immune response. These replicons should be safer than a live attenuated vaccine because they cannot cause disease in the host and they should be better than subunit vaccines because they can replicate in the host.

Applications: Prevention of severe and/or fatal human disease caused by dengue virus, a major health concern in tropical and subtropical regions.

Inventor: Xiaowu Pang (CBER/FDA).

Patent Status: U.S. Patent Application 10/656,721 filed 05 Sep 2003, claiming priority to 09 Mar 2001 (HHS Reference No. E–228–2000/0–US–03).

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

Licensing Contact: Peter A. Soukas, J.D.; 301/435–4646; *soukasp@mail.nih.gov.*

Dated: December 1, 2006.

Steven M. Ferguson,

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

[FR Doc. E6–21029 Filed 12–11–06; 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.

ARH3, a Therapeutic Target for Cancer, Ischemia, and Inflammation

Description of Technology: ADP-ribosylation is important in many cellular processes, including DNA replication and repair, maintenance of genomic stability, telomere dynamics, cell differentiation and proliferation, and necrosis and apoptosis. Poly-ADP-ribose is important in a number of critical physiological processes such as DNA repair, cellular differentiation, and carcinogenesis. Until recently, only one human enzyme, PARG, had been identified that degrades the ADP-ribose polymer. Another ADP-ribose, O-acetyl-ADP ribose, is formed via the deacetylation of proteins, such as acetyl-histone, by proteins in the Sir2 family. Sir2 proteins have been implicated in regulation of chromatin structure and longevity.

The NIH announces the discovery of a novel PARG-like enzyme, ARH3. ARH3 possesses PARG activity, yet is structurally distinct from PARG. ARH3 also hydrolyzes O-acetyl-ADP-ribose, and is the only protein recognized to date with such activity. ARH3 thus appears to function in two important signaling pathways, serving to regulate both poly-ADP-ribose and O-acetyl-ADP-ribose levels. It may affect chromatin structure through effects on both pathways. Since ARH3 structures differs from PARG or other enzymes that participate in these pathways, it may be possible to design specific inhibitors to target both the poly-ADP-ribose and Sir2 pathways. These drugs may be used as anticancer agents, radiosensitizers or antiviral agents, or for treating disorders involving oxidative damage, such as acute tissue injury, ischemia, and inflammation.

Applications: (1) Development of therapeutics for cancer or disorders associated with excessive DNA damage; (2) Development of therapeutics for diseases involving oxidative damage, such as acute tissue injury, ischemia and inflammation.

Market: (1) Patients with chemotherapy-resistant tumors, or with cancers that are genetically deficient in DNA repair; (2) Patients with inflammatory or ischemia/reperfusion diseases, particularly those associated with acute cardiovascular disease.

Development Status: Early stage.

Inventors: Joel Moss et al. (NHLBI).

Related Publications:

1. S Oka, J Kato, J Moss. Identification and characterization of a mammalian 39-kDa poly(ADP-ribose) glycohydrolase. J Biol Chem. 2006 Jan 13;281(2):705–713.

2. T Ono, A Kasamatsu, S Oka, J Moss. The 39-kDa poly(ADP-ribose) glycohydrolase ARH3 hydrolyzes *O*-acetyl-ADP-ribose, a product of the Sir2 family of acetyl-histone deacetylases. Proc Natl Acad Sci USA 2006 Nov 7;103(45):16687–16691. Epub 2006 Oct 30, doi 10.1073/pnas.0607911103.

Patent Status: U.S. Provisional Application No. 60/716,807 filed 12 Sep 2005 (HHS Reference No. E–347–2004/0-US–01); PCT Application No. PCT/US2006/035771 filed 12 Sep 2006 (HHS Reference No. E–347–2004/0–PCT–02).

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

Licensing Contact: Tara L. Kirby, PhD; 301/435–4426; *tarak@mail.nih.gov.*

Collaborative Research Opportunity: The Pulmonary Critical Care Medicine Branch in the National Heart, Lung, and Blood Institute is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the invention. Please contact Marianne Lynch in the NHLBI Office of Technology Transfer and Development by phone (301–594–4094) or e-mail (*lynchm@nhlbi.nih.gov*) for more information.

Antisera To Detect Phosphorylated Phosphoinositide-Dependent Kinase 1 (PDK–1)

Description of Technology: PDK–1 phosphorylates and activates a number of cellular kinases, and plays a major role in insulin and growth factor signaling. PDK–1 also represents a promising drug target for a number of cancers. Autophosphorylation at Ser244 (mouse) or Ser241 (human) is critical for PDK–1 activity.

Available for licensing are polyclonal rabbit antisera that specifically detect mouse PDK–1 protein phosphorylated at Ser244. These antisera are also expected to be specific for the human PDK–1 protein phosphorylated at Ser241.

Applications: (1) Tool for screening PDK–1 autophosphorylation inhibitors for cancer and other indications; (2) Tool for studying insulin and growth factor signaling.

Inventor: Michael J. Quon (NCCAM).

Publication: MJ Wick, FJ Ramos, H Chen, MJ Quon, LQ Dong, F Liu. Mouse 3-phosphoinositide-dependent protein kinase-1 undergoes dimerization and trans-phosphorylation in the activation loop. J Biol Chem. 2003 Oct 31;278(44):42913–42919.

Patent Status: HHS Reference No. E–330–2003/0—Research Tool.

Licensing Status: This technology is available as a research tool under a Biological Materials License.

Licensing Contact: Tara Kirby, PhD; 301/435–4426; *tarak@mail.nih.gov*

Collaborative Research Opportunity: The NIH, NCCAM, Diabetes Unit is seeking statements of capability or interest from parties interested in collaborative research to further

develop, evaluate, or commercialize phospho-specific PDK–1 antibody and insulin signaling. Please contact Michael J. Quon, Chief, Diabetes Unit, NCCAM, NIH at *quonm@nih.gov* for more information.

Dated: December 6, 2006.

Steven M. Ferguson,

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

[FR Doc. E6–21037 Filed 12–11–06; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Library of Medicine; Notice of 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 open to the public as indicated below, 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 portions of the meeting devoted to the review and evaluation of journals for potential indexing by the National Library of Medicine will be closed to the public in accordance with the provisions set forth in section 552b(c)(9)(B), Title 5 U.S.C., as amended. Premature disclosure of the titles of the journals as potential titles to be indexed by the National Library of Medicine, the discussions, and the presence of individuals associated with these publications could significantly frustrate the review and evaluation of individual journals.

Name of Committee: Literature Selection Technical Review Committee.

Date: February 22–23, 2007.

Open: February 22, 2007, 9 a.m. to 11 a.m.

Agenda: Administrative reports and program discussions.

Place: National Library of Medicine, Building 38, Board Room, 2nd Floor, 8600 Rockville Pike, Bethesda, MD 20894.

Closed: February 22, 2007, 11 a.m. to 5 p.m.

Agenda: To review and evaluate journals as potential titles to be indexed by the National Library of Medicine.

Place: National Library of Medicine, Building 38, Board Room, 2nd Floor, 8600 Rockville Pike, Bethesda, MD 20894.

Closed: February 23, 2007, 8:30 a.m. to 2 p.m.

Agenda: To review and evaluate journals as potential titles to be indexed by the National Library of Medicine.

Place: National Library of Medicine, Building 38, Board Room, 2nd Floor, 8600 Rockville Pike, Bethesda, MD 20894.

Contact Person: Sheldon Kotzin, MLS, Associate Director, Division of Library Operations, National Library of Medicine, 8600 Rockville Pike, Bldg 38/Room 2W06, Bethesda, MD 20894, 301–496–6921. *Sheldon_Kotzin@nlm.nih.gov.*

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.

(Catalogue of Federal Domestic Assistance Program No. 93.879, Medical Library Assistance, National Institutes of Health, HHS)

Dated: December 4, 2006.

Anna Snouffer,

Acting Director, Office of Federal Advisory Committee Policy, NIH.

[FR Doc. 06–9631 Filed 12–11–06; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HOMELAND SECURITY

Coast Guard

[USCG 2006–25522]

Exercise of Authority To Require Pilots To Submit Results of Annual Chemical Test for Dangerous Drugs and Extension of Deadline for Pilots To Submit Most Recent Annual Physical Examination

ACTION: Notice.

SUMMARY: By this notice, the Coast Guard is exercising authority currently set forth in Coast Guard regulations to require all first class pilots on vessels greater than 1600 gross registered tons (GRT), and other individuals who ''serve as'' pilots on certain types of vessels greater than 1600 GRT, to provide the passing results of their annual chemical test for dangerous drugs to the Coast Guard, subject to certain exceptions. In addition, the Coast Guard is extending the deadline for pilots to submit the most recent copy of their annual physical examination.

FOR FURTHER INFORMATION CONTACT: Mr. Stewart A. Walker, National Maritime Center. Phone: 202–493–1022, e-mail: *Stewart.A.Walker@uscg.mil*

DATES: Unless excepted under 46 CFR 16.220(c), each pilot must do the following: Submit the passing results of his or her most recent annual chemical test for dangerous drugs to the Coast Guard on or before April 11, 2007; submit the passing results of his or her annual chemical test for dangerous drugs to the Coast Guard no later than 30 calendar days after receiving the results of the test; and undergo a chemical test for dangerous drugs annually within 30 calendar days of the anniversary date of the individual's most recent chemical test for dangerous drugs.

In addition, the Coast Guard is extending the deadline for pilots to submit a copy of their most recent physical examinations until April 11, 2007. This information was initially requested to be submitted to the Coast Guard no later than December 27, 2006 in a **Federal Register** notice published on September 28, 2006 at 71 FR 56999.

SUPPLEMENTARY INFORMATION: On September 28, 2006, the Coast Guard provided notice that it is exercising its authority to require first class pilots on vessels greater than 1600 GRT, and those individuals who ''serve as'' pilots in accordance with 46 CFR 15.812(b)(3) & (c) on vessels greater than 1600 GRT, to submit copies of their annual physical examinations to the Coast Guard. 71 Fed. Reg. 56999. Copies of that notice, as well as this notice are available electronically by searching for docket number USCG–2006–25522 at *http://dms.dot.gov.* The purpose of the physical examination notice was to implement the recommendation made by the National Transportation Safety Board (NTSB), in their report on the 2003 allision of the Staten Island Ferry ANDREW J. BARBERI, that the Coast Guard require submission of annual pilot physicals. This notice is a continuation of the Coast Guard's efforts to fully implement the NTSB's recommendation.

Coast Guard regulations require that, unless excepted under 46 CFR 16.220(c), each pilot who is required to complete an annual physical examination must also pass a chemical test for dangerous drugs, and that he or she must submit the passing (i.e. negative) results of the chemical test to the Coast Guard when applying for license renewal, or when requested by the Coast Guard. 46 CFR 16.220(b). This includes first class pilots on vessels greater than 1600 GRT, and those individuals who ''serve as'' pilots in accordance with 46 CFR 15.812(b)(3) &