electronic, mechanical, or other technological collection techniques or other forms of information technology.

Direct Comments to OMB

Written comments and/or suggestions regarding the item(s) contained in this notice, especially regarding the estimated public burden and associated response time, should be directed to the: Office of Management and Budget, Office of Regulatory Affairs, New Executive Office Building, Room 10235, Washington, DC 20503, Attention: Desk Officer for NIH. To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact: Chris Thomsen, Chief, Cancer Information Service Branch, OCC, OD, NCI, Building 31, Room 10A16, 9000 Rockville Pike, Bethesda, MD 20892, or call non-toll-free number (301) 496–5583 ext. 239 or E-mail your request, including your address to: thomsenc@occ.nci.nih.gov

Comments Due Date

Comments regarding this information collection are best assured of having their full effect if received on or before June 16, 1997.

Dated: May 6, 1997.

Nancie L. Bliss,

OMB Project Clearance Liaison.

[FR Doc. 97–12782 Filed 5–14–97; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

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 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 (CDA) will be required to receive copies of the patent applications.

Agents That Bind To and Inhibit Human Cytochrome P450 2D6

HV Gelboin, FJ Gonzalez, KW Krausz (NCI)

OTT Ref. No. E–46–97/0 filed 22 Jan. 97

Licensing Contact: Leopold J. Luberecki, Jr., 301/496–7735 ext 223

This invention concerns monoclonal antibodies (MAbs) and other binding agents specific for the 2D6 subgroup of cytochrome P450 enzymes. The cytochrome P450s are the metabolic interface between xenobiotics and their metabolism in human and other species as well as for the metabolism of endobiotics. A large array of drugs, mutagens, carcinogens, pesticides, environmental chemicals, fatty acids, bile acids, and steroids are metabolized by individual forms of cytochrome P450. The invention involves the construction, isolation, and production of MAbs that specifically bind to human cytochrome P450 and 2D6 and that specifically inhibit the enzyme activity of human cytochrome P450 and lack specific binding to other human cytochrome P450s. These MAbs can be used to assess adverse reactions in patients to compounds and to identify populations that would exhibit different sensitivities to the therapeutic or toxic effects of compounds. Cytochrome P450 2D6, also known as debrisoquine hydroxylase, is the best characterized polymorphic P450 in the human population. Genetic differences in cytochrome P450 2D6 may be associated with increased risk of developing environmental and occupational based diseases. In addition, several drugs for treating cardiovascular and psychiatric disorders are known substrates of cytochrome P450 2D6, and these compounds could be more readily prescribed to normal metabolizers as assessed using the MAbs described in the invention. The list of compounds includes β-blockers and antiarrhythmics, psychoactive drugs including tricyclic antidepressants, and a variety of other commonly used drugs including codeine and dextromethorphan. A provisional patent application for this invention has been filed with the U.S. Patent and Trademark Office (PTO).

An adjunct technology to this invention that is available for licensing involves two inhibitory monoclonal antibodies to human P450 3A4 and human P450 2E1 that have been developed and filed as a separate patent application (U.S. Serial No. 08/599,808) with the PTO. The P450 3A4 has likely the largest number of known drug substrates than any other P450. The P450 2E1 also metabolizes some drugs and has high activity towards smaller molecules which are found in the environment and which may be toxic. (portfolios: Internal Medicine—Research Materials; Cancer—Research Materials, MAb based; Internal Medicine—Diagnostics; Cancer—Diagnostics, in vitro, MAb based)

Vanilloid Agonists for Desensitization of C-Fiber Sensory Afferent Neurons

PM Blumberg, T Biro, P Acs, G Acs (NCI)

Serial No. 60/030,999 filed 15 Nov 96

Licensing Contact: Leopold J. Luberecki, Jr., 301/496–7735 ext 223

Capsaicin has been proven to have therapeutic utility in the treatment of arthritis, pruritis, bladder hyperreflexia, allergic responses including rhinitis, and pain, including pain associated with cancer, peripheral neuropathies, and postherpetic neuralgia. For a number of these indications, applications have been found in veterinary as well as human medicine. Recent advances have identified capsaicin analogs with ultrapotency and with a more favorable spectrum of action, as well as subclasses of capsaicin receptors with different effects on desensitization. This invention describes a method of administering to a capsaicin-sensitive animal a therapeutically effective combination of capsaicin agonists and capsaicin-like antagonists which are more effective than the agonist alone at desensitizing a vanilloid responsive cell, and thereby improve the therapeutic index of the capsaicin agonist and overall treatment. Also described are pharmaceutical compounds which are effective in this method. (portfolios: Central Nervous System—Therapeutics, neurological, narcotics and analgesics; Internal Medicine—Therapeutics, other)

Sustained-Release Derivatives of Hydroxylated Analogs of Substituted 1-[2[bis(aryl)methoxy]-ethyl]-Piperazines and-Homopiperazines and Their Use As Noncompetitive Antagonists of Dopamine Reuptake Inhibitors

RB Rothman (NIDA), KC Rice (NIDDK), DB Lewis (NIDDK), D Matecka (NIDDK), JR Glowa (NIDDK)

Serial No. 60/030,248 filed 31 Oct 96

Licensing Contact: Leopold J. Luberecki, Jr., 301/496–7735 ext 223

Cocaine abuse and addiction is a major public health problem in the United States and several other

countries. The biomedical and psychosocial cost of cocaine abuse and addiction is considerable, and to date, there is no effective treatment for addiction. In an effort to develop an efficacious treatment for cocaine addiction, this invention describes sustained-release derivatives of hydroxylated analogs of substituted 1-[2bis(arly)methoxy]ethyl]-piperazines and-homopiperazines. These compounds bind to the dopamine transporter but do not inhibit dopamine reuptake, thereby providing a sustained increase in the level of extracellular dopamine and providing the drug abuser with some relief from drug craving due to dopamine deficiency, yet they simultaneously inhibit cocaine from further elevating the level of extracellular dopamine and increasing the probability of additional toxic side effects. These derivatives have been shown to produce moderate to long-acting attenuation of cocaine-induced activation of mesolimbic dopamine neurons in rhesus monkeys, resulting in decreased cocaine self-administration without concurrent effects on food response. The present invention provides these sustained-release derivatives, pharmaceutical compositions comprising the same, and a method of using such sustained release derivatives as a treatment for cocaine addiction. (portfolio: Central Nervous System—Therapeutics, psychotherapeutics, drug dependence)

Isolation and Use of Tissue Growth-Inducing FRZB Protein

FP Luyten (NIDR), M Moos Jr. (FDA), B Hoang (FDA), S Wang (FDA)
Serial No. 08/729,452 filed 11 Oct 96
Licensing Contact: Jaconda Wagner, 301/496–7735 ext 284

A secretable protein, named FRZB because of its homology to the *Drosophila* gene frizzled, has been isolated from cartilage. This protein appears to be involved in the formation of cartilage, bone, neural and muscle tissue. A pharmaceutical composition of this protein may be used as regenerative agent to treat degenerative disorders, (i.e., Huntingdon's, Alzheimer's, or spinal cord injuries), myodegenerative disorders (i.e., muscular dystrophy, myasthenia gravis, or myotonic myopathies) and osteodegenerative disorders (i.e., osteoporosis or osteoarthritis). In addition, FRZB directly interacts with the Wnt family of signaling molecules and inhibits their biological function *in vivo.* This provides the opportunity to selectively block Wnt driven diseases including neoplasias. (portfolios: Central Nervous System—Therapeutics, neurological, antiparkinsonian; Central Nervous System—Therapeutics, neurological, Alzheimer's; Central Nervous System—Therapeutics, neurologial, other; Internal Medicine—Therapeutics)

Novel Human Cancer Antigen of Tyrosinase-Related Proteins 1 and 2 and Genes Encoding Same

RF Wang, SA Rosenberg (NCI)
Serial No. 08/599,602 filed 06 Feb 96 and Serial No. 08/725,736 filed 04 Oct 96 (CIP)
Licensing Contact: Joseph Contrera, 301/496–7056 ext 244

Tumor infiltrating lymphocytes (TIL) from a melanoma patient showing regression were found to recognize epitopes from a protein designated gp75, now known as tyrosinase related protein 1 (TRP–1). The inventors found that the antigen recognized by the TIL was encoded by that gene but that was not the normal gene product. The TIL recognized a nine-amino acid peptide (ORF3P) which is the product of an alternative reading frame (ORF3). ORF3P cannot be lengthened or shortened without loss of antigenicity. The TRP–1 ORF3P antigen is only found in melanoma cells, melanocytes and normal retina. This technology was described in U.S. patent application 08/599,602 filed February 6, 1996.

The present invention is a CIP of 08/599,602 and was filed October 4, 1996. This CIP application contains a novel tumor antigen (TRP–2) which was recognized by CTL clones derived from TIL. However, TRP–2 was recognized by CTL clones which are capable of recognizing the ORF3P. A new antigenic peptide (TRP197–205) was identified from the TRP–2 product. The subject matter of both the parent and CIP applications were combined in a subsequent PCT application filed February 6, 1997.

The use of the methods described in the present invention could provide a form of cancer immunotherapy for melanoma. (portfolios: Cancer—Therapeutics, vaccines; Cancer—Diagnostics, in vitro, MAb based)

PFS25–28 Fusion as a Malaria Transmission Blocking Vaccine

DC Kaslow, MM Gozar (NIAID)
Serial No. 60/027,390 filed 30 Sept 96
Licensing Contact: Gloria Richmond, 301/496–7056 ext 268

Malaria is estimated to cause two to four million deaths per year, and 200 to 400 million people are infected annually with the deadliest of the protozoans that cause the disease, Plasmodium falciparum. The life cycle of the malarial parasite is very complex, involving stages that reside in both humans and mosquitoes. Vaccines that are able to inhibit the transmission of the disease at a variety of stages in the complex life cycle of the malarial parasite might provide an opportunity to effectively control and possible eradicate this disease. This invention relates to the generation of transmission-blocking antibodies to two sexual stage surface antigens, Pfs 25 and Pfs 28. Two issued patents cover the use of these antigens separately as transmission-blocking vaccines. The claims of the current invention relate to the production of fusion proteins between these two surface antigens that have increased potency as immunogens and ease of manufacture. (portfolios: Infectious Diseases—Vaccines, parasite)

Prostate Specific Antigen Oliog-Epitope Peptide

J Schlom, K Tsang, S Zaremba (NCI)
Serial No. 08/618,936 filed 20 Mar 96
Licensing Contact: Joseph Contrera, 301/496–7056 ext 244

Prostate Specific Antigen (PSA) is expressed in a majority of prostate cancers, and represents a potential target for immunotherapy. Previous studies have shown that two specific PSA peptides, PS1 and PS3, are capable of eliciting cytotoxic T-cell responses. The current invention embodies an oligopeptide, PSA–OP, which is comprised of the sequence for peptides PS1 and PS3. PS1 and PS3 are antigenic epitopes of PSA and are joined by a peptide linker sequence to form PSA–OP. PSA–OP has been shown, in vitro, to be effective in eliciting a cytotoxic T-cell response. This novel peptide, therefore, may be used in the development of vaccines for use in the prevention and treatment of prostate cancer. (portfolio: Cancer—Therapeutics)

Immortal Human Prostate Epithelial Cell Cultures and Their Applications in the Research and Therapy of Prostate Cancer

SL Topalian, WM Linehan, RK Bright, CD Vocke (NCI)
OTT Reference No. E–053–96/0 (USSN 60/011,042 filed 02 Feb 96) and OTT Reference No. E–017–97/0 (CIP of E–053–96/0)
Licensing Contact: Joseph Contrera, 301/496–7056 ext 244

The invention describes the further characterization of single cell clones derived from the prostate tumor cell lines disclosed in the earlier application (E–053–96/0). The isolation and characterization of long-term human prostatic epithelial cell cultures from

primary adenocarcinomas of the prostate is significant in that efforts to establish long-term cultures of cells of this type have been exceptionally difficult.

The present invention describes the characterization of single cell clones derived from the prostate tumor cell lines disclosed in the earlier application. These new clones exhibit traits which may indicate their usefulness as an in vitro model of human prostate cancer. The single cell clones are paired normal and tumor cell clones where the latter exhibit allelic loss of heterozygosity (LOH) indicating the presence of unique genetic deletions. This loss may suggest that these cells express unique proteins or antigens which might be of tremendous value in prostate cancer research. The subject matter of both the parent and CIP applications were combined in a subsequent PCT application filed January 30, 1997.

Possible uses of these cells include testing various anti-cancer agents and subtraction studies for identification of gene deletions. These lines could establish a new basis for possible cancer vaccines and also be used to develop monoclonal antibodies against specific prostate cancer antigens. (portfolios: Cancer—Therapeutics, vaccines; Cancer—Therapeutics, immunomodulators and immunostimulants)

Macrophage Migration Inhibitory Factor (MIF)

Graeme J. Wistow (NEI)
Serial No. 08/202,486 filed 28 Feb 94 (allowed); DIV of U.S. Patent 5,328,990 issued 12 Jul 94
Licensing Contact: Jaconda Wagner, 301/496–7735 ext 284

The protein known as macrophage migration inhibitory factor (MIF) was one of the first cytokines to be discovered. Thirty-years ago it was described as a T-cell-derived factor that inhibited the random migration of macrophages in vitro. Today, MIF is known to be a mediator of the function of macrophages in host defense and its expression correlates with delayed hypersensitivity and cellular immunity. It plays an important role in the inflammatory response and is associated with cell differentiation. As with other lymphokines, MIF could have therapeutic values in stimulating the immune system and other cells. Hardly abundant from other sources, the high concentration of the protein that has been found in the eye lens could be a useful source for research. The present invention provides the DNA that encodes MIF. A related invention provides a method for isolating MIF from the ocular lens. (portfolio: Ophthalmology—Therapeutics; Internal Medicine—Therapeutics, anti-inflammatory)

Dated: April 28, 1997.

Barbara M. McGarey,

Deputy Director, Office of Technology Transfer.

[FR Doc. 97–12783 Filed 5–14–97; 8:45 am]

BILLING CODE 4140–01–W

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; Notice of Closed Meeting

Pursuant to the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of a meeting of the President's Cancer Panel. This meeting will be closed in accordance with the provisions of section 552b(c)(9)(B), Title 5, U.S.C., for discussion and preparation of the Annual Report of the Chair to the President for 1996. These discussions could disclose information, the premature disclosure of which would be likely to significantly frustrate implementation of proposed action the Panel may plan to take.

Linda Quick-Cameron, Committee Management Officer, National Cancer Institute, Executive Plaza North, Room 630E, 6130 Executive Blvd., MSC 7410, Bethesda, MD 20892–7410 (301/496–5708) will provide a summary of the meeting and the roster of committee members upon request. Other information pertaining to the meeting may be obtained from the contact person indicated below.

Committee Name: President's Cancer Panel.

Date: May 22, 1997.

Place: La Guardia Marriott, 102–05 Ditmars Boulevard, E. Elmhurst, New York 11369.

Closed: 8:30 a.m. to 5:30 p.m.

Agenda: Finalization of the Annual Report of the Chairman to the President.

Contact Person: Maureen O. Wilson, Ph.D., Executive Secretary, National Cancer Institute, Building 31, Room 4A48, Bethesda, MD 20892–2473, Telephone: (301) 496–1148.

This notice is being published less than 15 days prior to the meeting due to the urgent need to proceed with the finalization of the Annual Report of the Chairman to the President.

Dated: May 8, 1997.

LaVerne Y. Stringfield,

Committee Management Officer, NIH.

[FR Doc. 97–12781 Filed 5–14–97; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Arthritis and Musculoskeletal and Skin Diseases; Notice of Meeting; National Arthritis and Musculoskeletal and Skin Diseases Advisory Council

Pursuant to Pub. L. 92–463, notice is hereby given of a meeting of the National Arthritis and Musculoskeletal and Skin Diseases Advisory Council to provide advice to the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS) on June 5, 1997, in Conference Room 6, Building 31, National Institutes of Health, Bethesda, Maryland.

The meeting will be open to the public June 5 from 8:30 a.m. to 12:00 p.m. to discuss administrative details relating to Council business and special reports. Attendance by the public will be limited to space available.

The meeting of the Advisory Council will be closed to the public on June 5 from 1:00 p.m. to adjournment in accordance with provisions set forth in secs. 552b(c)(4) and 552b(c)(6), Title 5 U.S.C. and sec. 10(d) of Pub. L. 92–463, for the review, discussion and evaluation of individual grant applications. These deliberations could reveal confidential trade secrets or commercial property, such as patentable material, and personal information concerning individuals associated with the applications, disclosure of which would constitute a clearly unwarranted invasion of personal property.

Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should contact Dr. Steven Hausman, Executive Secretary, National Arthritis and Musculoskeletal and Skin Diseases Advisory Council, NIAMS, Natcher Building, Room 5AS–13, Bethesda, Maryland 20892 (301) 594–2463.

A summary of the meeting and roster of the members may be obtained from the Extramural Programs Office, NIAMS, Natcher Building, Room 5AS–13, National Institutes of Health, Bethesda, Maryland 20892 (301) 594–2463.

(Catalog of Federal Domestic Assistance Program No. 93.846, Arthritis, Bone and Skin Diseases, National Institutes of Health)

Dated: May 9, 1997.

LaVerne Y. Stringfield,

Committee Management Officer, NIH.

[FR Doc. 97–12778 Filed 5–14–97; 8:45 am]

BILLING CODE 4140–01–M