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Selenium and Vitamin E Fail to Reduce Prostate Cancer Risk

Participants in the largest-ever prostate cancer prevention trial are receiving letters instructing them to stop taking their study pills, the supplements vitamin E and selenium, after an independent review concluded that the trial will not meet its primary endpoint of reducing prostate cancer risk by 25 percent. The 35,000 participants in the NCI-funded [Selenium and Vitamin E Cancer Prevention Trial \(SELECT\)](#) are being asked to continue in the study for approximately 3 more years.

It is important to keep following SELECT participants for a number of reasons, explained Dr. Eric Klein, director of the Center for Urologic Oncology at the Cleveland Clinic and one of the trial's co-chairs. "There is a small chance of a 'lag effect' of one or both supplements, meaning that their impact on rates of prostate or other cancers may not be apparent for a

few more years," he said.

The SELECT executive committee made the decision on the study medications based on the recommendation of the trial's Data Safety Monitoring Committee (DSMC). In its most recent review of the data in mid-September, the DSMC concluded that, with participants having been on their study medications for 5 years on average, there was little chance that one or both supplements would reduce the number of cases of prostate cancer. The review also revealed trends of increased prostate cancer risk with the use of vitamin E and increased adult-onset diabetes risk with the use of selenium, neither of which were statistically significant.

Launched in 2001, SELECT enrolled black men aged 50 or older and white men aged 55 or older—black men

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Cancer Research Highlights

Additional Genes Tied to Lung Cancer

A large-scale survey of genetic changes in lung tumors has identified 26 frequently altered genes in the most common form of the disease, lung adenocarcinoma. The discovery, reported in the October 23 *Nature*, increases the number of genes associated with lung cancer and expands the base of knowledge about the genetics of this deadly disease.

The Tumor Sequencing Project, a group of academic researchers funded by the [National Human Genome Research Institute](#), analyzed more than 600 genes in 188 lung tumors for noninherited mutations as well as gains and losses of DNA. Their integrated analysis supports the view of cancer as a disease in which core biological pathways may be altered by various

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Guest Director's Update

Oncology and the Physical Sciences Converge

Last week marked the third in a series of NCI-sponsored “think tanks” that I believe hold tremendous promise for advancing cancer research. These landmark meetings, “Physical Sciences-Based Frontiers in Oncology,” bring together top scientists from physics, physical chemistry, engineering, mathematics, cancer biology, and clinical oncology to collaboratively identify the most promising research questions and strategies at the intersection of these fields that could enable progress in cancer research.

Over the last three decades, cancer researchers have made significant progress in beginning to identify important molecular alterations in cancer, and some of these alterations are pointing to new approaches that capitalize on advances in areas ranging from specific genetic targets to biomarker-driven diagnostic tests. Cancer research, more than any other area of science, is producing unprecedented quantities of data and pushing back frontiers in areas such as systems biology and the use of nanotechnologies to interrogate and deliver drugs to cancer cells. It is becoming increasingly clear that we must begin to understand the complexity of cancer in terms of the interactive physics, mathematics,



Dr. Anna Barker

chemistry, and engineering of these systems at all scales.

These workshops represent the next logical step in the process of moving toward an understanding of cancer as an emergent and complex biological system.

Addressing questions such as: “How do cancers develop

and evolve in three-dimensional space over time?” and “What are the mechanics of metastatic disease?” are the domain of the physical sciences. Scientists from these disciplines have a great deal to offer in the ongoing effort to understand how tumors develop, evolve, metastasize, respond, and become resistant to our therapeutic and preventive interventions.

During the first of these workshops, held in February 2008 with approximately 100 attendees, four major themes emerged which represent important focus areas for NCI:

- The overall physics of carcinogenesis, including questions related to gradients, thermodynamics, and forces in cancer
- The role of evolution and evolutionary theory in cancer
- The complexity of cancer, an area which naturally leads to the development of highly sophisticated mathematical models of the cancer process (where we are [already starting to see](#) some headway)
- Information and information theo-

ry in cancer—the coding, decoding, transfer, and translation of information in cancer

This last area was the subject of last week’s think tank. A range of experts in information theory, physics, engineering, and mathematics considered the processes of information management across scales (from the molecular level to the organism) and developed a number of innovative research directions that provide opportunities to view cancer as an emergent information-driven system. Intriguing questions such as “How do cells decide?” created new views of information transfer and cell signaling in cancer.

All of these think tanks have engendered a real sense of excitement among attendees, surpassing anything I have experienced in a long time. I think this excitement derives from looking at cancer through a different prism and perhaps thinking new thoughts about how to attack this complex group of diseases. I doubt that so many experts from these disparate and important fields have ever gathered with a single focus—to enable the development of this new frontier in oncology.

I suspect that when NCI launches a systematic effort to support such work, we will see many transdisciplinary research teams evolve to write a new chapter in cancer research. The convergence of disparate areas of science has often produced true innovation, and I anticipate that the same will be true for these new emerging fields of study focused on understanding and controlling cancer.

We plan on holding more workshops in this series next year, with the hope of making them regular events to bring together individuals interested

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Cancer Research Highlights

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types of changes in multiple genes.

For instance, more than two-thirds of the tumors had at least one mutation affecting the MAPK pathway. Lung cancer patients with these changes may benefit from drugs that affect this pathway, the researchers suggest, noting that compounds called MEK inhibitors have produced promising results in mouse models of colon cancer.

The findings complement other large-scale genomic studies, including an [analysis](#) of glioblastoma brain tumors by [The Cancer Genome Atlas](#) (TCGA) project that appears in the same issue (the findings were published online in September).

In both the lung and brain studies, new mutations were identified in some of the same cancer-causing genes, noted Dr. Matthew Myerson of the Dana-Farber Cancer Institute, who was a leader of both studies. The overlap suggests that despite the diversity of genetic changes in cancer, some targeted therapies will be effective in multiple tumor types, he said in a conference call with reporters. Current examples of such drugs include [imatinib](#) (Gleevec) and [cetuximab](#) (Erbix).

Bevacizumab Tested as Imaging Agent

Combining a low dose of the anti-angiogenesis agent [bevacizumab](#) (Avastin) with a radioactive tracer may be an effective method for imaging tumors, according to findings presented October 22 at an international conference. In the study, Dr. Zheng

Jim Wang and colleagues from University of Texas Health Science Center at San Antonio and MPI Research, Inc., used bevacizumab with the radioactive tracer copper-64 (^{64}Cu) attached to it in mouse models of breast, lung, and pancreatic cancer.

The resulting tumor images, Dr. Wang reported at the [EORTC-NCI-AACR Symposium](#) on Molecular Targets and Cancer Therapeutics in Geneva, Switzerland, were superior to those obtained a day before in the same mice using what they said is considered “the gold standard” for tumor imaging—PET scanning with the imaging probe ^{18}F FDG. In addition, he noted, by using this combination as an imaging agent, the research team was able to detect tumors that were smaller and of an earlier stage than was possible with ^{18}F FDG.

The one-time administration of the compound uses a dose of bevacizumab that is 2.5 to 11.5 times less than the typical therapeutic dose given to patients. “Although we haven’t pursued the dose-contrast correlation study yet, we believe there still is some room to decrease the [bevacizumab] dose and still achieve satisfying results for tumor detection,” explained Dr. Wang.

Bevacizumab, which is approved in the United States for the treatment of advanced colorectal, breast, and lung cancers, targets vascular endothelial growth factor (VEGF), an important signaling protein that helps tumors develop the blood vessels needed to support their growth.

Stress, Depression Linked to Changes in Ovarian Tumors

Researchers have found that patients with ovarian cancer who experienced symptoms of depression and stress had elevated levels of an enzyme associated with the spread of cancer in [macrophages](#), influential cells in the tumor microenvironment. The enzyme, MMP9, is a matrix metallo-proteinase, which can induce cancer cells to invade and metastasize. The study included 56 women, and the findings appeared in the November 1 *Clinical Cancer Research*.

Dr. Susan K. Lutgendorf of the University of Iowa and her colleagues found that depressive symptoms, chronic stress, and low social support were strongly associated with increased levels of MMP9 in tumor-associated macrophages. Conversely, patients who had higher levels of social support had lower levels of MMP9 and VEGF, a growth factor that promotes the development of tumor blood vessels.

To look more closely at the mechanism by which these signals are elevated, the researchers exposed macrophages *in vitro* to hormones released during depression and stress, norepinephrine and cortisol, at concentrations similar to those found *in vivo* when a person is under stress. They found that this exposure increased production of MMP9 in the cells.

“These findings provide a new understanding of biobehavioral influences on the tumor microenvironment and may have implications for patient outcome and targeted pharmacologic and/or behavioral interventions for ovarian cancer patients,” the researchers wrote. ♦



Special Report

Colorectal Cancer Trials Support Gene Testing for Two Drugs

A trio of new studies adds to the growing evidence that patients with colorectal cancer should have their tumors tested for genetic mutations prior to starting therapy with [cetuximab](#) (Erbix) or [panitumumab](#) (Vectibix). Tumors with certain mutations are unlikely to respond to the drugs, and these patients should be spared the expense and side effects of the medications, the findings suggest.

Cetuximab and panitumumab are [monoclonal antibodies](#) that inhibit the epidermal growth factor receptor (EGFR), which plays a role in multiple cancers.

The new findings confirm and extend the results of prior studies, including a [retrospective analysis](#) of a major clinical trial reported this spring at the American Society of Clinical Oncology annual meeting that found cetuximab appeared to be ineffective in patients with mutated forms of a gene called *KRAS*. Since that study was presented, NCI has been modifying cetuximab trials to include testing for mutations in the *KRAS* gene. In Europe, treatment with panitumumab is restricted to patients with normal *KRAS* genes.

The first new study was a retrospective analysis of the only cetuximab trial to show a survival benefit for patients with advanced colorectal cancer. In the [CO.17](#) trial, the benefits of cetuximab were limited

to patients whose tumors carried normal, or unmutated, forms of the *KRAS* gene.

As the researchers [reported](#) last year, the cetuximab group lived, on average, 6 weeks longer than those who received supportive care. But only some patients benefited, and to understand why, the researchers went back and analyzed tumor samples from 394 of the 572 participants.

Some clear patterns emerged: Among patients with normal *KRAS* genes, survival was nearly double for the cetuximab group compared to the supportive care group (9.5 months versus 4.8 months). The progression-free interval was also superior in the group of patients treated with cetuximab, 3.7 months versus 1.9 months.

Among patients with mutated *KRAS* genes, however, there was essentially no difference in survival between the two treatment groups, the researchers reported in the October 23 [New England Journal of Medicine](#) (*NEJM*). *KRAS* mutations were present in 42 percent of the tumors.

“These results represent an exciting transition in the treatment of cancer,” said co-author Dr. Derek Jonker of the University of Ottawa. “Whereas in the past—and specifically in the CO.17 trial—we treated a large number of people and had a small effect, we now begin to have the ability to identify the patients who are most

likely to benefit and then tailor treatment based on the unique genetic makeup of each person’s cancer.”

The research to date leads to the “reasonable conclusion” that all patients with advanced colorectal cancer who are being considered for anti-EGFR therapy should undergo *KRAS* testing, according to an accompanying [editorial](#).

The clinical results are consistent with preclinical research on EGFR in colorectal tumors. The protein sits on the cell surface and controls a number of signaling pathways involved in cell growth. *KRAS* mutations can constitutively activate signaling pathways (for example, MAPK) normally controlled by EGFR. The researchers believe that because this activation occurs “downstream” of EGFR, drugs that inhibit the receptor will not affect the abnormal signaling.

Mutations in other genes, such as *BRAF*, may also impair the effectiveness of anti-EGFR therapy, the editorial notes. About 15 percent of colorectal cancers have disease-related mutations in the *BRAF* gene, which is part of the MAPK pathway.

A day after the *NEJM* study appeared, Italian researchers shared the results of another trial that showed that metastatic colorectal tumors with *BRAF* mutations did not respond to cetuximab or panitumumab. None of the patients with the mutations responded to these drugs, whereas all of the responders had normal *BRAF* genes.

The study included 113 patients, and *KRAS* mutations accounted for 30 percent of the non-responsive cases. *BRAF* mutations explained another 14 percent, leaving more than half of the nonresponsive cases unexplained.

Additional molecular markers will be *(continued on page 5)*

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needed to better define patients who are unlikely to benefit from EGFR-targeted treatment, said Dr. Federica Di Nicolantonio of University of Turin. She presented the results at the 20th [EORTC-NCI-AACR Symposium](#) on Molecular Targets and Cancer Therapeutics in Geneva, Switzerland.

A third new study looked at the *KRAS* status of 715 patients in four

panitumumab trials. None of the patients whose tumors had mutated *KRAS* genes responded to panitumumab (defined as tumor shrinkage or no tumor growth), compared with almost 14 percent of those with normal *KRAS* genes who did respond. Dr. Daniel Freeman of Amgen, Inc., the manufacturer of panitumumab, presented the results last week at the second [EORTC-NCI-ASCO Annual](#)

[Meeting](#) on Molecular Markers in Cancer in Hollywood, FL.

During a press briefing, Dr. Bruce Johnson, director of thoracic oncology at the Dana-Farber Cancer Institute, said that the results were timely, important, and “quite consistent” with the cetuximab study in the *NEJM*. ♦

By Edward R. Winstead

(Selenium continued from page 1)

have a higher incidence of prostate cancer and are typically diagnosed at an earlier age than white men—to be randomized to one of four arms: two placebo pills, selenium and placebo, vitamin E and placebo, or vitamin E and selenium. Although several earlier laboratory and animal model studies suggested selenium and vitamin E had cancer prevention properties, the primary impetus for launching SELECT was the results from two cancer prevention trials in which each supplement alone seemed to significantly decrease prostate cancer risk. Neither of those trials, however, was focused on prostate cancer prevention, noted Dr. Laurence Baker, professor of medicine and pharmacology at the University of Michigan School of Medicine and chair of the Southwest Oncology Group, which coordinated SELECT.

So SELECT was launched, he continued, to definitively answer whether either supplement alone or in combination could reduce prostate cancer risk. At the more than 400 participating sites, participant enrollment was remarkably swift, with all 35,000 patients enrolled within 3 years.

The slight increase in prostate cancer and diabetes seen thus far could be “due to chance,” stressed Dr. Scott Lippman, chair of the

Department of Clinical Cancer Prevention at the University of Texas M.D. Anderson Cancer Center, and a co-chair of the SELECT scientific steering committee. This is particularly true for the prostate cancer trend. “There is no biologic rationale to suggest that vitamin E causes prostate cancer,” he added.

In addition, noted Dr. Klein, the incidence of prostate cancer in the vitamin E/selenium arm was no different than for men taking placebo. “To explain this, one has to postulate that selenium cancels out any deleterious effect of vitamin E,” Dr. Klein

said. “This seems unlikely, given that selenium alone had no effect on the incidence of prostate cancer.”

Ongoing analyses should help to clarify the findings, they said.

The published evidence for any connection between selenium and diabetes is mixed, so continuing to follow SELECT participants should provide some of the strongest evidence yet on the true effect of selenium on diabetes risk, Dr. Klein continued. ♦

By Carmen Phillips

Cancer.gov Update



Web Site Compiles Research on the Food Environment

NCI's [Division of Cancer Control and Population Sciences](#) recently launched a new Web site at <https://riskfactor.cancer.gov/mfe> focused on measures of the food environment, including food stores, restaurants, schools, and worksites.

The site provides a searchable compilation of research articles on food available in these settings, as well

as some measurement instruments, and it allows users to submit articles to the compilation.

The site is intended to improve access to existing measures and stimulate the development of tools in this growing field of research—in turn, strengthening research on individual dietary behavior, informing policymaking, and helping to reduce the prevalence of obesity through targeted interventions. ♦



Profiles in Cancer Research

Dr. Debra Silverman

*Chief, Occupational and Environmental Epidemiology Branch,
Division of Cancer Epidemiology and Genetics, NCI*

When Dr. Debra Silverman caught her first glimpse of cancer research as a high school student in Brooklyn, she says, “It inspired something within me.” Her Advanced Placement biology class—a program which was then in its infancy—took a memorable field trip to nearby Memorial Sloan-Kettering Cancer Center. While there, the group visited a lab conducting early animal studies of tumorigenicity of constituents of tobacco smoke.

“Cancer seemed like a great puzzle to me at the time. We understood very little about what caused it. Smoking was very prevalent,” she recalls. “Early papers had come out in the 1950s about the connection between smoking and cancer, but the impact of that work hadn’t taken effect. I really wanted to do something important in my career, and that trip planted a seed of interest in research that later circumstances allowed to grow.”

Dr. Silverman continued to pursue that interest through college and graduate school in biostatistics, culminating with a doctorate in epidemiology at the Harvard School of Public Health, where she was one of the first non-M.D.s to complete the program and among the first women graduates.

Now an internationally recognized



expert on bladder cancer, Dr. Silverman has co-authored more than 50 papers on the topic. Her early work provided the [first national estimates](#) of the prevalence of occupationally induced bladder cancer. She was also the

first to demonstrate that truck drivers have an [increased risk of bladder cancer](#), and she [suggested](#) that motor exhaust, particularly exhaust from diesel engines, may play a role in the development of the disease.

She was also a key contributor to research that documented for the first time the pattern of risk of bladder cancer following smoking cessation. Her most recent [work suggests](#) that increased urinary frequency and increased water intake have independent protective effects on bladder cancer risk.

Dr. Silverman leads two large studies of bladder cancer that are revealing new insights into environmental determinants, for example, components in drinking water like arsenic and disinfection byproducts.

In the 1990s, her studies of risk factors for pancreatic cancer solidified the evidence that cigarette smoking contributes to this disease. She was also among the first to suggest that elevated body mass index, history of diabetes, and hyperinsulinemia play an etiologic role.

One of her most challenging projects thus far, she says, is a 16-year study of the effects of occupational exposure to diesel exhaust on lung cancer risk in a cohort of more than 12,000 non-metal miners who, because they work with diesel equipment in confined spaces underground, are exposed to diesel exhaust at levels an order of magnitude or more higher than those of other occupationally exposed groups. The study is expected to clarify the role diesel exhaust plays in lung cancer etiology.

Epidemiologic studies—whether in the occupational setting, such as the Diesel-Exposed Miners Study, or in the general population, such as the New England Bladder Cancer Study—are complex undertakings that often take a decade or more to complete. They involve state-of-the-art retrospective exposure assessment in the workplace and in the environment, in-depth interviews with participants, and the collection of biological samples from thousands of people.

“The methodologic challenges to studying occupational and environmental carcinogens require a huge commitment from the research team,” she explains. “This approach exemplifies the complex, long-term studies that are hallmarks of the NIH intramural research program.”

As chief of the NCI’s [Occupational and Environmental Epidemiology Branch](#), an appointment she received earlier this year, Dr. Silverman says she is excited to be leading the branch at a time when interdisciplinary research is becoming an increasingly important focus. In the era of genomic science, Dr. Silverman is championing the study of interactions between environmental/occupational hazards and genetic suscep-

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tibility. Her methodologic rigor in combining epidemiology, genetics, statistical analysis, and emerging technologies is one of the elements that make her so well suited for the job, says Dr. Joseph F. Fraumeni, Jr., director of NCI's [Division of Cancer Epidemiology and Genetics](#) (DCEG).

While carving out her reputation as an internationally respected epidemiologist, Dr. Silverman was able to raise two daughters, working part-time for many years. Juggling a scientific career with motherhood was a challenge, she says, and she now mentors many junior scientists who face the same work-life balance issues, a contribution for which she received the DCEG Mentoring Award.

"Cancer research is an extremely demanding career," she says, "but I think it's important for talented young women scientists to know it's possible to successfully balance a scientific career with the demands of raising a family." ♦

By Eleanor Mayfield

(Director's Update continued from page 2)

in pursuing and supporting science in these evolving fields. I am confident that these activities will help us to deconvolute the complexity of cancer in ways that we can only dream about now, and they will lead to new generations of more effective cancer interventions. ♦

Dr. Anna Barker

Deputy Director of NCI and Director of NCI's Advanced Technologies & Strategic Partnerships



Featured Clinical Trial

Comparing Surgical Treatment for Small NSCLC Tumors

Name of the Trial

Phase III Randomized Study of Lobectomy versus Sublobar Resection in Patients with Small Peripheral Stage IA Non-Small-Cell Lung Cancer (CALGB-140503). See the protocol summary at <http://www.cancer.gov/clinicaltrials/CALGB-140503>.

Principal Investigators

Dr. Nasser Altorki, CALGB; Dr. Harvey Pass, RTOG; Dr. Daniel Miller, ACOSOG; Dr. Kemp Kernstine, SWOG



Dr. Nasser Altorki

Why This Trial Is Important

Standard treatment for non-small-cell lung cancer (NSCLC) detected at a very early stage is surgical removal of the lobe of the lung in which the tumor is found (lobectomy).

Although lobectomy often results in long-term survival, patients may suffer from impaired lung function and may be less likely to be eligible for curative surgery if a second lung cancer develops.

Removing just a portion of the affected lung lobe (sublobar resection) has been shown in some [nonrandomized studies](#) to result in similar rates of survival as lobectomy for patients with small tumors (2 centimeters or smaller). However, these studies were not designed to prove definitively that sublobar resection is as good

as lobectomy in patients with these small tumors.

In this [randomized](#) phase III trial, patients with stage IA NSCLC measuring 2 centimeters or less and located in the outer third of the lung are randomly assigned to sublobar resection (either wedge resection or segmentectomy) or lobectomy. The researchers will follow the patients for 5 years to compare how long they live without their cancer recurring (disease-free survival). They will also compare how long the patients survive overall, their rates of lung cancer recurrence, and their lung function.

"Current practice is based on research conducted in the late 1980s," said Dr. Altorki. "We think that several developments have changed the way we should treat these small tumors. We now have much better staging and can zero in on smaller tumors on the surface of lung segments.

"If the intervention is successful, this trial is likely to change the way lung cancer is managed surgically for years to come, and this will especially benefit patients who have comorbidities such as emphysema," Dr. Altorki added.

For More Information

See the lists of entry criteria and trial contact information at <http://www.cancer.gov/clinicaltrials/CALGB-140503> or call the NCI's Cancer Information Service at 1-800-4-CANCER (1-800-422-6237). The toll-free call is confidential. ♦

Notes

Kington Named NIH Acting Director

On October 31, Dr. Raynard Kington stepped in as acting director of NIH, following Dr. Elias

Zerhouni's departure. Dr. Kington has served as principal deputy director of NIH since 2003 and worked closely with Dr. Zerhouni on the leadership, policy direction, and coordination of NIH's 27 institutes and centers. He previously served in several other positions at NIH and the Centers for Disease Control and Prevention, as well as being a senior scientist at the RAND Corporation.

Dr. Kington earned his undergraduate and medical degrees from the University of Michigan and completed his residency training in internal medicine at Michael Reese Hospital and Medical Center in Chicago. He attended the Wharton School of the University of Pennsylvania as a Robert Wood Johnson Clinical Scholar, earning his M.B.A. and his Ph.D. in health policy and economics.

Dr. Kington's research has focused on the role of social factors, especially socioeconomic status, as determinants of health. His current research includes studies of the health and socioeconomic status of black immigrants, differences in populations in willingness to participate in genetic research, and racial and ethnic differences in infectious disease rates.

SWOG Joins EDRN in Furthering Prostate Cancer Research

Dr. Ian Thompson, chair of the Department of Urology at the



Dr. Raynard Kington

University of Texas Health Science Center at San Antonio and an investigator with NCI's **Early Detection Research Network** (EDRN), was recently appointed chair of the Southwest Oncology Group (SWOG) Genitourinary Committee. Dr. Thompson proposed developing a close relationship with EDRN to improve correlative studies, specifically biomarker studies.

According to the proposal, all correlative studies involving biomarkers, at the concept stage, will come to the EDRN for review. This allows an opportunity to explore biomarkers that are not just diagnostic but also prognostic.

EDRN will use its secure Web site to provide online review and inform EDRN members of new concepts. If EDRN discovery and validation laboratories have a marker or technology that could be applied, they can contact the principal investigator of the therapeutic trial directly. The synergism between the two organizations will facilitate early identification of markers, modifications in trial design or study calendars, proper collection of specimens, recognition of any other preliminary data that are necessary to obtain, and, ultimately, a move towards individualized medicine.



Proposals Sought for 2009 NIH Director's Pioneer and New Innovator Awards

The 2009 NIH Director's Pioneer Awards and New Innovator Awards competition was launched on October 24. The programs, which are

part of the **NIH Roadmap for Medical Research**, support scientists who take innovative, potentially high-impact approaches to major challenges in biomedical or behavioral research.

Pioneer Awards provide up to \$2.5 million in direct costs over 5 years and are open to scientists at any career stage. New Innovator Awards provide up to \$1.5 million in direct costs over the same period and are for early career investigators who have not received an NIH regular research (R01) grant or similar funding. NIH expects to make 5 to 10 Pioneer Awards and up to 24 New Innovator Awards in September 2009.

Beginning this year, both award competitions will begin with a pre-application phase. More information about the Pioneer Awards, including application instructions and submission deadlines, can be found at <http://nihroadmap.nih.gov/pioneer>. Information about the New Innovator Awards, including applications instructions and submission deadlines, can be found at <http://nihroadmap.nih.gov/newinnovator>. ♦



A Shift in Strategy Needed to Win the Cancer Battle

In 1971—one year after I became chairman of the Department of Surgery at Howard University—President Richard Nixon declared a national war on cancer. Since that time, the United States has invested



billions of dollars in cancer research. The progress achieved in this area is a testament to the hard work and dedication of many researchers and health care professionals. Because of their efforts, many who face the diagnosis of cancer can be offered more hope than in years past.

However, cancer continues to impose a tremendous burden. Each and every day, 4,000 Americans are told they have cancer and an additional 1,500 lose their battle with this cunning enemy. Indeed, each cancer-related death should be viewed as a lost battle, a failure in the war we committed to winning so long ago.

In 2007 and 2008, the [President's Cancer Panel](#) heard from experts working in all areas of the National Cancer Program and concluded that a shift in strategy is needed. The Panel's recommendations, summarized below, are detailed in [Maximizing Our Nation's Investment in Cancer: Three Crucial Actions for America's Health](#).

First, preventing and treating cancer must become a national priority. The National Cancer Program requires strong leadership from

the highest level, beginning with the President of the United States. Support for cancer-related research must be strong and stable. Importantly, the emphases and design of our research programs must be carefully evaluated to ensure that our investment will translate into real progress in our clinics and our communities.

Second, all Americans must have timely access to needed health care and prevention measures. Research has taught us how to prevent, detect, and treat many cancers, but, sadly, effective interventions reach only a fraction of our population. This problem of care delivery is complicated by the fact that tens of millions of Americans are uninsured or underinsured. A new health care system must be forged that maintains a focus on wellness and provides all people with regular, coordinated medical care.

Third, the scourge of tobacco in America must end. Although the destructive effects of tobacco on health have been known for decades, it continues to be the leading cause of preventable death in the United States. Ridding the nation of tobacco is the single most important action needed to dramatically reduce cancer-related morbidity and mortality. Energetic efforts in this area are particularly critical in order to oppose the massive, unrelenting onslaught of tobacco industry marketing and product development.

Cancer is a complex enemy; however, we *cannot* allow ourselves to become

complacent about the suffering inflicted by this disease. Rather than being content with incremental progress, we must harness the collective will of our leadership and our nation to create a cancer-fighting enterprise capable of achieving significant and rapid reductions in cancer morbidity and mortality. ♦

Dr. LaSalle D. Leffall, Jr.

Chair, President's Cancer Panel

Cancer Disability Claims Fast-Tracked

The Social Security Administration (SSA) has launched a new “Compassionate Allowances” [initiative](#) to speed processing of disability claims for people with severe medical conditions. The initial [list](#) of 50 qualifying impairments for the initiative includes 25 cancers—lung, breast, brain, bone, ovarian, stomach, and certain leukemias, among others.

SSA disability benefits are intended to replace the loss of a person's income. However, it is left up to the applicant to determine how benefits are used. Once fully implemented, the initiative could speed the claims process from years to an average of 6 to 8 days, and benefit as many as a quarter million people.

More information, including how to contact SSA with questions, can be found at <http://www.social-security.gov> ♦