public as indicated below, 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.

The meeting 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/or contract proposals 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 and/or contract proposals, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

*Name of Committee:* Fogarty International Center Advisory Board.

*Date:* February 7-8, 2005.

*Closed:* February 7, 2005, 1:30 p.m. to Adjournment.

*Agenda:* To review and evaluate grant applications and/or proposals.

*Place:* National Institutes of Health, Lawton Chiles International House, Bethesda, MD 20892.

*Open:* February 8, 2005, 8:30 a.m. to Adjournment.

*Agenda:* A Report of the FIC Director on updates and overviews of new FIC initiatives. Topics to be discussed: Fogarty in Brazil: A Genealogy of Infectious Disease Training and Research.

*Place:* National Institutes of Health, Lawton Chiles International House, Bethesda, MD 20892.

*Contact Person:* Jean L. Flagg-Newton, PhD, Special Assistant to the Director, FIC, Fogarty International Center, National Institutes of Health, 9000 Rockville Pike, Building 31, Room B2C29, Bethesda, MD 20892, (301) 496-2968, [flaggnej@mail.nih.gov](mailto:flaggnej@mail.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.

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.

Information is also available on the Institute's/Center's home page: [www.nih.gov/fic/about/advisory.html](http://www.nih.gov/fic/about/advisory.html), where an agenda and any additional information for the meeting will be posted when available.

(Catalogue of Federal Domestic Assistance Program Nos. 93.106, Minority International Research Training Grant in the Biomedical and Behavioral Sciences; 93.154, Special International Postdoctoral Research Program in Acquired Immunodeficiency Syndrome; 93.168, International Cooperative Biodiversity Groups Program; 93.934, Fogarty International Research Collaboration Award; 93.989, Senior International Fellowship Awards Program, National Institutes of Health, HHS)

Dated: January 18, 2005.

**LaVerne Y. Stringfield,**

*Director, Office of Federal Advisory Committee Policy.*

[FR Doc. 05-1348 Filed 1-25-05; 8:45 am]

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## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

#### National Center for Research Resources; Notice of 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 open to the public as indicated below, 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.

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