PCT Application No. PCT/US03/05186 filed 21 Feb 2003 (DHHS Reference No. E–227–2001/0–PCT–02)

Licensing Contact: Matthew Kiser; 301/435–5236; *kiserm@mail.nih.gov*

The subject application discloses an isolated or purified nucleic acid molecule consisting essentially of a nucleotide sequence encoding a human or a non-human BORIS, or a fragment of either of the foregoing; an isolated or purified nucleic acid molecule consisting essentially of a nucleotide sequence that is complementary to a nucleotide sequence encoding a human or a non-human BORIS, or a fragment of either of the following; a vector comprising such an isolated or purified polypeptide molecule consisting essentially of an amino acid sequence encoding a human or a non-human BORIS, or a fragment or either of the foregoing; a cell line that produces a monoclonal antibody that is specific for an aforementioned isolated or purified polypeptide molecule; and the monoclonal antibody produced by the cell line; methods of diagnosing a cancer or a predisposition to a cancer in a male or female mammal; a method of prognosticating a cancer in a mammal; a method of assessing the effectiveness of treatment of a cancer in a mammal; a method of treating a mammal prophylactically or therapeutically for a cancer; and a composition comprising a carrier and an inhibitor of BORIS.

Use of IL–13 Inhibitors To Prevent Tumor Recurrence

Jay Berzofsky *et al.* (NCI).

PCT Application No. PCT/US01/51339 filed 22 Oct 2001 (DHHS Reference No. E–037–2001/1–PCT–02).

Licensing Contact: Catherine Joyce; 301/435–5031; e-mail: *joycec@mail.nih.gov*

This invention relates to the discovery of a role for IL–13 in the down-regulation of tumor immunosurveillance. Using a mouse model in which tumors show a growth-regression-recurrence pattern, the mechanisms for down-regulation of cytotoxic T lymphocyte-mediated tumor immunosurveillance was investigated. It was discovered that interleukin 4 receptor (IL–4R) knockout mice, and downstream signal transducer and activator of transcription 6 (STAT6) knockout mice, but not IL–4 knockout mice, resisted tumor recurrence. Thus, IL–13, the only other cytokine that uses the IL–4R-STAT6 pathway, was discovered to have a role in the down-regulation of tumor immunosurveillance. The use of an IL–13 inhibitor confirmed these results.

Additionally, loss of natural killer T cells (NKT cells) in CD1 knockout mice resulted in decreased IL–13 production and resistance to recurrence. Therefore, NKT cells and IL–13, possibly produced by NKT cells and signaling through the IL–4R-STAT6 pathway, are necessary for down-regulation of tumor immunosurveillance. Thus, the inventors have discovered a method of inhibiting tumor growth which comprises the administration of an IL–13 inhibitor. This invention is described in PCT application, PCT Publication No. WO 02/055100.

This technology is available for licensing on a non-exclusive basis.

Interleukin-2 Stimulated T-Lymphocyte Cell Death for the Treatment of Autoimmune Diseases, Allergic Disorders and Graft Rejection

Michael J. Lenardo (NIAID).

U.S. Patent 6,083,503 issued 07 Jul 2000 (DHHS Reference No. E–137–1991/0–US–03); U.S. Patent 5,989,546 issued 23 Nov 1999 (DHHS Reference No. E–137–1991/0–US–04).

Licensing Contact: Matthew Kiser; 301/435–5236; *kiserm@mail.nih.gov*

T-cell apoptosis induced by administration of IL–2 and antigen offers an important new treatment for allergic disorders, which are due to the effects of antigen-activated T-cells. Antigen-activated T-cells cause the release of harmful lymphokines and the production of immunoglobulin E by B cells. Presently available methods for treating allergies have limitations because they are nonspecific in their action and have side effects and limited efficacy. IL–2 and antigen stimulates the programmed death of only antigen-specific T-cells while leaving the rest of the patient's T-cells and other immune cells intact. This invention is also useful in treating HIV. Both fields of use, allergies and HIV, are available for licensing.

Interleukin-4 Stimulated T-Lymphocyte Cell Death for the Treatment of Autoimmune Diseases, Allergic Disorders and Graft Rejection

Michael J. Lenardo, Stefen A. Boehme, Jeffrey Critchfield (NIAID).

U.S. Patent 5,935,575 issued 10 Aug 1999 (DHHS Reference No. E–151–1992/0–US–11).

Licensing Contact: Matthew Kiser; 301/435–5236; *kiserm@mail.nih.gov*

The discovery that interleukin-4 (IL–4) predisposes T lymphocytes to programmed cell death (apoptosis) allows for a novel method of therapeutic intervention in diseases caused by the action of IL–4-responsive T cells. Specifically, the therapy induces the death of a subpopulation of T lymphocytes that are capable of causing disease. Current therapies may cause general death or suppression of immune responses involving T-cells, severely comprising a patient's immune system. This treatment affects only the subset of T cells that react with a specified antigen, thereby leaving a patients immune system uncompromised. This invention is useful in treating allergies and HIV complications. Both fields are available for licensing.

Dated: February 9, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–3526 Filed 2–18–04; 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, DHHS.

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.

SPATIAL for Altering Cell Proliferation

Ronald E. Gress, Francis A. Flomerfelt (NCI).

PCT Application No. PCT/US03/36874 filed 18 Nov 2003 (DHHS Reference No. E–177–2003/0–PCT–01).

Licensing Contact: Fatima Sayyid; (301) 435–4521; *sayyidf@mail.nih.gov.*

The present invention provides methods useful for altering cell proliferation by modifying SPATIAL, a gene expressed predominantly in thymus and lymph node, activity in cells. In some methods the thymocyte numbers in subjects with disease-associated immunodeficiencies are increased by administering an agent that inhibits SPATIAL activity. Other methods include but are not limited to increasing thymocyte number in a subject by administering an agent that interferes with an interaction between SPATIAL and Uba3.

Methods for the Treatment of Parkinson's Disease and Other alpha-synucleinopathies

M. Maral Mouradian and Eunsung Junn (NINDS)

U.S. Provisional Application No. 60/444,563 filed 02 Feb 2003 (DHHS Reference No. E–091–2003/0–US–01)

Licensing Contact: Norbert Pontzer; (301) 435–5502, *pontzern@mail.nih.gov*

Parkinson's disease (PD) is a neurodegenerative disorder characterized by the loss of dopaminergic neurons in the substantia nigra pars compacta. During the course of the disease, proteinaceous cytoplasmic inclusions known as Lewy bodies appear in the dopaminergic neurons. Several lines of evidence point to a key role for alpha-synuclein, a major constituent of Lewy bodies, in the pathogenesis of these disorders. In particular, the aggregation of this protein is believed to be deleterious to neurons. These inventors have now discovered that transglutaminase 2, also referred to as tissue transglutaminase, catalyzes alpha-synuclein cross-linking in vitro and in cultured cells. Evidence for the activity of this enzyme is also provided within the Lewy bodies in Parkinson's patients. The present invention provides novel methods for the treatment of Parkinson's disease and other alpha-synucleinopathies with inhibitors of transglutaminase, which can inhibit aggregation of alpha-synuclein. Also provided are screening assays for novel inhibitors of transglutaminase that may be used in the treatment of Parkinson's disease and other alpha-synucleinopathies. Further information may be found in Junn *et al.*, PNAS 2003 100(4): 2047–2052.

Dated: February 10, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–3527 Filed 2–18–04; 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 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 the following 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 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, Clinical Research.

Date: March 4, 2004.

Time: 8 a.m. to Adjournment.

Agenda: To review and evaluate grant applications.

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

Contact Person: Eva Petrakova, PhD, MPH, Scientific Review Administrator, Office of Review, National Center for Research Resources, National Institutes of Health, 6701 Democracy Boulevard, Room 1066, Bethesda, MD 20817–4874, (301) 435–0965, *petrakoe@mail.nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.306, Comparative Medicine; 93.333, Clinical Research; 93.371, Biomedical Technology; 93.389, Research Infrastructure, 93.306, 93.333, National Institutes of Health, HHS)

Dated: February 10, 2004.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 04–3520 Filed 2–18–04; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Diabetes and Digestive and Kidney Diseases; 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 Institute of Diabetes and Digestive and Kidney Diseases Special Emphasis Panel; Role of Icosanoids in Renal Function.

Date: March 9, 2004.

Time: 10:30 a.m. to 2:30 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, Two Democracy Plaza, 6707 Democracy Boulevard, Bethesda, MD 20892, (Telephone Conference Call).

Contact Person: Maxine A. Lesniak, PhM, Scientific Review Administrator, Review Branch, DEA, NIDDK, National Institutes of Health, Room 756, 6707 Democracy Boulevard, Bethesda, MD 20892–5452, (301) 594–7792, *lesniakm@extra.niddk.nih.gov.*

Name of Committee: National Institute of Diabetes and Digestive and Kidney Diseases Special Emphasis Panel; Mitochondrial Dysfunction: Role in Metabolic Syndrome.

Date: March 22, 2004.

Time: 2 p.m. to 5 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, Two Democracy Plaza, 6707 Democracy Boulevard, Bethesda, MD 20892, (Telephone Conference Call).

Contact Person: Paul A. Rushing, PhD, Scientific Review Administrator, Review Branch, DEA, NIDDK, National Institutes of Health, Room 747, 6707 Democracy Boulevard, Bethesda, MD 20892–5452, (301) 594–8895, *rushingp@extra.niddk.nih.gov.*

Name of Committee: National Institute of Diabetes and Digestive and Kidney Diseases Special Emphasis Panel; Obesity/Energy Balance.

Date: April 15, 2004.

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

Agenda: To review and evaluate grant applications.

Place: Bethesda Marriott Suites, 6711 Democracy Boulevard, Bethesda, MD 20817.

Contact Person: Paul A. Rushing, PhD, Scientific Review Administrator, Review Branch, DEA, NIDDK, National Institutes of Health, Room 747, 6707 Democracy Boulevard, Bethesda, MD 20892–5452, (301) 594–8895, *rushingp@extra.niddk.nih.gov.*

Name of Committee: National Institute of Diabetes and Digestive and Kidney Diseases Special Emphasis Panel; Training Applications.

Date: April 19, 2004.

Time: 3 p.m. to 4:30 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institutes of Health, Two Democracy Plaza, 6707 Democracy Boulevard, Bethesda, MD 20892, (Telephone Conference Call).