include, but are not limited to, the collection of data on the number of participants served, referral sources, kinds of services delivered, project goals, and other relevant data.

Respondents: State Access and Visitation Program Coordinators and administrators of state and/or local service providers.

ANNUAL BURDEN ESTIMATES

Instrument	Number of respondents	Number of responses per respondent	Average burden hours per response	Total burden hours
Grants to States: Access and Visitation Program Survey (1 additional year.—to collect FY 2001 program data in FY 2003).	324	1	15	4,860
State Child Access Program Survey (FY 2003, 2004, 2005).	324	1	15	4,860
Estimated Total Annual Burden Hours:.				Average 6,480 over 3 yrs. (9,720 in FY 2003; 4,860 in FY 2004; 4,860 in FY 2005.

Additional Information: Copies of the proposed collection may be obtained by writing to the Administration for Children and Families, Office of Administration, Office of Information Services, 370 L'Enfant Promenade, SW., Washington, DC 20447, Attn: ACF Reports Clearance Officer.

OMB Comment: OMB is required to make a decision concerning the collection of information between 30 and 60 days after publication of this document in the **Federal Register**. Therefore, a comment is best assured of having its full effect if OMB receives it within 30 days of publication. Written comments and recommendations for the proposed information collection should be sent directly to the following: Office of Management and Budget, Paperwork Reduction Project, 725 17th Street, NW., Washington, DC 20503, Attn: Desk Officer for ACF.

Dated: January 21, 2003.

Robert Sargis,

Reports Clearance Officer.

[FR Doc. 03–1971 Filed 1–28–03; 8:45 am]

BILLING CODE 4184–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

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

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.

Minimally Immunogenic Variant of Humanized COL–1 Antibody Against Carcinoembryonic Antigen (CEA)

Syed V.S. Kashmiri (NCI), Jeffrey Schlom (NCI), Eduardo A. Padlan (NIDDK)

DHHS Reference No. E–239–2002/0–US–01 filed 05 Sep 2002

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

Monoclonal antibodies (mAbs) show promise for the diagnosis and treatment of human cancers. COL–1 has a high affinity for carcinoembryonic antigen (CEA), and it reacts specifically to CEA. The present invention discloses humanized COL–1 (HuCOL–1) mAbs that are potentially minimally immunogenic and retain CEA binding affinity. Humanization of the antibody by ''abbreviated'' CDR grafting has reduced the risk of human anti-murine antibody response associated with the clinical use of murine mAbs for diagnosis and treatment of CEA expressing tumors. This invention also provides further methods of detecting and treating CEA expressing tumors.

Novel Broadly Cross-Reactive HIV Neutralizing Human Monoclonal Antibodies Selected From Fab Phage Display Libraries Using a Novel Strategy Based on Alternative Antigen Panning

Dimiter S. Dimitrov (NCI) and Mei-Yun Zhang (SAIC)

DHHS Reference No. E–144–2002/0–US–01 filed 05 May 2002 and

Novel Broadly Cross-Reactive HIV–1 Neutralizing Human Single-Chain Antibodies Derived From X5 by DNA Shuffling and Alternating Antigen Panning

Dimiter S. Dimitrov (NCI) and Mei-Yun Zhang (SAIC)

DHHS Reference No. E–144–2002/1–US–01 filed 05 May 2002

Licensing Contact: Sally Hu; 301/435–5606; *hus@od.nih.gov*

This invention (E–144–2002/0–US–01) identifies four antibodies, designed m12, m14, m16, and m18. These four antibodies were isolated from a human Fab phage display library using alternating antigen panning (AAP). All four antibodies bind to recombinant HIV envelope glycoproteins (Env) $gp12089.6$, $gp120JR–FL$ and $gp120IIIB$ with high affinity. Moreover, m12 binding to gp 120 or gp 140 is significantly enhanced in the presence of the receptor CD4. The second invention (E–144–2002/1–US–01) describes two scFv clones, designated M6 and M9 that were selected form phage-displayed X5 scFv mutants library by panning the library against $gp12089.6/IIIB$–CD4 complex using

the same strategy, alternating antigen panning strategy (AAP). M6 and M9 are more stable than previously reported HIV–1 antibody named X5 and have significant improved binding activities to $gp_{120IIIB}$. Both scFvs inhibit more efficiently membrane fusion mediated by envelope glycoproteins of primary HIV isolates with a broader spectrum compared to X5, indicating that scFv format may be a more proper format compared to Fab for HIV–1 neutralizing antibodies to inhibit virus infection and transmission. Furthermore, scFv is a single molecule with almost half size of Fab, which makes scFv more suitable for constructing bivalent and multivalent antibodies and antibody fusion proteins. Thus, since all six antibodies from the above two inventions cross-react with different HIV–1 isolates, these antibodies could be directly used for therapy of HIV–1 infected individuals. In addition, these antibodies can be also used for screening of peptide phage display libraries, libraries of Envs, and in general as tools for development of HIV vaccines.

A Mouse Model for Human Osteoarthritis

Laurent G. Ameye (NIDCR), Marian F. Young (NIDCR), Ake Oldberg (EM), Tianshun Xu (NIDCR)

DHHS Reference No. E–081–2002/0

Licensing Contact: Susan Carson; 301/435–5020; *carsons@od.nih.gov*

Osteoarthritis (OA) is the most common form of arthritis and affects more than 20 million Americans, costing billions of dollars in health care annually. Osteoarthritis is caused by the breakdown of joint cartilage, leading to a loss of the cartilage ''cushion'' between the bones of the joints. Risk factors associated with OA include age, obesity, traumatic injury and overuse due to sports or occupational stresses. There is no cure for OA and current treatments are directed at the symptomatic relief of pain, and at improving and maintaining joint function. There remains, however, a critical need both to develop OA treatments that focus on slowing down the degenerative process of the disease and for validated animal models to test these new treatments. NIH scientists at the NIDCR have generated a mouse model for osteoarthritis (FASEB J. (2002) 16, 673–680) that fills one part of this important gap.

The mouse model is a double knockout mouse that lacks biglycan and fibromodulin, two members of the small leucine-rich proteoglycan family, and that spontaneously develops OA. All the hallmarks of human osteoarthritis are present, including: progressive degeneration of the articular cartilage from early fibrillation to complete erosion, subchondral sclerosis, an absence of inflammation and development of osteophytes and cysts. Advantages over the existing models for osteoarthritis include: high phenotypic penetrance, early onset (at 1–2 months) and a rapid disease progression (between 3–6 months) which can be accelerated by moderate levels of exercise, such as treadmill running. These properties, combined with a normal life span, make the biglycan/fibromodulin-deficient mouse an ideal animal model for evaluating new drugs and treatments for osteoarthritis.

Ligands for FPR Class Receptors That Induce a Host Immune Response to a Pathogen or Inhibit HIV Infection

Ji Ming Wang *et al.* (NCI)

DHHS Reference Nos. E–267–1999/0–PCT–04 filed 04 Feb 2000 (PCT/US00/02842) and E–267–1999/0–US–05 filed 17 Jul 2002

Licensing Contact: Marlene Shinn-Astor; 301/435–4426; *shinnm@od.nih.gov*

The NIH announces a technology that relates a synthetic amino acid peptide that has been discovered to have chemotactic activity and the ability to activate both the FPR and FPRL1 receptors. This peptide has been found by NIH investigators to be a potent inhibitor of cellular response to chemokines including those chemokines that use the CCR5 receptor. It has been found that the activation of the FPRL1 by the peptide will in fact inhibit HIV–1 fusion to a cell and its infection through the CCR5 receptor. The peptide can potentially be used as a topical drug in the anal-vaginal tract to prevent or reduce the mucosal transmission of HIV–1. It also has the potential to be used as a vaccine adjuvant to prime a host response from a patient to a microbial infection. In addition, because of its interaction with the FPR and FPRL1 receptor it could be used to design drugs which interfere with responses due to the presence of excess quantities of chemokines. The peptide is short and contains a D-amino acid so that it is economical and easy to synthesize. Also, it may be more resistant to proteolytic degradation in vivo, which will prolong its half-life and therefore make it more effective as a treatment. It is available for immediate licensing and research collaborations via a Cooperative Research and Development Agreement (CRADA).

Dated: January 10, 2003.

Jack Spiegel,

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

[FR Doc. 03–1988 Filed 1–28–03; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Heart, Lung, and Blood 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 Heart, Lung, and Blood Institute Special Emphasis Panel.

Date: February 20–21, 2003.

Time: 7 p.m. to 5 p.m.

Agenda: To review and evaluate grant applications.

Place: Double Tree Rockville, 1750 Rockville Pike, Rockville, MD 20852.

Contact: Robert B. Moore, PhD, Review Branch, Room 7178, Division of Extramural Affairs, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, MD 20892, 301–435–0725.

(Catalogue of Federal Domestic Assistance Program Nos. 93.233, National Center for Sleep Disorders Research; 93.837, Heart and Vascular diseases Research, 93.838, Lung Diseases Research; 93.839, Blood Diseases and Resources Research, National Institutes of Health, HHS)

Dated: January 16, 2003.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 03–1983 Filed 1–28–03; 8:45 am]

BILLING CODE 4140–01–M