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Studies Show Gene Expression Activity in Leukemia Cells

Two studies, both in the August 5 *New England Journal of Medicine*, offer new insights to some of the underlying mechanisms of acute lymphoblastic leukemia (ALL), a leading form of cancer among children. The first study, by researchers at St. Jude Children's Research Hospital in Tennessee; Erasmus University–Sophia Children's Hospital in Rotterdam, the Netherlands; and the COALL cooperative group in Germany; identified sets of genes that are differentially expressed in leukemia cells resistant to each of four antileukemia drugs tested, and also showed that the pattern of expression of these genes was related to overall patient outcomes. The second study, by researchers at the National Cancer Institute (NCI), found that the loss of a key protein (Smad3) is specific to one form of childhood leukemia, but not to other pediatric and adult leukemias.

Dr. William Evans of St. Jude and colleagues compared total gene expression of leukemia cells taken from 173 ALL patients and grouped the samples that exhibited resistance to one of four drugs: prednisolone, vincristine, asparaginase, or daunorubicin. They found 124 genes and

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Director's Update

Refashioning the Clinical Trials System for a New Era of Opportunity

Robust, extensive, formidable: All of these terms aptly describe the system of clinical trials that underpins clinical cancer research in the United States. Our cancer clinical trial system is, in many respects, the envy of most research establishments; it has helped to save and/or extend the lives of millions of people in the United States and, no doubt, around the world.

It is also clear, however, that our clinical trials system has a number of shortcomings that hinder its effectiveness and limit our ability to make the rapid progress I believe can be achieved in this age of advanced technology and improved understanding of how various cancers operate at the genetic and molecular level. There is, for example, a significant degree of duplication of effort and fragmentation in the clinical trial system, which wastes resources and slows the clinical trials enterprise. In addition, many trials take many years and resources to complete, only to produce equivocal results. There are also problems with poor patient participation, inadequate reimbursement of trial costs, and complex regulatory requirements. Finally, and perhaps most importantly, there is a lack of a widely accepted bioinformatics platform to support a national clinical trials effort.

While the system is not broken, it does need to address these challenges.

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28 cDNA (potential genes) that are differently expressed in drug-resistant cells. "Interestingly, only three of those genes had previously been associated with drug resistance," said Dr. Evans, "so there are unexpected cellular mechanisms at work." There was also little gene overlap among the drugs, and no gene was present on all four lists. The expression profiles for drug resistance were similar across different ALL subtypes, indicating that the mechanism of disease resistance is independent of the molecular cause of the leukemia.

The exact level of drug resistance, whether moderate or high, also independently related to how well the patients generally responded to the drugs: The higher the resistance score, the greater the risk of relapse during treatment. After 10 years, more than 90 percent of the patients with drug-sensitive leukemia were symptomatically disease free, while only 60 percent of the patients with the most resistant cells were disease free. A second clinical trial of 98 patients, which used the same medications according to a different treatment protocol at a different institution, produced similar results.

While this study revealed some commonality among different leukemias, the discovery of Smad3 loss in T-cell ALL highlighted the fact that similar diseases can be quite different on the molecular level. NCI researchers, led by Dr. John Letterio, looked for the Smad3 protein, a key component of normal blood cells, in samples of leukemia cells collected from patients with one of several different forms of leukemia. Smad3 protein was absent in all 10 samples of childhood T-cell ALL, but present in specimens collected from patients with other leukemia subtypes, which included other forms of childhood leukemia (such as B-cell ALL) and adult T-cell leukemias (such as Sézary syndrome).

The researchers were intrigued by the biology behind Smad3's absence. The leukemia cells produced normal levels of Smad3 mRNA—the "instructions" that cells use to make protein—indicating that the Smad3 gene was turned on. Furthermore, the sequence of the Smad3 gene in patient samples was identical to the Smad3 gene found in healthy T-cells, so a genetic mutation was not the culprit. "We don't yet know the mechanisms behind this loss of Smad3 protein," said Dr. Letterio, "but two possibilities may be that protein synthesis is being blocked or that the protein is made but rapidly degraded."

Smad3 loss by itself does not lead to leukemia in mice, but spontaneous T-cell leukemia does develop when Smad3 expression is disrupted in mice lacking the gene for p27Kip1, another regulator of T-cell proliferation. The NCI researchers are now looking for other genetic and epigenetic alterations that might act along with Smad3 in the onset of T-cell ALL. ◆

(Director's Update continued from page 1)
This does not mean jettisoning the entire system. On the contrary, there are many aspects of our clinical trial program that function very well, from our strong biostatistical, data quality, and safety monitoring systems to the conduct of trials that are both disease- and modality-oriented.

The NCI Clinical Trials Working Group (CTWG), is developing new approaches to set priorities for developing and conducting clinical trials. Led by Drs. James Doroshow and Howard Fine, CTWG includes representatives from cancer centers, clinical trials cooperative groups, community clinical oncology programs, advocacy organizations, government agencies, and the research community at large.

CTWG will provide guidance to redesign the clinical system. Based on this work, early steps will focus on making infrastructure improvements, with an emphasis on bioinformatics through the cancer Biomedical Informatics Grid (caBIG) and the establishment of biorepositories and laboratories that can support clinical assays for biomarkers. Longer term, more ambitious areas of discussion will include trial review and prioritization procedures and potential revisions to the classic trial phases (phases I, II, III), with a focus on combination/targeted intervention studies.

Efforts that will dovetail with CTWG's work include the launch of caBIG and NCI's partnership with the Food and Drug Administration (FDA). When fully operational, caBIG will mark a revolutionary change in how clinical trials are conducted, offering researchers a new set of tools, including improved means of patient recruitment. Meanwhile, the NCI/FDA Interagency Oncology Task Force will help to streamline the regulatory process involved in launching and conducting cancer clinical trials.

Recent research advances further motivate us with their promise. Imatinib (Gleevec) proved that targeted therapies could have profound effects on patients, even those with late-stage disease. The use of "lymphochips" have shown the power and potential of gene profiling for lymphoma patients. And recent data on genetic mutations that are linked with response to gefitinib (Iressa) have sent researchers back to the lab to inform the next step of treatment development. As such discoveries become more frequent, NCI will ensure that our system is capable of moving the resulting interventions through well-designed, well-run clinical trials to deliver effective new interventions to cancer patients everywhere. ◆

Dr. Andrew C. von Eschenbach
Director, National Cancer Institute

Cancer Research Highlights

Studies Suggest Potential Link between HCV and NHL

Two case-control studies, one conducted in the United States and the other in Spain, have confirmed earlier findings of a possible link between hepatitis C virus (HCV) infection and non-Hodgkin's lymphoma (NHL). The studies' authors stressed, however, that it remains unclear whether HCV infection is associated preferentially with specific lymphoma subtypes. Both studies were published in the August *International Journal of Cancer*.

The population of the U.S.-based study came from four areas covered by the NCI Surveillance, Epidemiology, and End Results (SEER) program. Overall, 32 of the 813 patients (3.9 percent) with NHL were HCV infected, compared with 14 of 684 controls (2.1 percent). Even after adjusting for potentially confounding factors such as drug use and blood transfusion history, there was a nearly two-fold increase in NHL risk associated with HCV. In the Spanish study, which involved more than 1,100 participants, after excluding patients with HIV and those who had undergone organ transplantation, HCV was associated with a 58 percent increased risk of NHL.

The studies' findings are consistent with a number of earlier studies conducted in the United States and Europe, particularly Italy, in which HCV prevalence among NHL patients was as high as 37 percent. Other biological evidence tends to support such a link, explained Dr. Eric A. Engels of NCI's Division of Cancer Epidemiology and Genetics, the lead author of the U.S. study. For example, he noted that HCV infection has been linked to essential mixed cryoglobulinemia, a disorder that can evolve into NHL. In addition, patients with HCV infection often have chromosomal mutations that are associated with a high risk for NHL. In a study published in 2002, patients with splenic marginal zone NHL who were treated for HCV with an interferon-α-based regimen also saw complete regression of their NHL.

Even if HCV is confirmed to be a causal agent of NHL, Dr. Engels adds, "Most NHL cases in the United States would not be due to HCV because HCV infection is fairly uncommon in the United States and the HCV-associated relative risk is somewhat low."

Gene Transfer Technique Stimulates Immune System to Kill Tumor Cells

When normal cells are harmed by cancer treatments, the patient suffers. However, scientists at Minnesota's Mayo Clinic report that, in mice with metastatic melanoma, killing some of a class of normal skin cells stimulates the mice's immune systems to attack tumors throughout the body. They are the first to successfully demonstrate these effects using gene transfer.

Their "simple method to cure established tumors" by killing normal cells was published online in *Nature Biotechnology* on August 1. The researchers, led by Dr. Gregory Daniels, targeted normal melanocytes, pigment-producing cells in the skin and eyes which become cancerous in melanoma. To target the mice's melanocytes, researchers injected several compounds into the skin. Injections contained a circular piece of DNA with gene coding for HSVtk, a viral protein which converts the drug ganciclovir—also administered in the study—into a toxic form. The HSVtk plus ganciclovir combination killed normal melanocytes, stimulating the immune system, especially when cytomegalovirus was also injected into the skin.

Where melanocytes died, large, dense stretches of immune cells congregated. Melanocyte death appeared to break down some of the immune system's self-tolerance, which ordinarily protects melanoma tumor cells. All mice given HSVtk, ganciclovir, and cytomegalovirus treatment three times were cured of established melanoma. After 100 days, none showed evidence of cancer.

The researchers, whose work was funded in part by the National Institutes of Health (NIH), write that their approach "represents an important opportunity for the development of biological therapies for cancer." They note that in the clinical setting, doctors would have to design treatment carefully, taking into account such variables as tumor size and number of vaccinations, in order to decrease patients' risk of autoimmune disease.

Specific Signaling Pathway Activation Appears to Influence Gefitinib Response

Patients with non-small-cell lung cancer (NSCLC) whose tumor cells overexpress a specific signaling pathway appear to respond better to the targeted agent gefitinib (Iressa) than those in whom the pathway is not activated, according to a study published in the August 4 *Journal of the National Cancer Institute* (*JNCI*).

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Special Report

Impact of Cancer on Fertility Becomes Big Survivorship Issue

Cancer survivor Lindsay Nohr Beck started the Fertile Hope organization in 2001 when she learned that, although almost all patients who receive chemotherapy during their reproductive years are at risk for damage to their fertility capacity, only half of those individuals are informed by their doctors of those risks—and the options to counteract them.

"That's something we've worked really hard to change over the last few years," Ms. Beck recalls. "We're now at the 'tipping point.' This has become a hot topic and oncologists are listening. They want to know the options and where to refer patients." Years before, when Ms. Beck had a recurrence of cancer of the tongue at age 24, she was dismayed to find that there was little information available on options to preserve her childbearing ability after chemotherapy. At almost the last second, she found a local hospital that offered the then-new technology of egg freezing, which she was able to do before starting her treatment.

Clear signs that the impact of cancer on fertility has become a top survivorship issue were made evident at a 3-day international conference on "Parenthood After Cancer" held last March at the M.D. Anderson Cancer Center in Houston. Ms. Beck spoke at the conference along with about 50 other researchers, ethicists, and clinicians. The meeting drew 110 attendees from 13 countries.

Conference chairperson Dr. Leslie Schover, professor of behavioral science at M.D. Anderson, was pleased by the spirit of collaboration shown by the basic scientists and those clinicians involved in treating cancer patients and counseling them about risks and options for fertility-sparing treatment.

Attendees were informed about a number of exciting new research findings and reproductive technologies that are rapidly improving the outlook for cancer patients to have children without fear of endangering the fetus or mother. They included a report by Cornell University infertility specialist Dr. Kutluk Oktay who described a method for freezing ovarian tissue before a woman has chemotherapy and then reimplanting the tissue into the woman and reactivating its egg-producing capability. In a study reported in *Lancet* after the conference, Dr. Oktay reported that he and his colleagues were able to successfully reimplant ovarian tissue in a woman 6 years after the tissue was frozen, and harvest an oocyte that was fertilized in the lab, with development of an embryo. Unfortunately, a pregnancy did not result when the embryo was returned to the woman's uterus.

Dr. Schover said new developments were also reported on sperm banking, which has been an option for men for many years. "For prepubertal boys there are some ongoing, exciting experiments trying to take the stem cells that produce mature sperm cells from the testes, freeze them, and later

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Genetic Testing Shapes Fertility Plans

Women were less likely to want additional children after learning from genetic testing that they are carriers of a hereditary mutation associated with increased risks of breast and ovarian cancer, according to a study published in the May issue of *Cancer Epidemiology Biomarkers & Prevention*.

In a study of 101 men and women of childbearing age, who are descendants of a Utah family line with the inheritable BRCA1 gene mutation, researchers found that "female carriers were less likely to want additional children compared with female noncarriers" after being informed of their test results. When interviewed 4 months after the testing, only 28 percent of female carriers expressed an interest in having more children, compared with 67 percent of the noncarrier group.

The study also found that people who chose not to learn the results of their genetic testing also "have significantly lower intentions to have additional children than noncarriers."

In contrast, men who found out they are carriers of the BRCA1 mutation differed little from their noncarrier counterparts in the desire to have more children. However, none of the husbands of the female carriers "indicated a desire for additional children, while half the husbands of noncarrier wives did." ◆

A Conversation with Dr. Robert Croyle

Dr. Robert Croyle, director of NCI's Division of Cancer Control and Population Sciences, comments on the genetic testing study.

What are the main implications of this study?

For clinicians who provide genetic counseling, the data clearly indicate that the issue of family planning should be raised and discussed, ideally with both wives and husbands. The fact that no husband married to a BRCA1 mutation carrier desired more children reflects a complex family dynamic that could produce decision conflicts that were never anticipated prior to genetic testing.

What further research is being done or needs to be done in this area?

We need further research on fertility behavior in high-risk families and populations to better understand the role of perceived risk of inherited disease. The initial work in this area focused on the emotional impact of genetic test results on individuals tested, but more recent work has examined the more complex interplay of risk information and communication among high-risk family members. Our own work has shown that an individual's reaction to a particular test result is moderated by their siblings' tests results as well as other factors. ◆

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on be able transplant them back [to restore fertility]". Preteen boys are more likely than their female peers to be rendered infertile by cancer treatment, she noted.

Another study first reported at the conference by Harvard's Dr. Jonathan Tilly—later published in *Nature*—could overturn the long-held belief that a woman is born with a fixed number of eggs. Dr. Schover said Dr. Tilly's research in mice suggested that "there may be stem cells in the ovaries that continue to produce new primordial follicles" and germ cells. Dr. Tilly believes that this is likely also the case in humans, holding the promise of restoring or prolonging the fertility of young cancer patients, Schover explained.

Lindsay Beck applauded NCI's leadership, especially from Dr. Julia Rowland and NCI's Office of Survivorship, in making fertility an important issue of concern. She noted NCI's cosponsorship of the March conference and added, "NCI has been great in identifying and promoting survivorship as an area of interest, including working to address fertility."

Despite the rapid progress, much remains to be done to overcome the barriers of lack of information, insurance coverage, and doctor-patient communication about this important area, Ms. Beck said. "I try to make sure that patients feel hopeful, whether they're before treatment, in the midst of it, or afterwards. There are options every single step of the way to start a family and become a parent." ◆

Funding Opportunities

Clinical Trials: Oral Complications of Cancer Therapy

PAR-04-133
Application Receipt Dates: Nov. 1, 2004; March 1, July 1, Nov. 1, 2005; March 1, July 1, Nov. 1, 2006; March 1, July 1, Nov. 1, 2007

The National Institute of Dental and Craniofacial Research (NIDCR) and NCI invite applications for clinical research to reduce the incidence and severity of oral complications from cancer therapies. The purpose of this PA is to collect preliminary data to establish an adequate foundation for phase III clinical trials.

This PA will use the R21 award mechanism. For more information see http://cri.nci.nih.gov/4abst.cfm?initiativeparfa_id=2180. Inquiries: Dr. Roy S. Wu, rw51j@nih.gov

Pilot Studies: Oral Complications of Cancer Therapies

PA-04-134
Application Receipt Dates: Nov. 1, 2004; March 1, July 1, Nov. 1, 2005; March 1, July 1, Nov. 1, 2006; March 1, July 1, Nov. 1, 2007

NIDCR and NCI invite applications for clinical research to reduce the incidence and severity of oral complications from cancer therapies. Developmental, exploratory, and/or pilot studies in epidemiology, behavioral/social sciences, or other areas of clinical research may be needed to accelerate scientific progress in addressing this topic. The purpose of these studies is to collect preliminary data to establish a foundation that may lead to R01-level clinical research grants.

This PA will use the R21 award mechanism. For more information see http://cri.nci.nih.gov/4abst.cfm?initiativeparfa_id=2181. Inquiries: Dr. Roy S. Wu, rw51j@nih.gov. ◆

(Research Highlights continued from page 3)
Time to progression, response rate, and disease control rate were significantly better in patients with tumor cells in which the Akt signaling pathway was active, researchers from the Bellaria Hospital in Italy reported.

To conduct the study, the researchers measured the activity of the Akt and MAPK pathways—which are commonly overexpressed in lung, pancreatic, thyroid, and ovarian cancers—in 103 patients before gefitinib treatment. Patients whose tumors were positive for Akt phosphorylation, a measure of pathway activity, fared better than those whose tumors were negative for Akt phosphorylation. MAPK pathway status appeared to have no influence on response to the drug. Patients with an activated Akt pathway tended to be female and never have smoked.

Gefitinib, a drug that targets the epidermal growth factor receptor (EGFR), has proven to be remarkably effective in a small percentage of patients with NSCLC for whom other treatments have failed. As a result, researchers have been working to determine what genetic or molecular factors may influence gefitinib response. In April, two separate research teams reported that they had found specific EGFR mutations that correlated with patients' clinical response to gefitinib (see May 4 *NCI Cancer Bulletin*, p. 1). In a related editorial in *JNCI*, Dr. Mark G. Kris and colleagues from Memorial Sloan-Kettering Cancer Center, argued that the findings from the Italian study "do not supersede better predictors of response to gefitinib," referring to the studies published in April. "When patients with NSCLC enter the clinic today, treatment decisions must be guided by both clinical characteristics and EGFR mutation status," they said. ◆

Featured Clinical Trial

Novel Chemotherapy Agent for Solid Tumors and Lymphomas

Name of the Trial
Phase I Study of 17-Dimethylaminoethylamino-17-Demethoxygeldanamycin (17-DMAG) with Evaluation of HSP-90 Client Proteins in Patients with Solid Tumors and Lymphomas (NCI-04-C-0218). See the protocol summary at http://cancer.gov/clinicaltrials/NCI-04-C-0218.

Principal Investigators
Dr. Barbara Conley and Dr. Patricia Steeg, NCI's Center for Cancer Research

Why Is This Trial Important?
Heat shock proteins (HSPs) are found in every cell of the body. HSPs help cells survive stressful conditions (including heat, cold, nutrient starvation, and oxygen deprivation) by protecting other proteins. Under nonstressful conditions, HSPs help proteins achieve and maintain their proper shape. Furthermore, HSPs help regulate the activity of proteins involved in important cellular processes, such as cell division and signal transduction (i.e., the process by which cells receive and act on external signals, including hormones and growth factors). Researchers at NCI are investigating a particular HSP, called HSP-90, as a target for cancer therapy. HSP-90 is found in greater amounts in cancer cells than in normal cells.

In this trial, researchers are trying to determine whether 17-DMAG, an HSP-90 inhibitor developed by NCI, will help prevent cancer cells from growing in patients with solid tumors or lymphomas. Solid tumors include cancers of body tissues other than the blood, bone marrow, or lymphatic system.

Dr. Barbara Conley
Principal Investigator

"HSP-90's importance in the growth and survival of cancer cells was discovered in the lab of Dr. Len Neckers at NCI," said Dr. Conley. "That research, along with the work of other NCI and extramural investigators, potentially offers a new therapeutic target that is expressed in a wide range of cancer types."

Who Can Join This Trial?
Researchers seek to enroll 40 patients aged 18 or over with either solid tumor malignancy or lymphoma that is metastatic or unresectable and for which standard curative or palliative measures do not exist or are associated with minimal survival benefit. See the full list of eligibility criteria for this study at http://cancer.gov/clinicaltrials/NCI-04-C-0218.

Where Is This Trial Taking Place?
The study will be done at the NIH Clinical Center in Bethesda, Md.

Who to Contact
Contact the NCI Clinical Studies Support Center (CSSC) at 1-888-NCI-1937. The call is toll free and confidential. ◆

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

Notes

New DSMB Appointed for NLST

The Data and Safety Monitoring Board (DSMB) for the NCI-sponsored National Lung Screening Trial (NLST) has been reinstated, largely unchanged from its original configuration. As reported in the April 6 *NCI Cancer Bulletin*, DSMB members resigned on March 26 because of concern that the professional services contract under which they were secured did not include liability coverage for individual board members. Following the resignation, an interim DSMB, composed of scientists from the NIH, was appointed.

Dr. Christine Berg, of the Division of Cancer Prevention, one of the two NCI divisions administering NLST, said that DSMB members have now returned to their positions under a consulting agreement with the Rockville, Md., company Westat that includes liability insurance. "This agreement addresses the issue of liability that was of concern to DSMB members. With this agreement in place, the DSMB membership will be able to focus on the valuable service they provide to NCI and the NLST," commented Dr. Berg.

DSMBs provide expert review of a clinical trial's plan for monitoring patient safety and data collection as well as interim analyses of outcome data and cumulative toxicity data summaries. Liability issues relating to activities of members of all DSMBs are currently being considered by NIH. For more information on NCI and NIH policies on data and safety monitoring of clinical trials, go to http://deainfo.nci.nih.gov/grantspolicies/datasafety.htm (NCI) and http://grants.nih.gov/grants/guide/notice-files/not98-084.html (NIH).

CIS Nears 10 Millionth Caller

Since its inception in 1976, NCI's Cancer Information Service (CIS) has provided the latest and most accurate cancer information to patients, families, the public, and health professionals. Now, CIS is nearing a milestone in its history, expecting to receive its 10 millionth call for information within the next few months.

CANCER INFORMATION SERVICE™
A PROGRAM OF THE NATIONAL CANCER INSTITUTE
YOUR LINK TO CANCER INFORMATION

In 1976, CIS served 47,000 callers. In 2003, CIS received nearly 280,000 requests for service through several access points (telephone, online chat, e-mail).

CIS's trained information specialists are available Monday through Friday from 9:00 a.m. to 4:30 p.m. local time, and can provide information in either English or Spanish. Callers also have the option of listening to recorded cancer information 24 hours a day, 7 days a week. For cancer information from CIS, call 1-800-4-CANCER (TTY users: 1-800-332-8615). In addition, CIS information specialists offer online assistance at cancer.gov Monday through Friday from 9:00 a.m. to 10:00 p.m. Eastern Time through their LiveHelp service.

Merlino Appointed Acting Lab Chief

Dr. Glenn Merlino has been appointed Acting Chief of the Laboratory of Cell Regulation and Carcinogenesis in NCI's Center for Cancer Research. Dr. Merlino's work focuses on identifying candidate molecular targets or pathways for both mechanistic enlightenment and future therapeutic utility. His efforts are concentrated on two tumor types: cutaneous malignant melanoma and the pediatric malignancy rhabdomyosarcoma. Dr. Merlino is widely recognized for his achievements in providing unique models on these two tumor types and making outstanding scientific contributions. He has also been instrumental in his leadership role on the Animal Models Initiative, a program that fosters high-quality, cutting-edge basic research within the NCI intramural research program on the creation and use of mouse and other animal models of cancer and related disorders.

Workshop Held for Minority Investigators

To encourage minority investigator applications and successful competition for NCI funding, NCI's Division of Cancer Control and Population Sciences, Office of Women's Health, and Center to Reduce Cancer Health Disparities sponsored the Minority Investigator Career Development Workshop on July 21-23, 2004. The workshop provided information on the NCI grants process, technical skills training for grant writing, and sessions to enhance professional growth and development.

Approximately 130 midcareer and transitioning investigators from underrepresented racial and ethnic groups attended. Case studies of experiences obtaining funding were presented, as well as promotion and tenure experiences of other minority investigators. Specific topics included overviews of the NCI grants process and funding mechanisms; locating current funding opportunities at NCI; building collaborative research partnerships; grant writing; research methodology including statistical and qualitative methods, instrument development, and clinical trials design; mock grant reviews; and research publication. ◆

Featured Meetings

This is a list of selected scientific meetings sponsored by NCI and other organizations. For locations and times and a more complete list of scientific meetings, including NCI's weekly seminars and presentations open to the public, see the NCI Calendar of Scientific Meetings at http://calendar.cancer.gov.

NCI Advisory Committee Upcoming Meetings

Date	Advisory Committee
Aug. 30	President's Cancer Panel

Selected Upcoming Meetings of Interest

Date	Meeting	NCI Speakers
Aug. 11	"Pipeline" Symposium	Dr. J. Carl Barrett, Director, Center for Cancer Research; Dr. James H. Doroshow, Director, Division of Cancer Treatment and Diagnosis
Sept. 9-12	6th National Conference on Changing Patterns in Native Communities	Dr. Joseph F. Fraumeni, Jr., Director, Division of Cancer Epidemiology and Genetics
Sept. 13-14	First International Peritoneal Mesothelioma Meeting	Dr. Karen H. Antman, Deputy Director, Translational and Clinical Sciences; Dr. Raffit Hassan, Deputy Director, Laboratory of Molecular Biology, Center for Cancer Research; Dr. H. Richard Alexander, Head, Surgical Metabolism Section, Surgery Branch, Center for Cancer Research

NCI Exhibits

NCI Exhibits are presented at various professional and society meetings. Further information about the NCI Exhibits program can be found at http://exhibits.cancer.gov.

This *NCI Cancer Bulletin* is produced by the National Cancer Institute (NCI). NCI, which was established in 1937, leads a national effort to eliminate the suffering and death due to cancer. Through basic and clinical biomedical research and training, NCI conducts and supports research that will lead to a future in which we can 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://cancer.gov.

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