tumors, including breast cancer. Amplification is a common mechanism for upregulation of critical genes involved in cancer development and progression. Discovery of HER-2 oncogene amplification in breast cancer has led to a specific therapy for breast cancer patients with an activated HER-2 gene. Chromosomal region 17q23 is frequently amplified in breast cancer but the genes involved in this amplification are not yet known. Amplification of four previously known genes, S6K, TBX2, PAT1, RAD51C, has been identified in breast cancer cell lines and primary breast tumors. The amplification in cell lines leads to overexpression at the mRNA level. Thus, these genes represent putative targets for the 17q23 amplification and their upregulation may contribute to the genesis and progression of breast cancer.

**Inhibition of Cell Motility**

Donald P. Bottaro, Terrence R. Burke, Jr., Zhu-Jun Yao, Nese S. Atabay, Diane E. Breckenridge, Yang Gao (NCI)


Licensing Contact: Richard U. Rodriguez; 301/496–7056 ext. 287; e-mail: rodrigur@od.nih.gov

The present invention relates to a method of inhibiting cell motility induced by hepatocyte growth factor (HGF) and treating various diseases in a mammal. HGF stimulates mitogenesis, motogenesis and morphogenesis in a wide range of cellular targets including epithelial and endothelial cells, hematopoietic cells, neurons, melanocytes, and hepatocytes. These pleiotropic effects play important roles during development and tissue regeneration, but they are also implicated in several human cancers, including colon, breast, lung, thyroid and renal carcinomas, several sarcomas and gliomas. The ability of HGF to initiate a program of cell dissociation and increased cell motility coupled with increased protease production promotes aggressive cellular invasion and is linked to tumor metastasis. The methods of the present invention employ compounds, e.g., phosphotyrosine mimetics, to inhibit cell motility. A key advantage of this invention is that the peptides are free of cytotoxicity. Further development and use of this invention could serve a serious public need.

**Fibroblast Growth Factor-5 (FGF–5) Is a Tumor Associated T-cell Antigen for Human Renal Cell Cancer and Other Adenocarcinomas**

Ken-ichi Hanada and James C. Yang (NCI)


Licensing Contact: Elaine Gese; 301/496–7056 ext. 282; e-mail: gese@od.nih.gov

Renal cell carcinoma (RCC) is a form of kidney cancer caused when cells in the lining of the renal tubule undergo cancerous changes. The inventors have shown that fibroblast growth factor-5 (FGF–5) is a tumor associated antigen (TAA) for RCC and cancers of the breast and prostate. TAAs can be used to stimulate cytotoxic T-lymphocytes (CTL) which can be directed against specific tumor cells. This can be accomplished in at least two ways: (1) Activating a patient’s immune system by administering a vaccine containing the TAA, or (2) by removing a patient’s lymphoid cells, activating these cells ex vivo and then reintroducing these activated cells back into the patient to attack the tumor cells. The invention provides for methods of treating RCC and other adenocarcinomas with FGF–5 using the aforementioned approaches.

**Genetic System in Yeast for Functional Identification of Human p53 Mutations**

Michael A. Resnick, Alberto Inga (NIEHS)


Licensing Specialist: Vasant Gandhi; 301/406–7056 ext. 224; e-mail: gandhiv@od.nih.gov

The tumor suppressor gene p53, a key regulator of cellular mechanisms that maintain genome integrity, is the most commonly inactivated gene target associated with neoplastic transformation. About 50% of all human tumors express a mutated form of p53 and more than 80% of these mutations are missense, leading to single amino acid changes. This invention relates to human p53 mutations and identification methods using screening assays in the yeast Saccharomyces cerevisiae to functionally categorize expressed p53 mutant proteins. Additionally, the invention relates to methods of detecting or generating novel human p53 mutations with properties that can include toxicity in yeast and growth suppression in human cells, enhanced or reduced transactivation relative to wild type p53, altered promoter selectivity, and reactivation of common tumor mutations for the transactivation function of major p53 downstream genes. The invention also provides for screening of genetic factors, peptides and chemicals that mimic the toxic or supertransactivating mutations or inhibit p53 function.


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

[FR Doc. 00–19153 Filed 7–27–00; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Consensus Development Conference on Antenatal Corticosteroids Revisited: Repeat Courses

Notice is hereby given of the National Institutes of Health (NIH) Consensus Development Conference on “Antenatal Corticosteroids Revisited: Repeat Courses,” which will be held August 17–18, 2000, in Masur Auditorium of the NIH Clinical Center, 9000 Rockville Pike, Bethesda, Maryland, 20892. The conference begins at 8 a.m. on August 17, and at 8:30 a.m. on August 18 and is open to the public.

Preterm delivery is a major cause of death and illness in infants. Corticosteroid treatment of pregnant women delivering prematurely was first introduced in 1972 to enhance fetal lung maturity. Subsequent research has focused on the ability of glucocorticoids to reduce mortality and brain injury in preterm neonates. In 1994 the National Institutes of Health sponsored a Consensus Development Conference on the Effect of Corticosteroids for Fetal Maturation on Perinatal Outcomes to assess the effectiveness of antenatal glucocorticoid therapy. The Consensus Panel concluded, in part, that giving corticosteroids to pregnant women at risk for preterm delivery reduces the risk of death, respiratory distress syndrome, and intraventricular hemorrhage in preterm infants. The 1994 panel noted that optimal benefit of antenatal corticosteroid therapy last 7 days. The panel also noted that the potential benefits and risk of repeated administration of antenatal corticosteroids 7 days after the initial course are unknown and called for additional research on this issue.

The NIH is organizing this 1½ day conference to present research on repeat courses of antenatal corticosteroid therapy. After a day of presentations and audience discussion, an
independent, non-federal consensus development panel will weigh the scientific evidence and write a draft statement that will be presented to the audience on the second day. The panel’s statement will address these questions:

- Is the evidence on benefits and risks of repeat courses of antenatal corticosteroids sufficient to permit consensus recommendations?
- If so, what are the recommendations?
- If not, what additional information should be obtained?

On the final day of the conference, the panel’s draft statement will be read in public, at which time members of the public are invited to offer comments on the draft.

The primary sponsors of this meeting are the National Institute of Child Health and Human Development and the NIH Office of Medical Applications of Research. Co-sponsors include the National Institute of Nursing Research and the National Heart, Lung, and Blood Institute.

This is the 112th Consensus Development Conference held by the NIH in the 23-year history of the Consensus Development Program. Advance information about the conference and conference registration materials may be obtained from the NIH Consensus Program Web site—http://consensus.nih.gov. Conference information can also be obtained from Prospect Associates of Silver Spring, Maryland by calling (301) 592-3320 or by e-mail to antenatal@prospect.com. Prospect Associate’s address is 10720 Columbia Pike, Suite 500, Silver Spring, Maryland 20901-4437.


Ruth L. Kirschstein,
Acting Director, NIH.

[FR Doc. 00-19148 Filed 7-27-00; 8:45 am]
BILLING CODE 4140-01-M

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, Small Grants Program for Behavioral Research in Cancer Control.
Date: August 11, 2000.
Time: 8 am to 5 pm.

Agenda: To review and evaluate grant applications.
Place: Executive Plaza North, Conference Room J, 6130 Executive Plaza, Rockville, MD 20852.

Contact Person: C.M. Kerwin, PHD, Scientific Review Administrator, Special Review and Resources Branch, Division of Extramural Activities, National Cancer Institute, National Institutes Of Health, 6116 Executive Boulevard, Room 8086, Rockville, MD 20892-7405, 301/496-7421.

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, Cancer Control, National Institutes of Health, HHS)


LaVerne Y. Stringfield,
Director, Office of Federal Advisory Committee Policy.

[FR Doc. 00-19141 Filed 7-27-00; 8:45 am]
BILLING CODE 4140-01-M

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.

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Name of Committee: National Cancer Institute Special Emphasis Panel, Diet, Lifestyle and Cancer in U.S. Special Populations.