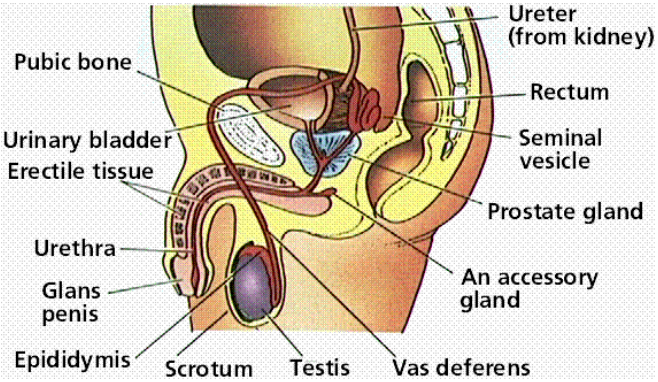
PROSTATE – MEN'S HEALTH



Normal function of the male genitourinary tract is essential to the overall health of a man's body. Because this system is susceptible to a number of conditions and disorders, special attention must be paid to its health in order to maintain healthy sexual function, proper elimination, and general vitality.

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Books: ***The China Study*** **by T. Colin Campbell**
Prostate Health in 90 Days:
http://www.mercola.com/forms/prostate_health_90days.htm

When the Body Says No **by Gabor Mate, MD**

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A MUST-SEE FILE: CANCER IS A FUNGUS

Source: David Rainoshek, M.A. research

This file is based on the work of two extraordinary researchers in the field of cancer research and treatment: Dr. Tullio Simoncini and Dr. Mark Sircus.

Both Drs. contend that *cancer is a fungus*, and can be treated and prevented with natural fungal-fighting agents such as sodium bicarbonate (baking soda) (also Nascent Iodine and Magnesium).

I have made a file on Dr. Mark Sircus' writing and research on the subject, which Members of Juice Feasting can download here:

Cancer is a Fungus

I also highly suggest you purchase and read these books:

***Winning the War on Cancer* by Mark Sircus**
***Sodium Bicarbonate: Rich Man's Poor Man's Cancer Treatment* by Dr. Mark Sircus**
***Cancer is a Fungus* by Dr. Tullio Simoncini**
***Iodine: Why You Need It* by David Brownstein, MD**
***Magnesium Medicine* by Dr. Mark Sircus**
***Rainbow Green Live Food Cuisine* by Dr. Gabriel Cousens, MD**
***Sick and Tired: Reclaim Your Inner Terrain* by Robert O. Young**

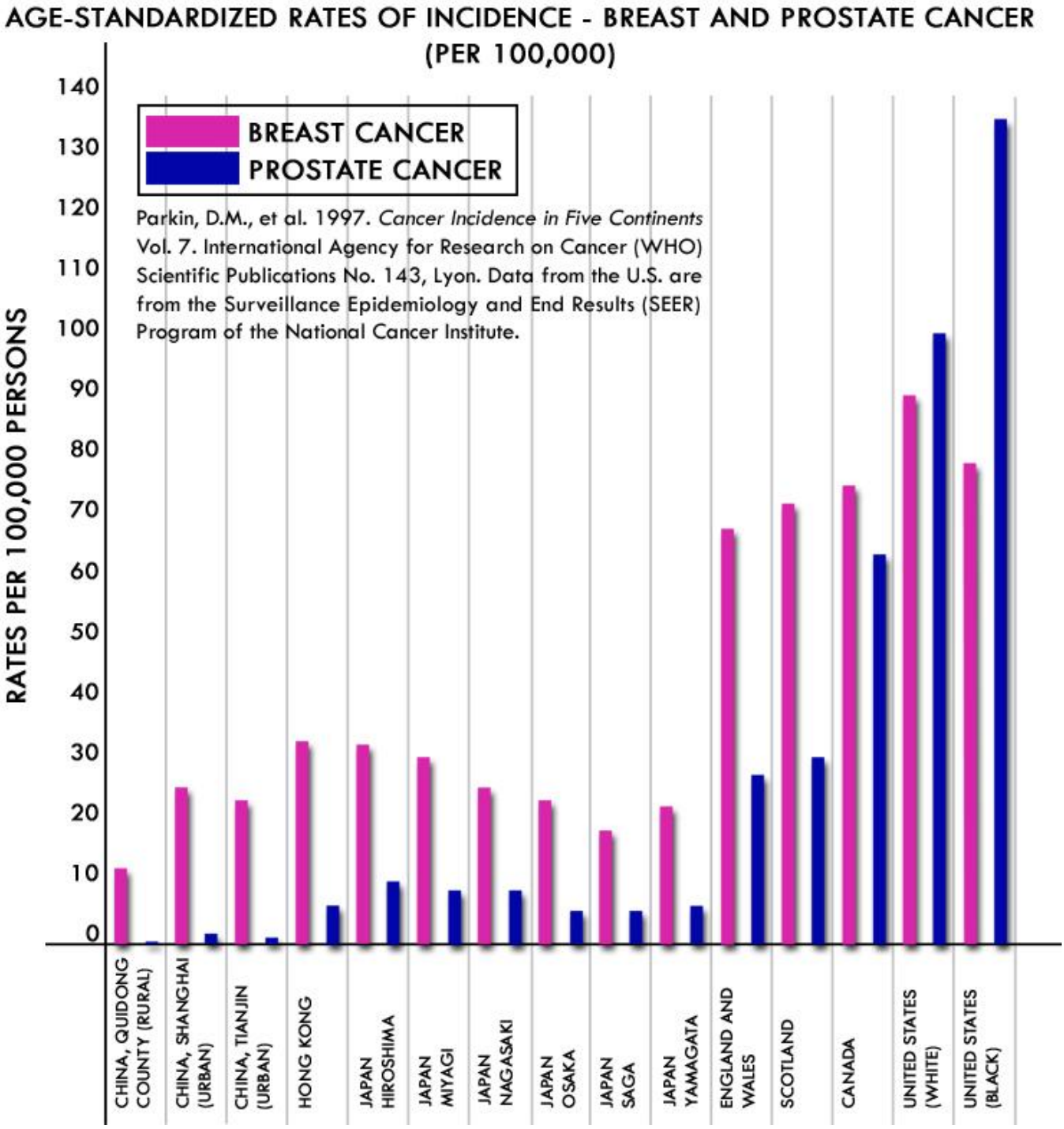
These will all give you an excellent idea about the ubiquitous disease-causing nature of fungus in our bodies, and what to do about it.

Blessings to you,

David Rainoshek, M.A.

www.JuiceFeasting.com

July 11, 2012



MANopause

By Lisa Marshall

Source: Alternative Medicine Magazine, June 2007

http://www.alternativemedicine.com/common/news/

It's no joke. Men go through the change of life, too. Although male menopause has been under the radar for years, the condition has gained credibility in recent years. What's a guy to do?

So you feel hot under the collar these days, but seldom under the sheets. Your middle is getting softer by the day, and your mood swings and night sweats are enough to drive your partner to the couch.

I know what you're thinking: It must be menopause.

But wait, you're a guy. Could you too be in for a "change of life?"

"This is coming for the Baby Boomers, and we are going to see an explosion of interest in it," says John Morley, MD, head of the geriatrics division at Saint Louis University Medical School, and a leading researcher on the subject of so-called "male menopause."

Scientists have long known that a man's testosterone level begins a slow downhill slide as early as age 30, dropping 1 percent a year on average after the age of 50. Add that to the fact that other sex hormones and brain chemicals also begin to fluctuate, and middle-aged

men can quite possibly look forward to an array of "change of life" symptoms, including loss of muscle mass, fatigue, depression, erectile dysfunction, and even hot flashes. But because the condition is exceedingly hard to test for—and historically difficult to discuss—it has remained under the radar for doctors and patients alike, says licensed psychotherapist Jed Diamond, author of the sentinel book *Male Menopause* (Sourcebooks,1997). "If you go to your average doctor as a guy and say 'I think I'm going through male menopause,' most of them will still laugh at you," says Diamond. "Men tend to deny anything in themselves that is remotely feminine."

As studies begin to link low testosterone with heart disease, cognitive decline, and bone loss, and aging Baby Boomers still insist on a thriving sex life well into their Golden years, organizations like the National Institutes of Health and the American Association of Clinical Endocrinologists have called for more research into the phenomenon of waning testosterone.
The burning questions: When and how should manopause be treated? And is treatment safe? Meanwhile, thousands are opting not to wait for the answer; instead they're flocking to specialty clinics for everything from testosterone shots to acupuncture and herbal supplements.

"I hit a wall and traditional medicine just couldn't help," says 50-year-old California filmmaker John Upton, who declined recommendations of anti-depressants and instead turned to Diamond for help. "I found myself, at 48, carrying around 70 pounds of body fat, not being able to get an erection, not feeling good, divorced twice and not in good shape at all. Jed said go to a doctor and get your hormones checked. I did, and they determined my testosterone was low."

What's in a name?

Despite more than three-dozen clinical trials on the subject and scores more in the works, the notion of male menopause is far from universally accepted. A widely heralded report commissioned in 2004 by the US National Institute of Aging and the National Cancer Institute concludes, "there is scant evidence that male menopause exists," and points out that "the likelihood a man will ever experience a major shut-down of hormone production similar to a woman's menopause, is remote." It also called for more research.

At the root of the controversy, says Morley, lies semantics: Because menopause, by definition, means the end of menses, calling the male experience by the same name often ruffles feathers. Some call it hypogonadism, which means low hormone production, but that can occur in men of all ages. Others call it andropause. Morley prefers Androgen Deficiency in the Aging Male (ADAM). "It will never be fully recognized until people can find one title they can agree on," he says.

Although menopause comes on fairly rapidly for all woman, halting production of progestin and estrogen and spelling the end of fertility, the male process comes on subtly and varies in severity, depending on the man's lifestyle, experts say. "It is a very slow, insidious, hard-to-figure-out process," says Todd Dorfman, MD, a Boulder physician who specializes in treating male menopause. "Men come in with one or two issues, (libido problems are typically Number One) and I have to drag the rest out of them."

And while the female "change of life" can lead to fairly specific health issues, such as rapid bone loss and hot flashes, linking testosterone-loss to conditions like weight gain, ED, and depression—all of which can have numerous other causes—can be tricky. Because men, even in their 30s and 40s, often turn to potentially-risky testosterone treatments to quell those changes (see "Hormone Replacement Therapy for Men?" on page TK), the subject remains controversial. "A lot of patients come to me and they are already on testosterone and they have never even had their levels measured," says Mark Carney, ND, LAc, of Denver, Colorado. "In my opinion, that is very poor medicine."

The test

So just how can you know for sure if low testosterone is the problem? Another tricky question. Testosterone levels normally fluctuate throughout the day (higher in the morning), from season to season (highest in the fall; lowest in the spring), and can vary according to stress levels and diet. So you can have a hard time distinguishing whether testosterone levels have truly dipped, or if you took the test at an inopportune time.

Assuming that you truly have age-related testosterone loss, another question then arises: How low is too low? A "normal" healthy adult male's total testosterone concentrations can

range anywhere from 300 ng/dL to 1,000 ng/dL. Those with levels of 200 ng/dL to 319 ng/dL are good candidates for therapy.

By those measures, one in 10 men between the ages of 40 and 60 has abnormally low testosterone levels, and after 75, the ratio rises to 3 in 10.
But because some men naturally produce more testosterone in their youth, those benchmarks can be misleading, says Diamond. "Let's say you have a guy in his 20s who has a testosterone level of 1,000 and by the time he gets to be 50, [levels drop] down to 500. He has lost half of his testosterone and is likely to have symptoms. On the other hand, you might have a guy who is at 400 in his 20s and drops to 250 (considered abnormal). He may not have any symptoms."

Dorfman says he runs an array of blood tests, asks patients to fill out a lengthy lifestyle questionnaire, and sits down for an in-depth interview with each one before making a diagnosis. If he can blame age-related hormone changes, he says he has good news: "It can be forestalled, and it can be reversed."

Natural solutions

To start building hormone levels naturally, look to the following three options as a good, safe way to start.

• **Exercise.** "There is a direct relationship between muscle mass and testosterone. One natural way a person can raise his testosterone levels is by getting into a weight-lifting program," says Carney, a naturopath who specializes in men's health. Studies have shown that as few as two sessions of strength training per week can increase muscle strength by more than 30 percent, while also boosting bone density (another victim of declining testosterone), speeding up metabolism, and pushing up production of testosterone and other sex hormones. Exercises that target several large muscle groups (like squats or bench presses) boost testosterone levels more than those that train isolated muscles (like curls).

Meanwhile, aerobic exercise boosts the production of feel-good neurotransmitters in the brain, which also have a tendency to get thrown out of balance as men age. So having a well-rounded exercise program helps.

Keeping weight in check also makes a difference, says Diamond. Because fat cells tend to convert circulating testosterone into estrogen, having too much fat around the middle can sabotage what little testosterone the body still produces. "If you are overweight, you are really working against yourself."

On the flip side, over-training and under-eating can also wreak havoc on testosterone production.

In one study, volunteer male soldiers undergoing an intense, eight-week training course also ate a restricted-calorie diet (about 1,200 calories less than what they needed). Their testosterone levels dipped to "castrate levels," far below normal, while their levels of sex hormone binding globulin (SHBG) which binds to testosterone and makes it less available to the body, went through the roof. Once they started getting enough calories again, their levels returned to normal.

• **Nutrition**. Men should also eat enough good carbohydrates, protein, and good fat, says Carney. Research shows that protein helps maintain lean muscle mass. Lack of carbohydrates can lead to decreased serotonin and, consequently, irritability. And it takes a certain amount of fat to keep testosterone production at healthy levels.

One recent study of 36 middle-aged, white, healthy men showed that switching to a strict, low-fat diet for eight weeks reduced circulating male hormone levels by 12 percent on average. Generally experts recommend that men get roughly 30 percent of their calories from fat (albeit good fat like that found in nuts, oily fish, and olive oil.) "Cholesterol is a building block of many of the hormones and if you don't have enough of it, you can't build the house," says Carney.

Another key piece of dietary advice: Cut back on the alcohol, which studies show also decreases testosterone levels. "Contrary to what many men think—that a few drinks make them sexier—they are really taking away their testosterone," says psychotherapist Diamond, who is also a Certified Addictions Counselor.

• **Herbs and Supplements.** Schuyler McHenry, ND, of Southwest College of Naturopathic Medicine, recommends B vitamins, which can help with stress and boost energy; C vitamins,

which can stabilize production of stress hormones; and herbs such as ashwagandha and ginseng (see "10 Herbs and Supplements to Quell Male Menopause Symptoms" on page TK).

McHenry also recommends acupuncture, herbs and Chinese patent formulas aimed at strengthening the kidneys, which are considered the hearth of male sexual energy in Chinese Medicine. "If there is anything that damages the kidneys, it can lead to weakness of the sexual organs," McHenry says.

Perhaps the Number One over-the-counter dietary supplement for addressing male menopause is dehydroepiandrosterone (DHEA), a building block for sex hormones that the body has naturally, but that also tends to decline rapidly with age. Sales of DHEA supplements (often derived from yams) jumped from just $1 million in 1998 to $48 million in 2004, according to *Nutrition Business Journal*, as studies have suggested it can improve skin, sex drive, mood, and strength in aging men. However, practitioners warn that DHEA is a hormone, and overuse of hormones can result in serious side effects. So before adding DHEA to your daily supplement regimen, have your blood DHEA levels tested and then have them retested periodically once you start taking it.

Other popular supplements used for male menopausal symptoms include fish oil, or omega-3 supplements, which have been shown to improve cognitive function, boost energy, and prevent heart attacks, and L-arginine, an amino acid that helps dilate constricted blood vessels associated with erectile dysfunction.

Bringing it all together

For John Upton, a combination of treatments has been the key to good health. He started with acupuncture, which he says lifted the "fog" he'd been in for years. "I remembered what it was like to be hopeful again."

Today, he takes dozens of dietary supplements daily, spending between $300 and $500 per month, eats a high-protein, low-glycemic index diet, lifts weights regularly, sees a counselor, and injects prescription testosterone to keep his levels within normal range. "The difference is stunning," he says. "I'd never want to go back."

Just how many American men are willing to go to such lengths, expense, and potential risk to slow down the ticking clock? That remains to be seen. But whether they should will likely remain a hot topic of conversation for some time.

Lisa Ann Marshall is a contributing editor for Alternative Medicine.

10 Herbs and Supplements to Quell Male Menopause Symptoms

DHEA (dehydroepiandrosterone). An over-the-counter supplement designed to mimic natural hormone building blocks, which decline in the body with age. Improves mood, exercise capacity, sex drive, and skin conditions such as lupus.

Maca (*Lepidium meyenii, L. Peruvianum*). A root used for centuries in Peru for its fertility and libido-enhancing properties.

Horny goat weed *(Epimedium sagittatum, D. Grandiflorum).* A Chinese herb, also called *yin yang huo*,used to increase libido and address erectile dysfunction and premature ejaculation.

Yohimbe (*Pausinystalia yohimba).* Some studies show that yohimbe, which comes from the bark of an African tree, can be effective in addressing erectile dysfunction.

Ginseng: This age-old standby promotes energy, stamina, and endurance, affects hormonal imbalance, and nourishes the kidneys, considered vital organs for supporting sexual health.

Ashwagandha (*Withania somnifera*). A powdered root used in the East as an aphrodisiac for 3,000 years.

Damiana *(Turnera diffusa; var. T. Aphrodisiaca).*A mood-elevating aromatic herb that helps calm anxiety.

Chaste tree (Vitex agnus-castus). Historically used to reduce male libido in monks and others entering the priesthood, it has since been used to help normalize hormonal changes

associated with male menopause.

L-arginine. An amino acid that helps dilate constricted blood vessels associated with ED. Should not be used in people who have had a heart attack

Fish oil or omega-3 supplements. Either improves cognitive function, prevents heart disease, and provides an energy source.
Sources: *The Male Herbal* (Sourcebooks, 1997), by herbalist James Green; Mark Carney, ND.

Hormone Replacement Therapy for Men?

By far the most controversial treatment, prescription testosterone, comes in the form of twice-monthly self-injections, prescription gels, or skin patches. According to IMS Health, a pharmaceutical market research firm, sales of prescription testosterone soared to $568 million in 2006, nearly double what they were in 2002, and with a host of new easier-to-use products in the pipeline those numbers will surely continue to rise.

Some studies have shown that supplemental testosterone can indeed restore sexual function and muscle strength, improve memory, prevent bone loss, and possibly protect against heart disease. But supplemental testosterone has potential risks: Too much can trigger aggression and cause breast enlargement; it also thickens blood, potentially increasing the risk of stroke; and it has been shown to cause sleep apnea in some men.

Although research to date remains inconclusive, some also fear excess testosterone may fuel the growth of prostate cancer. That concern has prompted many researchers—fans and critics of testosterone replacement alike—to call for more long-term health studies like the Women's Health Initiative (which ultimately exposed the risk of hormone replacement therapy in women). "We are lacking the Women's Health Initiative equivalent for men and we need that," says John Morley, MD, head of the geriatrics division at Saint Louis University Medical School. "Everybody knows that until we do one large study that includes side-effects, we won't have a clue."

Many experts say they prescribe testosterone in cases when needed, but only after rigorous

testing. Todd Dorfman, MD, a Boulder physician who specializes in treating male menopause, says that in some cases he can use other, more benign synthetic hormones, such as a self-injected "luteinizing hormone" intended to amplify the signal from the pituitary to the testes and jump-start the body into making its own testosterone. That way, he says, "I'm using the patient's own physiological mechanism to get him to produce his own testosterone"

When he does put men on testosterone supplementation, he prescribes plant-based bio-identical testosterone (which some believe is less disruptive to the hormone system). He also checks their Prostate Specific Antigen (PSA) levels every six months. "The bottom line is, there are no good quantifiable long-term papers out there yet about its safety," says Dorfman, "I very specifically describe to my patients the fact that I do not know long-term what the consequences will be." And their typical answer? They are willing to take the risk.

Are you Suffering from Male Menopause (a.k.a. Androgen Deficiency in Aging Men)?

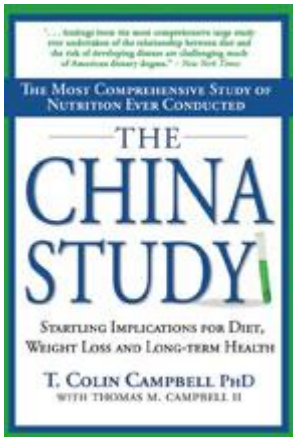
Answer the following questions to find out if you may be going through the "change of life." If you answer yes to question 1 or 7, or at least three of the other questions, you may have low testosterone and may want to discuss the results of this quiz with your health care provider.

1. Do you have a decrease in libido (sex drive)?
2. Do you lack energy?
3. Do you have a decrease in strength and/or endurance?
4. Have you lost height?
5. Have you noticed a decreased enjoyment of life?
6. Are you sad or grumpy?
7. Are your erections less strong?
8. Have you noticed a deterioration in your athletic performance?
9. Are you falling asleep after dinner?
10. Is your work performance deteriorating?

Source: John Morley, MD, Saint Louis University School of Medicine

PROSTATE CANCER

Source: *The China Study* by T. Colin Campbell (157-182), www.thechinastudy.com



I suspect that most people do not know exactly what a prostate is, even though prostate cancer is commonly discussed. The prostate is a male reproductive organ about the size of a walnut, located between the bladder and the colon. It is responsible for producing some of the fluid that helps sperm on its quest to fertilize the female's egg.

For such a little thing, it sure can cause a lot of problems. Several of my friends now have prostate cancer or closely related conditions, and they aren't alone. As one recent report pointed out, "Prostate cancer is one of the most commonly diagnosed cancers among men in the United States, representing about 25% of all tumors. . . ."[93] As many as half of all men seventy years and older have latent prostate cancer,94 a silent form of the cancer which is not yet causing discomfort. Prostate cancer is not only extremely prevalent, but also slow-growing. Only 7% of diagnosed prostate cancer victims die within five years.[95] This makes it difficult to know how and if the cancer should be treated. The main question for the patient and doctor is: will this cancer become life threatening before death comes from other causes?

One of the markers used to determine the likelihood of prostate cancer becoming life threatening is the blood level of prostate specific antigen (PSA). Men are diagnosed as having prostate problems when their PSA levels are above four. But this test alone is hardly a firm diagnosis of cancer, especially if the PSA level is barely above four. The ambiguity of this test leads to some very difficult decision-making. Occasionally my friends ask for my opinion. Should they have a little surgery or a lot? Is a PSA value of 6.0 a serious problem or just a wake-up call? If it's a wake-up call, then what must they do to reduce such a number? While I cannot speak to the clinical condition of an individual, I can speak to the research,

and of the research I have seen, there is no doubt that diet plays a key role in this disease.

Although there is debate regarding the specifics of diet and this cancer, let's start with some very safe assumptions that have long been accepted in the research community:

- Prostate cancer rates vary widely between different countries, even more than breast cancer.

- High prostate cancer rates primarily exist in societies with "Western" diets and lifestyles.

- In developing countries, men who adopt Western eating practices or move to Western countries suffer more prostate cancer.

These disease patterns are similar to those of other diseases of affluence. Mostly this tells us that although prostate cancer certainly has a genetic component, environmental factors play the dominant role. So what environmental factors are important? You can guess that I'm going to say plant-based foods are good and animal-based foods are bad, but do we know anything more specific? Surprisingly, one of the most consistent, specific links between diet and prostate cancer has been dairy consumption.

A 2001 Harvard review of the research could hardly be more convincing [96]:

> . . . twelve of. . . fourteen case-control studies and seven of. . . nine cohort studies [have] observed a positive association for some measure of dairy products and prostate cancer; *this* is *one of the most consistent dietary predictors for prostate cancer in the published literature* [my emphasis]. **In these studies, men with the highest dairy intakes had approximately double the risk of total prostate cancer, and up to a fourfold increase in risk of metastatic or fatal prostate cancer relative to low consumers.**96

Let's consider that again: dairy intake is "one of the most consistent dietary predictors

for prostate cancer in the published literature," and those who consume the most dairy have double to quadruple the risk.

Another review of published literature done in 1998 reached a similar conclusion:

> In ecologic data, correlations exist between per capita meat and dairy consumption and prostate cancer mortality rate [one study cited]. In case control and prospective studies, the major contributors of animal protein, meats, dairy products and eggs have frequently been associated with a higher risk of prostate cancer. . . [twenty-three studies cited]. Of note, numerous studies have found an association primarily in older men [six studies cited] though not all [one study cited] The consistent associations with dairy products could result from, at least in part, their calcium and phosphorous content.[97]

In other words, an enormous body of evidence shows that animal-based foods are associated with prostate cancer. In the case of dairy, the high intake of calcium and phosphorus also could be partly responsible for this effect.

This research leaves little room for dissent; each of the above studies represents analyses of over a dozen individual studies, providing an impressive bulk of convincing literature.

THE MECHANISMS

As we have seen with other forms of cancer, large-scale observational studies show a link between prostate cancer and an animal-based diet, particularly one based heavily on dairy. Understanding the mechanisms behind the observed link between prostate cancer and dairy clinches the argument.

The first mechanism concerns a hormone that increases cancer cell growth, a hormone that our bodies make, as needed. This growth hormone, **Insulin-like Growth Factor 1 (IGF-1)**, is turning out to be a predictor of cancer just as cholesterol is a predictor for heart disease. Under normal conditions, this hormone efficiently manages the rates at which cells "grow"-

that is, how they reproduce themselves and how they discard old cells, all in the name of good health.

Under unhealthy conditions, however, IGF-1 becomes more active, increasing the birth and growth of new cells while simultaneously inhibiting the removal of old cells, both of which favor the development of cancer [seven studies cited98]. So what does this have to do with the food we eat? It turns out that consuming animal-based foods increases the blood levels of this growth hormone, IGF-1. 99-101

With regard to prostate cancer, people with higher than normal blood levels of IGF-1 have been shown to have 5.1 times the risk of advancedstage prostate cancer.98 There's more: when men also have low blood levels of a protein that binds and inactivates IGF-1,102 they will have 9.5 *times the risk of advanced-stage prostate cancer,98* Let's put a few stars by these numbers. They are big and impressive-and fundamental to this finding is the fact that we make more IGF-1 when we consume animalbased foods like meat and dairy.99-101

The second mechanism relates to vitamin D metabolism. This "vitamin" is not a nutrient that we need to consume. Our body can make all that we need simply by being in sunlight fifteen to thirty minutes every couple of days. In addition to the production of vitamin D being affected by sunlight, it is also affected by the food that we eat. The formation of the most active form of vitamin D is a process that is closely monitored and controlled by our bodies. This process is a great example of our bodies' natural balancing act, affecting not only prostate cancer, but breast cancer, colon cancer, osteoporosis and autoimmune diseases like Type 1 diabetes. Because of its importance for multiple diseases, and because of the complexity involved in explaining how it all works, I have provided in Appendix C an abbreviated scheme, just enough to illustrate my point. This web of reactions illustrates many similar and highly integrated reaction networks showing how food controls health.

The main component of this process is an active form of vitamin D produced in the body from the vitamin D that we get from food or sunshine. This active or "supercharged" D produces many benefits throughout the body, including the prevention of cancer, autoimmune diseases and diseases like osteoporosis. This all-important **supercharged D** is not something that you get from food or from a drug. A drug composed of isolated

supercharged D would be far too powerful and far too dangerous for medical use. Your body uses a carefully composed series of controls and sensors to produce just the right amount of supercharged D for each task at exactly the right time.

As it turns out, our diet can determine how much of this supercharged D is produced and how it works once it is produced. Animal protein that we consume has the tendency to block the production of supercharged D, leaving the body with low levels of this vitamin D in the blood. If these low levels persist, prostate cancer can result. Also, persistently high intakes of calcium create an environment where supercharged D declines, thus adding to the problem.

So what food substance has both animal protein and large amounts of calcium? *Milk and other dairy foods.* This fits in perfectly with the evidence that links dairy consumption with prostate cancer. This information provides what we call biological plausibility and shows how the observational data fit together. **To review the mechanisms:**

- Animal protein causes the body to produce more IGF-1, which in turn throws cell growth and removal out of whack, stimulating cancer development.

- Animal protein suppresses the production of "supercharged" D.

- Excessive calcium, as found in milk, also suppresses the production of "supercharged" D.

- "Supercharged" D is responsible for creating a wide variety of health benefits in the body. Persistently low levels of supercharged D create an inviting environment for different cancers, autoimmune diseases, osteoporosis and other diseases.

The important story here is how the effects of food-both good and bad-operate through a symphony of coordinated reactions to prevent diseases like prostate cancer. In discovering the existence of these networks, we sometimes wonder which specific function comes first and which comes next. We tend to think of these reactions within the network as independent. But this surely misses the point. What impresses me is the multitude of

reactions working together in so many ways to produce the same effect: in this case, to prevent disease.

There is no single "mechanism" that fully explains what causes diseases such as cancer. Indeed, it would be foolish to even think along these lines. But what I do know is this: the totality and breadth of the evidence, operating through highly coordinated networks, supports the conclusions that consuming dairy and meat are serious risk factors for prostate cancer.

FLAX OIL (ALA) & PROSTATE CANCER

"Witch Hunt" or Cause for Concern?

Udo Erasmus, PhD, http://www.udoerasmus.com/articles/udo/flax_prostate.htm

Introduction

Consumers, retailers, practitioners, and media people are hearing that flax oil can increase prostate cancer. So many have asked me for clarification on this issue that I have assembled here the information that I consider helpful. I have looked up the research studies, and have added to what I've found, my own experience of working with flax and other oils for the past 20 years.

The following report summarizes what I have discovered so far. As new information brings further clarity, I will update this article.

I'll strive to give you a balanced view. I want neither to over-emphasize the safety nor the potential toxicity of flax oil, and I hope that the information that follows provides you with much needed insight into this topic.

Promoters of flax oil have touted its benefits, but have not adequately addressed the down side of exclusive use of flax oil. Flax oil has benefits and shortcomings. Ignored, its shortcomings can lead to serious health problems.

Flax is very rich in n-3 and low in n-6 (n-3: n-6 ratio is usually between 3.5: 1 and 4: 1).

Exclusive use of flax oil can lead to n-6 deficiency within 2-8 months. Using CLA (conjugated linoleic acid, a trans- fatty acid which is produced by shifting a double bond and twisting the molecule of the n-6 EFA, LA) in addition to flax oil can lead to n-6 deficiency symptoms even sooner than flax oil used alone.

N-6 deficiency symptoms from too much flax oil can be reversed either by lowering n-3 intake or by increasing n-6 intake.

The list of symptoms of n-6 deficiency garnered from research is long, and is found in an overview article on this web site called Fats that Heal Fats that Kill. I have experienced myself and have seen in other people using flax oil exclusively the following symptoms: dry eyes, skipped heart beats, thin skin, joint pain, eczema and psoriasis-like skin problems, increased susceptibility to infection, and deterioration of immune function.

Context

A recent review article points out that prostate cancer is increasing, and is the second leading cause of cancer deaths in the Western world. The "etiology of prostate cancer remains unclear, course and progression are unpredictable, and definite treatment is not yet established". Lifestyle and diet could contribute to the progression from small, latent, non-metastatic tumors to clinically significant, invasive, metastatic lesions.1

Research on the involvement of fats and fatty acids in prostate cancer has led to inconsistent conclusions. Most of the available information comes from epidemiological (or population) studies. Direct data from animal and human studies are limited.1

Further confusion results from the fact that results from rat studies cannot be automatically generalized to humans, because rats and humans metabolize fats differently. Also, rats don't fry steaks, don't use salad dressings and mayonnaise made with oils that have been highly processed, and don't eat butter that has been exposed to light and air, sometimes for weeks. The reason I make this point will become clear a little later.

Studies done on cell cultures do not take into account the effects of fats on glands and organs, which can affect tumor development and tumor growth. In particular, some fatty acids up- or down-regulate the functions of genes, and it appears that some fatty acids also change the effectiveness of hormones even if they don't change hormone levels present in tissues.

A One-sided View...

Within this context, the suggestion has been made in published literature that flax oil should not be used because it can increase prostate cancer. The Prostate Forum2 lists six studies showing positive correlation between ALA (in serum, adipose tissue, and red blood cell membranes) and prostate cancer. Of the six studies, one showed no correlation3. One found a small (not statistically significant) positive correlation.4 Four studies found a strong positive

correlation between ALA and prostate cancer5,6,7,8. At least two other studies have also shown a correlation of alpha-linolenic acid with increased prostate cancer.9,10

According to Prostate Forum, several labs have found that ALA is one of the most powerful growth stimulants for human prostate cancer cells in tissue culture.2 The Prostate Forum has recommended against the use of flax oil by men with prostate cancer because flax oil is the richest available food source of ALA. The reasoning is that this oil should cause the most prostate cancer because it contains the most ALA.

Sources of ALA Used in Studies that Support one-sided View...

The 'ALA' in human population (epidemiologic) studies comes from two main sources: vegetable oil, and red meat animal products. Both were shown to correlate with similar increases in prostate cancer. In cell studies, chemically 'pure' fatty acids are usually used. In the cited studies, the source of ALA-the n-3 EFA that is 5 times more easily destroyed by light, oxygen, and heat than LA (the n-6 EFA)-was foods that have been processed destructively and treated with great carelessness. Let me illustrate this point.

In one of the epidemiologic studies, five sources of ALA - butter, red meat, bacon, salad dressing, and mayonnaise-were listed.

The animal sources included butter, red meat and bacon. Butter is extensively exposed to light and air between the time the cow is milked and the time the butter is consumed. Butter also contains some trans- fatty acids which, research suggests, may also correlate with increased cancers.

Red meat and bacon are rich sources of iron, a pro-oxidant that can damage EFAs (especially the n-3, ALA). Both are usually fried, and it has been known for at least 30 years that frying damages EFA molecules. Damage caused by frying is well documented in research.

All three animal sources of ALA usually contain traces of sex hormones, pesticides, and antibiotics. Sex hormones are known to increase the growth of certain cancers, especially

those of prostate and breast. Many pesticides have cancer-causing properties. Antibiotics lower immune function. No one knows whether the synergy of these three can augment the detrimental effects of each of them individually, but chances are good that this is the case.

The vegetable sources included salad dressing and mayonnaise. These are made from soybean and/or canola oils that have been destructively processed by degumming, refining, bleaching, and deodorizing (so-called 'RBD oils'). While these oils are generally free of hormones and antibiotics, they can contain carcinogenic pesticides. Insecticides, herbicides (weed killers) and fungicides are used in agriculture. Of these, the fungicides have the most potential for increasing cancer.

Some Other Views

Interestingly, a study done with flax grain has shown that flax inhibits the growth of prostate cancer.6 The study was short-term, and therefore does not predict what would happen if flax grain was used as the sole source of fat for a long time, measured in years.

Another study showed that prostatic alpha-linolenic acid was lower in cancerous prostate glands that exhibited perineural invasion, seminal vesicle involvement, and stage T3 tumors.7

In a review article on n-3 fatty acids and cancer, the author makes the observation that the effect of n-3 polyunsaturated fatty acids (PUFAs) on cancer depends on "background levels of n-6 PUFAs and antioxidants, and this could account for previously inconsistent results in experimental carcinogenesis." He also makes the observation that "n-3 PUFAs appear to be excellent substrates for lipid peroxidation in situations where an oxidative stress is involved, such as in the action of several cytotoxic agents in the treatment of cancer."8

Other researchers found that the ratio of n-3/n-6 PUFAs decreased in the following order: normal, benign prostatic hyperplasia, and prostate cancer. This indicates that n-3 inhibit prostate problems. They conclude that the ratio of n-3/n-6 may have an important association with the benign or malignant state of prostatic disease.9

Yet other researchers suggests that among fatty acids, the n-6 derivative arachidonic acid (AA), delivered in larger than normal quantities to prostate cancer cells in tissue culture by LDL cholesterol via over-expression of its receptor (LDLr), increases the activity of the cancer-related genes c-fos and cox-2.10

In 1994, one review suggested that for prostate cancer, fat consumption should be decreased to 15% of calories. The antioxidant mineral selenium and vitamin E should be supplemented, and a soy product should be used.11

Another study shows that the same n-6 derivative AA, stimulates growth and division of prostate cancer cells (both hormone-sensitive and hormone-insensitive) by increasing lipoxygenase enzyme activity (increasing inflammation). The researchers show that if you block this enzyme, the prostate cancer cells self-destruct (apoptose) very rapidly.12 This could be achieved by inhibitor molecules, by decrease of AA in the medium (or diet), and by increase of n-3 fatty acids that inhibit the production of AA. By the way, AA is found in meat, eggs, and dairy products.

One further study showed a positive association between prostate cancer and animal fat, as well as the n-3 EFA (ALA). It also showed an inverse association between the antioxidant vitamin C and prostate cancer.13

A study in 1985 showed that GLA, ALA, AA, and EPA killed prostate cancer cells in tissue culture, but did not affect the normal cells with which they were cultured. The normal cells continued to grow normally. When essential fatty acids were not present, the prostate cancer cells overgrew the normal cells.14

In 1991, the view from research was that diets containing high levels of n-6 fatty acids enhance tumorigenesis in animals, and that diets with equivalent levels of n-3 fatty acids diminish tumorigenesis.15

A 1999 publication concludes that the combination of fatty acids makes a difference. In this study, GLA, ALA, and EPA increase the death of prostate cancer cells. A slight increase of

cancer cell death was obtained when ALA was combined with AA, OA, or GLA. But ALA with LA or EPA had no effect or even decreased prostate cancer cell deaths.16

A study with another prostate cancer cell line reports that GLA and EPA, which inhibit an important enzyme in carcinogenesis (urokinase-type plasminogen activator [uPA]), suppress cell proliferation (growth and division). Low EPA and high uPA levels have been reported in cancer. ALA, LA, and AA also suppressed cell proliferation in this study.17

Another study found that rats grow faster when vitamin E is given along with linseed oil (which is refined, bleached, deodorized flax oil), grow slower if linseed oil was given without vitamin E, and grow even slower in the presence of pro-oxidant.18

A study in women found that only ALA, but not saturates, monounsaturates, or long chain polyunsaturates n-3 or n-6, had a protective effect on breast cancer.19

A 1999 study found that mutation of the androgen receptor (AR) gene as a cause of prostate cancer is rare, and that over-expression of the AR gene seems to be the most common alteration in hormone-refractory prostate cancer.20 A question left unanswered is what causes this over-expression.

A study published in 2001 concludes that a high intake of both red meat and dairy products is associated with a two-fold increase in risk of prostate cancer.

The reason for the association with red meat remains unexplained.21

Another 2001 study found that a short term (3 month) low fat, fish oil (EPA and DHA) enriched diet increased the n-3/n-6 ratio in plasma and adipose tissue. Also, cyclooxygenase (COX-2) expression decreased in 4 of 7 patients.22 COX-2 produces inflammation, which is involved in cancer.

Finally, a study found that DHA and EPA decreased expression of several genes that are up regulated by androgen in LNCaP prostate cancer cells. They thereby reduced androgen-

mediated cell growth of this prostate cancer cell line. DHA increased the proto-oncoprotein c-jun.23

What can one conclude from all of these studies? Science has become so technical that we get lost in a sea of details that defies common sense; then we get confused. This confusion makes it easier for 'high-tech' industries to benefit, whose drug products suppress symptoms without effecting cures.

One major problem with these studies is the isolation in which they are carried out. In Nature, EFAs are found along with many other substances. In the lab, substances are isolated into chemically pure forms, which are easier to manage, but may be far out of line with what happens in a body fed by whole foods containing hundreds or even thousands of interacting (synergistic) ingredients.

I will attempt to address the contradictory findings of the studies by applying some common sense, and add some overlooked details that may help us practically.

N-3 related Causes of Prostate Cancer: Common Sense

EFAs are chemically very active molecules. The body cannot make them. They are required for vital functions in all cells and tissues. We cannot live without them. They must be provided by foods.

The big question that begs to be answered is why substances that are absolutely required for health can at the same time give you cancer and kill you. It doesn't make sense. N-3s, in particular, have a long history of anti-cancer benefits. If they have anti-cancer properties, why are they causing cancer? Essential nutrients, which the body must have for life and for health, cannot easily be both pro-cancer and anti-cancer at the same time.

So the question that must be answered is what other issues are being overlooked when medical professionals (untrained in nutrition-in this case, ALA and flax oil), issue edicts against the use of essential nutrients.

Here are my thoughts.

These thoughts come from 20 years of pursuing practical answers regarding the application of fats to health.

Processing damage of ALA, the most fragile of essential nutrients, must be considered as a possible cause of increased prostate cancer. As ALA consumption increases, so does the amount of damaged, toxic breakdown products of ALA resulting from careless treatment of this essential nutrient. Unless care is taken to protect ALA from being damaged and thereby being made toxic by light, air, and heat, health problems based on the toxicity of altered molecules of ALA should be expected.

Pro-oxidants. According to the study that compared high and low intakes of ALA in humans,5 the strongest risk factor was the consumption of red meat. Red meat is rich in iron, which has strong pro-oxidant action that can speed up the damage done to EFAs by light, oxygen, and heat. That's true outside as well as inside the body.

Because of ALA's far higher fragility, we should expect ALA to be damaged far more extensively than LA. As a result, far more toxicity should come from diets with higher ALA intake in association with pro-oxidants that lead to free radical formation and oxidation products.

Related information shows that red meat consumption correlates with increased cancer in general. White meats from chicken and turkey, which contain as much ALA as red meat does, show less of a correlation with cancer than red meat. Consumption of high-fat fish, which contains more n-3 than red meat, and in the form of EPA and DHA, that are even more fragile to damage done by light, air, and heat, lowers cancer risk factors. And raw high-fat fish, in the form of Japanese sushi or sashimi, correlates with the least cancer.

These findings do not provide proof, but the trend is clear. It suggests that ALA or the other n-3 do not increase prostate cancer, but that the n-3 molecules damaged during

commercial processing and food preparation: cooking, frying, and especially barbecuing may well do so.

Antioxidant depletion. Research has consistently shown that increased intake of EFAs increases the need for antioxidants. EFAs are high-energy fuel. In the body, they build a strong fire. A strong fire throws more sparks than a weak one. Those who fear the EFAs suggest that we should lower intake. That means, turn down the fire. Taken to its logical conclusion, that would mean that we should put the fire out, because if there's no fire, there'll be no sparks that can do damage. It's stupid advice. If we are dead, then we need no more antioxidant spark control because there's no more fire. What would be the point of that?

A more viable solution is to make the strongest fire possible, and to make sure that there's good spark control. Antioxidant protection should accompany our increased intake of EFAs. N-3 fatty acids, being more chemically active than n-6, require a higher antioxidant intake for spark control. But higher n-6 intake too, requires more antioxidants.

The richest source of antioxidants is fresh green vegetables. They contain hundreds, if not thousands of different kinds of antioxidants. The seeds themselves are also rich sources of antioxidants. And research has shown that 400-800mg of vitamin E daily reduces cardiovascular risk by over 75%, while 200ug of selenium daily reduce cancer risk by over 50%. These two powerful antioxidants, as well as zinc, manganese, vitamin C, vitamin A (or carotene), as well as sulfur-containing amino acids, alpha-lipoic acid, glutathione, coenzyme Q10, turmeric, ginger, garlic, and onions, all provide antioxidant protection to the body. Certain herbs, and mushrooms like maitake also help.

Lack of Phytosterols. Phytosterols (plant sterols) have been shown to inhibit many cancers. One of the pioneers in natural treatments of cancer, Dr. Emanuel Revici, worked from the notion that there are two causes of cancer: lack of EFAs, and lack of (phyto)sterols. His methods reversed the cancers of many patients, and Revici himself was a testimony to his own methods. He died a few years ago at the age of 102. Unfortunately, much of his work is now lost.

Phytosterols, which are found in the membranes of all cells of all plants, seeds, and unrefined oils, are not present in animals. They inhibit sterol reactions: cholesterol, the male and female steroid hormones androgen (testosterone) and estrogens (estradiol, estriol, progesterone), and cortcosteroids (aldosterone, cortisol, and others). They therefore slow down the growth of steroid hormone-specific cancers, including some types of prostate cancer.

Too much ALA in relation to LA is another factor that needs to be addressed. N-3 and n-6 EFAs compete in the body for space on the enzymes that convert them into derivatives and eicosanoid hormones. Hence the ratio between them must be such that adequate amounts of both are converted.

A ratio of 2: 1 of n-3 to n-6 will do this. So might a ratio of 1: 4. In healthy people, a wide range of ratios is possible. In people with degenerative conditions, an emphasis on n-3 seems to be more effective. That's because n-3 intake has dropped to 1/6th of what people obtained in their diet 150 years ago, while n-6 intake has doubled over the past 100 years.

This problem can be caused by the exclusive use of flax oil.

Flaxseed, used as the only source of fats in the diet, can also cause this problem. Both flax and flax oil have an n-3: n-6 ratio of 3.5 or even 4: 1. Using such a ratio will result in the n-6 EFA being crowded out from the enzymes. And that will lead to n-6 deficiency symptoms.

The list of n-6 symptoms is long, but relevant here is the fact that n-6 deficiency leads to deterioration of immune function, which in turn can lead to increased cancer growth.24 A comprehensive list of n-3 and n-6 deficiency symptoms is found in the book Fats That Heal Fats That Kill.

The same problem concerning high n-3 with low n-6 similarly affects the growth of other cancers.

Other toxic influences that accompany EFAs can also affect cancers. For instance, antibiotics used in feeds end up in meat. These antibiotics can inhibit immune function. Hormones and pesticides contained in meat, butter, and other dairy products can also affect cancer initiation and growth.

In vegetable oils, the packaging can also be an issue. Fillers, plasticisers, stabilizers, mould releasers, and other industrial chemicals unsuitable for human consumption but present in plastics may dissolve in oils, and can then affect the body after the consumption of oil.

Packaging oils in clear glass or plastic, especially those that contain n-3 (canola and soybean) is inadvisable, because it exposes oils and n-3s to the destructive influence of light.

In some plastics, heavy metals like lead and aluminum are present. In some plastic containers, the pigment used to render plastic opaque to light-carbon black, a cousin of soot-contains Polycyclic Aromatic Hydrocarbons (PAHs). These, formed when carbon reacts with itself in a situation of incomplete burning, are carcinogenic.

The Cause of the Increase in Prostate Cancer: Research

Authors of published studies have been clear that the correlation of ALA with increased prostate cancer is not proof that ALA causes prostate cancer, and point out that that the mechanisms involved in this finding remain unknown. These researchers have suggested that several possibilities need to be explored. These include:

- Oxidation products of ALA formed during cooking of meat;
- Damage done to ALA molecules during processing;
- Lack of balancing molecules such as phytosterols and antioxidants, which are found in seeds, but are removed or damaged during processing and cooking practices;
- Free radical formation from fatty acid oxidation;

- ALA-based free radicals (products of processing) that can damage genetic material (DNA) and lead to tumor formation;
- Decrease in the level of antioxidants, because they are used up to deal with ALA-based free radicals produced in the body;
- Too low a ratio of LA: ALA (or too high a ratio of ALA: LA);
- Alterations in eicosanoid synthesis;
- Changes in cell membrane composition, affecting permeability and receptor activity;
- Interference with 5-alpha-reductase activity; and
- EFAs may increase steroid hormone production that is important in androgen sensitive growth. (Actually, EFAs appear to decrease steroid hormone levels. Apparently they make hormones-insulin, thyroid, androgens, and others-work better, and therefore smaller amounts of them are needed to get hormones' normal job done).

Differences between Oils Made With or Without Health in Mind

I learned about the highly sensitive n-3 ALA in 1981. **I have emphasized since that time that ALA should never be subjected to the destructive influences of light, oxygen, and high temperatures.** One or more of these destructive influences is involved during:

Commercial and home frying, deep frying, and sautéing
Processing (deodorization) involved in the production of the cooking (RBD) oils that line the shelves of grocery, convenience, and health food stores
Hydrogenation, a process used to make margarine and shortening
Partial hydrogenation of oils used in making shelf-stable convenience foods.

Damage done to ALA molecules by light, air, and heat can produce highly toxic unnatural molecules.24 ALA forms more toxic breakdown products due to processing damage than does the n-6 EFA.24 Destructive processing is likely the cause of some of the changes that lead to increased prostate cancer. A more comprehensive story of how EFAs are damaged is found in the book *Fats That Heal Fats That Kill*.

What Should We Do To Protect Our Prostate Gland?

Born in 1942, I'm in the age group of men that should pay attention to the condition of their prostate gland. I cannot give you medical advice or make decisions for you, but I can tell you what I do.

I do not use, and recommend against the use of flax oil by itself, but do recommend this n-3-rich oil in combination with n-6 richer oils to get the n-3/n-6 ratio right. Flax is a great source of essential n-3 but is deficient in the equally essential n-6.

The prostate gland appears to be quite sensitive to environmental toxins. Among these may be plasticisers and contaminants present in plastics. Because of environmental concerns and our very limited knowledge of the effects of these molecules on health, I recommend against using plastics for packaging, especially liquids (water, oil, milk, juices, vinegar, alcohol, tinctures, etc.). Liquids move, continually washing the inside of their container. Any molecules present in plastic containers (fillers, plasticisers, stabilizers, mould releasers, slip agents, sheen agents, contaminating metals such as lead (Pb) or aluminum (Al)), which might dissolve in the liquid contained, may drift from the plastic into the food. Drift of molecules from plastic into liquid is the reason why water in plastic bottles can acquire a 'plastic' taste.
One is less likely to taste plastic in oils than in water. However, due to the chemical similarity of oil molecules and plastic molecules, oils swell plastics, opening pores in this non-natural synthetic material that make the drift of such molecules into oils even more likely than the drift of molecules from plastic into water.

I do use and recommend an oil blend containing flax with sunflower and sesame oils from organically grown seeds, made with health in mind, and in the right n-3: n-6 ratio to prevent n-6 deficiency. In fact, I created the formula for such a blend, and I use it daily with my food.

I do insist that my oil blend is packed in brown glass, further protected by a box to keep out all light, and further protected by refrigeration in factory, store, and home to extend freshness.

I also use and recommend zinc, selenium, antioxidants, phytosterols, saw palmetto, broccoli and other cruciferous vegetables, anti-inflammatory herbs, and maitake extracts or mushrooms as part of a prostate nourishing nutritional program.

I use and recommend optimum intake of all components of health: 20 minerals; 14 vitamins; 8-11 essential amino acids; 2 essential fatty acids; detoxifying fiber; digestive enzymes; friendly bowel microorganisms; antioxidants; herbs (phytonutrients); filtered water; clean air; sunlight; and fuel.

I engage in and recommend physical activity (work or exercise) to stay fit. I indulge myself in and recommend rest; sufficient sleep; recreation; the passionate pursuit of worthwhile goals; time spent with friends; a sense of humor; good balance between work and play; heart-felt gratitude; and faith in the grand scheme of things.
I use ALA on a daily basis, combined with LA in my oil blend, as part of my program for health, along with lots of fresh organic green foods, proteins, support for digestion, and carbohydrate intake limited to the amount I burn. I take supplements of minerals, vitamins, antioxidants, and herbs.

I do not worry about ALA from oils made with health in mind causing me prostate cancer. After all, common sense insists and research confirms that ALA (undamaged and accompanied by sufficient undamaged LA and natural antioxidants), is essential for life and for health.
Updated: November 9, 2002

Abbreviations used in this Article + Basic facts about 'essential' fats ...

EFAs = Essential Fatty Acids are substances from fats that must be provided by foods because the body cannot make them, and yet must have them for health.

EFAs exist in two families: omega-3 (n-3) and omega-6 (n-6). From these two, the body can make several derivatives, as well as eicosanoid 'hormones', and other active substances.

N-3s never turn into n-6s in our body, and n-6s cannot turn into n-3s. N-3 and n-6 EFAs do, however, have some overlapping functions.

As a result of overlap in functions, n-6 can cover some symptoms of n-3 deficiency. But, as a quirk of nature, n-3 cannot cover most symptoms of n-6 deficiency. This leads to a situation where, although n-3 deficiency is far more widespread, n-6 deficiency symptoms are easier to identify.

For many years, this quirk of nature led to the mistaken notion that n-6 are more important than n-3 (or that n-3 are not essential at all). As a result, instead of focusing on bringing the missing n-3 EFA back into the diet, much work has been done with oils rich in the n-6 derivative GLA.

(Essential) N-3 = omega-3 fatty acids include:

1. ALA (alpha-linolenic acid; abundant in flax, and present in small quantities in hemp, walnut, soybean, and canola); given enough ALA to start with, the body converts ALA into SDA, EPA, and DHA in various tissues, according to need; conversion varies, depending on several factors, and ranges from less than 5% to 36% per day of the amount of ALA consumed;
2. SDA (stearidonic acid; present in a few exotic seeds);
3. EPA (eicosapentaenoic acid; parent of Series 1 eicosanoid hormones; found in fish oils);
4. DPA (docosapentaenoic acid);
5. DHA (docosahexaenoic acid; the major brain n-3; also found in eyeball (retina), red-brown algae, and fish oils).

ALA = Alpha-Linolenic Acid is the omega 3 (n-3) EFA. It is sometimes shortened to LNA.
ALA is very fragile to destruction by light, oxygen (air), and heat, and must therefore be protected from these influences. If this is not done, ALA molecules change from natural and beneficial to unnatural and toxic. ALA is destroyed about 5 times faster than LA, the n-6 EFA.

ALA is deficient in the diets of most people in affluent societies. Due to processing damage, shelf life considerations, and changes in food choices, average intake of n-3 has decreased to less than 20% of what was present in common diets 150 years ago. Even back then, n-3 intake was less than optimal because only a few foods are rich in n-3.

About 90-95% of the population gets less n-3 than required for good health (making n-3 the essential nutrient most often lacking in people's foods) and n-3 is therefore the most therapeutic of all of the essential nutrients (20 minerals, 14 vitamins, 8-11 amino acids, 2 fatty acids).

N-3s improve more than twice as many health problems as do n-6!
N-3s are more effective for:

raising energy levels, stamina, and performance;

improving concentration, learning, calmness, behavior, and IQ;

lowering cardiovascular risk factors;

inhibiting cancer growth and metastasis;

increasing insulin sensitivity;

speeding the healing of wounds due to accidental injury, physical exertion, and surgery;

decreasing inflammation and joint pain;

dampening the symptoms of auto-immune diseases;

improving bone mineral metabolism;

improving weight management; and

Increasing fat burning, decreasing fat production, and increasing fat burn-off as heat (thermogenesis).

However, too much n-3 (e.g. the use of flax and flax oil as the only source of EFAs in the diet) can lead to n-6 deficiency and thereby work against the health of cells, tissues, glands, and organs. Thus the ratio of n-3 to n-6 in the diet is a highly important consideration.

(Essential) N-6 = omega-6 fatty acids include:

LA (linoleic acid; abundant in safflower, sunflower, and corn; present in medium quantities in soybean, sesame, pumpkin seed, and almond; present in small quantities in canola, peanut, and olive); given enough LA to start with, the body converts LA into GLA, DGLA, and AA in various tissues, according to need;

GLA (gamma-linolenic acid; present in evening primrose oil); GLA can partially cover n-3 deficiency; a main reason for its benefits comes from being used in an n-3 deficient population; in people consuming an n-3-rich, n-6-balanced diet, GLA is not nearly as impressive as it is in treating n-3 deficient people;

DGLA (dihomogamma-linolenic acid; parent of Series 1 eicosanoid hormones);

AA (Arachidonic acid; the major brain n-6; parent of Series 2 eicosanoid hormones; found in meat, eggs, and dairy products).

LA = Linoleic Acid = the omega-6 (n-6) EFA.

LA is abundant in the diets of most people in affluent societies, its intake having doubled during the past 100 years due to increased use of corn and safflower oils.

Diets too high in LA (and too low in n-3) are associated with increased cancer. Damaged n-6 molecules due to processing, removal of antioxidants and phytosterols, and concomitant lack of n-3 are likely responsible for this problem.

LA is essential to life and to health, and must be present in the diet.

LA is sensitive to destruction by light, oxygen (air), and heat (but 5 times less sensitive than n-3), and should be protected from these destructive influences. If this is not done, LA molecules can change from natural and beneficial, to unnatural and toxic.

People on low fat or no fat diets are likely to get insufficient n-3 and n-6.

N-3: N-6 Ratio: Both n-3 and n-6 are essential to health and must come from the diet because the body cannot make them. However, too much n-3 can crowd out the n-6 (as can happen with flax and flax oil used exclusively as the source of EFAs in the diet), and lead to n-6 deficiency. Too much n-6 can crowd out n-3 (as is the case in 'normal' Western diets), and lead to n-3 deficiency.

The ratio of n-3 to n-6 is important and must be carefully considered. I have seen the best results for health using an n-3: n-6 ratio of 2: 1. (Non-essential) N-9 = omega-9 = OA (oleic acid)

The body can itself make this monounsaturated fatty acid, and OA is therefore not essential. Essential means that the body cannot make it, cannot live without it, and must therefore obtain it from an outside source, i.e. food or supplement.

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POMEGRANATE: PROSTATE CANCER

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Can pomegranates prevent prostate cancer? A new study offers promise

MADISON - The juice of the pomegranate, say researchers at University of Wisconsin Medical School, shows major promise to combat prostate cancer - the most common invasive cancer and the second-leading cause of cancer death in American men.

With more than 230,000 new cases of prostate cancer expected to be diagnosed this year alone in the U.S. and the outlook poor for patients with metastatic disease, researchers are looking for new strategies to combat the disease. Earlier research at Wisconsin and elsewhere has shown that the pomegranate, a fruit native to the Middle East, is rich in anti-oxidant and anti-inflammatory activity and is effective against tumors in mouse skin. In fact, pomegranate juice has higher anti-oxidant activity than do red wine and green tea, both of which appear promising as anti-cancer agents.

The UW research team aimed to find out if the extract from pomegranates would not only kill existing cancer, but help prevent cancer from starting or progressing. Using human prostate cancer cells, the team first evaluated the fruit extract's effect, at various doses, on those cells cultured in laboratory dishes. They found a "dose-dependent" effect - in other words, the higher the dose of pomegranate extract the cells received, the more cells died.

The research team then progressed to tests in mice that had been injected with prostate cancer cells from humans and developed malignancies. The 24 mice were randomly divided into three groups. The control group received normal drinking water, while the animals in the second and third groups had their drinking water supplemented with .1 percent and .2 percent pomegranate extract respectively. The doses for the mice were chosen to parallel how much pomegranate juice a typical healthy human might be willing to eat or drink daily.

The results were dramatic: the mice receiving the higher concentration of pomegranate extract showed significant slowing of their cancer progression and a decrease in the levels of prostate-specific antigen (PSA), a marker used to indicate the presence of

prostate cancer in humans. The animals that received only water had tumors that grew much faster than those in the animals treated with pomegranate extract.

"Our study - while early -- **adds to growing evidence that pomegranates contain very powerful agents against cancer, particularly prostate cancer**," says lead author Dr. Hasan Mukhtar, professor of dermatology in the UW Medical School. "There is good reason now to test this fruit in humans - both for cancer prevention and for treatment."

The next step in the evaluation of pomegranates for cancer prevention and treatment is to conduct tests in humans, according to Mukhtar.

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The other members of the research team are Arshi Malik, Farrukh Afaq, Vaquar Adhami, Deeba Syed and Sami Sarfaraz, all research scientists in the department of dermatology. The Wisconsin research was funded by the National Institutes of Health.

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PROSTATE CANCER: ADJUVANT THERAPY (VITAMINS, MINERALS, TRACE ELEMENTS, AND HERBAL PREPARATIONS)

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Life Extension Magazine, http://www.lef.org/protocols/prtcl-134.shtml

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Dr. Strum is on the Life Extension Medical Advisory Board, and Dr. McDermed is from the Prostate Cancer

Research Institute (PCRI) in Los Angeles, California. Drs. Strum and McDermed are proponents of a holistic medical strategy that combines peer-reviewed conventional scientific publications with new findings in the areas of nutrition and supportive care of the patient.

We have routinely employed natural therapies in our holistic approach to oncology and internal medicine disorders for almost 30 years. Such therapies are based on published peer-reviewed literature and not hearsay from individuals or companies that appear to be in the business of medicine. The amount of literature in this area has grown exponentially. We are now at a phase in our acceptance of such approaches that medical studies are being conducted to verify the benefits of such "nutriceuticals" in pilot clinical trials with endpoints that are objectively evaluable. Most of the recommendations in this section are directed to one or more phases of prostate cancer (PC) management: prevention, early-stage treatment, late-stage treatment, and/or maintenance phase of active treatment. During radiation therapy, there is concern that the use of high doses of antioxidants will protect tumor cells from the cell-killing effects of radiation. This is a controversial issue. There is a randomized study indicating significant reduction in the radiation-induced side effects of cystitis and proctitis using SOD (Orgotein) in patients receiving radiation therapy (RT) for bladder cancer. This study involved 448 patients. It showed a dramatic lessening in the radiation therapy-induced side effects in the SOD treated arm (Sanchiz et al., *Anticancer Res.*, 1996). The differences in anticancer results in the treatment arm receiving Orgotein versus placebo are still not known. Orgotein is an injectable SOD drug approved in Europe, but not in the United States.

Nutritional Recommendations for Active PC

We use the nutritional approaches during the preventive phase in men at risk for developing PC. We employ the same strategy during the initial use of androgen deprivation therapy (ADT) and during the off-phase of ADT for the purpose of slowing the growth rate of PC as much as possible. In patients with active PC, we take a more aggressive stance on the dosing of these agents and also suggest the use of additional agents that appear promising in their activity against PC. However, it is important to indicate that the rationale for the use of many of these adjuncts is based on studies involving human cell lines of PC grown in animal models, usually mice. Clinical studies in humans have either not been

started or are in progress but are too preliminary to report at this time.

Selenium

As noted above, we suggest the use of selenomethionine derived from yeast. We have seen no selenium toxicity at doses as high as 800 mcg a day. Larry Clark, at the University of Arizona, has recently initiated clinical trials using 800, 1600, and 3000 mcg a day of selenium in patients involved in watchful waiting. At 800 mcg a day, physician monitoring and consideration of selenium blood levels is advised. Please note that selenium blood levels will be elevated in most patients taking any selenium supplementation due to the fact that normal selenium levels were based on population sampling from men and women not taking selenium supplements. If selenium toxicity occurs, the most common signs are abnormalities in nail growth with loss of nails, hair loss, lack of appetite with weight loss, and a garlic-like odor to the breath. Personally, I have only seen hair loss, anorexia, and weight loss in one patient who received a dose of 300,000 mcg of selenium a day. That patient had a drop in his PSA and has had a slow recovery of PSA; he has been off therapy after ADT for 7 years. Carefully controlled trials with selenium such as at the University of Arizona are critically important to our understanding of how best to use selenium. We urge patients doing watchful waiting to join in such trials if this is possible. Call Trish Wilkens or Jennifer Hart at (520) 321-7798 (extension 19 or 23) for further information on this clinical trial.

Genistein

The use of soy and genistein in the prevention of PC was discussed above. Genistein has been proposed as an effective agent to prevent the expression of metastatic capacity in hormone dependent cancers. In a cell-culture system, genistein appeared to be cytotoxic and inhibitory of PC cell proliferation (Geller et al., Prostate, 1998). Genistein and soy products therefore play a potential major role in established PC. Cancer cells use the enzyme tyrosine kinase as a growth factor. Soy genistein is a potent inhibitor of tyrosine kinase activity. The effects of protein kinase inhibitors on human prostate cell growth have been extensively investigated. Other biological activities of genistein have been demonstrated in animal models and include the following:

- Inhibition of DNA topoisomerase

- Inhibition of protein tyrosine kinase
- Antiangiogenesis by modulation of FGF (fibroblast growth factor)
- Inhibition of EGF (epidermal growth factor)

In active PC patients, we are exploring higher doses of genistein using Life Extension UltraSoy Extract. Each 700 mg capsule of UltraSoy Extract contains 134 mg of genistein, 122 mg of daidzein, and 24 mg of glycetein. Currently, we are advising two of these capsules a day and are trying to arrange for serum genistein levels.

Synthetic Vitamin D, Bisphosphonates, Calcium Citrate

Other adjunctive therapies known to have an effect on PC include the use of vitamin D. Published studies using more potent synthetic vitamin D analogs such as Rocaltrol or Calcitriol have shown a slowing effect on PC growth (Gross et al., *J. Urol.*, 1998). These analogs affect the p27Kip1 oncogene that results in over-expression of enzymes that inhibit part of the tumor cell cycle (Koike et al., *Proc. Annu. Meet. Am. Assoc. Cancer Res.*, 1997). In short, synthetic vitamin D analogs cause a G1 arrest in the cell cycle by over-expression of cyclin-dependent kinase inhibitors (CDKIs). We routinely use 0.5 mcg of Rocaltrol at bedtime. Rocaltrol requires a physician's prescription. When we employ Rocaltrol, we do so in a comprehensive setting of improving bone integrity. As mentioned earlier, the use of ADT results in an increase in bone resorption due to activation of bone-resorbing cells called osteoclasts. Excessive bone resorption leads to release of bone-derived growth factors that have been shown to play an important role in increasing PC growth. We block this bone resorption by using drugs in the bisphosphonate family. Examples of such drugs currently in use include alendronate (Fosamax), pamidronate (Aredia), and most recently Risedronate (Actonel). The proper use of these agents necessitates physician supervision. As bisphosphonates block excessive bone resorption, they favor bone growth that allows for calcium utilization. Therefore, we routinely combine calcium supplementation when employing bisphosphonate use. We enhance calcium absorption with Rocaltrol or Calcitriol and at the same time get a second benefit from these agents due to their effect on slowing the growth rate of PC cells. Our bone integrity approach therefore involves

1. Bisphosphonate Compound(s).

- **Actonel**--30 mg 1 hour before breakfast taken with water or

- **Fosamax**--10 mg 1 hour before breakfast taken with water and/or
- **Aredia**--30 mg 1 hour before breakfast taken with water or
- **Fosamax**-10 mg 1 hour before breakfast taken with water and/or
- **Aredia**-30 mg intravenously for the first dose (over 1.5 hours); followed every 2 weeks by 60 to 90 mg (over 1.5 hours). Patients unable to tolerate Fosamax or those whose insurance does not allow them to qualify for Aredia (bone metastases are currently an insurance requirement) may use Miacalcin nasal spray once a day to decrease bone resorption and enhance bone formation. Patients with severe bone resorption who are not responding to one of these agents may require the combination of two anti-osteoclastic agents. We monitor the effectiveness of the above therapies with the Pyrilinks-D urine test to quantitate bone resorption. Bone mineral density (BMD) evaluations every 6 to 12 months, as well as periodic serum calcium levels (part of a routine chemistry panel) are also part of the monitoring process. You are encouraged to work with your physician(s) on these issues.

2. Calcium citrate.

500 mg with dinner and 500 mg at bedtime. Calcium citrate is much better absorbed than calcium carbonate. Utilization of calcium at night will lower excessive bone resorption by 20%. Calcium intake during the day has no such effect (Blomsohn et al., *J. Clin. Endocrinol. Metab.*, 1994).

3. Synthetic vitamin D (1, 25-dihydroxycho-lecalciferol) as Rocaltrol.

0.5 mcg at bedtime. The use of synthetic vitamin D at bedtime lowers the urinary calcium excretion. This suggests enhanced utilization of calcium and also diminishes the risk of calcium-based kidney stone formation. We are not concerned about the additional use of low doses of ordinary vitamin D (D3) that is commonly added to most of the available calcium citrate products. However, we do recommend monitoring of the serum calcium levels to make sure that calcium balance is appropriate. In addition, the use of magnesium at a dose of at least half the daily intake of calcium will decrease the formation of calcium oxalate stones.

4. Exercise.

We do encourage the use of exercise to decrease excessive bone resorption. This should be in the form of both aerobic and muscle-building exercises. We recommend reading Ken Cooper's Anti-Oxidant Revolution as well as Barry Sears's Anti-Aging Zone for detailed exercise programs and other important information.

Antimetastatic Agents

The inhibition of new blood vessel formation to block the growth and spread of PC is currently under investigation. Androgen deprivation therapy (ADT) is known to have this antiangiogenesis effect as well as genistein. Other agents that have an effect on cancer cell invasiveness include green tea polyphenols. Green and black tea are derived from the same plant, Camellia sinensis. However, only green tea is rich in the flavonol group of polyphenols known as catechins. The fermentation process used in making black tea destroys the biologically active polyphenols of the fresh leaf. The catechins as a group have significant free radical scavenging ability and are potent antioxidants. Four catechins are found in green tea leaves:

epicatechin (EC)
epigallocatechin (EGC)
epicatechin gallate (ECG)
epigallocatechin gallate (EGCG)

Of these four factions EGCG is the most important to the PC patient. Pharmacological activity extends beyond its actions as an antioxidant and free radical scavenger. Epigallocatechin-3 gallate (EGCG) acts against urokinase, an enzyme often found in large amounts in human cancers (Jankun et al., *Nature*, 1997). Urokinase breaks down the basement membrane of cell junctions, which may be a key step in the process of tumor cell metastasis, as well as tumor growth (Ennis et al., *Proc. Annu. Meet. Am. Assoc. Cancer Res.*, 1997). EGCG attaches to urokinase and prevents these actions.

GTP also inhibits ornithine decarboxylase (ODC), resulting in a decrease in polyamine synthesis and cell growth (Carlin et al., *J. Urol.*, 1996). Inhibitors of 5-alpha-reductase (5AR) may be effective in the treatment of 5-alpha-dihydrotestosterone-dependent abnormalities, such as benign prostate hyperplasia, PC, and certain skin diseases. The green tea catechins

are potent inhibitors of type-1 but not type-2 5AR (Liao and Hiipakka, *Biochem. Biophys. Res. Commun.*, 1995). They also inhibit accessory sex gland growth in rats. These results suggest the certain tea gallates can regulate androgen action in target organs. The 5AR inhibitor Proscar is predominantly a type-2 inhibitor.

Long-term consumption of tea catechins is common in China and Japan. The frequency of the latent, localized type of PC does not vary significantly between Eastern and Western cultures, but the clinical incidence of metastatic PC is generally lower in Japan and other Asian countries, in contrast to the common occurrence of metastatic PC in Europe and the United States. One possible explanation is that EGCG consumption in green tea in Asian countries prevents the progression and metastasis of PC cells. This explains the lower mortality rate due to PC and breast cancer in Asian countries as compared to Western countries.

In a study investigating the effect of intraperitoneal injections of different catechins on the growth of the human PC cell lines PC-3 and LnCaP, and the human breast cancer cell line MCF-7 grown in nude mice, EGCG was found to play a key role (Figure 1). The injection of EGCG slowed the growth of tumors when administered to the control mice on day 14, while the growth of tumors accelerated when EGCG was stopped in the PC-3 line on day 14. Inhibition of PC-3 growth was EGCG specific; it was not seen with EC, EGC, or ECG (Liao et al., Cancer Lett., 1995). The galloyl group of EGCG appears to be necessary for tumor growth inhibition since EGC is not active. EGCG accounts for about 50% of the solid matter in the hot water extract of green tea that is consumed as a beverage.

Figure 1:EGCG Effect on PC-3 Growth

Green tea is prepared from lightly steamed and dried leaves of the tea plant. The steaming process leaves the polyphenol activity intact. The polyphenol activity varies with climate, season, horticultural practices, and the position of the leaf on the harvested shoot. The Life Extension Foundation makes a 95% green tea extract that contains a high level of the active polyphenol EGCG. The polyphenolic profile of Green Tea 95% Extract is EGCG 35%. Each 350 mg capsule is an extract of green tea leaves containing 122.5 mg of EGCG. One 350 mg capsule of Green Tea 95% Extract is equivalent to 4 to 10 cups of Japanese green tea. Green Tea 95% Extract is available in decaffeinated form, or in a lightly caffeinated extract

that contains 10 to 20 mg of caffeine. We suggest that GT 95% be used at a dose of 1 capsule 3 times a day in patients with active PC and perhaps once a day as prevention against PC. We would suggest that GT be taken with food to avoid stomach upset. GT should be kept in a dry, cool location and out of direct light.

Lycopene

Recent studies have shown a statistically significant inverse relationship between the ingestion of tomatoes, tomato sauce, and pizza with the development of prostate cancer. In a 6-year study by Giovannucci et al. (*J. Natl. Cancer. Inst.*, 1995) involving the intake of carotenoids and retinol in 47,894 men, lycopene-rich foods significantly lowered the risk of PC. Men who ingested 10 or more servings of tomatoes in several forms (sauce, juice, raw, or on pizza) had a 41% reduction in PC, while those who ate four to seven servings a week had a 22% reduction. Tomatoes and tomato sauce contain high amounts of lycopene, a carotenoid. Lycopene is the most predominant carotenoid in plasma and in various tissues, including the prostate gland. Lycopene is the most efficient scavenger of singlet oxygen among the common carotenoids. Lycopene is not converted to vitamin A. The major contributors to the specific carotenoids are shown below:

Carotenoid Class	Vegetable or Fruit
ß-carotene	Carrots, yams, sweet potatoes, spinach
α-carotene	Carrots, mixed vegetables
Lutein	Spinach, broccoli, kale, mustard, chard
Lycopene	Tomatoes, tomato sauce, pizza, tomato juice
ß-cryptoxanthin	Oranges

Another study evaluated the effect of lycopene on the development of mammary cancers in

a mouse model. This showed a significant suppression of tumor growth in those mice receiving a diet supplemented with lycopene. Decreases in thymidylate synthetase within the breast tissue, lower levels of serum-free fatty acids, and decreased plasma prolactin levels by the pituitary were characteristic of the lycopene-supplemented group (Nagasawa et al., *Anticancer Res.*, 1995). Interestingly, the source of lycopene was a beta-carotene-rich algae called Dunaliella bardawil.

Recently, Kucuk et al. reported on 30 men with localized PC scheduled for radical prostatectomy. They were randomly assigned to receive either 15 mg of lycopene (Lyc-o-Mato, LycoRed, Beer Sheva, or Israel) orally twice daily, or no intervention for 3 weeks prior to surgery. Prostate specimens were step-sectioned, entirely embedded, and evaluated for pathological stage, Gleason score, the volume of PC, as well as the extent of PIN (a pathological finding often associated with PC) in the gland. The specimens were also examined for biomarkers of cell proliferation, differentiation, and apoptosis. Comparisons were made between intervention and control groups. Serum and tissue lycopene levels increased by 22% in the intervention group. At RP, within the treated group, 8 of 12 patients (67%) had organ-confined PC, and 84% had tumors < 4 cc, compared to 44% and 55%, respectively, in the control group. Lesser glandular involvement by PIN was also observed in the intervention group. The expression of biomarkers of proliferation decreased, whereas the markers of differentiation and apoptosis increased in the intervention group. Serum PSA level also decreased significantly in the intervention group but not in the control group. The results suggest a role for lycopene in PC prevention. This is a very exciting study, and the full report should be published shortly. We currently advise patients with active PC to include 30 mg a day of lycopene in their diet.

Lifestyle Changes to Prevent and Treat Prostate Cancer

- **Restrict Total Caloric Intake to 500 Calories a Meal**

  We believe that diet should be regarded as having serious biochemical relevance to the health of the individual. You are, for the most part, what you eat. Western society, and especially the United States, are over-consumers of calories. Excessive caloric consumption is a significant factor that adversely effects longevity. Caloric

restriction has been shown to be an important factor in augmenting the immune system and improving longevity. We need to rethink how much food we need to eat. Our ideal body weight should be taken seriously. If we were to do this alone, we would virtually eliminate diabetes, hypertension, hypercholesterolemia, stroke, heart disease, and a significant amount of cancer from our lives. Patients should strive at a general figure of 500 calories a meal, and 100 calories per snack. Modifications of this are based on the level of activity, age, and body surface area. Nutritional software or nutritional counseling should be an integral part of our approach to good health.

- **Eliminate Smoking, Reduce Alcohol Consumption, and Exercise Properly**

If we were to eliminate major factors relating to oxidative damage such as cigarette smoking and excessive alcohol consumption, in conjunction with dietary restrictions, we would eliminate 80% of disease as we know it today. In the context of caloric excess, we have additional co-factors such as lack of routine exercise and over consumption of dietary fat. *The Anti-Oxidant Revolutio*n, 1994, by Kenneth Cooper, M.D., focuses on the causal association of over-exercise and the generation of injurious freeradicals with resultant increases in degenerative diseases and cancer. We agree with Cooper that exercise should be low impact and that we should routinely use free radical scavengers, especially at times when we are more physically active, and certainly when we are exposed to excessive free radical damage, i.e., sunlight, high altitude, and activities that generate tissue damage. It is ironic that we bring our automobiles in for a periodic oil change to remove the products of oxidative damage, but we do not attempt a similar maneuver for our own bodies to prevent oxidative damage due to the wear and tear of everyday life.

- **Avoid Excessive Carbohydrate Intake to Prevent Hyperinsulinemia and the Generation of Unfavorable Eicosanoids**

The dietary fat issue is significant. There are studies that show dietary fat to increase the growth rate of PC in animal models of human PC. However, the emphasis on dietary fat per se has taken attention away from caloric over-consumption. Fat

excess, however, is linked to excessive calorie consumption, since fat contains twice as many calories, gram for gram, as protein or carbohydrate. In addition, the ratio of protein to carbohydrate in our meals is related to how our body reacts to the intake of food and how it handles calories that are ingested. The reader is advised to read Barry Sears's book *The Zone*, 1995, and *Anti-Aging Zone*, 1999, for an in-depth discussion of the dangers of over-consumption of carbohydrates and the ill effects of hyperinsulinemia that occur as a result. Our patients are advised to incorporate Sears's approach into their lives while consuming fewer calories a day and exercising moderately. The value of generating favorable eicosanoids is discussed in detail in both of these books. The free radical-generating fatty acid called arachidonic acid, an unfavorable eicosanoid, has been shown to stimulate PC cell growth. The molecular pathway of arachidonic stimulation involves the inflammatory enzyme 5-lipooxygenase. Recent papers show that inhibition of arachidonic acid leads to PC programmed cell death, or apoptosis (Ghosh and Myers, *Proc. Natl. Acad. Sci.* USA, 1998). Lipooxygenase also is involved in the formation of abnormal blood clots. Nutrients that specifically inhibit 5-lipooxygenase include garlic. Fish oil supplements (EPA), an omega-3 fatty acid, have been shown to suppress arachidonic acid formation. Prostaglandins are synthesized from arachidonic acid by the enzyme cyclooxygenase. A particularly dangerous prostaglandin is PGE2, which is involved in many chronic inflammatory diseases. The administration of PGE2 to prostate, breast, and colon-cancer cells resulted in increased cellular proliferation. An ibuprofen derivative called Flurbiprofen inhibited PGE2-induced PC cell growth (Tjandrawinata et al., *Br. J. Cancer*, 1997). Aspirin, ibuprofen, and fish oil are other available agents that inhibit PGE2 synthesis. The eicosanoid pathways are shown in Figure 2.

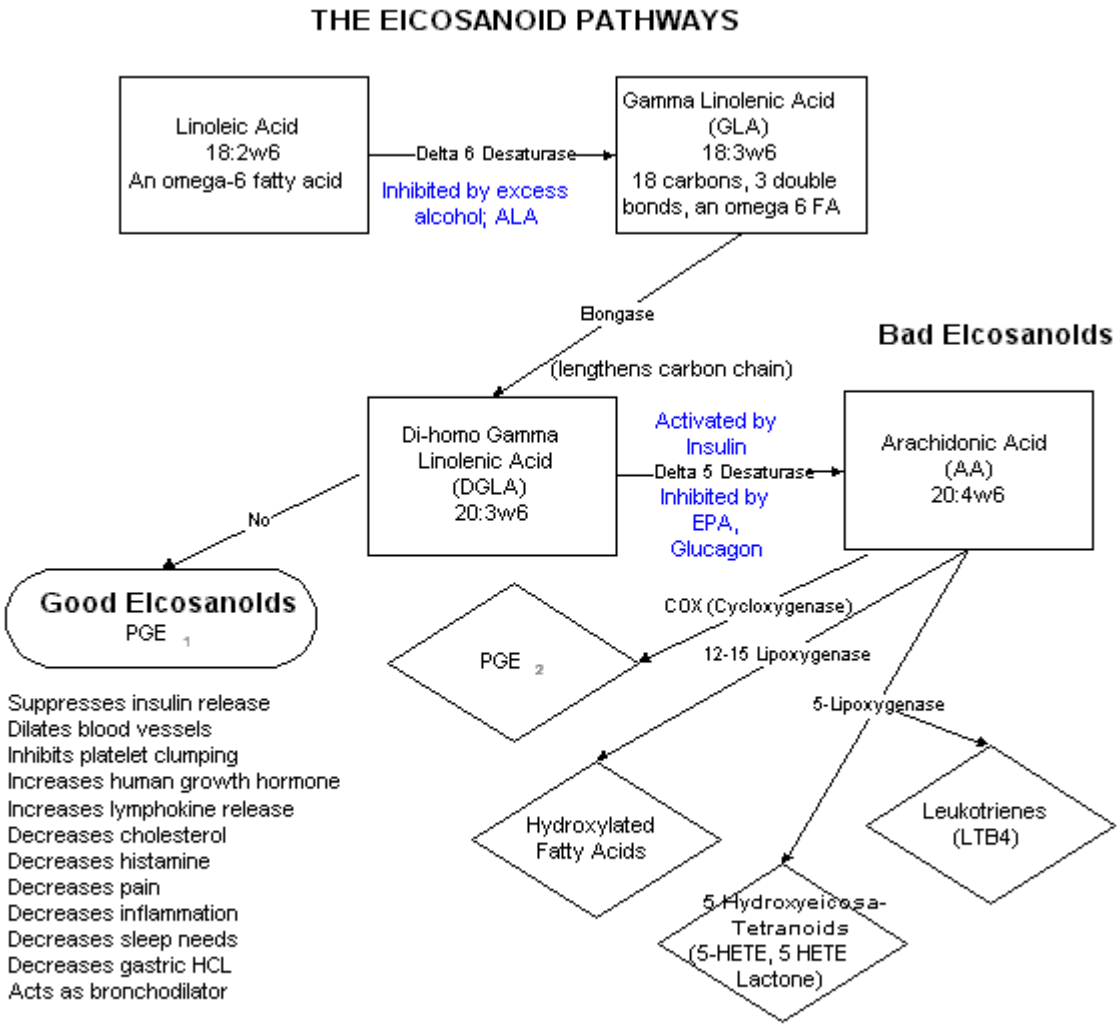


Figure 2. Eicosanoid Pathways

- **Use Free Radical Scavengers (Selenium and Vitamin E) to Prevent Oxidative Damage**

  In conjunction with dietary restriction of calories and alteration in the nature of the calories consumed as well as moderating our exercise, there is evidence that aging, degenerative disease, and cancer are all expressions of varying degrees of cellular oxidative damage. In fact, fat itself induces the generation of fatty acid peroxides that generate damaging free radicals. The concept here is that living organisms are subject to oxidation just as metal is subject to rusting. As part of aging, we see the sequelae of such oxidation manifested in the graying of hair, short-term memory

loss, cataract formation, gum and jaw recession, vascular disease, cardiac disease, degenerative joint disease, and sun-induced skin changes ranging from wrinkling to skin cancer. The majority of items in health food stores today are antioxidants.

In regards to PC, there are now studies that show that vitamin E and selenium use will decrease the incidence as well as the mortality from PC. The ATBC study by Heinonen et al. (*J. Natl. Cancer Inst.*, 1998) demonstrated a 32% decrease in the incidence of PC and a 41% lower mortality rate from PC in men taking alpha-tocopherol (vitamin E). Another study by Fleshner et al. (*J. Urol.*, 1998) showed a reduction in growth rates of transplanted LNCaP cells in athymic mice induced by a high-fat diet (40.5%) by dl-alpha tocopherol (synthetic vitamin E). The landmark study by Clark et al. (*JAMA,* 1996) provided evidence that 200 mcg of selenium could reduce the incidence of PC by 63%. This is consistent with the observation that selenium inhibited the growth of DU-145-an androgen-independent human cell line of PC-by 50% at a selenium dose of 1 × 10-6 M and by 98% at a dose of 10-4 M. For comparison, selenium serum levels in humans living in high selenium areas may be as high as 10-6 M (Webber et al., *Biochem. Biophys. Res. Commun.*, 1985). Our recommended vitamin E dose for prevention is 400 to 1000 IU a day as mixed tocopherols. Mixed tocopherols contain synthetic vitamin E (d-alpha-tocopherol and dl-alpha-tocopherol) as well as natural vitamin E. A study by Moyad et al. (in press, 1999) indicates gamma tocopherol has more anti-PC activity then conventional d-alpha-tocopherol.

The selenium dose recommended for prevention is 400 mcg a day. This is best given as selenomethionine, usually derived from yeast. Selenium works best in conjunction with vitamin E, which enhances its activity. Vitamin E works best in association with beta-carotene and vitamin C. We recommend 1000 mg of vitamin C to be taken after each meal to prevent fatty acid peroxide generation. In a likewise manner, coenzyme Q10 has been shown to prevent the oxidation of LDL cholesterol. In fact, the prevention of fatty acid oxidation may be just as important as decreasing fat consumption. We suggest coenzyme Q10 be taken at a dose of 200 mg a day. An added benefit of coQ10 is the improvement in heart function and diabetic control as well as the treatment of periodontal disease. CoQ10 works best when given with

vitamins E and C.

- **Use Genistein to Decrease Cell Adhesion, Slow Proliferation, and Decrease Metastatic Potential**

Incidences of PC are higher in the Western world than in Asia, where soy is consumed as part of the normal diet, producing higher levels of genistein in the blood, which in turn appear to prevent the expression of metastatic capacity in hormone-dependent cancers. Studies have shown that, in a cell-culture system, genistein appears to be cytotoxic and inhibitory of PC cell proliferation (Santibanez et al., *Anticancer Res.*, 1997; Peterson and Barnes, Prostate, 1993). Genistein's protein-tyrosine kinase-inhibiting effects have been identified as a cancer-prevention mechanism. One study examined genistein's effect upon cell adhesion as one possible mechanism by which it could be acting as an antimetastatic agent. A morphogenic analysis revealed that genistein caused cell flattening in a way that prevented metastatic adhesion of PC cell lines. We advise patients to eat a diet rich in soy products such as tofu, soy beans (edamame), soy milk, and miso. We recommend a breakfast and dinner drink that contains soy milk, isolated soy powder, many of the vitamins mentioned above, and strawberries. We use a Vita-Mix blender to pulverize the vitamins and add them to this drink. The protein-to-carbohydrate ratio of this drink is also close to the desired 3:4 ratio that Sears considers "zone" favorable. Life Extension makes a 700 mg Ultra Soy product that contains 134 mg of genistein per capsule as well as the isoflavones daidzein, and glycitein. We would suggest that clinical trials be initiated that would determine the genistein oral intake associated with blood genistein levels similar to those found in Asian men. Currently, we recommend 100 to 200 mg of genistein a day in addition to a diet high in soy products. We also believe that the major source of protein in our diet should come from soy.

- **Decrease Cell Proliferation with Pygeum and Silymarin**

Pygeum extract also has been shown to specifically inhibit prostate-cell proliferation by inhibiting protein kinase C enzyme activity (Yablonsky et al., *J. Urol.*, 1993).

Silymarin has been shown to have an anti-PC effect by virtue of increasing the levels of p27 (Zi et al., Cancer Res., 1998; Gali et al., *Proc. Annu. Meet. Am. Assoc. Cancer Res.*, 1994). Silymarin also has protective effects against liver cell injury and skin cancer (Kropacova et al., *Radiat. Biol. Radioecol.*, 1998; Agarwal et al., *Proc. Annu. Meet. Am. Assoc. Cancer Res.*, 1995; Katiyar et al., *J. Natl. Cancer Inst.*, 1997).

Product availability: Ultra Soy Extract, lycopene, silymarin, Natural Prostate Formula, Gamma E tocopherol, Vitamin C, Super Green Tea Extract Capsules Lightly Caffeinated, selenium and vitamin E can be ordered by phoning 1-800-544-4440 or order **OnLine**

EATING YOUR WAY TO PROSTATE CANCER

By William Faloon

Reviewed and critiqued by Stephen B. Strum, MD, FACP (Life Extension Scientific Advisory Board Member)

Online: http://www.lef.org/magazine/mag2007/feb2007_cover_prostate_01.htm

Cancer cells lurk in the prostate glands of most aging men, yet only one in six is ever diagnosed with prostate cancer. Scientists have discovered an enzyme called 5-lipoxygenase (5-LOX) that promotes the development of full-blown prostate cancer, along with angiogenesis and metastasis. The good news is that this lethal enzyme can be significantly reduced by eating a healthier diet and using supplements that specifically inhibit 5-LOX production.

Cancer cells lurk in the prostate glands of most aging men, yet only one in six men is ever diagnosed with prostate cancer.1 If one looks at what is required for a single cancer cell to develop into a detectable tumor, it becomes obvious that natural barriers exist to protect people against full-blown cancer.

Unfortunately, the dietary choices of most men living in the modern Western world circumvent the body's natural protective barriers. The end result is that we unwittingly provide biological fuel for existing prostate cancer cells to propagate and metastasize.

Good news: If you understand the biological roles of diet and specific nutrients, you'll be able to achieve a considerable amount of control over whether isolated cancer cells in your prostate gland will ever show up as a clinically diagnosed disease.

So keep reading, because what you learn here can make a difference between bad news and good news in your future.

The impact of the food we ingest on cell growth and death is so pronounced that it can be identical to the effects displayed by anti-cancer drugs. Unlike synthetic drugs, however, the proper dietary constituents produce no side effects and confer additional health benefits.

All cancers begin when genes that regulate cellular proliferation become so damaged that they can no longer control normal cell division. For example, scientists are actively engaged in clinical research using selenium because it helps protect specific genes that enable cells to divide normally.2-5 The limitation of a nutrient like selenium, however, is that it may not be able to reverse accumulated damage (mutations) to such cell-regulating genes.

Prostate cancer with infiltration into bladder, lymph nodes, and urethra

Prostate cell genes are especially prone to mutations early in the course of human life. This has been demonstrated by autopsy findings of prostate cancer cells in younger men who never knew they had the disease.6

Doctors continue to wonder why so many men with active cancer cells in their prostate glands do not progress to overt disease. One answer may relate to the discovery of a particular enzyme that prostate cancer cells use to propagate, infiltrate, and metastasize. A large volume of published research indicates that this enzyme functions via multiple pathological pathways to facilitate prostate cancer at various stages.7-21 The encouraging news is that this enzyme can be suppressed via dietary modification and the use of dietary supplements, many of which are already being utilized by health-conscious men today.

OMEGA 3 FATTY ACIDS: THE FIRST LINE OF DEFENSE

Diets high in omega-6 fats and saturated fats are associated with greater prostate cancer risk, whereas increased intake of omega-3 fats from fish has been shown to reduce risk.22-29 Based on consistent epidemiological findings across a wide range of human populations, scientists have sought to understand why eating the wrong kinds of fat (saturated and omega-6 fats) provokes a stimulatory effect on prostate cancer.

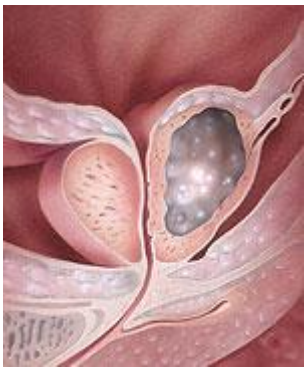
To ascertain what happens after we eat bad fats, all one has to do is look at the metabolic breakdown pathways that these fats follow in the body, as shown in the chart on the right (Figure 1). For example, let us assume that for dinner, you eat a steak (a source of saturated fat) and a salad, along with a typical salad dressing of soybean and/or safflower oils (sources of omega-6 fats).

As can be seen in Figure 1, both saturated and omega-6 fats convert to arachidonic acid in the body, whereas the meat itself contains arachidonic acid. One way that the body rids itself of excess arachidonic acid is by producing a dangerous enzyme called 5-lipoxygenase (5-LOX). New studies show conclusively that 5-LOX directly stimulates prostate cancer cell proliferation via several well-defined mechanisms.2,26,30-36 In addition, arachidonic acid is metabolized by 5-LOX to 5-HETE, a potent survival factor that prostate cancer cells utilize to escape destruction.31,37-40

Figure 1 clearly demonstrates how consuming a diet of foods rich in arachidonic acid directly provokes the production of the dangerous 5-LOX enzyme, which can promote the progression of prostate cancer. In addition to 5-HETE, 5-LOX also metabolizes arachidonic acid to leukotriene B4, a potent pro-inflammatory agent that causes destructive reactions throughout the body and inflicts severe damage to the arterial wall.41-47

One reason that fish oil supplements have become so popular is that their beneficial EPA/DHA fatty acids can help reduce production of arachidonic acid in the body. As shown in Figure 1, if arachidonic acid levels are reduced, there would be a corresponding suppression of 5-LOX, 5-HETE, and leukotriene B4.

Once one understands the lethal 5-lipoxygenase (5-LOX) cascades, it is easy to see why people who excessively consume foods rich in arachidonic acid, and those who do not reduce the production of excessive arachidonic acid metabolites, are setting themselves up for prostate cancer and a host of inflammatory diseases (including atherosclerosis).2,30,35,48,49



Prostate tumor confined to prostate gland.

5-LOX IS OVER-EXPRESSED IN PROSTATE CANCER

Based on studies showing that consumption of foods rich in arachidonic acid is greatest in regions with high incidences of prostate cancer,26,30,35,49 scientists sought to determine how much of the 5-LOX enzyme is present in malignant versus benign prostate tissues.

Figure 1. Flow chart showing how the body metabolizes common foods via the 5-lipoxygenase (5-LOX) pathway.

Using biopsy samples taken from living human patients, the researchers found that 5-LOX levels were an astounding six-fold greater in malignant prostate tissues compared to benign tissues. This study also found that levels of 5-HETE (a 5-LOX metabolite that prevents prostate cancer destruction) were 2.2-fold greater in malignant versus benign prostate tissues.33

The scientists concluded this study by stating that selective inhibitors of 5-LOX may be useful in the prevention or treatment of patients with prostate cancer.

5-LOX PROMOTES TUMOR GROWTH FACTORS

As the evidence mounts that ingesting "bad fats" increases prostate cancer risk, scientists are evaluating the effects of 5-LOX on various growth factors involved in the progression, angiogenesis, and metastasis of cancer cells.

One study found that 5-LOX activity is required to stimulate prostate cancer cell growth by epidermal growth factor (EGF) and other cancer cell proliferating factors produced in the

body. When 5-LOX levels were reduced, the cancer cell stimulatory effect of EGF and other growth factors was diminished.30

In a mouse study, an increase in 5-LOX resulted in a corresponding increase in vascular endothelial growth factor (VEGF), a key growth factor that tumor cells use to stimulate new blood vessel formation (angiogenesis) into the tumor. 5-LOX inhibitors were shown to reduce tumor angiogenesis along with a host of other growth factors.50 In both androgen-dependent and androgen-independent human prostate cancer cell lines, the inhibition of 5-lipoxygenase (5-LOX) has consistently been shown to induce rapid and massive apoptosis (cancer cell destruction).26,49,51-54

NUTRIENTS THAT SUPPRESS 5-LOX

Health-conscious people already take nutrients like fish oil that help to lower 5-LOX activity in the body.20,21 Studies show that lycopene and saw palmetto extract also help to suppress 5-LOX.51,55-68 The suppression of 5-LOX by these nutrients may partially account for their favorable effects on the prostate gland.

As humans age, however, chronic inflammatory processes can cause the over-expression of 5-LOX in the body. For maturing males, the result of excess 5-LOX may be the epidemic of prostate cancer observed after the age of 60.

Based on the cumulative knowledge that 5-LOX can promote the invasion and metastasis of prostate cancer cells, it would appear advantageous to take aggressive steps to suppress this lethal enzyme. The good news is that a natural 5-lipoxygenase (5-LOX) inhibitor is now available and has been added to a popular formula used to maintain healthy prostate function.

In addition to potentially suppressing prostate cancer, the successful inhibition of 5-LOX should also slow the progression of atherosclerosis.

CANCER-PROMOTING EFFECTS OF 5-LOX

Tumor Growth Factor	Cellular Effects	Inhibited by
Epidermal Growth Factor (EGF)	Stimulates tumor cell proliferation	5-Loxin®
Vascular Endothelial Growth Factor (VEGF)	Stimulates angiogenesis, tumor growth, and metastasis	5-Loxin®
Tumor Necrosis Factor-Alpha (TNF-α)	Induces matrix metalloproteinases, increases invasiveness and metastasis; Induces NF-kappaB, 5-Loxin® increases cell adhesion molecules (I-CAM, V-CAM)	5-Loxin®

Figure 2. 5-lipoxygenase (5-LOX) acts as biological fuel for cancer cells by stimulating EGF (epidermal growth factor), VEGF (vascular endothelial growth factor), and other growth factors. Tumor growth factors that enhance cancer cell proliferation, invasiveness, and metastasis can be inhibited by a natural product called 5-LOXIN®.

5-LOXIN®: NATURE'S 5-LOX INHIBITOR

Specific extracts from the Boswellia plant selectively inhibit 5-lipoxygenase (5-LOX).69,70 This is not surprising when one considers that various boswellia extracts have been used for centuries in India as anti-inflammatory agents.71

In several well-controlled human studies, boswellia has been shown to be effective in alleviating various chronic inflammatory disorders.72-82 Scientists have discovered that the specific constituent in boswellia responsible for suppressing 5-LOX is AKBA (3-O-acetyl-11-keto-B-boswellic acid). Boswellia-derived AKBA binds directly to 5-LOX and inhibits its activity.70 Other boswellic acids only partially and incompletely inhibit 5-LOX.70,83

Methods to extract high concentrations of AKBA from boswellia have been intensively investigated due to AKBA's potential in treating chronic inflammatory disorders. Even in standardized boswellia extracts, however, biologically active AKBA makes up only 2-5% of the final product.

Several years ago, researchers discovered how to obtain an economically viable boswellia extract standardized to contain a greater than 30% concentration of AKBA. This 30% AKBA extraction discovery was patented and given the trademark name "5-LOXIN®. When tested against the best commercial boswellia compounds, 5-LOXIN® exhibited better inhibitory action against 5-LOX.

MULTIPLE DANGERS OF EXCESS ARACHIDONIC ACID

In response to arachidonic acid overload, the body increases its production of enzymes like 5-lipooxygenase (5-LOX) to degrade arachidonic acid. Not only does 5-LOX directly stimulate cancer cell propagation,49,89-98 but the breakdown products that 5-LOX produces from arachidonic acid (such as leukotriene B4, 5-HETE, and hydroxylated fatty acids) cause tissue destruction, chronic inflammation, and increased resistance of tumor cells to apoptosis (programmed cell destruction).30,37,99-103

It is important to understand that 5-LOX is not the only dangerous enzyme the body produces to break down arachidonic acid. As can be seen in Figure 3, both cyclooxygenase-1 and cyclooxygenase-2 (COX-1 and COX-2) also participate in the degradation of arachidonic acid.

COX-1 causes the production of thromboxane A2, which can promote abnormal arterial blood clotting (thrombosis), resulting in heart attack and stroke.104-109 COX-2 is directly involved in cancer cell propagation,110-113 while its breakdown product (prostaglandin E2) promotes chronic inflammation.103,114,115 Most health-conscious people already inhibit the COX-1 and COX-2 enzymes by taking low-dose aspirin,106,115-119 curcumin,120-132 green tea,133-135 and various flavonoids such as resveratrol.136-138

A more integrative approach to this problem, however, would be to also reduce levels of arachidonic acid, which is the precursor of 5-HETE and leukotriene B4. In fact, if we focus on the metabolic pathways involved in arachidonic acid production and metabolism, we can understand why selective inhibitors of only the COX-2 enzyme, such as Vioxx® and Celebrex®, may be associated with an increased risk of heart attack and stroke.139,140 The fault lies not within the specific drug (Vioxx®, for example), but rather in a misguided approach that involves blocking only one of the pathways leading from arachidonic acid metabolism (the COX-2 pathway), while ignoring the three other enzymatic pathways (COX-1, 5-LOX, 12-15 LOX) through which arachidonic acid can be metabolized.

Vioxx® primarily blocks the COX-2 metabolic pathway of arachidonic acid, yet Americans taking this class of drug continued to overindulge in foods rich in arachidonic acid, which resulted in excess production of toxic 5-HETE, 12-15-HETE, and hydroxylated fatty acids. A focus on decreasing consumption of arachidonic acid—as well as inhibiting arachidonic acid production by means of fish oil and reducing consumption of insulin-stimulating carbohydrates—was completely ignored by the physicians who prescribed these drugs.

While Merck, the manufacturer of Vioxx®, is now being sued, Vioxx® was not the sole cause of the side effects seen in patients taking this drug. The primary culprit was the failure of scientists and physicians to take into account the basic biochemistry of omega-6 fatty acid and arachidonic acid metabolism. If patients prescribed COX-2 inhibitors were (1) advised to decrease their intake of omega-6 fats and arachidonic acid, (2) shown how to block arachidonic production by increasing their fish oil consumption and decreasing their

carbohydrate intake, and (3) advised to take steps to inhibit the COX-1 and 5-LOX pathways, the side effects attributed to Vioxx® may never have occurred.

5-LOXIN® DECREASES INFLAMMATION, INVASIVE POTENTIAL, TUMOR CELL ADHESIVENESS, AND ANGIOGENESIS

A rat study was conducted to evaluate the efficacy of 5-LOXIN® compared to the popular anti-inflammatory drug ibuprofen. 5-LOXIN® reduced inflammation by 27%, compared to 35% for ibuprofen.84 Another rat study compared 5-LOXIN® to the anti-inflammatory steroid drug prednisone. 5-LOXIN® reduced inflammation by 55%, which was similar to the prednisone used in the study.79,85 The significance of these findings is that prednisone and ibuprofen can be toxic when used chronically, whereas natural 5-LOXIN® is free of side effects.

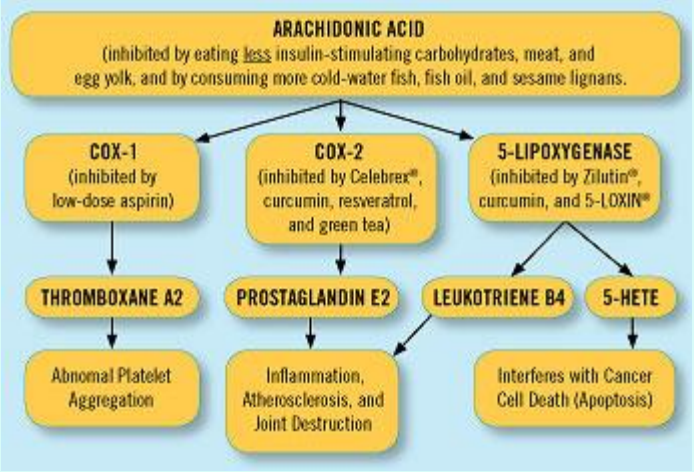
Ibuprofen has demonstrated anti-cancer effects, most probably due to its inhibition of cyclooxygenase-2 (COX-2), another enzyme that cancer cells use to facilitate their growth and survival. As you have just learned, 5-LOXIN® functions to block the 5-LOX enzyme. Since the effects of 5-LOXIN® and ibuprofen may be either additive or synergistic, a clinical trial of a combination of these agents is warranted.

Tumor necrosis factor-alpha (TNF-α) is a dangerous pro-inflammatory cytokine that often increases in aging people. In a gene-chip study, 5-LOXIN® blocked the expression of many genes that are sensitive to the pathological effects of TNF-α.84

From the standpoint of keeping prostate cancer cells in check, 5-LOXIN® was shown to prevent the TNF-α-induced expression of a protein-degrading enzyme called matrix metalloproteinase (MMP). Cancer cells use the MMP enzyme to tear apart natural barriers in the body that would normally encase them. Prostate cancer cells are notorious for inducing the production of this enzyme that causes containment structures within the prostate gland to vanish, thus enabling the mutated (cancerous) prostate cells to break through healthy prostate tissue and eventually metastasize.86

FIGURE 3. ARACHIDONIC ACID'S DESTRUCTIVE CASCADE

To better understand the pathways by which arachidonic acid can cause arthritic, carcinogenic, and cardiovascular conditions, the flow chart below shows how arachidonic acid cascades down into damaging compounds in the body.



Prostate cancer cells use adhesion molecules (known as VCAM-1 and ICAM-1) to facilitate their spread throughout the body. 5-LOXIN® was shown to prevent the up-regulation of these adhesion molecules, which are directly involved in inflammatory processes.85 Chronic inflammation is tightly linked to the induction of aberrant angiogenesis used by cancer cells to facilitate the growth of new blood vessels (angiogenesis) into tumors.87

DAILY USE OF ASPIRIN MAY DECREASE PROSTATE RISKS

Most Life Extension members take aspirin to reduce their risk of heart attack and stroke. A new study shows that taking one aspirin tablet (325 mg) every day may lower the risk of prostate enlargement.

Researchers studied 2,447 men over 12 years, examining them every other year. After adjusting for age, diabetes, hypertension, and other factors, they found that men who took

a daily aspirin or another NSAID (like ibuprofen) reduced their risk of moderate or severe urinary symptoms by 27% and lowered their risk of an enlarged prostate by 49%. Even more intriguing was the finding that men who consumed aspirin or another NSAID were 48% less likely to have an elevated level of prostate-specific antigen (PSA), the protein measured in the blood that helps detect prostate cancer.141

Aspirin inhibits the cyclooxygenase (COX-1 and COX-2) enzymes, which are also involved in the arachidonic acid inflammatory pathway. Like 5-lipoxygenase, COX-2 is known to promote the proliferation of prostate cancer cells.114

CONCLUSION

A plethora of research documents the role of chronic inflammatory mediators such as 5-lipooxygenase (5-LOX) and tumor necrosis factor-alpha (TNF-α) in the manifestation and progression of prostate and other cancers.7-19,30,31,33,49,52-54,87,88

The typical American diet is high in omega-6 fatty acids, saturated fats, and arachidonic acid. Over-consumption of these foods, and under-consumption of omega-3 fatty acids, contribute significantly to systemic chronic inflammatory states.

Boswellia extracts have been thoroughly studied as natural remedies for inflammatory disorders. A patented extract from boswellia called 5-LOXIN® has potent ability to inhibit the enzyme 5-LOX, preventing the formation of protein-degrading enzymes, and protecting against inflammation-induced events that can promote tumor angiogenesis.

EATING YOUR WAY TO PROSTATE CANCER: WHAT YOU NEED TO KNOW

- Prostate cancer cells are present in most men, yet only one in six men is ever diagnosed with the disease. Natural barriers help to protect some men from developing clinically diagnosable prostate cancer.
- Poor dietary choices can break down the body's innate defenses against the development of prostate cancer, while fueling its proliferation and spread. Consuming a healthy diet and specific protective nutrients can provide significant support against prostate cancer.
- A comprehensive strategy to fight prostate cancer should focus on inhibiting the 5-lipoxygenase (5-LOX) enzyme, which is central to the cancer's propagation, infiltration, and spread. This can be done by limiting intake of foods that contain or

stimulate arachidonic acid and thus increase 5-LOX production, such as red meat, egg yolks, dairy products, saturated and omega-6 fats, and high-glycemic carbohydrates. Healthier dietary choices are cold-water fish, fish oil, and sesame lignans.

- Certain nutrients protect the prostate by suppressing 5-LOX activity and production of metabolites. These include fish oil,20,21 lycopene,51 and saw palmetto.68 A novel extract of the boswellia plant called 5-LOXIN® strongly inhibits 5-LOX.
- 5-LOXIN® exerts powerful anti-inflammatory effects and blocks the expression of enzymes and cytokines that can lead to the proliferation and spread of cancer.
- Excess arachidonic acid in the body stimulates not only prostate cancer, but also processes that lead to heart attack, stroke, and chronic inflammation. Health-conscious people can reduce arachidonic acid's toxic effects by eating a healthy diet and utilizing cyclooxygenase (COX) inhibitors, which inhibit the toxic byproducts of arachidonic acid metabolism. COX inhibitors include aspirin, curcumin, green tea, and resveratrol.
- Blocking both the LOX and COX pathways, in addition to making wise dietary choices, is essential to limiting the dangerous effects of arachidonic acid, including cancer and heart disease.

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CURCUMIN (TURMERIC) **AND PROSTATE CANCER: KEY RESEARCH FINDINGS**

Source: **www.prlabs.com**

Curcumin (a major component of turmeric) **inhibits proliferation, induces apoptosis, and inhibits angiogenesis prostate cancer cells.**

Reference: Curcumin inhibits proliferation, induces apoptosis, and inhibits angiogenesis of LNCaP
prostate cancer cells in vivo. Dorai T, Ccao YC, Dorai B, et al. *Prostate* 2001;47:293-303

BACKGROUND

Earlier work from our laboratory highlighted the therapeutic potential of curcumin (turmeric), used as a dietary ingredient and as a natural anti-inflammatory agent in India and other Southeast Asian countries. This agent was shown to **decrease the proliferative potential and induce the apoptosis potential of both androgen-dependent and androgen-independent prostate cancer cells** in vitro, largely **by modulating the apoptosis suppressor proteins and by interfering with the growth factor receptor signaling pathways** as exemplified by the EGFreceptor.

To extend these observations made in vitro and to study the efficacy of this potential anticancer agent in vivo, the growth of LNCaP cells as heterotopically implanted tumors in nude mice was followed.

METHODS

The androgen-dependent LNCaP prostate cancer cells were grown, mixed with Matrigel and injected subcutaneously into nude mice. Experimental group received a synthetic diet containing 2% curcumin for up to 6 weeks. At the end point, sections taken from the excised tumors were evaluated for pathology, cell proliferation, apoptosis, and vascularity.

RESULTS

Curcumin causes a marked decrease in the extent of cell proliferation as measured by the

BrdU incorporation assay and a significant increase in the extent of apoptosis as measured by an in-situ cell death assay. Moreover, a significant decrease in the microvessel density as measured by the CD31 antigen staining was also seen.

CONCLUSIONS

Curcumin could be a potentially therapeutic anti-cancer agent, as it **significantly inhibits prostate cancer growth**, as exemplified by LNCaP in vivo, and has the potential to prevent the progression of this cancer to its hormone refractory state.

Mushroom compound suppresses prostate tumors

Source: Contact: Stephanie Harrington
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A mushroom used in Asia for its medicinal benefits has been found to be 100 per cent effective in suppressing prostate tumour development in mice during early trials, new Queensland University of Technology (QUT) research shows.

The compound, polysaccharopeptide (PSP), which is extracted from the 'turkey tail' mushroom, was found to target prostate cancer stem cells and suppress tumour formation in mice, an article written by senior research fellow Dr Patrick Ling in the international scientific journal *PLoS ONE* said.

Dr Ling, from the Australian Prostate Cancer Research Centre-Queensland and Institute for Biomedical Health & Innovation (IHBI) at QUT, said the results could be an important step towards fighting a disease that kills 3000 Australian men a year.

"The findings are quite significant," Dr Ling said.

"What we wanted to demonstrate was whether that compound could stop the development of prostate tumours in the first place.

"In the past, other inhibitors tested in research trials have been shown to be up to 70 per cent effective, but we're seeing 100 per cent of this tumour prevented from developing with PSP.

"Importantly, we did not see any side effects from the treatment."

Dr Ling said conventional therapies were only effective in targeting certain cancer cells, not cancer stem cells, which initiated cancer and caused the disease to progress.

During the research trial, which was done in collaboration with The University of Hong Kong and Provital Pty Ltd, transgenic mice that developed prostate tumours were fed PSP for 20 weeks.

Dr Ling said no tumours were found in any of the mice fed PSP, whereas mice not given the treatment developed prostate tumours. He said the research suggested that PSP treatment could completely inhibit prostate tumour formation.

"Our findings support that PSP may be a potent preventative agent against prostate cancer, possibly through targeting of the prostate cancer stem cell population," he said.

He said PSP had been previously shown to possess anti-cancer properties, and 'turkey tail' mushrooms (known as Coriolus versicolor or Yun-zhi) had been widely used in Asia for medicinal benefits.

However, Dr Ling said it was the first time it had been demonstrated that PSP had anti-cancer stem cell effects.

Although 'turkey tail' mushrooms had valuable health properties, Dr Ling said it would not be possible to get the same benefit his research showed from simply eating them.

NEW TREATMENT FOR PROSTATE CANCER GIVES 'PERFECT RESULTS' FOR NINE IN TEN MEN: RESEARCH

Source: http://www.telegraph.co.uk/health/healthnews/9206425/New-treatment-for-prostate-cancer-gives-perfect-results-for-nine-in-ten-men-research.html

A new treatment for prostate cancer can rid the disease from nine in ten men without debilitating side effects, a study has found, leading to new hope for tens of thousands of men.

It is hoped the new treatment, which involves heating only the tumours with a highly focused ultrasound, will mean men can be treated without an overnight stay in hospital and avoiding the distressing side effects associated with current therapies.

A study has found that focal HIFU, high-intensity focused ultrasound, provides the 'perfect' outcome of no major side effects and free of cancer 12 months after treatment, in nine out of ten cases.
Traditional surgery or radiotherapy can only provide the perfect outcome in half of cases currently.
Experts have said the results are 'very encouraging' and were a 'paradigm' shift in treatment of the disease.

It is hoped that large scale trials can now begin so the treatment could be offered routinely on the NHS within five years.

NUTRITION AND THE PROSTATE

Source: *The China Study* by T. Colin Campbell (157-182), www.thechinastudy.com

We will briefly consider three all too common problems with the male prostate: infection, enlargement and malignancy.

PROSTATITIS (Infection of the prostate)

Bacterial infection of the prostate may be acute or chronic. A nonbacterial prostatitis is actually more common. (*Merck Manual*, 14th ed., pp 1566-1567) Saturation doses of vitamin C are at least as effective as antibiotics in any of these conditions. We know this through the work of Frederick R. Klenner, M.D., Robert Cathcart, M.D. and other physicians who have used very large doses of vitamin C to cure infections for decades. Vitamin C is admittedly nonspecific, but no more so than the pharmaceutical antibiotics that are given for infection no matter where in the body it may be. Vitamin C has the advantages of being cheaper and considerably safer than drugs. Saturation of vitamin C is indicated by diarrhea, so one takes just less than the amount that would produce loose bowels. It will be a lot, measured in grams and not milligrams. The need for vitamin C will diminish as the infection subsides. A maintenance dose effectively helps to prevent a recurrence.

If there were a mineral that could be as important for the prostate as vitamin C is, it would be zinc. Infection or other stress results in lower blood serum zinc levels in general and lower prostate levels in particular. **In prostatitis, zinc levels are only ONE-TENTH of those in a normal prostate.** (Fair and Heston,1977; Pfeiffer, 1978) One time-tested prostate remedy is eating pumpkin seeds. It is no surprise that pumpkin seeds are a good source of zinc, as are shellfish (especially oysters, which would account for still more folklore) and nutritional yeast. A daily zinc supplement totaling 50 to 100 milligrams is frequently

recommended in the natural healing literature, and that amount cannot be faulted by medical literature.

Since men lose zinc in every seminal emission, their need for the mineral is higher than a woman's. Research by Dr. Irving M. Bush and the Center for the Study of Prostatic Diseases in Chicago employed 50 to 100 mg of zinc per day for as long as 4 months to as little as only two weeks. There was prompt improvement in 70 per cent of the cases.

Not bad for just a single mineral. (Taylor DS. Nutrients can remedy prostate problems. *Today's Living*, February 1990, p 12-13.)

ENLARGED PROSTATE; BENIGN PROSTATIC HYPERTROPHY or HYPERPLASIA (BPH)

The *Merck Manual* has historically indicated surgery as "definitive" therapy for this common condition. Medication is now commonly prescribed first, one of the more popular being finasteride ("Proscar") manufactured, conveniently enough, by the Merck company. Proscar is actually a somewhat dangerous substitute for an herbal remedy, as at least half of all pharmaceuticals are. The classic herb pirated in this case is the **saw palmetto berry**.

Saw palmetto is a shrub that grows down south in Georgia and Florida along the ocean. The leaves are palm-like, and the stems are saw-toothed, hence the name. According to *The Herb Book* by Dr. John Lust, a teaspoon of the dark-colored berries is steeped in one cup of water, and that is taken once or twice daily. There are no side effects or contraindications listed. This is in sharp contrast with the drug Proscar. Proscar has many serious side effects, and they are stated on the package insert, published in advertisements for the drug, and listed in the *Physician's Desk Reference* (*PDR*). The *PDR* is available in any bookstore or library, and you will find a copy at all drug counters. European studies have confirmed that saw palmetto berries are a statistically significant therapy for enlarged prostate. They are clearly a safer treatment, and cheaper as well.

Zinc is as helpful with enlarged prostates as it is with inflamed ones, since zinc deficiency results in prostate enlargement. Very few men obtain even the low US RDA of 15 milligrams of zinc a day, and this would explain a lot. Supplemental doses, commonly between 50 and 100 mg daily, may help shrink a swollen prostate. Toxicity of zinc is very low. Side effects of diarrhea and anemia begin at about 500 mg daily, vastly more than anyone would need to

take. (Even at that level, supplemental iron and copper alleviate the side effects.) How effective is zinc therapy? **Dr. Irving Bush (mentioned previously) of the Chicago Medical School and researchers from Cook County Hospital studied over 5,000 patients and have confirmed that zinc prevents prostate enlargement.**

Vitamin C would almost certainly be of benefit to the enlarged prostate. At the very least, infection would be avoided. Additionally, men with enlarged prostates report that vitamin C's modest diuretic effect makes urination easier.

Lycopene, the natural antioxidant pigment that makes tomatoes red, has been demonstrated to slow or even halt the growth of benign prostatic hyperplasia (BPH). In a recent study, men got results when given a mere 15 mg of lycopene per day. (Schwarz S, Obermüller-Jevic UC, Hellmis E, Koch W, Jacobi G, Biesalski HK. Lycopene inhibits disease progression in patients with benign prostate hyperplasia. *J Nutr*. 2008 Jan;138(1):49-53.)

PROSTATE CANCER

This is the one we're really worried about. There is much that can be done to prevent this number two cancer killer of American men. Adequate, even abundant, nutrients strengthen the body's immune system and prevent cancer. As mentioned above, optimum prostate health requires vitamin C and zinc in particular.

Some widely-publicized reports (such as *J Natl Cancer Inst*. 2007 May 16;99[10]: p 754-64) would have you believe that zinc supplementation actually encourages or even *causes* prostate cancer This is unlikely for the following reasons:

1. The study authors themselves actually said that "no association was observed between multivitamin use and risk of localized prostate cancer."

2. The zinc doses examined were low, usually only modest variants of RDA-multivitamin levels of 11 mg/day.

3. The data was collected "as part of a self-administered, mailed food-frequency questionnaire." This is far from the most reliable form of research.

4. While the study "found an increased risk of advanced and fatal prostate cancers" among extra-multivitamin users, the very same tablets also contain vitamin C, vitamin D, beta-carotene, and selenium. All of these nutrients have repeatedly been shown to *fight* cancer. This is another reason to suspect statistical invalidity in this study.

5. Men often do not begin taking vitamin and zinc supplements until after they are diagnosed as having prostate cancer. More cancer patients would be taking more zinc, which would certainly skew the results. Furthermore, this is not causation, this is correlation.

THE REAL STORY:

There is no doubt whatsoever that diet has a major role in allowing - or stopping - prostate cancer. For example, a Harvard University School of Public Health study indicated that you are 250% more likely to suffer advanced prostate cancer if you eat red meat every day than if you eat red meat only once a week. The message is clear and generally ignored: move your diet in the direction of vegetarianism, and start today (*USA Weekend*, December 3-5, 1993, p 14).

Vitamin D fights prostate cancer. Be sure to read this very important article by John J. Cannell, M.D.: http://www.vitamindcouncil.com/cancerProstate.shtml To learn more, I recommend a free and quick Medline search (http://www.ncbi.nlm.nih.gov/sites/entrez) for papers by "Holick MF." My interview with Dr. Holick is posted at http://www.doctoryourself.com/news/v6n6.rtf This may also be useful to you: http://www.doctoryourself.com/dvitamin.htm

Eating a lot of lycopene-rich, fresh **tomatoes** has been shown to radically reduce your prostate cancer risk. (A Medline search at http://www.ncbi.nlm.nih.gov/sites/entrez will bring up dozens of supporting studies.)

Soy products appear to have a special benefit against prostate cancer. **Japanese men have especially low death rates from prostate cancer, even thought they get the disease as often as American men do.** The Japanese eat a lot of tofu, tempeh, miso, soy milk and other soy foods. Even animals fed a lot of soybeans have far less prostate cancer than others. There are at least two specific substances in soybeans that seem to help fight

cancer: genistein and isoflavinoids. These natural chemicals are especially effective against the hormone-dependent cancers, which includes prostate cancer. (Soybean products may lower prostate cancer, *Lancaster Intelligencer-Journal*, January 12, 1994)

Prostate cancer is very slow growing. Because of this, radical measures such as radiation or surgery are often reasonably postponed. This "watchful waiting," to see if surgery is truly needed, is advocated by more and more doctors. Obviously, regular medical examination and follow-up is important. Although there is question as to whether it actually saves lives, the Prostatic Specific Antigen (PSA) blood test is one way to monitor the prostate's condition. The actual benefits of surgery and radiation therapy are statistically quite small. After ten years, only slightly more of the treated patients are still alive than those that did nothing at all (Prostate cancer cure questioned, *Associated Press*, January 27, 1994).

In the mean time, an especially good diet and appropriately generous use of supplements may positively influence the situation. It certainly cannot hurt to have lots of raw salad foods, sprouts, and fresh vegetable juices every day. Natural health research has continually emphasized these measures to help fight cancer. A particularly good example is the work of Max Gerson, M.D. Dr. Gerson used a mostly raw food and fresh vegetable juice diet for cancer patients with remarkably good results. He also used substantial quantities of vitamin supplements. His entire program is set forth in a tremendously valuable book entitled ***The Gerson Therapy***, by Charlotte Gerson and Morton Walker (2001) NY: Kensington Publishing Corp. ISBN 1-57566-628-6 (paperback, 371 pages, plus appendixes and index http://www.doctoryourself.com/gersontherapy.html .)

To learn more about how to do the Gerson Therapy, you can start with these pages:
http://www.doctoryourself.com/gersonbio.htm
http://www.doctoryourself.com/gersonmovie.html

http://www.doctoryourself.com/gersonspeech.html is a transcript of a speech by Dr. Gerson himself.

http://www.doctoryourself.com/bib_gerson_therapy.html is a bibliography of published clinical studies showing the demonstrated benefits of the Gerson treatment.

http://www.doctoryourself.com/bib_gerson.html is a bibliography of all of Dr. Gerson's scientific writings.

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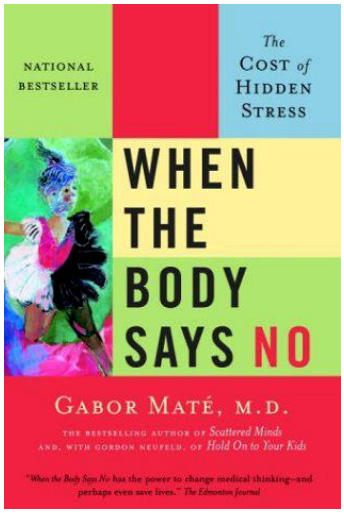
For more information, you might also want to read *DOCTOR YOURSELF* (http://www.doctoryourself.com/saulbooks.html) and *FIRE YOUR DOCTOR!* (http://www.doctoryourself.com/review.html).

CHAPTER 8: SOMETHING FOOD COMES OUT OF THIS [PROSTATE CANCER AND MIND/EMOTIONS/WAYS OF BEING]

Source: *When the Body Says No* by Dr. Gabor Mate



ED WAS DIAGNOSED AFTER his general practitioner found a small nodule during a routine rectal exam. "I went for a biopsy," he reports, "and they did six hits on the prostate. They found an irregularity in one hit. Prostate cancer. Since then I've looked at all the options, and it was all either slash, burn or poison. I've spoken with a lot of men who have had their prostate removed, and some who have had radiation. It's been pretty lousy for most of them."

"You haven't had any medical treatment?" I ask Ed.

"I've been to a naturopath, and I am doing hypnotherapy, and I've been doing a lot of looking at myself and how I've lived my life."

Ed's colorful phrase, "slash, burn or poison" refers to the three major types of treatment currently offered for prostate cancer: surgery, radiation and chemotherapy. Although some patients come through such treatments without harm, others suffer unpleasant

consequences such as urinary incontinence and impotence. A review of over a hundred thousand prostatectomy cases published in 1999 concluded that "complications and re-admission after prostatectomy are substantially more common than previously recognized." [1]

Those risks might be acceptable if the treatments available cured disease or saved lives, but the evidence is ambivalent at best. The loud public campaigns urging men to undergo screening tests for prostate cancer by means of the rectal digital exam or the prostate specific antigen (PSA) blood tests have no proven scientific basis. **"I think it's important for people to realize that once we find their prostate cancer, we still have no evidence that treatment works," Timothy Wilt, associate professor of medicine at the Minneapolis Veterans Affairs Medical Center**, told The New York Times. [2] "And that's really the whole crux of the screening issue: If treatment doesn't work, why are we using the PSA to look for tumors?"

Supporters of aggressive medical approaches ought to be disheartened by statistics gathered by Dr. Otis Brawley, a medical oncologist and epidemiologist at the U.S. National Cancer Institute. In places where screening is widely practiced, the incidence of diagnosed prostate cancer goes up, and the number of men being treated increases, but the death rate from prostatic malignancy remains unchanged." If anything, prostate cancer mortality rates were slightly higher in the intensely screened areas. Also disturbing are findings published in The Journal of the National Cancer Institute, that men aggressively treated for prostate cancer had a higher chance of dying of other cancers than men who did not receive any medical intervention.[4]

Although some prostate cancer probably should receive treatment, at this point it is not known exactly who would benefit from intervention. **Most prostate cancers are very slow to develop, so much so that the man is likely to die before the malignancy triggers any health problems, if it ever would**. In others cases, the cancer is so aggressive that by the time of diagnosis, treatment makes no difference. **Since there is no reliable way of deciding when treatment works, what are people who "survive" their prostate cancer really surviving their treatment or their disease?** In the case of prostate malignancy, medicine as it is commonly practiced simply does not apply the usual scientific standards.

Public opinion is based on the common-sense view that the sooner a condition is discovered, the more likely doctors will be able to cure it. Convinced that medical intervention saved their lives, celebrities like Gen. Norman Schwarzkopf, the golfer Arnold Palmer or the Canadian federal cabinet minister Allan Rock all diagnosed with prostate cancer after screening tests act as persuasive advocates of early diagnosis. Men need to let science, not the latest public figure endorsing PSA testing, help them make a decision about prostate cancer screening and treatment, Dr. Otis Brawley told The Journal of the American Medical Association. [5]

Despite scientific confusion, bias toward treatment is powerful. Few doctors are willing to let nature take its course in the face of potential disease, even if the value of intervention is questionable. And men, even if well informed, may choose to "do something" rather than tolerate the anxiety of inaction. But patients always deserve to be told what is known about prostate cancer - and, just as important, all that remains unknown.

Prostate cancer was the first human malignancy to be linked with hormonal influences. Just as cancer of the breast may improve in women who have their ovaries removed, so castration leads to a shrinking of prostate tumors, due to diminished levels of androgens, or male hormones. Orchidectomy, the surgical removal of the testicles, remains part of the treatment arsenal, as does the administration of powerful medications blocking the effects of the male hormones. Such "chemical castration" is the first-line treatment now offered men with metastatic prostate cancer.

Given the strong connection between hormone levels and emotions, it is striking how completely medical research and medical practice have ignored psychological influences on the causation of prostate cancer and have eschewed more holistic approaches to its treatment. There has been virtually no investigation of personality or stress factors in prostate malignancy. Textbooks ignore the subject.

The neglect of potential links between stress, emotions and prostate cancer is all the less justifiable given what is already known. By their thirties, many men will have some cancerous cells in their prostate, and by their eighties, the majority are found to have them. By the age of fifty, a man has a 42 per cent chance of developing prostate cancer. Yet relatively few

men at any age will progress to the point of overt clinical disease. In other words, the presence of cancerous prostate cells is not unusual even in younger men, and it becomes the norm as men get older. Only in a minority does it progress to the formation of a tumor that causes symptoms or threatens life. It is worth asking how stress may promote the development of malignant disease. What personality patterns or life circumstances may interfere with the body's defense mechanisms, allowing the already-present cancer cells to proliferate?

As I arrived to interview Ed, a wiry man with a body and face of someone years younger than his age of forty-four, he turned to his wife, Jean, who was just leaving to go shopping. "It's a pain in the ass," he said, "but I have to go and look at so-and-so's truck for him. It's not starting."

"Let me ask you something right away," I begin.

"Sure. "

"You're saying that looking at this guy's truck is a pain in the ass. Now that's an interesting metaphor, anatomically, when used by somebody who has cancer of the prostate. How easy has it been in your life to say no to things that were actually more of a pain in the ass than a benefit to you?"
"I really don't say no. I try to help people all the time."

"Even if it's a pain?"

"Yeah. Even if it's not the greatest time for me, or I should be doing other things that are more important for me. I like to help people out."

"What happens if you don't?"

"I feel bad about it. Guilty."

Ed, leader of a country music band, used to do cocaine, mescaline and marijuana, "two or three joints a day, my whole youth. A problem for me ever since my childhood has been alcohol." Ed tells me about his first adult relationship, which lasted ten years. He lived with an older woman whose two children he helped to bring up, drinking daily to suppress his unhappiness. That relationship came to an end when his partner had an affair.

"I threw in the towel. I said, I don't want to put up with this. I never screwed around, even though I felt like it. From that day on, I quit drinking for a year and a half, started jogging and doing what I wanted to do. I had this free feeling, like this huge weight was off my chest. I could do anything I wanted to and I felt so good about myself."

"How much are you drinking these days?"

"Maybe about four beer a day. Every day. "

"What does it do for you?"

"Jean and I got hooked up, and her problems become my problems, and it just gets heavier and heavier and heavier, and then I start with alcohol again."

"So in some ways you are not happy in this marriage."

"I guess the biggest thing is the control factor. I've allowed Jean to take control of this marriage, because of her multiple sclerosis and because she came from such an abusive marriage.* She was dictated to, told what clothes to wear and all that kind of stuff. What in turn it's done is made me cower in this marriage."

"So you see yourself as being controlled. How do you feel about that?"

"I'm resentful."

"And how do you deal with it?" "I hide it."

"You don't tell her that you don't like it?"

"No. I don't."

"What does that remind you of?"

"My childhood? Exactly."

Although Ed had told me previously that he had had a "very great upbringing," it soon became evident that he had felt controlled by his parents and full of guilt if he failed to meet their expectations. He recalled he had received what he called "deserved spankings," which, on further inquiry, turned out to have been beatings with a belt administered by his father, from about age eight on." He believed that that was the best way of doing things."

"What do you believe?"

"Well, now, I don't think that was the best thing he could do, but you really don't have much choice when you're a young child. I wanted to be a good person. When you're a child looking at your father, you don't know what he's supposed to be, because you want your dad to be perfect, and you want to be a perfect child."

One of the puzzling features of prostate malignancy is that while testosterone the hormone people have been led to believe is responsible for male aggression seems to promote its growth, this cancer is most typically a disease of older men. Yet the body's production of testosterone declines with aging. Nor have men with prostate cancer been shown to have higher than average blood levels of testosterone. As with estrogen receptors in breast cancer, it appears the sensitivity of tumor cells to normal concentrations of testosterone must have been altered.

Like hormone secretion by the adrenal glands and the ovaries, the synthesis of testosterone by the testicles is under the complex feedback control of the hypothalamic-pituitary system in the brain. That network, highly reactive to stress and emotions, sends a cascade of biological substances into circulation. Emotional factors can directly influence male sex-

hormone functioning for good or ill - just as the female hormone estrogen from the ovaries, or adrenalin, cortisol and other hormones from the adrenal glands, are affected by psychic events. It so happens that in a small series of patients, surgical removal of the brain's pituitary gland did show positive results in the treatment of prostate cancer. [6]

Testosterone gets a bad rap. If one wishes to compliment a woman's self-confidence or assertiveness, one will assert that she "has balls." A Canadian columnist wrote in praise of Margaret Thatcher, the iron-willed or merciless, depending on one's vantage point former British prime minister, that she had "10 times more testosterone than the men." Meanwhile, male destructiveness and hostile aggression are frequently blamed on testosterone. In actual fact, high levels of the hormone are more an effect than a cause.

Victory or defeat was shown to alter not only the hormonal balance but even the brain cells in a species of fish, the African cichlid. "In defeat, the fish's hypothalamic cells shrink with consequent declines in reproductive hormones and shrinkage of the testes." If the situation is manipulated to permit defeated fish to become dominant, there is a dramatic growth of the cells in the hypothalamus that produce a gonadotropin-releasing hormone (GRH), which stimulates the pituitary to produce hormones that act on the testes. The testes, in turn, will now increase in size, and the fish's sperm counts will improve. "Most importantly, this research has clearly demonstrated . . . that it is the behavioral changes [i.e., the attainment of dominant status] that lead to the subsequent physiologic changes." [7]

As highly evolved creatures, we may like to believe that our gonadal functioning is not as readily susceptible to life's ups and downs as that of the lowly African cichlid. In fact, human hormone levels, like those in our African fish, may follow rather than precede changes in dominance relationships. Prof. James Dabbs, a social psychologist at Georgia State University in Atlanta, has researched the interaction of testosterone and behavior. According to a report in The New York Times, after reviewing his nearly forty studies he has concluded that while testosterone does increase libido, "there is no proof it causes aggression." On the other hand, there is proof that emotional states can rapidly alter testosterone production: "Dr. Dabbs tested fans before and immediately after the 1994 World Cup of soccer final between Italy and Brazil. In what Dr. Dabbs considers proof of the axiom 'basking in reflected glory,' testosterone levels swelled among the victorious Brazilians and sank among

the dejected Italians."[8] Not surprisingly, then, gonadal function is affected by psychological states in both men and women. In depressed men, the secretion of testosterone and other hormones connected with sexual functioning were found to be significantly diminished.[9] A hormone-dependent malignancy like that of the prostate may be highly susceptible to bio chemical influences related to stress and emotional states.

Cancer of the prostate is the second commonest malignancy of men. Only cancer of the lung occurs more frequently. Calculations vary, but in the United States in 1996 as many as 317,000 new cases were estimated, and about 41,000 deaths. [10] About 20,000 new cases are diagnosed in Canada each year.

Environmental factors must be significant. Japanese men migrating to Hawaii and the continental United States were found to have a higher incidence of the disease than those natives of the country who stayed in Japan: over two and a half times as great. Yet on autopsies of men without clinical disease, similar rates of inactive malignant cells were found regardless of geography.l1 The question, then, is, Why do these inactive cells develop into cancerous tumors in one environment but not in another? There are highly suggestive epidemiological findings to indicate that stress crucially influences who will and who will not suffer illness and death from prostate cancer.

Family history increases the risk for prostate cancer, but it is not a major factor in most instances. No specific cancer-inducing environmental agents have been identified comparable to, say, cigarettes and lung cancer. Saturated fats may play a role. Given the wide geographic variation, so may genetic influences. The disease is most prevalent in the Scandinavian countries, least in Asia. The single racial/ethnic group at highest risk in the world are African Americans, among whom prostate cancer is twice as common as among the U.S. white population.

"African-American men have a poorer survival rate than whites for all stages of prostate cancer when the cancer is diagnosed at younger ages." [12] One could ascribe this higher death rate to the reduced access to medical care generally available to lower-middle-class and working-class people in the US. health system. However, the racial differences in prostate malignancy cut across class lines. **In any case, greater access to medical care has**

not so far been shown to have any positive effect on survival. We could possibly attempt to explain the difference in death rates by referring to genetic factors, except that American blacks experience prostate cancer at a sixfold rate compared with black men in Nigeria. Here, too, the presence of clinically "silent" prostate cancer cells is the same in the two groups. [13]

Now, if environmental factors such as caloric intake were responsible for the development of the disease, one would not expect much difference in the death rate between American whites and blacks. As it stands, only about 10 per cent of the black/white variation in cancer rate has been estimated to be due to the intake of saturated fats.[14] If, on the other hand, genetic influences were decisive, disease rates between blacks in the US. and Nigeria ought to be much closer than they are.

The historical, social and economic position of black people in US. society has undermined cohesion in black communities and black families and has imposed greater psychological stress on African Americans than their Caucasian fellow citizens or that blacks in Africa find themselves under. There is a parallel here in the higher occurrence of elevated blood pressure among American blacks. Hypertension is a condition clearly related to stress. In an analogous example, the rates of an autoimmune disease, rheumatoid arthritis, suffered by blacks in South Africa under apartheid increased as they migrated to the city from their native villages, even if in strict financial terms they may have gained by the move. The major factor would seem to be the psychological pressures of living in an environment where official racism directly and overtly deprived people of autonomy and dignity, while it uprooted people from their traditional family and social supports.

A finding consistent with what we have seen elsewhere in relationship to disease and emotional isolation is that men who are currently married, compared with men who are divorced or widowed, are less likely to be diagnosed with prostate cancer.[15] While I was not able to find in the literature any other investigation specific to prostate cancer and psychological factors, one study did look at men who had greater dependency needs than a comparable group that is, men who were less able to experience themselves as individuated, self-reliant adults. This study concluded that dependent men were more likely to develop a number of diseases, including prostate and other cancers.[16]

What would be the practical implications if a holistic perspective gained more research support and was incorporated into the medical view of prostate cancer? First, the promotion of anxiety-producing examinations and tests would cease, at least until we had definite proof of their usefulness. In June, 1999 the US. Postal Service planned to issue a stamp urging "annual checkups and tests" for cancer of the prostate. The New England Journal if Medicine warned against such foolishness, pointing out that the message was "inconsistent with current scientific evidence and thinking within the medical community." [17] Second, we would not subject tens of thousands of men to invasive and potentially harmful surgery and other equally unproven interventions without fully informing them of the uncertainty that shrouds the treatment of prostate cancer.

A holistic approach that places the person at the centre, rather than the blood test or the pathology report, takes into account an individual life history. It encourages people to examine carefully each of the stresses they face, both those in their environment and those generated internally. In this scenario the diagnosis of prostate cancer could serve as a wake-up call rather than simply a threat. In addition to whatever treatment they may choose to receive or not receive, men who are encouraged to respond reflectively, taking into account every aspect of their lives, probably increase their chances of survival.

A transformation appears to have affected Rudy Giuliani, diagnosed with prostate cancer in April 2000, in the midst of his Senate race against Hillary Clinton. The former mayor of New York City has been described as a driven man, "a robo-mayor immune to fatigue, fear, or self-doubt," who "lived and breathed the work ethic." [18] He completely identified with his role, slept only four hours a day and worked most of the other twenty. It was said of him that he could not abide being away from the centre of the action. He had to have a hand in everything, needing to be in control, "barking orders like a general." He had failed to show compassion to suffering individuals and groups and had displayed emotional tightness to an extreme degree. After his diagnosis, he made a remarkable public confession. Referring to his cancer, he said:

> It makes you figure out what you're all about and what's really important to you and what should be important to you - you know, where the core of you really exists. And I

guess because I've been in public life for so long and politics, I used to think the core of me was in politics. . . It isn't.

There is something good that comes out of this. A lot of good things come out of it. I think I understand myself a lot better. I think I understand what's important to me better. Maybe I'm not completely there yet. I would be foolish to think that I was in a few weeks. But I think I'm heading in that direction.

In contrast to prostate cancer, another hormone-related cancer of the male genital tract - that of the testicle - has been a success story of medical and surgical oncology. Whereas this rare disease used to be the third leading cause of cancer death among young men, it is no longer even in the top five. The cure rate with early diagnosis is now over 90 per cent. As the remarkable story of the quadruple Tour de France champion, Lance Armstrong, demonstrates, even men with advanced metastatic disease have hope of full recovery with a judicious combination of surgery, radiation or chemotherapy and determination.

When I was working in palliative care, an oncologist at the British Columbia Cancer Agency asked me to speak with Francis, a thirty-six- year-old with cancer of the testicle not because he needed palliation, but because he didn't. Although the tumor had spread to his abdomen by the time Francis was diagnosed, with appropriate treatment he still had a better than fifty-fifty chance of a complete cure. The problem was that he was refusing all medical intervention. The oncologist hoped that my counseling skills might help to reverse his patient's negative attitude.

The medical statistics promising cure - or, at least, prolonged life - did not interest Francis. He based his refusal on religious grounds, arguing that since God sent him this disease, it would be impious of him to resist it. He said he was not afraid of treatment he simply felt it was wrong to even consider it. I tried to approach his obstinate denial of life from every angle that came to mind. Was it some childhood guilt that he felt merited punishment? It was evident that personally Francis was isolated in life, with no family or close ones. Was he depressed? Was this a form of medical suicide?

I asked, non-believer as I was, whether perhaps it was blasphemous in him to claim to know God's will. If God, indeed, had sent him the cancer, could He not have intended it as a challenge for Francis to overcome and learn from? Further, if God was the source of the illness, was He not finally also the source of the medical knowledge that made a cure highly probable?

I asked all these questions, but mostly I just listened to Francis. What I heard was the voice of a very confused and lonely man who was adamant in his refusal to save his life. He stuck firmly to what he felt were unshakeable religious principles, despite the express disagreement about his ideas from the elders of his church. They told him that his interpretation of their denomination's teaching was wayward and unjustified. They offered to support him through treatment and convalescence, all to no avail.

Francis is one of three or four men I have ever seen with cancer of the testicle. Although the incidence of this malignancy is rising, in the United States there are only about six thousand new cases each year, in Canada about one-tenth that number. There have been no studies of the emotional or personal histories of the men who develop it, only of the psychological consequences. There are remarkable similarities between what little I did learn of Francis's life, the published autobiography of Lance Armstrong and the experiences of Roy, a young man I knew well, whom I interviewed for this chapter.

Armstrong first noticed a slight swelling of his testicle in the winter of 1996 and began to feel uncharacteristically short of breath next spring. His nipples felt sore, and he had to drop out of the 1997 Tour de France owing to a cough and low-back pain. "Athletes, especially cyclists, are in the business of denial," Lance Armstrong writes. [19] It wasn't until September, when he coughed blood and his testicle became painfully enlarged, that he finally sought medical attention. By then the cancer had spread to his lungs and brain.

When it comes to cancer of the testicle, it is not only cyclists who are in the business of denial. Thirty-year-old Roy first felt the swelling in his left testicle in mid-2000 but put off going to his family doctor for another eight months. In the meantime, he told no one. "I was a little embarrassed and secondly I was afraid of getting bad news," he says. According to a British study, such reluctance to get help is not un typical with this disease: "Delayed

diagnosis is common, but is more often due to delay in seeking medical advice than to delay in the correct diagnosis being made by the physician. . . The maximum period of delay between symptoms and orchidectomy was three years, with a ... mean delay of 3.9[20] months' ...

It may be that young men are simply loath to accept that there is anything wrong with them, particularly with their sexual organs. But logic would suggest the opposite: if masculinity were the issue, young men would likely run for help as soon as they noticed an abnormality with their testes just as they do, for example, when they notice their hair thinning owing to familial baldness. Certainly when we look at Roy's life and at the autobiography of Lance Armstrong, we see deeper motives for the denial of their disease.

I have known Roy and his family since he was eight. I was their doctor for twenty years, until I left my practice in 2000. I discovered that Roy had been treated for testicular cancer when I dropped in for a quick visit to my old office a few months ago. By happenstance it was the same afternoon Roy was there for a checkup. By then I had already read Lance Armstrong's book, *It's Not about the Bike: My Journey Back to Life.* The parallels in the lives of Roy and Lance were eerie. Perhaps the similarities in their response to disease were more than coincidental.

Long before his cancer, Armstrong had developed a pattern of emotional repression. One of his close friends described him as "kind of like an iceberg. There's a peak, but there is so much more below the surface."

Armstrong never knew his biological father, whom he contemptuously dismisses as his "DNA donor." His mother, Linda Mooneyham, the daughter of divorced parents, was seventeen and abandoned when she gave birth to Lance, her first son. Linda's father, an alcoholic Vietnam veteran, gave up drinking, to his credit, the day his grandson was born.

Linda was a spirited and independent-minded young woman but, given her circumstances, also a very needy one, hardly an adult. As Lance was to write, "In a way, we grew up together." When Lance was three, Linda remarried. The stepfather, Terry Armstrong, is described by Lance as "a small man with a large mustache and a habit of acting more

successful than he really was." He professed Christian principles but, despite them, beat Lance regularly: "The paddle was his preferred method of discipline. If I came home late, out would come the paddle. Whack. If I smarted off: I got the paddle. Whack. It didn't hurt just physically, but also emotionally. So I didn't like Terry Armstrong. I thought he was an angry testosterone geek, and as a result, my early impression of organized religion was that it was for hypocrites."

As the adolescent Lance was to learn, his stepfather also engaged in extramarital affairs. "I could have dealt with Terry Armstrong's paddle. But there was something else I couldn't deal with," writes Lance, referring to his stepfather's infidelities. The marriage broke up.

Roy is also the first-born, the child of an ill-tempered and violent man who used to beat his wife and his son. "I remember one thing that my dad did. He tied my wrists and tied my ankles and put me out in the backyard. I don't remember how long he left me out there, but what really bothered me was the that guy who lived upstairs was looking out the window at me and laughing at me. How the fuck can you do that to a kid? Obviously it bothers me to this day."

"Was your mom around?"

"I think my mom was at work." Roy looked upon his mother as his ally. Very early he took on the role of defending her against her husband's violence.

Lance Armstrong's mother was also unable to protect her son from being beaten. It is inevitable that a child in that situation would have deep hurt around that failure - and anger not only at the abusive stepfather but also at the mother who could not keep him safe. Lance seems unaware of any such feelings - and that is the source, I believe, of his propensity to deny and ignore his pain. "If it was a suffer-fest," Lance writes about his teenage attraction to endurance sports, "I was good at it."

As indicated in the passage quoted above, he had greater difficulty enduring his mother's betrayal by her husband than his own harsh treatment.

The child of an unhappy mother will try to take care of her by suppressing his distress so as not to burden her further. His role is to be self-sufficient and not "needy" recall my reflexive suppression of a limp after minor knee surgery. When twenty-five-year-old Lance was given his cancer diagnosis, he was quite unable to tell his mother directly." I wasn't strong enough to break it to my mother that I was sick," he writes. He accepted the offer of a close friend to inform her on his behalf.

Linda rose to the challenge with great strength, love and courage, supporting Lance through the nightmare of a highly uncertain prognosis, the bewildering difficulties of making the appropriate treatment decisions and the travails of brain surgery and chemotherapy. Her son's automatic reflex to protect her was rooted not in their adult realities but in the childhood experiences that had programmed his coping style.

The result of Roy's childhood relationship with his parents, he says, was that "in the past I've always seemed to put other people's happiness before my own. My self-esteem was very low, so I thought socially that if I made others happy, then they would accept me. I'd try to satisfy them, doing what I thought they would want me to do."

"How would you do that?"

"By not being honest with myself or others. Always going along with what they wanted to do, or not being honest with them if they said something that hurt. I would just let that go.

"A few years ago I had a business with two partners. As far as I was concerned, we were all equal, but it seemed like the way they were running the show, it was all them. They were in charge. My opinion didn't matter. Things like that hurt, but I just suppressed it and kept it in and didn't say anything. 1 didn't know how to deal with it."

The crucial difference, I believe, between Lance Armstrong and Roy on the one hand, and Francis on the other, is that the first two had had enough love in their lives to hold on to the part of themselves that allowed for the development of a fighting spirit. Unlike Francis, they also both received powerful caring and support from family and friends when they were diagnosed.

I strongly suspect that repression plays a role in the onset of testicular malignancy. It would be worthwhile for someone to undertake a study in which men with the disease were carefully interviewed about how they experienced their lives emotionally. One aspect deserving attention would be the patients' level of closeness to and identification with their mothers. There is - I don't believe coincidentally - an uncanny resemblance in looks between Lance's mother, and his wife, Kik. In a photograph of the three of them in Armstrong's riveting memoir, one can hardly tell the two women apart.

One of the lessons Roy spontaneously drew from his experience of cancer was to refuse to orient his behaviors any longer to pleasing others without considering the cost to himself. "Whatever I do now, it is definitely not to please anyone else," he says. "What is going to make me happy? Is this what I want to do? I've tried it the other way in the past, and it didn't work out for me. "

Francis was admitted to palliative care, in the end. The cancer eventually spread to his liver, causing a painful distension of that organ. He died quite soon, sooner than we doctors had anticipated.

Chapter 8 References

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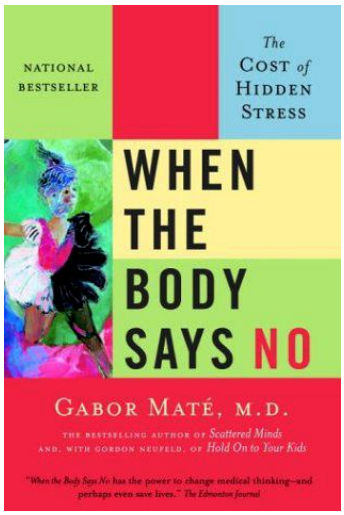
CHAPTER 9: IS THERE A CANCER PERSONALITY?

Source: *When the Body Says No* by Dr. Gabor Mate



IT WAS LATE AUTUMN OF 1990 when Jimmy married Linda. The wedding took place in the chapel of Vancouver Hospital's palliative care unit, five days before he died of the skin cancer that had invaded his spine. The bride was eight months pregnant. Except for his father, all Jimmy's family had gathered to witness the ceremony and to be with him in his final weeks. A month and a day after I pronounced Jimmy's death, I attended the birth of their daughter, Estelle, just as I had helped deliver Linda's two older children from her first marriage. Jimmy wasn't much for doctors. Although he and Linda had been together five years, I had met him only that summer when he visited the office with persistent back pain. It turned out to be the sign of spinal metastases from a skin cancer that had been excised from his leg some years before.

The original condition, malignant melanoma, is a life-endangering tumor of melanocytes, the pigmented cells in the skin. A deadly disease with a ready tendency to spread to other organs, melanoma often strikes people in the prime of life. I did not get to know Jimmy very well, but from our first meeting he impressed me as extraordinarily likeable. He was thirty-one years old, polite and friendly, with sandy-colored light brown hair, blue eyes, a complexion sprinkled with freckles and a broad, Irish, open-faced look about him. The exposure of fair-skinned individuals to ultraviolet radiation is the major physical risk factor for malignant melanoma. People of Celtic origin appear to be especially vulnerable, particularly if, like Jimmy, they have light-colored hair, freckles and blue or grey eyes. Dark-skinned ethnic groups are at little risk for skin cancer-in Hawaii, skin cancer is forty-five times less common among non-Caucasians than in Caucasians.'

Local dermatologists conduct a "sunscreen patrol" on the beaches of Vancouver in the summertime as a public service, warning sunbathers of the danger they are courting. It is unfortunate that repression is not as easily remediable a problem as inadequate sunscreen. Malignant melanoma has been the subject of some of the most persuasive research evidence linking repression and the development of cancer.

Jimmy's condition deteriorated very quickly, and the chemotherapy and radiation made him feel worse. "I've had enough," he finally said. "This is crazy. I'm dying, and I don't need to be

dying as sick as I am" Soon after that, his legs became paralyzed, triggering his admission to palliative care. Death followed within a few weeks. Until I left my practice two years ago, Linda and her children remained my patients. When I called her recently, she agreed to be interviewed for this book, as did Donna, Jimmy's older sister.

I asked Linda to describe her late husband's personality. "Jimmy was easygoing, laid-back and relaxed. He loved to be around people. I had to think when you asked me about what kind of stresses there were in his life. He wasn't a very stressed out kind of person. Now, he was a drinker. He had to drink pretty much every day. That's why I wouldn't marry him all those years, because of the drinking. He had beer every day-at least four or more."

"Did it change him at all?"

"Only if he had a lot more than that.... Then he became this very big, lovable bear who wanted to tell everybody how much he loved them. When he drank, he just wanted to hug people. Guys, too, like they were his big brothers. He needed to say to a guy, `You're my buddy," and then he would cry.

"He wasn't a violent man, he wasn't angry or frustrated. He was sad. He had a lot of sadness in him, and I don't know why.

"There's only one thing I can think of, some secret that he had about his father that he didn't want to tell me. He couldn't talk about it. He did not talk about his emotions. He did not share anything, really."

"What kind of a childhood had he had?"

"He grew up in Halifax. He always said he was a happy kid. His parents stayed together. Both his parents were alcoholics-the father, from what I understand, drank a lot for a long time. I think the mother started when Jimmy was a teenager."

As I found out later from Jimmy's sister, Donna, his senior by two years, their father had been a heavy drinker throughout their childhood. Donna and I had two conversations. "I felt

very comfortable with my childhood," she told me at first. "My younger siblings have a different perspective ... but I believe we had a very good upbringing. Very happy household...

"Jimmy was a real little boy, a happy kid.We'd play all the time.We'd go out into the backyard and have water fights-you know, those little spray guns. I just see him as a kid with a real happy face"

"How do you recall your parents?"

"My father was the nicest, friendliest man around. He was a very funny man. He was always joking around with us, play-fighting with us, tickling. He used to mimic, used to talk like Donald Duck. People would come over and say, `Get your father to talk like a duck.'

"He was a comical person, but you had to listen to him. We'd joke around with him, but when Dad spoke, the ground shook.... When he was annoyed or angry, when enough was enough-that was it. If he told us to do something, you did it."

"Why?"

"Because if not, you'd be punished and yelled at."

Donna married and moved to a different town when she was nineteen. Jimmy stayed with his parents until the age of twenty-two. On what was to have been a brief trip to Vancouver, ostensibly to see a friend, he called to tell his parents he would not be back. He did not return, except for a rare visit.

"He just called and said he wasn't coming home. He left a letter in his top drawer, explaining it."

"He escaped."

"He did. And the reason why, I remember him saying to my parents, `Hey, I couldn't tell you, because I didn't want to hurt you.. "'

"So Jimmy had the feeling that it would hurt his parents for him to be an independent person."

"All of us were made to feel that way. For our mother, her children were her world. They were her everything. She tried to do the best she could, but she was very attached to us- even to nay detriment but especially to Jimmy's. In retrospect, I realize we were far too attached, to an unhealthy degree. I think at some point you have to let your children go. I think emotionally, she didn't let go. I felt obligated, and many times Jimmy did, too. Normally your parents would try to understand and accept your separateness as you got older."

"Jimmy's escape to the West Coast physically doesn't mean that he liberated himself internally."

"Of course he didn't, no. He felt terrible. He felt very, very had. He did it, but he also had to live with the feelings."

According to Donna, Jimmy found the burden of his parents' emotional pain unbearable even at the end of his life. "Just before the Labor Day weekend, my brother phoned me. He told me what was going on with the melanoma, but he said, `You know, Donna, I can't phone Mom and Dad, because emotionally I can't handle it. Could you do it for me?' I said sure, I'll do it. So he said, `Just make sure that they don't call me all upset and crying and everything, because I couldn't take it. "

I suggested to Donna that perhaps what she had recalled as Jimmy's childhood "real happy face" might not have been a genuine face at all. At least in part, it could have been a coping mechanism Jimmy adopted in reaction to his parents' anxieties and anger. It was a way of avoiding the painful impact of their emotions on himself. Soothing his parents' feelings was accomplished by negating his own.

Donna called me back a few days later. Our conversation had brought to the forefront many memories. She needed to talk. "

After you and I spoke, I just went on about my day. I went to bed at night. About four o'clock in the morning I woke up. It was just incredible how many things came out and just kept going through my mind.

"You had mentioned Linda saying that Jimmy had a lot of sadness in him, maybe to do with his dad. I knew Jimmy really, really well, and yes, there was a lot of sadness. I can go way back to the beginning, remembering when he was little. The only time I can recall my dad doing anything with my brother was a little bit of roughhousing on the carpet in the living room. And I see a bunch of smiles and laughs. But other than that, there was never any participation in Jimmy's life. Never went to the hockey games. Never played with him.

"The crazy thing is that our father always said that he loved us, but he could be so hurtful. I have a brother who is quite heavy, and he'd ridicule him in front of people. He'd say some terrible things to him. And to Jimmy, too.

"I was never angry with my father-I've always covered up for him, maybe knowingly, maybe in an unknowing way. That night, all of a sudden, I got so angry. I started to think of Jimmy and all the things that happened as he was growing up and throughout his life. I kept thinking of all the times my father raised his voice. If he was trying to fix something and he didn't have the right tools, or the screws fell on the floor, or if something didn't happen exactly the way it was supposed to happen, he would scream and yell, and we were scared. We just fled. All of a sudden I remembered his voice and the screaming and the yelling, and I thought, This is not how you should live. This is not what we should have experienced.

"Even at the end ... My father came out to see Jimmy - they drove from Halifax. Actually, my sister and her husband did all the driving; my father drank all the way. They arrived a couple of weeks before Jimmy had to go into palliative care. My father walked into the apartment and sat there sipping his beer, not wanting even to go into the bedroom to see his son, to see Jim.

"We were trying to cover up. We didn't want Jimmy to realize that his father couldn't face seeing him - was afraid to see what he was going to look like. Finally, Dad built up enough courage and went into the room, and asked, `Jimmy, can I get you anything? Is there something that you want?'

"My father came out, went to the fridge, and all of a sudden he said, `How come there's no apple juice here? I don't believe this!' And he started ranting and raving at all of us in the apartment. We were stunned. Got his coat on and stomped off to the store and came back with apple juice for Jimmy.

"Then my father went home, and that was it. He never saw Jimmy in the hospital. He went back to Halifax and never saw him again. And the funny thing is, well ... you know Linda was pregnant with Estelle and they got married five days before Jimmy died.

"He was semi-comatose that day."

"Yes, he was drowsy. We'd had to increase his pain medication rapidly." "Well, one of the things I keep remembering is this.... After the wedding, he was weak, but he held his hand up and said, `Look, look, *just like Dad's ring*.' And his wedding band was identical to my father's. It's funny, those were the words that came out of Jimmy's mouth. *Just like Dad's ring*."

Jimmy's mode of emotional coping has been extensively documented among melanoma patients. An elegant study in 1984 measured the physiological responses to stressful stimuli of three groups: melanoma patients, people with heart disease and a control cohort with no medical illness. Each person was connected to a dermograph, a device that recorded the body's electrical reactions in the skin as the subject looked at a series of slides designed to elicit psychological distress. The slides displayed statements of an insulting, unpleasant or depressing nature, such as "You're ugly," or "You have only yourself to blame." As their physiological responses were being registered, the participants were asked to record their subjective awareness of how calm or disturbed they felt on reading each statement. The researchers thus secured a printout of the actual level of distress experienced by the

nervous system of each subject and simultaneously a report of the subjects' conscious perception of emotional stress.

The physiological responses of the three groups were identical, but the melanoma group proved most likely to deny any awareness of being anxious or of being upset by the messages on the slides. **"This study found that patients with malignant melanoma displayed coping reactions and tendencies that could be described as indicating `repressiveness: These reactions were significantly different from patients with cardiovascular disease, who could be said to manifest the opposite pattern of coping. 12**

The melanoma group was the most repressed among the three groups; the cardiac patients appeared to be the least inhibited. (It is not, as it may seem, that the reactivity of the cardiac patients is healthy. In between repression and hyper-reactiveness is a healthy median.) This study demonstrated that people can experience emotional stresses with measurable physical effects on their systems-while managing to sequester their feelings in a place completely beyond conscious awareness.

It was in relationship to melanoma that the notion of a "Type C" personality was first proposed, a combination of character traits more likely to be found in those who develop cancer than in people who remain free of it.Type A individuals are seen as "angry, tense, fast, aggressive, in control"-and more prone to heart disease. Type B represents the balanced, moderate human being who can feel and express emotion without being driven and without losing himself in uncontrolled emotional outbreaks. Type C personalities have been described as "extremely cooperative, patient, passive, lacking assertiveness and accepting.... The Type C individual may resemble Type B, since both may appear easygoing and pleasant, but ... while the Type B easily expresses anger, fear, sadness and other emotions, the Type C individual, in our view, suppresses or represses `negative' emotions, particularly anger, while struggling to maintain a strong and happy facade."'

Could it be disease itself that changes someone's personality, affecting his coping style in a way that may not reflect how he had functioned in life before the onset of illness? Jimmy's

story, related by his wife and sister, illustrates that repression, "niceness" and lack of aggression are lifelong patterns, having their origins in early childhood. As the researchers who studied physiological stress responses in melanoma patients noted, "When people are diagnosed with a disease-whether cancer or cardiovascular-they do not precipitously change their usual ways of coping with stress or suddenly develop new patterns.... Under stress, people usually mobilize their existing resources and defenses"

How do psychological stresses translate into malignant skin lesions? Hormonal factors likely account for the fact that the number of melanoma tumors is increasing in bodily sites not exposed to sunlight. Researchers have suggested that hormones may be over stimulating the pigment-producing cells.[4]

The Type C personality traits associated with melanoma have been found in studies of many other cancers as well. In 1991 researchers in Melbourne, Australia, investigated whether any personality traits were a risk factor in cancer of the colon or the rectum. Over six hundred people, newly diagnosed, were compared with a matched group of controls.

Cancer patients, to a statistically significant degree, were more likely to demonstrate the following traits: "the elements of denial and repression of anger and of other negative emotions . . . the external appearance of a `nice' or `good' person, a suppression of reactions which may offend others, and the avoidance of conflict....

The risk of colorectal cancer with respect to this model was independent of the previously found risk factors of diet, beer intake, and family history."' Self-reported childhood or adult unhappiness was also more common among the bowel cancer cases. **We have already noted similar traits among patients with breast cancer, melanoma, prostate cancer, leukemias and lymphomas, and lung cancer.**

In 1946 researchers at Johns Hopkins University began a long-term prospective study to establish whether there are psychobiological characteristics in young people that could help predict susceptibility to future disease states. In the course of the subsequent eighteen

years, 1,130 white male students enrolled in medical school underwent psychological testing. They were questioned regarding their emotional coping styles and childhood relationships with parents. Biological data-pulse, blood pressure, weight and cholesterol levels-were also recorded, as were habits such as smoking, coffee drinking and alcohol intake. At study's end, nearly all the subjects had graduated and most were doctors, their ages ranging from thirty to over sixty. At this point, their health status was reviewed; the majority were healthy, but in about equal numbers some had developed heart disease, high blood pressure, mental illness, cancer or had committed suicide.

When the researchers conceived of the project, they had not expected to find that cancer would be associated with any pre-existing psychological factors. However, their data showed just such a connection. There were striking similarities between those who had been diagnosed with cancer and the suicide group: "Our results appear to agree with findings that cancer patients `tend to deny and repress conflictual impulses and emotions to a higher degree than do other people." 6

The researchers found that both for the healthy majority and for each disease category there was a distinctive set of psychological traits. **The lowest scores for depression, anxiety and anger had been originally recorded for the medical students who later developed cancer.**

They had also reported being the most distant from their parents.

Of all the groups, the cancer subjects were the least able to express emotion.

Does that mean there is a "cancer personality"? The answer is neither a simple yes nor a no.

Melanoma illustrates the futility of simplistic reductions to a single origin. Fair skin alone cannot be the cause of this cancer, since not everyone with fair skin will develop melanoma. Ultraviolet damage to the skin by itself cannot be sufficient, since only a minority of light-complexioned persons who suffer sunburns will end up with skin cancer. Emotional

repression by itself also cannot account for all cases of malignant melanoma, since not all people who are emotionally repressed will develop either melanoma or any other cancer. A combination of these three circumstances is potentially lethal.

While we cannot say that any personality type causes cancer, certain personality features definitely increase the risk because they are more likely to generate physiological stress. Repression, the inability to say no and a lack of awareness of one's anger make it much more likely that a person will find herself in situations where her emotions are unexpressed, her needs are ignored and her gentleness is exploited. Those situations are stress inducing, whether or not the person is conscious of being stressed. Repeated and multiplied over the years, they have the potential of harming homeostasis and the immune system. It is stress-not personality per se-that undermines a body's physiological balance and immune defenses, predisposing to disease or reducing the resistance to it.

Physiological stress, then, is the link between personality traits and disease. Certain traits-otherwise known as coping styles-magnify the risk for illness by increasing the likelihood of chronic stress. **Common to them all is a diminished capacity for emotional communication. Emotional experiences are translated into potentially damaging biological events when human beings are prevented from learning how to express their feelings effectively. That learning occurs-or fails to occur-during childhood.**

The way people grow up shapes their relationship with their own bodies and psyches. The emotional contexts of childhood interact with inborn temperament to give rise to personality traits. Much of what we call personality is not a fixed set of traits, only coping mechanisms a person acquired in childhood. There is an important distinction between an inherent *characteristic*, rooted in an individual without regard to his environment, and *a response to the environment*, a pattern of behaviors developed to ensure survival.

What we see as indelible traits may be no more than habitual defensive techniques, unconsciously adopted. People often identify with these habituated patterns, believing them to be an indispensable part of the self. They may even harbor self-loathing for certain traits-for example, when a person describes herself as "a control freak." In reality, there is no innate human inclination to be controlling. What there is in a "controlling" personality is deep anxiety. The infant and child who perceives that his needs are unmet may develop an obsessive coping style, anxious about each detail. When such a person fears that he is unable to control events, he experiences great stress. Unconsciously he believes that only by controlling every aspect of his life and environment will he be able to ensure the satisfaction of his needs. As he grows older, others will resent him and he will come to dislike himself for what was originally a desperate response to emotional deprivation. The drive to control is not an innate trait but a coping style.

Emotional repression is also a coping style rather than a personality trait set in stone. Not one of the many adults interviewed for this book could answer in the affirmative when asked the following: When, as a child, you felt sad, upset or angry, was there anyone you could talk to-even when he or she was the one who had triggered your negative emotions? In a quarter century of clinical practice, including a decade of palliative work, I have never heard anyone with cancer or with any chronic illness or condition say yes to that question. Many children are conditioned in this manner not because of any intended harm or abuse, but because the parents themselves are too threatened by the anxiety, anger or sadness they sense in their child-or are simply too busy or too harassed themselves to pay attention. "My mother or father needed me to be happy" is the simple formula that trained many a child - later a stressed and depressed or physically ill adult - into lifelong patterns of repression.

Jill, a Chicago filmmaker diagnosed with advanced ovarian cancer, admits to being a perfectionist. A friend of hers told me that she had felt concern during the year prior to the diagnosis as she watched Jill endure a stressful experience. "I felt at the time that this is going to be more than psychologically damaging," the friend said.

"About three years ago Jill got into a collaboration on a video. The production company didn't do a great job. It became a horrendous nightmare for her, because her expectations

were that she had to come through on a project. Once she's agreed to it, it has to be very high quality. She spent three or five times as much time as she was compensated for. That was, I believe, a big trigger for Jill's body to say, I can't stand this."

My interview with Jill herself was illuminating for It's combination of disarming honesty and psychological denial. Jill told revealing stories of stresses in her relationships with her parents and her spouse, without for a moment accepting that these may have contributed to the onset of her illness. She is fifty, highly articulate, with a tendency to go into a labyrinth of details on every topic. I sensed that was her way of keeping anxiety at bay. She appeared uncomfortable with even brief silences in the conversation. At our first meeting, Jill was still wearing a wig, having lost her hair because of chemotherapy.

She had adopted a mothering role in her marriage. When her husband, Chris, suffered an acute but debilitating illness, she cared for him with maternal concern and devotion, calling the doctors, nursing him at nights, ensuring that he was well looked after while she was at work. All this time she was preparing a presentation she was about to give at a national conference and conducted an evening study group for aspiring filmmakers. She led such a group the night before she left for the conference, packing at two in the morning and catching an early flight.

It was shortly after her stint of caring for her husband that she experienced the first symptoms of ovarian cancer. The contrast in caretaking by husband and wife was dramatic. Chris made no medical inquiries on her behalf over several months, seemingly oblivious to her pain and weight loss, despite that fact that she was "living on Advils." "Strangers in elevators would ask me if I was well," she says. As often happens with ovarian cancer, doctors took several months to arrive at the diagnosis.

The first thing Jill said when informed she had ovarian cancer was " 'My poor husband and my poor mother.' I am a pillar of strength for them. I felt sorry for them, because they would lose that support."

The gynecological oncologist explained to the couple that the prognosis for survival past five years was poor, given the stage at which Jill's disease had been diagnosed. Chris was in

denial. "He didn't seem to have heard that," Jill says. "I needed to talk about what I just been told, but in the car on the way home Chris just kept saying how we're going to fight and beat this. He actually didn't remember what the specialist had said about the prognosis, not even afterwards. It completely bypassed him."

As she faced her surgery, Jill had to deal with her mother's decision to stay with her. "She was not going to come. She's really used to being the centre of attention, and she doesn't like flying. But everyone was saying to her, 'Your daughter is going into hospital, and you're not going to be there?' So in response to that she had to be a mother and really come."

"If that's how you saw it, how did you feel about her coming?"

"At the very beginning I was happy that she wasn't coming. I didn't want her. I knew she was using me to be a good mother, but I've always taken care of my mother since my dad died-he had asked me to."

"My guess is that you've taken care of her since you were born."

"Okay, since I was born. My dad used to say to me, you know, leave her be. He was so very protective, exasperated with her, but he really loved her in some twisted way. He also had a great understanding of her limitations, and at his own expense he accommodated her as much as he could.

"Once my father picked me up at the airport as I came back from a major work trip to Southeast Asia. I was exhausted. My mother was a teacher, and Dad wanted to drive me to her school. 'So you can say hello to your mom she's waiting there with all her pupils,' he said. I said, 'No Dad, I don't want to go. I'm very tired. I've had an emotionally draining trip. I just want to go and be by myself." Do this for your mom. You know she is really looking forward to this.' He actually drove me there, and she was waiting with all the kids, and he made me put this rice paddy hat on that I'd bought so that I would entertain them. She was doted on like this all her life-and he knew that she needed to be honored that way. She could show the kids her daughter had been away, and now she was back to see her. I played that role to please my dad, and it happened all the time."

"Wouldn't you encourage your children to assert themselves, not be drawn into taking care of somebody in that sense? Jill, you've got this serious disease, this major operation coming up, and your mother not only comes, she stays with you a whole month."

"And she's very demanding. For a whole month I was catering to her. You know, it's true, I'm very dutiful, I am really very dutiful. I take care of her. I went through it and talked about it with my friends, and a lot of them said not to let her come.

"It went through my head many times, If one of my kids were having surgery, and if they didn't want me to come, I would accept it. However, I would hope that they would feel comfortable that I would be there. With my mother, if I was going to feel guilty and miserable because I didn't provide for her also, that would have been a greater stress for me."

Jill's recollection of her childhood is not that she was a compliant child but that she was rebellious. "I wasn't such a good kid as an adolescent. My father said that he would never wish that I would have a kid like me. I was quite a handful for them. As a teenager, I was considered very difficult. I did well at university, but I just didn't like school. Then I got married somebody professional. So I turned out good for my parents, after all."

Jill's mother died last year, since our interview. Her daughter felt a need to look after her even in death. The obituary she wrote eulogized her mother for having travelled a long distance to be with her and to nurse her after her surgery for ovarian cancer.

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MEN'S HEALTH

Source: *Alternative Medicine: The Definitive Guide* (782-795)

It is estimated that 10-30 million men in the U.S. are experiencing some form of impotence, of which only about 200,000 per year seek medical help. – **Tom Kruzel, N.D.**

STATISTICALLY, MEN experience higher rates of heart disease, cancer, chronic lung disorders, liver diseases, and diabetes than women, all of which contribute to an average life expectancy seven years less than women. These conditions, however, are not unique to In men. Therefore, when a person thinks of men's health, the focus is primarily on disorders of their genitourinary organs.

Because the genitourinary tracts of men and women are distinct, each requires its own specific set of health requirements.

Women's health issues have been well publicized and discussed with relative openness. Men's health issues, on the other hand, are just beginning to be seen as equally important. This is partly due to men in general, who traditionally have been less likely to seek medical attention than women, especially for minor problems, which often serve as warning signs for more serious underlying illness. The result is higher mortality rates resulting from the advanced state of the illness when it is eventually discovered. Social and economic pressures, as well as a lack of understanding of what constitutes normal function, also contribute to men's ambivalence in issues of health.

Types of Men's Health Disorders

The health of the genitourinary organs reflects a man's overall well-being, according to Dana Ullman, M.P.H., Director of Homeopathic Educational Services, in Berkeley, California. Individual sexual habits can also influence a person's state of health, just as the health of the body and the reproductive system can be a determining factor for sexual habits and sexual drive. The most common men's health concerns include **impotence**, **benign prostatic hypertrophy (BPH)**, **prostatitis**, and **prostate cancer**.

Impotence

Impotence is defined as the inability to sustain a satisfactory erection to perform intercourse and ejaculation. "It is estimated that 10-30 million men in the United States are experiencing some form of impotence, of which only about 200,000 per year seek medical help," states Tom Kruzel, N.D., of Scottsdale, Arizona. Although impotence has long been associated with aging, Dr. Kruzel points out that growing older is not necessarily an inevitable cause. "Rather, the amount and force of ejaculation decreases, while the recovery period between ejaculations becomes longer," he says. "This varies from person to person, depending on their overall health and vitality."

There are two forms of impotence, primary and secondary. "**Primary impotence** is rare and is almost always associated with severe psychopathology," says Dr. Kruzel. "This may include unreasonable fear of engaging in intercourse, fear of intimacy, or extreme feelings of guilt or anxiety about the act of intercourse. Low male sex hormone levels can also be a

cause." In cases of primary impotence, men are unable to engage in sexual intercourse.

Secondary impotence is the more common type of impotence and Dr. Kruzel estimates that it is related to psychological causes about 80% of the time. "Men who suffer from secondary impotence are only able to engage in intercourse 25% of the time," he says. "This is most often situational in nature, as the person may have become bored with the relationship or the place and time may be less than optimal. Emotional factors such as low self-esteem, immaturity, performance anxiety, and depression can also playa role."

> **By age 50, about 30% of men will start to experience difficulties with urination related to enlargement of the prostate gland.** – Tom Kruzel, M.D.

One way of evaluating the cause of impotence is to evaluate whether or not an erection occurs while sleeping, which generally happens during REM (rapid eye movement) sleep. The usual method of doing this is called the "stamp test," which involves placing a ring of paper around the penis prior to bedtime. If the ring has been disrupted in the morning, the man has experienced an erection at some time during the night. This indicates that the cause of impotence is most likely due to psychological factors rather than to physical causes.

Illness, such as diabetes and heart disease, can contribute to impotence, according to Dr. Kruzel, as can certain endocrine disorders such as hypothyroidism and hypopituitarism. Vascular diseases can playa role due to their effects on the vessels of the penis. Neurological disorders from injury or brain disease (Parkinson's disease or multiple sclerosis) have also been found to be causes, as have the various surgeries of the genitalia. Medications (antihypertensives, antidepressants, and psychotropics) may cause impotence, especially in elderly men taking multiple prescription drugs. Other causes of impotence include low levels of testosterone, zinc deficiency, high cholesterol, and alcohol or drug addiction. [1]

See Addictions, Diabetes, Heart Disease, Hypertension, Multiple Sclerosis

Benign Prostatic Hypertrophy (BPH)

According to Dr. Kruzel, by the age of 50, about 30% of men will start to experience difficulties with urination related to enlargement of the prostate gland or benign prostatic hypertrophy (BPH). **Increases in the number of times a man has to urinate**, along with a frequent sensation of having to urinate (especially at night), are among some of the early signs. In addition, **a reduction in the force and caliber of urination** is also characteristic of prostatic enlargement. Instability of the detrusor muscle (the outer muscle layer of the bladder) can even result in urinary incontinence. "This occurs in up to 3% of patients with BPH," Dr. Kruzel says. "These symptoms often lead to an increased sense of frustration and embarrassment, as well as the disruption of normal activities."

Enlargement of the prostate is usually caused by an abnormal overgrowth or swelling of the tissue of the prostate, which then blocks the urethra (opening from the bladder). Problems associated with this condition usually continue to worsen with age, increasing in incidence to about 50% of all men by the age of 60 and up to 80% past age 70.2 Most physicians consider this to be a normal consequence of aging.

The growth of prostate tissue that results in BPH occurs due to the hormone testosterone, which is produced by the testicles and the adrenal glands, according

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CHAPTER 9: IS THERE A CANCER PERSONALITY?

Source: *When the Body Says No* by Dr. Gabor Mate