novo mutations in the *HRPT2* tumour suppressor gene in familial isolated hyperparathyroidism (FIHP),'' J. Med. Genet. (2004 Mar) 41(3):e32, doi: 10.1136/jmg.2003.012369.

Dated: May 4, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–10607 Filed 5–10–04; 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.

Mouse Model With Targeted Disruption of the Neurofibromatosis Type-1 (Nf1) Gene

Neal G. Copeland *et al.* (NCI).

DHHS Reference No. E–162–2004/0—Research Tool

Licensing Contact: Jesse S. Kindra; 301–435–5559; *kindraj@mail.nih.gov*.

This invention relates to a mouse model having a targeted disruption of the neurofibromatosis type–1 (NF1) gene. This mouse model is useful as a research tool in studying some forms of human neuron diseases/injuries in addition to juvenile chronic myelomonocytic leukemia (JMML).

The neurofibromatosis (NF1) gene shows significant homology to mammalian GAP and is an important regulator of the Ras signal transduction pathway. To study the function of NF1 in normal development and to develop a mouse model of NF1 disease, the inventors have used gene targeting in ES cells to generate mice carrying a null mutation at the mouse Nf1 locus. Although heterozygous mutant mice, aged up to 10 months, have not exhibited any obvious abnormalities, homozygous mutant embryos die in utero. Embryonic death is likely attributable to a severe malformation of the heart. Interestingly, mutant embryos also display hyperplasia of neural crest-derived sympathetic ganglia. These results identify new roles for NF1 in development and indicate that some of the abnormal growth phenomena observed in NF1 patients can be recapitulated in neurofibromin-deficient mice. In addition, lethally-irradiated wild type mice transplanted with fetal liver cells taken from NF1 null embryos develop a form of juvenile chronic myelomonocytic leukemia (JMML) that is very similar to what is seen in children with NF1 disease. This mouse model can be used to test various therapeutic treatments for this disease.

Novel Antisense Oligonucleotides Targeting Folate Receptor Alpha

Mona S. Jhaveri, Patrick C. Elwood, Koong-Nah Chung (NCI).

U.S. Patent Application No. 10/093,523 filed 11 Mar 2002, U.S. Pat. App. Pub. No. U.S. 2003/0050267 A1 (DHHS Reference No. E–321–2000/0–EIR–00).

Licensing Contact: Thomas P. Clouse; 301/435–4976; *clousetp@mail.nih.gov*.

Ovarian cancer is the fifth leading cause of cancer death for women in the United States. Drug resistance of ovarian tumors to chemotherapy is a common problem resulting in only 20 to 30 percent overall 5-year survival rates.

Folate is a vitamin that is required for cell survival. Some cancer cells, including ovarian carcinomas, have an abundance of a folate-binding protein termed the human alpha folate receptor (ahFR). It is believed that elevated levels of ahFR in cancer, relative to normal cells, contribute to the cellular malignant phenotype by mediating increased folate uptake or by generating positive regulatory growth signals.

This invention comprises a DNA-based therapy that selectively targets and diminishes the levels of ahFR using antisense oligonucleotides that block the transcription of the ahFR gene. Studies have shown that this invention significantly decreases proliferation of cultured cancer cells and sensitizes these cells to treatment with chemotherapeutic drugs. Further development of ahFR-targeted antisense oligonucleotides and related compounds has potential therapeutic value for a range of cancers that express increased levels of ahFR, including cancers of the ovary, cervix, uterus, and brain.

Dated: May 5, 2004.

Steven M. Ferguson,

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

[FR Doc. 04–10689 Filed 5–10–04; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer 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 Cancer Institute Special Emphasis Panel, Ovarian Cancer SPORE.

Date: May 18, 2004

Time: 8:30 a.m. to 5 p.m.

Agenda: To review and evaluate grant applictions.

Place: The Crystal City Marriott at National Airport, 1999 Jefferson Davis Highway, Arlington, VA 22202.

Contact Person: Olivia Preble Bartlett, PhD, Chief, Research Programs Review Branch, Division of Extramural Activities, National Cancer Institute, 8th Floor, Room 8121, 6116 Executive Boulevard, Rockville, MD 20892–7405. (301) 594–2501; *op2t@nih.gov*.

This notice is being published less than 15 days prior to the meeting due to the timing limitations imposed by the review and funding cycle.

(Catalogue of Federal Domestic Assistance Program Nos. 93.392, Cancer Construction; 93.393, Cancer Cause and Prevention Research; 93.394, Cancer Detection and Diagnosis Research; 93.395, Cancer Treatment Research; 93.396, Cancer Biology Research; 93.397, Cancer Centers Support; 93.398, Cancer Research Manpower; 93.399,