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Link Between 1p36 Deletions and Cancer Revealed

A team of scientists at Cold Spring Harbor Laboratory and Stanford University has identified a tumor-suppressor function for Chd5, a member of the chromatin remodeling protein (CHD) family that maps to the 1p36 region of human chromosome 1. Their results are published February 9 in *Cell*.

Mutations in 1p36 are coincident with many diseases, including neural cancers, melanoma, hematopoietic cancers, and epithelial cancers of the thyroid, cervix, colon, and breast. These mutations are usually deletions,

sparking a widespread search for a tumor suppressor as the missing link.

Finding this link was the ultimate goal, says lead author Dr. Alea A. Mills, of Cold Spring Harbor Laboratory. "There was a risk," she says. "If we had hit a haploinsufficient gene, for example, we wouldn't have been able to study cancer in these models, but may have discovered other interesting genes in the process. It takes a big commitment to see this sort of chromosome engineering project through to the end."

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Director's Update

Guest Director's Update by Dr. Anna Barker

Mapping the Cancer Genome



Dr. Anna Barker, NCI Deputy Director for Advanced Technologies & Strategic Partnerships

The following captures some of the highlights from an article in the March 2007 Scientific American magazine that I wrote with National Human Genome Research Institute (NHGRI) Director Dr. Francis Collins. It represents one of the first detailed discussions of The Cancer Genome Atlas in a publication that is widely read by a variety of audiences.

Writing in *Science* in 1986, Dr. Renato Dulbecco, a pioneering cancer researcher and Nobel Laureate declared, "We are at a turning point." Discoveries in preceding years had made clear that much of the deranged behavior of

cancer cells stemmed from damage to their genes and alterations in their functioning. "We have two options," he wrote. "Either try to discover the genes important in malignancy by a piecemeal approach, or...sequence the whole genome."

Over the span of 2 decades, Dr. *(continued on page 2)*



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<http://www.cancer.gov>

(Link Revealed continued from page 1)

Using embryonic stem cells, Dr. Mills' team created mouse models with a region corresponding to human 1p36 that was deleted or duplicated. This allowed them to study what happens when the region was lost (as in human cancers) or when an extra copy was added.

They found that cells with an extra copy of the region proliferated poorly and underwent senescence, but deletion of the same region caused immortality. *In vivo*, mice with duplications had excessive apoptosis, while those with deletions were normal but cancer prone.

Once they pinpointed an important tumor suppressive region that corresponded to 1p36, Dr. Mills' team tested which of the candidate genes in the area was the tumor suppressor by seeing which one was able to correct the poor proliferation of cells with an extra copy of the region when knocked down by RNAi. Only one, *Chd5*, scored positive. Importantly, when *Chd5* was depleted in normal cells, the result was the same as when the whole region was deleted. Thus, *Chd5* was the tumor suppressor gene in the region.

Chd5 is related to a family of proteins known as chromatin remodelers, proteins that help unravel DNA so that genes can be read by a cell's transcription machinery.

"The role of *Chd5* as a chromatin remodeler is based purely on its homology to other members of the *Chd* family—there has been no functional data before this," says Dr. Mills. "How this affects tumorigenesis is not well understood, but a better knowledge in this arena is likely to offer novel ways to design more effective anticancer therapies."

The methods behind this research are an important contribution and will have significant implications for mouse modeling and cancer, says Dr. Stephen Chanock, who leads the Genomic Variation Section of NCI's Pediatric Oncology Branch within the [Center for Cancer Research](#) and the Core Genotyping Facility within the [Division of Cancer Genetics and Epidemiology](#).

"Not only have they identified a functionally important novel tumor suppressor mapping to human 1p36, but they have provided a useful road map of where and how to go after other similar genes; moreover, they've used a technically clever and very interesting way to accomplish this," Dr. Chanock says.

For Dr. Javed Khan, who leads the Oncogenomics Section of NCI's Pediatric Oncology Branch, an important aspect of this research is the finding that heterozygosity of a single gene, *Chd5*, predisposed cells to malignancy and that the wild-type locus was retained in immortalized cells, as well as in spontaneous tumors. This finding implies that the "two-hit hypothesis"—which states that if one copy of a gene is defective, then the other copy must be deleted or silenced for a cancer to develop—is not necessary for tumorigenesis in certain cancers. "This study provides convincing evidence that haploinsufficiency of a single gene can lead to certain cancers and this represents a paradigm shift," says Dr. Khan. ♦

By Brittany Moya del Pino

(Director's Update continued from page 1)

Dulbecco's vision has moved from pipe dream to reality. Less than 3 years after the Human Genome Project's (HGP's) completion, NIH

has launched the pilot stage of an effort to create a comprehensive catalog of the genomic changes involved in cancer: [The Cancer Genome Atlas \(TCGA\)](#).

HGP laid a solid foundation for TCGA by creating a standardized reference sequence of the 3 billion DNA base pairs in the genome of normal human tissues. Now TCGA—a pilot project focused on three cancers—will characterize a number of genomic alterations as well as the DNA sequences of specific genes and regions of DNA from tumor tissues and compare them with normal cells to identify the major genetic changes that drive the cancer development process.

Several recent developments have provided proofs of concept that identifying specific genetic changes in cancer cells can point us toward improved methods of diagnosis, treatment, and prevention.

For example, in 2001 the Wellcome Trust Sanger Institute began to use genomic sequencing technologies to explore cancer. A year later, the group found that a gene called *B-RAF* was mutated in about 70 percent of the malignant melanoma cases they examined. A variety of researchers set their sights on this potential new therapeutic target, and 5 years later, the most promising of these therapies are being tested in clinical trials. Other research groups have zeroed in on genetic mutations linked to certain types of breast cancer, colon cancer, leukemia, and other cancers to develop new therapies and high-value molecular diagnostics that will point physicians to those chemotherapeutic agents to which a specific patient is most likely to respond.

A strategy used at the beginning
(continued on page 7)



Cancer Research Highlights

Disparities Found in Colonoscopy Use by Those on Medicare

Screening rates for colorectal cancer in the United States lag far behind those for breast and cervical cancer, despite [research](#) showing that the high mortality rates could be lowered significantly by detecting colorectal cancer at an early stage when it is more treatable. A study in the February 12 *Archives of Internal Medicine* of nearly 600,000 Medicare recipients aged 65 or older living in Illinois, Florida, and New York shows that women, nonwhites, and people with low income or educational levels were less likely to get a colon screening test than were men, whites, and people with higher incomes and education levels.

Over a 2-year period, 18.3 percent of all men and women in the study group had a colon screening test, although women were less likely to undergo invasive screening tests such as [colonoscopy](#). The frequency of screening by both sexes diminished with age, but more so in women.

Most (89.5 percent) beneficiaries in the study were white; blacks, Hispanics, and all other racial and ethnic groups were aggregated together as nonwhite, and were 48 percent less likely to get any screening test than were white participants. Beneficiaries living in areas with the highest proportion of high school graduates were 52 percent more likely to get screened. In general, living in a zip code with a higher per capita income increased the likelihood of

getting screened, though nonwhites in the highest income group were actually less likely to be screened. Whites were more likely to get a colonoscopy as their income increased; nonwhites were not.

“[Further research](#) is needed to determine the basis for the observed ongoing disparities to develop interventions to reduce and eliminate these differences,” wrote Dr. Ashwin N. Ananthakrishnan from the Medical College of Wisconsin in Milwaukee, lead researcher on the study.

Switching Hormone Therapies Reduces Mortality from Breast Cancer

Pooled results from two randomized clinical trials, published online ahead of print in *Cancer*, indicate that women taking [tamoxifen](#) after surgery for breast cancer who switch to an aromatase inhibitor after 2 or 3 years have improved survival compared with women who continue tamoxifen for an additional 2 or 3 years.

The investigators combined the results from the GROCTA 4B trial, which tested the aromatase inhibitor aminoglutethimide, and the ITA trial, which tested the aromatase inhibitor [anastrozole](#) (Arimidex). Combination of the data was planned into the design of the ITA trial, which was performed by the same collaborative group as GROCTA 4B.

In GROCTA 4B, investigators randomly assigned women who had already been taking tamoxifen for an average of 3 years to either continue taking tamoxifen at the same dose for

an additional 2 years or to switch to aminoglutethimide for an equivalent period of time. The design of ITA mirrored that of GROCTA 4B, except that women assigned to switch drugs were given anastrozole.

In the combined analysis, all-cause mortality and breast cancer-related mortality were both significantly improved in women who switched to one of the aromatase inhibitors. Although the trials had several limitations, including the fact that neither trial reached its recruitment goal, the results mirror those of other recently published studies, explained the authors. “The present data and mortality benefits emerging from the most recent reports of the other switching trials...reinforce the indication of early switching to an aromatase inhibitor in women presently receiving adjuvant treatment with tamoxifen,” they wrote.

Lung Cancer Incidence Rates High Among Women Who Have Never Smoked

While smoking remains the predominant cause of lung cancer, a new study reveals that incidence rates of lung cancer among people who have never smoked (never smokers) are higher in women than in men. The study results were published in the February 10 *Journal of Clinical Oncology*. This study is unlike previous studies that focused mainly on mortality rates and that found men had higher lung cancer mortality rates than women.

Dr. Heather A. Wakelee of the Stanford Comprehensive Cancer Center and colleagues calculated the incidence of lung cancer among never smokers, former smokers, and smokers aged 40 to 79 from six large cohort populations: the
(continued on page 4)

(Highlights continued from page 3)

Nurses' Health Study, the Health Professionals Follow-Up Study, the California Teachers Study, the Multiethnic Cohort Study, the First National Health and Nutrition Examination Survey Epidemiologic Follow-Up Study, and the Swedish Lung Cancer Register in the Uppsala/Orebro region. Lung cancer incidence was calculated as new cases per person-year.

Lung cancer incidence rates among female never smokers aged 40 to 79 ranged from 14.4 to 20.8 per 100,000 person-years, while incidence rates among male never smokers aged 40 to 79 ranged from 4.8 to 13.7 per 100,000 person-years.

While researchers have not pinpointed the underlying cause of the greater incidence of lung cancer in female never smokers, they have identified the following as potential risk factors: secondhand smoke; occupational exposures such as asbestos, chromium, or arsenic; environmental exposures such as domestic radon; indoor pollutants; previous lung disease; dietary factors; family history; and genetic factors.

Dr. Adi F. Gazdar of the University of Texas Southwestern Medical Center in Dallas and Dr. Michael J. Thun of the American Cancer Society wrote in an accompanying editorial, "Clearly, lack of an understanding of the factors responsible for lung cancers in never smokers is a major deficiency that must be addressed before we can explore preventive strategies." Also, the differences the investigators found in the histological types of lung cancer, as well as the genetic differences between nonsmokers and smokers, could have implications for improving treatments and outcomes of lung cancer.

Gene Involved in Brain Development Has Role in Tumors

A gene that helps control the growth of stem cells during brain development also plays a role in regulating the growth of malignant glioma, a deadly brain cancer, according to a new study. The gene, *Olig2* (oligodendrocyte lineage transcription factor 2), produces a protein found only in the nervous system that controls the activity of other genes. Experiments in mice suggest that this protein could be a potential target of therapies that are specific to the nervous system.

Drs. Charles Stiles and David Rowitch of the Dana-Farber Cancer Institute led the study, which appeared in the February 15 *Neuron*. Using tumor tissue from patients with glioma, the researchers found that the *Olig2* protein was expressed in two types of cells—cancer stem cells and progenitor cells—that contribute to the growth of glioma. Further experiments in mice indicated that the protein helps regulate a cellular process, or pathway, involved in tumor growth.

Together with previous research, the new findings suggest that *Olig2* controls a critical pathway involved in the proliferation of normal and malignant stem cells in the central nervous system. The results also highlight the benefits of stem cell research in understanding human cancers, says lead author Dr. Keith Ligon of Dana-Farber.

The researchers describe *Olig2* as a "gateway" gene for the development of brain tumors for several reasons. First, the *Olig2* protein is crucial for the development of neural stem cells and their progeny specifically in the

central nervous system. Second, the activity of *Olig2* is deregulated in brain cancer. And finally, *Olig2* activity is required for the formation of some tumors.

When the researchers blocked *Olig2* activity in mice that develop brain cancer, 91 percent of the animals did not form tumors. "Our findings identify this core transcriptional regulator as an important candidate for anti-tumor therapeutics," the researchers conclude. ♦

Legislative Update

NIH Continuing Resolution Approved

On February 14, the FY2007 Continuing Appropriations Resolution was passed by the Senate; it was signed into law by President Bush on February 15. NIH received a total appropriation of \$28.9 billion, an increase of \$619.5 million (2 percent) more than the FY2006 appropriation. A large portion of this increase—\$483 million—goes toward funding the NIH common fund.

Although NCI will continue to be funded at the FY2006 level of \$4.79 billion, NCI and other institutes will not be required to contribute to the common fund this year, which could translate into approximately \$42.8 million for NCI.

For a more in-depth look at the NCI budget process and how it affects the National Cancer Program, please see the [recent article](#) by NCI Director Dr. John E. Niederhuber, "A Look Inside the National Cancer Institute Budget Process: Implications for 2007 and Beyond." ♦



Spotlight

A New Tobacco Threat?

In 1983, newly retired New York Yankees star Bobby Murcer recorded “Skoal Dippin’ Man,” his first and only country music hit. “Just a pinch between my cheek and gum,” Murcer twanged, “makes me feel like a long home run.”

At the time, Skoal and other [smokeless tobacco](#) (ST) products, including chewing tobacco and snuff, were near their peak popularity among teens and young adults, including use by approximately one in five high school seniors.

Two decades later, things are remarkably different. By 2003, ST use among 12th-grade boys had dropped to less than 13 percent, and use among girls remained very low. While the reasons for the decrease are unclear, researchers suggest that increases in excise taxes and extensive antitobacco efforts, though primarily aimed at cigarettes, likely played a role.

Some recent developments, however, have tobacco control researchers and advocates worried that these important gains could be erased. In addition to periodontal problems and cardiovascular disease, ST has been most closely associated with increased risks of oral and pancreatic cancer, so any increase in its use is concerning. That concern is compounded by the results of a new study demonstrating that ST use is strongly associated with smoking initiation.

Although a spit-free ST product called snus has been popular in some Scandinavian countries for several

decades, similar products have only recently been introduced in the United States.

Because users don’t need to spit, these small, tea bag-like pouches are easier to use and far more discreet than traditional “moist snuff” like Copenhagen or loose-leaf products like Red Man. They typically come in small, decorative tins, and several brands are available in flavors like mint and cinnamon.

U.S. Smokeless Tobacco (UST), Inc., which manufacturers Skoal, launched its first spit-free ST product, Revel, in 2004. Instead of “Just a pinch,” marketing materials for Revel proclaim “No smoke, no spit, no boundaries.” UST began test marketing its first Skoal-branded spit-free product, Skoal Dry, last year in Louisville, KY, and Austin, TX.

The ease of use and gum-like flavors are reminiscent of earlier history, says Dr. Mark Parascandola, from NCI’s [Tobacco Control Research Branch](#).

ST use began to surge among teens and young adults in the 1970s, he explains, when tobacco companies introduced “products with lower nicotine content and attractive flavorings, which made them more accessible to new users.” Users would eventually graduate to products with higher nicotine content.

“With these new products,” he continues, “there is reason to be concerned we could see a similar phenomenon.”

That very scenario is happening in Norway, where, over the past decade, daily use of snus has tripled among teens and young adults. According to Dr. Karl E. Lund, research director at the Norwegian Institute for Alcohol and Drug Research, the company that controls 95 percent of the European snus market, Swedish Match, makes what he calls “starter kits.”

“This is snus sold in glamorous metal boxes...where the snus is seasoned with different kinds of fruit flavors,” he explains. “[They are] easy to use for snus novices.”

Last October, Swedish Match North America announced a partnership with one of the largest U.S. cigarette companies, Lorillard Tobacco Company, to develop new smokeless products for the U.S. market.

Lorillard, however, is playing catch-up to Philip Morris and R.J. Reynolds (RJR), the two biggest U.S. cigarette manufacturers, both of which launched spit-free products last year.

RJR’s product, Camel Snus, is being test marketed in Austin and Portland, OR, but can also be ordered by phone or via the Internet. Philip Morris’ product, Taboka, is being test marketed in Indianapolis.

“That’s a very big concern for the public health community,” says Dr. Herbert H. Severson, a tobacco control researcher at the Oregon Research Institute in Eugene. “Most of us [in tobacco control] believe the companies can be successful in increasing the market for oral tobacco.”

Count Dr. Michael Siegel, a tobacco control researcher at Boston University, among the believers. In order to be successful, he argues, “they have to have young people use it.”

Both Philip Morris and RJR explic-
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(Spotlight continued from page 5)

itly state in marketing materials and media reports that these new products are for adult smokers searching for a smoking alternative, particularly in smoke-free workplaces or restaurants.

In the case of Taboka, says Dr. Scott Tomar, an ST researcher from the University of Florida, its low nicotine content and Philip Morris' marketing approach suggest it is being positioned, at least for now, as a "situational substitute" for smokers.

Camel Snus, on the other hand, seems to be targeted toward younger users, Dr. Tomar adds. It's being promoted in bars and music magazines geared toward younger audiences, and the marketing materials suggest using it at rock concerts and clubs.

With these new products, the potential for a surge in use among teens and young adults, Dr. Tomar says, is considerable.

"The history of [ST] products in the U.S. and in Sweden and Norway is that it's almost entirely adolescent and young adult males who initiate use," he says.

A big question is what happens after that, because several studies have suggested that ST use among teens can be a smoking gateway.

A 2003 study led by Dr. Tomar, for instance, found that teens using ST at study entry were three times more likely to be smokers 4 years later than those who had not used ST. But a research team from Penn State disputed the findings, arguing that the study failed to account for other smoking initiation risk factors, such as whether parents or close friends smoke or engage in deviant behavior.

However, a new study of nearly 2,300 seventh- and ninth-graders led by Dr. (continued on page 7)



Featured Clinical Trial

Neoadjuvant Treatment of Breast Cancer Using Aromatase Inhibitors

Name of the Trial

Phase III Randomized Study of Neoadjuvant Therapy Comprising Exemestane Versus Letrozole Versus Anastrozole in Postmenopausal Women with Estrogen Receptor Positive Stage II or III Breast Cancer (ACOSOG-Z1031). See the protocol summary at <http://cancer.gov/clinicaltrials/ACOSOG-Z1031>.

Principal Investigators

Dr. Matthew Ellis and Dr. John Olson, American College of Surgeons Oncology Group; Dr. Laura Esserman, Cancer and Leukemia Group B

Why This Trial Is Important

Women with breast cancer that grows in response to the hormone estrogen often benefit from treatment with drugs known as aromatase inhibitors (AIs). AIs block the ability of an enzyme in the body called aromatase to make estrogen.

Researchers want to compare AI therapy and chemotherapy as neoadjuvant (before surgery) treatments for breast cancer, but first they need to determine which AI(s) to use in such a comparison. Neoadjuvant therapy may shrink tumors enough to allow a woman to undergo breast-sparing surgery instead of mastectomy.

In this trial, women with estrogen-responsive (i.e., estrogen receptor-positive) breast cancer will be treated with one of three AIs—**exemestane**

(Aromasin), **letrozole** (Femara), or **anastrozole** (Arimidex)—before surgery to determine which AI(s) to use in a future trial that compares neoadjuvant AI therapy and neoadjuvant chemotherapy. If major differences between the AIs cannot be found, more than one AI may be used in the future trial.

"Our long-term aim is to establish AI therapy as a standard neoadjuvant treatment option," said Dr. Ellis. "Another goal of this trial is to help us define a patient population that

is likely to benefit from neoadjuvant AI treatment. Through genomic analysis we will determine the molecular basis for differences in aromatase inhibitor response."

Who Can Join This Trial

Researchers seek to enroll 375 postmenopausal

women with ER-positive stage II or stage III breast cancer. See the list of eligibility criteria at <http://cancer.gov/clinicaltrials/ACOSOG-Z1031>.

Study Sites and Contact Information

Study sites in the United States and elsewhere are recruiting patients for this trial. See the list of study contacts at <http://cancer.gov/clinicaltrials/ACOSOG-Z1031> or call NCI's Cancer Information Service at 1-800-4-CANCER (1-800-422-6237) for more information. The toll-free call is confidential. ♦



Dr. Matthew Ellis

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

(Spotlight continued from page 6)

Severson scheduled to be published later this month—which controlled for known smoking initiation risk factors—found that ST use at baseline was independently associated with a more than 2.5-fold increased risk of smoking 2 years later.

That's why any tobacco use needs to be discouraged with young people, Dr. Severson says.

Among teens and parents, he adds, "we have evidence that indicates they believe smokeless tobacco is a safer alternative. What I'm trying to tell people is whether it's safer or not, it can lead to nicotine dependence." ♦

By Carmen Phillips

CCR Grand Rounds

February 27: Dr. Monica Justice, Associate Professor, Baylor College of Medicine. "Mouse Models of Hematologic Diseases."

March 6: Dr. Zena Werb, Professor and Vice Chair, Department of Anatomy, University of California, San Francisco. "Deciphering the Tumor Microenvironment During Breast Cancer Progression."

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

(Director's Update continued from page 2)

of HGP was to test protocols and technology before scaling up to full DNA sequence "production." Similarly, TCGA is beginning with a pilot project to develop and test the scientific framework that will be needed if we are to ultimately map all the genomic abnormalities involved in cancer. Over the next 3 years, NCI and NHGRI will devote \$100 million to compiling an atlas of genomic changes in glioblastoma, lung cancer, and ovarian cancer.

These cancers were chosen for several reasons, including their value in gauging the feasibility of expanding this project to a much larger number of cancer types. Only if this pilot phase achieves its goals will we move forward with a full-fledged project to develop a complete cancer "atlas."

As detailed in the article, the road ahead is fraught with scientific, technological, and policy challenges—some of which are known and others as yet unknown. Among the uncertainties to be resolved: Will new sequencing technologies deliver on their early promise in time to make this effort economically feasible? How quickly can we improve and expand our toolbox for systematically detecting epigenetic changes and other large-scale genomic alterations involved in cancer, especially those associated with metastasis? How can

we harness the power of computational biology to create algorithms and data portals that will prove useful to basic biologists, clinical researchers and, eventually, health care professionals on the front lines? The list goes on.

In *Scientific American*, Dr. Collins and I compared our position with TCGA to that of the 19th-century explorers Lewis and Clark, as they ventured up the Missouri River into the largely uncharted Northwest Territory. Although they did not find the water route across the continent, their detailed maps proved valuable to a fledgling nation in ways that President Thomas Jefferson, who dispatched the explorers, could never have imagined.

For the sake of all those whose lives have and will be touched by cancer, we can only hope our 21st century expedition into cancer biology exceeds even Renato Dulbecco's grandest dreams. ♦

Funding Opportunities

For a complete listing of current NCI funding opportunities, please go to the HTML version of today's *NCI Cancer Bulletin* at http://www.cancer.gov/nci-cancerbulletin/NCI_Cancer_Bulletin_022007/page8 ♦

70
YEARS
OF EXCELLENCE
IN **CANCER**
RESEARCH

If Memory Serves...

In November 1937, NCI awarded its first research grants, which totaled \$90,925 by the end of that fiscal year. The first recipients were Dr. Louis F. Fieser of Harvard—\$27,550 to study chemical structures and carcinogenesis; Dr. E. O. Lawrence of the University of California, Berkeley—\$30,000 to study the development of a cyclotron for cancer therapy; and Dr. Edward Wallace of the University of Cincinnati—\$1,500 for the study of the pituitary gland and cancer. ♦

For more information about the birth of NCI, go to <http://www.cancer.gov/aboutnci/ncia>.



Community Update

M.D. Anderson Awarded CEO Cancer Gold Standard Accreditation

The University of Texas M.D. Anderson Cancer Center has been awarded the [CEO Cancer Gold Standard accreditation](#). M.D. Anderson is the first NCI-designated Cancer Center and the first health care system in the nation to receive this accreditation.

It was awarded by the [CEO Roundtable on Cancer](#) which was established in 2001 in response to a challenge from former President George H.W. Bush, who called on a group of corporate executives to “do something bold and venturesome about cancer within your corporate families.” The Roundtable focuses on three key cancer-fighting objectives: risk education, early detection, and quality care. Only 12 organizations have received the distinction.

Organizations that adopt the CEO Cancer Gold Standard offer a series of benefits and programs to their employees and their families that lower the risk of cancer through lifestyle changes. These include elimi-

nating the use of tobacco, exercising regularly, and maintaining a healthy diet. Gold Standard companies also cover preventive screening costs so that more cancers will be detected at the earliest possible stage when treatment can improve the outcome. Finally, a Gold Standard organization provides access to the best-available treatment, including clinical trials, when a cancer diagnosis becomes a reality for an employee or a family member.

“To be recognized for the emphasis we place on our employees’ health and well-being in the fight against cancer and to be the first health care system to receive the distinction is an incredible honor,” said Dr. Georgia Thomas, executive director of M.D. Anderson’s Department of Employee Health and Well-Being. “In recent years, as the research between healthy lifestyle habits and one’s risks for cancer have become only more evident, M.D. Anderson has reinvigorated its emphasis on the wellness of our employees.”

M.D. Anderson offers its employees gym membership discounts, smoking cessation programs, cancer screening opportunities, use of exercise equipment stationed around the institution, and healthy choices in the cafeteria.

“The CEO Cancer Gold Standard accreditation challenges us not only to maintain these programs, but to expand upon them, to meet the needs of our diverse employee population,” Dr. Thomas noted. “Our commitment to the principle of CEO Cancer Gold Standard is permanent, as it fully embodies our mission to eliminate cancer, and that starts with M.D. Anderson employees and their family members.”

New health-conscious programs already underway for M.D. Anderson employees in 2007 include the “Rock Steady” weight management group and employee walking and running clubs. Also this year, M.D. Anderson plans to expand on the number of exercise equipment stations across the campus.

In addition to M.D. Anderson, the CEO Roundtable recently announced five other newly accredited organizations: the American Legacy Foundation, Enzon Pharmaceuticals, Jenner & Block, the SAS Institute, and Quintiles Transnational Corp. ♦

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> ♦

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

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

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