COMMON CANCERS – BREAST, PROSTATE, AND LARGE BOWEL

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Cancer is a Fungus
Breast Cancer Awareness Month: File
Breast Cancer: File

Books:

Prostate Health in 90 Days
http://www.mercola.com/forms/prostate_health_90days.htm

The No-Dairy Breast Cancer Prevention Program **by Jane A. Plant, Ph.D.**

Winning the War on Cancer **by Dr. Mark Sircus**

Crazy Sexy Cancer **by Kris Carr**

Articles:

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People:

Dr. Simoncini (Cancer is a Fungus)
Dr. Mark Sircus
Dr. T. Colin Campbell, PhD

Kevin Gianni
Gabriel Cousens, MD
Dr. Thomas Lodi
Dr. Lorraine Day, MD
Suzanne Somers
Kris Carr

Integral Nutrition: **Plant-Based Diet: No Animal Proteins of Any Kind**
Prostate Health: See Prostate File

Conventional: **Chemotherapy, Radiation, Surgery**

Terms:

NOTE FROM DAVID RAINOSHEK, M.A.

Source: www.*JuiceFeasting.com* and www.RevolutionaryWebinars.com

Cancer is such a huge topic, and you are receiving excellent information and insight on this site about it… from Women's Health (Day 31) to Men's Health (Day 32) to The Seven Stages of Disease, and all the info about moving your Center of Gravity up the Spectrum of Diet to plant-based and beyond….

That being said, here are some of the top elements that you can bank on to apply to a cancer preventative and healing regimen. Of course… ask your medical professional before applying any of these in a cancer situation. ☺

- **Sodium Bicarbonate (Baking Soda)**
- **Magnesium Chloride**
- **Nascent iodine**
- **ALA (Alpha Lipoic Acid)**
- **THC (medical marijuana oil)**
- **Natural Vitamin C**
- **Selenium**
- **Probiotics**
- **Proteolytic Enzymes (serrapeptase)**
- **Spirulina**
- **Chlorella**

- **Glutathione**
- **Pure water**
- **Healing Clays**
- **Sunlight for natural vitamin D supplementation**
- **Vitamin D3 Supplementation**
- **Infrared Saunas or an infrared BioMat**

DO NOT UNDERESTIMATE ANY OF THESE!!! These are top cancer preventatives and healing agents after decades and decades of collaborated knowledge and experience in the field of preventative and healing oncology.

See *Winning the War on Cancer* and *Sodium Bicarbonate: Rich Man's Poor Man's Cancer Treatment* by Dr. Mark Sircus for an excellent look at all of these elements listed above.

Also familiarize yourself with *Cancer is a Fungus* by Dr. Simoncini.

Thank you. Enjoy the file. The main take-home from this file is that *processed dairy* (pasteurized, homogenized, hormone-injected, antibiotic-treated, corn-fed, pesticide-laden cow milk) is a major carcinogen in westernized societies worldwide.

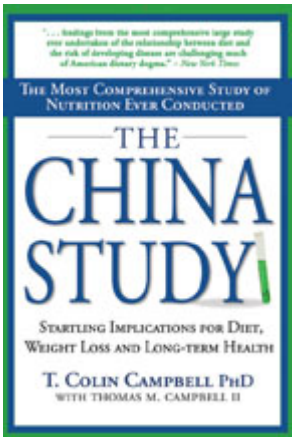
For more on my thoughts on the work of Dr. T. Colin Campbell in particular (including a critique of his work) see Day 37 of the Juice Feasting Program.

With love,

David Rainoshek, M.A.
July 5, 2012

COMMON CANCERS: BREAST, PROSTATE, LARGE BOWEL (COLON AND RECTAL)

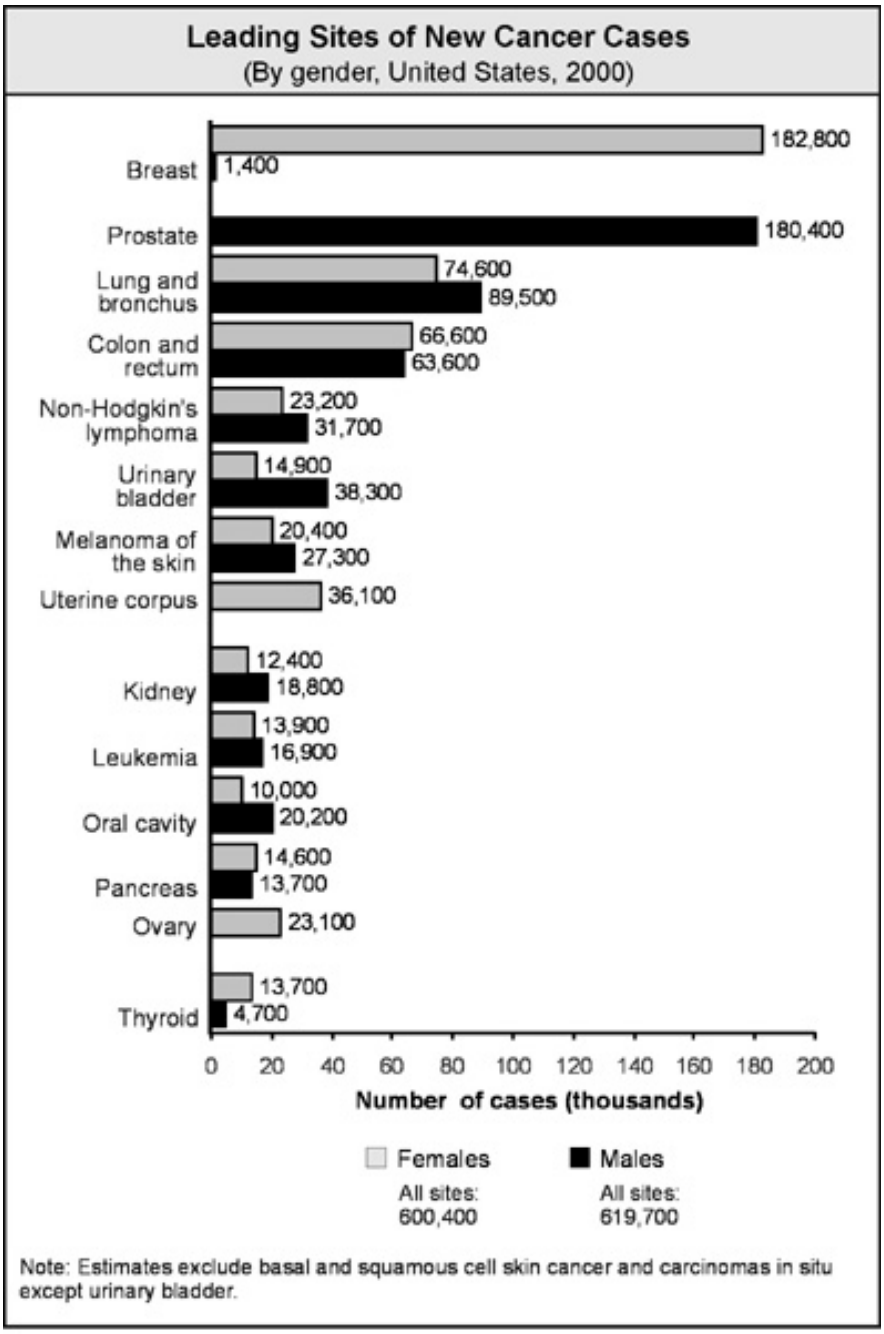
Source: *The China Study* by T. Colin Campbell (157-182)



MUCH OF MY CAREER HAS BEEN CONCENTRATED ON THE STUDY OF CANCER. My laboratory work was focused on several cancers, including those of the liver, breast and pancreas, and some of the most impressive data from China were related to cancer. For this lifetime work, the American Institute for Cancer Research kindly presented me with their Research Achievement award in 1998.

An exceptional number of books have summarized the evidence on the effects of nutrition on a variety of cancers, each with their own particularities. But what I've found is that the nutritional effects on the cancers I've chosen to discuss here are virtually the same for all cancers, regardless of whether they are initiated by different factors or are located in different parts of the body. Using this principle, I can limit my discussion to three cancers, which will allow me space in the rest of the book to address diseases other than cancer, demonstrating the breadth of evidence linking food to many health concerns.

I have chosen to comment on three cancers that affect hundreds of thousands of Americans and that generally represent other cancers as well: two reproductive cancers that get plenty of attention, breast and prostate, and one digestive cancer, large bowel-the second leading cause of cancer death, behind lung cancer.



Source: American Cancer Society. *Surveillance Research,* 2000.

BREAST CANCER

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

It was spring almost ten years ago. I was in my office at Cornell when I was told that a woman with a question regarding breast cancer was on the phone.

"I have a strong history of breast cancer in my family," the woman, Betty, said. "My mother and grandmother both died from the disease, and my forty-five-year-old sister was recently diagnosed with it. Given this family problem, I can't help but be afraid for my nine-year-old daughter. She's going

to start menstruating soon and I worry about her risks of getting breast cancer." Her fear was evident in her voice. "I've seen a lot of research showing that family history is important, and I'm afraid that it's inevitable that my daughter will get breast cancer. One of the options I've been thinking about is a mastectomy for my daughter, to remove both breasts. Do you have any advice?"

This woman was in an exceptionally difficult position. Does she let her daughter grow up into a deathtrap, or grow up without breasts? Although extreme, this question represents a variety of similar questions faced every day by thousands of women around the world.

These questions were especially encouraged by the early reports on the discovery of the breast cancer gene, BRCA-I. Headline articles in the *New York Times* and other newspapers and magazines trumpeted this discovery as an enormous advance. The hoopla surrounding BRCA-I, which now also includes BRCA-2, reinforced the idea that breast cancer was due to genetic misfortune. This caused great fear among people with a family history of breast cancer. It also generated excitement among scientists and pharmaceutical companies. The possibility was high that new technologies would be able to assess overall breast cancer risk in women by doing genetic testing; they hoped they might be able to manipulate this new gene in a way that would prevent or treat breast cancer. Journalists busily started translating selective bits of this information for the public, relying heavily on the genetic fatalistic attitude. No doubt this contributed to the concern of mothers like Betty.

"Well, let me first tell you that I am not a physician," I said. "I can't help you with diagnosis or treatment advice. That's for your physician to do. I can speak about the current research in a more general way, however, if that is of any help to you."

"Yes," she said, "that's what I wanted."

I told her a little bit about the China Study and about the important role of nutrition. I told her that just because a person has the gene for a disease does not mean that they are destined to get the cancer: prominent studies reported that only a tiny minority of cancers can be solely blamed on genes.

I was surprised at how little she knew about nutrition. She thought genetics was the only factor that determined risk. She didn't realize that food was an important factor in breast cancer as well.

We talked for twenty or thirty minutes, a brief time for such an important matter. By the end of the conversation I had the feeling that she was not satisfied with what I told her. Perhaps it was my conservative, scientific way of talking, or my reluctance to give her a recommendation. Maybe, I thought, she had already made up her mind to do the procedure.

She thanked me for my time and I wished her well. I remember thinking about how often I receive questions from people about specific health situations, and that this was one of the most unusual.

But Betty wasn't alone. One other woman also talked to me regarding the possibility of her young daughter undergoing surgery to remove both breasts. Other women who already had one breast removed wondered whether to have the second breast removed as a preventative measure.

It's clear that breast cancer is an important concern in our society. One out of eight American women will be diagnosed with this disease during their lifetimes-one of the highest rates in the world. Breast cancer grassroots organizations are widespread, strong, relatively well funded and exceptionally active

compared to other health activist organizations. This disease, perhaps more than any other, incites panic and fear in many women.

When I think back to that conversation I had with Betty, I now feel that I could have made a stronger statement about the role nutrition plays in breast cancer. I still would not have been able to give her clinical advice, but the information I now know might have been of more use to her.

So what would I tell her now?

RISK FACTORS

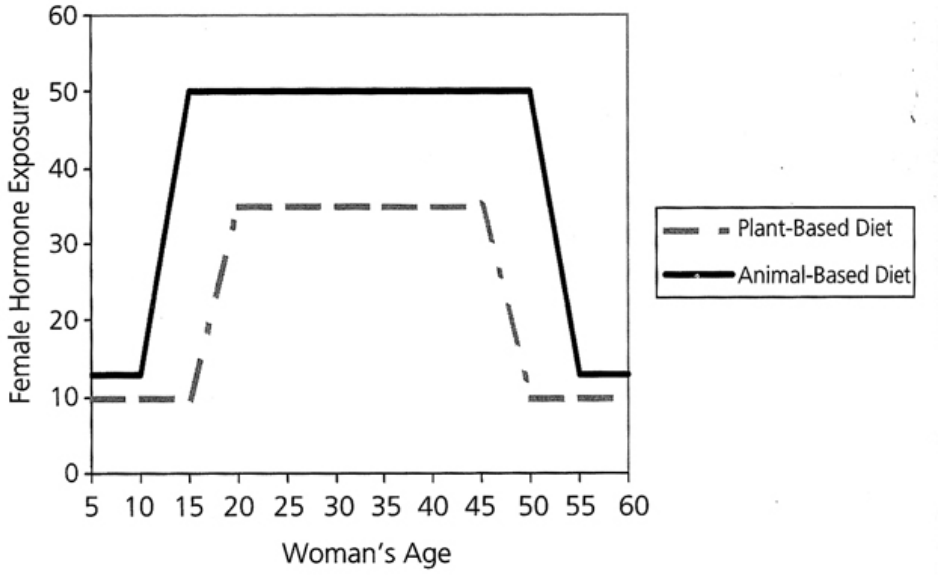
There are at least four important breast cancer risk factors that are affected by nutrition, as shown in Chart 8.1. Many of these relationships were confirmed in the China Study after being well established in other research.

CHART 8.1: BREAST CANCER RISK FACTORS AND NUTRITIONAL INFLUENCE

Risk of breast cancer increases when a woman has...	A diet high in animal foods and refined carbohydrates...
...early age of menarche (first menstruation)	...lowers the age of menarche
...late age of menopause	...raises the age of menopause
...high levels of female hormones in the blood	...increases female hormone levels
...high blood cholesterol	...increases blood cholesterol levels

With the exception of blood cholesterol, these risk factors are variations on the same theme: exposure to excess amounts of female hormones, including estrogen and progesterone, leads to an increased risk of breast cancer. Women who consume a diet rich in animal-based foods, with a reduced amount of whole, plant-based foods, reach puberty earlier and menopause later, thus extending their reproductive lives. They also have higher levels of female hormones throughout their lifespan, as shown in **Chart 8.2.**

CHART 8.2: DIETARY INFLUENCE ON FEMALE HORMONE EXPOSURE OVER A WOMAN'S LIFETIME (SCHEMATIC)



According to our China Study data, lifetime exposure to estrogen[1] is at least 2.5-3.0 times higher among Western women when compared with rural Chinese women.

This is a huge difference for such a critically important hormone.[2] To use the words of one of the leading breast cancer research groups in the world,[3] "there is overwhelming evidence that estrogen levels are a critical determinant of breast cancer risk."[4, 5] Estrogen directly participates in the cancer process.[5,7] It also tends to indicate the presence of other female hormones[8-12] that playa role in breast cancer risk.[5, 7] Increased levels of estrogen and related hormones are a result of the consumption of typical Western diets, high in fat and animal protein and low in dietary fiber.[3, 13-18]

The difference in estrogen levels between rural Chinese women and Western women[19] is all the more remarkable because a previous report[20] found that a mere 17% decrease in estrogen levels could account for a huge difference in breast cancer rates when comparing different countries. Imagine, then, what 26-63% lower blood estrogen levels and eight to nine fewer reproductive years of blood estrogen exposure could mean, as we found in the China Study.

This idea that breast cancer is centered on estrogen exposure[3, 21, 22] is profound because diet plays a major role in establishing estrogen exposure. This suggests that the risk of breast cancer is preventable if we eat foods that will keep estrogen levels under control. The sad truth is that most women simply are not aware of this evidence. If this information were properly reported by responsible and credible public health agencies, I suspect that many more young women might be taking very real, very effective steps to avoid this awful disease.

THE COMMON ISSUES

GENES

Understandably, women who are most afraid of this disease have a family history of breast cancer. Family history implies that genes do playa role in the development of breast cancer. But I hear too

many people say, in effect, that "it's all in the family" and deny that they can do anything to help themselves. **This fatalistic attitude removes a sense of personal responsibility for one's own health and profoundly limits available options.**

It is true that if you have a family history of breast cancer, you are at an increased risk of getting the disease.[23,24] However, one research group found that less than 3% of all breast cancer cases can be attributed to family history.[24] Even though other groups have estimated that a higher percentage of cases are due to family history,[25] the vast majority of breast cancer in American women is not due to family history or genes. But genetic fatalism continues to define the nation's mindset.

BRCA-1 and BRCA-2

Among the genes that influence breast cancer risk, BRCA-1 and BRCA-2 have received the most attention since their discovery in 1994.[26-29] These genes, when mutated, confer a higher risk both for breast and ovarian cancers.[30, 31] These mutated genes may be passed on from generation to generation; that is, they are inherited genes.

In the excitement over these discoveries, however, other information has been ignored. **First, only 0.2% of individuals in the general population (1 in 500) carry the mutated forms of these genes.**[25] Because of the rarity of these genetic aberrations, only a few percent of the breast cancer cases in the general population can be attributed to mutated BRCA-1 or BRCA-2 genes.[32,33] Second, these genes are not the only genes that participate in the development of this disease32; many more will surely be discovered. Third, the mere presence of BRCA-1, BRCA-2 or any other breast cancer gene does not guarantee disease occurrence. Environmental and dietary factors playa central role in determining whether these genes are expressed.

A recent paper[31] reviewed twenty-two studies that assessed the risk of breast (and ovarian) cancer among women who carried mutated BRCA-1 and BRCA-2 genes. Overall, disease risk was 65% for breast cancer and 39% for ovarian cancer by age seventy for BRCA-l women, and 45% and 11%, respectively, for BRCA-2 women. Women with these genes certainly face high risks for breast cancer. But even among these high-risk women, there is still good reason to believe that more attention to diet is likely to pay handsome rewards. *About half of the women who carry these rare, potent genes do not get breast cancer.*

In short, although the discovery of BRCA-1 and BRCA-2 added an important dimension to the breast cancer story, the excessive emphasis given to these particular genes and genetic causation in general is not warranted.

I do not mean to diminish the importance of knowing all there is to know about these genes for the small minority of women who carry them. But we need to remind ourselves that these genes need to be "expressed" in order for them to participate in disease formation, and nutrition can affect this. We've already seen in chapter three how a diet high in animal-based protein has the potential to control genetic expression.

SCREENING AND NON-NUTRITIONAL PREVENTION

With all of this new information regarding genetic risk and family history, women are often encouraged to get screened for breast cancer. Screening is a reasonable step, especially for women who may have tested positive for the BRCA genes. But it's important to remember that doing a

mammography or getting a genetic test to see if you harbor BRCA genes does not constitute prevention of breast cancer.

Screening is merely an observation to see whether the disease has progressed to an observable state. Some studies[34-36] have found that groups of women who undergo frequent mammography have slightly lower mortality rates than groups of women who do not undergo frequent mammography. This implies that our cancer treatments are more likely to be successful if the cancer is found at an earlier stage. This is likely to be true, but there is some concern over the way statistics are used in this debate.

One of the statistics used to support early detection and the ensuing treatments is that once diagnosed with breast cancer, the likelihood of surviving for at least five years is higher than ever before.[37] What this really means is that with the aggressive campaign for regular screening, many women are discovering their breast cancer at an earlier stage of disease. When disease is discovered at an earlier stage it is less likely to lead to death within five years, *regardless of treatment*. ***As a consequence, we may have an improved five-year survival rate simply because women find out that they have breast cancer earlier in the disease progression, not because our treatments have improved over time.***[38]

Beyond the current screening methods, there are other non-nutritional options for prevention that have been promoted. They are especially of interest to women who have a high risk of breast cancer due to family history and/or to the presence of the BRCA genes. These options include taking a drug such as tamoxifen and/or mastectomy.

Tamoxifen is one of the most popular drugs taken to prevent breast cancer,[39,40] but the long-term benefits of this option are not clear. One major U.S. study showed that tamoxifen administered over a period of four years to women at increased risk of breast cancer reduced the number of cases by an impressive 49%.[41] This benefit, however, may be limited to women whose estrogen levels are very high. It was this result that led the U.S. Food and Drug Administration to approve use of tamoxifen by women who met certain criteria.[42] Other studies suggest that the enthusiasm for this drug is not warranted. Two less substantial European trials[43, 44] have failed to show any statistically significant tamoxifen benefit, raising some doubt about how dramatic the benefit really is. Moreover, there is the additional concern that tamoxifen raises the risks for stroke, uterine cancer, cataracts, deep vein thrombosis and pulmonary embolism, although the overall benefits of breast cancer prevention are still believed to outweigh the risks.[42] Other chemicals have also been investigated as alternatives to tamoxifen, but these drugs are encumbered by limited effectiveness and/or some of the same troublesome side effects.[45,46]

Drugs such as tamoxifen and its newer analogues are considered *anti-estrogen* drugs. In effect, they work by reducing the activity of estrogen, which is known to be associated with elevated breast cancer risk.[4, 5] **My question is quite simple: why don't we ask why estrogen is so high in the first place, and once we recognize its nutritional origin, why don't we then correct that cause?** We now have enough information to show that a diet low in animal-based protein, low in fat and high in whole plant foods will reduce estrogen levels. Instead of suggesting dietary change as a solution, we spend hundreds of millions of dollars developing and publicizing a drug that mayor may not work and that almost certainly will have unintended side effects.

The ability of dietary factors to control female hormone levels has long been known in the research

community, but a recent study was particularly impressive.[47] **Several female hormones, which increase with the onset of puberty, were lowered by 20-30% (even 50% lower levels for progesterone!) simply by having girls eight to ten years of age consume a modestly low-fat, low animal-based food diet for seven years.**[47] These results are extraordinary because they were obtained with a modest dietary change and were produced during a critical time of a young girl's life, when the first seeds of breast cancer were being sowed. These girls consumed a diet of no more than 28% fat and less than 150 mg cholesterol/day: a moderate plant-based diet. I believe that had they consumed a diet devoid of animal-based foods and had they started this diet earlier in life, they would have seen even greater benefits, including a delay in puberty and an even lower risk of breast cancer later in life.

Women at high risk for breast cancer are given three options: watch and wait, take tamoxifen medication for the remainder of their lives or undergo mastectomy. **There should be a fourth option: consuming a diet free of animal-based foods and low in refined carbohydrates, aided by regular monitoring for those at high risk.** I stand by the usefulness of this fourth option even for women who have already had a first mastectomy. **Using diet as an effective treatment of already-diagnosed disease has been well documented in human studies with advanced heart disease,[48,49] clinically documented Type 2 diabetes (see chapter seven), advanced melanoma[50] (a deadly skin cancer) and, in experimental animal studies,[51] liver cancer.**

ENVIRONMENTAL CHEMICALS

There is another breast cancer conversation that has been taking place for some years now. It concerns environmental chemicals. These widely distributed chemicals have been shown to disrupt hormones, although it is not clear which hormones in humans are being disrupted. These chemicals may also cause reproductive abnormalities, birth defects and Type 2 diabetes.

There are many different types of offending chemicals, most of which are commonly associated with industrial pollution. One group, including dioxins and PCBs, persist in the environment because they are not metabolized when consumed. Thus they are not excreted from the body. Because of this lack of metabolism, these chemicals accumulate in body fat and breast milk of lactating mothers. Some of these chemicals are known to promote the growth of cancer cells, although humans may not be at significant risk unless one consumes excessive quantities of meat, milk and fish. Indeed, 90-95% of our exposure to these chemicals comes from consuming animal products-yet another reason why consuming animal-based foods can be risky.

There is a second group of these environmental chemicals that are also commonly perceived to be significant causes of breast52 and other cancers. They are called PAHs (Polycyclic Aromatic Hydrocarbons) and are found in auto exhaust, factory smoke stacks, petroleum tar products and tobacco smoke, among other processes common to an industrial society. Unlike the PCBs and dioxins, when we consume PAHs (in food and water), we can metabolize and excrete them. But there is a snag: when the PAHs are metabolized within the body, they produce intermediate products that react with DNA to form tightly bound complexes, or adducts (see chapter three). This is the first step in causing cancer. In fact, these chemicals have recently been shown to adversely affect the BRCA-1 and BRCA-2 genes of breast cancer cells grown in the laboratory.[53]

In chapter three, I described studies in my laboratory showing that when a very potent carcinogen is

put into the body, the rate at which it causes problems is mostly controlled by nutrition. Thus the rate at which PAHs are metabolized into products that bind to DNA is very much controlled by what we eat. Very simply, consuming a Western-type diet will increase the rate at which chemical carcinogens like PAHs bind to DNA to form products that cause cancer.

So when a recent study found slightly increased levels of PAH-DNA adducts in women with breast cancer in Long Island, New York,[54] it may well have been that these women were consuming a more meaty diet, which increased the binding of the PAHs to DNA. It is entirely possible that the quantity of PAHs being consumed had nothing to do with increasing breast cancer risk. In fact, in this study, the number of PAHDNA adducts in these women seem to be *unrelated* to PAH exposure.54 How is this possible? Perhaps all of the women in this Long Island study consumed a relatively uniform, low level of PAHs, and the only ones who subsequently got breast cancer were the ones who ate a diet high in fat and animal protein, thus causing more of the ingested PAHs to bind to their DNA.

In this same Long Island study, breast cancer was not associated with PCBs and dioxins, the chemicals that can't be metabolized.[55] As a result of the Long Island study, the hype associating environmental chemicals with breast cancer has been somewhat muted. This and other findings suggest that environmental chemicals seem to playa far less significant role for breast cancer than the kind of foods we choose to eat.

HORMONE REPLACEMENT THERAPY

I must briefly mention one final breast cancer issue: whether to use hormone replacement therapy (HRT), which increases breast cancer risk. HRT is taken by many women in order to alleviate unpleasant effects of menopause, protect bone health and prevent coronary heart disease.[56] However, it is now becoming widely acknowledged that HRT is not as beneficial as once thought, and it may have certain severe side effects. So what are the facts?

I am writing this commentary at an opportune time because the results of some large trials of HRT use have been released in the last year.[56] Of special interest are two large randomized intervention trials: the Women's Health Initiative (WHI)57 and the Heart and Estrogen/Progestin Replacement Study (HERS).[58] Among women who take HRT, after 5.2 years the WHI trial is showing a 26% *increase* in breast cancer cases while the HERS study is seeing an even greater 30% increase.[59] These studies are consistent. It appears that increased exposure to female hormones, via HRT, does indeed lead to more breast cancer.

It has been thought that HRT is associated with lower rates of coronary heart disease.[56] However, this is not necessarily true. In the large WHI trial, for every 10,000 healthy postmenopausal women who took HRT, there were seven more women with heart disease, eight more with strokes and eight more with pulmonary embolism[57]—the opposite of what had been expected. HRT may *increase* cardiovascular disease risk after all. On the other hand, HRT did have a beneficial effect on colorectal cancer and bone fracture rate. Among every 10,000 women, there were six fewer colorectal cancers and five fewer bone fractures.[57]

So how do you make a decision with such information? Just by adding and subtracting the numbers we can see that HRT may well be the cause of more harm than good. We can tell each individual woman to make her own decision depending on which disease and which unpleasantry she fears the most, as many physicians are likely to do. But this can be a tough decision for women who are having a difficult time with menopause. These women must choose between living unaided through the

emotional and physical symptoms of menopause in order to preserve a low risk of breast cancer, or taking HRT to manage their menopause discomforts while increasing their risk of breast cancer and, possibly, cardiovascular disease. To say that this scenario troubles me would be an understatement. We have spent well over a billion dollars on the research and development of these HRT medical preparations, and all we get is some apparent pluses and probably even more minuses. Calling this troubling doesn't begin to describe it.

Instead of relying on HRT, I suggest that there is a better way, using food. The argument goes like this:

- During the reproductive years, hormone levels are elevated, although the levels among women who eat plant-based diets are not as elevated.
- When women reach the end of their reproductive years, it is entirely natural for reproductive hormones of all women to drop to a low "base" level.
- As reproductive years come to an end, the lower hormone levels among plant eaters don't crash as hard as they do among animal eaters. Using hypothetical numbers to illustrate the concept, the levels of plant eaters may crash from forty to fifteen, rather than sixty to fifteen for animal eaters.

- These abrupt hormone changes in the body are what cause menopausal symptoms.
- Therefore, a plant-based diet leads to less severe hormone crash and a gentler menopause.

This argument is eminently reasonable based on what we know, although more studies would be helpful. **But even if future studies fail to confirm these details, a plant-based diet still offers the lowest risk for both breast cancer and heart disease for other reasons.** It might just be the best of all worlds, something that no drug can offer.

In each of the various issues involving breast cancer risk (tamoxifen use, HRT use, environmental chemical exposure, preventive mastectomy), **I am convinced that these practices are distractions that prevent us from considering a safer and far more useful nutritional strategy.** It is critical that we change the way we think about this disease, and that we provide this information to the women who need it.

LARGE BOWEL CANCER (INCLUDING COLON AND RECTUM)

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

At the end of June 2002, George W Bush handed the presidency over to Dick Cheney for a period of roughly two hours while he underwent a **colonoscopy**. Because of the implications President Bush's colonoscopy had for world politics, the story made national news, and colon and rectal screening were briefly thrust into the spotlight. Across the country; whether the comedians were making jokes or the news anchors were describing the drama, everybody was suddenly, briefly, talking about this thing called a colonoscopy and what it was for. It was a rare moment in which the country turned its focus to some of the most prolific killer diseases, colon and rectal cancers.

Because colon and rectal cancers are both cancers of the large bowel, and because of their other similarities, they often are grouped together under the term colorectal cancer. Colorectal cancer is the

fourth most common cancer worldwide, in terms of overall mortality.60 It is the second most common in the United States, with 6% of Americans getting the cancer during their lifetime?? Some even claim that, by age seventy, one-half of the population of "Westernized" countries will develop a tumor in the large bowel and 10% of these cases will progress to a malignancy.61

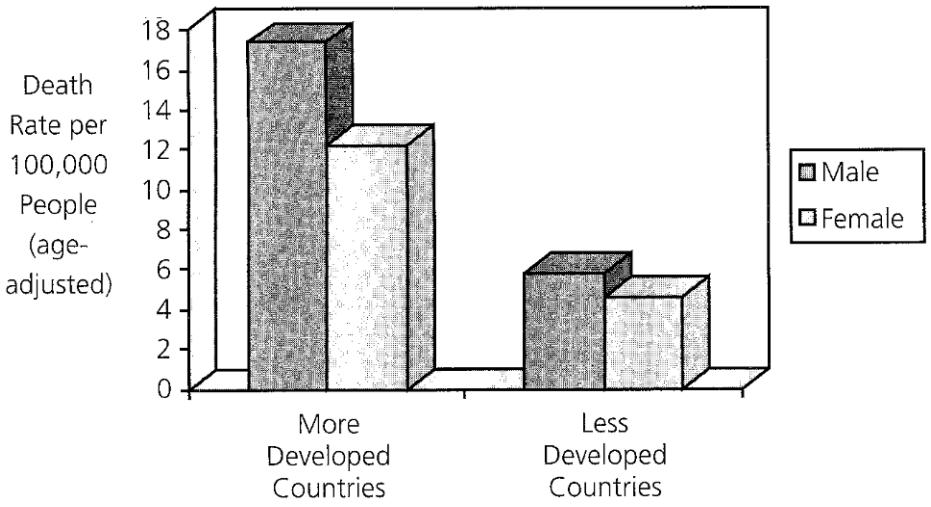
GEOGRAPHIC DISPARITY

North America, Europe, Australia and wealthier Asian countries Oapan, Singapore) have very high rates of colorectal cancer, while Africa, Asia and most of Central and South America have very low rates of this cancer. For example, the Czech Republic has a death rate of 34 [19] per 100,000 males, while Bangladesh has a death rate of 0.63 per 100,000 males! [62,63] **Chart 8.3** shows a comparison of average death rates between more developed countries and less developed countries; all these rates are age-adjusted.

The fact that rates of colorectal cancer vary hugely between countries has been known for decades. The question has always been why. Are the differences due to genetics, or to environment?

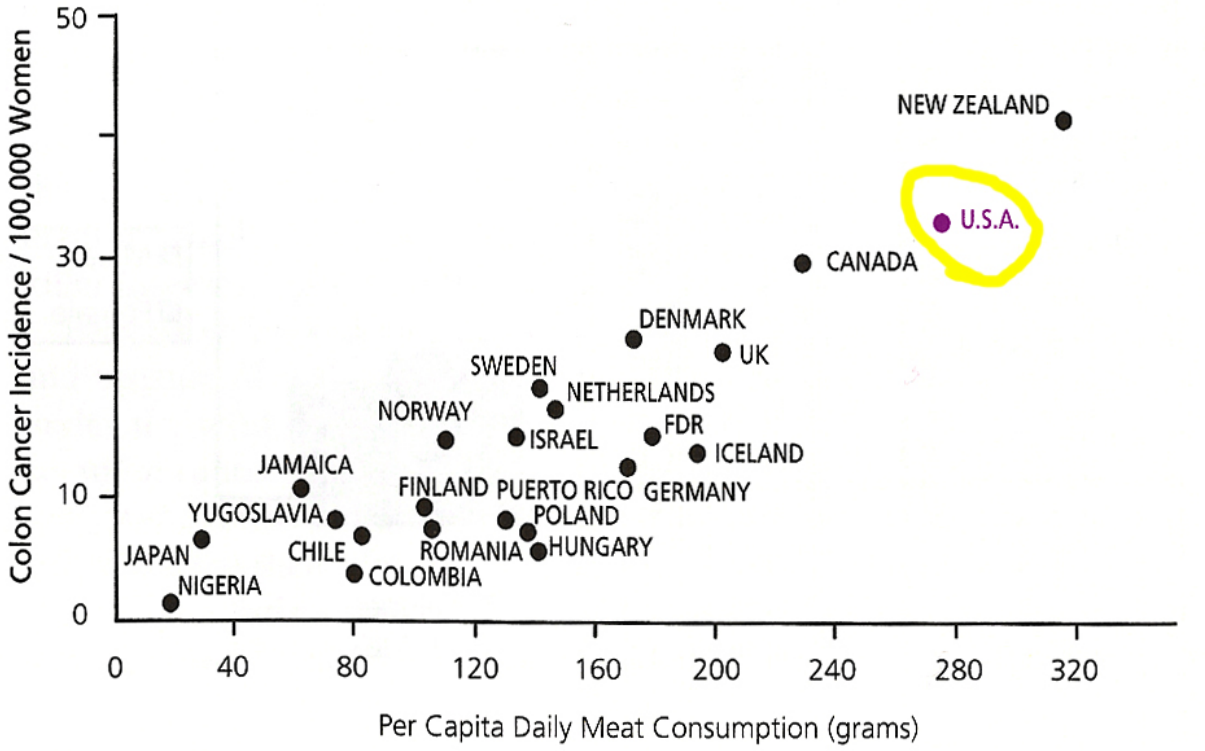
It seems that environmental factors, including diet, play the most important roles in colorectal cancer. Migrant studies have shown that as people move from a low-cancer risk area to a high-cancer risk area, they assume an increased risk within two generations.64 This suggests that diet and lifestyle are important causes of this cancer. Other studies have also found that rates of colorectal cancer change rapidly as a population's diet or lifestyle changes.64 These rapid changes in cancer rates within one population cannot possibly be explained by changes in inherited genes. In the context of human society; it takes thousands of years to get widespread, permanent changes in the inherited genes that are passed from one generation to the next. Clearly, something about environment or lifestyle is either preventing or enhancing the risk of getting colorectal cancer.

CHART 8.3: COLORECTAL CANCER DEATH RATE IN "MORE DEVELOPED" COUNTRIES AND "LESS DEVELOPED" COUNTRIES



In a landmark paper published almost thirty years ago, researchers compared environmental factors and cancer rates in thirty-two countries around the world.65 One of the strongest links between any cancer and any dietary factor was between colon cancer and meat intake. Chart 8.4 shows this link for women in twenty-three different countries.

CHART 8.4: FEMALE COLON CANCER INCIDENCE AND DAILY MEAT CONSUMPTION



In this report, countries where more meat, more animal protein, more sugar and fewer cereal grains were consumed had far higher rates of colon cancer.65 Another researcher whom I mentioned in chapter four, Denis Burkitt, hypothesized that intake of dietary fiber was essential for digestive health in general. He compared stool samples and fiber intakes in Africa and Europe and proposed that colorectal cancers were largely the result of low fiber intake.66 Fiber, remember, is only found in plant foods. It is the part of the plant that our body cannot digest. Using data from another famous study that compared diets in seven different countries, researchers found that eating an additional ten grams of dietary fiber a day lowered the long-term risk of colon cancer by 33%.67 There are ten grams of fiber in one cup of red raspberries, one Asian pear or one cup of peas. A cup of just about any variety of bean would provide significantly more than ten grams of fiber.

From all this research, it seems clear that something can be said for the importance of diet in colorectal cancer. But what exactly stops colon and rectal cancer? Is it fiber? Is it fruits and vegetables? Is it carbohydrates? Is it milk? Each of these foods or nutrients has been suggested to playa role. The debate has raged, and solid answers are seldom agreed upon.

THE SPECIFIC CURE

Most of the debate over the past twenty-five years on dietary fiber and its link to large bowel cancer began with Burkitt's work in Africa. Because of Burkitt's prominence, many people have believed that fiber is the source of colorectal health. Perhaps you have already heard that fiber is good for preventing colon cancer. At least you probably have heard that fiber "keeps things running well." Isn't that what prunes are known for?

Yet nobody has ever been able to prove that fiber is the magic bullet for preventing colorectal cancer. There are important technical reasons why a definitive conclusion regarding fiber is difficult to make.68 Each of these reasons is related either directly or indirectly to the fact that dietary fiber is not a single, simple substance producing a single, simple benefit. Fiber represents hundreds of substances, and "its" benefits operate through an exceptionally complex series of biochemical and physiological events. Each time researchers assess the consumption of dietary fiber, they must decide which of the hundreds of fiber sub-fractions to measure and which methods to use. It is nearly impossible to establish a standard procedure because it is virtually impossible to know what each fiber sub-fraction does in the body.

The uncertainty of having a standard procedure prompted us to measure fiber in more than a dozen ways in our China Study. As summarized in chapter four, as consumption of almost all of these fiber types went up, colon and rectal cancer rates went down.69 But we could make no clear interpretations70 as to which type of fiber was especially important.

Despite the uncertainties, I continue to believe that Burkitt's [66] initial hypothesis that *fiber-containing diets* prevent colorectal cancers is correct and that some of this effect is due to the aggregate effect of all the fiber types. In fact, the hypothesis that dietary fiber prevents large bowel cancers has become even more convincing. In 1990, a group of researchers reviewed sixty different studies that had been done on fiber and colon cancer. [71] They found that most of the studies supported the idea that fiber protects against colon cancer. They noted that the combined results showed that the people who consumed the most fiber had a 43% lower risk of colon cancer than the people who consumed the least fiber.[71] Those who consume the most vegetables had a 52% lower risk than those who consume the least vegetables.71 But even in this large review of the evidence, researchers noted, "the data do not permit discrimination between effects due to fiber and non-fiber effects due to vegetables."[71] So is fiber, all by itself, the magic bullet we've been looking for? We still, in 1990, didn't know.

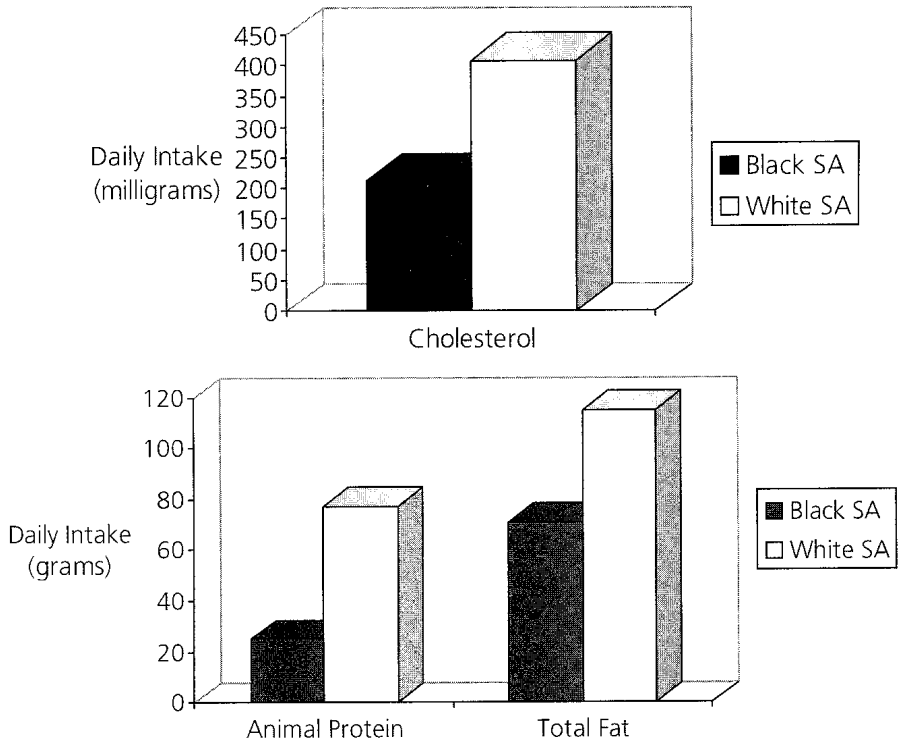
Two years later, in 1992, a different group of researchers reviewed thirteen studies that had compared people with and without colorectal cancer (case-control design).[72] They found that those who had consumed the most fiber had a 47% lower risk of colorectal cancer than those who consumed the least. [72] In fact, they found that if Americans ate an additional thirteen grams of fiber a day *from food sources* (not as supplements), about a third of all colorectal cancer cases in the U.S. could be avoided.[72] If you'll remember, thirteen grams, in real world terms, is the amount found in about a cup of any variety of beans.

More recently, a mammoth study called the EPIC study collected data on fiber intake and colorectal cancer in 519,000 people across Europe.[73] They found that the 20% of people who consumed the most fiber in their diet, about thirty-four grams per day, had a 42% lower risk of colorectal cancer than the 20% who consumed the least fiber in their diet, about thirteen grams per day.[73] It's important to note once again that, as with all of these studies, dietary fiber was obtained in food, not as supplements. So all we can say is "fiber-containing diets" seem to significantly reduce the risk of colorectal cancer. We still can't say anything definitive about isolated fiber itself. This means that attempts to add isolated fiber to foods may not produce benefits. But consuming plant foods naturally high in fiber is clearly beneficial. These foods include vegetables (the non-root parts), fruits and whole grains.

In reality, we can't even be sure how much of the prevention of colorectal cancer is due to fiber-

containing foods, because as people eat more of these foods they usually consume less animal-based foods. In other words, are fruits, vegetables and whole grains protective, or is meat dangerous? Or is it both? A recent study in South Africa helped to answer these questions. White South Africans have seventeen times more large bowel cancer than black South Africans, and this was first thought to be due to the much higher consumption of dietary fiber among black South Africans provided by unrefined maize.[74] However, in more recent years, black South Africans have been increasingly consuming commercially *refined* maize-meal-maize minus its fiber. They now eat even less fiber than the white South Africans. Yet, colon cancer rates among blacks remain at a low level,[75] which calls into question how much of the cancer-protective effect is due to dietary fiber alone. A more recent study[76] showed that the higher colon cancer rates among white South Africans could well be due to their elevated consumption of animal protein (77 vs. 25 g/day) , total fat (115 vs. 71 g/day) and cholesterol (408 vs. 211 mg/day), as seen in Chart 8.5.

CHART 8.5: INTAKE OF ANIMAL PROTEIN, TOTAL FAT AND CHOLESTEROL AMONG BLACK AND WHITE SOUTH AFRICANS



The researchers suggested that the much higher colon cancer rates among white South Africans may be more related to the quantity of animal protein and fat in their diets than their lacking the protective factor of dietary fiber. [76]

What is clear is that diets naturally high in fiber and low in animal-based foods can prevent colorectal cancer. Even in the absence of more specific details, we can still make important public health recommendations. *The data clearly show that a whole foods, plant-based diet can dramatically lower colorectal cancer rates.* We *don't need to know which fiber* is *responsible, what mechanism* is *involved or even how much of the effect* is *independently due to fiber.*

OTHER FACTORS

It has been recently noted that the same risk factors that promote colorectal cancer, a diet low in fruits and vegetables and high in animal foods and refined carbohydrates, can also promote insulin resistance syndrome.[77-79] From there, **scientists have hypothesized that insulin resistance may be responsible for colon cancer.[**77-82] Insulin resistance was described in chapter six as a diabetic condition. And what's good for keeping insulin resistance under control is also good for colon cancer: a diet of whole, plant-based foods.

This diet happens to be very high in carbohydrates, which have recently been under assault in the marketplace. Because carbohydrate confusion persists, let me remind you that there are two different types of carbohydrates: **refined carbohydrates** and **complex carbohydrates**. Refined carbohydrates are the starches and sugars obtained from plants by mechanically stripping off their outer layers, which contain most of the plant's vitamins, minerals, protein and fiber. This "food" (regular sugar, white flour, etc.) has very little nutritional value. Foods such as pastas made from refined flour, sugary cereals, white bread, candies and sugar-laden soft drinks should be avoided as much as possible. But do eat whole, complex carbohydrate-containing foods such as unprocessed fresh fruits and vegetables, and whole grain products like brown rice and oatmeal. These unprocessed carbohydrates, especially from fruits and vegetables, are exceptionally health-promoting.

CALCIUM AND COLON CANCER

You also may have heard that calcium is beneficial in fighting colon cancer. This, of course, gets extended to the argument that cow's milk fights colon cancer. It has been hypothesized that high-calcium diets prevent colon cancer in two ways: first, it inhibits the growth of critical cells in the colon,[83, 84] and second, it binds up intestinal bile acids. These bile acids arise in the liver, move to the intestine and are thought to get into the large bowel and promote colon cancer development. By binding these bile acids, calcium is said to prevent colon cancer.

One research group demonstrated that high-calcium diets-generally meaning diets high in dairy foods-inhibit the growth of certain cells in the colon,[84] *but this effect was not entirely consistent for the various indicators of cell growth. Furthermore,* it is *not clear whether these presumably favorable biochemical effects really lead to less cancer growth.* [83,85] Another research group demonstrated that calcium does reduce the presumably dangerous bile acids, but also observed that *a high-wheat diet did an even better job of reducing the bile acids.*[86] But-and this is the really odd part-when a combination high-calcium and high-wheat diet was consumed, the binding effect on bile acids was weaker than for each individual supplement taken alone.[86] It just goes to show that when individually-observed nutrient effects are combined, as in a dietary situation, the expected may become the unexpected.

I doubt that a high-calcium diet, obtained through calcium supplements or through calcium-rich cow's milk, has a beneficial effect on colon cancer. In rural China where calcium consumption is modest and almost no dairy food is consumed,[87] colon cancer rates are not higher; instead they are much lower than in the U.S. **The parts of the world that consume the most calcium, Europe and North America, have the highest rates of colorectal cancer.**

Another lifestyle choice that is clearly important for this disease is exercise. Increased exercise is convincingly associated with less colorectal cancer. In one summary from the World Cancer Research Fund and the American Institute for Cancer Research, seventeen out of twenty studies found that

exercise protected against colon cancer.[64] Unfortunately, there seems to be no convincing evidence as to why or how this occurs.

SCREENING FOR TROUBLE

The benefits of exercise bring me back to President George W Bush. He is known to enjoy staying physically fit with a regular running routine, and that is undoubtedly one of the reasons why he received a clean bill of health when he had a colonoscopy. But what is a colonoscopy anyway, and is it really worth the effort to get checked? When people go to the doctor to get a colonoscopy, the doctor inspects the large bowel using a rectal probe and looks for abnormal tissue growth. The most commonly found abnormality is a polyp. Although it is not yet clear exactly how tumors are related to polyps, most scientists would agree88,89 that they share similar dietary associations and genetic characteristics. Those people who have noncancerous problems in the large bowel, such as polyps, often are the same people who later develop cancerous tumors.

So getting screened for polyps or other problems is a reasonable way to establish risk for large bowel cancer in the future. But what if you have a polyp? What is the best thing to do? Will surgical removal of the polyp lessen colon cancer risk? A nationwide study has shown that, when polyps were removed, there was a 76-90% decrease in the expected cases of colon cancer.89,90 This certainly supports the idea of routine screening.89,91 It is commonly recommended that people get a colonoscopy once every ten years starting at the age of fifty. If you have a higher risk of colorectal cancer, it is recommended that you start at the age of forty and screen more frequently.

How do you know if you are at a higher risk for colorectal cancer? We can very roughly assess our personal genetic risk in several ways. We can consider the probability of our getting colon cancer based on the number of immediate family members who already have the disease, we can screen for the presence of polyps, and we now can clinically test for the presence of suspect genes.92

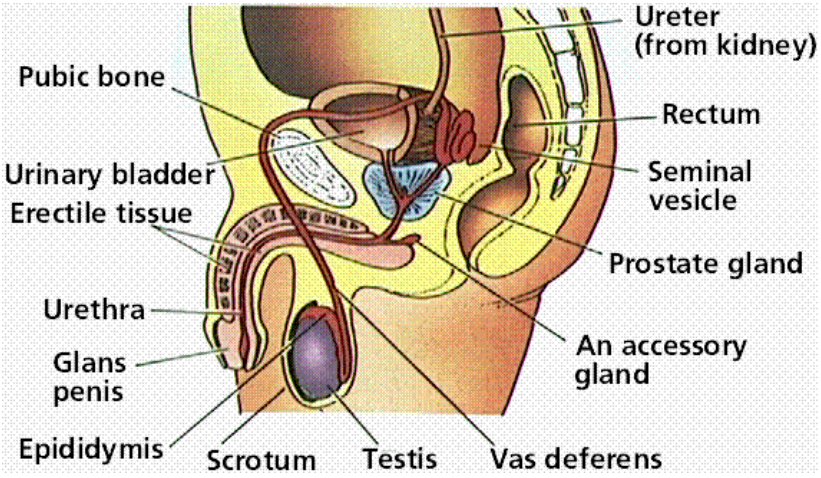
This is an excellent example of how genetic research can lead to a better understanding of complex diseases. However, in the enthusiasm for studying the genetic basis for this cancer, two things often get overlooked. First, the proportion of colon cancer cases attributed to known inherited genes is only about 1-3%.89 Another 10-30%89 tend to occur in some families more than others (called familial clustering), an effect possibly reflective of a significant genetic contribution. These numbers, however, exaggerate the number of cancers that are solely "due to genes."

Except for the very few people whose colon cancer risk is largely determined by known inherited genes (1-3%), most of the family-connected colon cancer cases (i.e, the additional 10-30%) are still largely determined by environmental and dietary factors. After all, place of residence and diet are often shared experiences within families.

Even if you have a high genetic risk, a healthy plant-based diet is capable of negating most, if not all, of that risk by controlling the expression of these genes. Because a high-fiber diet can only prevent colon cancer-extra fiber won't ever *promote* colon cancer-dietary recommendations should be the same regardless of one's genetic risk.

PROSTATE CANCER

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



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.

BRINGING IT TOGETHER

Roughly half a million Americans this year will go to the doctor's office and be told that they have cancer of the breast, prostate or large bowel. People who get one of these cancers represent 40% of all new cancer patients. These three cancers devastate the lives of not only the victims themselves, but also their family and friends.

When my mother-in-law died of colon cancer at the age of fifty-one, none of us knew that much about nutrition or what it meant for health.

It wasn't that we didn't care about the health of our loved ones-of course we did. We just didn't have the information. Yet, over thirty years later, not much has changed. Of the people you know who have cancer, or are at risk of having cancer, how many of them have considered the possibility of adopting a whole foods, plant-based diet to improve their chances? I'm guessing very few of them have done so. Probably they, too, don't have the information.

Our institutions and information providers are failing us. Even cancer organizations, at both the national and local level, are reluctant to discuss or even believe this evidence. Food as a key to health represents a powerful challenge to conventional medicine, which is fundamentally built on drugs and surgery (see Part IV). The widespread communities of nutrition professionals, researchers and doctors are, as a whole, either unaware of this evidence or reluctant to share it. Because of these failings, Americans are being cheated out of information that could save their lives.

There is enough evidence now that doctors should be discussing the option of pursuing dietary change as a potential path to cancer prevention and treatment. There is enough evidence now that the U.S. government should be discussing the idea that the toxicity of our diet is the single biggest cause of cancer. There is enough evidence now that local breast cancer alliances, and prostate and colon cancer institutions, should be discussing the possibility of providing information to Americans everywhere on how a whole foods, plant-based diet may be an incredibly effective anti-cancer medicine.

If these discussions were to happen, it is possible that, next year, fewer than 500,000 people would go to the doctor's office and be told they have cancer of the breast, prostate or large bowel. The year after that, even fewer friends, coworkers and family members would be given the most dreaded of all diagnoses. And the following year, even fewer.

The possibility that this future could be our reality is real, and as long as this future holds such promise for the health of people everywhere, it is a future worth working for.