Proteinaholic

HOW OUR OBSESSION WITH MEAT IS KILLING US AND WHAT WE CAN DO ABOUT IT

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Cancer: Fleshing Out the Protein Connection

I don’t think I have to tell you this, but cancer is a big deal. Nothing strikes fear into a person more than the “C word.” In fact, while cancer is the second-leading cause of death, we spend double to triple on cancer research than on heart disease research.

Cancer can strike anybody at any age and any race. We have been in a war on cancer for four decades and yet cancer is poised to take over as the number one cause of death. We still seem baffled as to the best way to treat cancer, though our therapies are far more advanced than our ability to prevent. We talk a big game about prevention, but really when Western medicine says prevention, it means mammograms and colonoscopies. These are not preventive medicines, they are early diagnostic tools.

Why do we get cancer? You think I am going to say protein, don’t you? I wish it were so simple. So many things have to happen in order to get a cancer. Genetics do play a strong part. We have found certain tumor suppressor genes and tumor promoter genes that are linked with certain cancers, but just having a gene doesn’t mean you will get cancer. Something has to activate the gene. It could be an environmental toxin, it could be our food, it could be the sun, and obviously it could be a combination.

In fact, it is said that we always have cancers forming and dissipating in our bodies all the time. So maybe you go out in the sun and this causes
a gene mutation in a cell. That doesn’t mean you will necessarily get cancer. Our cells have an ability to repair DNA damage. We also have specialized cells that search the body for abnormal cells and extinguish them. The important thing is that we foster an environment where either we avoid the instigator or enable the repair.

So I cannot say that animal protein causes cancer. What I can say is that animal protein causes various reactions in the body that have been shown to be carcinogenic, and animal protein has been strongly correlated with cancer. In this chapter, we’ll see epidemiologic evidence linking animal foods to cancer incidence and mortality. We’ll also discover randomized controlled trials that prove a strong link between diet and cancer. But if we are going to say animal protein contributes to cancer development and progression, we first need to establish a mechanism of action, or probable cause.

**Probable Cause**

It’s time to peer into the microscope and look at the mechanisms by which particular foods and diets may contribute to cancer progression or prevention. Identifying biological plausibility is important to weed out possible correlations found in the population studies that may be due to random chance. For example, if you study several hundred thousand people over twenty years and consider enough variables, you may find associations that are purely due to chance, such as wearing a yellow polo shirt on Tuesdays being linked with less prostate cancer. Here’s a real-life example of a coincidence that looks real: you can predict stock market performance based on the winner of the Super Bowl. The market has had an up year following an NFC win and a down year after an AFC win over 80 percent of the time. Unless someone can find an explanatory mechanism linking the two data sets, we have to treat it as a whimsical coincidence. The following sections discuss the mechanisms connected to the development of many cancers.

**Cooking-Derived Bad Guys: HCAs**

Heterocyclic amines (HCAs) are chemicals formed in meat, or any animal protein, that is cooked at a high temperature or over an open flame (Sugimura 1997, 2000). You know those grill marks that commercials for meat
products like to display? Well, if you scrape off that grilled substance and send it to a lab, technicians will find a formaldehyde-like substance called “heterocyclic amine.”

2-Amino-1-methyl-6-phenylimidazo(4,5-b)pyridine (PhIP) is one such compound that has been well studied. The HCAs are implicated in many cancers, so it’s reasonable to ask if it’s the animal protein by itself, or the way meat is often seared or grilled, that is contributing to cancer development. One study of 1,600 people in North Carolina found that not only was meat consumption associated with colon cancer, but the strength of the association varied with cooking method. The worst way of cooking meat was pan fried and well done (Butler, Sinha, et al. 2003). This is likely due to that fact that the HCAs form when you burn animal protein.

Several animal studies link PhIP in cooked meat with prostate cancer. A 2005 prospective study of over 29,000 men confirmed that increased amounts of cooked meat lead to an increased risk of prostate cancer, and named PhIP specifically as a likely carcinogen (Cross, Peters, et al. 2005).

Another cancer closely linked to HCA, and PhIP in particular, is breast cancer. A large study of postmenopausal women in Long Island found that grilled, barbecued, and smoked meat was associated with an increased risk of breast cancer (Steck, Gaudet, et al. 2007). Again, the mediating mechanism here appears to be HCAs and PhIP. A 2011 in vitro (test tube) study published in Toxicology lent support to this hypothesis. Researchers exposed breast cancer cells to PhIP and watched what happened. The cells began dividing and actually became so invasive that they started to digest and then go through the basement membrane (the matrix of tissue that separates the epithelial layer from deeper tissue) (Lauber and Gooderham 2011). This is kind of a chilling finding, if you think about it; this one chemical found in cooked meats converted cancer cells from early stage to malignant. This was just a lab study, but research on actual humans does confirm this correlation.

An intriguing 2009 study reinforced the HCA/breast cancer connection. It sought to examine the breast tissue of women who had not been diagnosed with breast cancer to look for evidence of precancerous changes that might be linked to diet. There’s one huge problem with this methodology, however: it’s not ethical to perform breast biopsies on healthy women. The researchers solved this problem by enrolling participants who were undergoing breast reduction surgery; their breast tissue was already going to be removed. The women undergoing the surgery were interviewed
about their diet, and their breast tissue was analyzed for the presence of DNA adducts (pieces of DNA that have bonded to a carcinogen and signal the beginning of cancer development). The study found that total HCA intake, and consumption of fried meat, beef, and processed meat were all correlated to precancerous damage to breast tissue (Rohrmann, Lukas Jung 2009). The scary thing is these women had no idea that this was happening in their breasts. They thought they had normal breasts, and all had negative mammograms, which are required prior to breast reduction.

While red meat, processed meat, and meat cooked at high temperatures appears to contribute to cancer formation via HCAs (and possibly other compounds, including polycyclic aromatic hydrocarbons (PAHs), fruits and veggies seem to mitigate those effects. In vitro studies show that eating fruits and veggies can block PhIP effects, a phenomenon that may have partially obscured the link between animal protein and cancer in the EPIC-Oxford study, which we will see in the next section. Since the meat eaters in that study were consuming unusually large quantities of fruits and veggies compared to the general population, they may have been somewhat protected from PhIP’s carcinogenic effects (Edenharder, Sager et al., 2002). So the data in the EPIC-Oxford study may suggest animal protein does not cause breast cancer, or is weakly correlated, while in actuality the effect is being neutralized by the high plant content in the diet.

**Heme Iron and N-Nitroso Compounds**

We ingest two types of iron from food: heme iron and nonheme iron. Animal foods are the only sources of heme iron, which is often assumed to be nutritionally superior since we absorb it more efficiently into our bloodstream. Evidence is mounting, however, that links heme iron to various cancers, including colorectal cancer (Qiao and Feng 2013). We’re not exactly sure how heme iron contributes to cancer, but one promising theory links heme iron consumption to the creation of unstable N-nitroso compounds (NOC) in the body (Bingham, Hughes, et al. 2002). One of the EPIC studies showed that the more NOC produced, the higher the risk of gastrointestinal cancer; rectal cancer, specifically. A 2003 randomized controlled trial found that feeding people meat led to higher levels of NOC in their stool. When they were fed an equivalent amount of vegetable protein, their levels of NOC were much lower. And here’s the fascinating part about this study: supplementation with 8 grams
of heme iron spiked NOC levels, while adding ferrous (nonheme) iron had no such effect (Cross, Pollock, et al. 2003). So the “good-for-us” iron from meat may turn out to be the missing ingredient in turning nitrates into cancer-causing NOC.

An interesting 2010 study supports this theory. Researchers fed 14 participants a high-red-meat diet for 8 days, followed by 8 days of a combination of red meat and fish, then 8 days of a high-fish diet. Their stool samples were examined during each phase of the trial. The study found more NOC and heme iron on the red meat diet, and less on the fish diet (Joosen, Lecommandeur, et al. 2010). And another study by the same team found more NOCs and more DNA damage in the group eating red meat and processed meat than in those on a vegetarian diet (Joosen, Kuhnle, et al. 2009).

As with HCAs, eating more plants appears to reduce the effects of heme iron. Beets, for example, are high in nitrates, but there’s no evidence that those chemicals are converting into NOC; beets have fewer amino acids than meat and are chock-full of antioxidants. It appears this may block the conversion of plant sources of nitrates into NOC. The Mediterranean diet consists of many foods with high antioxidant ability and also have the ability to chelate iron; that is, take it out of the bloodstream and remove it from the body (El and Karakaya 2004). Greens in particular are highly protective. This finding may also contribute to explaining the small differences found by EPIC-Oxford between meat eaters and vegetarians. The meat eaters were consuming a fair amount of plant-based food, which may have been partially canceling the toxic effects of heme iron.

**IGF1: Another Smoking Gun**

Insulin-like growth factor 1 (IGF1) is a human growth hormone produced in the liver and other tissues. We produce it all our lives, and in the proper amounts it helps us grow new tissue and synthesize new cellular DNA. We need IGF1 in the right quantities. Too little IGF1 is correlated with dwarfism, while if you are born with a disease where you make too much IGF1, then you develop acromegaly (giantism). You may remember the wrestler André the Giant. He had acromegaly. The large body and large forehead are a giveaway. The sad thing about people with acromegaly is they die an early death either from heart failure or from cancer. Studies show that people with acromegaly caused by high levels of IGF1 have high levels of...
breast, prostate, and colon cancer (Jenkins 2006; Epstein 2001).

The reason people with high IGF1 levels develop cancer can be explained. If you put cancer cells (or normal cells) in a culture dish and expose them to IGF1, the cells will activate and grow. Accelerated cell growth is part of cancer development. IGF1 can also promote cancer by inhibiting cell death. Cell death may not sound like a good thing, but it is. When normal cells become damaged, they activate a self-suicide process known as “apoptosis.” Cancer cells lose this ability, which is one reason cancer can spread so aggressively at the expense of the rest of the body (Pollak 1998).

It’s known that prostate cells are highly sensitive to IGF1 effects (Cohen and Peehl 1994). The 1998 Physicians Health Study associates increased IGF1 with higher rates of prostate cancer (Chan, Stampfer, et al. 1998). And it turns out there’s a relationship between our diets and the IGF1 levels in our bodies. Restricting energy and animal protein intake causes IGF1 to fall. This is a crucial finding, as many studies show that calorie restriction slows aging and increases longevity. The trouble is, very few people are willingly to adopt a “mild starvation” diet to increase their life span. Fortunately, it appears that caloric restriction isn’t necessary; it’s the accompanying restriction in animal protein that decreases IGF1 and thereby increases longevity (Fontana, Weiss, et al. 2008).

The more animal protein we consume, the more circulating IGF1. Vegans, for example, have much less circulating IGF1 than meat eaters. And in addition to making our own IGF1, we can consume it in our diets. Milk cows are routinely treated with growth hormones to get them to milk-producing age as fast as possible. This IGF1 gets into their milk, and we absorb it into our bodies when we drink that milk (even when pasteurized) (Epstein 2001). Even hormone-free, organic milk contains IGF1 (after all, baby calves, for whom nature intended the milk, need to grow a lot over a short period of time). And its high protein content also increases IGF1 levels in humans.

One way that a vegan diet may protect against cancer is by limiting IGF1 synthesis and absorption in the body. A 2002 study, spurred by observations that Asian countries had lower incidences of cancer compared to Western societies, explored IGF1 as a possible cause. Researchers found that vegans had significantly lower levels of IGF1 and significantly higher levels of a protein that binds IGF1 and takes it out of active circulation. They concluded that a diet rich in essential amino acids (the ones
found extensively in meat) was associated with higher IGF1 levels (Allen, Appleby, et al. 2002). And for “manly” men concerned about getting enough protein, another study found that despite having lower IGF1 levels, the vegan men actually had higher testosterone levels than vegetarians or meat eaters (Allen, Appleby, et al. 2000).

Several studies link IGF1 to breast cancer (Pollak 1998; Toniolo, Bruning, et al. 2000; Rollison, Newschaffer, et al. 2006). The Nurses’ Health Study found a strong correlation in premenopausal women even though the dietary range of participants was quite narrow (Hankinson, Willett, et al. 1998). A 2010 meta analysis of 17 prospective studies found that increased IGF1 levels are associated with hormone sensitive breast cancer in both pre- and postmenopausal women (Key, Appleby, et al. 2010).

Breast and prostate cancer are just the beginning. IGF1 has also been associated with colon cancer and I am sure it has an effect on others as science is just starting to study this interesting hormone (Epstein 2001).

**Carnitine, Choline, and TMAO**

Another mechanism linking meat and prostate cancer (and possibly others) comes from ingestion of certain nutrients—nutrients some people actually supplement with. I mentioned carnitine and choline as possible causes of heart disease in the cardiac chapter. Well, there may also be a link when we look at cancer, especially prostate cancer. Eggs in particular are very high in choline. A prospective study of men already diagnosed with prostate cancer found that consuming high amounts of eggs and the skin of chickens doubled the risk of cancer progression or recurrence. Those men deemed “high risk” for recurrence were even more susceptible; their risk of recurrence or progression of prostate cancer quadrupled compared to men who ate the smallest amount of eggs and chicken skin (Richman, Stampfer, et al., 2010). And the Health Professionals Follow-up Study found that those men who ate the most eggs increased their risk of dying from prostate cancer by 70 percent compared with the men who ate the fewest (Richman, Kenfield, et al. 2012). Interestingly, the actual prostate cancer cells were found to have high choline levels. Why does this cause cancer? Researchers are not sure. There are several possible mechanisms that are being investigated (Richman, Kenfield, et al. 2011, 2012).
Reduction in Protective Bacteria

Butyrate, a substance formed when carbohydrates are fermented in the large intestines, helps with gene repair and apoptosis (cell death) (Bingham 1999). Both of these activities are protective against cancer and are specifically linked to reduced risk of adenomas of the colon, which are pre-cancerous lesions. Nondigestible carbohydrates found in fruit, fructooligosaccharides, help promote bifidobacter, a beneficial bacteria that produces butyrate, which in turn protects the lining of the colon. A 2007 randomized controlled trial put people on first a high-carb, then a low-carb diet. The low-carb diet led to a significant reduction in butyrate production, which was accompanied by decreases in key protective bacteria (Duncan, Belenguer, et al. 2007). A 2013 study of people with adenomas found a relationship between fiber intake, butyrate formation, and subsequent risk of adenomas (Chen, Yu, et al. 2013).

A 2011 randomized controlled trial confirmed this relationship. Researchers placed 17 obese men on a “weight-maintenance diet” consisting of 12 percent protein, 37 percent fat, and 51 percent carbs (total calories: 2,824) for seven days. They spent the next four weeks on a high-protein/moderate-carb diet (28% protein, 37% fat, 36% carbs, 2,018 total calories), and the four weeks after that on a high-protein/low-carb diet (28% protein, 67% fat, 5% carbs, 1,923 total calories). Stool samples showed that the high-protein/low-carb diet “increased concentrations of hazardous metabolites” and significantly decreased concentrations of cancer-protective metabolites, including butyrate. The researchers concluded that despite the reduced calorie intake, which is known to be beneficial, long-term adherence to a high-protein/low-carb diet may increase the risk of colon cancer (Russell, Gratz, et al. 2011).

We know that diet has a rapid effect on our bowel bacteria. Worse yet, it has been shown that a diet heavy in meat alters the gut bacteria rapidly and allows colonization of certain bacteria that may cause inflammation in the bowels (David, Maurice, et al. 2014).

Methionine

Methionine is an essential amino acid, which means that humans cannot synthesize it and must get it in our diets. It appears in high concentrations in eggs, fish, and meat, and in some seeds and nuts, and in much lower lev-
els in fruits, vegetables, and legumes. We need some methionine, but is too much methionine too much of a good thing?

Research has shown that restricting methionine increases longevity in rodents. It’s been known for a while that caloric restriction increases life span; now we’re starting to gain a better understanding of the underlying mechanisms. It’s not all calories, but those from animal foods, that tend to shorten life. Methionine restriction appears to reduce cancer risk by preventing oxidation of mitochondria (López-Torres and Barja 2008), a phenomenon that’s been documented in rodents and mammals, as well as in human cancers (Epner, 2001). Researchers are continuing to test the hypothesis that methionine restriction may increase life span in humans (McCarty, Barroso-Aranda, et al. 2009).

Some low-carb promoters, most notably Robert Lustig, see sugar as the root of all dietary evils and recommend attacking cancer by starving it of sugars. The problem with this approach is that all cells need sugar, so starving the cancer requires starving the whole body. Also, when people have to eliminate sugar and carbs they turn to meat, and therefore eliminate the antioxidants while increasing cancer-causing chemicals. Not good for curing cancer.

In an excellent 2003 article, researchers reviewed a number of in vitro (lab) and in vivo (in the body) studies showing that methionine restriction harms cancer cells but not normal ones. It appears that cancer cells must have methionine to reproduce. They suggest that methionine restriction may become a helpful additional therapeutic strategy in conjunction with chemotherapy (Cellarier, Durando, et al. 2003). Instead of starving cancer cells by starving yourself, adopt a plant-based diet that naturally lowers your methionine, an essential nutrient for cancer cells to divide, without harming your natural ability to heal.

**Acidosis**

As we saw in Chapter 9, an animal-protein-heavy diet creates a dangerous condition in the body known as acidosis. Evidence is beginning to emerge suggesting that chronic acidosis may act at a molecular/cellular level in ways that promote cancer formation (Robey 2012). So a low bicarbonate level on a blood test, such as the one Gary Taubes publicized to show the world how healthy he is, should be a wake-up call to take action to prevent cancer as well as diabetes.
Neu5Gc

Neu5Gc is a form of sialic acid that is found in animals, but not native to humans. Meat eaters can start expressing Neu5Gc after eating meat, however. After a person consumes an animal containing Neu5Gc, their body will incorporate it into the glycoproteins found on the surface of their cells. The problem is, even though the body is now expressing and assimilating Neu5Gc, it still doesn’t quite recognize it as part of itself. When faced with a potential alien invader, the body creates antibodies, causing an inflammatory immune response (Tangvoranuntakul, Gagneux, et al. 2003). The ongoing antibody/antigen reaction may generate chronic inflammation that may contribute to carcinomas (Varki 2008). Cancer cells have been know to express Neu5Gc, which can only come from animals. Meanwhile, people who avoid animal proteins do not express Neu5Gc.

Hormone Plasma Levels

Some cancers are induced by specific hormones, including breast and prostate cancers. These hormonally induced cancers can be affected by diet, in that different foods affect the plasma levels of hormones in our bodies. Seventh-day Adventist males, for example, have one-third the mortality from prostate cancer as other Californians, and they consume 6 percent fewer fat calories. Vegetarians have different levels of various hormones than meat eaters, including lower estrogen levels (despite whatever nonsense you may have heard about soy). It’s clear that we can reduce our cancer risk by changing our hormonal concentrations (Howie and Schultz 1985).

Also, we know that early onset of menses strongly increases the risk of breast cancer (Cheng Buyken, et al. 2012). It has been well established that children who eat more animal protein are at higher risk of developing premature puberty (Günther, Karaolis-Danckert, et al. 2010). Meanwhile, a diet low in animal protein but high in plants and soy exposes children to isoflavones, which are nutrients that have been shown to prevent early periods and to lower lifetime risk of developing breast cancer (Cheng, Remer, et al. 2010).

Slaughterhouse Chemicals

We don’t know much about the effects of the many chemicals used during the raising, slaughter, processing, and packaging of the meats produced by
our industrial farm system. That doesn’t mean there isn’t reason for concern: one study found that men working in a poultry slaughtering/processing plant got cancer of the penis almost nine times more frequently than the general population (Johnson, Ndetan, et al. 2010). The animals are given lots of hormones and antibiotics and ingest food that has all kinds of pesticides and chemicals. These can all have an effect on our health.

**Thermoresistant Viruses**

The final animal-protein-based cancer mechanism we’ll look at is thermoresistant viruses (that is, they don’t die even when cooked at high temperatures). Population studies show a clear and consistent relationship between red meat consumed and colon cancer. Red meat appears to increase colon cancer much more than fish and chicken, despite the formation of similar chemicals when cooked, including IGF1, HCAs, TMAO. It’s become clear over the past decade that some cancers are triggered by viruses. Some researchers suspect that certain viruses found in cows initiate colon cancer (zur Hausen 2012).

**The Protective Role of Phytonutrients**

Part of the problem with our current reductionist philosophy when it comes to food is this false notion that carbs are bad, which inevitably leads to avoidance of fruits in an attempt to eat more animal protein. While I think we have shown there are harmful ingredients in animal protein, there is also the opportunity cost that comes from filling your belly with animal protein instead of plants.

Phytonutrients (nutrients synthesized only in plants) can foil cancer development through a large array of mechanisms. A 1991 review identified many potential cancer-fighting compounds in plants, “... including carotenoids, vitamins C and E, selenium, dietary fiber, dithiolthiones, glucosinolates and indoles, isothiocyanates, flavonoids, phenols, protease inhibitors, plant sterols, allium compounds, and limonene.” It concludes that parts of fruits and veggies are essential for our growth and maintenance and hypothesizes that cancer is a maladaptive response to their deficiency (Steinmetz and Potter 1991). Cancer as a deficiency in fruits and
veggies? An interesting possibility. If you look at the NHANES data on what we eat, we consume a surplus of protein but are well below the RDA for fiber consumption.

Flavonoids are a large family of phytonutrients shown to have powerful positive effects on our health. So far science has identified about six thousand of them, but there’s no reason to suspect there aren’t thousands more. They include the anthocyanidins found in berries and other fruit, the flavones from parsley, lettuce, apples, and oranges, and the flavonols in onions, almonds, sweet potatoes, and garbanzo beans.

There’s reason to believe that fruits and veggies reduce cancer risk in and of themselves, even if animal protein consumption is not reduced. The EPIC study showed that increased flavonoid consumption led to a decrease in gastric cancer in women (Zamora-Ros, Agudo, et al. 2012). A 2003 meta-analysis found “weak evidence” for the protective effect of fruits and vegetables for all cancers, but it was able to pinpoint some specific links, despite various flaws the authors identified in the individual studies. These included the finding that fruit consumption lowers the risk of bladder and lung cancer (Riboli and Norat 2003).

So what’s the bottom line? If we eat our fruits and veggies, can we avoid cancer even if we don’t dial back our animal protein consumption? Several studies suggest the answer is no. While fruits and veggies are crucial, it’s just as important to avoid the toxins found in meat, eggs, and dairy. Colon cancer is strikingly rare in black Africans: the prevalence is less than 1 case per 100,000 people. For comparison, the rate among white South Africans is about twenty times higher. Researchers wondered about the huge discrepancy, and a 1999 study of low-risk black South Africans and high-risk white South Africans looked at many dietary factors to see which ones might be significant contributors. According to the authors, “The diets of all the black subgroups were characterized by a low animal product and high boiled maize-meat content, whereas whites consumed more fresh animal products, cheese, and wheat products. Blacks consumed below RDA quantities of fiber (43% of RDA), vitamin A (78%), C (62%), folic acid (80%) and calcium (67%), whereas whites consumed more animal protein (177% of RDA) and fat (153%).” The researchers determined that the low rates of colon cancer among black South Africans could not be explained by known “protective factors,” as their diet consisted largely of boiled maize. Instead, they found, the difference was the lack of “aggressive” factors, specifically “excess animal protein and fat” (O’Keefe, Kidd, et al. 1999).
This finding is supported by an analysis of the Health Professionals Follow-up Study of 48,000 men (Giovannucci, Rimm, et al. 1994). They found a significant risk of colon cancer specifically linked to animal protein consumption, one that was independent not only of fruit and veggie consumption, but also of saturated fat, total fat, and animal fat. In other words, animal protein all by itself increased colon cancer in middle-aged American men.

A 2005 meta-analysis of 13 large-scale prospective studies following 725,000 men for between 6 and 20 years likewise found that fiber intake is only weakly correlated with decreased rates of colorectal cancer. It may be that avoiding the meat protein is a bigger deal than eating lots of produce (Park, Hunter, et al. 2005). And if you’re still wondering if the problem with animal foods is simply an effect of factory farming, a 2009 study out of Uruguay found a definite association between meat consumption and colon, gastric, and pharyngeal cancer. Interestingly, their animals were grass fed and hormone free, just like the Paleo people recommend, yet the cancer risk was still strong (Aune, De Stefani, et al. 2009). The HCA, PhIP, acid, IGF1, and so on, occur in grass-fed meat as much as they do in animals from industrial farms.

Epidemiology of Cancer and Diet

So we have established a probable cause. But if this cause is accurate we should see an effect. There should be a causal link between animal protein and actual cancer. The best way to prove this would be to start a study of people and randomize them to a high animal protein or low animal protein group. We would then have to make sure they stay on the diet by providing them the actual food and monitor every bite they take. Both groups would have to live the exact same lives. Same exposure to toxins, same exercise, and so on. And this study would ideally go many years. Study length really matters; most cancers take decades to get big enough to cause symptoms and thus get our attention. One article estimated the latency period between diet and cancer development at 15 to 20 years, except for prostate cancer, which required 28 years to manifest (Grant 2014). As we saw in Chapter 9, it’s quite rare to study a population for decades. It’s expensive, it’s difficult, and perhaps most important, it’s potential career suicide for
less established scientists working under constant pressure of “publish or perish.” But, if we are following two groups for only two years, it is unlikely that, if one of the diets is causing a cancer, that cancer would rear its ugly head in just two years. In fact, if a person in one group did develop a cancer, was that cancer from the experimental diet or the diet they ate before the experiment began?

All is not lost, however. There are many studies that look at the interplay of animal protein and cancer, following hundreds of thousands of people for many years, in many different countries. These studies tend not to randomize to a diet since it hard for people to stick to a diet. Instead they follow people who already eat a certain way and compare those that eat lots of protein versus those who do not. Lots of issues still arise. There is something called a healthy volunteer effect, where people who volunteer for a study tend to be healthier to begin with (Struijk, May, et al. 2015). Because they are healthier we are less likely to see differences between groups, especially if not a long-term study. The other issue with this is we tend to see little difference between the study groups. If two groups are actually eating a similar diet that only differs a little in amount of meat, we are less likely to see an actual significant difference in the study.

Finally there are plenty of confounding factors, meaning while animal protein may cause cancer, it may also be that one group has more smokers, or one group is heavier. Thankfully, we have very advanced statistical analysis that can eliminate these biases that may influence the data. The problem with the statistics is that scientists tend to overadjust. They are so concerned with eliminating bias that sometimes they actually eliminate a real correlation.

The point here is that there are many great long-term studies that draw attention to the link between animal protein and cancer. People may say that this is just a correlation, but I will tell you that if there is a correlation in multiple studies from multiple parts of the world, and these correlations have been put through rigorous analysis, then you better believe there is something to the connection.

You cannot just look at one individual study. You have to look at the entirety of the research, from the lab studies, to the randomized trials to the epidemiology.

For instance, let’s look at prostate cancer. Many studies document the fact that there is a much lower incidence of prostate cancer in Asia compared to America (Jemal, Center, et al. 2010). There could be many reasons
for this. It could be genetic, although migration studies of people moving to America show that, despite their genes, they actually get prostate cancer. Migrations studies are fascinating. We tend to think that cancer is all genetics but we find that cultures that have low rates of cancer seem to get the "big C" when they move to America. Take breast cancer, for example. Asian countries typically show low incidence of breast cancer, but Asian communities in the United States soon develop breast cancer at the same rate as other Americans (Deapen, Liu, et al. 2002). Breast cancer is also rapidly increasing in Asian and African countries as they adopt Western lifestyles and diets (Kelsey and Horn-Ross 1993).

So if the prostate cancer difference is not genes, could it be due to not testing for prostate cancer appropriately? This is true in parts of Asia, but Japan does have an active PSA testing. In addition, as Japanese men move to America, their rates of prostate cancer approximate ours.

So if it is not genetic, could it be environmental? Certain pollutants could be to blame but the best way to get polluted is in what you eat. Looking at diets in Japan versus America, Japanese eat far more soy but far less overall protein, more fruits and veggies, and far less meat. So they are not being exposed to all the carcinogenic chemicals we discussed earlier.

I would assume that the phytochemicals in fruits and veggies would be protective against prostate cancer, but a study looked at a large population in Europe and found that the amounts of fruits and veggies eaten didn’t seem to make a difference in occurrence of prostate cancer (Key, Allen, et al. 2004). In fact, while the Adventist Health Study does show plant eaters have lower rates of prostate cancer, they still have large amounts. This could be due to dairy consumption. Milk and dairy are still consumed by vegetarians. So maybe it is not how many plants but rather how few animals that matters.

The Seventh-day Adventists offer us a great ability to further investigate this dilemma because, as we have seen, they have a fairly large group of vegans that we can compare to vegetarians and to meat eaters. It turns out that vegans have lower prostate cancer compared to vegetarians, and vegetarians have lower cancer rates than meat eaters (Fraser 1999). So maybe it is the lack of animal protein. This is a maybe. These epidemiologic studies show correlation but not necessarily causation.

However, there are other factors out there that can lead us to a better idea of causal relationships. As we have seen, we know that if you put cancer cells (or normal cells) in a culture dish and expose them to IGF1, the
cells will activate and grow. Does this mean that IGF1 causes cancer? Not necessarily, but we do know that people who have acromegaly caused by high levels of IGF1 also have high levels of breast, prostate, and colon cancer. And we have shown that high IGF1 levels are associated with prostate cancer independent of having acromegaly.

To delve deeper we know that IGF1 release in the body is enhanced by consumption of animal protein. As we have seen, vegans have much lower circulating IGF1 than meat eaters. We also know that growth hormone given to cows gets into milk and is absorbed when we drink the milk even if it has been pasteurized. So could a vegan diet given to prostate cancer patients reduce the growth of cancer by reducing IGF1?

Well, Dr. Dean Ornish had the unique opportunity to test this hypothesis (Ornish, Weidner, et al. 2005). In 2005, he and his colleagues did a study in which they randomized 93 early-stage prostate cancer patients into a low-fat vegan diet (along with his lifestyle modification program) or a “standard of care” control group and followed them for a year (Ornish, Weidner, et al. 2005). During that whole period, those eating the low-fat vegan diet consumed more protective and fewer harmful dietary factors than the control group. Once put on the vegan diet, they ate more fiber (59 grams at year’s end, compared to 31 at baseline), more lycopene, a cancer-fighting phytonutrient (34,464 mcg/day vs. 8693), and fewer saturated fats (from 20 grams/day down to 5) and cholesterol (from 200/mg per day down to 10) (Dewell, Weidner, et al. 2008). Again, note that the lycopene came from whole foods, not pills.

They then specifically looked at the progression of their cancer by monitoring their serum prostate-specific antigen (PSA), a measure of the presence of prostate cancer. They could see that the experimental group was avoiding carcinogens and eating protective substances—but was it slowing down or reversing the cancer?

The first thing the researchers noticed was that none of the lifestyle group, but six members of the control group, had to undergo treatment for worsening PSA. After one year, they found that the plant-based patients’ PSA levels had dropped by 4 percent, while the control group’s PSA levels had actually increased by 6 percent. Then Ornish and his team performed a fascinating experiment: they took serum from both groups and mixed it with prostate cancer cells. To their amazement, the serum from the lifestyle group was eight times more effective at stopping prostate cancer cells from replicating when compared to the control group. That’s right: the
vegan diet and other lifestyle changes literally made their blood poisonous to cancer. And both findings, the PSA changes and the serum’s ability to inhibit cancer growth, were associated with the degree to which the men adhered to the lifestyle protocol. The closer they got to “perfect” compliance, the better their numbers.

Ornish got curious about the mechanism by which his protocol reversed prostate cancer progression. He came across the work of Nobel Prize–winning scientist Elizabeth Blackburn, who discovered the health significance of chromosomal telomeres. Telomeres are caps that sit on the ends of our chromosomes and protect them from unraveling. You might think of them as the plastic protectors at the ends of shoelaces. Blackburn postulated that it’s possible to determine a person’s health, and even their potential life span, by looking at their telomeres. Originally, scientists thought of telomere length as fate: determined at birth, and unchangeable. Born with short telomeres? That’s a shame; you’re probably going to die young, possibly of an opportunistic cancer just waiting for a telomere to wear out.

Ornish has always assumed that most of us can change our health outcomes through our own efforts, regardless of the genetic hand we’ve been dealt at birth. So he teamed with Blackburn to test this theory. They recruited 30 men with low-risk prostate cancer that had been identified by biopsy, measured their telomere length, and put them on the vegan diet and lifestyle program for three months. Sure enough, the lifestyle modification lengthen the telomeres by 29 percent in just 90 days (Ornish, Lin, et al. 2008). This is incredible. Ornish showed that changing diet actually has an effect on a chromosomal level!

My point in this long diatribe is that you cannot look at just one study but need to look at the totality of the medical science to understand the underlying story.

The Epidemiology of the Protein/Cancer Link

In this section, I’ll take you on a tour of epidemiological studies, that is, large-scale studies of populations. Some just take static snapshots, while others follow participants for years and even decades. All have been published in leading peer-reviewed journals and have used strict protocols to
eliminate bias. We’ll examine the strengths and weaknesses of the studies, and slowly and carefully connect the dots and arrange the pixels to come up with a relatively clear picture, especially given our knowledge of the probable cause.

EPIC Evidence

As we’ve seen, the EPIC study is one of the most comprehensive large-scale epidemiologic studies ever conducted. It found that gastric cancer risk rose with increased consumption of total meat, red meat, and processed meat (González, Jakszyn, et al. 2006). The risk was even greater in people infected with H. pylori bacteria, a common infection now known as the main cause of ulcers (rather than stress or Advil). H. pylori itself may have a food connection, being better able to survive and flourish in the presence of high-protein diets. Further examination of EPIC data linked meat and processed meat consumption to colon and rectal cancer. The more fiber people ate, the less cancer they got. Fish consumption also appeared protective, but it’s not clear if fish itself helps fight tumors, or if fish was simply replacing land meats and was therefore “less worse.”

Other findings of EPIC: fruit helps prevent lung cancer, and saturated fat found in animal products may be associated with increased breast cancer (González 2006a, 2006b).

But then some researchers looked at a subset of the EPIC data and threw a big wrench in the works. The subset was known as EPIC-Oxford and consisted of a large number of vegetarians living in and around Oxford, England, where that sort of diet was much more common than other parts of Europe, and their meat-eating neighbors. In the EPIC-Oxford data, the incidence of colon cancer rose in vegetarians compared to meat eaters. True, the effect had dissipated in the 2014 data, but still. What in the world was going on?

The EPIC-Oxford data have been trumpeted by low-carb advocates as proof of the inferiority of a vegan diet. Funny that they always complain about epidemiologic studies, until one goes their way. But as we look at other studies, EPIC-Oxford really becomes an outlier.

A meta-analysis of several studies shows red meat and processed meat were associated with increase risk of colorectal cancer (Norat, Lukanova, et al. 2002). Another meta-analysis of multiple epidemiologic studies showed a 2.5 times greater risk of developing colon cancer in those eat-
ing animals. It also showed the less fiber, the more cancer, independent of whether the person was eating meat or not.

In 2007, the World Cancer Research Fund and the American Institute for Cancer Research published their report, “Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective.” In the following four years, researchers conducted ten prospective studies based on the recommendations in this report (many of which advised reducing animal consumption and eating more fruits and veggies). A 2011 meta-analysis of these studies showed that red meat and processed meat definitely correlate with colorectal cancer. This was very high level data (Chan Lau, et al. 2011).

A huge prospective Korean study followed over two million people for seven years. It looked at people who ate meat four days a week and those who ate meat just once a week. Researchers found a large and significant effect of meat eating on risk of colorectal cancer (Kim, Park, et al. 2011). And keep in mind that even the “heavy” meat eaters in this study were consuming considerably less animal protein than the average American.

So why did EPIC-Oxford show increased risk? Well, several reasons. As you can see above, one of the protective effects of being vegetarian/vegan is presumably eating more plants and therefore more fiber. However, this group of vegetarians didn’t eat nearly the amount of fiber you would expect for plant eaters. Only 20 grams, which is only slightly higher than the meat eaters. Other studies on this group show that they don’t take B₁₂ and many don’t take calcium. Not the healthiest group of vegetarians. We can only conclude that while they were not eating meat, they must have been eating processed junk food.

In addition, the meat eaters were actually quite healthy meat eaters, compared to rest of the country. Their meat consumption was very moderate to low, and they controlled for vegetable consumption, meaning those meat eaters who didn’t consume any fruits and veggies were discarded. Furthermore, they controlled for weight. Weight is an independent risk of cancer, so controlling for weight is appropriate, but one benefit of a plant-based diet, as we saw in the last chapter, is weight loss. So by controlling for weight they are eliminating one way the plant-based diet protects against cancer.

Finally, there was definitely a healthy volunteer effect. We can tell this because as the data goes out longer, the increased cancer rate has diminished. Taking all this into mind, the EPIC-Oxford becomes a true outlier.
Breast Cancer Studies

One of the most feared and common cancers in America is breast cancer. What does the evidence show?

A 2003 meta-analysis of case/control and cohort studies showed that fat and animal protein intake were associated with increased risk for breast cancer. Saturated fat and meat specifically were identified as culprits (Boyd, Stone, et al. 2003). Another study, the Women’s Intervention Nutrition Study (WINS) looked at women who had already been diagnosed with breast cancer. It showed that when these women decreased their fat intake (generally correlated with decreased animal food consumption), they lived longer without relapse (Chlebowski, Blackburn, et al. 2006).

On the other hand, two large-scale studies failed to find a link between diet and breast cancer. The Women’s Health Initiative (WHI) did not show that women who reduced their fat intake reduced their cancer risk (Willett 2010). Comparing WHI and WINS is instructive. The women eating “low fat” in the WHI study were still getting 25 to 28 percent of their daily calories from fat, and there’s reason to believe that the participants underreported their fat consumption. By contrast, the low-fat WINS cohort were getting just 20 percent of their calories from fat. The WHI simply didn’t look at sufficient variation in diet to spot meaningful differences. The group who were supposed to eat a normal diet actually reduced their fat, so the two groups ended up looking too similar to find a difference.

The concept of “sick populations” is helpful here. First proposed by Geoffrey Rose in 1985 (Rose 1985), it posits that we won’t be able to find causation in a trait that is universally present in a population. Take smoking, for example. We know that smoking is highly correlated with lung cancer. Now imagine a population in which everyone smokes. Lung cancer will be common, but won’t afflict everyone. The scariest thing about the epidemic will be its apparent randomness. Scientists will look for genetic susceptibility, for lifestyle factors, for environmental factors, and they may make a few weak correlations here and there. But the smoking (literally) gun will be invisible, since there’s no way to conduct a study that compares smokers to nonsmokers.

Another breast cancer study suffering from the same design problem was the Women’s Healthy Eating and Living (WHEL) study, a random-
ized trial that examined whether women previously diagnosed with early-stage invasive breast cancer who reduced fat and increased fruit and veggie consumption also reduced their risk of additional breast cancer events and early death. The study randomized women into a diet intervention or a control group. The dietary changes had no apparent effect. One obvious problem with the study design was its focus on fat rather than protein. This meant that participants could still eat lots of lean meat that wasn’t accounted for in the analysis. Another problem, albeit a positive one, was that the control group appeared to increase their fruit and vegetable consumption nearly as much as the intervention group.

Additionally, some of the WHEL study participants had cancerous masses greater than one centimeter in diameter and yet were not given an aromatase inhibitor, the standard of care. Recurrence of cancer in these women may therefore have been due to failure to adhere to proper protocol rather than the low-fat diet. Finally, the researchers committed a classic “response bias” error in their reporting. Only 45 percent of study participants in the low-fat group completed their food surveys, yet it was assumed that nonresponders complied with the dietary recommendations to the same extent as responders. Logically, we would expect that people who didn’t comply with the reporting requirements would also be less adherent to the low-fat diet (Pierce, Natarajan, et al. 2007).

In one very large study run by the National Institutes of Health (NIH) and the American Association of Retired Persons (AARP), researchers followed 189,000 postmenopausal women for an average of four years. Those women who ate the most fat had highest likelihood of getting breast cancer. There was a fairly wide range of fat intake, which allowed researchers to see clearly the effect of dietary fat on breast cancer (Thiébaut, Kipnis, et al. 2007). And the EPIC study, which looked at 334,000 women over 12 years, found that diets rich in fiber, especially from vegetable sources, decreased breast cancer risk regardless of menopausal status (Ferrari, Rinaldi, et al. 2013).

One reason to lend more credence to EPIC, the huge Korean study, WINS, and the NIH-AARP study, aside from their sheer size and length of study, is the wide variation in diets. Researchers often divide study populations into quintiles, or fifths, for ease of comparison. Each quintile would contain the same number of participants. Let’s imagine a study of 1,000 people asking if animal protein consumption is related to colon cancer. The data might look like this:
As you can see, the variance in animal protein intake is quite high. Quintile 1 is comprised of vegans, Quintile 2 might be vegetarians or flexitarians who consume meat just a couple times a week, while Quintile 5 consists of heavy meat eaters. If there’s a link between animal protein and colon cancer, this study is likely to find it. Therefore, in our made-up data, we can see a big difference in cases of colon cancer between the first and fifth quintiles. And indeed, the real-life studies that showed a significant relationship between animal protein or saturated fat intake and cancer incidence studied populations similarly varied in their dietary patterns.

Now suppose another study, using exactly the same methodology, chose a population that was less diverse. Pretend, in fact, that this study looked at just the people in Quintiles 3, 4, and 5 of the first study. Now the chart looks like this:

<table>
<thead>
<tr>
<th>Quintile</th>
<th>Number of people</th>
<th>Avg daily animal protein intake (g/day)</th>
<th>Number of cases of colon cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>120</td>
<td>30</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>120</td>
<td>45</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>120</td>
<td>50</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>120</td>
<td>67</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>120</td>
<td>80</td>
<td>5</td>
</tr>
</tbody>
</table>

The 600 people who were in the bottom three quintiles have now been divided into five quintiles. Because the bottom of the range of animal protein intake has been cut off, it now looks like there’s either a slight connection or no connection between protein and cancer. Due to a statistical anomaly, some might even point to the fact that Quintile 4 has less colon cancer than Quintile 3 as proof that steak and butter are protective against
colon cancer! WHI and WHEL are good examples of this type of study. They found no significant differences in outcomes because they examined no significant differences in dietary habits.

The best epidemiologic data comes from the Adventist Health Studies. They were well designed and collected trustworthy data. They were also long-term, prospective studies that looked at large groups of people. They looked at a community that was similar in many ways except for diet, making it easier to see the effects of dietary differences in the absence of other potentially confounding factors. Finally, since many Adventists were completely vegan, while others were vegetarian (eating eggs and dairy), pescetarian (adding fish), and omnivorous, the dietary differences between the quintiles was huge.

One Adventist Health Study found that vegetarians developed significantly less colon, prostate, and bladder cancer than meat eaters. Legumes and fruits were specifically found to be protective against pancreatic cancer (Fraser 1999).

These findings agreed with a couple of extremely large-scale and long-term studies, the Health Professionals Follow-up Study that followed over 37,000 men from 1986 to 2008, and the Nurses’ Health Study, which looked at over 83,000 women from 1980 to 2008. These studies clearly showed that the more red meat and processed meat in the diet, the more cancer. And the cancer death rate was significantly lower in those who ate less meat overall (Pan, Sun, et al. 2012). And these striking findings may have underestimated the harm of meat, as the study appears to have systematically overadjusted for participant weight.

An 11-year study of 1,904 German vegetarians also contradicted the EPIC-Oxford finding that vegetarians didn’t seem protected against cancer. Researchers found that being vegetarian was associated with a significant decrease in cancer deaths, especially those caused by cancers of the GI tract. Overall, being vegetarian reduced risk of death 50 percent in men and 25 percent in women compared to the general population (Chang-Claude, Frentzel-Beyme, et al. 1992).

One of the largest studies ever, another joint effort of NIH-AARP, looked at almost half a million people from 1995 to 2005 (Daniel, Cross, et al. 2011). Researchers found that when red meat was replaced by chicken and fish, the risk of certain cancers decreased by modest but significant amounts. Careful statistical analysis of the data set suggests not that chicken and fish are protective, but rather less carcinogenic than red meat.
So by going to a better source (less saturated fat, less heme iron, more omega-3) it appears that chicken and fish are good. What they didn’t do, because they didn’t have enough of a comparison group, is look at what happens if you substitute meat with beans. Now that would have some interesting results, I would bet.

Please don’t get misled by people who use this data to promote fish and chicken consumption. First of all, there’s a big difference between wild-caught fish and farmed fish. Wild-caught fish have long-chain fatty acids, which may provide a protective effect because the greater concentration of omega-3 decreases the important omega-6/omega-3 ratio, decreasing inflammation. But most of the fish we get in the United States are farmed. They get omega-6 fatty acids from their grain feed, rather than the omega-3s they would get from their natural diet of algae. These omega-6s are not anti-inflammatory and therefore are not protective against cancer (Larsson, Kumlin, et al. 2004). And even wild-caught fish are not an unmitigated health food; even studies that find significant protective effects recommend limiting consumption to two servings per week because of heavy metal, PCB, and dioxin contamination (Mozaffarian and Rimm 2006).

Chicken is no better, and probably considerably worse, than fish. Studies show higher rates of lymphoma in chicken eaters (Rohrmann, Linseisen, et al. 2011). Even EPIC-Oxford showed that for lymphatic cancers, including leukemia, multiple myeloma, and lymphoma, a vegetarian or vegan diet is highly protective (Key, Appleby, et al. 2009b). And while correlation does not equal causation, it is interesting to note that our consumption of chicken has spiked over the past few years, as have our lymphoma rates.

Let’s look at one more meta-analysis, a 2012 study that specifically looked at cardiovascular disease mortality and cancer incidence in vegetarians compared to the general population. When you look at the study design, you can see several factors that would tend to bias the study toward showing no benefit to vegetarianism. It included several studies suffering from the “healthy volunteer” effect, in which the control groups were healthier than the general population. Also, some of the vegetarians included in the analysis were actually “semi-vegetarian.” And it also included the EPIC-Oxford data. Despite all these strikes against, the meta-analysis concluded that vegetarians get roughly 18% less cancer than meat eaters (Huang, Yang, et al. 2012).
Research on Other Specific Cancers

So far we’ve been looking at the big picture around diet and cancer, paying specific attention to breast cancer. Now I’d like to summarize some of the epidemiologic research on other cancers.

Pancreatic Cancer

One study followed 190,000 residents of Hawaii for seven years. Named the Multiethnic Cohort Study, it tried to eliminate genetic variability as a factor by including men and women of African American, Japanese, Latino, Native Hawaiian, and Caucasian origin. By study’s end, 468 subjects had developed pancreatic cancer. Researchers found that those who consumed the most meat, and specifically processed meat, were 50 percent more likely to develop pancreatic cancer than those who ate the least. Researchers noted that they could not find correlations between pancreatic cancer and saturated fat levels in meat and dairy, which suggests that the animal protein specifically is the problem. They hypothesized that the way the meat was cooked may have increased its carcinogenic properties (Nöthlings, Wilkens, et al. 2005).

Other Cancers of the GI Tract

Epidemiologic evidence from the EPIC study of over half a million Europeans showed that eating more veggies and fiber reduced the risk of stomach and colon cancer, while red and processed meat increased the chances of developing those cancers (González 2006a; Bingham, Day, et al. 2003). EPIC also found that consuming dietary fiber reduced the risk of colon cancer, especially on the left side. If the people eating the least fiber would double their intake, they would reduce their colon cancer risk by 40 percent. This finding highlights one of the biggest problems with our society’s protein addiction: it makes us avoid fiber. Combining a high-fiber diet with the added benefits of reducing or eliminating meat is powerful medicine indeed.

An Australian study that followed 37,000 people for nine years showed that as red meat and processed meat consumption went up, so did rates of rectal cancer (English, MacInnis, et al. 2004). And the NIH-AARP study that looked at half a million men and women between the ages of 50 and 71 showed that high red meat and processed meat consumption was signif-
icantly associated with cancers of the colon, esophagus, and liver (Cross, Leitzmann, et al. 2007).

**Renal Cell Cancer**

That same NIH-AARP study showed the quintile highest in fiber, fruit, and veggies had a 20 percent lower risk of developing renal cell cancer. The foods that made the biggest difference were cruciferous veggies, legumes, and whole grains (Daniel, Park, et al. 2013). Another large study (following half a million people for an average of nine years) showed that meat intake is related to renal cell cancer (Daniel, Cross, et al. 2012). An analysis of the Health Professionals Follow-up Study showed that men who ate more fruits and veggies had substantially lower incidence of developing renal cell cancer (Lee, Giovannucci, et al. 2006). And a 2007 meta-analysis of multiple studies confirmed the association between meat and renal cell cancer. This study implicated poultry as well (Faramawi, Johnson, et al. 2007).

**Bladder Cancer**

Cruciferous veggies significantly decrease uro-epithelial cancers like bladder cancer, due to sulforaphane, a chemical found in cauliflower and other veggies (Michaud, Spiegelman, et al. 1999). In the Health Professionals Follow-up there is a clear decreased risk in developing bladder cancer in the people eating the most broccoli and cauliflower. A full 40 percent reduction in risk from the highest consumer to the lowest.

**Lung Cancer**

The NIH-AARP study also found that eating large quantities of red meat and processed meat increased the risk of lung cancer by 16 percent (Cross, Leitzmann, et al. 2007). The EPIC study likewise found a reduction in lung cancer risk from increased fruit consumption (González 2006a).

**Epidemiologic Advice**

When I share my dietary views on Facebook, the most common argument made by meat eaters who don’t want to hear the bad news is this: “Correla-
tion doesn’t equal causation.” Which translates to, “I don’t want to accept the fact that thousands of published studies following tens of millions of people for dozens of years have found clear and dramatic linkages between eating animal protein and developing cancer because you can’t prove a direct causal link.” To which I can only reply, using my best Facebook manners, “SMH.”* By that criterion of proof, we can’t prove that cigarettes are harmful. We can’t even prove that shooting someone with a gun can kill them because sometimes people get shot and live.

If you’re addicted to an animal-rich diet and don’t want to change, that’s no business of mine. If you don’t care about your long-term health, again, that’s your decision. But if you want to eat in a way that gives you a great quantity and quality of life, please don’t dismiss the very best evidence we have with a lazy appeal to 50-cent words.

In 2004, a hugely important article, “Diet, Nutrition and the Prevention of Cancer,” was published in the journal *Public Health Nutrition* (Key, Schatzkin, et al. 2004). Two of the authors, Timothy Key and Naomi Allen, were involved in many of the studies we’ve looked at. Lending further credence to the article was coauthor Walter Willett, professor of epidemiology and nutrition at the prestigious Harvard School of Public Health. The article summarized everything known about the links between diet and cancer at that time.

From the article: “Among the diet-related factors, overweight/obesity convincingly increases the risks of several common cancers. After tobacco, overweight/obesity appears to be the most important avoidable cause of cancer in populations with Western patterns of cancer incidence. Among non-smoking individuals in these populations, avoidance of overweight is the most important strategy for cancer prevention.”

As we’ve already seen, a plant-based diet helps people lose weight. Even if it didn’t accomplish anything else, that’s reason enough to adopt one. Getting to a healthy weight is probably the most important thing you can do to prevent cancer.

The World Cancer Research Fund, with the American Institute for Cancer Research, has been publishing updated reports on what’s known about diet and cancer for decades. Their most recent report, written by a panel chaired by Michael Marmot of University College, London, came out in 2007. I give extra credit to this report, titled “Food, Nutrition, Physi-

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* Shaking my head.
Cancer: Fleshing Out the Protein Connection

The process was to use a systematic approach to examine all the relevant evidence using predetermined criteria, and assemble an international group of experts who, having brought their own knowledge to bear and having debated their disagreements, arrived at judgments as to what this evidence means. Both parts were crucial: the systematic review and, dare I say, the wisdom of the experts.

The report was based on three years of study and debate, in which the expert panel reviewed close to one thousand papers. The findings that were released were considered unanimous among those experts despite the fact that they had disagreed over many of the smaller details. Marmot summarizes, “None of our recommendations is based on ‘could be’ conclusions. All are based on judgments that evidence was definite or probable.” This is about as complete a scientific review as possible. You don’t need to believe me, but I don’t know how anybody can doubt such an intensive review by the world’s experts. I am sure many will, though.

The report clarified two controversial issues. First, the cancer rate has been increasing. It’s not simply a factor of better reporting or earlier detection. Second, only a small percentage of cancer is genetic. Most cases can be prevented by lifestyle and other environmental improvements.

Ready for the diet and lifestyle recommendations of the most comprehensive review of the literature by an unbiased panel of the best-regarded experts in the world? Here goes:

1. Be as lean as possible, within the normal range of body weight. [Aim for the lower end of normal body mass index (BMI). In this book, we have seen that a plant-based diet is the best way to maintain low body weight.]
2. Be physically active as part of everyday life.

3. Limit consumption of energy-dense foods. Avoid sugary foods. [In other words, eat foods low in fat and high in fiber and water content. Foods low in fat and high in fiber are plant foods, not animal proteins.]

4. Eat mostly foods of plant origins. [The personal recommendation within this public health goal is to have at least five servings of fruits and veggies each day, and include pulses and unprocessed cereals with every meal.]

5. Limit intake of red meat and avoid processed meat. [The public health goal within the recommendation is to consume less than 300 grams (\( \frac{2}{3} \) of a pound) per week. Being completely vegetarian does reduce cancer risk, but we can’t rule out the possibility that the difference is due to other aspects of a healthy lifestyle.]


7. Limit consumption of salt. Avoid moldy cereals (grains) or pulses (legumes).

8. Aim to meet nutritional needs through diet alone. [That is, without dietary supplements.]

The panel found convincing evidence that fruits and vegetables decrease risk of cancer of the mouth, pharynx, esophagus, lung, stomach, colon, and rectum. Eating more plants probably reduces cancer of the pancreas, breast, bladder, and possibly in liver, ovary, uterine and prostate.

Since the report was published in 2007, additional information has come to light that add even more weight and specificity to the panel’s recommendations. A 2013 study applied the first six of the recommendations to one of the EPIC databases (and added breastfeeding for women) and compared those who adhered to them closely with those who didn’t. The results were staggering: people who answered “mostly yes” to those recommendations had a 34 percent lower risk of dying in the twelve-year period of the study than those who answered “mostly no.” They also had a significantly lower risk of developing cancer or coronary artery disease (CAD) (Vergnaud, Romaguera, et al. 2013).

That the plant-based diet is protective against both cancer and heart
disease makes sense, as the panel’s recommendations are very similar to American Heart Association’s (AHA) advice for achieving ideal cardiac health in its Strategic Impact 2020 plan. To prevent CAD, the plan recommended the following:

1. Get active.
2. Eat better: more fish, veggies, and fiber and less saturated fat and added sugar.
3. Manage blood pressure.
4. Lose weight.
5. Control cholesterol.
6. Reduce blood sugar.
7. Stop smoking.

Another 2013 study looked at cancer incidence in people based on their adherence to the AHA guidelines. The researchers looked at data from the Atherosclerosis Risk in Communities (ARIC) study of over 13,000 people who had been observed for up to 17 years for signs of heart disease. It turned out that those who followed at least five of the recommendations had a 50 percent decreased risk of cancer compared to those who followed none of them (Rasmussen-Torvik, Shay, et al. 2013). And the relationship held in the middle groups as well, with the study authors reporting “a significant, graded, inverse association” between the number of heart healthy behaviors and combined cancer incidence, with the exception of nonmelanoma skin cancers. So, the better the weight, the less the high blood pressure, the better your sugars, the more likely you avoid cancer. Best way to do all those things? Cut back on animal protein and increase fruits and veggies.

One more thing. I don’t want you leaving this section entertaining even the wisp of a thought that you can get the benefits of eating plants by taking supplements. In a very carefully designed and run case/control study of premenopausal women with breast cancer, researchers found that vegetable intake was strongly and inversely related with the cancer, but when they included supplements or consumption of component parts in the analysis, the relationship was much weaker. The whole vegetable, not the extracted or synthesized nutrient components, proved protec-
The researchers weren’t sure why whole plant foods were superior to supplements of vitamins C and E, folic acid, individual carotenoids, and dietary fiber, but hypothesized that those ingredients of plants may have worked together synergistically to reduce breast cancer risk. Also, they surmised, “other unmeasured factors” in whole plant foods may also fight cancer. So don’t be fooled by your neighbor in a pyramid marketing scheme, offering you a pill that has all the vegetable and fruit you need. That is just silly.

**Summing Up**

So what are the conclusions we can responsibly draw from all the research I’ve shared in this chapter? Have we “proved” that a vegan diet can prevent or cure all cancers? No, we haven’t. But we’ve done serious damage to the lazy and irresponsible dismissal of evidence on the grounds that “correlation doesn’t equal causation.”

We’ve seen that populations that eat less meat tend to have less cancer. Adventist studies show that vegans are more protected than vegetarians and moderate meat eaters. We know that vegans have lower levels IGF1 and PhIP, and we know people with high IGF1 are predisposed to a variety of cancers. Ornish and Blackburn have shown us that a low-fat vegan diet can control prostate cancer, not just by altering our hormonal balance but also changing our very chromosomes. Have we proved that we can cure or prevent prostate or other cancers by going vegan? Not yet. But where there is this much smoke, there has got to be a fire, and this one is a barn burner.