(NCRR), the National Institutes of Health (NIH) will publish periodic summaries of proposed projects to be submitted to the Office of Management and Budget (OMB) for review and approval.

Proposed Collection: Title: Inventory and Evaluation of Clinical Research Networks. *Type of Information Collection Request:* Revision of OMB # 0925–0550. *Expiration:* 07/31/08. *Need and Use of Information Collection:* Through the original data collection, the IECRN project identified and surveyed clinical research networks to obtain data for two purposes: (1) To create a web-based inventory of clinical research networks that can be accessed by the clinical research community and the general public and (2) to prepare a detailed description of existing network practices from a sample of identified networks. The current request is to continue collecting data for the first purpose only. The instrument known as the *Core Survey* will be used to collect information to confirm that the respondent is truly a clinical research network, plus basic characteristics about each identified clinical research network to be included in the web-based inventory. The information for the inventory database includes the network's name, address, contact information, funding sources, age, geographic coverage, size, composition, and populations and diseases of focus. Permission to post the network's data in the web-based public inventory will be requested, and only those networks that agree will have their information posted. Currently the inventory includes ''network profiles'' for approximately 270 clinical research networks. While this number is believed to represent most of the existing networks, some networks have not yet been identified, are unaware of the existence of the inventory, or are newly formed since the original data collection occurred. In addition, each network in the inventory is requested annually to update the information posted in its ''network profile'' to ensure that the inventory is complete and accurate. *Frequency of Response:* Once (*Core Survey*), Annually (*Network Updates*). *Affected Public:* Individuals. *Type of Respondents:* Health Professionals (Physicians and others involved in research networks).

TABLE A 12.1—ESTIMATE OF ANNUAL HOUR BURDEN AND ANNUALIZED COST TO RESPONDENTS

Type of respondent	Number of responses	Frequency of response	Length of response	Annual hour burden	Hourly wage rate	Respondent cost
Core Survey:						
Principal Investigator	20	1	0.25 (15 minutes)	5	$70.00	$350.00
Annual Update:						
PI/network contact	280	1	.1667 (10 minutes)	46.7	70.00	3,269.00
Total				51.7		3,619.00

The annualized cost to respondents is estimated at: $3,619.00. There are no Capital Costs to report. There are no Operating or Maintenance Costs to report.

Request for Comments: Written comments and/or suggestions from the public and affected agencies are invited on one or more of the following points: (1) Whether the proposed collection of information is necessary for the proper performance of the function of the agency, including whether the information will have practical utility; (2) The accuracy of the agency's estimate of the burden of the proposed collection of information, including the validity of the methodology and assumptions used; (3) Ways to enhance the quality, utility, and clarity of the information to be collected; and (4) Ways to minimize the burden of the collection of information on those who are to respond, including the use of appropriate automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

FOR FURTHER INFORMATION CONTACT: To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact Dr. Jody Sachs, National Center for Research Resources, NIH, Room 917, 6701 Rockledge Drive, Bethesda, MD 20892–4874, or call 301–435–0802.

Comments Due Date: Comments regarding this information collection are best assured of having their full effect if received within 60-days of the date of this publication.

Dated: March 18, 2008.

Jody Sachs,

Project Officer, NCRR, National Institutes of Health.

[FR Doc. E8–5816 Filed 3–21–08; 8:45 am]

BILLING CODE 4140–01–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, HHS

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.

HIV Monoclonal Antibodies

Description of Technology: This technology describes several hybridomas that produce monoclonal antibodies (mAbs) useful in HIV research applications. The mAbs are specific for either gp41 or gp120. In particular, the hybridomas producing mAbs designated D19, D56, M12, T8 and T24 (all anti-gp120), and T32 and T33 (gp41 specific) were found to be of particular utility. Additional hybridomas expressing mAbs disclosed in the publications may also be available.

Applications: HIV research.

Development Status: Murine hybridomas available; T32 mAb available.

Inventors: Bernard Moss, Patricia Earl, Christopher Broder, and Robert Doms (NIAID).

Publications:

1. PL Earl, CC Broder, RW Doms, B Moss. Epitope map of human immunodeficiency virus type 1 gp41 derived from 47 monoclonal antibodies produced by immunization with oligomeric envelope protein. J Virol. 1997 Apr;71(4):2674–2684.
2. U.S. Patents 6,039,957 and 6,171,596 (gp140 mAbs).
3. PL Earl, CC Broder, D Long, SA Lee, J Peterson, S Chakrabarti, RW Doms, B Moss. J Virol. 1994 May;68(5):3015–3026 (gp140 mAbs).

Patent Status:

HHS Reference No. E–109–2008/0 (anti-gp41mAbs)—Research Tool. Patent protection is not being pursued for this technology.

HHS Reference No. E–200–1993/1 (anti-gp140 mAbs).

Licensing Status: Available for biological materials licensing only; the IP that includes descriptions of the anti-gp120 and gp41 mAbs is available for exclusive or non-exclusive licensing.

Licensing Contact: Susan Ano, PhD; 301–435–5515; *anos@mail.nih.gov.*

Collaborative Research Opportunity: The NIAID/DIR/LVD is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize HIV Monoclonal Antibodies. Please contact either Michael Pizali or Dana Hsu at 301–496–2644 for more information.

Epoxy-guaiane Cancer Inhibitors: New Class of Natural Products Isolated from the African Plant *Phyllanthus englerii*

Description of Technology: The present invention involves the observation of renal selective inhibitory activity by the extracts of the African plant Phyllanthus englerii. Bioassay-guided fractionation of the purified extracts revealed a series of novel chemical entities which are named Englerin A–F. The englerins and their derivatives are useful in the treatment of a number of cancers, particularly renal cancer. The englerins exhibit selective and potent renal cell inhibitory activity *in vitro.*

These compounds are recoverable in reasonable yield from natural product extracts and are considered to be reasonably tractable for synthetic chemistry schemes. Sufficient supply of several analogs had been extracted from repository samples for identification and initial biological characterization. Subsequent five-dose testing in the NCI60 screening panel indicated and confirmed impressive renal-selective activity.

Applications: The new chemical entities can be potential cancer therapeutics, especially for renal cancer.

Advantages:

There is reasonable yield and recovery of the compounds from the natural product extracts.

The synthetic chemistry schemes for synthesis of these compounds are considered to be reasonably tractable.

Development Status: Proof of concept in vitro studies have been completed and further in vitro and in vivo animal model studies are ongoing.

Inventors: John A. Beutler et al. (NCI).

Relevant Publication: S. Sutthivaiyakit et al. A novel 29-nor-3,4-seco-friedelane triterpene and a new guaiane sesquiterpene from the roots of Phyllanthus oxyphyllus. Tetrahedron 2003 Dec 8;59(50):9991–9995.

Patent Status: U.S. Provisional Application No. 61/018,938 filed 04 Jan 2008 (HHS Ref. No. E–064–2008/0–US–01).

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: Surekha Vathyam, PhD; 301–435–4076; *vathyams@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute Molecular Targets Development Program is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize epoxy-guaiane cancer inhibitors. Please contact John D. Hewes, Ph.D. at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

VEGF-B as a Therapeutic Agent for Neurodegenerative Disease

Description of Technology: This technology identifies vascular endothelial growth factor–B (VEGF–B) as a potent inhibitor of apoptosis in neuronal and other types of cells, and highlights its ability to rescue these cells from apoptosis in the brain and retina. Members of the VEGF family of proteins are noted for their angiogenic and blood vessel permeabilizing abilities. Some members of this family, such as VEGF–A, may promote neurogenesis; however, the neuroprotective effects are accompanied by inherent angiogenic and vessel permeabilizing activities, which make VEGF–A treatment unsuitable for clinical use as neuroprotective agents. The inventor has recently discovered that unlike the other VEGF family members, the neuroprotective effects of VEGF–B are not associated with undesired angiogenesis or increased blood vessel permeability, but rather through inhibiting apoptosis via suppressing the expression of the apoptotic/cell death related genes (1). This discovery, that the use of VEGF–B can protect endangered neurons from death and avoid the undesirable effects associated with other VEGF family members, makes it a promising candidate for the treatment of neurodegenerative and other diseases that involve neuronal impairment and/or excessive apoptosis, such as muscular dystrophy, stroke, brain injury, myocardial infarction, ischemic renal damage, etc.

In-vivo trials have already demonstrated the efficacy of VEGF–B as a therapeutic agent. VEGF–B has shown efficacy in mouse models suffering from optic nerve crush injury (ONC). ONC induces the apoptotic death of retinal ganglion cells (RGCs) in the retina. However, intravitreal administration of a single dose of the VEGF–B protein significantly restored the number of RGCs by 1.7 fold, demonstrating the potential use of the protein in treating degenerative ocular diseases, such as glaucoma. Similar results were obtained when exogenous administration of VEGF–B to the brain cortex was shown to significantly reduce ischemia-induced stroke volume and to protect neurons from apoptosis in the brain. Further, intracerebroventricular injection of VEGF–B in mutant knockout mice lacking the gene for VEGF–B (VEGFB–KO) has caused a complete reversal of neuronal impairment and restored neurogenesis back to normal levels.

Applications: VEGF–B as a powerful therapeutic agent for use in a wide range of therapeutic intervention regimes where neuronal repair and inhibition of apoptosis are required.

Inventors: Xuri Li (NEI).

Relevant Publications

1. Yang Li, Fan Zhang, Nobuo Nagai, Zhongshu Tang, Shuihua Zhang, Pierre Scotney, Johan Lennartsson, Chaoyong Zhu, Yi Qu, Changge Fang, Jianyuan Hua, Osamu Matsuo, Guo-Hua Fong, Hao Ding, Yihai Cao, Kevin G. Becker, Andrew Nash, Carl-Henrik Heldin, and Xuri Li. VEGF–B inhibits apoptosis via VEGFR–1-mediated suppression of the expression of BH3-only protein genes in mice and rats. J Clin Invest. 2008 Mar 3;118(3):913–923. Published online 2008 Feb 7, doi 10.1172/JCI33673.
2. Yunjuan Sun, Kunlin Jin, Jocelyn T. Childs, Lin Xie, Xiao Ou Mao, David A. Greenberg. Vascular endothelial growth factor–B (VEGFB) stimulates

neurogenesis: Evidence from knockout mice and growth factor administration. Dev Biol. 2006 Jan 15;289(2):329–335.

Patent Status: U.S. Provisional Application No. 60/972,780 filed 15 Sep 2007 (HHS Reference No. E–154–2007/0–US–01).

Licensing Status: Available for exclusive or non-exclusive licensing.

Licensing Contact: Jasbir (Jesse) S. Kindra, J.D., M.S.; 301–435–5170; *kindraj@mail.nih.gov*.

Collaborative Research Opportunity: The National Eye Institute, NIH, Office of Scientific Director, Unit of Retinal Vascular Neurobiology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize VEGF–B as a therapeutic agent in treating various types of degenerative (neural, vascular, muscular, etc.) diseases, and to study the molecular and cellular mechanisms involved. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Rapid *Clostridium botulinum* Diagnostic for Food Safety and Biodefense Applications

Description of Technology: The urgent need for a rapid diagnostic test capable of detecting all serotypes of *C. botulinum* is well known. Botulinum neurotoxins (BoNTs) are the most potent biological toxins known and are categorized as category A biodefense agents because of lethality and ease of production. BoNTs are also one of the most deadly agents associated with food poisoning. Current diagnostic methods include clinical observation of symptoms that could be mistaken for other neurological conditions and a mouse protection bioassay that takes as long as four days and has a number of disadvantages. The subject technology utilizes unique PCR primers for the detection of the non-toxin non-hemaglutinin (NTNH) gene of *C. botulinum*; this gene is highly conserved in all *C. botulinum* toxin types and subtypes. Thus, samples that contain botulinum can be determined regardless of serotype involved, providing a universal means of diagnosis. Further, the technology describes different PCR primers and flurogenic probes for a BoNT-specific assay. The type-specific assay can be used independently or in conjunction with the universal assay described above. The universal and type-specific assays were successfully used first to identify positively botulinum DNA samples in a test of botulinum and non-botulinum clostridia species then to determine the toxin type. The diagnostic testing described by the subject technology requires less significantly less time than the current gold standard diagnostic tests.

Applications: Universal diagnostic test for *C. botulinum*; Diagnostic test for *C. botulinum* capable of detecting all seven toxin types; Combination diagnostic; Food safety applications; Biodefense applications.

Development Status: Fully developed.

Inventors: Daniel C. Douek *et al.* (VRC/NIAID).

Patent Status: U.S. Provisional Application No. 60/884,539 filed 11 Jan 2007 (HHS Reference No. E–046–2007/0–US–01); PCT Patent Application No. PCT/US2008/50872 filed 11 Jan 2008 (HHS Reference No. E–046–2007/0–PCT–02).

Licensing Status: Available for non-exclusive or exclusive licensing.

Licensing Contact: Susan Ano, PhD; 301/435–5515; *anos@mail.nih.gov*.

Collaborative Research Opportunity: The NIAID is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize ''Rapid *Clostridium botulinum* Diagnostic for Food Safety and Biodefense Applications.'' Please contact either Rosemary Walsh or Barry Buchbinder at 301–496–2644 for more information.

Prolidase Expression Construct Useful as Anti-Angiogenesis Screen

Description of Technology: The technology describes a prolidase expression construct and a method of using the construct to isolate stable transfectants with high prolidase expression. Specifically, a human colorectal cancer cell line (RKO) was transfected with a plasmid (pcDNA3.1) expressing prolidase cDNA. Using this cell line, the inventors found that extracellular matrix degradation is associated with the prolidase-dependent activation of the hypoxia/inflammation pathway. The construct and transfectants can also be used to study other regulatory functions of prolidase.

Applications

Prolidase as a target for anti-angiogenesis drugs: Angiogenesis, a prerequisite for tumor growth, requires proteolysis of the extracellular matrix (ECM). Prolidase participates in the degradation of the ECM by hydrolyzing collagen dipeptides having C-terminal proline or hydroxyproline. Current anti-angiogenic approaches target matrix metalloproteinase activity, but this can cause musculoskeletal complications. By modulating prolidase activity to inhibit the degradation of the ECM, it may be possible to provide an alternative anti-angiogenic approach with fewer side effects. The prolidase construct and transfected cell lines could be used as a screen for prolidase modulators, which could be developed as anti-angiogenesis agents.

Prolidase as a target for anti-inflammatory drugs and wound-healing agents: Inherited prolidase deficiency is also associated with defective wound healing, extensive skin alterations, and immunodeficiency. Products from the prolidase activity screen may also have potential use in patients with prolidase deficiency, chronic inflammation, or problematic wound healing.

Development Status: Pre-clinical stage.

Inventors: Yongmin Liu (NCI), Arkadiusz Surazynski (NCI), James M. Phang (NCI), Sandra K. Cooper (NCI/SAIC), Steven P. Donald (NCI).

Publication: A Surazynski, SP Donald, SK Cooper, MA Whiteside, K Salnikow, Y Liu, JM Phang. Extracellular matrix and HIF–1 signaling: The role of prolidase. Int J Cancer. 2008 Mar 15;122(6):1435–1440.

Patent Status: HHS Reference No. E–235–2006/0—Research Material. Patent protection is not being sought for this technology.

Licensing Status: This invention is available for licensing through a Biological Materials License.

Licensing Contact: David A. Lambertson, PhD; 301/435–4632; *lambertsond@mail.nih.gov*.

Dated: March 17, 2008.

Steven M. Ferguson,

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

[FR Doc. E8–5813 Filed 3–21–08; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Diabetes and Digestive and Kidney Diseases; Notice of Closed Meetings

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 meetings.

The meetings 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