Patent Status: U.S. Provisional Patent Application filed 07 Apr 2006 (HHS Reference No. E–336–2005/0–US–01).

Licensing Status: This technology is available for licensing under an exclusive or non-exclusive patent license.

Licensing Contact: Michelle A. Booden, PhD; 301/451–7337; *boodenm@mail.nih.gov*.

Collaborative Research Opportunity: The NCI Center for Cancer Research Nanobiology Program is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize monoclonal antibodies to treat human diseases. Please contact Melissa Maderia at *maderiam@mail.nih.gov* or by phone at (301) 846–5465 for more information.

Immortal Human Prostate Epithelial Cell Cultures as a Prostate Cancer Model

Description of Technology: The National Institutes of Health has multiple immortalized, malignant, human, adult prostate epithelial cell lines available for license. They are useful as models in epithelial cell oncogenesis studies and in the diagnosis and treatment of prostate cancer.

The cell lines were generated from primary adenocarcinomas of the prostate. Long-term cultures were established by immortalizing cells with human papillomavirus (HPV) transforming proteins. The cultures were characterized and single-cell clones with unique genetic characteristics were selected based on allelic loss of heterozygosity (LOH). Tissue-matched normal cell lines are available also, useful for the appropriate controls.

The invention also encompasses polyclonal and monoclonal antibodies directed to the cell lines, which may be useful as immunotherapeutics.

Applications: (1) Screening tool to identify novel genes unique to or overexpressed in prostate cancer; (2) Raising of prostate cancer-reactive antibodies, useful as immunotherapeutics or diagnostics; (3) Screen for compounds that kill tumor cells and represent potential therapeutic agents; (4) Identification of prostate cancer antigens to develop recombinant prostate cancer vaccines.

Inventors: Susan L. Topalian, W. Marston Linehan, Robert K. Bright, Cathy D. Vocke (NCI).

Publication: R.K. Bright, *et al.*, ''Generation and genetic characterization of immortal human prostate epithelial cell lines derived from primary cancer specimens,'' *Cancer Res.* 1997 Mar 5;57(5):995–1002.

Patent Status: U.S. Patent 6,982,168 issued on 07 May 2003 (HHS Reference No. E–053–1996/0–US–03).

Licensing Status: Available for non-exclusive internal use and biological material license.

Licensing Contact: Michelle A. Booden, PhD; 301/451–7337; *boodenm@mail.nih.gov*.

Collaborative Research Opportunity: The NCI Center for Cancer Research, Surgery Branch, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact Brian W. Bailey, PhD, at 301/451–2158 or *bbailey@mail.nih.gov* for more information.

Dated: June 21, 2006.

David R. Sadowski,

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

[FR Doc. 06–5867 Filed 6–27–06; 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.

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.

Treatment of Inflammatory Bowel Disease (IBD) Using NF–KB Decoy Polynucleotides

Warren Strober (NIAID), Ivan Fuss (NIAID), Atsushi Kitani (NIAID), and Stefan Fichtner-Feigl (NIAID)

U.S. Patent Application No. 11/125,919 filed 10 May 2005 (HHS Reference No. E–108–2005/0–US–01); PCT International Application filed 10 May 2006 (HHS Reference No. E–108–2005/0–PCT–02)

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

Inflammatory Bowel Diseases (IBDs; Crohn's disease and ulcerative colitis) are chronic inflammatory disorders affecting almost 1 million people in the developed world at an estimated annual cost of one billion dollars in lost work days. Current treatments include corticosteroids, 5-aminosalicylates and immunomodulators but novel and more effective therapies without adverse side effects continue to be needed. NIH researchers have previously shown that a variety of immunomodulators affecting the Th1 and Th2 T cell responses which underlie Inflammatory Bowel Diseases can be used to treat IBD disease models and have now extended this work by inhibiting NF–KB transcriptional activity in a variety of animal models using decoy oligodeoxynucleotides (decoy ODNs).

Dr. Strober and colleagues at the National Institute of Allergy and Infectious Diseases (NIAID) have shown that intrarectal (i.r.) or intraperitoneal (i.p.) administration of decoy ODNs encapsulated in a viral envelope (HVJ–E) prevented and treated a model of acute trinitrobenzene sulfonic acid-induced (TNBS-induced) colitis, a model for Crohn's disease, as assessed by clinical course and the effect on Th1 cytokine production. NF–KB decoy ODNs were also shown to be an effective treatment of a model of chronic TNBS-colitis, inhibiting both the production of IL–23/Il–17 and the development of fibrosis that characterizes this model. Treatment of TNBS-induced inflammation by i.r. administration of NF–KB decoy ODNs did not inhibit NF–KB in extraintestinal organs and resulted in CD4+ T cell apoptosis, suggesting that such treatment is highly focused and durable. Additionally, NF–KB decoy ODNs also prevented and treated oxazolone-colitis, a mouse model for ulcerative colitis, and thus affected a Th2-mediated inflammatory process. In each case, decoy administration led to inflammation clearing effects, suggesting a therapeutic potency

applicable to human IBD [J. Clin. Invest. (2005) 115, 3057–3071].

Available for licensing are methods for treating or preventing the inflammatory response of IBDs by intrarectally or intraperitoneally administering a therapeutic effective amount of NF–KB decoy ODN. Claims are directed to treatment of Th1 and Th2 inflammatory response and these studies suggest that NF–KB decoy ODNs targeting the consensus NF–KB binding site and encapsulated in a viral envelope represent an effective approach for the treatment of IBDs.

Related IBD technologies available for licensing also include IL–13 modulators and inhibitors (HHS Reference No. E–131–2002/0–PCT–02, WO 2004/001655, filed 14 June 2002) and IL–13 mutant and chimeric molecules (HHS Reference No. E–003–2005/0–US–01, U.S. Provisional Patent Application No. 60/671,624 filed 15 April 2005).

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Treatment and Prevention of Inflammatory Bowel Disease (IBD) using Mutant and Chimeric IL–13 Molecules

Warren Strober (NIAID), Ivan Fuss (NIAID), Peter Mannon (NIAID), Jan Preiss (NIAID), Raj Puri (FDA), Koji Kawakami (FDA), Stefan Fichtner-Feigl (NIAID), and Atsushi Kitani (NIAID)

U.S. Provisional Patent Application No. 60/671,624 filed 15 April 2005 (HHS Reference No. E–003–2005/ 0-US–01); PCT International Application filed 14 April 2006 (HHS Reference No. E–003–2005/0–PCT–02)

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

Ulcerative colitis (UC) is a chronic inflammatory disease of the colorectum and affects approximately 400,000 people in the United States. The cause of UC is not known, although an abnormal immunological response to bacterial antigens in the gut microflora is thought to be involved. Present treatments for UC include anti-inflammatory therapy using aminosalicylates or corticosteroids, as well as immunomodulators and diet. However, 25–40% 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 (oxazolone 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 haptenating 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 Natural Killer T (NKT) cells, rather than conventional CD4+T cells, mediate oxazolone colitis and are the source of IL–13 as well as being activated by CD1-expressing intestinal epithelial cells. Tissue removed from ulcerative colitis 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. Invest. (2004) 113, 1490–1497]. Building on their previous work, scientists at NIAID and FDA have shown that an Il-13 chimeric fusion protein linked to an effector molecule was able to prevent colitis in a mouse model of ulcerative colitis.

Available for licensing are methods for treating or preventing the inflammatory response of IBD by inhibiting the binding of IL–13 to IL–13 receptors on NKT cells. Additionally, these mutant and chimeric Il-13 molecules are able to block the chronic inflammatory response that results in fibrosis as seen in Crohn's disease. Preventing the inflammatory response of colitis by either modulating or blocking IL–13 and NKT cell activity continues to be an effective therapeutic approach in animal models of colitis with implications for the treatment of human ulcerative colitis and for the treatment of fibrosis associated with Crohn's disease.

Related IBD technologies available for licensing also include IL–13 modulators and inhibitors (HHS Reference No. E–131–2002/0–PCT–02, WO 2004/001655, filed 14 June 2002) and NF-kappa B decoy oligonucleotides [HHS Reference No. E–108–2005/0–US–01, U.S. Patent Application No. 11/125,919, filed 10 May 2005; J. Clin. Invest. (2005) 115, 3057–3071].

In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Dated: June 21, 2006.

David R. Sadowski,

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

[FR Doc. 06–5868 Filed 6–27–06; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Center for Research Resources; 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 Center for Research Resources Special Emphasis Panel, CTSA Center Grants #1.

Date: July 11–12, 2006.

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

Agenda: To review and evaluate grant applications.

Place: Double Tree Rockville, 1750 Rockville Pike, Rockville, MD 20852.

Contact Person: Sheryl K. Brining, PhD, Scientific Review Administrator, Director, Office of Review, NCRR, National Institutes of Health, 6701 Democracy Boulevard, 1 Democracy Plaza, Room 1074, MSC 4874, Bethesda, MD 20892–4874. (301) 435–0811. *sb44k@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 Center for Research Resources Special Emphasis Panel, CTSA Center Grants #2.

Date: July 20–21, 2006.

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

Agenda: To review and evaluate grant applications.

Place: Double Tree Rockville, 1750 Rockville Pike, Rockville, MD 20852.

Contact Person: Guo Zhang, PhD, Scientific Review Administrator, National Center for Research Resources/OR, National Institutes of Health, 6701 Democracy Boulevard, 1 Democracy Plaza, Room 1064, Bethesda, MD 20892–4874. (301) 435–0812. *zhanggu@nih.gov*.