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TOWARD THE FUTURE OF CANCER PREVENTION

University of Texas M. D. Anderson Cancer Center
by Renee Twombly

Can most types of cancers be prevented?

It's a question that has emerged in the past 20 years, given advances in screening and early diagnosis, rapid developments in genetics and molecular biology, and progress in the treatment of early disease and in next-generation targeted therapies.

And finding answers is one of the top goals of The University of Texas M. D. Anderson Cancer Center, which has one of the largest cancer prevention research programs in the world.

M. D. Anderson was among the first to begin dedicated prevention research efforts in the late 1970s. A decade ago, nine faculty were working on 23 projects - a pursuit that was regarded as trend-setting at the time. The cancer center's focus on prevention has grown so much in recent years that the 48 faculty, involved in 140-plus research projects and clinical programs valued at more than $20 million in 2005 alone, just moved into the new Cancer Prevention Building.

In addition to housing faculty offices, the building's Cancer Prevention Center and new Behavioral Research and Treatment Center provide advanced early detection and risk-reduction services and state-of-the-art biobehavioral and psychosocial research venues.

These two centers involve only a sliver of the basic and applied research under way. In short, the researchers, physicians, nurses, employees and volunteers that staff this building aim to bring about a future that may some day be free of cancer.

They also are the first to say that attaining this goal will not be easy; that prevention will require developing a wide variety of strategies and associated tactics to curtail the variety of different diseases, all called cancer, that have now emerged as the number one killer of Americans under age 85.

"Prevention is very broad," says Bernard Levin, M.D., vice president and head of the Division of Cancer Prevention and Population Sciences. "It is not just prevention of cancer development, but includes advances in diagnosis and treatment that reduce suffering and mortality from the disease."

In short, "prevention," as oncologists use the term spans the gamut from stopping cancer from ever developing to improving cure rates through earlier detection, thereby preventing recurrence and death. Prevention also encompasses preventing suffering from cancer by controlling pain and meeting psychosocial needs.

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Because we see prevention as so inclusive, the task we have set for ourselves is very difficult and won't likely be accomplished for decades," Levin says. "But if we can lessen the odds that even one person will develop cancer, or suffer or die from it, we have moved one step closer to our goal. It is that march of progress over time that will make a difference in the future."

Developing a model of cancer prevention

Debate exists on how many cancer deaths are preventable in principle - estimates range from 50 percent to 80 percent - but most researchers agree that tobacco use (mostly smoking) accounts for the majority. Today, cigarette smoking claims about 438,000 premature deaths in the U.S. annually. It is responsible for up to one-third of all cancer deaths and accounts for 20 percent of annual U.S. mortality due to all causes, according to the federal Centers for Disease Control and Prevention.

And while lung cancer is tobacco's primary killer, smoking also is responsible for many other types of tumors. Since the same carcinogens that cause lung cancer also affect the lining of the entire respiratory tract and are absorbed by the blood and then excreted as waste, smoking is a major cause in cancers of the oral cavity, pharynx, larynx, esophagus, pancreas, stomach, kidney and bladder, among others. The American Cancer Society states that smoking damages almost every organ in the body.

The cumulative consequence of other lifestyle factors on cancer risk such as obesity, physical activity, diet/nutrition and alcohol use, as well as infectious agents and occupational exposures, is not fully known, although some experts say it may approach that of tobacco use.

Given the certainty that the number one cause of cancer illness and death is also the most preventable, scientists to date have aimed much of cancer prevention science on smoking. "Because tobacco is responsible for an impressive one-third of cancers, prevention efforts naturally begin with it," Levin says.

But he and colleagues in the Division of Cancer Prevention and Population Sciences have moved beyond solely delivering advice to stop smoking.

They are developing a comprehensive program that not only devises innovative behavioral and pharmacological approaches to smoking prevention and cessation, but burrows down to the molecular level on every aspect related to prevention.

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For example, researchers at M. D. Anderson are looking at brain physiology; variations in genes that "favor" smoking and other addictive behaviors; genes that either protect people from developing cancer or put them at greater risk; and genes that either aid or thwart cancer treatment.

The goals of such research, Levin says, are to be able to:

* Predict those people who might be most susceptible to smoking and to help them resist smoking initiation;
* Provide more effective cessation assistance to those who are already smoking;
* Help prevent development of cancer by use of chemoprevention strategies;
* Understand the biological processes that make some smokers more susceptible to different cancers; and
* Offer tailored treatments based on tumor and genetic profiles in each patient to help prevent further disease.

If such a global program can reduce tobacco-related cancers, then the same approach might work for cancers influenced by poor nutrition, lack of exercise and excess body weight, and other such factors, Levin says. Add in prevention screening and it makes sense why Levin says "the future of cancer prevention is an integrated approach."

Biobehavior in the cancer formula

Two facts about smokers rivet cancer researchers: the notion that not everyone who tries cigarettes becomes addicted, and the knowledge that only a fraction of long-term smokers (about 15 percent) will develop lung cancer, although tobacco also is responsible for one-third of all cardiovascular deaths under age 85.

Innate differences exist between non-smokers and smokers in terms of "biobehavior," such as a need for nicotine, the way different societal cultures view smoking and how they respond to clinical treatment. Within the division's three groups - the Department of Health Disparities Research, the Department of Behavioral Science and the Department of Epidemiology - are investigating aspects of these topics, often in collaboration.

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Differences also are likely between smokers in their physiological responses - how their bodies vary in susceptibility to the cancer-causing compounds in cigarettes - which implies that agents might be designed that help prevent cancer from developing or treat it more effectively if it does. To explore these topics, other teams of researchers in the Department of Epidemiology and the Department of Clinical Cancer Prevention are working together.

The Department of Behavioral Science is unique in the United States, says its chair clinical psychologist Ellen R. Gritz, Ph.D. "It is a fully established department, with resources and faculty, as opposed to a program, which many cancer centers have."

This department "focuses on the human side of cancer - the continuum from risk behaviors that cause or contribute to cancer to the psychosocial factors that affect treatment outcome, adjustment and survival," Gritz says.

"Our goal in smoking-related research is to detect those who are susceptible to nicotine, identify the best ways to prevent these persons from beginning to smoke and, if they do, determine how best to break the nicotine addiction that can result," Gritz says.

The Department's longstanding efforts in this field have helped the institution enroll thousands of smokers in numerous smoking cessation studies. Among their notable achievements to date are:

* Development of a "scheduled smoking" approach to quitting, in which a smoker is prompted by a hand-held computer to smoke on a schedule with increasing intervals between prompts;
* Creation of a teen-savvy computerized classroom program (ASPIRE - A Smoking Prevention Interactive Experience) that has resulted in lower rates of smoking in high school; and
* Increased smoking cessation among junior high school students using comput erized, personal health status feedback techniques.

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Work is ongoing on several dozen other tobacco-related studies. Among them are:

* A randomized, controlled trial in 16 Texas rural and urban communities, which aims to design and test an intervention protocol for training physicians and pharmacists to effectively counsel their patients for smoking cessation;
* A project which tests a motivational intervention protocol for smoking cessation among students at the University of Houston. Individual smoking cessation treatment sessions are combined with internet "cyber-support" available 24/7;
* Research to derive factors that predict onset of smoking in white, Hispanic and African-American youth;
* Multiple studies on the role of depression in smoking behavior and smoking cessation. For example, one involves tracking depression in pregnant smokers, based on earlier findings that depression makes it harder for smokers to quit;
* Research on special populations of smokers, including low income, multi-ethnic HIV-positive persons;
* Research that examines the cognitive processes underlying addiction, such as the physical response in the brain to drugs used to treat nicotine dependence; and
* An investigation of why some cancer patients continue to smoke, and how they can be helped to stop during treatment and throughout survival.

The risk-related and behavioral research methods used to study smoking have been adapted by researchers in the department to look at other preventable cancers, such as skin cancer, melanoma and colon cancer, Gritz says. Among these current projects are studies that develop "interventions" that reduce sun exposure in preschool children, as well as in high-risk melanoma patients and their first degree relatives. Another project is examining the psychosocial aspects of genetic testing and counseling for people with a genetic risk of developing colon cancer.

Prevention also means addressing the psychosocial needs of patients with cancer, with the goal of providing the best opportunities for regaining health. "About 15 percent to 20 percent of patients have emotional or psychological needs that have not been adequately dealt with," Gritz says. "We have been so focused on disease treatment that those important elements have traditionally been given lower priority."

In the Behavioral Research and Treatment Center, studies are carried out on tobacco prevention and cessation and a range of other behavioral and psychosocial research topics. These include social interaction, exercise and sleep patterns. Other clinical research projects in Behavioral Science focus on addressing sexuality following various cancers, discussing parenting post-cancer treatment and studying the role of acupuncture, yoga and Chinese medicine in the integrative medical treatment of cancer.

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Finally, Gritz plans to tackle ways to prevent cancers associated with obesity. Excess body weight is said to be responsible for about 10 percent of breast and colorectal cancers, and up to 40 percent of kidney, esophageal and endometrial cancers. "Finding ways to thwart the obesity epidemic that is arising in this country will not only help prevent these cancers, but other health issues such as heart disease and diabetes," she says.

Defining genes of risk and benefit

More than 80 percent of all lung cancer cases occur in people who have smoked cigarettes, but what accounts for the fact that only a small percentage of tobacco users will develop the disease? Why are some people more at risk?

That's one of the central issues being researched at the Department of Epidemiology, along with its corollary: why do some people with lung cancer fare better with treatment than others?

Now expand these questions into other tobacco-associated cancers such as those that occur in the bladder, kidney and esophagus, and to other non-smoking related cancers such as melanoma, brain, prostate and lymphoma, and that gives you an idea of the mission that Department Chair Margaret Spitz, M.D., has undertaken since 1995. "Although an element of chance is likely to play a role in the complex, multi-step process leading to cancer development, there is mounting evidence that genetic factors also influence susceptibility to cancer-causing exposures," she says.

Finding those genetic factors that determine risk of developing cancer, as well as those that confer benefit from treatment, is the focus of the 213 employees in the department - the largest in the division.

"The diversity of human beings is remarkable," Spitz says. "The fact that some smokers develop lung cancer while others don't suggests that there are differences among smokers in susceptibility to the cancer-causing compounds in cigarettes.

"Individuals respond differently to environmental exposures," she says. "They process chemicals differently, and they have a wide range of susceptibility to the undesirable side effects of treatments. Such differences could be explained by variation in our genes."

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Humans have a series of overlapping mechanisms to deal with the consequences of harmful environmental exposure, and these molecular pathways are all under genetic control, she says. "In our genes are thousands of small variations that may mean more - or less - production of an enzyme or protein that contribute to our diversity and explain our different risks of developing disease," Spitz says.

For example, her research has revealed a possible hereditary component to nicotine addiction and an inability to quit, showing some smokers receive more pleasure from nicotine than others because of genetic differences in the brain's dopamine reward pathway.

Another process under genetic control that could explain susceptibility is DNA repair capacity. These systems help maintain the integrity of genes by continually fixing the damage that occurs to DNA from exposure to harmful chemicals as well as to the daily assault of cosmic X-rays and UV light. If errors in this repair system occur, DNA damage can result in unstable genes and an increased cancer risk.

"Some people just have better DNA repair function than others," Spitz says. "If we can find out why, it may enable us to identify those at risk for cancer at an earlier age and to tailor intervention therapies for each individual."

Researchers in the department have studied variations in many DNA repair genes to see how they affect lung cancer risk. In these published studies, they report that patients with a variety of different cancers have significantly poorer capacity to repair DNA damage compared to those who do not develop the cancer.

Specifically:

* One finding demonstrated that individuals who don't eat enough dietary folate (a vitamin found in some fruits and vegetables), and who had genetic instability, are at much greater risk of developing bladder cancer. Folate is crucial to DNA synthesis and repair, and cigarette smoking (the major cause of the disease) puts this system under stress, the researchers say.

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The same genes that are implicated in cancer risk also may be involved in prediction of patient outcome, Spitz says. Among recent discoveries are that:

* Patients with esophageal cancer who had the best treatment outcomes were those that had gene variants that were less effective at neutralizing the killing power of cancer treatments. For example, patients treated with radiation treatment, who inherited less-effective variants of a gene (XRCC1) that repairs DNA damage from radiation, exhibited longer survival.

* People with more efficient DNA repair function who were given chemotherapy, particularly platinum-based drugs like cisplatin, had a lower overall survival rate than those with less efficient DNA repair.

While faulty DNA repair genes may put a person at risk for developing cancer, they also may benefit them when that cancer is being treated, Spitz says. "Such detailed genetic information can help us develop targeted interventions depending upon individual risk, which will promote cancer prevention and earlier detection as well as improve patient treatment and outcome."

Currently, researchers in the Department of Epidemiology are studying more than 3,000 patients diagnosed with lung, head and neck, bladder, kidney or esophageal cancer. Similar approaches are ongoing for other cancers including melanoma, glioma, lymphoma, and breast and prostate cancer. They ask these patients questions relating to their smoking status, diet, occupation, exposure to chemicals and family history, and then collect urine, blood and tissue cells.

From these samples, they are applying novel molecular "assays," or tests that gauge the biological importance of various genes or proteins.

Among the molecules being investigated in these assays are:

* Nicotine addiction genes;
* Gene variants involved in metabolism of chemicals, hormones and folic acid;
* DNA repair genes;
* Agents that push cells to mutate, or change;
* Length of telomeres (protein caps that stabilize chromosomes);
* Genes that control the cell cycle;
* Genes involved in inflammation;
* Methylation (addition of methyl groups that destroy gene function); and
* Genes that control a cell's "microenvironment."

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The combined findings eventually will provide a molecular road map to risk of cancer development as well as optimal cancer treatment. If oncologists knew who would be most susceptible to cancer development, it may be possible to use agents or behavior modifications as preventatives. If cancer does develop, oncologists may be able to tailor treatment to an individual's own genetic profile.

The department also has launched the first long-term effort to study health outcomes and risk factors in the Mexican-American population in the Houston metropolitan area, research paid for by philanthropy and tobacco industry settlement funds. Over many years, the study aims to enroll more than 100,000 Mexican-Americans in Texas, and to date more than 10,000 have joined. The study will follow the residents and collect biological samples to relate mortality and disease incidence to genetic, environmental and occupational exposures, diet, other lifestyle factors and health behaviors. A smaller five-year investigation, funded for $2.9 million by the National Cancer Institute, will specifically look at patterns of smoking experimentation and initiation in Mexican-American adolescents - why they begin smoking, how addiction sets in, what may help prevent their smoking and how to help these young smokers quit.

"We may one day be able to answer the 'why me' question - 'why did I get cancer' - or perhaps we might be able to prevent cancer from occurring at all," Spitz says. "It won't happen overnight, or even in my lifetime, but we're definitely moving in the right direction."

A daily dose of prevention

Will the patient of tomorrow be given a cocktail of daily drugs that will help prevent or reduce the chance of cancer developing?

This cocktail might include refined forms of anti-inflammatory drugs to prevent colon cancer, trace minerals to protect against prostate cancer, or proven versions of ancient remedies, such as turmeric spice for breast cancer and cups of green tea daily to repress oral cancer.

M. D. Anderson is devoted to finding preemptive strikes - ways to block cancer from ever starting or from becoming clinically apparent. These efforts are being spearheaded by Scott Lippman, M. D., chair of the Division's Department of Clinical Cancer Prevention, Levin and other researchers campus-wide.

M. D. Anderson was among the first to look for agents that may help prevent cancer - some three decades ago, beginning with the innovative work of Waun Ki Hong, M.D. - and now is seen as a national leader in the field of chemoprevention, Levin says.

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Four of five classes of chemopreventive agents the National Cancer Institute has said are promising and are "considered priority substances for study" are being investigated here. Some of the efforts involve national trials being led by M. D. Anderson researchers. Those compounds are retinoids, nonsteroidal anti-inflammatory drugs (NSAIDs), calcium compounds and selective estrogen receptor modulators (SERMs).

The research represents a completely new way of thinking about cancer, says Hong, head of the Division of Cancer Medicine at M. D. Anderson and a pioneer in the field. "Cancer doesn't begin with the appearance of a tumor, just as cardiac disease doesn't start with a heart attack," he says. "And just as we can control the risk of a heart attack with medication, we want to control the process of cancer development with drugs and supplements."

Hong launched the first chemoprevention clinical trial of its kind when he and a team of researchers demonstrated that smoking impaired the ability of vitamin A and its chemical cousin, retinoids, to keep cells healthy. In the early 1990s, they demonstrated that daily doses of retinoids could stop precancerous growths in the mouth and oral cavity from turning into cancer. They proved, for the first time, that cancer could be reversed. That work has led to examining other formulas of retinoic acids and other, unrelated agents.

Current strategies of "chemoprevention" - the use of natural or synthetic substances to reduce the risk of developing cancer - are less geared toward preventing all cancer than toward preventing specific major cancers, Lippman says.

"Great clinical strides have been made in breast, colorectal and prostate cancer prevention," Lippman says. For example, he says that tamoxifen (Nolvadex®) reduced breast cancer risk by 50 percent in the Breast Cancer Prevention Trial, and finasteride (Propecia®, Proscar®) reduced prostate cancer risk by 25 percent in the Prostate Cancer Prevention Trial.

But men and women haven't flocked to get prescriptions for either agent because, as Lippman points out, these two large-scale trials indicated that some serious side effects came along with preventive benefits. "This stand-off between agent risks and benefits has raised a major focus of cancer prevention - tailoring interventions to specific groups of people.

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"Efforts to identify people at a very high cancer risk and likely to benefit from and not be harmed by particular agents will be crucial to the future of cancer prevention," Lippman says. He leads an effort at M. D. Anderson and several other cancer centers to understand prostate cancer risk and how finasteride changed this risk in the Prostate Cancer Prevention Trial.

Other chemopreventive agents have proven to be effective. M. D. Anderson researchers have found that:

* A low-dose baby aspirin proved effective as a modest colon cancer chemopreventive. A randomized clinical trial of more than 1,000 participants found it reduced the number of precancerous polyps by 19 percent. Robert Bresalier, M.D., chair of the Department of Gastrointestinal Medicine and Nutrition, helped lead this national effort.
* Celecoxib, a non-steroidal anti-inflammatory drug (NSAID) known by the trade name Celebrex®, reduced the number of colon polyps in people who have familial adenomatous polyposis (FAP), in which hundreds of precancerous polyps form in the colon and rectum. The study, led by researchers at M. D. Anderson and St. Mark's Hospital, London, in collaboration with the National Cancer Institute, led to federal approval of Celebrex for FAP patients.
* Supplements of selenium and vitamin E unexpectedly reduced the incidence of prostate cancer by up to two-thirds in trials testing them for different cancers. These observations led to the ongoing international Selenium and Vitamin E Cancer Prevention Trial (SELECT) in more than 32,000 patients. M. D. Anderson leaders of SELECT include Lippman and Elise D. Cook, M.D., who led the national effort to recruit minority men, especially African-Americans, who have the highest risk of prostate cancer in the world. Cook's successful campaign resulted in the highest percentage of African-Americans ever recruited to a large-scale cancer prevention trial.
* The spice curcumin (found in turmeric and curry powders) has shown dramatic results in preventing cancer in animal studies, and has led to clinical studies at M. D. Anderson with patients that have pancreatic cancer or multiple myeloma. A trial with breast cancer patients is expected to begin in 2005. Patients in these trials take curcumin capsules daily.

A number of ongoing chemoprevention trials at M. D. Anderson are focusing on reducing chronic inflammation, which has lately been associated with cancer development. An agent of current interest in these investigations is Celebrex, given its proven ability to prevent inherited colon cancer. Celebrex works by blocking cycloxygenase-2, or "COX-2," an enzyme that is over-produced when cells become inflamed. Studies have shown, however, that many tumors, including those for small-cell lung cancer, also contain a lot of COX-2, possibly because of the body's natural immune reaction to the cancer.

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The precise way Celebrex, or any preventive agent, works is never simple, however. Novel laboratory studies of Imad Shureiqi, M.D., at M. D. Anderson show that 15-lipoxygenase-1 and related signaling pathways are at least as important as COX-2 to the workings of Celebrex and other NSAIDs. "M. D. Anderson is a world leader in research to better understand agent mechanisms," Lippman says. "And this research will hasten the arrival of safe and effective preventive drugs into the hands of the people who need them."

Some of M. D. Anderson's studies with Celebrex were halted after news that the agent's sister drug, Vioxx®, was associated with an increase in cardiovascular problems. Later, a slightly increased risk of cardiovascular disease was also found in one of the polyp prevention trials using Celebrex. Another multi-center international trial of Celebrex was also halted; this one evaluated the role of the agent in preventing recurrence of precancerous colon polyps and Levin is co-principal investigator.

After consultation with the National Cancer Institute, the following M. D. Anderson studies investigating use of Celebrex as a chemopreventive have been re-opened:

* An international trial in FAP looking at use of Celebrex combined with eflornithine (DFMO), a drug used to treat African sleeping sickness, but which is suspected of having anti-cancer properties. This study is led by Patrick Lynch, M.D., in the Department of Gastrointestinal Medicine and Nutrition.
* A clinical trial testing whether Celebrex can repair precancerous lung damage in current and former smokers. Jonathan Kurie, M.D., in the Department of Thoracic/Head and Neck Medical Oncology, is the principal investigator.
* An international trial testing use of Celebrex in children who are carriers of the mutated FAP gene, and who have little or no evidence yet of polyps.

Hong's work also has led to a major international program of M. D. Anderson in collaboration with Nordic investigators to prevent oral cancer with two molecular-targeted drugs (Celebrex and erlotinib, also known as Tarceva®) in people at extremely high risk of coming down with and dying from this disease. A molecular marker, aneuploidy (an abnormal amount of chromosomes in a cell), signals the risk of these people, "highlighting how important accurate risk detection is for effective chemoprevention," Lippman says.

As promising as some of the research has been, none of M. D. Anderson's chemoprevention experts, including Lippman and Levin, suggest that people take a little Celebrex here, a dose of aspirin there, or swallow tablets of curcumin with a dash of vitamin E as a way to "self medicate" against cancer.

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They all stress most chemoprevention studies now test people who are at higher risk of developing cancer, such as former smokers, as a way to predict whether they will help those who are not at risk. It will take decades, they say, to prove that any substance can substantially reduce the risk of a disease in the average person without producing side effects. These studies will require giving young and healthy volunteers a drug for many years and then waiting until they have aged to see whether volunteers who used the agent developed fewer diseases compared to those who didn't. "First, we must do no harm," Lippman says.

Levin emphasizes that chemoprevention must not be substituted for other important lifestyle habits such as avoiding tobacco, eating a nutritious diet, exercising and managing body weight: "If you do these things and make sure you are adequately screened, you may be able to reduce your odds of developing cancer by 50 percent - and that is a conservative estimate," he says.

"While we should be modest in claiming our work will lead to new chemoprevention advances in the coming years, we have promising leads from the laboratory that will enable us to conduct even better and more informative trials in the future," Levin says.

Offering cancer prevention to all

Preventing cancer with a pill is a nice idea that will likely take years to achieve, but there is much that can be done now to help many Americans, says David Wetter, Ph.D., chair of the fourth "arm" of the prevention division, the Department of Health Disparities Research.

Wetter is referring to the fact that "underserved" populations in the United States shoulder a disproportionate burden of cancer, and the researchers he leads are finding ways to reduce those inequities.

Wetter's new department, up and running only since April, builds on the pioneering work of the Center for Research on Minority Health, and reflects M. D. Anderson's dedication to addressing cancer in those who are disadvantaged. It is the only department of its kind in the country and the current staff of three faculty members is expected to triple within four years.

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Underserved populations today bear an unequal burden of cancer, with higher rates of incidence, severity and death, says Wetter, who has long researched smoking behavior in underserved and minority populations, as well the effects of gender on the ability to break the habit. Wetter has spent much of his career investigating new treatment approaches for smoking cessation, including palm top computer-delivered treatments, innovative telephone and face-to-face therapies, and meditation.

An example of such disparity is the fact that African-Americans have much higher rates of prostate cancer and Hispanic women have a greater incidence of cervical cancer, compared to other ethnic/racial groups. While biological differences may explain some of this increased burden, most is due to social inequities, such as lack of cancer screening and access to primary care, increased poverty and lower educational levels.

"We know that poor neighborhoods often do not have grocery stores with fresh vegetables and fruit, but only convenience stores that primarily sell snack food, cigarettes and alcohol," Wetter says. "There are often no sidewalks, so it is not safe to walk. These factors set up conditions that promote the development of cancer and other diseases.

"This is just a simple illustration of the pervasive problems that exist, and it is critical that we address health disparities in all its forms, from the molecular to the societal," he says.

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Examples of the kind of work that is already under way within the department are:

* The African-American Nutrition for Life Project, or "A NU-LIFE," designed to address the fact that breast cancer occurs more frequently in African-American women who have not experienced menopause than women of any other racial ethnic population. This four-year, $1.8 million study is following 200 Houston-area African-American women between the ages of 25 and 45 to determine if a low-fat, high-fiber diet affects breast cancer risk. According to Lovell Jones, Ph.D., principal investigator of the study and director of M. D. Anderson's Center for Research on Minority Health, the study will determine how levels of total body fat, dietary fat and fiber intake, circulating triglycerides and free fatty acids impact estrogen levels. Previous studies have shown that a high-fiber, low-fat diet can reduce estrogen levels in women and that women with lower estrogen levels have a lower risk of breast cancer.

* A research center focused on how environmental contaminants affect health and cancer risk. The largest study in this project, known as "EXPORT," examines biomarkers of genetic susceptibility to pesticide exposure in Mexican-American migrant and seasonal farm workers women and their children. Another aspect of the center is to look at the town of Fresno, Texas, and its almost 7,000 residents, of which 50 percent are Latino and 27 percent African-American. Fresno is located near two Superfund clean-up sites and a landfill, so researchers will try to determine the effect of environmental exposure on the health of these residents.

* A low cost intervention that uses a palm top computer to help smokers in the African-American community resist the urge to use cigarettes. The tiny computer delivers messages, personalized to the individual, to bolster the motivation to quit in the face of "real world stressors," according to Wetter, who leads the study. While the impact of socioeconomic disparities on cancer risk has long been recognized, the conditions are only getting worse, Wetter says. The underserved population is growing nationwide, and is especially prevalent in Texas and some other states, says Wetter. The Hispanic population is expected to become the largest ethnic group in the state soon, and this group is often not insured, he says. "Lack of insurance is a tremendous risk factor for cancer development because there is a lack of access to primary prevention, screening and care."

Research can parse out factors that may lead to improved population health, according to Wetter. "It must have a real world impact and will likely have policy implications and be community based," he says. "It is critical that we create an environment in which differences in health due to race, ethnicity, socioeconomic status, gender and other factors cease to exist."