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.,