Dated: July 31, 2000.

Margaret M. Dotzel,

Associate Commissioner for Policy.

[FR Doc. 00–20343 Filed 8–10–00; 8:45 am]

BILLING CODE 4160–01–F

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Office of the Director; Notice of Meeting

Pursuant to Pub. L. 92–463, notice is hereby given of a meeting of the Advisory Committee to the Director, NIH.

The entire 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 inform the Contact Person listed below in advance of the meeting. The meeting will take place via conference call with the members. A speaker phone will be installed in the conference room for the public to listen to the discussion.

Name of Committee: Advisory Committee to the Director, NIH.

Date: September 14, 2000.

Time: 4–5 p.m.

Agenda: To discuss and provide advice on the final Report from the Working Group on NIH Oversight of Clinical Gene Transfer Research.

Place: National Institutes of Health, 1 Center Drive, Building 1, Room 151, Bethesda, Maryland 20892.

Contact: Ms. Janice C. Ramsden, Special Assistant to the Principal Deputy Director, NIH, National Institutes of Health, Building 1, Room 235, Bethesda, Maryland 20892, *jr52h@nih.gov*, Telephone: (301) 496–0959.

Dated: August 4, 2000.

LaVerne Y. Stringfield,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 00–20371 Filed 8–10–00; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute (NCI); Rational Design of Hepatocyte Growth Factor (HGF) Agonists and Antagonists

An opportunity for a Cooperative Research and Development Agreement (CRADA) is available for collaboration with the NCI intramural Structural Biophysics Laboratory 9SBL) to rationally design agonists and antagonists to hepatocyte growth factor (HGF). Collaborative projects will focus upon cancer and/or areas of infectious diseases of high public health significance and high national and international priority.

AGENCY: National Cancer Institute, National Institutes of Health, PHS, DHHS.

ACTION: Notice of an opportunity for Cooperative Research and Development Agreement (CRADA).

SUMMARY: Pursuant to the Federal Technology Transfer Act of 1986 (FTTA, 15 U.S.C. 3710; and Executive Order 12591 of April 10, 1987, as amended by the National Technology Transfer and Advancement Act of 1995), the National Cancer Institute (NCI) of the National Institutes of Health (NIH) of the Public Health Service (PHS) of the Department of Health and Human Services (DHHS) seeks one Cooperative Research and Development Agreement (CRADA) with a pharmaceutical or biotechnology company to rationally design agonists and antagonists to hepatocyte growth factor (HGF). The CRADA would have an expected duration of one (1) to five (5) years. The goals of the CRADA include the rapid publication of research results and timely commercialization of products, methods of treatment or prevention that may result from the research. The CRADA Collaborator will have an option to negotiate the terms of an exclusive or non-exclusive commercialization license to subject inventions arising under the CRADA and which are the subject of the CRADA Research Plan.

ADDRESSES: Proposals and questions about this CRADA opportunity may be addressed to Jeffrey W. Thomas, Technology Development and Commercialization Branch, National Cancer Institute-Frederick Cancer Research and Development Center, Fairview Center, Room 502, Frederick, MD 21701 (phone: 301–846–5465; fax: 301–846–6820; e-mail: jeffreyt@mail.nih.gov).

Scientific inquiries should be submitted to Dr. R. Andrew Byrd, Chief, Structural Biophysics Laboratory, National Cancer Institute-Frederick Cancer Research and Development Center, Bldg. 538, Room 120, Frederick, MD 21702–1201 (phone: 301–846–1407; Fax: 301–846–6231; e-mail rabyrd@ncifcrf.gov).

EFFECTIVE DATE: Inquiries regarding CRADA proposals and scientific matters may be forwarded at any time. Confidential, preliminary CRADA proposals, preferably two pages or less, must be submitted to the NCI on or before September 11, 2000. Guidelines for preparing final CRADA proposals will be communicated shortly thereafter to all respondents with whom initial confidential discussions will be have established sufficient mutual interest.

SUPPLEMENTARY INFORMATION:

Technology Available

The Structural Biophysics Laboratory, DBS, NCI is seeking a collaborative partner to pursue the rational design of hepatocyte growth factor (HGF) agonists and antagonists. HGF is a secreted, heparin-binding protein that stimulates mitogenesis, motogenesis, and morphogenesis in a wide spectrum of cellular targets including epithelial, endothelial, and hematopoietic cells, as well as hepatocytes. HGF and its receptor, c-Met, are essential for embryonic development, and HGF signaling contributes to tissue repair and organ homeostasis throughout adulthood. Inherited activating mutations in c-Met are associated with renal papillary carcinoma, and ligand-stimulated pathway activation has been implicated in the growth, neovascularization, invasiveness, and metastasis of several other human tumors. The restorative, as well as the deleterious potential of this pathway make it a promising target for therapeutic intervention against several degenerative and neoplastic diseases. The HGF gene encodes full-length HGF, and two truncated isoforms (NK1 and NK2) which consist of the N-terminal domain (N) linked to the first one (K1) or two (K1+K2) kringle domains. Both truncated isoforms are motogenic; NK1 also retains the mitogenic and morphogenic potency of HGF, while NK2 is a competitive antagonist of these activities. The primary heparin and receptor binding sites of HGF reside in the N and K1 domains, respectively. Three dimensional structures of N and NK1 obtained using NMR spectroscopy and X-ray crystallography suggest that ligand dimerization, augmented by heparin binding, may facilitate receptor activation. This information provides the basis for [1] determining the solution structure of an NK1-heparin complex; [2] locating K2 in NK2 to learn the structural basis for its antagonistic properties; [3] identifying receptor binding residues in K1, [4] creating NK1 and NK2 mutants with altered heparin and receptor binding properties, and [5] assessing these proteins as activators or inhibitors of HGF signaling using cultured cells and intact animals. Achieving these goals will help elucidate the mechanism by which HGF