TABLE 1.—ESTIMATED ANNUAL REPORTING BURDEN[1]

21 CFR Section	No. of Respondents	Annual Frequency per Response	Total Annual Responses	Hours per Response	Total Hours
100.2(d)	1	1	1	10	10

[1] There are no capital costs or operating and maintenance costs associated with this collection of information.

The reporting burden for § 100.2(d) is insignificant because enforcement notifications are seldom used by States. During the last 3 years, FDA has not received any enforcement notifications. Since the enactment of section 403A(b) of the act (21 U.S.C. 343–1(b)) as part of the Nutrition Labeling and Education Act of 1990, FDA has received only a few enforcement notifications. Although FDA believes that the burden will be insignificant, it believes these information collection provisions should be extended to provide for the potential future need of a State government to submit enforcement notifications informing FDA when it intends to take enforcement action under the act against a particular food located in the State.

Dated: September 7, 2005.

Jeffrey Shuren,

Assistant Commissioner for Policy.

[FR Doc. 05–18223 Filed 9–13–05; 8:45 am]

BILLING CODE 4160–01–S

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.

Soluble Fragments of the IGF1R Ectodomain

Dimiter S. Dimitrov et al. (NCI)
HHS Reference No. E–144–2005/0—Research Tool
Licensing Contact: Michelle A. Booden; 301/451–7337; *boodenm@mail.nih.gov.*

The type 1 insulin-like growth factor (IGF) receptor (IGF1R) is over-expressed by many tumors and mediates proliferation, motility, and protection from apoptosis. Agents that inhibit IGF1R expression or function can potentially block tumor growth and metastasis.

The present invention relates to the identification of soluble fragments of the IGF1R ectodomain, where these fragments bind IGF-I, IGF-II, or the various other ligands of IFG1R. The identified fragment may be useful for identifying agents that block IGF1R and may act as a strong dominant negative inhibitor of tumor growth by blocking the IGF1R pathway. The invention also encompasses other IGF1R fragments or derivatives of the original fragments, methods of identifying IGF1R fragments or other similar fragments in the IGF1R ectodomain, methods of using said fragments to block binding of ligands, and methods of producing antibodies against the IGF1R fragments.

The technology is available for licensing under a biological material license. In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Polymer-Linked Pseudomonas Exotoxin Immunotoxin

Ira Pastan (NCI) et al.
U.S. Provisional Application No. 60/636,007 filed 12 Dec 2004 (HHS Reference No. E–121–2005/0-US–01)
Licensing Contact: Jesse Kindra; 301/435–5559; *kindraj@mail.nih.gov.*

Molecules based on monoclonal antibodies hold the promise of highly selective therapeutics. However, their efficacy can be limited by poor tissue penetration, rapid renal clearance and an immune response to the antibody. The present technology provides an immunotoxin that is modified to overcome such limitations.

The technology relates to polymer-conjugated immunotoxins targeted to the mesothelin tumor cell antigen. These polymer-immunotoxin conjugates possess an enhanced therapeutic index and may provide improved methods of treating tumors and cancers expressing the mesothelin antigen.

Tumor Suppressor Gene Caliban

Mark A. Mortin et al. (NICHD)
U.S. Provisional Application filed 06 Jun 2005 (DHHS Reference No. E–118–2005/0-US–01)
Licensing Contact: Jesse S. Kindra; 301/435–5559; *kindraj@mail.nih.gov.*

This invention relates to the identification of a tumor suppressor gene named Caliban from Drosophila melanogaster. The inventors have demonstrated that Caliban is very similar to the corresponding human gene and they have shown that the human gene is inactive in human lung cancer cells but active in normal lung cells. For the first time, it has been shown that when full length Caliban is expressed in human lung cancer cells they lose many of their tumorigenic properties. Hence, using gene therapy to replace the inactive gene with full length Caliban may treat cancer. Details of this were published in Bi *et al.*, ''Drosophila caliban, a nuclear export mediator, can function as a tumor suppressor in human lung cancer cells,'' Oncogene advance online publication, August 15, 2005; doi:10.1038/sj.onc.1208962.

This invention also provides a biomarker assay that can be used to determine if the fly or human tumor suppressor Caliban gene product is functioning in cells. This assay uses a peptide from the fly gene Prospero, named HDA, which when fused to a reporter such as green fluorescent protein, is exported from the nucleus when Caliban is working.

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

SIPA–1 Gene and SIPA–1 Inhibitor for the Treatment, Prevention and Diagnosis of Cancer

Kent Hunter et al. (NCI)

U.S. Provisional Application No. 60/649,365 filed 02 Feb 2005 (HHS Reference No. E–082–2005/0-US–01); U.S. Provisional Application No. 60/657,943 filed 02 Mar 2005 (HHS Reference No. E–082–2005/1–US–01); U.S. Provisional Application No. 60/695,024 filed 29 Jun 2005 (HHS Reference No. E–216–2005/0–US–01)

Licensing Contact: Mojdeh Bahar; 301/435–2950; *baharm@mail.nih.gov.*

The technology relates to methods and compositions of matter used to identify and treat metastatic cancer. Using genetics, the inventors identified the mouse Sipa-1 gene as a possible metastasis modifying gene. Further analyses revealed that Sipa-1 expression levels correlate with metastasis. The inventors developed compounds that modulate Sipa-1 expression and reduce metastasis in animal models. The inventors also identified single nucleotide polymorphisms (SNPs) present in the mouse Sipa-1 gene that, if also present in humans, could serve as the basis for diagnosing cancer and metastasis.

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

Diagnostic Tool for Diagnosing Benign Versus Malignant Thyroid Lesions

Steven K. Libutti et al. (NCI)

PCT Patent Application No. PCT/US05/12289 filed 11 Apr 2005 (HHS Reference No. E–124–2004/2–PCT–01)

Licensing Contact: Mojdeh Bahar; 301/435–2950; *baharm@mail.nih.gov*.

The present invention relates to methods for the diagnosis and staging of thyroid cancer. The invention employs analysis of gene expression using microarrays or quantitative RT-PCR to distinguish between malignant and benign tumors. Primer and probe sequences are described that represent a six gene or ten gene model for diagnosing benign and malignant thyroid cancer. Analysis of the expression of these genes in thyroid lesions taken from patients could be used for molecular classification of the lesions. As analysis of thyroid lesions by traditional means, such as fine needle biopsy with cytologic examination, can result in indeterminate results, the current invention may provide a superior method for molecular diagnoses of thyroid cancer.

This research is described, in part, in Mazzanti et al., ''Using gene expression profiling to differentiate benign versus malignant thyroid tumors,'' Cancer Res. 2004 Apr 15 64(8):2898–2903.

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

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

Liyanage Perera et al. (NCI)

U.S. Provisional Application No. 60/433,703 filed 16 Dec 2002 (HHS Reference No. E–243–2002/0–US–01); PCT Application No. PCT/US03/39967 filed 15 Dec 2003, which published as WO 2004/058278A1 on 15 Jul 2004 (HHS Reference No. E–243–2002/1–PCT–01); U.S. Patent Application filed 14 Jun 2005 (HHS Reference No. E–243–2002/1–US–02)

Licensing Contact: John Stansberry; 301/435–5236; *stansbej@mail.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-expression of IL–15 with antigens during the immunization process, according to the current invention, leads to induction of CD8+ memory T cells with higher avidity 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 SK Oh et al., ''Coadministration of HIV vaccine vectors with vaccinia viruses expressing IL–15 but not IL–2 induces long-lasting cellular immunity,'' Proc. Natl. Acad. Sci USA 2003 Mar 18;100(6):3392–3397, online publication 10.1073/pnas.0630592100.

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

Amelioration of Inflammatory Arthritis Targeting the Pre-ligand Assembly Domain (PLAD) of Tumor Necrosis Factor Receptors

Michael J. Lenardo et al. (NIAID)

U.S. Provisional Application No. 60/694,015 filed 24 Jun 2005 (HHS Reference No. E–095–2000/2–US–01)

Licensing Contact: Mojdeh Bahar; 301/435–2950; *baharm@mail.nih.gov*.

The invention relates to compositions of matter and methods for treating arthritis by modulating Tumor Necrosis Factor Alpha (TNFalpha) signaling. TNFalpha plays a key role in the pathogenesis of numerous diseases including rheumatoid and septic arthritis, and other autoimmune and inflammatory diseases. TNFalpha mediates its effects through receptors that contain a Pre-ligand Assembly Domain (PLAD). The inventors have discovered compounds that interfere with PLAD can block the effects of TNFalpha in vitro. Treatment of mice with these compounds in vivo ameliorated disease in several models of arthritis. Therefore, the compositions and methods of the current invention may lead to novel arthritis treatments.

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

Disubstituted Levendustin A Analogs (Including Adaphostin) and Pharmaceutical Compositions Comprising the Analogs

Venkatacha L. Narayanan et al. (NCI)
U.S. Patent Application No. 09/623,000 filed 25 Aug 2000 (DHHS Reference No. E–013–1998/0–US–07)
Licensing Contact: John Stansberry; (301) 435–5236; *stansbej@mail.nih.gov*.

Chronic myelogenous leukemia (CML) is almost universally associated with a translocation that juxtaposes the Bcr and Abl genes. Because the resulting kinase, p210 $^{Brc/Abl}$, is found exclusively in malignant hematopoietic cells there has been considerable interest in identifying inhibitors of this enzyme. Adaphostin induces cytotoxicity in human leukemia cells by down-regulating p210 $^{Bcr/Abl}$, inducing DNA damage and initiating apoptosis. Adaphostin exhibits selectivity for CML myeloid progenitors in vitro and retained its catholicity when cytotoxicity mesylate-resistant K562 cells were examined. Adaphostin may kill a wide range of human leukemia cells and may be effective against other cancer types. The present invention provides pharmaceutical compositions comprising effective amounts of adaphostin. The compound and composition of the present invention may be used for treating human leukemia and other proliferative diseases.

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

Heterologous Boosting Immunizations

Ronald S. Chamberlain et al. (NCI)
U.S. Patent Application No. 09/171,086 filed 22 Jan 1999 (HHS Reference No. E–087–1996/0-US–04); U.S. Patent Application No. 09/838,987 filed 20 Apr 2001 (HHS Reference No. E–087–1996/0–US–05); U.S. Patent Application No. 11/007,115 filed 08 Dec 2004 (HHS Reference No. E–087–1996/0–US–06); PCT Application No. PCT/US97/06632 filed 21 Apr 1997, which published as WO 97/39771 on 30 Oct 1997 (HHS Reference No. E–087–1996/0–PCT–02); and Canadian Patent Application Serial No. 2,252,406 (HHS Reference No. E–087–1996/0–CA–03)
Licensing Contact: Michelle A. Booden; 301/451–7337; *boodenm@mail.nih.gov*.

The identification of tumor-associated antigens and the cloning of DNA sequences encoding them have enabled the development of anticancer vaccines. Such vaccines target tumors by stimulating an immune response against the antigens. One method of vaccination involves the delivery of antigen-encoding DNA sequences, and a number of recombinant vectors have been used for this purpose. To optimize the efficacy of recombinant vaccines, Dr. Steve Rosenberg and colleagues at the NCI have developed treatment regimens that use two different vectors (*i.e.*, heterologous boosting).

The present invention describes the method of heterologous boosting immunizations, which in essence is the use of a priming vaccination and a boosting vaccination using two different recombinant vectors that contain a similar or different tumor associated antigen (TAA). The use of different recombinant vectors unexpectedly increases and maintains the immune response to most tumor-associated antigens included in the vectors. The claims are directed, but not limited to, various recombinant viral vectors: poxvirus, vaccine, adenovirus, etc. Additional embodiments and claims are directed, but not limited to, melanoma tumor antigens such as Mart1, gp100, or Hep B surface antigen. These tumor antigen expressing recombinant vectors are coupled with distinctly different recombinant vectors, which express various cytokines and co-stimulatory and accessory molecules such as B7–1, B7–2, ICAM–1, etc. This therapeutic intervention could be directed toward multiple human carcinomas but, with respect to this technology, has been customized as a therapeutic intervention for melanoma.

This technology is available under an exclusive or non-exclusive license. In addition to licensing, the technology is available for further development through collaborative research opportunities with the inventors.

Dated: September 2, 2005.

Steven M. Ferguson,

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

[FR Doc. 05–18168 Filed 9–13–05; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Center for Complementary & Alternative Medicine; 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 Complementary and Alternative Medicine Special Emphasis Panel, Clinical Science.

Date: October 20–21, 2005.

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

Agenda: To review and evaluate grant applications.

Place: Bethesda Park Hotel, 8400 Wisconsin Avenue, Bethesda, MD 20814.

Contact Person: Jeanette M. Hosseini, Scientific Review Administrator, National Center For Complementary and Alternative Medicine, 6707 Democracy Blvd., Suite 401, Bethesda, MD 20892, 301–594–9096.

Dated: September 6, 2005.

Anthony M. Coelho, Jr.,

Acting Director, Office of Federal Advisory Committee Policy.

[FR Doc. 05–18171 Filed 9–13–05; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Library of Medicine; Notice of Meeting

Pursuant to section 10(a) of the Federal Advisory Committee Act, as amended (5 U.S.C. appendix 2), notice is hereby given of the tenth and final meeting of the Commission on Systemic Interoperability.

The meeting will be open to the public, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the meeting.

The mission of the Commission on Systemic Interoperability is to submit a report to the Secretary of Health and Human Services and to Congress on a comprehensive strategy for the adoption and implementation of health care information technology standards that includes a timeline and prioritization for such adoption and implementation. In developing that strategy, the