NATIONAL CANCER INSTITUTE

NCI Cancer Bulletin

A Trusted Source for Cancer Research News

June 24, 2008
Volume 5 | Number 13

In this issue:

A Publication of the
National Cancer Institute
U.S. DEPARTMENT OF
HEALTH AND HUMAN SERVICES
National Institutes of Health
NIH Publication No. 05-5498
http://www.cancer.gov

Master Protein Controls Multiple Myeloma Cells

Despite the constellation of abnormal genetic changes that drive multiple myeloma, these cancer cells are controlled by a single protein, researchers reported online in *Nature* this week.

The study suggests that multiple myeloma cells are dependent upon the cancer-promoting genetic activities controlled by a protein called IRF4. It further suggests that breaking their "addiction" to this protein could possibly treat the disease.

Achilles' heel: In the lab, blocking one protein caused multiple myeloma cells to die.

In 10 different laboratory models of multiple myeloma, Dr. Louis M. Staudt of NCI's Center for Cancer Research and his colleagues found that interfering with the production of IRF4 caused the myeloma cells to die.

"We were surprised to find that multiple myeloma cells with many different genetic abnormalities were all exquisitely sensitive to IRF4 inhibition," says Dr. Staudt. "This suggests that therapeutic targeting of IRF4 might be broadly effective in this disease."

The IRF4 protein, a transcription factor that regulates the activity of multiple genes, is normal in most myeloma cells. But it acquires an expanded genetic repertoire and directs a broad program of activity, including genes that are active in cell metabolism and other basic functions. The loss of IRF4 in myeloma cells, then, results in wide-

(continued on page 4)

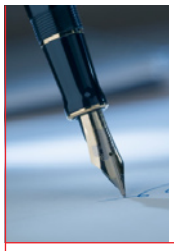
Cancer Research Highlights

Outreach Improves Breast Cancer Detection in African Americans

African American women face a greater risk of death from breast cancer, in part because they tend to be diagnosed at later stages of the disease than white women. New findings published online today in *Cancer* suggest that this disparity in stage of diagnosis can be reduced, using a program that pairs educational outreach to the community with patient navigators who advise and guide diagnosed patients.

Dr. Sheryl G. A. Gabram and colleagues analyzed data from breast cancer patients, 89 percent of whom were African American, treated between 2001 and 2004 at the Avon Foundation Comprehensive Breast

(continued on page 3)



Guest

Director's Update

Cancer Survivorship Research Conference Maps New Challenges

It was very exciting and professionally gratifying for me to join the nearly 500 researchers, patient advocates, cancer care specialists, public health officials, and others at this year's fourth biennial cancer survivorship research conference held June 18–20 in Atlanta, GA. I remember attending the first such meeting in 2002, which was co-sponsored by NCI's Office of Cancer Survivorship in collaboration with the American Cancer Society's Behavioral Research Program. In 2006, the Lance Armstrong Foundation became a full partner as the meeting steadily expanded in scope.

I was struck by the dramatic growth and sophistication of the science presented during last week's meeting. It highlighted key topics and critical questions facing those living with a history of cancer and their caregivers and health care providers. It featured forward-looking research focusing on the unique physical, psychosocial, behavioral, and economic outcomes associated with cancer survivorship.

New findings and insights were presented by a growing cadre of investigators who are dedicating their considerable expertise, talent, and time in this still relatively young field of cancer research. This year, we received and considered 220 abstracts for presentation, a new record for the conference. We selected 12 of the top-ranked abstracts for podium presentations, revealing a wealth of new data and directions for future research.

One of the major plenary sessions focused on new directions in biobehavioral research. A chronic inflammation model proposed at this session could explain two of the most troublesome, persistent effects of cancer treatment—fatigue and depression. Another plenary delved into the burgeoning research examining the impact of cancer on family caregivers. We have only recently come to describe empirically the hidden costs (physical, psychological, and economic) of caregiving and the critical need to intervene not just with cancer patients but also with the family members and friends who provide the bulk of patients' long-term care.

The conference also served as a springboard for mapping out future research and clinical directions in this dynamic and fast-growing scientific area. For example, I was privileged to moderate a fascinating symposium on studies of new groups of cancer survivors: those surviving second cancers, recurrence, and advanced disease. As cancer becomes for many a chronic disease, this poses new challenges for us to address.

I was also drawn to the closing session on survivorship research and the media, which featured a lively dialogue with several well-respected print, radio, and TV journalists. I found the discussions and insider perspective very informative and hope the session will stimulate researchers to think about how to connect with the media and how we can promote the science of cancer survivorship, get more visibility for this work, and have our research results disseminated more widely.

The demand for such information is indicated by new data from NCI's Health Information National Trends Survey (HINTS), which show that 63 percent of cancer survivors report looking for cancer information, compared with only 27 percent of people with no personal or family history of cancer. We need to meet that rising demand with evidence-based information and interventions.

It is also clear that survivorship research is of growing interest to scientists, policy leaders, and clinicians around the country. It has been a focus of emphasis in recent years by the Institute of Medicine, the American Society of Clinical Oncology, leaders of the NCI-designated Cancer Centers, and NCI's Community Cancer Centers Program (NCCCP) pilot.

This survivorship research conference has highlighted the gaps in our knowledge base, what it is going to take to address those gaps, and what we need to do to get there. I know we will! ◆

Dr. Julia H. Rowland
Director, NCI Office of Cancer Survivorship

Cancer Research Highlights

(continued from page 1)

Cancer Center at Grady Memorial Hospital in Atlanta. During that period, 125 trained community health advocates (CHAs) conducted more than 1,000 interactive presentations to increase awareness and encourage breast self-examinations and screening mammography at churches, schools, workplaces, and health fairs in the communities from which the hospital draws many of its patients. Twenty CHAs received further training as patient navigators and helped diagnosed patients maximize their use of services and treatments.

Three years after the program was launched, a cross-sectional study of all breast cancer patients at the primary community hospital showed that the proportion of women diagnosed with *in situ* breast cancer doubled, while those diagnosed with stage IV breast cancer fell by more than a third.

The authors believe the results have "implications on prognosis, and ultimately outcome, for these women if recommended treatment guidelines are followed." They note that the cross-sectional study design could not definitively prove that the intervention caused the shift in stage of diagnosis they observed, but they are continuing prospective work to link the effect of CHAs and patient navigators to particular patient outcomes.

Osteopontin Helps Tumors Harness Bone Marrow Cells for Growth

Researchers at the Whitehead Institute for Biomedical Research and their colleagues have shown that the protein osteopontin, released by breast carcinoma cells, can induce the growth of distant, inactive tumors through the recruitment of bone marrow cells (BMCs). Their report appeared June 13 in *Cell*.

The investigators injected mice with an indolent (slow-growing) breast cancer cell line in one flank, and in the other flank they injected either one of two vigorously growing breast carcinoma lines for the test group or a placebo of matrigel, the liquid vehicle used to support cells for injection, for the control group.

Mice in the test group showed approximately 10 times the tumor growth in the indolent cells as control mice that received the placebo. The cause of the growth was not metastasis of the active cells to the indolent tumor site, but rather the recruitment of BMCs to tumors in both flanks, which in turn encouraged growth of the injected cells.

After analyzing the plasma levels of 80 known human cytokines, the investigators identified osteopontin as a necessary factor for recruitment of BMCs and subsequent tumor growth. Osteopontin is a glycoprotein involved in inflammation, angiogenesis, and metastasis, and has been found in higher levels in cancer patients with metastatic disease. However, when the researchers induced a cell line that does not normally express osteopontin to produce the protein, this alone was not sufficient to trigger tumor growth, indicating the involvement of other cellular factors.

"Our results provide evidence that bone marrow functional activation can be governed on a systemic level by endocrine factors that are released by certain instigating tumors," conclude the authors.

LYN Kinase May Contribute to Imatinib Resistance in CML

Imatinib (Gleevec), the standard treatment for chronic myelogenous leukemia (CML), works by targeting a protein called BCR-ABL, which is expressed only in leukemia or lymphoma cells. Unfortunately, many patients eventually become resistant to treatment with imatinib, and their cancer progresses. Mutations in BCR-ABL that occur over time account for some but not all cases of imatinib resistance, and researchers believe that other proteins may play a role.

In a study published online today in the *Journal of the National Cancer Institute*, investigators from the University of Texas M.D. Anderson Cancer Center and colleagues showed that persistent activation of a protein called LYN kinase may be one of the factors involved in imatinib resistance, independent of any mutations found in BCR-ABL.

The investigators tested both established, BCR-ABL-mutation-negative CML cell lines and patients with BCR-ABL-mutation-negative CML. In patients and cells sensitive to imatinib treatment, the drug suppressed the overactivation of LYN kinase; however, in imatinib-resistant cells and patients, LYN kinase remained activated after imatinib treatment. When LYN kinase activity was reduced—with gene silencing in the cells or with the drug dasatinib

(continued on page 4)

(Highlights continued from page 3)

in patients—the cells died and the patients responded to treatment.

Interestingly, in imatinib-sensitive cell lines and cells taken from patients, the activation of LYN kinase appeared to be regulated by BCR-ABL, but not in imatinib-resistant cells.

Although the investigators cautioned that their cell samples were small, thereby restricting their ability to perform repeat analyses, their results "support the presence of…complex mechanisms of targeted drug resistance in CML patients. The association and mechanism of LYN activation in imatinib resistance warrants further study in additional patients," they concluded.

Mortality Risks Highlight the Effect of Smoking

Charts published June 18 in the *Journal of the National Cancer Institute* compare side-by-side an individual's chances of dying within 10 years from various causes according to age, sex, and smoking status.

Earlier versions of these charts were published 5 years ago by the same NCI-supported researchers, who are affiliated with the U.S. Department of Veterans Affairs and institutions in Vermont and New Hampshire; the new charts include updated mortality data and a revised algorithm to calculate the risks.

For current smokers, former smokers, and those who have never smoked, the researchers calculated age-specific risk of death (number of cases per 1,000 people) from heart disease; stroke; lung, colon, or prostate cancer; pneumonia; flu; AIDS; chronic obstructive pulmonary disease; accidents; and all causes combined. They used data from the National Center for Health Statistics and the U.S. Census Bureau.

Among the most striking trends in the charts are the fact that men of all ages are more likely than women to die in the next 10 years, and that smoking has the detrimental effect of adding about 5 years to the age of a woman and 10 years to the age of a man.

The charts are meant to help clinicians and patients discuss health risks, note the authors. "[They] provide two basic elements that people need if they are to make sense of the health threats they face: the magnitude of the risk and some context… [to] help people understand where to focus risk reduction efforts." The charts may be especially useful in smoking prevention and cessation efforts, they add.

More information from NCI on quitting smoking can be found at http://www.smokefree.gov and from the toll-free tobacco quitline at 1-800-QUITNOW (784-8669). ◆

(Master Protein continued from page 1)

spread disruptions to these essential functions, a process the researchers call "death by a thousand cuts."

Multiple myeloma is a cancer that involves a type of white blood cells called plasma cells, which are antibody-producing cells that develop from the immune system's B cells.

The disease currently has no cure, but patients may benefit from chemotherapy, stem cell transplantation, or newer treatments such as bortezomib (Velcade) or thalidomide.

To discover the role of IRF4, the researchers screened a genetically diverse collection of myeloma cells for "essential" genes using a technique known as RNA interference. They observed the dependence on IRF4 in myeloma cells across genetic subtypes of the disease.

The list of oncogenes regulated by IRF4 in myeloma cells includes *MYC*, which plays an important role in multiple myeloma and other cancers. In an unexpected finding, IRF4 and *MYC* form a feedback loop: IRF4 activates *MYC*, and *MYC*, in turn, activates IRF4, as well as abnormal gene networks regulated by IRF4.

The study is an example of so-called "non-oncogene addiction." In this situation, cancer cells become dependent on the activity of a gene that does not have mutations or other hallmarks of oncogenes.

Based on studies of mice with a form of IRF4 and laboratory experiments, the researchers are hopeful that a therapeutic window may exist in which treatments directed at the production of IRF4 might kill myeloma cells while sparing normal cells.

Transcription factors are considered challenging molecules to inhibit, but recent success in targeting the transcription factors p53 and BCL-6 "provides hope that IRF4 can be exploited as the Achilles' heel of multiple myeloma," the researchers note. ◆

By Edward R. Winstead

Featured Meetings and Events

A calendar of scientific meetings and events sponsored by the National Institutes of Health is available at http://calendar.nih.gov/app/MCalWelcome.aspx ◆

Special Report

Sparing Patients Treatments They Don't Need

Hippocrates would approve. The early days of personalized medicine are as much about "doing no harm" as about matching patients with the most appropriate therapies.

In cancer, a handful of molecular markers can help guide clinical decisions. For instance, tests can identify candidates for drugs such as imatinib (Gleevec). At the same time, markers are emerging that can identify patients who should *not* receive a treatment, and as new research suggests, this is valuable information.

"Patients are well served when physicians can forego a particular therapy and avoid the treatment's toxic effects," said Dr. Richard Schilsky of the University of Chicago, who writes about predictive biomarkers in the June *Lancet Oncology*.

"There really are no highly reliable biomarkers that predict a positive response to a cancer therapy," he continued. "The estrogen receptor for breast cancer is probably the best one going."

The biology of cancer is so complex that biomarker studies are more likely to find, say, a genetic change that causes resistance to a drug than a single biomarker that predicts a positive response.

One such marker of resistance is the gene *KRAS* in colorectal tumors. Patients with advanced tumors that carry *KRAS* mutations appear not to benefit from the addition of cetuximab to chemotherapy, as researchers reported this month at the American Society of Clinical Oncology (ASCO) annual meeting.

Hippocrates wrote in Epidemics, *"As to diseases, make a habit of two things—to help, or at least, to do no harm." Contrary to popular belief, the Hippocratic Oath does not include the phrase "First, do no harm."*

Patients with these mutations (one-third of those with the disease) should be spared the side effects and cost of cetuximab and a related drug, panitumumab, researchers said at the meeting. In Europe, cetuximab and panitumumab are approved for colorectal cancer only in patients with normal *KRAS* genes.

The new results, supported by other studies, were a tipping point. Within days, NCI and its Cooperative Group partners stopped enrolling patients on colorectal cancer clinical studies using cetuximab until this new information on *KRAS* can be appropriately incorporated into the trials.

As new drugs continue to expand treatment options, it will be even more important to know which ones to withhold.

"We are in an exciting era of targeted agents, and there are hundreds more in the pipeline," said Dr. Julie Gralow of the Fred Hutchinson Cancer Research Center at the ASCO meeting. "How are we going to afford these? One answer is that we need to identify patients who are likely to respond and [then] hold the money and hold the toxicity."

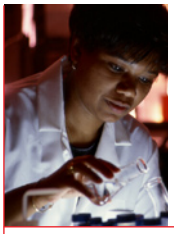
A goal for the field is to develop biomarkers in parallel with new drugs, so that the biomarkers can be validated in final-stage clinical trials. Validation and reliable testing is critical, because errors could deprive patients of a valuable treatment or expose them to unnecessary risk.

A decade after the approval of the breast cancer drug trastuzumab (Herceptin), researchers are still exploring the best way to identify candidates for the drug. Dr. Schilsky noted that while the current test is considered a positive predictor, only about 25 percent of advanced *HER2*-postive tumors respond to the drug. "So, the test may be better at identifying non-responders," he said.

Another biomarker test designed to help patients avoid an unnecessary treatment is Oncotype DX. A clinical trial (TAILORx) is asking whether the genomic test can identify women with ER-positive, early stage breast cancer whose risk of recurrence is sufficiently low that they can safely avoid chemotherapy after surgery.

Beyond what biomarkers can do for individual patients, many researchers believe they could speed the devel-

(continued on page 6)



Featured Clinical Trial

Treatment for Advanced Carcinoid Tumors

Name of the Trial
Phase III Randomized Study of Depot Octreotide Acetate and Interferon alfa-2b versus Depot Octreotide Acetate and Bevacizumab in Patients with Unresectable Metastatic or Locally Advanced, High-Risk Neuroendocrine Carcinoid Tumor (SWOG-S0518). See the protocol summary at http://cancer.gov/clinicaltrials/SWOG-S0518.

Dr. James Yao

Principal Investigator
Dr. James Yao,
Southwest Oncology Group

Why This Trial Is Important
Carcinoid tumors originate most often in neuroendocrine cells of the gastrointestinal tract, although they can arise elsewhere in the body. While most carcinoid tumors grow slowly, they are often resistant to treatment and can be life threatening when advanced. Currently, there is no treatment that has been proven to stop or slow the growth of advanced carcinoid tumors, and patients with these tumors face a dim prognosis.

Doctors are eager to find new ways to treat advanced carcinoid tumors. One strategy being studied is the inhibition of tumor angiogenesis. Carcinoid tumors tend to produce a lot of blood vessels and may be susceptible to antiangiogenesis therapy.

The monoclonal antibody bevacizumab (Avastin) has been shown to inhibit tumor angiogenesis and is approved by the FDA to treat several different cancer types. In this trial, patients with advanced carcinoid tumors that have spread (metastasized) or that cannot be surgically removed (unresectable) will be randomly assigned to receive the drug octreotide acetate along with either bevacizumab or interferon alfa.

The combination of octreotide acetate and interferon alfa is often used for refractory carcinoid syndrome, a collection of symptoms—including flushing, abdominal pain, and diarrhea—caused by hormones secreted by advanced carcinoid tumors.

"In our previous phase II study comparing these combinations, the addition of bevacizumab to octreotide acetate led to rapid and sustained decreases in tumor blood flow, resulting in disease stabilization in most patients and even producing partial responses in some patients," said Dr. Yao. "Additionally, patients receiving bevacizumab were more likely to have stable disease at 18 weeks than patients who received interferon.

"With this phase III trial, we hope to confirm these results and possibly establish bevacizumab as a standard therapy for patients with these difficult to treat tumors."

For More Information
See the lists of entry criteria and trial contact information at http://cancer.gov/clinicaltrials/SWOG-S0518 or call the NCI's Cancer Information Service at 1-800-4-CANCER (1-800-422-6237). The toll-free call is confidential. ◆

(Special Report continued from page 5)

opment of new drugs by enrolling patients who are "likely responders" in clinical trials. Fewer patients might then be needed to meet the goal of the study, and this could save money, as well.

Some companies are searching for biomarkers in preclinical studies, but large trials may be needed to find useful markers. The evidence on *KRAS* and cetuximab, for instance, came from 1,200 patients in many different trials.

Preclinical studies often don't produce biomarker candidates, and "you may only begin to relate a biomarker to efficacy after treating hundreds or thousands of patients," said Dr. Eric K. Rowinsky, the chief medical officer at ImClone Systems, which initially developed cetuximab. The company starts biomarker studies for all of its experimental drugs early in their development, he said.

Many experts believe it is only a matter of time before all patients with colorectal cancer have their tumors tested for *KRAS* mutations following surgery. It also seems likely that physicians will use combinations of positive and negative predictive markers in the future.

"The *KRAS* story is tremendous," said Dr. Rowinsky. "We've always known in oncology that most patients won't respond, and we have become accustomed to that. We have kind of shut our eyes to the reasons. This story tells us we should always look for the reasons patients do or do not respond to any therapy." ◆

By Edward R. Winstead

FDA Update

FDA Warns of Fake Cancer Cures Online

The Food and Drug Administration (FDA) sent warning letters on June 17 to 23 U.S. companies and 2 foreign individuals that have been marketing a variety of fraudulent cancer "cures" on the Internet. The agency also warns consumers against purchasing the bogus cancer treatments.

The Internet has provided a mechanism for bogus cancer cures to flourish, according to an FDA press release. Although the products being sold online are not regulated by the FDA, the agency has authority to issue the warnings because the product marketers claim their products "cure, treat, mitigate, or prevent disease" without having undergone testing for safety and effectiveness. Therefore, the agency considers them unapproved new drugs marketed in violation of the Federal Food, Drug, and Cosmetics Act.

"These warning letters are an important step to ensure that consumers do not become the victim of false 'cures' that may cause greater harm to their health," said FDA Associate Commissioner for Regulatory Affairs Margaret Glavin.

More information about the FDA warnings, including a list of the 125 fake cures consumers should avoid, can be found on the FDA Web site. ◆

Cancer.gov Update

Web Portal for Cancer Advocates Launched

Last week, NCI launched a new portal on Cancer.gov intended for the advocacy community. The portal, Science Serving People, offers information on the human and economic burden of cancer; NCI's budget and research priorities; NCI's collaborations with the public and private sectors; and the return on investment the institute provides the nation. Users can also review cancer-related legislation and Congressional testimony.

NCI's Office of Advocacy Relations led the portal's production by listening to the concerns of advisory boards and cancer advocates seeking ways to stay connected to NCI. To view the portal, click this tile:

Funding Opportunities

Following are newly released NCI research funding opportunities:

Pediatric Brain Tumor Consortium
Announcement Number: RFA-CA-08-026
Letter of Intent Receipt Date: July 15, 2008
Application Receipt Date: Aug. 15, 2008

This is a renewal of RFA-CA-04-501 and will use the U01 award mechanism. For more information see http://researchportfolio.cancer.gov/initiativedetail.jsp?InitiativeID=3890. Inquiries: Dr. Malcolm Smith—smithm@ctep.nci.nih.gov.

Millennium Promise Awards: Non-Communicable Chronic Diseases Research Training Program
Announcement Number: PAR-08-175
Letter of Intent Receipt Dates: Aug. 31, 2008; Aug. 31, 2009; Aug. 31, 2010
Application Receipt Dates: Sept. 29, 2008; Sept. 29, 2009; Sept. 28, 2010

This funding opportunity will use the D43 award mechanism. For more information see http://researchportfolio.cancer.gov/initiativedetail.jsp?InitiativeID=3889. Inquiries: Dr. Aron Primack—aron_primack@nih.gov.

Centers for Innovation in Membrane Protein Production for Structure Determination
Announcement Number: RFA-RM-08-019
Letter of Intent Receipt Date: Sept. 21, 2008
Application Receipt Date: Oct. 21, 2008

This is a renewal of RFA-RM-04-009 and will use the P50 award mechanism. For more information see http://researchportfolio.cancer.gov/initiativedetail.jsp?InitiativeID=3888. Inquiries: Dr. John C. Norvell—norvellj@nigms.nih.gov. ◆

Notes

NCAB Hears of Changes to the NIH Roadmap

At its quarterly meeting last week, the National Cancer Advisory Board (NCAB) heard an update and plans for changes to the NIH Roadmap for Medical Research.

Dr. Dinah Singer, director of NCI's Division of Cancer Biology, provided a historical overview of the Roadmap mission and vision; the scientific programs that have been funded; and a description of the new "Transformative R01" grants, a 5-year pilot program with funding opportunities to be announced this year.

Six areas of special emphasis for fiscal year 2009 have been identified: new protein-capture technologies, the science of behavior change, functional variation in mitochondrial disease, 3-D tissue models, the transition from acute to chronic pain, and pharmacogenomics.

A videocast of the 2-day meeting can be found at http://videocast.nih.gov/PastEvents.asp.

New HMO Cancer Research Network Booklet Available Online

A new publication about NCI's HMO Cancer Research Network (CRN) is now available online. It describes the CRN's research agenda, accomplishments, capacity, and future research potential. It also serves as a "user's guide" for potential collaborators. The brochure outlines how researchers can become partners with CRN members to utilize the network's unique research resources and scientific expertise.

The CRN provides a framework for leading and working with others to address cancer research challenges in the areas of prevention, early detection, treatment, survivorship, surveillance, and end-of-life care. The CRN is also uniquely positioned to study the quality of cancer care in community-based settings.

The network includes research organizations affiliated with 14 large health care delivery systems covering nearly 11 million individuals and enables access to community-based health care systems data and health informatics, allowing for large, multicenter, multidisciplinary intervention research that addresses the spectrum of cancer control issues.

Abstracts Being Accepted for Cancer Immunology and Immunotherapy Symposium

NCI's Center for Cancer Research (CCR) will host a 2-day, national symposium called "Cancer Immunology and Immunotherapy: Realizing the Promise" on September 11–12 in Masur and Lipsett Auditoriums on the NIH campus.

Sponsored by CCR's Center of Excellence in Immunology, this meeting will host leaders in the field of cancer immunology and immunotherapy, include recent advances in both translational and clinical research, and provide a forum for discussion and debate on promising immunologic approaches to prevent and treat cancer.

Abstracts are being accepted for the poster session. The deadline for submission is July 15. Registration is free, but seating is limited. Online registration and instructions for abstract submission can be found at http://web.ncifcrf.gov/events/CancerImmunology/default.asp.

The full program can be found at http://web.ncifcrf.gov/events/CancerImmunology/program.asp. For conference-related questions, contact Karen Kochersberger at kochersbergerks@mail.nih.gov or 301-228-4027.

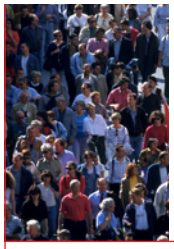
Disparities Summit Scheduled for July 14–16

The 2008 Cancer Health Disparities Summit will take place July 14–16 at the Bethesda North Marriott Conference Center and Hotel in Bethesda, MD. The theme of this year's conference is "Eliminating Cancer Health Disparities Through Science, Training, and Community."

Summit '08 will share evidence-based prevention, screening, treatment, and survivorship interventions from the Community Networks Program, Minority Institution and Cancer Center Partnerships, and Patient Navigation Research Program.

This year's summit will also showcase scientific efforts in diversifying the cancer health disparities training pipeline and provide grant-writing basics, components to build a successful research career, and a mock review session for the Continuing Umbrella of Research Experiences (CURE) program.

The summit is sponsored by NCI's Center to Reduce Cancer Health Disparities. On-site registration will be available. For more information about this meeting, go to: http://www.cancermeetings.org/CHDSummit08/index.cfm. ◆



Community Update

Undergrads Try Integrative Science

It's June, the month when college students around the country finish their spring semester and head home to save money from summer jobs, or take summer classes so they can graduate on time. But a select group of 13 has bigger plans.

These students are recipients of Integrative Cancer Biology Program (ICBP) Cancer Research Fellowships, a program in its third year that places sophomores and juniors for 9 weeks in laboratories that study cancer as a complex integrative system, developing mathematical and computational models for diagnosis and treatment of the disease.

"One of the challenges for today's cancer researcher is incorporating large and diverse information about the disease. This increasingly requires multiple expertise and is a fundamental aspect of the ICBP," says Dr. Dan Gallahan, who directs the program and is deputy director in NCI's Division of Cancer Biology. "Through this fellowship, undergraduates are exposed to multiple disciplines addressing real cancer problems."

"The students who we select are exceptionally bright, but they're not sure what a career in research really means," explains Dr. Betty Tarnowski, ICBP education and outreach coordinator. "This program gives them the exposure to determine if research is something they would like to pursue."

Most of the ICBP fellows are biology majors, but many of them study math, engineering, computer science, or physics.

University of South Florida student, Arthur Edwards, is working this summer at the Lawrence Berkeley National Laboratory.

Anastasia Krymkowski, a double major in applied mathematics and computer science at the University of Vermont, with a minor in art, is working this summer with Dr. Thomas Deisboeck, who directs the ICBP Center at Massachusetts General Hospital, and with Dr. Santosh Kesari at the Dana-Farber Cancer Institute. She is using brain cancer stem cells and mathematical analysis to help develop computer models of tumor signaling networks.

"I enjoy thinking spatially, and modeling a virtual tumor seemed like the perfect fit for that," says Ms. Krymkowski, who is still weighing her options after graduation. "I've always known that were I to pursue a career in a medical field, I'd want to study something that has a devastating impact on people, so I'm definitely satisfied to be working on this project."

Arthur Edwards, another ICBP fellow who is studying bioengineering at the University of South Florida, is being mentored by Dr. Paul Spellman at the Lawrence Berkeley National Laboratory. Most of his friends are taking it easy at the beach this summer, he says, while he's using Bayesian models to look at gene expression, gene copies, and methylation patterns in breast cancer.

"I'll be asking a lot of questions, since I don't really have too much training in this field," he says. His career plans include graduate school, but he's not sure yet what area of research will be his focus. "After this summer I'll have a better idea."

Students have much to gain from these fellowships: the chance to try their hands at cancer research with some of the nation's top scientists as mentors, as well as earn a stipend and explore a different city. But they're not the only ones who benefit from the arrangement.

"These eager students bring a refreshing new perspective to our challenging projects," says Dr. Deisboeck, whose mentoring last summer encouraged Christina Birch to begin graduate school this fall studying biological engineering at MIT, which hosts an ICBP Center, so that she can continue the integrative approach to research.

"We are delighted to contribute to the next wave of students who are trained in both the quantitative and quality aspects of cancer research," he says. "They are who this field needs desperately in order to move ahead." ◆

By Brittany Moya del Pino