commercialize in vitro assembly of protein microarrays. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Methods and Compositions for High-Throughput Detection of Protein/Protein Interactions Ex Vivo

Description of Technology: This invention relates to methods and compositions for the high-throughput detection of protein-protein interactions using a lambda phage display system. One of the central challenges in systems biology is defining the interactome, or set of all protein-protein interactions within a living cell, as a basis for understanding biological processes for early diagnosis of disease and for drug development. The invention provides a novel proteomic toolbox for high-throughput medical research based in combining phage lambda protein display and recent advances in manipulation of the phage's genome. The method uses the bacteriophage lambda vector to express proteins on its surface, and is based on the use of mutant phage vectors such that only interacting phages will be able to reproduce and co-infect an otherwise non-permissive host and produce plaques.

Application: The invention allows for the characterization of bacteriophage display libraries that could be easily adapted to be used in large-scale functional protein chip assays.

Inventors: Sankar Adhya and Amos Oppenheim (NCI).

Patent Status: U.S. Patent Application No. 11/719,925 filed 22 May 2007 (HHS Reference No. E–264–2004/0–US–03).

Licensing Status: Available for licensing.

Licensing Contact: Jeffrey A. James, PhD; 301–435–5474; *jeffreyja@mail.nih.gov.*

Therapeutic Methods Based on In Vivo Modulation of the Production of Interferon Gamma

Description of Technology: The technology offered for licensing is in the field of Therapeutics. More specifically, the technology relates to biological ligands and their use as modulators of the production of Interferon gamma as a means to treat a broad spectrum of diseases. The invention describes and claims antibodies and other ligands that can stimulate Natural Killer (NK) immune cells to produce Interferon gamma which contributes to the combat against foreign pathogens. Conversely, the invention also describes and claims methods that can inhibit such Interferon gamma production for treatment of diseases where excess of Interferon is not desirable. The invention also describes methods and assays to identify both inducing and inhibiting ligands.

The license agreement may include biological materials, such as monoclonal antibodies that were made and identified by the inventors as Interferon gamma stimulators.

Interferon-gamma is a potent antiviral and antimicrobial substance produced by natural killer (NK) white blood cells. NK cells are activated during infections by viruses and by other intracellular pathogens, such as parasites and bacteria. Soluble substances, such as interleukins, produced by infected cells activate NK cells to secrete interferon-gamma. Injection of interleukins into patients to stimulate NK cells to secrete interferon-gamma has not been a successful therapeutic approach because of the toxicity involved. The invention is based on the discovery by the inventors that activation of the KIR2DL4 receptor expressed by all NK cells stimulates them to produce interferon-gamma. The invention claims monoclonal antibodies and derivatives thereof, as well as natural and synthetic ligands of KIR2DL4 that can be utilized to stimulate interferon-gamma production by NK cells without any other stimulus. The possibility of inducing interferon-gamma production by NK cells without the toxic side effects of interleukins could be an effective therapy for various types of infections and of cancers. Also claimed in the invention are methods of treating various cancers and viral infections, methods of treating autoimmune disease, and methods of administration of the antibody or derivatives thereof. Certain diseases benefit from reduction in the amount of Interferon gamma. The instant invention claims such ligands that are capable of inhibiting KIR2DL4 from producing interferon gamma. It also describes methods of identifying such ligands.

Applications:

- Therapeutics of infectious diseases, cancer and autoimmune diseases
- The mAbs can be used as research reagents

Advantages: Absence of toxicity as compared with current methods such as IL–2 treatment.

Development Status: The inventors generated monoclonal antibodies that have demonstrated stimulation of Interferon gamma production. Proof of concept has been demonstrated.

Market: The technology lends itself to treatment of viral and microbial-caused infectious disease and possibly as therapy for certain cancers and autoimmune disease. Collectively, these medical areas represent a huge market of multi billion dollars and thus significant commercial opportunities.

Inventors: Eric O. Long and Sumati Rajagopalan (NIAID).

Relevant Publications:

1. S Rajagopalan, J Fu, EO Long. Cutting edge: induction of IFN-gamma production but not cytotoxicity by the killer cell Ig-like receptor KIR2DL4 (CD158d) in resting NK cells. J Immunol. 2001 Aug 15;167(4):1877–1881.

2. A Kikuchi-Maki, TL Catina, KS Campbell. Cutting edge: KIR2DL4 transduces signals into human NK cells through association with the fc receptor gamma protein. J Immunol. 2005 Apr 1;174(7):3859–3863.

3. S Rajagopalan, YT Bryceson, SP Kuppusamy, DE Geraghty, A van der Meer, I Joosten, EO Long. Activation of NK cells by an endocytosed receptor for soluble HLA–G. PLoS Biol 2006 Jan;4(1):e9.

Patent Status: U.S. Patent 7,435,801 issued 14 Oct 2008 (HHS Reference No. E–255–2000/0–US–03); U.S. Patent Application No. 12/249,703 filed 10 Oct 2008 (HHS Reference No. E–255–2000/0–US–04); both entitled ''Antibodies and Other Ligands Directed Against KIR2DL4 Receptor for Production of Interferon-Gamma''.

Licensing Status: Available for licensing. Monoclonal antibodies made by the inventors and identified as stimulators may be available and provided with the license agreement.

Licensing Contacts: Uri Reichman, PhD, MBA; 301–435–4616; *UR7a@nih.gov;* Rung C. Tang, JD, LLM; 301–435–5031; *tangrc@mail.nih.gov.*

Dated: April 17, 2009.

Richard U. Rodriguez,

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

[FR Doc. E9–9348 Filed 4–22–09; 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.

Identification of Subjects Likely To Benefit From Copper Treatment

Description of Technology: Menkes disease is an infantile onset X-linked recessive neurodegenerative disorder caused by deficiency or dysfunction of a copper-transporting ATPase, ATP7A. The clinical and pathologic features of this condition reflect decreased activities of enzymes that require copper as a cofactor, including dopamine-β-hydrolase, cytochrome c oxidase and lysyl oxidase. Recent studies indicate that ATP7A normally responds to N-methyl-D-aspartate receptor activation in the brain, and an impaired response probably contributes to the neuropathology of Menkes disease. Affected infants appear healthy at birth and develop normally for 6 to 8 weeks. Subsequently, hypotonia, seizures and failure to thrive occur and death by 3 years of age is typical. Occipital horn syndrome (OHS) is also caused by mutations in the copper transporting ATPase ATP7A, although its symptoms are milder than Menkes syndrome, including occipital horns and lax skin and joints.

Treatment with daily copper injections may improve the outcome in Menkes disease if commenced within days after birth; however, newborn screening for this disorder is not available and early detection is difficult because clinical abnormalities in affected newborns are absent or subtle. Moreover, the usual biochemical markers (low serum copper and ceruloplasmin) are unreliable predictors in the neonatal period, since levels in healthy newborns are low and overlap with those in infants with Menkes disease. Although molecular diagnosis is available, its use is complicated by the diversity of mutation types and the large size of ATP7A (about 140kb). Thus, there is a need for improved methods for early detection of infants with Menkes disease or OHS in order to improve outcomes.

This technology relates to methods of identifying individuals who may benefit from treatment with copper, particularly those having Menkes disease or Occipital Horn Syndrome.

Inventor: Stephen G. Kaler (NICHD).

Publication: SG Kaler, CS Holmes, DS Goldstein, JR Tang, SC Godwin, A Donsante, CJ Liew, S Sato, N Patronas. Neonatal diagnosis and treatment of Menkes disease. N Engl J Med. 2008 Feb 7;358(6):605–614.

Patent Status: PCT Application No. PCT/US2008/078966 filed 06 Oct 2008 (HHS Reference No. E–186–2008/0–PCT–01).

Licensing Status: Available for licensing.

Licensing Contact: Fatima Sayyid, M.H.P.M.; 301–435–4521; *Fatima.Sayyid@hhs.nih.gov*.

Collaborative Research Opportunity: The National Institute of Child Health and Human Development, Division of Intramural Research, Molecular Medicine Program, Unit on Pediatric Genetics, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize population-based newborn screening for Menkes disease and related disorders of copper transport in order to identify subjects likely to benefit from copper injections and other treatments. Please contact Alan Hubbs, PhD at 301–594–4263 or *hubbsa@mail.nih.gov* for more information.

Polyclonal Antibody Against Bloom's Syndrome Protein (BLM) for Research and Diagnostic Use

Description of Technology: Investigators at the National Institutes of Health have generated a polyclonal antibody against Bloom's syndrome protein (BLM). The BLM protein is a DNA helicase enzyme and a key component of the DNA damage response signaling pathway. Several protein kinases including ATM, DNA–PK, and ATR can mediate the phosphorylation of BLM. The polyclonal antibody is generated by using a phosphorylated peptide belonging to the N-terminus of BLM. The antibody shows a rapid phosphorylation of BLM on threonine 99 (T99p-BLM) following DNA damage by anti-cancer agents and could serve as a therapeutic marker of drug action on DNA. The antibody is also useful for microscopic and biochemical analysis of DNA damage signaling.

Applications:

- A therapeutic marker of drug action on DNA
- A diagnostic indicator of inherent genomic instability

Inventors: Yves Pommier and V. Ashutosh Rao (NCI)

Patent Status: HHS Reference No. E–053–2006/0—Research Tool. Patent protection is not being sought for this technology.

Licensing Status: Threonine 99 specific polyclonal antibody against the BLM protein is available for licensing.

Licensing Contact: Betty Tong, PhD; 301–594–6565; *tongb@mail.nih.gov*.

Dated: April 16, 2009.

Richard U. Rodriguez,

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

[FR Doc. E9–9345 Filed 4–22–09; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute on Deafness and Other Communication Disorders; Amended Notice of Meeting

Notice is hereby given of a change in the meeting of the National Institute on Deafness and Other Communication Disorders Special Emphasis Panel, April 28, 2009, 1 p.m. to April 28, 2009, 4 p.m., National Institutes of Health, Bethesda, MD which was published in the **Federal Register** on April 6, 2009, 7415501.

The meeting will be held April 29, 2009. The meeting is closed to the public.

Dated: April 15, 2009.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. E9–9204 Filed 4–22–09; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute on Drug Abuse; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. App.), 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.,