HRSA Reports Clearance Officer on (301) 443–1129.

Comments are invited on: (a) Whether the proposed collection of information is necessary for the proper performance of the functions of the agency, including whether the information shall have practical utility; (b) the accuracy of the agency's estimate of the burden of the proposed collection of information; (c) ways to enhance the quality, utility, and clarity of the information to be collected; and (d) ways to minimize the burden of the collection of information on respondents, including through the use of automated collection techniques or other forms of information technology.

Proposed Project: Ryan White Comprehensive AIDS Resources Emergency (CARE) Act: CARE Act Data Report (CADR) Form: (OMB No. 0915–0253)—Revision.

The CARE Act Data Report (CADR) form was created in 1999 by HRSA's HIV/AIDS Bureau. It is designed to collect information from grantees and their subcontracted service providers, who are funded under Titles I, II, III, and IV of the Ryan White CARE Act of 1990, as amended by the Ryan White CARE Act Amendments of 1996 and 2000 (codified under Title XXVI of the Public Health Services Act). All Titles of the CARE Act specify HRSA's responsibilities in the administration of grant funds, the allocation of funds, the evaluation of programs for the population served, and the improvement of the quantity and quality of care. Accurate records of the providers receiving CARE Act funding, the services provided, and the clients served continue to be critical to the implementation of the legislation and thus are necessary for HRSA to fulfill its responsibilities. CARE Act grantees are required to report aggregate data to HRSA annually. The CADR form is used by grantees and their subcontracted service providers to report data on seven different areas: service provider information, client information, counseling and testing services, medical services, and other services provided/clients served, demographic information, and the Health Insurance Program. The primary purposes of the CADR are to: (1) Characterize the organizations from which clients receive services; (2) provide information on the number and characteristics of clients who receive CARE Act services; and (3) enable HAB to describe the type and amount of services a client receives. In addition to meeting the goal of accountability to the Congress, clients, advocacy groups, and the general public, information collected on the CADR is critical for HRSA, State, and local grantees, and individual providers to assess the status of existing HIV-related service delivery systems.

The response burden for grantees is estimated as:

Title under which grantee is funded	Number of grantee respondents	Responses per grantee	Hours to coordinate receipt of data reports	Total hour burden
Title I Only	51	1	40	2,040
Title II Only	59	1	40	2,360
Title III Only	365	1	20	7,300
Title IV Only	90	1	20	1,800
Subtotal	565			13,500

The response burden for service providers is estimated as:

Title under which grantee is funded	Number of respondents	Responses per provider	Hours per response	Total hour burden
Title I Only	976	1	26	25,376
Title II Only	857	1	26	22,282
Title III Only	166	1	44	7,304
Title IV Only	122	1	42	5,124
Funded under more than one title	681	1	50	34,050
Subtotal	2,802			94,136
Total for providers and grantees	3,367			107,636

Send comments to Susan G. Queen, Ph.D., HRSA Reports Clearance Officer, Room 14–45, Parklawn Building, 5600 Fishers Lane, Rockville, MD 20857. Written comments should be received within 60 days of this notice.

Dated: May 20, 2004.

Tina M. Cheatham,

Director, Division of Policy Review and Coordination.

[FR Doc. 04–12012 Filed 5–26–04; 8:45 am]

BILLING CODE 4165–15–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.

AT8, a Hybridoma Cell Line Producing a Monoclonal Antibody (MAb) Specific for Ly49G, a Mouse Natural Killer (NK) Cell Receptor

Andrew J. Makrigiannis (NCI).
DHHS Reference No. E–131–2004/0—Research Material.
Licensing Contact: Cristina Thalhammer-Reyero; 301/435–4507; *thalhamc@mail.nih.gov*.

This MAb is useful for identifying and isolating specific subpopulations of mouse NK cells using flow cytometry and fluorescence activated cell sorting (FACS). The AT8 antibody is also useful as a reagent to study the innate immune system using mouse models. This antibody has been described in Makrigiannis *et al.*, ''Independent Control of *Ly49g* Alleles: Implications for NK Cell Repertoire Selection and Tumor Cell Killing,'' J. Immunol. 2004 172:1414–1425.

Materials and Methods for Inhibiting Wip1

Dmitry Bulavin, Galina BeLova, Albert J. Fornace, Jr. (NCI).
U.S. Patent Application filed 12 Mar 2004 (DHHS Reference No. E–340–2003/0-US–01), a CIP of PCT/US03/08997 filed 21 Mar 2003, which published as WO 03/083103 on 01 Oct 2003 (DHHS Reference No. E–002–2002/0-PCT–02).
Licensing Contact: Jesse S. Kindra; 301/435–5559; *kindraj@mail.nih.gov*.

Wild-type p53-induced phosphatase 1 (Wip1) is a MG^{2+}-dependent serine/threonine protein phosphatase that is expressed in response to ionizing or ultra-violate radiation in a manner that is dependent on the tumor suppressor gene product p53. Wip1 has been shown to dephosphorylate and inactivate p38 MAP kinase, which in its activated state functions to activate p53 for the induction of apoptosis and transcription in response to environmental stress, thereby rendering Wip1 anti-apoptotic.

Further studies have indicated that Wip1 is a candidate proto-oncogene involved in tumorigenesis. Therefore, Wip1 represents an attractive new target for cancer therapy. Accordingly, the present invention relates to methods and compositions of inhibiting Wip1 in a cell. Inhibition of Wip1 would be expected to reduce tumor cell viability either alone or in combination with cytotoxic agents.

Genes Expressed in Prostate Cancer and Methods of Use

Ira Pastan, Tapan Bera, and Byungkook Lee (NCI).
U.S. Provisional Patent Application No. 60/461,399 filed 08 Apr 2003 (DHHS Reference No. E–148–2003/0-US–01); PCT Application has been filed.
Licensing Contact: Brenda Hefti; 301/435–4632; *heftib@mail.nih.gov*.

This invention is a novel gene, called New Gene Expressed in Prostate (NGEP). This gene appears to be expressed only in prostate. This gene has two known splice variants of significantly different size. The shorter splice variant encodes a cytoplasmic protein, while the longer splice variant encodes a plasma membrane protein.

This patent application contains claims to the polypeptide, NGEP, nucleotides encoding NGEP, antibodies that bind NGEP polypeptides, and methods of using these polypeptides, polynucleotides, and antibodies.

The presence of the protein on the cell surface and the selective expression in prostate and prostate cancer make this a potential target for prostate cancer diagnostics and therapeutics. Potential therapeutics could be gene-based, vaccines, antibodies, or immunoconjugates. Further information can be obtained by viewing a recent publication by the inventors (PNAS v.104 no.9, p.3050–3064, March 2, 2004).

BASE, a New Cancer Gene, and Uses Thereof

Ira Pastan, Kristi Egland, James Vincent, Byungkook Lee, and Robert Strausberg (NCI).
PCT Application No. PCT/US03/39476 filed 10 Dec 2003 (DHHS Reference No. E–321–2002/0–PCT–02).
Licensing Contact: Brenda Hefti; 301/435–4632; *heftib@mail.nih.gov*.

The present invention identifies a new gene expressed in breast cancers. The gene undergoes alternative splicing, and is expressed as one of two polypeptides. Both splice variants appear to be secreted proteins, and therefore good potential therapeutic targets. The patent application claims BASE polypeptides, nucleic acids, gene therapy and vaccine uses, and antibodies. This novel gene target might be useful as a breast cancer marker for diagnostics, or as a target for breast cancer therapeutics.

IL–21 Critically Regulates Immunoglobulin Production

Warren J. Leonard, Katsutoshi Ozaki, and Rosanne Spolski (NHLBI).
U.S. Provisional Patent Application 60/393,215 filed 01 Jul 2002 (DHHS Reference No. E–211–2002/0–US–01); PCT/US03/20370 filed 26 Jun 2003, which published as WO 04/003156 on 08 Jan 2004 (DHHS Reference No. E–211–2002/0–PCT–02).
Licensing Contact: Brenda Hefti; 301/435–4632; *heftib@mail.nih.gov*.

The invention includes a mouse in which the IL–21 receptor gene is disrupted by homologous recombination, the disruption being sufficient to prevent expression of the IL–21 receptor and thus to inhibit the action of IL–21. The invention also includes a mouse in which both the IL–21 receptor gene and the IL–4 gene are simultaneously disrupted in fashions being sufficient to inhibit the action of IL–21 and the production of IL–4. In a homozygous state, these mutations produce a mouse that has diminished B cell function.

This invention also relates to the use of agents that inhibit the interaction of IL–21 with the IL–21 receptor to modulate an immune response. This invention may be used to alter B cell activity, to treat a subject with Job's disorder, to treat an allergic reaction in a subject, or prevent an allergic reaction in a subject.

Novel Anti-CD30 Antibodies and Recombinant Immunotoxins Containing Disulfide-Stabilized Fv Fragments

Ira H. Pastan *et al.* (NCI).
U.S. Provisional Application No. 60/387,293 filed 07 Jun 2002 (DHHS Reference No. E–135–2002/0–US–01); PCT Application No. PCT/US03/18373 filed 07 Jun 2003, which published as WO 03/104432 on 18 Dec 2003 (DHHS Reference No. E–135–2002/1–PCT–01).
Licensing Contact: Brenda Hefti; 301/435–4632; *heftib@mail.nih.gov*.

The present invention discloses the creation of new anti-CD30 stalk antibodies and anti-CD30 dsFv-immunotoxins, which have shown good cytotoxic activity.

CD30 is a member of the tumor necrosis factor receptor super family. It is an excellent target due to its high expression in malignant Reed Sternberg cells of Hodgkin's Lymphoma (HL) and in anaplastic large cell lymphomas (ALCL), and due to its expression in only a small subset of normal lymphocytes. Previous attempts to target CD30 include the scFv immunotoxin Ki-4 that has shown specific binding to CD30-positive lymphoma cell lines and killed target cells.

The immunotoxins of the present invention are more stable and have higher affinity for CD30 then their predecessors. Research thus far has

shown that the dsFv-immunotoxins are able to kill a variety of CD30-positive lymphoma cell lines in vitro as well as CD30-transfected A431 cells via specific binding to CD30.

As claimed in this patent application, some of the antibodies do not bind to CD30 released from cells, although they do bind to cell associated CD30. This enhancement further increases the ability of immunotoxins and other immunoconjugates to target and treat lymphomas expressing CD30.

Dated: May 20, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–11970 Filed 5–26–04; 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 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 Cancer Institute Special Emphasis Panel; Special Emphasis Panel for K25 Grant Applications.

Date: June 15, 2004.

Time: 3 p.m. to 5 p.m.

Agenda: To review and evaluate grant applications.

Place: Holiday Inn Georgetown, 2101 Wisconsin Avenue, NW., Washington, DC 20007.

Contact Person: Lynn M. Amende, PhD, Scientific Review Administrator, Resources and Training Review Branch, Division of Extramural Activities, National Cancer Institute, 6116 Executive Boulevard Room 8105, Bethesda, MD 20892–8328, 301–451–4759, *amendel@mail.nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399, Cancer Control, National Institutes of Health, HHS)

Dated: May 20, 2004.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 04–11981 Filed 5–26–04; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; 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 a meeting of the National Cancer Institute Board of Scientific Advisors.

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.

Name of Committee: National Cancer Institute Board of Scientific Advisors.

Date: June 24–25, 2004.

Time: June 24, 2004, 8 a.m. to 6 p.m.

Agenda: Director's Report; Ongoing and New Business; Reports of Program Review Group(s); and Budget Presentation; Reports of Special Initiatives; RFA and RFP Concept Review; and Scientific Presentations.

Place: National Institutes of Health, Building 31, C Wing, 6 Floor, Conference Rm. 10, 9000 Rockville Pike, Bethesda, MD 20892.

Time: June 25, 2004, 8:30 a.m. to 1 p.m.

Agenda: Ongoing and New Business; Reports of Program Review Group(s); and Budget Presentation; Reports of Special Initiatives; RFA and RFP Concept Review; and Scientific Presentations.

Place: National Institutes of Health, Building 31, C Wing, 6 Floor, Conference Rm. 10, 9000 Rockville Pike, Bethesda, MD 20892.

Contact Person: Paulette S. Gray, PhD, Executive Secretary, Acting Director, Division of Extramural Activities, National Cancer Institute, National Institutes of Health, 6116 Executive Boulevard, 8th Floor, Rm. 8141, Bethesda, MD 20892, 301–496–4218.

Any interested person may file written comments with the committee by forwarding the statement to the Contact Person listed on this notice. The statement should include the name, address, telephone number and when applicable, the business or professional affiliation of the interested person.

In the interest of security, NIH has instituted stringent procedures for entrance into the building by non-government employees. Persons without a government I.D. will need to show a photo I.D. and sign-in at the security desk upon entering the building.

Information is also available on the Institute's/Center's homepage: *http://deainfo.nci.nih.gov/advisory/bsa.htm,* where an agenda and any additional information for the meeting will be posted when available.

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399, Cancer Control, National Institutes of Health, HHS).

Dated: May 20, 2004.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 04–11982 Filed 5–26–04; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Human Genome Research 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 section 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 Human Genome Research Institute Initial Review Group; Genome Research Review Committee.

Date: June 3, 2004.

Time: 11:30 a.m. to 3 p.m.

Agenda: To review and evaluate grant applications.

Place: Building 31, Bethesda, MD 20814 (Telephone Conference Call).

Contact Person: Ken D. Nakamura, PhD, Scientific Review Administrator, Office of Scientific Review, National Human Genome Research Institute, National Institutes of Health, Bethesda, MD 20892, 301 402–0838.