Prostate Cancer

Prevention and Cure

Lee Nelson, M.D.

At the Prostate Cancer Foundation (formerly CaP CURE), we get a lot of calls for information on prostate cancer. From now on, we intend to tell callers to read this book first, then call back if they still have questions. This is the best book on prostate cancer that we've seen and we see almost all of them.

Howard Soule, Ph.D., Chief Science Officer,
Prostate Cancer Foundation

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by Lee Nelson, M.D.

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Introduction

At age 55, I underwent a routine PSA (prostate-specific antigen) blood test. I'd spent the previous five years living in remote parts of Asia where this screening test for prostate disease was unavailable. A visiting doctor friend of mine (general practitioner) had done a digital rectal exam (the dreaded "finger wave") three years prior, and he found that my prostate was generally enlarged, but there were no areas of hardness. The prostate border was smooth and regular. I'd had minor urinary symptoms for years, getting up two to three times each night to urinate. This was compatible with my age and the moderately enlarged prostate my doctor friend felt when he examined me, but it was certainly no cause for concern. Benign prostatic hypertrophy (BPH), as this condition is called, is quite common in men over 50 and usually responds to medicinal treatment.

I started taking a remedy containing saw palmetto oil to shrink the prostate gland. I also started taking a new over-the-counter "wonder drug" called DHEA. I really liked the effects of DHEA. I felt much more energetic and focused. As you'll read later, however, DHEA and prostate cancer don't mix.

When my PSA (prostate specific antigen) came back at 11.2 (normal is less than 4.0), I was shocked, to put it mildly. I had been extremely health conscious since graduating from medical school nearly 30 years earlier. I ate very little red

meat, used only "good" oils, exercised regularly, and took antioxidant-rich vitamin supplements. Surely this was a lab error. I repeated the test and the result was the same. From my medical training, I knew that at my age a PSA of this magnitude meant that the probability of prostate cancer was high.

I sought out a top-notch urologist who did another digital rectal exam (DRE). Unlike the one three years earlier, this test revealed an area of hardness in the right base of the prostate. A few more blood tests and a prostate biopsy later and the diagnosis was confirmed—prostate cancer.

I felt scared and confused. What to do? As I had done so often in medical school, I decided to find out as much as possible about this disease and its treatment. What I discovered was one of the most muddled areas in modern medicine.

Recognized experts in the field diametrically disagreed with each other as to the preferred treatment. To be sure, there were many options, but none was a clear-cut magic bullet. And there were many nasty potential side-effects to consider. Despite my medical training, given my level of confusion, I found it hard to imagine how someone who was not a doctor could successfully navigate the maze of options.

Indeed, most don't. They simply put themselves in the hands of their local urologist, do what he says, and hope for the best.

My purpose in writing Prostate Cancer—Prevention and Cure is to familiarize as many readers as possible with the current state of the art in the diagnosis and treatment of prostate cancer. This book is not only for someone who's been diagnosed with prostate cancer, but also for anyone who's a potential candidate for it—in other words, all men. It's also for women whose partners are either prostate cancer patients or who simply want to understand the disease in the event that it enters their lives.

In writing this book I reviewed more than 2,000 papers in the medical literature. Armed with this background information, I then spoke with many of the top doctors in the field of prostate cancer—urologists, medical oncologists, and radia-

tion oncologists. Each expert has his own view of the disease: which treatment to use, how to evaluate side-effects, and an overall approach, or philosophy, for dealing with prostate cancer at different stages.

However, this book has been written with the best interests of the patient as the overriding paradigm. I've tried to demystify the way doctors think in order to provide you with clearer insights, allowing you to take control of your illness. Then, once you've gotten rid of the cancer cells, I teach you the most optimal course for staying healthy.

The fact is, there's only one person whom you can rely upon to significantly improve your chances of cure from prostate cancer: You! Your active involvement in managing your disease is likely to increase your chances of success.

From this book and other sources, learn all you can. Ask questions, even if you think they might be dumb or embarrassing (such as those that concern sexual implications, incontinence, or bowel problems). Do not blindly accept medical pronouncements.

Optimistic and informed action is likely to improve your overall health and outlook.

Cancer is usually a defining event in someone's life. It can be viewed as a curse or a wake-up call. If you choose to behave as a passive victim, you are less likely to survive, and the quality of your life may erode. However, if you choose to view it as a catalyst for change in lifestyle and thinking, it can become a blessing in disguise. The course you set now will shape the balance of your life. It's up to you.

SECTION I:

WHAT IS PROSTATE CANCER?

Prostate Cancer Basics

In the year 2000, roughly 500,000 American men died of cancer. If you're between the ages of 45 and 64 your chances of dying from cancer are greater than from any other cause; if you are older than 65, cancer deaths are surpassed only by deaths from heart disease.

The two most common cancers for men are skin cancer and prostate cancer. Cancer of the prostate is second only to lung cancer as the leading cause of male cancer deaths. According to the American Cancer Society, in 1998 about 184,000 American men were newly diagnosed with prostate cancer and 39,000 died from it. In 2002, new cases were estimated to be 189,000, with 30,200 deaths. While new cases have increased slightly during the past four years, deaths from prostate cancer have decreased by about 23%. This dramatic decrease in death rate is probably attributable to both earlier detection and better treatment. If you're over 50, statistically you have a 50% chance of getting prostate cancer at some point in your life.

The prostate (not prostrate) gland is about the size and shape of a plump chestnut. The gland is surrounded by a protective sheath called the prostate capsule. The gland/capsule is located in the space between the bladder and the rectum.

The urethra, through which both urine and semen flow, passes through the middle of the prostate. When the size of the prostate increases due to either benign or malignant growth, it can compress the urethra, producing symptoms. These symptoms may include frequent urination, especially at night, start-and-stop urination, difficulty in starting the stream of urine, and reduction in the rate of flow.

The prostate is an expendable gland. You can live quite nicely without it. Its limited functions include providing fluid that may help transport sperm and protecting the urinary system against bacterial infections. It's not required for erections or fertility. As men age, the prostate becomes a liability.

Most aging men are well aware of the annoying symptoms of an enlarged prostate, which is caused by cell proliferation, whether benign (non-cancerous) or malignant. Benign prostatic hypertrophy (BPH), as it's called, is a common problem. Fortunately, the symptoms of BPH can usually be controlled by medicines or herbal formulas.

Another common prostate problem is prostatitis, or inflammation of the prostate. It's usually caused by bacteria. Often the invading bacteria are difficult to identify. Symptoms of prostatitis can mimic those of BPH or prostate cancer. Prostatitis can also dramatically raise blood levels of PSA (prostate specific antigen). Measuring the PSA is a screening test for prostate cancer (see "Screening and Diagnosis"). Prostatitis can sometimes be mistaken for prostate cancer; when the two occur together, prostatitis can make the cancer seem more advanced than it really is.

Prostate cancer may produce symptoms identical to BPH or prostatitis. But it can also grow for years without causing any symptoms at all. The cause of prostate cancer is unknown. About 9%-15% of the cases are hereditary. Inflammation from hit-and-run infections, hormone levels, and lifestyle factors like diet and exercise are thought to play a role in the development, growth, and spread of prostate cancer.

What all these possible causes have in common is that they damage the DNA, or the genetic blueprint, of prostate

cells. The DNA of our cells is perpetually being damaged by internal and external factors, and our cells are constantly repairing DNA damage. A delicate balance exists between factors that stimulate cell growth and agents that trigger cell death. This balance is controlled by proteins. The production of these proteins is controlled by the DNA of genes. One gene produces one specific protein.

Some of these proteins turn cells "on," some turn cells "off." If the DNA in particular genes is damaged, this fine on-off balance is disrupted. Genes that stimulate prostate cell growth may act unimpeded by genes that suppress cell growth, if these "suppressor" genes have been damaged (see "Risk Factors: Age").

For example, some genes control a built-in suicide program in cells. In due course they program cells to die. This process of programmed cell death is called "apoptosis." You will see this term often when you read about prostate cancer. When genes that control apoptosis are damaged, cells continue to live longer than normal. Much longer. New cells continue to be formed, but old cells don't die. This leads to clumps of cells growing out of control: cancer.

As these clumps of aberrant cells continue to grow, they require more nutrients. When a clump of cancer cells reaches no more than one cubic millimeter, about the size of the head of a pencil, it requires new blood vessels to provide nutrients for continued growth. The cancer cells secrete proteins that stimulate cells lining small adjacent blood vessels to grow, thereby creating new minute vessels. This process is called "angiogenesis" (blood-vessel development). It's important to understand that no cancer, no matter which kind, can grow without new blood vessels. In fact, one of the most fertile fields of anti-cancer research is the development of "anti-angiogenesis" agents, drugs that interfere with the ability of cancer cells to form new blood vessels. Prostate cancer is no exception. Anti-angiogenesis is discussed in detail in the chapter titled "New and Future Developments."

As prostate cancer cells continue to grow, cells can break

off from the clump and be carried away in either the blood or lymph systems. Most of these cells are killed by the body's immune system. Special lymphocytes (white blood cells) called T-cells, and other lymphocytes called natural killer (NK) cells, attack and kill these circulating cancer cells. But as their number increases, some cancer cells may slip through the body's defenses. They may wind up establishing themselves far away from their original source. Here they grow, forming new clumps and stimulating the formation of new blood vessels. These distant groups of cancer cells are called "metastases." These cells grow faster than the surrounding normal cells and don't die. Like weeds in a garden, they eventually crowd out the cells in the organs where they settle, compromising their function.

Prostate cancer has a particular affinity for bone. Most prostate cancer cells that break off of a clump wind up in the bone or bone marrow. In fact, the cause of death for most men who die from prostate cancer is complications from bone metastases. One of the most important goals in the treatment of prostate cancer, as you will read in the chapter on bisphosphonates, is to prevent it from getting established in bones. Accomplishing this goes a long way toward improving your chances of recovery.

Prostate cancer is not a single disease. In other words, it exhibits different characteristics in different individuals. This makes intuitive sense, since there are undoubtedly multiple causes. It's not surprising, therefore, that some men have very slow growing cancers and other men have aggressive cancers. Actually, the two are probably different diseases entirely. As such, it makes sense to treat them differently. We will discuss treatment options later. For now, suffice it to say that men who have slow-growing cancers have more treatment options and generally less disease than men with aggressive cancers. Determining whether you're dealing with a tortoise or a hare is a major role of the tests your doctor will order for you.

Prostate cancer usually develops in more than one location in the gland (multi-focal). By age 90, virtually all men

have microscopic pockets of prostate cancer. This has been confirmed by autopsy results. But most men die with their prostate cancer, rather than from their cancer. As you will read, there are things you can do, steps totally within your control, to delay the development and growth of this cancer. Understanding the risk factors and lifestyle factors that affect your chances is a good start.

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RISK FACTORS

Who gets prostate cancer? Who's at risk? Some risk factors, like age, race, and family history, are well-established. Recent studies also present strong evidence for dietary factors, hormonal influences, lack of exposure to sunlight, and environmental contaminants as significant influences on the incidence of prostate cancer. Some medical conditions, like diabetes and obesity, appear to predispose men to prostate cancer. Other potential risk factors, including vasectomy, baldness, and body type, are more controversial.

If you have known risk factors, you can increase your level of vigilance to reduce your risk. Annual PSA blood testing and digital rectal exam (DRE) from as early as age 40 can help in early detection and treatment of prostate cancer. Dietary and lifestyle changes may slow a developing cancer. Thus, there are steps you can take if you are at increased risk. Let's look at each risk factor.

Age

Not much you can do about this one. The older you are, the greater your risk of getting prostate cancer. You may have heard this truism: "All men will die from prostate cancer if they don't die of something else first." This indicates how common prostate cancer is in aging men. And it is common. Upwards of 20% of all American men will get clinically signifi-

cant prostate cancer sometime in their lives. Many more will have microscopic pockets of prostate cancer found at autopsy that caused them no trouble in life. In fact, virtually 100% of men lucky enough to make it to 90 have evidence of cancer in their prostate tissue. No other cancer is so prevalent. Yet most men die from other causes.

It's generally believed that these microscopic cancer foci are precursors (forerunners) of clinical prostate cancer. Interestingly, these small pockets of cancer occur pan-culturally—Japanese, Chinese, and Thai men are just as likely to harbor them as Swedes, Frenchmen, or African-Americans. But clinical prostate cancer is a far different matter. Here, other factors seem to come into play changing insignificant microscopic lesions into a potentially life-threatening disease. Both hereditary and environmental forces may influence this unwanted metamorphosis.

That brings us back to age. The chance of a man 39 years old or younger developing this active clinical form of prostate cancer is less than 1 in 10,000. From 40 to 59 the odds dramatically plummet, to 1 in 78. Between 60 and 79 they drop again, to 1 in 6!

Why does age make such a difference? A big part of the reason seems to be the increased tendency for DNA to be damaged over time. DNA controls all cell processes. It's the stuff that genes are made of—the building blocks of life. Destructive environmental forces, such as radiation, pesticides, heavy metals, and free radicals, damage DNA. As we've seen, our bodies are constantly repairing damaged DNA. But if destructive elements overwhelm our ability to mend DNA, cells become abnormal. Over time, this can lead to cancer. As we age, it appears, there may be a build-up of damaged DNA. Our bodies may not be as efficient repairmen.

One class of genes that's pivotal in keeping prostate cancer at bay is the tumor suppressors briefly mentioned earlier. There are at least several of these. As the mysteries of the human genome are unraveled (the complete genome has now been mapped), more of these tumor-suppressing genes are likely

to be discovered. The proteins produced by these genes are a large family of natural proteins that block numerous pathways involved in progression to malignancy.

One key suppressor gene is called p53; this gene is crucial for DNA repair. When it's altered, damaged DNA can't be mended. About half of the men with advanced prostate cancer have mutations (alterations) in p53. By special testing, changes in p53 can be detected in a prostate biopsy specimen. If p53 has mutated, the chance of the cancer recurring after treatment is higher. For this reason, some oncologists (cancer specialists) will treat patients who have altered p53 at the time of diagnosis more aggressively. So it may be worthwhile requesting this test when you have a biopsy that shows prostate cancer.

It's likely that advancing age increases the chances of gene mutations. A probable example of this phenomenon comes from a potential risk factor reported in the American Journal of Epidemiology in late 1999. Investigators from Boston University Medical School found that a father's age (not the mother's) at the time of conception affected the chances of his sons developing prostate cancer. Men who have male children early in life impart far less risk of prostate cancer to their offspring than men who reproduce later in life. Men aged 38 or more had about a 70% greater chance of having an affected son than men under age 27. Fathers between 27 and 38 had less risk of having an affected son than older men, but their risk was still 20%-30% higher than men whose age was less than 27 years old. The researchers postulated that the reason for this previously undiscovered risk factor might be genetic changes (mutations) to sperm cells due to advancing age.

Age may also predict the ability to cure prostate cancer. A recent study out of Johns Hopkins University discovered that older men who underwent radical prostatectomy (removal of the prostate by surgery) had less chance than younger men of being cured of their disease. In fact, the researchers were surprised to discover that age was a better predictor of disease outcome than the highly informative PSA levels. PSA is the global blood test used to screen men for prostate cancer.

Although controversial, it's generally agreed that PSA levels above 4.0 nanograms per milliliter (ng/ml) are abnormal. In men with prostate cancer aged 40 to 50 with PSA levels greater than 10 ng/ml and who had no detectable lump in the prostate on rectal examination, the chance of surgical cure was 73%. This compared with only a 49% chance of cure in men 61 to 73 years of age with comparable PSA levels, rectal exam status, and identical surgery. So advancing age seems not only to predispose men to the development of clinically significant prostate cancer, it also appears to reduce the chances of cure.

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Race

Race significantly influences the incidence of prostate cancer. Asians have very low rates, Caucasians intermediate rates, and African-Americans very high rates of prostate cancer development. The difference in death rates between African-Americans and American whites is surprisingly large. As a report from the National Institutes for Health (NIH) puts it, "The disparity in mortality rates from prostate cancer is greater between white and black men than for any other type of cancer in the U.S. and possibly the world." Several studies confirm that African-Americans have a 35% higher chance of developing prostate cancer, and a more than 220% chance of dying from it, than white men.

If the gap between African-Americans and western Caucasians is large, the disparity between African-Americans and Asians is cavernous. African-Americans are between 50 and

200 times as likely to die of prostate cancer than their various Asian counterparts.

Meanwhile, Asian men living in the United States have more chance of getting prostate cancer than men who remain in Asia. Put it all together and it's highly probable that both environmental and hereditary forces are at work in determining the racial make-up of who gets prostate cancer and who doesn't.

Why are African-Americans more prone to develop prostate cancer and much more likely to die from it than American whites? This is a hotly debated topic in the corridors of a number of centers for prostate cancer. The definitive answer remains elusive. A combination of factors seems to be involved.

One element that has received a lot of attention in medical publications of late is that African-Americans are less likely to be screened for prostate cancer. This may be due both to a lack of awareness and a lack of access to PSA blood testing and digital rectal exams. Without proper screening, black men often have significantly more advanced and aggressive disease at the time they first seek medical attention. Often they will have troublesome symptoms that lead them to seek medical help.

One multi-institutional study from North Carolina showed a statistically significant inverse correlation between income level and health-insurance status and advanced prostate cancer in black men. The study found that black men with lower income levels and/or no health insurance were more likely to have advanced cancer than those with higher income and health insurance. No such correlation was observed in white men in North Carolina.

Another study showed significantly lower levels of literacy in black men showing up at the doctor's office with prostate cancer than in whites. In a study of 212 lower-income-level men diagnosed with prostate cancer in Louisiana and Illinois, only 48% of African-Americans possessed at least a sixth-grade literacy level; 91% of Caucasian men of comparable income

had achieved at least sixth-grade literacy. Interestingly, in this study, when adjustments were made for literacy, age, and geographic location, race was no longer a predictor of advanced disease. The authors postulate that low literacy rates in African-Americans may lower their awareness to the availability of screening. They suggest that literature designed specifically to literacy level might go a long way toward rectifying the increased risk among black men.

Although screening is getting the lion's share of racial attention lately, it's unlikely to be the whole story. For one thing, African-Americans had higher incidence and mortality before screening became popular. Also, researchers at Wayne State University's Harper Hospital led by Dr. Powell found that black men have a greater risk of recurrent cancer than white men if the cancer has spread beyond the prostate. These investigators observed that African-American men and Caucasians develop prostate cancer at similar ages. But across all age groups, black men have metastatic cancer much more often than whites. They concluded that the reason black men have worse outcomes is due to more aggressive cancers in black men under 70 years of age. Why? See below.

These findings were confirmed by research from the Southwest Oncology Group (SWOG). At the May 2000 meeting of the American Urological Association (AUA), Dr. Thompson revealed that in the SWOG data black men had more extensive disease and more bone pain than whites. Additionally, race independently predicted survival outcome when variables like age, bone pain, etc. were factored out.

Besides race, other considerations that might affect risk in African-American men include diet, hormonal influence, infections, and response to sunlight.

There is now considerable evidence, though not yet conclusive, that consuming fat may stimulate changes from dormant to active prostate cancer. Some investigators have noted that African-Americans have more fat in their diets than whites. Similarly, whites take in more fat than Asians. Mortality from prostate cancer correlates with fat intake.

Not only the amount of fat in a man's diet may predispose him to prostate cancer, but how much its cooked also seems to make a difference. Cancer-causing compounds, called heterocyclic amines (HAs), are formed when meat is cooked. According to a recent study, meat type, intake rate, cooking method, and meat doneness all influence the amount of carcinogenic HAs that are formed. Pan-fried meats produce the most HAs. Surprisingly, chicken (with skin) is the largest source of HAs among the different types of meat.

This study showed that African-American men over age 30 consume about three times as much HAs as whites. A national survey showed a preference amongst African-American men for well-done meat, which increases the HA content. About $^{2}/_{3}$ of HA content is comprised of a compound that causes prostate DNA to mutate and induces prostate cancer in rats.

A recent study by the National Cancer Institute (NCI) showed that increased amounts of animal-fat intake doubled the risk of blacks getting prostate cancer, but did not significantly increase the risk in whites. This lends support to the idea that cooking with its increased HA content is a significant risk for prostate cancer and predominately affects black men.

The risk of prostate cancer progressing to advanced disease is also increased by animal-fat intake, but in this case both blacks and whites with comparable fat intake are equally affected. Since HAs have been implicated in the initiation of prostate cancer and the amount of animal fat consumed has been associated with the cancer progressing and spreading, this NCI finding makes sense.

Young black men have been found to have a higher level of circulating testosterone than white men of equivalent age—about 15% higher. Testosterone levels also appear to decrease more slowly in aging black men. Although these hormonal differences may account for some of the black-white difference, this has not been proven.

African-American men have a higher incidence of a known prostate cancer risk factor called prostatic intra-epithelial hyperplasia (PIN). Thought to be a precursor to cancer,

PIN is an area of inflammation and cellular growth within the prostate. It may be part of a process that starts with an infection in the prostate and becomes an area of smoldering inflammation. Then, under hormonal influences primarily from DHT and estrogens, DNA changes may occur. A study by Dr. Sakr at Wayne State University noted that blacks had more areas of PIN in their prostates and that the PIN tended to be of higher grade (more abnormal) than in a comparable group of white men. This difference started when men were in their twenties.

The researchers compared the prostates of black and white men in several ways. At autopsy they found that extensive higher-grade PIN was present in 7% (25/364) of black men less than 50 years of age compared to 2% (4/208) of white men. When they examined prostates removed surgically in a group of 1,200 men, they consistently found more extensive and higher grade PIN in African-Americans, especially in those under 50. In men with disease discovered by PSA testing and no evidence of cancer upon digital rectal exam, 33% of blacks had extensive high-grade PIN compared with only 12% of whites, a highly significant difference.

Dr. Sakr concludes: "Our findings suggest an important role for high-grade PIN in the development of clinically significant, potentially aggressive prostate cancer in African-American men."

Yet another ingredient that may influence African-American susceptibility is response to sunlight. Here, there is some conflicting data. While blacks seem to have reduced ability to convert sunlight into vitamin D, recent data show that vitamin D levels are equivalent in black men whether or not they have prostate cancer. Vitamin D is thought to play a protective role in halting the growth of prostate cancer. What significance, if any, it has in relation to the higher death rate from prostate cancer in African-Americans is unclear. It seems prudent, however, for all men with prostate cancer to periodically check their vitamin D blood levels. If low, whether the man is black

or white, he'll probably benefit from supplemental vitamin D. The active form of vitamin D, known as calcitriol (Rocaltrol), is available only by prescription and must be taken under doctor's care (see "Sun Exposure" later in this chapter and the section on vitamin D in the "Nutrition" chapter).

A great deal of work is being done to reduce the risk of death from prostate cancer in African-American men. One technique that shows great promise is interactive screening. Dr. Myers and colleagues report in a recent issue of the journal Cancer that understandable printed material, combined with telephone follow-up, lead to a far greater turnout for a screening examination and PSA blood test when compared with men who only received a letter inviting them to be screened. Undoubtedly better screening will help close the gap between black and white men.

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Heredity

Regardless of race, the risk of prostate cancer escalates for a man when his immediate male relatives (father, son, or brother) are diagnosed with this disease. If your father (or son) is affected, your risk doubles. If your brother is affected, your risk triples. One study reported that if your brother or father got prostate cancer at 50 or younger and another first-degree relative (brother, father, son) is also diagnosed, your chances of developing prostate cancer increase seven-fold!

Overall genetic factors are believed to account for about 9%-15% of all cases of prostate cancer. However, for men under age 55, the chance of genetically determined disease skyrockets. In this group 43% of all instances of prostate cancer are thought to be due to genetic factors.

Due to the increased risk, for men in families with known prostate cancer, especially when the onset in a close relative occurs early in life (age 50 or less) or if more than one first-degree relative is involved, initial screening for prostate cancer should begin at age 35. Normally, screening is not recommended until age 50.

Generally, although hereditary prostate cancer starts earlier

than non-hereditary prostate cancer, it does not appear to differ significantly in its characteristics or survival patterns.

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Obesity and Fat Consumption

Besides being at greater risk for heart disease and diabetes, obese men appear to be more vulnerable to aggressive prostate cancer than men who are overweight, or of normal weight. Dr. Amling and associates reported at the AUA meeting in May 2000 that 20% of a group of 860 men who had surgery for prostate cancer were obese. These men had significantly more aggressive prostate cancers on average, they got cancer much earlier in life, and they had more advanced cancer than slimmer men. Dietary factors are an obvious place to begin in accounting for these differences.

Obese men have high levels of insulin-like growth factor-1, a hormone that has been associated with markedly increased chances of developing prostate cancer (discussed in depth later in this section). If the increased risks of heart disease, diabetes, and other debilitating conditions are not persuasive enough to compel obese men to lose weight, perhaps the increased risk of dying from prostate cancer added to the list will help provide sufficient motivation.

One popular theory floating around the halls of prostate cancer academic centers is that various kinds of fat might stimulate the change of dormant prostate cancer to the more

dangerous clinically significant form. Since microscopic prostate cancer is essentially equally prevalent for all cultures, diet, specifically fat consumption, is one element that might make a difference. A 2002 study from the Fred Hutchison Cancer Research Institute in Seattle, Washington, shows an association between fat intake and total caloric intake, not with the initiation of prostate cancer, but rather with its spread (see "Nutrition").

Fat intake is discussed in detail in the chapter "Nutrition." Suffice it to say here that animal fat (saturated fat) and polyunsaturated oils containing either linoleic acid (corn oil, soybean oil, safflower oil, etc.) or linolenic acid (flax oil) are potential contributors to the promotion of prostate cancer. Char-broiling or frying animal or fish fat produces cancer-causing substances known as heterocyclic amines that cling to the surface of fat molecules. Heavy ingestion of these carcinogens may increase risk.

In addition, nutrients that have medical evidence to support a protective effect against damage from fat ingestion include selenium, vitamin E, soy protein (but not soybean oil), green tea, cruciferous vegetables and sprouts, silymarin, and curcumin (see "Nutrition"). Olive oil and oil from fish appear to be helpful. However, since olive oil is high in calories, it's probably best to use only the necessary amounts for cooking and salad dressing. Olive oil should be the predominant oil consumed. Other beneficial oils are macadamia nut, avocado, and walnut oils.

HELPFUL HINT

Avoid eating animal fat. If you eat fish, eat it raw (sushi and sashimi), steamed, or poached, to minimize heterocyclic amines consumption.

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Environmental Hazards

More and more evidence is now coming out on the link between environmental contaminants and cancer. In mid-2000, the Washington Post published a summary of a report yet to be released from the Environmental Protection Agency (EPA) on dioxin. Dioxin is a bi-product of waste incineration, as well as paper-pulp production and other industrial sources. You may be aware that Agent Orange, the defoliant used in the Vietnam War, has been shown to significantly increase the risk of a variety of cancers. Dioxin is the prime active ingredient of Agent Orange. The EPA now concedes, according to the Post, that dioxin is a "human carcinogen."

Accumulating in animal fats, fish fats, and dairy products, dioxin is ubiquitous—people worldwide have measurable blood-dioxin levels. This toxin accumulates over time and the effects are cumulative. The EPA now estimates the risk of cancer from dioxin consumption to be 10 times as high as previous estimates. Cancer of the lung and lymphoma are increased by dioxin exposure. Since toxin exposure is usually not organ-specific, it's likely that dioxin increases the chances of getting a variety of cancers. I would not be at all surprised to ultimately discover that prostate cancer is one of these.

Another risk factor for cancer is traffic pollution. Two recent Scandinavian studies showed a clear-cut increase in lung cancer in people exposed to prolonged periods of heavy traffic. In a Swedish study, 30-year exposure resulted in a 40% increase in the chances of getting lung cancer for both smokers and non-smokers. Ten years of exposure increased lung cancer probabilities by 20%.

One contaminant in auto-exhaust fumes is cadmium, a heavy metal. Although cadmium in polluted air has not been directly linked to prostate cancer, prostate cancer is an

occupational hazard for workers exposed to cadmium in battery manufacturing. Other substances that increase the risk of prostate cancer are pesticides, metallic dust, liquid fuels, lubricating grease and oil, and aromatic hydrocarbons. Dr. Kristan Aronson found that in Montreal, Canada, men working in aircraft manufacture, gas and water utilities, and around jet fuels had an increased risk of prostate cancer. Farmers regularly exposed to pesticides and herbicides also appear to be more vulnerable. Dr. Aronson estimates that about 10% of prostate cancer cases are due to occupational hazards, primarily exposure to environmental toxins.

Lack of Exposure to Sunlight

As you'll read in the Nutrition chapter, there is considerable evidence that vitamin D may retard the growth and spread of prostate cancer. Vitamin D production depends upon the skin's exposure to sunlight—specifically UV-B rays, according to Dr. William Grant. Demographically, the incidence of prostate cancer in the United States increases incrementally as you head south to north. The farther north you live, the greater your chances of getting prostate cancer. This may be due to a deficiency of vitamin D.

Although most studies of cancer and sunlight use average yearly amounts, Dr. Grant believes that lack of winter sun is the key variable. The long northern winter may deprive men of UV-B rays, impairing their ability to make vitamin D. This may account for increases in prostate cancer oc-

HELPFUL HINTS

Avoid walking or jogging in areas with heavy traffic. Select organic produce whenever possible to reduce pesticide ingestion. Thoroughly soak all fruits and vegetables in water before eating. Peel apples, pears, persimmons, and peaches, if not organically grown. Buy organic strawberries whenever possible. Straw-berries, although rich in a nutrient associated with a reduction in prostate cancer, are one of the most highly sprayed fruits.

currence in northern areas. By the way, according to Dr. Grant, winter UV-B exposure also reduces the risk of colon and breast cancer. It appears that those winter vacations to sunny destinations may have even greater benefits than mere stress reduction.