Subject, City, and State	Effective date
UNGAR-SARGON, JULIAN, NEW YORK, NY	09/05/96
VANDER KOOI, PAUL, ORANGE, IA	09/11/96
VENANZI, ENZO J., BLACKWOOD, NJ	09/09/96
WESSER, DAVID ROBERT, ARDSLEY, NY	09/05/96
FEDERAL/STATE EXCLUSION/SUSPENSION:	
CARLONI, EDMUND L., HARTSDALE, NY	09/11/96
CENTRAL CARE, INC., CONGERS, NY	09/09/96
FILIZ, GENCER, BROOKLYN, NY	09/09/96
GALLARDO, ANNETTE, CONGERS, NY	09/09/96
GOMES, GERSON, FLUSHING, NY	09/09/96
JEAN-BAPTISTE, ROOSEVELT, CHICAGO, IL	09/11/96
KLIMOVA, LUBOV T., BROOKLYN, NY	09/09/96
RIVAS, LUIS, BRONX, NY	09/09/96
OWNED/CONTROLLED BY CONVICTED/EXCLUDED:	
AMERICAN MEDICAL SERVICES, INC., VOORHEES, NJ	09/10/96
DEW TRANSPORTATION, TALLULAH, LA	09/15/96
MEDICINE SHOPPE, COLORADO SPRINGS, CO	09/10/96
SAFETY MEDICAL TRANSPORTATION, PENSACOLA, FL	09/15/96
DEFAULT ON HEAL LOAN:	
ANTHONY, STEVEN M., LAKE ELSINORE, CA	09/10/96
AVER, REVA B., PORTLAND, OR	09/10/96
BAKER, YVETTE KETAI, LITTLE ROCK, AR	09/15/96
BEAVER, BART D., EVERGREEN PARK, IL	09/11/96
BONO, JOHN E., SAN JOSE, CA	09/10/96
CARY, HUNSDON R., ARLINGTON, TX	09/15/96
CLUNES, LINDSAY C., CORVALLIS, OR	09/10/96
CONTI, ANTHONY N., MEDFORD, NY	09/09/96
DIBENEDETTO, MAURO V., MASSAPEQUA, NY	09/09/96
DRABINSKY, GERALD S., LOS ANGELES, CA	09/10/96
EARLY, GARY M., KIRKSVILLE, MO	09/11/96
FISHKIN, WILLIAM H., SAN FRANCISCO, CA	09/10/96
GARCIA, ROBERT WILLIAM, ODESSA, TX	09/15/96
GERVASI, CININA M., LONG BEACH, NY	09/09/96
HEALEY, CHRISTINE J., WINTHROP, MA	09/11/96
HITCHCOCK, PHILIP R., DUNSMUIR, CA	09/10/96
JAFAR, WIDAD J., WHITEHALL, PA	09/10/96
JUBERT, ANGELA K., NASHVILLE, TN	09/12/96
KASHANI, MORTAZA, HILLSIDE, IL	09/10/96
KETTERER, CYNTHIA L., PHILADELPHIA, PA	09/10/96
KRATT, THOMAS WILLIAM, NACOGDOCHES, TX	05/29/96
LAYMAN, KEVIN WAYNE, ALMA, AR	09/15/96
LINVILLE, MICHAEL R., SOMERVILLE, MA	09/11/96
MEISNER, JOHN T., BOGALUSA, LA	09/15/96
MELTON, TERRY L., OLD HICKORY, TN	09/12/96
MILLER, KEVIN DWAYNE, EUNICE, LA	09/15/96
MIRRIONE, JOHN J., WASHINGTON TWNSHP, NJ	09/09/96
MOINI, KIAN, FOUNTAIN VALLEY, CA	09/10/96
ODETTE, CAROLINE K., HOUSTON, TX	09/15/96
ONYEKAHA, EBERE N., MAPLEWOOD, NJ	09/09/96
RESNANSKY, ALEXANDER, RICHFIELD SPRNG, NY	09/09/96
REYNOLDS, BOB R., BAKERSFIELD, CA	09/10/96
SAUNDERS, RONALD W., SAN ANTONIO, TX	09/15/96
SIGH, EDWARD R., FT. STOCKTON, TX	09/15/96
SIMON, MICHELLE R., THOUSAND OAKS, CA	09/10/96
SLAVIN, TIMOTHY, FLORAL PARK, NY	09/09/96
TAYLOR, ROGER A., SAN MATEO, CA	09/10/96
VERBARO, DENNIS S., CHESTER, NJ	09/09/96
WATSON, PERRY F., GRANVILLE, OH	09/11/96
WILBERT, LEONARD BRIAN, VINITA TERRACE, MO	9/11/96

Dated: September 9, 1996.

William M. Libercci,

Director, Health Care Administrative Sanctions, Office of Enforcement and Compliance.

[FR Doc. 96–23568 Filed 9–13–96; 8:45 am]

BILLING CODE 4150–04–P

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, HHS.

ACTION: Notice.

The inventions listed below are owned by agencies 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 U.S. companies and may also be available for licensing.

ADDRESSES: Licensing information and copies of the U.S. patent applications and issued patents listed below may be obtained by contacting Ken Hemby at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7735 ext 265; fax: 301/402–0220). A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Expression of Early Lung Cancer Detection Market P31 in Neoplastic and Non-Neoplastic Respiratory Epithelium

JL Mulshine (NCI)
Filed 02 Oct 95
Serial No. 08/538,711

Lung cancer is the most frequent cause of cancer death in both males and females in the United States. Metastatic lung cancer is almost uniformly fatal. Methods for earlier detection of lung carcinoma may help increase survival rates by allowing earlier treatment. Recently, there have been efforts to detect antigens associated with lung carcinoma in the sputum of patients. The basis of this approach is the identification of early, potentially pre-neoplastic changes in cells shed from bronchial epithielium. This invention identifies P31 as a candidate for a lung carcinoma associated antigen which can be detected in sputum using immunological methods. This invention describes production and use of antibodies specific for P31 antigen which are highly correlated with patients having increased age and prolonged smoking history, and is selective for neoplastic tissue or tissue proximally associated with neoplasms. This invention further defines a method by which other candiate early lung cancer detection markers can be evaluated. Issuance of a patent on this invention is currently pending. (portfolio: Cancer—Diagnostics, in vitro, MAb based; Cancer—Research Reagents, MAb based)

Colon Mucosa Gene Having Down-Regulated Expression in Colon Adenomas and Adenocarcinomas

CW Schweinfest, TS Papas (NCI)
Filed 17 Apr 95
Serial No. 08/424,567 (FWC of 08/026,045)

Tumor suppressor genes that are down-regulated in colon adenomas and adenocarcinomas have been identified and isolated that may be valuable for the study and treatment of these disorders as well as for detecting and identifying other tumor suppressor genes. Colorectal cancer is a significant problem in the U.S., with 130,000 new cases per year and more than 65,000 deaths per year. Colorectal cancer is a multistep process involving the loss of function of so-called tumor suppressor genes as well as the activation of oncogenes. Studies in cell cultures have shown that the transfer of wild-type tumor suppressor genes to colon cancer cells lacking this gene suppresses tumorigenicity. cDNAs encoding an mRNA that is down-regulated in adenocarcinomas and adenomas of the colon have been isolated and cloned. The mRNA encodes a polypeptide of about 84,500 daltons. This down-regulated in adenoma (DRA) gene maps to chromosome 7, in which abnormalities have previously been linked to colorectal carcinomas. The polypeptide product of the cDNA may be used for studying the process of tumorigenesis and suppression. In addition, the DRA gene and/or polypeptide may be valuable as therapy for colon cancer or for staging colon tumors. Finally, this invention includes nucleotide probes for detecting and isolating other tumor suppressor genes. (portfolio: Cancer—Diagnostics, in vitro, DNA based; Gene-Based Therapies—Diagnostics)

Screening Assays for Compounds That Cause Apoptosis

CC Harris, XW Wang, JH Hoeijmakers (NCI)
Filed 19 Dec 94
Serial No. 08/359,316

This application discloses a method for screening compounds for those which have the property of inducing programmed cell death, or ''apoptosis'', and which therefore are candidates for treating cancers caused by a loss of ability to induce apoptosis. Apoptosis is a normal body mechanism for controlling the growth of cells; the loss of ability to induce apoptosis in cells with defective DNA replication is associated with the formation of certain cancers. One major pathway for monitoring cells for transformation and for inducing apoptosis in transformed cells involves the nuclear protein coded for by the p53 tumor suppressor gene. Mutations in the p53 gene have been linked to a number of human cancers. The screening assays are based on the knowledge that the p53 dependent apoptosis pathway involves the interaction of the p53 protein with XPB or XPD proteins of the disease *Xeroderma pigmentosum* (XP), or both. The application discloses in *vitro* diagnostic assays for two of the eight genetic forms of XP, specifically those related to defects in the B or D groups. The assays capitalize on the p53/XP protein interaction by using the ability of compounds with certain binding properties to induce apoptosis to detect the defects indicative of XP. The application also describes a peptide which interferes with the binding of p53 to XPB or XPD protein and may thus be capable of inducing apoptosis in cells susceptible to p53-mediated apoptosis. Issuance of a patent for this invention is currently pending. (portfolio: Cancer—Diagnostics, in vitro, DNA based; Cancer—Research Reagents, DNA based; Gene-Based Therapies—Diagnostics)

Cancer-Related Autocrine Motility Factor, Autotaxin

ML Stracke, LA Liotta, E Schiffmann, HC Krutzsch (NCI)
Filed 28 Nov 94
Serial No. 08/346,455

Many types of tumor cells have been found to produce proteins termed ''autocrine motility factors''. Cell motility plays an important role in the metastasis of tumor cells. The present novel motility factor autotaxin has been isolated and molecular cloned. The cDNA encoding the entire autotaxin protein contains 3251 base pairs, and has an mRNA size of approximately 3.3 kb. The full-length deduced amino acid sequence of autotaxin comprises a protein of 915 amino acids. Autotaxin was found to hydrolyze the type I phosphodiesterase substrate p-nitrophenyl thymidine-5′ monophosphate. Autotaxin stimulates both random and directed migration of human A2058 melanoma cells at picomolar concentrations.

The patent application includes claims to the autotaxin protein and cDNA and antibodies thereto. These materials may be useful in the development of cancer diagnostics and therapeutics. (portfolio: Cancer—Diagnostics, in vitro, MAb based; Cancer—Therapeutics, biological response modifers; Cancer—Therapeutics, immunoconjugates, MAb)

The IRS Family of Genes

MF White, XJ Sun, JH Pierce (NCI)
Filed 03 Oct 94
Serial No. 08/317,310

Insulin Receptor Substrate (IRS) is a polypeptide that has recently been shown to play a role in activation of downstream responses to insulin as well as a possible role in models of obesity or insulin resistance. This invention discloses IRS–2 polypeptide. An IRS–2 polypeptide, insulin receptor substrate-2, specifically binds the insulin receptor, the interleukin-4 receptor, interleukin-13 receptor, insulin-like growth factor, or IL–15 receptor. Disclosed is a method of diagnosis of an insulin-related disorder, such as diabetes, or immune related diseases relating in human or other mammals. A method of measuring the effect of treatment, using a cell or tissue sample the misexpresses the IRS–2 gene. The invention also discloses the manufacture of a transgenic animal that expresses a mutant form of the IRS–2 gene and is useful as a model for the study of insulin-related disorders or other disorders characterized by unwanted cell growth. (portfolio: Cancer—Diagnostics, in vitro, DNA based; Cancer—Diagnostics, in vivo, other; Cancer—Therapeutics, gene therapy, vectors; Cancer—Therapeutics, gene therapy, genes; Cancer—Therapeutics, biological response modifiers, growth factors; Gene-Based Therapies—Therapeutics, gene therapy, therapeutic genes)

Antigenic Matrix Metalloproteinase Peptides

LA Liotta, W Stetler-Stevenson, H Krutzch (NCI)
Serial No. 07/830,313 filed 26 Feb 90
U.S. Patent 5,372,809 issued 13 Dec 94

Inhibitory synthetic peptides have been made which incorporate various regions of the type IV collagenase purified from human melanoma cells. These peptides have been used to generate antibodies against specific domains within the type IV collagenase molecule. These peptides have also been shown to inhibit matrix metalloproteinases, and therefore may be useful in the treatment of matrix metalloproteinase-related disease states such as arthritis, tumor growth, invasion and metastasis, inappropriate angiogenesis, and certain inflammatory conditions, such as sarcoidosis. The peptides are suitable for administration by any means which provides ready transmission into the circulation, such as injection, infusion, inhalation, or buccal or sublingual administration. Opthalmologic administration via eye drops may also be possible. In addition to their inhibitory properties, the peptides may also be used to generate antibodies that recognize matrix metalloproteinases and antibodies that recognize collagenase IV specifically. Collagenase IV-specific antibodies are particularly advantageous, since the enzyme shares significant sequence

homology with other matrix metalloproteinases. Antibodies made with certain of the peptides are capable of distinguishing activated and non-activated forms of collagenase IV. Hence, the peptides have potential applications as both therapeutic and diagnostic agents. (portfolio: Cancer—Research Reagents; Cancer—Diagnostics, in vitro, DNA based)

Cell Matrix Receptor System And Use In Cancer Diagnosis And Management

LA Liotta, NC Rao, V Terranova (NCI)

Serial No. 06/481,934 filed 04 Apr 83

U.S. Patent No. 4,565,789 issued 21 Jan 86

A method of diagnosis and management of cancer, particularly breast cancer, is provided. The method involves interfering with the mechanism by which tumor cells adhere to the various membranes and tissues of the body, enabling replication, using cell receptors specific for the laminin molecule. The laminin molecule normally adheres to collagen IV of the membranes and tissues. The novel laminin molecule disclosed binds the cell receptor of the tumor cell because it has an affinity for the receptor but it does not have an affinity for collagen IV which is part of the membranes and tissues of the body.

Other applications include possible burn therapy through the promotion of adhesion and growth of epithelial cells, which form the covering of most internal organs and outer surface layers of skin.

Secondly, this invention provides a method for evaluating the effectiveness of chemotherapeutic agents designed to affect the receptor in cancer cells. The invention discloses a kit for detecting the presence of metastasizing cancer cells having this cell receptor. A method of separation of metastatic cancer cells expressing the cell receptor from a mixed population of cells is also provided.

Also provided is a method of detecting breast cancer using radiolabelled antibodies specific to the cell receptor. (Portfolio: Cancer—Diagnostics, in vitro, MAb based; Cancer—Diagnostics, in vivo, conjugate chemistry; Cancer—Diagnostics, in vitro, other; Cancer—Research Reagents, MAb based; Cancer—Miscellaneous; Cancer—Therapeutics, biological response modifiers, growth factors; Internal Medicine—Therapeutics, anti-inflammatory.)

Dated: August 21, 1996.

Maria C. Freire,

Director, Office of Technology Transfer.

[FR Doc. 96–23634 Filed 9–13–96; 8:45 am]

BILLING CODE 4140–01–M

National Institute on Deafness and Other Communication Disorders; Notice of Meeting of the Deafness and Other Communication Disorders Programs Advisory Committee

Pursuant to Pub. L. 92–463, notice is hereby given of a meeting of the Deafness and Other Communication Disorders Programs Advisory Committee.

Date: October 28, 1996.

Place: National Institutes of Health, 9000 Wisconsin Avenue, Building 31C, Conference Room 6, Bethesda, MD 20892.

Time: 8 am to 5 pm.

Purpose/Agenda: To hold discussions on Extramural Research programs.

Contact Person: Ralph F. Naunton, M.D., Director, Division of Human Communication, NIH/NIDCD, 6120 Executive Boulevard, MSC 7180, Bethesda, MD 20892–7180, 301–496–1804.

The entire meeting will be open to the public, with attendance limited to space available. A summary of the meeting and a roster of the members may be obtained from Dr. Naunton's office. For individuals who plan to attend and need special assistance such as sign language interpretation or other reasonable accommodation, please contact Dr. Naunton prior to the meeting.

(Catalog of Federal Domestic Assistance Program No. 93.173 Biological Research Related to Deafness and Communication Disorders)

Dated: September 6, 1996.

Margery G. Grubb,

Senior Committee Management Specialist, NIH.

[FR Doc. 96–23564 Filed 9–13–96; 8:45 am]

BILLING CODE 4140–01–M

Prospective Grant of Exclusive License: Immunotoxins With In-Vivo T Cell Suppressant Activity and Methods of Use and Immunotoxins

AGENCY: National Institutes of Health, Public Health Service, DHHS.

ACTION: Notice.

SUMMARY: This notice in accordance with 35 U.S.C. 209(c)(1) and 37 CFR 404.7(a)(1)(I) that the National Institutes of Health (NIH), Department of Health and Human Services, is contemplating the grant of an exclusive world-wide license to practice the inventions embodied in U.S. Patent Number 5,167,956, and entitled; ''Immunotoxins With In-Vivo T Cell Suppressant Activity and Methods of Use'', Patent Applications USSN 08/308,730, 60/008,104 and 60/015,459, and corresponding U.S. and foreign patent applications, all entitled; ''Immunotoxins With In-Vivo T Cell Suppressant Activity And Methods Of Use'' and U.S. Patent Number 5,208,021, and entitled; ''Immunotoxins'' and corresponding foreign patent applications to Sandoz Pharma Ltd., Basel, Switzerland. The patent rights for NIH inventors in these inventions have been assigned to the United States of America.

The prospective exclusive license will be royalty-bearing and will comply with the terms and conditions of 35 U.S.C. 209 and 37 CFR 404.7. The prospective exclusive license may be granted unless within sixty (60) days from the date of this published notice, NIH receives written evidence and argument that establishes that the grant of the license would not be consistent with the requirements of 35 U.S.C. 209 and 37 CFR 404.7.

The field of use for this prospective exclusive license may be limited to ''Induction of Tolerance to Transplanted Organs''. The field of use for this prospective exclusive license for U.S. Patent Number 5,208,021 will exclude, at a minimum, fields of use of, ''for therapeutic treatment of all cancers'' and ''for therapeutic treatment of all muscle diseases and disorders.''

A major goal in transplant immunobiology is the development of specific immunologic tolerance to organ transplants. This therapy holds the potential of freeing patients from the side effects of continuous pharmacologic immunosuppression and its attendant complications and costs. Dr. David Neville's laboratory at the National Institute for Mental Health, NIH has developed immunotoxins (IT) targeted to the pan-T cell marker CD3 (anti-CD3-IT) and demonstrated that it has a profound immunosuppressive effect on human and rhesus T cells in vivo. A collaboration with Dr. Stewart Knechtle's laboratory (University of Wisconsin, Madison) has shown that a 3-day administration of anti-CD3 IT in rhesus monkeys can transiently deplete T cells to <1% of initial val–es in both the blood and lymph node compartments. Donor lymphocytes were injected intrathymically in some animals. All monkeys with T cell depletion had prolonged allograft survival. Tolerance was confirmed by skin grafting in 5 of 6 long-surviving recipients (>150 days). No other drug or treatment regimen has come close to achieving these results. In a collaboration with Dr. Judith Thomas' laboratory (University of Alabama,