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FY 07 President's Budget for NCI Highlighted at NCAB Meeting

NCI leadership presented the institute's proposed budget for fiscal year 2007 to the National Cancer Advisory Board (NCAB) at today's meeting. President Bush's FY 2007 budget request to Congress this week included a request of $4.754 billion for NCI—a decrease of $39.7 million from the funds available for the current year (See chart, above). For the entire National Institutes of Health (NIH), the funds available are $28.6 billion, the same amount from FY 2006.

Key Components of FY 07 President's Budget Request for NCI

	FY 2006	FY 2007	Change	
Appropriation	$4,841,774	$4,753,609		
1% Rescission	-48,418			
Adjusted Appropriation	4,793,356	4,753,609	-$39,747	-0.8%
Contained within the Budget:				
NIH Roadmap Contribution	42,834	57,382	14,548	34.0%
AIDS Research	253,666	244,104	-9,562	-3.8%

(dollars in thousands)

Among the policies and initiatives included within the President's request are several with direct implications for NCI: *(continued on page 2)*

Director's Update

Guest Update by Dr. John E. Niederhuber

Clinical Trial Program Restructuring Advancing Quickly

A new NCI organizational structure, designed to oversee the institute's entire clinical trials enterprise, was unveiled today at the NCAB meeting.

The reorganization is the direct result of recommendations issued last June by the Clinical Trials Working Group (CTWG). At the NCAB meeting, Dr. James Doroshow, director of the Division of Cancer Treatment and Diagnosis and co-chair of CTWG, described the structural components that will support this effort, which include the Clinical Trials Advisory Committee (CTAC), Clinical Trials Operations Committee (CTOC), and the Coordinating Center for Clinical Trials (CCCT).

CTAC is an external oversight committee, governed by the provisions of the Federal Advisory Committee Act, that will advise the NCI Director on the institute's Clinical Trials Program and will include members of NCAB as well as other NCI advisory boards and additional cancer clinical trials experts. CTAC will oversee implementation of CTWG initiatives, including a review of the system to evaluate and measure the effects of the implementation. CTAC also will *(continued on page 2)*

(FY 2007 Budget continued from page 1)

- Genes, Environment and Health study—$7.8 million is included within the NCI budget to be a part of the overall NIH initiative
- "Pathway to Independence" training awards—an NIH $15 million initiative of which $1.8 million is included within the NCI budget
- Average cost of competing Research Project Grants will remain the same average as in FY 2006
- Inflationary increases for direct recurring costs in noncompeting continuation awards will not be provided, except for cases in which NCI has committed to a programmatic increase
- NRSA stipend levels will remain at the FY 2006 level

NCI Director Dr. Andrew C. von Eschenbach commented, "We are committed, as we go through this period of making difficult choices between those programs, that we will always put scientific excellence as the critical and number one criteria in making those fiscal decisions." However, he continued, in addition to scientific merit, "we must also make those decisions within the context of strategic priorities." One priority is to maintain the "pipeline for the development of our intellectual capital, particularly in the development of young scientists and new investigators across the continuum of basic science to clinical research," he said.

NCI will also "work very hard going forward to leverage our resources and to find opportunities where we can partner and collaborate with other agencies, NIH institutes, and other sectors of the cancer research community," Dr. von Eschenbach added.

Appropriation hearings on the President's FY 2007 budget request are scheduled to begin on March 15 in the Senate and March 16 in the House of Representatives.

NCI management also provided updates to NCAB on several major initiatives. A report for implementation of the Clinical Trials Working Group (CTWG) recommendations was presented by Dr. James Doroshow, director of the Division of Cancer Treatment and Diagnosis (DCTD). The plan is discussed in the *NCI Director's Update* in this issue of the *NCI Cancer Bulletin*.

NCI Deputy Director Dr. Anna Barker also provided a status report on The Cancer Genome Atlas (TCGA) pilot project being conducted as a partnership between NCI and the National Human Genome Research Institute (NHGRI) to assess the feasibility of sequencing genomic changes in human tumors on a large scale. NCI and NHGRI launched the pilot project on December 13.

Dr. Robert Croyle, director of the Division of Cancer Control and Population Sciences (DCCPS), presented examples of current issues in tobacco control research. Some of the collaborations between NCI and the U.S. Centers for Disease Control and Prevention (CDC) were highlighted by Dr. Corinne Husten, acting director of CDC's Office on Smoking and Health.

The webcast of the public sessions of this NCAB meeting can be viewed at http://videocast.nih.gov. ◆

By Bill Robinson

(Director's Update continued from page 1)

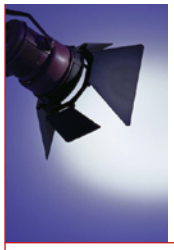
provide advice on the use of "correlative science funds," additional funding allotted to specific clinical trials for correlative science and quality-of-life studies.

CTOC is an internal NCI committee, chaired by the Deputy Director for Clinical and Translational Sciences, that includes the directors of every NCI division, branch, or center involved in clinical trials. Based in the NCI Director's Office, CTOC will coordinate clinical trials programs across NCI and will make recommendations to improve the cost-effectiveness and reduce duplication and overlap among NCI components involved in clinical trials. CTOC will also evaluate new Requests for Applications and Program Announcements for clinical trials prior to review by the NCI Executive Committee

Project management for the implementation of all CTWG initiatives will be provided by the Coordinating Center for Clinical Trials (CCCT). CCCT will support a number of significant activities, including coordinating new disease-specific steering committees for prioritization of phase III trials, the investigational drug steering committee for phase I and II trials, and working groups that will develop critical new tools for clinical investigators, as well as measures to improve clinical trial operational efficiency.

Two disease-specific cancer steering committees have begun to take shape. On January 26, the gastrointestinal steering committee met for the first time at the American Society of Clinical Oncology Gastrointestinal Cancer Symposium. The second steering committee, for gynecologic malignancies, began discussions at last month's Gynecologic Oncology Group meeting.

The CTWG implementation plan will help to integrate successful, but functionally diverse, elements of the current clinical trials enterprise. By embracing this restructuring, NCI and the cancer community will be positioned to ensure that advances in our understanding of the biological basis of cancer are rapidly translated into improved patient outcomes. ◆



Spotlight

Capturing Biomarkers of Melanoma's Progression

Skin cancers are the most common of all cancers. One in five Americans will develop skin cancer in their lifetime—about a million of us each year. More than 95 percent of these are basal cell or squamous cell carcinomas—nonmalignant moles or nevi—which can easily be removed by surgery or treatment.

Melanoma, the most deadly form of skin cancer, evolves through various stages from a benign mole, to a primary tumor, and eventually to a metastatic lesion. These stages can be recognized by unique patterns of gene activity, suggesting that melanoma progression can be studied and staged as a series of distinct molecular events.

Increasingly, molecular profiling data are collected for solid tumors such as those associated with breast and colon cancers to identify biomarkers that will be helpful in early detection. Similar approaches for the field of skin cancer research are lagging behind. There is a need for human skin cancer biopsies because the lesions are small and often removed in a setting outside an oncology practice—in a dermatologist's or family physician's office.

This barrier to biomarker research in melanoma was discussed in mid-January at a conference in Santa Fe, N.M. The "Markers of Tumor Progression, Diagnosis and Prognosis" session highlighted the need for tissue samples from melanoma patients.

Biomarker issues also were on the minds of more than 50 prominent melanoma researchers last October, when they met in Gaithersburg, Md., for the "Resources for Melanoma Research Workshop," according to Dr. Magdalena Thurin, program director in NCI's Cancer Diagnosis Program (CDP). The conclusions from this meeting urged the research community to establish a high-quality melanoma tissue sample resource to support discovery and development research. "Without a more reliable source of well-characterized human melanoma tumor tissue for systematic and meaningful studies, we can't pursue the most urgent issues in melanoma research," says Dr. Thurin.

CDP and the Specialized Programs of Research Excellence (SPORE) skin program at NCI addressed this issue by arranging for more active collection of the tissues that would be needed to build tissue micorarrays (TMAs) that will contribute to the discovery of biomarkers for melanoma. The Tissue Array Research Program (TARP), within NCI's Center for Cancer Research (CCR) began to assemble TMA slides consisting of a systematic grid of paraffin-embedded tissue taken from tumors in a number of different patients.

Scientists use TMAs to determine whether an immunohistochemical assay or antibody for their candidate biomarker is present. TMAs provide researchers "a high-throughput laboratory platform to explore the molecular features of the progression pathway, from a nevus all the way to metastatic melanoma," says Dr. Stephen Hewitt, TARP's manager.

"We saw a chance to bring some rational order to the process of supplying quality tissue to foster melanoma research," he says. "From NCI's point of view, everyone is on the same team." A second melanoma TMA was produced as a SPORE-sponsored effort.

"Newly improved high-throughput genomic and proteomic approaches are beginning to make inroads into the molecular characterization of this disease," says Dr. Dorothea Becker, a member of the University of Pittsburgh Melanoma Center and co-chair of the October workshop. "A number of promising genes, proteins, and other candidate biomarkers have emerged in the last few years that may help to predict and assess the risk of developing melanoma, serve to identify melanoma at an early stage, and become good targets for much-needed new therapies for advanced-stage melanomas."

The NCI-supported first-generation TMAs "are only a good beginning," says Dr. Hewitt. "This resource should help move things forward, but what we really need to do is partner with private practitioners, dermatologists, local oncologists, and melanoma patients to develop a strategy to obtain high-quality, well-annotated tissue samples that can be used for basic and discovery research."

The October 2005 workshop produced a list of the 33 most promising biomarker candidates. Each has shown some promise as a way to detect or treat melanoma, or to provide insights into progression or an early warning of metastasis. ◆

By Addison Greenwood

Cancer Research Highlights

Low-Fat Diet May Have Small Impact on Breast Cancer in Women

Results from the Women's Health Initiative (WHI) Dietary Modification (DM) prevention study show that reducing dietary fat and increasing fruits, grains, and vegetables may reduce risk of invasive breast cancer in some women, but has no effect on invasive colorectal cancer. Although more than 19,500 postmenopausal women followed the modified diet, the overall breast cancer risk reduction of 9 percent was not statistically significant after an average of 8.1 years. Women whose diets were highest in fat before they entered the study, however, were 22 percent less likely to develop breast cancer than the comparison group.

Dr. Ross L. Prentice of the Fred Hutchison Cancer Research Center in Seattle and colleagues write that the risk reduction seen in this and other subgroups "would not be expected if the intervention had no effect on breast cancer risk." Citing other trends in the data in favor of DM, they note the benefit increased to 15 percent among women who most closely followed the dietary regimen. Also, citing the small impact of DM on breast cancer, which begins to occur after about 4 years and appears to be increasing, the authors note that "the health implications of a low-fat dietary pattern may take years to be fully realized." In an accompanying editorial, Dr. Aman U. Buzdar from the University of Texas M.D. Anderson Cancer Center said the study is another indication that breast cancer oncologists "are beginning to understand which approaches may be effective for particular subsets of patients."

Dr. Leslie Ford of NCI's Division of Cancer Prevention noted that "Breast cancer is an exceedingly complex disease. The more we learn about the molecular underpinnings of the disease, the better we will understand how a healthy eating pattern and exercise may contribute to a reduction in risk for some women."

Regarding the colorectal study, lead author Dr. Shirley A.A. Beresford of the University of Washington in Seattle and colleagues also say that currently planned longer follow-up "may reveal delayed benefit," though, unlike the breast cancer study, "no time trends…have been seen."

The WHI DM study is the largest randomized controlled clinical trial of low-fat dietary interventions ever conducted. Results were published in the February 8 *Journal of the American Medical Association*.

Leukemia Risk after Hodgkin Lymphoma Reduced in Recent Decades

The risk of developing acute myeloid leukemia (AML) after treatment for Hodgkin lymphoma (HL) has decreased over the last three decades. In the early 1980s, the chemotherapy regimen changed in a way that may have influenced the development of secondary AML in HL survivors, a new study reports.

To determine whether patients treated after 1984 have decreased risks for AML, the researchers analyzed data on more than 35,000 1-year Hodgkin lymphoma survivors who were reported to population-based cancer registries in North America and in Nordic countries between 1970 and 2001.

Of the survivors, 217 developed AML. The risks were higher for patients over age 35 at the time of their HL treatment, and if that treatment occurred between 1970 and 1984 (versus 1985–2001). The decline over time in the risk of secondary AML was particularly apparent among HL survivors who initially received any chemotherapy, the researchers report in the February 1 *Journal of the National Cancer Institute* (*JNCI*).

The analysis showed that excess absolute risk for secondary AML was highest during the first 10 years after HL diagnosis but remained elevated thereafter. Excess absolute risk is the risk that occurs in addition to the "background" risk for a disease that exists in the general population.

In the analysis, Dr. Lois B. Travis of NCI and colleagues simultaneously evaluated the effects of age, calendar year of and time since HL diagnosis, and initial course of treatment. More research is needed to correlate decreases in risk of secondary AML with changes in HL therapy because population-based cancer registries do not contain detailed data on treatment regimens or information on subsequent therapy.

The findings "demonstrate an overall reduction, although not elimination, in the burden of AML over calendar year time," the researchers write. "This likely reflects in part the changes in chemotherapy (given initially or at relapse) that were implemented over the last few decades."

(continued on page 5)

(Highlights continued from page 4)

IL-12 Demonstrates Unique Capabilities in Neuroblastoma Mouse Model

Immunotherapy using the cytokine interleukin 12 (IL-12) in mouse models of advanced neuroblastoma induced complete tumor regression and long-term survival in the majority of mice, NCI researchers report. In this study, the team discovered that IL-12 achieved this regression not only by improving the immune system's ability to recognize tumor cells, but also by potently inhibiting a key protein that supports tumor survival.

"This is the first evidence, to our knowledge, that IL-12 can counteract this mechanism of tumor self-defense," said the study's senior author, Dr. Jon Wigginton, of the Pediatric Oncology Branch in NCI's CCR.

In the study, published in the February 1 *JNCI*, the NCI team, led by Dr. Tahira Khan, discovered that IL-12 treatment inhibits the activation of Akt, a "prosurvival" protein that helps tumor cells avoid cell death, or apoptosis.

To conduct the study, the research team delivered scheduled doses of IL-12 via intraperitoneal injection to cohorts of mice bearing primary and/or metastatic neuroblastomas. In addition to assessing tumor regression and survival, the researchers treated other mice identically with IL-12 to determine IL-12's impact on the expression of pro-apoptotic genes, phosphorylated/activated Akt, and overall tumor cell apoptosis.

IL-12 administration was associated with increases in circulating levels of several pro-apoptotic cytokines, as well as potent inhibition of Akt's "prosurvival" activity. In addition, there was marked tumor cell apoptosis within the tumor microenvironment.

According to Dr. Khan, the data provide a sound preclinical rationale for the investigation of IL-12 in children with neuroblastoma and suggest that IL-12 might be an excellent choice for combination therapy with specific targeted Akt inhibitors.

Researchers Use Systems Biology Approach to Cancer Drug Development

As researchers gain a greater understanding of the complexity of the molecular events leading to the development of cancer, more interest has been shown in using systems-biology approaches to develop targeted cancer therapeutics. These approaches treat cancer as a complex biological system that can be modeled to provide predictions of behavior, prognosis, and response to therapy. Two papers published in the January 19 *Nature* highlight results obtained from systems-biology-based examinations of cell-signaling deregulation.

One study, from the NCI-funded Integrative Cancer Biology Program (ICBP) center at Duke University, examined whether a cancer cell's unique gene expression signature can indicate which oncogenic signaling pathways are activated, and if that signature can be used to guide treatment targeting those pathways.

The researchers used recombinant adenoviruses to activate known oncogenic pathways in human cell cultures, and detected the expressed genes that were highly correlated with each active pathway by using microarray analysis. These gene-expression signatures were then tested in mouse models with similar oncogenic pathway mutations. The signatures determined from human cells correlated with the matching mutations in the mouse models.

The signatures were then used to predict pathway deregulation in a series of breast cancer cell lines before treatment with drugs targeting those pathways. In each case there was a close correlation between the probability of pathway deregulation and the response to the targeted drugs.

A second paper in the same issue of *Nature* examined the variations in mutations of a known oncogenic pathway. A team lead by investigators from Memorial Sloan-Kettering Cancer Center, and including Dr. Todd Golub, ICBP center director at Dana Farber Cancer Institute, focused on mutations in the genes that encode RAS and BRAF, two signaling proteins in the MEK-ERK signaling pathway, which is known to be deregulated in many tumor types. The investigators originally hypothesized that cells with either mutation would become dependent on aberrant signaling downstream in the pathway, and that inhibition of MEK in cells with either mutation would stop cell growth.

RAS-mutant cells and BRAF-mutant cells were both treated with CI-1040, a selective inhibitor of MEK. Surprisingly, while BRAF-mutant cells were extremely sensitive to the drug, RAS-mutant cells were not. The investigators then tested a panel of drugs on another set of cancer cell lines with known RAF or BRAF mutations. Compounds that inhibited MEK were effective against BRAF mutants, but did not have a significant effect on RAS mutants.

The investigators proposed that RAS- and BRAF-mutant cells are probably not equally dependent on aberrant signaling mechanisms involving MEK. They concluded that any clinical trials testing MEK inhibitors must stratify patients by BRAF-mutation status. ◆

Funding Opportunities

Exploratory Grants for Increasing the Utilization and Impact of NCI's Cancer Information Service

Announcement Number: RFA-CA-06-015
Letter of Intent Receipt Date: March 20, 2006.
Application Receipt Date: April 19, 2006.

This funding opportunity will use the R21 award mechanism. For more information, see http://cri.nci.nih.gov/4abst.cfm?initiativeparfa_id=3333. Inquiries: Dr. Linda Squiers—squiersl@mail.nih.gov

Innovative Technologies for Molecular Analysis of Cancer (SBIR)

Announcement Number: RFA-CA-07-006
Letter of Intent Receipt Dates: Feb. 8, April 26, and Aug. 28, 2006. Application Receipt Dates: Feb. 22, May 26, and Sept. 26, 2006.

This is a renewal of RFA-CA-06-005. This funding opportunity will use the R43 and R44 award mechanisms. For more information, see http://cri.nci.nih.gov/4abst.cfm?initiativeparfa_id=3334. Inquiries: Dr. Gregory J. Downing—downingg@mail.nih.gov

Innovative Technologies for Molecular Analysis of Cancer (STTR)

Announcement Number: RFA-CA-07-007
Letter of Intent Receipt Dates: Feb. 8, April 26, and Aug. 28, 2006. Application Receipt Dates: Feb. 22, May 26, and Sept. 26, 2006.

This is a renewal of RFA-CA-06-005. This funding opportunity will use the R41 and R42 award mechanisms. For more information, see http://cri.nci.nih.gov/4abst.cfm?initiativeparfa_id=3335. Inquiries: Dr. Gregory J. Downing—downingg@mail.nih.gov

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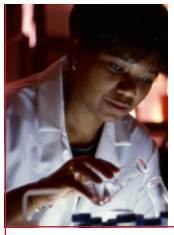


Featured Clinical Trial

Targeted Treatment for Cutaneous T-Cell Lymphoma

Name of the Trial
Phase II Study of LMB-2 Immunotoxin in Patients with CD25-Positive Cutaneous T-Cell Lymphoma (NCI-04-C-0142). See the protocol summary at http://cancer.gov/clinicaltrials/NCI-04-C-0142.

Principal Investigator
Dr. Robert J. Kreitman, NCI CCR

Dr. Robert J. Kreitman

Why This Trial Is Important
Cutaneous T-cell lymphoma (CTCL) is a type of non-Hodgkin lymphoma that includes mycosis fungoides and Sézary syndrome. CTCL is caused when cancerous T lymphocytes (or T cells) migrate out of blood vessels and invade the skin, where they give rise to a variety of abnormalities, including thickened skin plaques, reddening, and at more advanced stages, malignant tumors. In the most advanced stages, cancerous T cells spread into the lymphatic system and internal organs. CTCL is highly treatable when diagnosed at its earliest stages, but there is no curative treatment available for advanced disease.

Researchers are testing the effectiveness of an immunotoxin called LMB-2 in killing CTCL cells. LMB-2 is a laboratory-created monoclonal antibody fragment attached to a bacterial toxin. It binds to a protein called CD25, which is found on the surface of many human lymphocytes. CD25 is also present on CTCL cells in approximately half of all cases, where it appears more abundantly than on normal lymphocytes. The greater abundance of CD25 allows LMB-2 to target malignant T lymphocytes with great specificity. Once LMB-2 binds to CD25 on the cell surface, the toxin is taken up by the lymphocytes, causing them to die.

Patients will receive up to 6 courses of LMB-2 over approximately 6 months, providing their disease does not progress. Patients who respond completely to LMB-2 may receive further treatment.

"In a phase I trial of LMB-2 that involved patients with a variety of hematologic malignancies," said Dr. Kreitman, "the only patient with CTCL had a clinically significant partial response lasting more than 6 months."

Who Can Join This Trial
Researchers will enroll 16 to 27 patients aged 18 and older and diagnosed with advanced-stage CD25-positive CTCL. See the complete list of eligibility criteria at http://cancer.gov/clinicaltrials/NCI-04-C-0142.

Study Site and Contact Information
The trial is taking place at the NIH Clinical Center in Bethesda, Md. For more information, contact the NCI Clinical Studies Support Center at 1-888-NCI-1937. The toll-free call is confidential. ◆

An archive of "Featured Clinical Trial" columns is available at http://cancer.gov/clinicaltrials/ft-all-featured-trials.

Notes

SNP500Cancer Data Now on caGrid

The SNP500Cancer database is now available on the caGrid—the result of a collaborative effort between NCI's Core Genotyping Facility (CGF) and the NCI Center for Bioinformatics. The SNP500Cancer project seeks to validate known or newly discovered single nucleotide polymorphisms (SNPs) and other classes of genetic variants of potential importance to molecular epidemiology studies of cancer and other diseases. SNP500Cancer is a combined effort of NCI's Cancer Genome Anatomy Project and CGF.

The project provides enabling technology to cancer researchers and molecular epidemiologists in the form of data (polymorphism sequences, population frequencies, haplotype estimations, and validated assays) and DNA samples with sequence-verified genotypes. The presence of this dataset on the caBIG™ Grid is a major step in the creation of the data resources and analytical services that is the goal of the caBIG™ program. For more information, go to https://cabig.nci.nih.gov.

Leaf Proposes Electronic Marketplace of Cancer Data

Clifton Leaf, senior editor at large of *Fortune* Magazine, spoke at CCR Grand Rounds on January 31. In his talk, "Cancer Epidemiology, Google, and the Dangerous Legacy of GIGO (Garbage In, Garbage Out)," Mr. Leaf described the Google search engine as the most efficient marketplace of ideas in the world. He called for a medical research enterprise based on Google that would include all available biomedical research data. The new marketplace would allow researchers to integrate and cross-check data currently found in small, segregated "markets." Enormous, open-access marketplaces correct themselves, he said, and in such environments concerns about "garbage in, garbage out" are no longer relevant.

Scholarships Available for Breast Cancer Conference

Scholarships based on need are now available for the National Breast Cancer Coalition Fund's annual training conference, April 29–May 2, in Washington, D.C. Attendees will participate in 3 days of plenary sessions and workshops on cutting-edge breast cancer research, quality health care efforts, and public policy developments. Breast cancer advocates will hear from more than 70 leading researchers and policy makers. For more information, go to http://www.stopbreastcancer.org. ◆

CCR Grand Rounds

February 14: Dr. Anthony Atala, William H. Boyce Professor and Chair, Department of Urology, Wake Forest University School of Medicine. "Regenerative Medicine: New Approaches to Health Care in the 21st Century."

February 21: Dr. Richard R. Love, Professor of Medicine Hematology/Oncology; Professor of Public Health, Epidemiology/Biometrics, Ohio State University Comprehensive Cancer Center. "The Case for a Paradigm Shift from Tumor to Host in Adjuvant Treatment of Breast Cancer."

CCR Grand Rounds are held 8:30 to 9:30 a.m. at the NIH campus in Bethesda, Md., in the Clinical Center's Lipsett Amphitheater. ◆

(Funding Opportunities continued from page 6)

Application of Emerging Technologies for Cancer Research (STTR)

Announcement Number: RFA-CA-07-009
Letter of Intent Receipt Dates: Feb. 8, April 26, and Aug. 28, 2006. Application Receipt Dates: Feb. 22, May 26, and Sept. 26, 2006.

This is a renewal of RFA-CA-06-006. This funding opportunity will use the R41 and R42 award mechanisms. For more information, see http://cri.nci.nih.gov/4abst.cfm?initiativeparfa_id=3336. Inquiries: Dr. Gregory J. Downing—downingg@mail.nih.gov

Innovations in Cancer Sample Preparation (SBIR)

Announcement Number: RFA-CA-07-010
Letter of Intent Receipt Dates: Feb. 8, April 26, and Aug. 28, 2006. Application Receipt Dates: Feb. 22, May 26, and Sept. 26, 2006.

This is a renewal of RFA-CA-06-007. This funding opportunity will use the R43 and R44 award mechanisms. For more information, see http://cri.nci.nih.gov/4abst.cfm?initiativeparfa_id=3337. Inquiries: Dr. Gregory J. Downing—downingg@mail.nih.gov

Innovations in Cancer Sample Preparation (STTR)

Announcement Number: RFA-CA-07-011
Letter of Intent Receipt Dates: Feb. 8, April 26, and Aug. 28, 2006. Application Receipt Dates: Feb. 22, May 26, and Sept. 26, 2006.

This is a renewal of RFA-CA-06-007. This funding opportunity will use the R41 and R42 award mechanisms. For more information, see http://cri.nci.nih.gov/4abst.cfm?initiativeparfa_id=3338. Inquiries: Dr. Gregory J. Downing—downingg@mail.nih.gov ◆

Featured Meetings and Events

A calendar of scientific meetings and events sponsored by the National Institutes of Health (NIH) is available at http://calendar.nih.gov. ◆

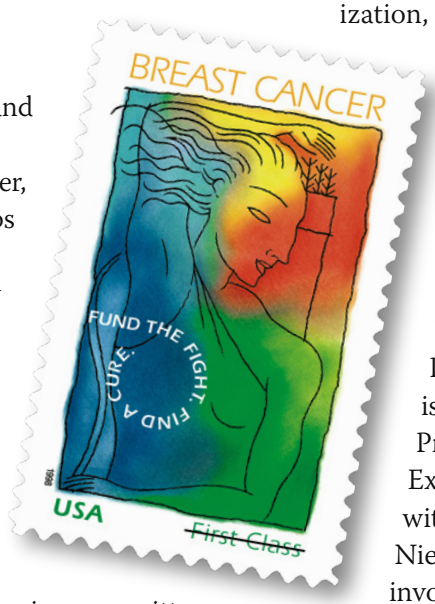
Community Update

Breast Cancer Stamp Will Fund Premalignancy Research

NCI's Executive Committee (EC) recently approved a comprehensive program in breast cancer premalignancy research that includes the areas of prevention, etiology, biology, diagnosis, and molecular epidemiology. Funding for this unique program will come from the highly successful Stamp Out Breast Cancer Act, enacted in 1997, under which the U.S. Postal Service created and sells a special-issue stamp with a surcharge above the first-class postage rate to support breast cancer research.

The distribution of funds from the sale of the Breast Cancer Stamp is set at 70 percent to NIH and 30 percent to the Department of Defense. As of November 2005, NCI had collected more than $33.5 million and has awarded (in obligations plus commitments) more than $25.4 million in grants and contracts, through Insight Awards and an RPG Exceptions Program.

Last fall, during the EC's discussions on how to spend the stamp money in 2006, NCI Deputy Director and Deputy Director for Translational and Clinical Sciences Dr. John Niederhuber strongly urged that it be used to address multiple aspects of breast cancer around a unifying theme, and include intramural components and extramural divisions and centers. "I view it as another opportunity to create a collaborative and integrated scientific program across NCI divisions and centers and, by working together, fill research gaps and synergistically reach new discoveries and interventions," he explained.



At the recent inaugural meeting of a transdisciplinary NCI steering committee for the Breast Cancer Premalignancy Program, a wide-ranging, integrated research plan that encompasses the following elements from all NCI research divisions and centers was presented:

- Molecular epidemiology and biology of mammographic density (Division of Cancer Epidemiology and Genetics)
- Evaluation of different decision-making approaches to chemoprevention (Division of Cancer Prevention)
- Evaluation of strategies for early detection of breast cancer (Division of Cancer Control and Population Sciences, with matching funds from the American Cancer Society)
- Biology of breast premalignancy (Division of Cancer Biology)
- Isolation, propagation, characterization, and imaging of tumor stem cells (Center for Cancer Research)
- MRI-guided therapy with targeted SPIO carbon nanostructures (Division of Cancer Treatment and Diagnosis).

The Breast Cancer Premalignancy Program is similar to the Special Programs of Research Excellence (SPORE) within NCI and NIH, Dr. Niederhuber observed, and involves work on breast cancer stem cells, pathways, the microenvironment, molecular target identification (biomarkers), imaging, drug discovery, and translation.

"I have no doubt that the Breast Cancer Premalignancy Program will lead to real advances in understanding and preempting the biological changes that lead to this terrible disease," he added. "That knowledge gives me a special sense of gratification every time I receive a letter with one of the 'Fund the Fight and Find a Cure' stamps." ◆

The *NCI Cancer Bulletin* is produced by the National Cancer Institute (NCI). NCI, which was established in 1937, leads the national effort to eliminate the suffering and death due to cancer. Through basic, clinical, and population-based biomedical research and training, NCI conducts and supports research that will lead to a future in which we can identify the environmental and genetic causes of cancer, prevent cancer before it starts, identify cancers that do develop at the earliest stage, eliminate cancers through innovative treatment interventions, and biologically control those cancers that we cannot eliminate so they become manageable, chronic diseases.

For more information on cancer, call 1-800-4-CANCER or visit http://www.cancer.gov.

NCI Cancer Bulletin staff can be reached at ncicancerbulletin@mail.nih.gov.