Committee Names	Dates of Meetings
Anti-Infective Drugs Advisory Committee	March 24, September 11–12
Antiviral Drugs Advisory Committee	July 20–21
Arthritis Advisory Committee	April 11, June 8–9, September 11–12, November 9–10
Cardiovascular and Renal Drugs Advisory Committee	May 1–2, July 20–21, October 19–20
Dermatologic and Ophthalmic Drugs Advisory Committee	May 4–5
Drug Abuse Advisory Committee	October 19–20
Endocrinologic and Metabolic Drugs Advisory Committee	May 18–19, July 13–14, October 5–6, December 7–8
Gastrointestinal Drugs Advisory Committee	April 12
Medical Imaging Drugs Advisory Committee	May 22–23, October 30–31
Nonprescription Drugs Advisory Committee	June 22–23, July 13–14, October 19–20, December 7–8
Oncologic Drugs Advisory Committee	March 16–17, June 5–6
Peripheral and Central Nervous System Drugs Advisory Committee	October 26
Pharmacy Compounding Advisory Committee	May 15–16
Psychopharmacologic Drugs Advisory Committee	June 28–29, November 2–3
Pulmonary-Allergy Drugs Advisory Committee	November 6–7
CENTER FOR FOOD SAFETY AND APPLIED NUTRITION	
Food Advisory Committee	September 14–15
CENTER FOR DEVICES AND RADIOLOGICAL HEALTH	
Device Good Manufacturing Practice Advisory Committee	No meetings planned
Medical Devices Advisory Committee	
Anesthesiology and Respiratory Therapy Devices Panel	May 25–26, September 7–8, November 2–3
Circulatory System Devices Panel	May 2–3, September 25–26
Clinical Chemistry and Clinical Toxicology Devices Panel	March 24, June 29–30, September 14–15, December 14–15
Dental Products Panel	April 6–7, May 23–24, July 18–19, October 3–4
Ear, Nose, and Throat Devices Panel	May 26, June 23, July 20–21, September 22
Gastroenterology-Urology Devices Panel	April 13–14, August 31–September 1, November 30–December 1
General and Plastic Surgery Devices Panel	June 12–13, September 11–12, December 4–5
General Hospital and Personal Use Devices Panel	May 1–2, August 7–8, November 6–7
Hematology and Pathology Devices Panel	June 12, August 8, November 7
Immunology Devices Panel	June 16, September 15, December 8
Medical Devices Dispute Resolution Panel	To be determined
Microbiology Devices Panel	June 21–22, November 16–17
Molecular and Clinical Genetics Panel	June 23, September 15, December 15
Neurological Devices Panel	March 31, May 11–12, August 17–18, November 16–17
Obstetrics-Gynecology Devices Panel	April 10–11, July 24–25, October 9–10
Ophthalmic Devices Panel	March 17, May 11–12, July 27–28, September 21–22, November 8–9
Orthopaedic and Rehabilitation Devices Panel	March 18, May 4–5, August 24–25, November 16–17
Radiological Devices Panel	May 15, August 14, November 6
National Mammography Quality Assurance Advisory Committee	July 10, December 11
Technical Electronic Product Radiation Safety Standards Committee	June 21–22
CENTER FOR VETERINARY MEDICINE	
Veterinary Medicine Advisory Committee	September 15
NATIONAL CENTER FOR TOXICOLOGICAL RESEARCH	
Advisory Committee on Special Studies Relating to the Possible Long-Term Health Effects of Phenoxy Herbicides and Contaminants	No meetings planned
Science Board to the National Center for Toxicological Research	May 1–2

Dated: March 17, 2000.

Linda A. Suydam,

Senior Associate Commissioner.

[FR Doc. 00–7429 Filed 3–24–00; 8:45 am]

BILLING CODE 4160–01–F

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute: Opportunity for a Cooperative Research and Development Agreement (CRADA) for the Research, Purification, and Further Development of a Factor(s) That Inhibits Human Immunodeficiency Virus (HIV) Replication

AGENCY: National Institutes of Health, PHS, DHHS.

ACTION: Notice.

SUMMARY: The National Cancer Institute (NCI) seeks a Cooperative Research and Development Agreement (CRADA) Collaborator to aid NCI in the further characterization and commercial development of a factor(s) that inhibits the replication of the Human Immunodeficiency Virus (HIV). NCI recently discovered that leukocytes stimulated with infectious or ultraviolet-inactivated influenza A virus produce a factor(s) that inhibits the replication of both CCR5- and CXCR4-tropic HIV–1 viral isolates. The factor(s) inhibits replication of the virus after

The National Cancer Institute's Experimental Immunology Branch has identified a factor that is produced by leukocytes when exposed to influenza virus which inhibits HIV replication.

viral binding but prior to reverse transcription. NCI has performed the initial characterization of the HIV–1 replication-inhibiting factor(s). The discovery of this factor(s) raises the possibility that immunization with recombinant influenza viral constructs and/or ultraviolet (UV)-inactivated influenza offers an immune-based therapeutic strategy that could be used to treat HIV-infected patients. NCI is looking for a CRADA Collaborator with a demonstrated record of success in protein purification and HIV therapeutics for the eventual use of this factor(s) in the clinical treatment of patients suffering from Acquired Immunodeficiency Syndrome (AIDS). The proposed term of the CRADA can be up to five (5) years.

DATES: Interested parties should notify this office in writing of their interest in filing a formal proposal no later than May 26, 2000. Potential CRADA Collaborators will then have an additional thirty (30) days to submit a formal proposal. CRADA proposals submitted thereafter may be considered if a suitable CRADA Collaborator has not been selected.

ADDRESSES: Inquiries and proposals regarding this opportunity should be addressed to Holly Symonds Clark, Ph.D., Technology Development Specialist (Tel. # 301–496–0477, FAX # 301–402–2117), Technology Development and Commercialization Branch, National Cancer Institute, 6120 Executive Blvd., Suite 450, Rockville, MD 20852. Inquiries directed to obtaining patent license(s) for the technology described in U.S. Provisional Patent Application Serial No. 60/162,262, filed October 29, 1999 for ''Leukocyte-Derived Anti-Viral Factors'' (Shearer *et al.*) (NCI), should be addressed to J.P. Kim, J.D., M.B.A., Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Blvd., Suite 325, Rockville, MD 20852, (Tel. 301–496–7056, ext. 264; FAX 301–402–0220).

SUPPLEMENTARY INFORMATION: A Cooperative Research and Development Agreement (CRADA) is the anticipated joint agreement to be entered into with NCI pursuant to the Federal Technology Transfer Act of 1986 and Executive Order 12591 of April 10, 1987 as amended by the National Technology Transfer Advancement Act of 1995. NCI is looking for a CRADA partner to aide NCI in the characterization and commercial development of the HIV replication-inhibiting factor. The expected duration of the CRADA would be from one (1) to five (5) years.

NCI has discovered a system in which leukocytes can produce an anti-HIV factor following exposure to an influenza virus. Specifically, NCI has found that the factor or factors secreted by the leukocytes inhibit retroviral replication prior to reverse transcription and formation of the provirus. The influenza virus to which the leukocytes are exposed causing them to generate anti-HIV activity include infectious influenza virus and UV-inactivated influenza virus. NCI has found that exposure of the leukocytes to the influenza virus can inhibit viral isolates that use different coreceptors for binding CD4.

The generation of the influenza-stimulated anti-HIV factor(s) can be mediated in the absence of CD4+ or CD8+ cells, and it does not appear to require the presence of both subsets. Thus, it is possible that the anti-HIV factor could be produced in patients exhibiting low CD4 counts. NCI has determined that the anti-HIV factor(s) presently claimed do not include several of the known chemokines or cytokines.

NCI predicts that the influenza-stimulated anti-HIV factor(s) offers the following advantages: 1. The anti-HIV activity appears to be independent of the presence of both CD4+ and CD8+ cells and of ability to generate strong T cell proliferative responses to flu, as well as of influenza-stimulated production of the Th1 cytokine, IFN-gamma. 2. Influenza-stimulated peripheral blood mononuclear cells (PBMCs) from HIV+ patients can generate anti-HIV activity that is as potent as cells from HIV-donors, and this activity appears to be independent of a patient's T helper responses to influenza. 3. Flu-stimulated anti-HIV–1 activity is broadly reactive in that it inhibits HIV–1 isolates that use different coreceptors for entry, and is therefore not a beta-chemokine. 4. NCI's demonstration that inhibition occurs prior to HIV reverse transcription distinguishes it from the CD8 anti-viral factor (CAF), which inhibits at transcription. 5. The fact that UV-inactivated flu can stimulate anti-HIV activity indicates the potential clinical feasibility of immunizing HIV+ patients. NCI believes that the utilization of an attenuated form of live influenza virus might represent the best form of immunization to HIV–1.

The described methods are the subject of U.S. Provisional Patent Application Serial No. 60/162,262, filed on October 29, 1999 by the Public Health Service on behalf of the Federal Government. Furthermore, the initial report and characterization of the invention is described in: J. Virol., in press, May 2000.

Under the present proposal, the goal of the CRADA will be to enhance the development of the influenza-stimulated, anti-HIV factor(s) in the following areas:

1. Further purification and characterization of the factor(s).
2. Determination of the factor's mechanism of viral replication inhibition.
3. Determination as to whether or not the factor(s) is unique by cloning and sequencing the gene.
4. Utilization of the SIV/macaque model to determine efficacy of flu-based therapy.
5. Development of clinical trials to test the efficacy of the flu-based therapy.

Party Contributions

The role of the NCI in the CRADA may include, but not be limited to:

1. Providing intellectual, scientific, and technical expertise and experience to the research project.
2. Providing the CRADA Collaborator with information and data relating to the influenza-stimulated, anti-HIV factor(s).
3. Planning research studies and interpreting research results.
4. Carrying out research to validate the anti-viral activities of the influenza-stimulated factor(s).
5. Publishing research results.
6. Developing additional potential applications of the factor(s).

The role of the CRADA Collaborator may include, but not be limited to:

1. Providing significant intellectual, scientific, and technical expertise or experience to the research project.
2. Planning research studies and interpreting research results.
3. Providing technical and/or financial support to facilitate scientific goals and for further design of applications of the technology outlined in the agreement.
4. Publishing research results.

Selection criteria for choosing the CRADA Collaborator may include, but not be limited to:

1. A demonstrated record of success in the areas of protein purification, characterization and therapeutic development.
2. A demonstrated background and expertise in immunological sciences and AIDS therapeutics.
3. The ability to collaborate with NCI on further research and development of this technology. This ability will be demonstrated through experience and expertise in this or related areas of technology indicating the ability to contribute intellectually to ongoing research and development.

4. The demonstration of adequate resources to perform the research and development of this technology (*e.g.* facilities, personnel and expertise) and to accomplish objectives according to an appropriate timetable to be outlined in the CRADA Collaborator's proposal.

5. The willingness to commit best effort and demonstrated resources to the research and development of this technology, as outlined in the CRADA Collaborator's proposal.

6. The demonstration of expertise in the commercial development and production of products related to this area of technology.

7. The level of financial support the CRADA Collaborator will provide for CRADA-related Government activities.

8. The willingness to cooperate with the National Cancer Institute in the timely publication of research results.

9. The agreement to be bound by the appropriate DHHS regulations relating to human subjects, and all PHS policies relating to the use and care of laboratory animals.

10. The willingness to accept the legal provisions and language of the CRADA with only minor modifications, if any. These provisions govern the distribution of future patent rights to CRADA inventions. Generally, the rights of ownership are retained by the organization that is the employer of the inventor, with (1) the grant of a license for research and other Government purposes to the Government when the CRADA Collaborator's employee is the sole inventor, or (2) the grant of an option to elect an exclusive or nonexclusive license to the CRADA Collaborator when the Government employee is the sole inventor.

Dated: March 20, 2000.

Karen Maurey,

Deputy Chief, Technology Development and Commercialization Branch, National Cancer Institute, National Institutes of Health.

[FR Doc. 00–7380 Filed 3–24–00; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute: Opportunity for a Cooperative Research and Development Agreement (CRADA) for the Screening, Development and Commercialization of Novel Inhibitors of GADD45 Polypeptide Activity for the Treatment of Cancer

AGENCY: National Institutes of Health, PHS, DHHS.

ACTION: Notice.

The National Cancer Institute's Laboratory of Human Carcinogenesis (LHC) has created and characterized *in vitro* and *in vivo* methods designed to screen for modulators of GADD45 polypeptide activity. Furthermore, LHC has developed methods for sensitizing proliferating cells to DNA damaging agents by inhibiting GADD45 polypeptide activity. Identification of novel inhibitors of GADD45 using LHC's screening assays would provide potential new treatments for cancer.

SUMMARY: The National Cancer Institute (NCI) seeks a Cooperative Research and Development Agreement (CRADA) Collaborator to aid NCI in the screening, development and commercialization of novel compounds for the treatment of cancer. These methods focus on the identification of small molecule inhibitors of GADD45 polypeptide activity.

NCI has developed a series of *in vitro* and *in vivo* assays to screen for modulators of GADD45 polypeptide activity. These assays may identify novel small molecule inhibitors of GADD45 activity that, when used in conjunction with current chemotherapeutics, reduce the toxicity of and enhance the effectiveness of current treatments of cancer. NCI is looking for a CRADA Collaborator with a demonstrated record of success in cancer diagnostics and therapeutics. The proposed term of the CRADA can be up to five (5) years.

DATES: Interested parties should notify the Technology Development and Commercialization Branch of the NCI in writing of their interest in filing a formal proposal no later than May 26, 2000. Potential CRADA Collaborators will then have an additional thirty (30) days to submit a formal proposal. CRADA proposals submitted thereafter may be considered if a suitable CRADA Collaborator has not been selected.

ADDRESSES: Inquiries and proposals regarding this opportunity should be addressed to Holly Symonds Clark, Ph.D., Technology Development Specialist (Tel. # 301–496–0477, FAX # 301–402–2117), Technology Development and Commercialization Branch, National Cancer Institute, 6120 Executive Blvd., Suite 450, Rockville, MD 20852. Inquiries directed to obtaining patent license(s) for the technology described in U.S. Provisional Patent Application Serial No. 60/126,069, filed March 25, 1999, for ''Methods for Identifying Modulators of GADD45 Polypeptide Activity'' (Harris *et al.*) should be addressed to Vasant Gandhi, J.D., Ph.D., Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Blvd., Suite 325, Rockville, MD 20852, (Tel. 301–496–7056; FAX 301–402–0220).

SUPPLEMENTARY INFORMATION: A Cooperative Research and Development Agreement (CRADA) is the anticipated joint agreement to be entered into with NCI pursuant to the Federal Technology Transfer Act of 1986 and Executive Order 12591 of April 10, 1987 as amended by the National Technology Transfer Advancement Act of 1995. NCI is looking for a CRADA partner to collaborate with NCI in the further development and commercialization of screening assays and methods relating to the analysis of small molecule inhibitors of GADD45 polypeptide activity. The expected duration of the CRADA would be from one (1) to five (5) years.

Mammalian cells cycle through a series of ordered stages that involve various cellular components during normal cellular growth (for reviews: 1, 2). A normal cell can arrest cell cycle progression when DNA damage is incurred. Cell cycle ''checkpoints'' exist at two different stages in cell cycle progression: the G1 to S (replication) stage and the G2–M (mitosis) stage. These checkpoints are essentially stages in which the cell ''stalls'' its cell cycle to repair any damaged DNA that may exist prior to entry into mitosis. The G2–M checkpoint prevents the improper segregation of chromosomes likely to be important in human tumorigenesis (3, 4). The G2-specific kinase composed of Cdc2 and cyclin B1 is a regulator of the cell cycle transition from G2 to M (1). NCI has recently reported the identification of one of the gene products that controls the G2–M checkpoint: the ubiquitously expressed polypeptide, GADD45. GADD45 was originally identified on the basis of its rapid transcriptional induction following ultraviolet (UV) irradiation (5). Induction of GADD45 has also been observed following various types of pathological stimuli including various environmental stresses, hypoxia, IR, genotoxic drugs and growth factor withdrawal (6). The GADD45-induced G2/M checkpoint is at least in part mediated through inactivation of the Cdc2/cyclin B1 kinase (1).

NCI believes that the GADD45-mediated G2–M checkpoint could be a new target for the development of anti-cancer agents. Inhibitors of GADD45 activity at the G2–M checkpoint could destroy the cell's ability to stall its proliferative cycle to correct damaged DNA. Cancer cells are often deficient in the G1–S checkpoint, thus, the G2–M