Subject city, state	Effective date
GREENVILLE, AL	
WAGHER, LISA KATHLEEN ... WILLIAMS, AZ	05/20/2003
WASSIF, ANTHONY A ..... RESEDSA, CA	05/20/2003
WEINBERG, ROBERT PAUL .. BOSTON, MA	05/20/2003
WEINBERG, MARC ..... COLUMBIA, MD	05/20/2003
WHITE, MARGARET ..... ASHEVILLE, NC	05/20/2003
WILSON, JOHN STROTHER ... MONTGOMERY, AL	05/20/2003
WILTFANG, CHRIS CHARLES ST SIMONS ISLAND, GA	05/20/2003
WITTRUP, LADONNA ALBERTINA ..... GREELEY, CO	05/20/2003
FEDERAL/STATE EXCLUSION/ SUSPENSION	
NATHAN, SYLVESTER ARTHUR ..... WOODRIDGE, IL	05/20/2003
NATOLI, DENNIS ..... RIVER EDGE, NJ	05/20/2003
SMOOTS, OLIVIA L ..... SACRAMENTO, CA	05/20/2003
OWNED/CONTROLLED BY CONVICTED ENTITIES	
BLOSSOM HILL DENTAL CARE ..... SAN JOSE, CA	05/20/2003
CALIXTO GARCIA MEDICAL CTR INC ..... MIAMI, FL	05/20/2003
DIAMOND DENTAL SERVICES, LLP ..... POCATELLO, ID	05/20/2003
FAMILY FIRST HEALTHCARE, INC ..... WICHITA, KS	05/20/2003
HOI LEE D D S ..... RENTON, WA	05/20/2003
NAT'L MEDICAL EQUIP & SUPPLIES ..... PLANTATION, FL	05/20/2003
PRINCIPLE MEDICAL MGMT, INC ..... HOLLYWOOD, CA	05/20/2003
ROLYJANE HOME CARE, INC MIAMI, FL	05/20/2003
SOLOMON'S INVALID COACH, INC ..... NEWARK, NJ	05/20/2003
UNION PHARMACY ..... MIAMI, FL	05/20/2003
DEFAULT ON HEAL LOAN	
BARBATO, BEVERLY V ..... SALEM, WV	04/03/2003
BILYEU, STUART WILLIAM .... ANN ARBOR, MI	05/20/2003
CROW, JOE A ..... ROCKY FORD, CO	05/20/2003
DIEDE, CLYDE A ..... WINNER, SD	05/20/2003
FLEMING, JAMES E JR ..... E CLEVELAND, OH	05/20/2003
LESINSKI, MARY CATHERINE CLEVELAND, OH	04/14/2003
MARTINEZ, JOSE R ..... MINEOLA, TX	05/20/2003
OWNERS OF EXCLUDED ENTITIES	
PARKER, CHARLIE A ..... MINNEAPOLIS, MN	05/20/2003
PARKER, ROBERTA CATO .... MINNEAPOLIS, MN	05/20/2003
CIVIL MONETARY PENALTY	
MATTHYS, JERRY A ..... SHAWNEE MISSION, KS	10/01/2002

Dated: May 1, 2003.

Katherine B. Petrowski,

Director, Exclusions Staff Office of Inspector General.

[FR Doc. 03–11681 Filed 5–14–03; 8:45 am]

BILLING CODE 4150–04–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 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 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.

Triggering Receptors Expressed in Myeloid Cells (TREM) Like Transcript (TLT–1), A Novel Inhibitory Receptor of Platelets and Uses Therefore

Daniel W. McVicar (NCI), A. Valence Washington (NCI), Laura Quigley (NCI)

DHHS Reference No. E–097–2003/0 filed March 16, 2003

Licensing Contact: Jeff Walenta; 301/435–4633; *walentaj@od.nih.gov.*

The human immune response involves a complex series of molecular interactions to produce a beneficial response to foreign invasion within the body. These molecular interactions orchestrate the specific responses of innate and adaptive immunity. When these interactions break down, immune related disorders such as cancer and sepsis arise.

This invention describes an advance in understanding the regulation of the immune response. Triggering Receptors in Myeloid Cells (TREM) recently were discovered to modulate innate and adaptive immunity. Specifically, TREM1 amplifies the response to sepsis in innate immunity by activating neutrophils and other leukocytes; and TREM2 potentiates dendritic cell maturation in adaptive immunity. This invention describes a new inhibitory TREM like Transcript, TLT–1.

TLT–1 is the first inhibitory receptor discovered to reside within the TREM gene locus. This discovery implies the receptor has an important regulatory role in both innate and adaptive immunity. Structurally, TLT–1 also possesses inhibitory domains that indicate this regulatory function. TLT–1 is highly expressed in peripheral blood platelets and may modulate many other types of myeloid cells. Potential therapeutic implications are for immune disorders, cancer, septic shock, infectious disease, stroke, heart disease, myocardial infarction, vascular disorders, and other platelet-associated disorders.

17–AAG Treatment of Diseases Sensitive to c-KIT Down Regulation

Gerard Fumo and Len Neckers (NCI)

DHHS Reference No. E–256–2002 filed 22 Oct 2002

Licensing Contact: George Pipia; 301/435–5560; *pipiag@od.nih.gov.*

This invention describes the use of 17-allylamino-17-demethoxygeldanamycin (17-AAG), a derivative of geldanamycin, which inhibits mutated KIT protein kinase activity (the product of proto-oncogene c-KIT). This kinase has been identified as the protein responsible for transformation of certain human cell types into pathologic cells. The invention is predicated on the discovery of a new method of inhibiting the activity of a mutated, constitutively active form of the tyrosine kinase, KIT. The method involves the administration of 17-AAG to a cell expressing the

mutated KIT protein, whereby the activity level of KIT in the cell is reduced. The invention may prove to be useful for treating diseases such as mastocytosis, gastrointestinal stromal tumors (GIST), mast cell leukemia, myelogenenous leukemia, and testicular cancer, all of which are associated with mutations in the c-KIT proto-oncogene.

Recombinant Vaccinia Viruses Expressing IL–15 and Methods of Using the Same

Liyanage Perera et al. (NCI)

Serial No. 60/433,703 filed 16 Dec 2002

Licensing Contact: Jonathan Dixon; 301/435–5559; *dixonj@od.nih.gov.*

Vaccinia-based vaccines have a proven record of being effective vaccines in humans as well as in animals. However, accumulating evidence reveals the need for technology to improve the immune responses such vaccines generate.

The present invention discloses recombinant vaccinia viruses capable of expressing interleukin 15 (IL–15), and methods for modulating immune responses using such viruses. This invention shows that by inserting the human IL–15 gene into the vaccinia genome, more effective vaccines can be generated against infectious agents and cancer. Currently, IL–2 has been approved by the FDA for use in the treatment of patients with metastatic renal cell carcinoma or with metastatic melanoma. It has been used as a component of cancer vaccines and in various approaches for the treatment of AIDS. However, administration of IL–2 is associated with activation-induced cell death (AICD), and may lead to death of T-cells that recognize the antigens expressed in the tumor cells. Thus, IL–15 may be a superior agent in the treatment of cancer, or as a component of a vaccine directed towards cancer or infectious agents. Co-delivery of IL–15 with antigens during the immunization process, according to the current invention, leads to induction of CD8+ memory T cells that proliferate more effectively in vivo and persist much longer in the immunized individual in addition to enhancing the levels and persistence of antigen specific antibodies thus providing substantially longer lasting cellular and humoral immunity.

This invention has the potential to be used in a variety of ways, including: (i) An improved, more efficacious vaccine candidate for smallpox, (ii) for incorporation into existing vaccinia based vaccines to enhance and confer superior long lasting immune response to viral and cancer antigens, or (iii) as a valuable source material for IL–15 production, especially should IL–15 be proven as an alternate of more efficacious cytokine than IL–2.

This research has been described, in part, in Proc. Natl. Acad. Sci USA 2003 Mar 18; 100(6):3392–3397.

DNA-Binding Polyamide Drug Conjugates

Zoltan Szekely, Humcha K. Hariprakasha, Marek W. Cholody, Christopher J. Michejda (NCI)

DHHS Reference No. E–060–2002/2–PCT–01 filed 27 Feb 2003 (PCT/US03/06006)

Licensing Contact: George Pipia; 301/435–5560; *pipiag@od.nih.gov.*

Many current anti-cancer drugs have the DNA of cancer cells as their principal target. However, in most instances, the drugs are not selective and are plagued by toxicities, which are frequently dose limiting. The present invention seeks to enhance anti-tumor selectivity and decrease unspecific toxicity. It has been known that various polyamides can target the minor groove of DNA, and rules have been devised to ascertain the sequence-reading properties of the component residues of the polyamide chain. The present invention utilizes sequence-selective polyamide technology together with groups that modify DNA, either by sequence-selective alkylation or strand cleavage. The DNA-modifying moieties that are used for this purpose are novel derivatives based on the cyclopropylbenzindole (CBI) core structure. These compounds alkylate the DNA only when bound into the minor groove, and they provide some DNA-sequence recognizing capability of their own. The DNA-modifying agents are either embedded in the polyamide chain as components of the chain or are located at the termini. These compounds are highly toxic to cancer cells that over-express a targeted DNA sequence (*e.g.* the c-Myc oncogene promoter sequence) and are much less toxic to non-cancerous tissue. The compounds of the present invention represent a novel method for targeting DNA of cancer cells.

SH2 Domain Binding Inhibitors

Terrence R. Burke, Jr., et al. (NCI)

DHHS Reference No. E–262–2000/1 filed 28 Jun 2002

Licensing Contact: George Pipia; 301/435–5560; *pipia@od.nih.gov.*

Signal transduction processes underlie the transfer of extracellular information to the interior of the cell and ultimately to the nucleus. A variety of signal transduction processes are critical for normal cellular homeostasis, with protein-tyrosine kinases (PTKs) playing central roles in many of these pathways. Examples of such PTKs include the PDGF receptor, the FGF receptor, the HGF receptor, members of the EGF receptor family, such as the EGF receptor, erb-B2, erb-B3 and erb-B4, the src kinase family, Fak kinase and the Jak kinase family. Protein-tyrosine phosphorylation that results from the action of PTKs can modulate the activity of certain target enzymes as well as facilitate the formation of specific multi-protein signaling complexes through the actions of homologous protein modules termed Src homology 2 (SH2) domains, which recognize specific phosphotyrosyl containging sequences. A malfunction in this system through tyrosine kinase overexpression and/or deregulation can be manifested by various oncogenic and hyperproliferative disorders, including cancers, inflammation, autoimmune disease, hyperproliferative skin disorders, psoriasis and allergy/asthma, etc. The disclosed compounds, *e.g.* peptides, preferably, macrocyclic peptides, are Grb2 SH2 domain signaling antagonists with enhanced binding affinity. The claims of the current application are directed to compositions of matter and methods of use which provide for the diagnosis, testing and treatment of the aforementioned disease states.

Dated: May 7, 2003.

Steven M. Ferguson,

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

[FR Doc. 03–12102 Filed 5–14–03; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; 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