

Cancer Control – The New Protocol (CTP9.6)
Basic Principles, Strategies, and Interventions

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Chapter 1 – Cancer: A Systemic Disease

Cancer is systemic meaning that it affects the whole body. At the earliest stages of a tumor's existence, years before it is first commonly detected, a cancerous tumor has been sending out cancer cells throughout the body, most (but not all) of which collect back at the same site since the site has been "prepared" suitably by the initial tumor. Even for the smallest detected tumors, this has been going on for several years before the tumor has been detected. This means it is never the case that "they got it all."

For example, breast cancer has been ongoing for at probably at least 8 years before it can be detected by any of today's technologies. So your cancer has been systemic in your body for quite some time. From this recognition, just as an aside, the notion that you can check your lymph nodes to see if it has spread is utter nonsense. This is one reason why published research shows that cancer found in the lymph nodes (and removed where found) has no impact on the patient's mortality.

Virtually all cancer patients have some cells in some lymph node whether detected or not, so it is not a distinguishing feature among cancer patients; all have it. Hence its detection (or not) in so-called sentinel nodes is a useless procedure. Removing the lymph nodes causes enormous pain and grief, all for naught.

This is just another example of how cancer treatment is not rational in so many respects. Given cancer's systemic nature, the first implication is that cancer must be tackled on various fronts to successfully eliminate or at least control it. The preoccupation with modern oncology fixated on the tumor in place, slicing it out with surgery, killing it with radiation, and poisoning it with chemotherapy is a woefully inadequate and ill-conceived strategy. A whole body approach is not just helpful but in fact is mandatory.

In other words, since the cancer has been ongoing for many years, stop thinking it is all there in the tumor found. Since it takes many years for a tumor to be detected, when they find a metastatic site a few months after the first site has been found, as is often the case, this tells you that the "new" cancer in fact was there for years, just slightly behind the found tumor in terms of time to detection; it was there all along.

A tumor does not just jump off and skip to another part of the body. A tumor does not break off and like an iceberg float to some part of the body like the brain. You have had all this cancer all along. Thus you want to treat cancer as a systemic or whole body disease — not just some focused spot where it was first found.

For example, it was reported in a case study [*NEJM*, 2003; 348(6):567-8] in the *New England Journal of Medicine*, one of the world's most respected medical journals, the case of a 47-year-old woman who had a melanoma lesion removed 16 years before her death (not related to cancer), and she seemed cancer-free up to the time of her death when she died of a brain hemorrhage.

Her kidneys were donated and transplanted to two people. However about 18 months after the kidney transplant, the first organ recipient was found to have melanoma, and then a few months later, the second organ recipient was also found to have melanoma. What does this tell us? The woman organ donor was suppressing any clinical manifestation of cancer during her life, but the recipients' immune system, suppressed in order to keep the body from rejecting the organ transplant, allowed the latent cancer in her

donated organs to erupt. As Dr. Ralph Moss concluded regarding these cases, “The health of the immune system was critical ... a fully functioning immune system prevented cancer from reasserting itself in the original donor. The *lack* of an unhindered immune system also resulted in the reappearance of cancer in the unfortunate recipients.”

So in short, her cured but deadly melanoma skin cancer was in fact systemically hanging around in all her organs. Once the immune suppression was initiated for the organ transplant people, the cancer quickly asserted itself. For this reason, if you are now cancer-free after some oncology treatment, you still need to stay on some maintenance protocol to prevent “recurrence” of a cancer that never went fully away. You need to read this book.

Chapter 2— Fundamentals: Tackling the Existing Cancer Cell Itself

One principle approach is to tackle cancer at its cellular *environmental* level. We will be describing how to do this with supplements in the next chapters. Another important strategy tackles the cancer cell itself. To accomplish this, we must recognize that once a cell has become malignant, it uses sugar and carbohydrates for fuel. Carbohydrates become sugar as soon as we eat them so sugar and carbohydrates are essentially the same thing from cancer's point of view. So it becomes imperative to reduce your sugar and carbohydrate consumption to as low a level as possible to starve cancer cells. For example, a 160 pound person should consume no more than about 60 grams of carbohydrates (and/or sugars) per day. Less is better.

It has long been widely known that cancer cells survive with a different mechanism than normal cells. They survive by feeding off of glucose for their survival. Thus sugar is *the* requirement for cancer cells. Recall that carbohydrates also are converted into sugar by the body quite directly and promptly. So sugar and carbohydrates are cancer's friend and requirement and your enemy.

Just what is a carbohydrate? If it is not a protein such as chicken, meat, fish, or eggs, or it is not a fat from butter or cream or cheese or oil, or it is not a green leafy vegetable like spinach, then it is most likely a carbohydrate. Carbohydrates are all those wonderful sugars and starches from bread, pastry, fruits, most vegetables, candy, ice cream, pasta, potatoes, cereal, corn, rice, and so much more. Nearly all fruits and very many vegetables (e.g., potatoes, corn, peas, etc.) along with most grains are all carbohydrates.

Further it does not matter whether it is a simple sugar or complex starch, whether it has a low or high glycemic value — the carbohydrates become sugar in the body. For each 4-5 carbohydrates you eat, it is the equivalent of one teaspoon of sugar. The arithmetic goes something like this: there are about 4 calories per carb. Since sugar has 16 calories per teaspoon, then the teaspoon of sugar is about 4 carbs. There are some round-off issues involved but these are the approximate equivalences. Actually we do not need any carbohydrates at all in our diet (although they taste good, and many are otherwise helpful) since we can make glucose from parts of protein and fat.

So if you currently have a diagnosed cancer, in order to starve your cancer, you want a very low carbohydrate and sugar intake. For a 160 pound adult, or about 73 kilograms, you would ideally consume no more than 240 calories in carbohydrates and sugars which is the same as about 60 grams of carbohydrates (the back of food packages will normally list the amount of carbohydrates as so many grams per serving). Actually much less is better. For many, this will be very difficult to do. Simply try to maximize your protein and fat intake, along with those foods low in sugar and carbohydrates, as much as you humanly can. It could be a matter of survival. To borrow from the advertising world, it doesn't taste as good as surviving feels.

Chapter 3 – Strategies: Two Fundamentally Different Cancer Interventions

As we begin to develop specific interventions, it will be important at this point to identify another important principle, to make a critical distinction. Many cancer patients will mix and match many “good ideas” as if anything can be combined with anything. This is categorically not true. As my colleague Dr. Stephen Martin pointed out, there are two different strategies that cannot be usefully combined.

The first is an immune system boosting strategy. In this first case, we may take supplements, particularly antioxidants for example, that boost our immune system, and with its boost, one’s own system’s ability to fight cancer is initiated or accentuated. Then it is up to our enhanced immune system to undertake cancer eradication. This can be an effective strategy.

The second strategy is called a cytotoxic one. In this instance, one takes supplements known to kill cancer cells, or halt cancer cell progress, or aid in turning cancer cells back into normal cells [yes, this is possible for some of the cancer cells]. It is not directed toward enlisting the immune system with its own “killer” cells. This too can be effective.

But the two cannot be successfully combined. Immune boosting requires certain immune factors to be advanced or up-regulated while cytotoxic efforts require the very same factors to be down-regulated, to be eliminated. The cytotoxic strategy typically leads to cancer cell death, called apoptosis, by causing the cell to self-destruct by way of an oxidizing process, to some degree just as chemo-therapeutic agents do.

This destruction process however can be halted by antioxidants which allow the cancer cell to survive. This is not the desired outcome. So when you read the literature and/or visit the web and add one or another supplement, each of which may be useful to boost your immunity or alternatively to caused cancer cell death, the two may be cancelling each other out.

Putting together a useful protocol is much harder than it appears to the naked eye. I have tried to accommodate these conflicting prospects with the protocols below. Overall they are cytotoxic – killing cancer cells – and mega-anti-oxidant strategies will undercut their effectiveness. I will have much more to say about this issue in later sections.

To identify what I have chosen, let me state that I have created a cytotoxic cancer cell protocol. I have worked with both immune boosting and cytotoxic strategies with many clients for many years. From this experience, I have found the following: if you do not have cancer as yet, you should undertake an immunity boosting strategy. So here you take antioxidants, eat blueberries, and so forth.

If you have had overt cancer, the cytotoxic strategy is wiser since we need to diminish your cancer burden. Note that I did not say eliminate it. Neither this protocol nor any chemo and radiation will ever do that. If we can just push it back to either undetectable levels – or – just push it back to a stable level, we will have accomplished all that is ever possible. One client I have, with prostate cancer, has remained stable for about 10 years so far. My personal opinion is that he will be good for at least another 10. Actually I think he will like most men die *with* prostate cancer, not *from* prostate cancer.

The point of this discussion is that wise protocol construction requires a lot of background information. Finally let me make the matter even more complicated. My former colleague Dr. Jerome Block (former head of Medical Oncology at the Harbor-UCLA Medical Center and a former director of research for the National Cancer Institute as just part of his sterling credentials) and I published a paper about the role of antioxidants in chemotherapy treatments. Here is an important paragraph from that paper:

*The view that antioxidants' preventative role in free-radical formation may be an antagonist to the goal of free-radical formation by cytotoxic drugs is further challenged by other recent research. In both tissue culture and a variety of animal model systems, **substances such as vitamin E, carotenoids, and ascorbic acid**, viewed classically as antioxidants, **have often been shown to be antioxidants in certain systems and pro-oxidants in others, depending on the system studied and the dosages explored**; in differing studies, one can demonstrate both antioxidant and pro-oxidant intracellular effects of a wide variety of putative antioxidants.⁸⁶⁻⁹⁷ In some instances, the pro-oxidant effects of these agents will influence intracellular levels of glutathione, often converting reduced glutathione (GSH) to oxidized glutathione (GSSH) and removing thereby important intracellular defense mechanisms against cytotoxic agents such as commonly used chemotherapy. In other instances intracellular oxidation products of antioxidants⁹⁶ often generated by interaction with H₂O₂^{70,98} enhance drug cytotoxicity or promote apoptosis and will have important oxidative DNA damaging effects in tumor systems and normal tissues; these effects may be concentration-dependent.⁹³ Such results indicate that the notion of antioxidant versus pro-oxidant drug action must be viewed with respect to their actions being concentration-dependent,^{28,74,90,93} sequence-dependent,⁹⁸ and/or may vary depending on the target markers measured.^{54,56} Experimental data with ascorbic acid (vitamin C) typifies this dichotomy of antitumor effects with antioxidants. While in some systems ascorbic acid will antagonize apoptosis and some antiproliferative drug effects,¹⁴⁷⁻¹⁵⁰ when combined with the clinically useful anticancer drug arsenic trioxide (As₂O₃), ascorbic acid enhances antitumor effects.*

In short, what we demonstrate from the peer-reviewed medical literature is that sometimes antioxidants act as oxidants! It all depends on context. I invite you to read the entire paper – just google my name and my colleague Dr. Block's name ["steven evans jerome block"] and about our last 10 publications come up. Click on the ninth one entitled, *Reprint of article published in the Journal of the American Nutraceutical Association, Vol. 4 No. 1, 2001, 11-19, A Review of Recent Results Addressing the Potential Interactions of Antioxidants with Cancer Drug Therapy*].

In developing this protocol, these are just some of the issues that needed to be considered.

Chapter 4 – Metastasis: Your Primary Enemy and Protocol A

Once cells become fully malignant, they will typically not reverse back. Although we will later address how these may then be directly killed, the key strategy is to (a) prevent any more malignant cells from forming, (b) prevent any metastasis of the cancer to other parts that will actually kill you such as to the brain or liver, and © kill already malignant cells. In particular, if you halt the tumor process where it is, assuming you do not yet have a fatal condition such as a tumor in your brain that is stopping your critical functions, then the strategy of “holding the fort” can enable you to live out your life in this chronic but acceptable state.

In short, a cancer patient almost always dies of metastasis, not the original cancer itself. Stopping metastasis is the key. If you already have metastasis, do not despair. If you can halt further cancer as we will hope to accomplish in the Protocols below as well as halt further metastasis with the protocol in this section, you may at least extend your survival significantly, perhaps indefinitely. If you already have metastasis, it is not possible to predict the benefit since it depends on the extent of the metastasis. Nonetheless I believe that patients will benefit at least to some important measure by following this protocol.

Now most differentiated malignant cancer cells have a very short life span. They may be able to divide multiple times, but eventually they die out on their own. The danger to the cancer patient is from the cancer stem cells which do not die out. Please note carefully: The KEY to controlling cancer is to control the growth of *cancer stem cells* since these cells are the ones that keep producing the differentiated malignant cells that characterize all cancers.

Killing the *already* differentiated cancer cells that characterize breast, prostate, brain, lung, etc. cancers is great, but it is a relatively short-term victory. One must kill the *cancer stem cells* which unfortunately are extremely resistant to chemo drugs and radiation. In fact, over time, chemo drugs may and do make these stem cells even more malignant (producing more cancer cells). This results in additional tumors over time which may also be more aggressive than before.

When adult stem cells replicate, they produce two *different* cells. This is quite unlike the cell division of most other cells which produce two *identical* copies of the original cell. Stem cells, on the other hand, both self-renew by producing one copy of the mother stem cell and another copy of a cell called a multipotent progenitor cell (MPC).

This MPC cell can form a diversity of different cell types in a particular tissue. If the stem cells or progenitor cells become mutated, they form self-replicating cancer cells. Standard chemotherapy often kills the "children" of these stem cells, but typically not the stem cells themselves. So naturally, the cancer will return over time. Killing the cancer stem cells is obviously a priority. It is worth noting that both radiation and chemotherapy are extremely ineffective in killing cancer stem cells.

A second crucial discovery is that cancer uses blood clots to metastasize to other sites. So as you would expect, people who were on blood thinners (to eliminate blood clots) such as Coumadin had no metastasis of their cancer when these people were studied. You will not need to be on any prescription blood thinners. We will itemize a simple over-the-counter product that will greatly reduce blood clots and

hence reduce metastasis. Stopping the metastatic process as well as halting new cancer creation is the goal of the new protocols. There is an enormous amount of evidence that this can be achieved in a very economical and straightforward manner.

In the book *The Hidden Story of Cancer*, there is a quote from the provost of the M.D. Anderson Cancer Center, Raymond DuBois, M.D., Ph.D.: “*Keep in mind that almost no one dies of primary cancer ... a patient almost always dies of metastasis.*” This is indeed true. Most patients do not stop to think about the fact that a lump, wherever, is in itself not necessarily dangerous unless it is in the middle of the brain, in the liver, etc. A lump just sitting there in the breast or prostate, for example, is not doing much harm at all.

However when the cancer spreads to the brain for example, we have a much greater problem. It might spread throughout the bones, causing pain, a disruption of metabolic processes, etc. — all of which can be deadly. So stopping metastasis is of utmost importance. We have some very significant tools to do this.

We are now in the position of combining the principles above to begin the construction of our first protocol. This first part is applicable to all cancers.

1. Meriva [high-powdered curcumin]

An extremely effective anti-inflammatory is curcumin, the main spice that makes up curry and which comes from the turmeric plant. This has been known for decades, if not centuries. The main problem is that curcumin is hardly bio-available. This has now been remedied. A new product called Meriva is simply curcumin bound up at the molecular level with an oil (phosphatidylcholine) which transports the curcumin into the cell. So Meriva will be an important component in our protocol.

It is found at the Swanson Vitamin web site [www.swansonvitamins.com] as a product called Meriva, product #SWU493, a 500 mg capsule, \$9.99 for 60 capsules. One should take at least one twice a day, with some food. If you have had more advanced cancer, take two capsules twice a day. I should note that I also found a similar product at Life Extension Foundation (www.lef.org). They have a 400 mg capsule of Super Bio-curcumin, item #00407, \$35 for 60 capsules so it is also a perfectly good product but since it is 350% more for a capsule that is only 80% as much curcumin, that is the only reason I have identified the Swanson one.

Slightly less convenient, but actually technically more powerful, is a combination product of a high-level curcumin product along with extra bioperine which multiplies its power. To follow this alternate, select Swanson’s SWH084, a high-level curcumin product, plus SWU308, a bioperine product. Take these two **together** (this is absolutely essential) twice a day but double this dose – so 2 of each – twice a day if you had a more advanced cancer or have ongoing cancer.

2. Isoleucine

There is yet more. In 2007, Japanese scientists found that the oral administration of the amino acid isoleucine completely prevented the metastasis of colon cancer cells into the liver. Once colon cancer

metastasizes to the liver, the cancer is considered untreatable. They found that the amino acid isoleucine inhibits the synthesis of VEGF, the major promoter of blood vessel growth into tumors. You can access the abstract by typing in the word Pubmed and the number 17409434 into a google search. The first "hit" will be the abstract published in *Cancer Research*, 67(7): 3263-8, Ap 1, 2007.

Isoleucine does not kill cancer cells directly. But it does inhibit the *synthesis* of VEGF, thereby inhibiting metastasis. This is a fairly monumental observation. Although the study referenced above used a colon cancer model, the ability of isoleucine to inhibit VEGF synthesis almost certainly applies to all tissues, including brain tumors.

Isoleucine is an essential amino acid. This means it must be obtained from the diet. The concentration of isoleucine in foods varies tremendously. Egg whites contain the highest concentration of isoleucine. Isoleucine can also be purchased in bulk. The site musclematrix.com sells a 1000 gram container for \$50. I suggest you take 5 grams of isoleucine twice a day in juice. The 1000 grams would last 100 days (hence \$15 per month, not counting shipping). If you order enough for more than 2+ months (which is likely needed), then your shipping costs are divided out over the larger purchase.

Note: You can use a scale to measure out 5 grams. I found a somewhat inexpensive one at www.saveonscales.com which has the ProScale 600 Luxe for \$24.90 plus shipping (uses 2 AAA bat.) However one of our clients has found that 5 grams is precisely a level measured teaspoon so this might eliminate your need for a scale.

3. Cimetidine

In 1994, a study ["Short-course cimetidine and survival with colorectal cancer." *Lancet*, 1994, Dec. 24-31; 344(8939-8940): 1768-9.] demonstrated that just 7 days of treatment with cimetidine [i.e., *Tagamet*, the over-the-counter drug for heart-burn] decreased the 3-yr. colorectal cancer mortality from 41% to 7%. In another study ["Cimetidine increases survival of colorectal cancer patients with high levels of sialyl Lewis-X and Lewis-A epitope expression on tumor cells. *Br J Can* 2002 (86) 161-7], patients with aggressive colon cancer had a 84% 10-year survival rate when treated with cimetidine for one year after surgery compared to a 23% 10-yr. survival for patients not treated. This is quite remarkable.

As we will note shortly, this may well apply to many other cancers as well. However I would bet you dollars to donuts oncologists have not communicated these results to their patients assuming they even know about them. The key to the usefulness of cimetidine is whether the tumor expresses what are called Lewis antigens, which in the study reported, 75% of the tumors did.

If the tumor does not express these antigens, cimetidine conferred *no advantage at all*. In the study reported, when the tumors expressing Lewis antigens were viewed, cimetidine-treated patients had a survival rate of 90.7% while the Lewis-tumors untreated with cimetidine had a 33.7% survival rate. Wow. Recognize this: Other cancers such as breast and pancreatic cancer also express Lewis antigens! The dose level for the studies was 800 mg, either all at once at night or divided into two 400 mg doses.

In short, if you actually know whether your tumor expressed Lewis antigens because they checked at the time, act accordingly. If you don't, it might be very wise to undertake a regimen of cimetidine. Note

that a one-time-only 7 day treatment yielded huge outcomes, so if you do not know your tumor status, it would likely be very wise to take a regimen.

I would suggest a 7-10 day regimen just in case there is more benefit to accrue with a slightly longer time. You can then stop the regimen altogether after 10 days. It will either help or not. No further cimetidine is needed. Cimetidine can upset your stomach, so after 7 days, if you are feeling upset, then just stop.

Note that many companies make a generic cimetidine product [such as Walgreens] which is the same as the more expensive brand-name product called Tagamet. Getting the more inexpensive form is perfectly fine. As noted, the dose level for the studies was 800 mg, either all at once at night before bed time or divided into two 400 mg doses, one before bed time and the other 2+ hours after eating. This short and relatively inexpensive one-time-only supplement (for 7-10 days use) would seem like a very worthwhile venture to take, on the chance that your cancer happened to express those Lewis antigens.

4. Super CitriMax for Cancer Control

A scientific paper demonstrated that the enzyme ATP citrate lyase (ACL) was *fundamentally* important in cancer cell growth. If this enzyme is inhibited, cancer cell growth stops. PERIOD. This is so important, I have inserted a reprint of the Abstract of a key article, published in *Cancer Cell*. 2005 Oct 8(4):311-21 discussing this point.

“ATP citrate lyase inhibition can suppress tumor cell growth.”

Hatzivassiliou G, Zhao F, Bauer DE, Andreadis C, Shaw AN, Dhanak D, Hingorani SR, Tuveson DA, Thompson CB. Department of Cancer Biology, Abramson Family Cancer Research Institute, University of Pennsylvania, Philadelphia, PA 19104, USA.

Many tumors display a high rate of glucose utilization, as evidenced by 18-F-2-deoxyglucose PET imaging. One potential advantage of catabolizing glucose through glycolysis at a rate that exceeds bioenergetic need is that the growing cell can redirect the excess glycolytic end product pyruvate toward lipid synthesis. Such de novo lipid synthesis is necessary for membrane production and lipid-based posttranslational modification of proteins. A key enzyme linking glucose metabolism to lipid synthesis is ATP citrate lyase (ACL), which catalyzes the conversion of citrate to cytosolic acetyl-CoA. ACL inhibition by RNAi or the chemical inhibitor SB-204990 limits in vitro proliferation and survival of tumor cells displaying aerobic glycolysis. The same treatments also reduce in vivo tumor growth and induce differentiation.

What we learn from the above paper is that when a cancer cell grows, it expands two-fold. It must make new lipids and cholesterol for continued membrane synthesis. Lipids and cholesterol derived from diet are useless. The lipids/cholesterol **must** be synthesized in the cell. Inhibitors of ACL do not induce apoptosis in cancer cells. To the contrary, they stop cancer cells from growing while forcing them to differentiate into normal cells. Many cancer cells cannot differentiate (back) into normal cells, but some can.

Now we review the following report published in *Oncogene*. 2005 Sep 15;24(41):6314-22.

“ATP citrate lyase is an important component of cell growth and transformation.”

Bauer DE, Hatzivassiliou G, Zhao F, Andreadis C, Thompson CB. Department of Cancer Biology, Abramson Family Cancer Research Institute, University of Pennsylvania School of Medicine, Philadelphia, PA 19104, USA.

Cell proliferation requires a constant supply of lipids and lipid precursors to fuel membrane biogenesis and protein modification. Cytokine stimulation of hematopoietic cells directly stimulates glucose utilization in excess of bioenergetic demand, resulting in a shift from oxidative to glycolytic metabolism. A potential benefit of this form of metabolism is the channeling of glucose into biosynthetic pathways. Here we report that glucose supports de novo lipid synthesis in growing hematopoietic cells in a manner regulated by cytokine availability and the PI 3 K/Akt signaling pathway. The net conversion of glucose to lipid is dependent on the ability of cells to produce cytosolic acetyl CoA from mitochondria-derived citrate through the action of ATP citrate lyase (ACL). Stable knockdown of ACL leads to a significant impairment of glucose-dependent lipid synthesis and an elevation of mitochondrial membrane potential. Cells with ACL knockdown display decreased cytokine-stimulated cell proliferation. In contrast, these cells resist cell death induced by either cytokine or glucose withdrawal. However, ACL knockdown significantly impairs Akt-mediated tumorigenesis in vivo. These data suggest that enzymes involved in the conversion of glucose to lipid may be targets for the treatment of pathologic cell growth.

In summary, ACL inhibitors stop cancer cell growth, but they do not necessarily kill the cells. In fact, they induce many cancer cells to develop into normal cells. We suspect the appearance of newly differentiated cells in a tumor mass is due to the action of ACL inhibitors on resident cancer stem cells. The lack of ACL activity may actually correct the defective cancer stem cells and allow them to produce normal cells again.

The scientists used experimental drugs to inhibit this enzyme. Naturally, the un-availability of these drugs doesn't help patients at all. However my brilliant colleague Dr. Stephen Martin began looking around for a natural inhibitor of this enzyme. And he found one — a really great, inexpensive and easily available one. It is being used as a weight loss supplement sold over-the-counter on the Internet. By the way, it is a poor weight loss product so don't even worry that this effect will arise.

What Dr. Martin found was that ATP citrate lyase is naturally inhibited by hydroxycitric acid. This molecule is an analog of citrate which blocks ACL activity. And, lo and behold, the product Super CitriMax, is a highly concentrated form of hydroxycitric acid. *Super CitriMax* is sold as a weight loss product since it increases weight loss by blocking lipid synthesis. As I noted above, do not worry too much about this aspect since its effectiveness for weight loss is actually extremely low. I did not personally see any weight loss from its use whatsoever.

The key is that *Super CitriMax* is a very bioavailable form of hydroxycitric acid. In addition, the following study fortunately shows that *Super CitriMax* is extremely safe for human consumption [the Abstract below tells us *Super CitriMax* is a safe product to take — you can skip its details if you wish].

Published in *Food Chem Toxicol.* 2004 Sep 42(9):1513-29.: Soni MG, Burdock GA, Preuss HG, Stohs SJ, Ohia SE, Bagchi D. “Safety assessment of (-)-hydroxycitric acid and *Super CitriMax*, a novel calcium/potassium salt.”

*(-)-Hydroxycitric acid (HCA) is a principle constituent (10-30%) of the dried fruit rind of *Garcinia cambogia*, a plant native to Southeastern Asia. The dried rind has been used for centuries throughout Southeast Asia as a food preservative, flavoring agent and carminative. Extensive experimental studies show that HCA inhibits fat synthesis and reduces food intake. The*

objective of this review is to systematically review the available safety/toxicity literature on HCA to determine its safety in-use. The primary mechanism of action of HCA appears to be related to its ability to act as a competitive inhibitor of the enzyme ATP-citrate lyase, which catalyzes the conversion of citrate and coenzyme A to oxaloacetate and acetyl coenzyme A (acetyl-CoA), primary building blocks of fatty acid and cholesterol synthesis. Super CitriMax, a novel calcium/potassium-HCA extract (HCA-SX), is considerably more soluble and bioavailable than calcium-based HCA ingredients. Acute oral toxicity studies in animals demonstrate that CitriMax (50% HCA as calcium salt) has a low acute oral toxicity. In a subchronic study in rats, the gavage administration of HCA-SX at doses up to 2500 mg/kg/day for a period of 90 days caused a significant decrease in body weight and reduction in feed consumption without any adverse effects. The structure, mechanism of action, long history of use of HCA and other toxicity studies indicate that HCA-SX is unlikely to cause reproductive or developmental effects. HCA-SX was not mutagenic in the presence or absence of metabolic activation in Ames genotoxicity assays in strains TA98 and TA102. HCA-SX-induced increases in number of revertants in other strains (TA100 and TA1535 in the absence of metabolic activation and in strain TA1537 in the presence of metabolic activation) but these were not considered as biologically indicative of a mutagenic effect. In several, placebo-controlled, double-blind trials employing up to 2800 mg/day HCA, no treatment-related adverse effects were reported. There is sufficient qualitative and quantitative scientific evidence, including animal and human data suggesting that intake of HCA at levels up to 2800 mg/day is safe for human consumption.

For a second article on product safety, see *Mutat Res.* 2005 Nov 11;579(1-2):149-62. Epub 2005 Aug 1. This article concludes that ... *Both Ca and K act as buffers in pH homeostasis. HCA-SX has been shown to increase serotonin availability, reduce appetite, increase fat oxidation, improve blood lipid levels, reduce body weight, and modulate a number of obesity regulatory genes without affecting the mitochondrial and nuclear proteins required for normal biochemical and physiological functions.*”

There are different CitriMax products on the market. Purchase *ONLY* the trademarked Super CitriMax brand. It is manufactured by one company which in turn sells it to other supplement manufacturers. Try to purchase a product that doesn't contain other additional ingredients. The dose is 3 grams a day, taken as 1.5 grams twice a day. This is the minimum dose. Most Super CitriMax capsules contain 700 mg of hydroxycitrate concentrate from an Asian fruit. So you will need 4 of these capsules as a minimum dose per day. Given its power and usefulness, if you have cancer, consider 6 capsules per day.

This product could be a stand-alone treatment for many leukemias and some cancers. As an individual product, it would stop cancer cell growth and induce differentiation. In particular, I found on the Swanson site [www.swansonvitamins.com or call 800-437-4148] a suitable product. It is product #SWD051, \$9.49 for 120 capsules with each capsule delivering 750 mg of Super CitriMax. Take at least 2 capsules twice a day; a bottle will last a month, but if possible, take 6 a day so a bottle will last 20 days.

5. Nattokinases

There is one more very important anti-metastatic supplement you should take. It has been recognized that it is frequently a blood clot that causes cancer metastasis to seed into tissue at a distance from the original tumor. It was reported as early as 1958 that the number of deaths from cancer is dramatically

decreased by over 80% if blood clots are eliminated [*Archives of Pathology*, vol 66, Oct 1958]. In a study in 1997, the incidence of cancer deaths was one-eighth the expected number for patients on permanent anti-coagulant therapy. The study covered 1569 patient-years and there was no cancer deaths by metastasis in the entire group [see Horn, R. *Health and Survival in the 21st Century*, Chapter 13, 1997, HarperCollins: Australia]. There is yet more evidence for the substantial elimination of metastasis through the reduction of blood clots.

Now we have a simple and safe supplement that will substantially reduce your propensity for blood clots. It is made from fermented natto, a vegetable, skimming off the enzyme nattokinase which has quite significant anti-clotting properties as well as being able to dissolve formed clots. Natto has the consistency of boiled okra and smells worse than boiled mustard greens. There is fortunately a supplement that is easy to take. One product is called Nattokinase, available from Swanson as product #SWU419, \$8.99 for 30 capsules with each capsule rated at 2000 fibrinolytic units or FU's— a good rating. Take 1 capsule twice a day (every 8 hours); if you can afford it, take 3 a day, 8 hours apart.

Summary of Key Recommendations: Protocol A

1. Meriva	Swanson's SWU493	\$9.99 for 60 caps	2 capsule twice a day
2. Isoleucine	musclematrix.com	\$50 for 1000 grams	5 grams twice a day
3. Cimetidine (Tagamet)	Walgreens Brand or other		800 mg a day for 7-10 days
4. Super CitraMax	Swanson's SWD051	\$9.49 for 120 caps	3 capsules twice daily
5. Nattokinase	Swanson's SWU419	\$8.99 for 30 caps	2-3 capsules daily

Follow a diet extremely low in sugars/carbohydrates [also see diet considerations in Chapter 8 below].

The summary of the information analyzed so far is that if you have ***any*** cancer, you can (1) keep it in place, (2) slowly change some of the cancer cells back to normal, and (3) stop the metastatic process which will prevent the cancer from progressing. This alone is a remarkable advance. In the next sections, we shall address added cancer therapies. The Protocol A above is applicable for all cancers.

Chapter 5 – The 2nd Level Cancer Protocol: Protocol B

Next, we shall begin the second part of the protocol with eight key supplements. These supplements are applicable to all watchful waiting situations as well as other overt ongoing cancers. If you are in remission from any serious cancer, you should also follow this protocol. Chapter 7 discusses more details about which protocols might be right for you. The only exceptions in this protocol are the supplements DIM and pomegranate which only apply to estrogen-related cancers such as prostate cancer, breast cancer, ovarian cancer, and colon cancer.

To motivate you and support the choices presented, we begin with a detailed discussion of why the literature supports these choices. Do not skip this part. You need to appreciate the depth of support for these choices or they will just be arbitrary supplements that otherwise make no sense. Don't worry if not every part of this discussion is clear. Read to get the gist of the argument.

Note: We shall use the following citation form. If an eight number is given in brackets, to get the citation, enter this number in a google search along with the word PUBMED. The first hit that comes up will be the actual Abstract for the article quoted so hit that choice and you will have the Abstract along with the citation for the Abstract.

1. Sodium Selenite

The following is an excellent review article on the relationship between selenium and cancer development [15387717]. This 2004 article is focused on the cofactor role of selenium in the enzymatic activity of various enzymes. Most of these enzymes are involved in anti-oxidant activities. In the last three years, it has become clear that selenium itself can profoundly reduce glutathione levels in cancer cells and induce oxidative stress. This results in apoptosis (cell death).

A Brief Review

We obtain selenium from plants which extract the selenium from the soil. The soil content of selenium differs across the world. Selenium is highly deficient in the soils of certain areas of China. Europe and the southern USA states are also relatively selenium deficient. Certain plants highly concentrate selenium from the soil. They include garlic, broccoli, leeks and onions. Some of these plants may accumulate 1,000 to 10,000 micrograms of selenium per gram of plant weight. These organic selenium compounds take many different forms. In this discussion, we are ONLY focusing on the form of selenium called sodium selenite. If you already express frank cancer, then this product is MUCH more toxic to cancer cells than the organic, plant derived forms which is the one of choice for a preventive role. Sodium selenite can be purchased as a supplement and is 80% bioavailable.

Some have been concerned that a daily dose of selenium in excess of 200 micrograms/day was toxic. This is incorrect. The NOAEL (i.e., No Adverse Effect Level) of selenium per day is 819+/-126 micrograms a day. Interestingly, a 12 month study found that doses of 1600 micrograms of selenium a day produced no adverse symptoms. The RDA for selenium is only 55 micrograms/day. The immune system and the thyroid gland require higher concentrations of selenium for maximal activity.

There appears to be a strong relationship between selenium deficiency and the incidence of different cancers. Many long term studies on this topic are presently being conducted. We suspect that the preventative role of selenium in these cancers is related to the activation of anti-oxidant enzymes. Our focus here is on addressing already-present cancers.

When sodium selenite enters the cell, it interacts with two molecules of glutathione producing hydrogen selenide (H₂Se). Hydrogen selenide is used as a building block for selenoproteins. However, excess hydrogen selenide activates the synthesis of superoxide anion, a powerful oxidant. The following study shows that sodium selenite strongly depletes glutathione in the cell while stimulating the production of superoxide anion [11585738].

Some of the data reported in this article is quite amazing. In tests on prostate cancer cells, the authors found:

1. 3 microM sodium selenite induced apoptosis in almost 100% of the cells.
2. 2 microM sodium selenite reduced the level of active glutathione in the cells to near zero.

Sodium selenite is clearly a powerful apoptosis inducing agent. It accomplishes this goal by depleting cellular glutathione while concurrently increasing the level of superoxide anion. Oxidative stress induces the activation of some genes and the inactivation of others. See the following articles [10699758, 10331077]. Oxidative stress protects cells against apoptosis by the activation of HSP-70 and NF-kappaB synthesis. [See for example 15706092].

We can block the activation of HSP-70 and NF-kappaB with other natural supplements, thus increasing the apoptosis inducing efficacy of oxidative stress. This is the very nature of product synergy. In our selenium literature research, we found some incredible new studies. Recall that an activated Cox-2 enzyme produces prostaglandins such as PGE₂. PGE₂ inhibits the immune response and promotes cancer cell growth and survival.

As the following study shows, selenium inhibits the synthesis of the Cox-2 enzyme by activating the AMPK pathway. Selenium activates this pathway via oxidative stress. Further, selenium is a MORE powerful apoptosis inducing agent than various chemo drugs which rapidly induce resistance [17047069].

The AMPK pathway is a MASTER metabolic sensor. If glucose levels decrease, this enzyme shuts down all unnecessary biochemical pathways on a short term basis in an effort to preserve ATP levels. This means reduced protein synthesis, fatty acid synthesis, isoprenoid synthesis, cholesterol synthesis, lipolysis, fatty acid oxidation, etc. The following review article has excellent diagrams and can be read online [12960015].

The prolonged activation of the AMPK pathway will induce apoptosis in cancer cells. Interestingly, the AMPK enzyme also inhibits the activity of the mTOR pro-growth pathway. This pathway is also inhibited by glutamine [See for example 16613876].

AMPK activates a number of tumor genes which inhibit cellular proliferation. One of the most important is p53, the master tumor suppressor in the body. AMPK phosphorylates and activates the p53 molecule. [See the following 15866171].

The following short article is a mini-review of the previous article. It has a nice diagram showing that p53 is activated by many forms of oxidative stress. AMPK is in good company [16054073].

More Review

High levels of selenium induce oxidative stress in cancer cells. This oxidative stress is a direct effect of the selenium molecule. Two molecules of glutathione are neutralized by every molecule of selenite. This new selenium molecule DIRECTLY activates the production of superoxide anion, a ROS molecule. Oxidative stress activates AMPK and AMPK phosphorylates and activates p53. Selenium is known to activate p53 by oxidative stress, but the mechanisms were not known. They are now [15252149, 17172431, 16891474, 15274301].

What Else Do We Know about Selenium?

Sodium selenite induces endoplasmic reticulum stress. This results in apoptosis. The saturated fat palmitate does the same thing. See [16204082]. Selenium activates AMPK which inhibits fatty acid synthesis. This may be the pathway by which selenium induces endoplasmic reticulum stress [See 17283163].

The following article is truly exciting. It shows that selenium, acting as an anti-inflammatory agent, activates the synthesis of the prostaglandin 15d-PGJ2. This prostaglandin is a powerful anti-inflammatory agent. Among other roles, it activates the genetic factor PPARgamma, a major inhibitor of inflammatory hormones (and HIV synthesis). This article also demonstrates, for the first time, that 15d-PGJ2 is an endogenous or natural inhibitor of NF-kappaB activation. This explains why selenium is such a powerful anti-cancer agent. It promotes oxidative stress while simultaneously inhibiting NF-kappaB activity. Chemo drugs CANNOT do this. They activate NF-kappaB activity. Selenium requires an active Cox-1, but not Cox-2 enzyme in order to stimulate the synthesis of 15d-PGJ2 [See 17439952].

2. Melatonin

The "sleep" hormone melatonin inhibits the synthesis of the 5-lipoxygenase enzyme via its activation of nuclear receptors. The nuclear factor RZR/ROR is the receptor for melatonin in normal and cancer cells. The activation of this nuclear receptor inhibits the synthesis of the 5-lipoxygenase gene, thereby promoting apoptosis in specific cancers.

Specifically, the activation of the ROR receptor promotes apoptosis in androgen-independent prostate cancer cells. The ROR nuclear receptor, activated by melatonin, also counteracts the growth promoting effects of 5-lox gene products.

You may be aware that different cancers are not biochemically alike. Some cancers depend on specific growth pathways that play little or no role in the growth of other cancers. This is especially true of prostate cancer. Prostate cancer typically over expresses the enzyme 5-lipoxygenase (5-lox). This enzyme, like the Cox-2 enzyme, uses the omega-6 fatty acid arachidonic acid as a substrate to produce growth factors such as 5-HETE. 5-HETE is a known activator of multiple growth pathways.

The following study shows that the inhibition of 5-lox activity triggers a MASSIVE apoptosis in prostate cancer cells. Clearly, the 5-lox enzyme and its product 5-HETE plays a FUNDAMENTAL role in prostate cancer growth and survival [9789062].

5-lipoxygenase inhibitors induce death by an enhanced membrane expression of TRAIL receptors on the membranes of cancer cells. In addition, these inhibitors also activate the JNK death pathway in cancer cells. The combination of sodium selenite and specific 5-lipoxygenase inhibitors such as melatonin may be an effective method for controlling prostate cancer development.

Melatonin has been shown to be non-toxic in doses up to 150 milligrams a day. It is a perfect anti-estrogen dependent or independent cancer drug. This means it can be used against ovarian and cervical cancer as well as other cancers such as prostate, breast, etc., with no fear of toxicity. Melatonin as a hormone is effective in very low concentrations, and it's almost impossible for a cancer cell to become resistant to its anti-cell growth effects.

There is another feature about melatonin worth noting. Just as the drug Tamoxifen inhibits estrogen binding to its receptor so that cells are not encouraged to grow, melatonin does the same thing. Tamoxifen also kills estrogen-insensitive breast cancer cells by a pathway called JNK. This pathway is activated by virtually all chemotherapeutic drugs and radiation therapies. Melatonin does the same thing. Also melatonin inactivates the aromatase pathway just as aromatase inhibitors do (although Tamoxifen doesn't) [15683469]. So all in all, melatonin is extremely valuable, and your author would personally choose it over Tamoxifen any day, which by the way, increases your chances of endometrial cancer while melatonin does not.

3. Flaxseed

Several studies have indicated the huge impact that flaxseed can have as an ongoing food supplement. For example, Canadian scientists enrolled 50 women newly diagnosed with breast cancer to study the effects of flaxseed on breast cancer progression [results were similarly obtained for prostate cancer too]. [15897583].

One group received a daily muffin for 30 days with each muffin containing *50 mg of flaxseed lignans* while patients in the second group ate ordinary muffins. Actual tumor biopsies were performed prior to and after the 30 day period. The results showed that the flaxseed was extremely efficient, in these low doses, in *killing the breast cancer cells!* The results were dramatic. The growth index in the cancer cells was reduced 34.2%, while the level of programmed cell death increased 30.7%. Women expressing the HER2 (c-erbB2) metastatic oncogene saw its expression decrease a dramatic 71%. All in just 30 days. **These are extraordinary biopsy-verified results!**

As we noted there is the direct analogue to prostate cancer. In addition, there is reason to believe flaxseed will be applicable to other cancers as well. We shall repeat below our own developed muffin recipe incorporating this research, for convenience.

Muffin Recipe: In one bowl, mix the following:

1½ cups of flour [preferably unbleached]

12 Tablespoons of flaxseed [use the flaxseed brand mentioned below] — about ¾ cup

½ cup of packed brown sugar

1 Tablespoon of baking powder

¼ teaspoon of salt [this is needed]

1 teaspoon of cinnamon, and ¼ teaspoon nutmeg

In a separate bowl, mix 1 large egg, 1 cup of milk [preferably skimmed], & ¼ cup oil [preferably olive]

Combine wet ingredients into dry and mix.
Spray a muffin or cup cake pan with an oil spray
Spoon evenly into 8 cupcake bins
Bake at 400 degrees for about 18-19 minutes

We stated above that somewhat similar results with prostate cancer. A study from Duke University Medical Center divided 161 men, who were scheduled to have their prostate removed 30 days later, into four groups [See *Journal of Clinical Oncology*, 2007 ASCO Annual Meeting Proceedings Part I. Vol 25, No. 18S (June 20 Supplement), 2007: 1510].

One took 30 grams of flaxseed for 30 days, one had a low-fat diet with the flaxseed, a third had just the diet, and the fourth had nothing. Examining the removed tumors 30 days later, those taking the flaxseed (irrespective of diet) had significantly slower rate of tumor growth.

Technically flaxseed contains *pre*-lignans. Once eaten, lignans are produced in the intestine by the stomach's bacteria acting on the pre-lignans. What has been discovered is that these lignans (a) inhibit the activation of the AKT enzyme [to be explained in a moment] and (b) increase the expression of a protein called p53 by blocking its degradation in the cell. So although it does not incorporate all the subtlety, we can shorten the science by saying that "flaxseed inhibits AKT and increases p53."

AKT is the critical switch for the development of all cancers!! If AKT were **totally** inhibited, cancer would cease to exist. AKT inactivates the GSK-3 pathway which means it **halts** a crucial enzyme that (a) promotes many cancer cell pro-death pathways and (b) promotes chromosome stability. So again, putting AKT to bed halts some of its destructive actions. AKT also facilitates blood supply to tumors, which we want to halt. The role of p53 is also hugely significant since it is the major cell death protein in the cell. P53 permits the cell to be killed by the body, rather than remain an immortal cancer cell or remain active for ongoing viral replication. So cancer works to increase AKT and decrease p53 while flaxseed precisely reverses these actions.

Moreover flaxseed inhibits estrogen-induced growth in cancer cells and inhibits VEGF, a key growth factor for cancer cells. And the list just continues. **The end point is that there is no dietary product more powerful in the direct killing of all cancer types than flaxseed.**

Note you *must* use flaxseed with a high standardized lignan level. You **cannot** buy just any flaxseed at the local store. It is extremely unlikely to have the proper amount and type of (SDG) lignans needed. The product must be reliably standardized for its SDG lignan count. Work up from a tablespoon to 2 tablespoons a day or higher (build up slowly). If you have cancer, work up to 6 tablespoons per day. For a good product, go to Swanson Vitamin Company's site, at www.swansonvitamins.com and look for product #SP029, Spectrum Essentials Flaxseed, a 14 ounce bag of ground, 100% organic, cold-milled flaxseed made by Spectrum Essentials with at least 98 mg of SDG lignans (the desired type) per 2 tablespoons. It is currently \$4.34 per bag and at 2 tablespoons per day, it will last a month.

4. DIM

The addition of diindolylmethane (DIM) may be very helpful to enable the body to maintain a properly balanced level of estrogen. Estrogen-related cancers such as breast, ovarian, and cervical cancers, as well

as prostate cancer and colon cancer may well have imbalanced levels of estrogen as part of their causative factors. DIM is known to restore proper estrogen balance.

DIM is a crucial ingredient in broccoli, cabbage, kale, cauliflower, etc. that makes these vegetables so good for us as cancer fighters. Eating them has long been associated with a reduced risk of various cancers. When these vegetables are eaten, a compound in the vegetables breaks down into DIM in the stomach. Many researchers believe that DIM is the most critical compound delivering the anti-cancer benefit from these vegetables.

As far back as 1978, researchers first discovered that “DIM had an inhibitory effect on mammary tumor formation.”¹ [i.e., DIM can contribute to breast cancer prevention]. A 1996 study provided evidence that “DIM induces apoptosis [cell death] in human cancer cells.”² [i.e., DIM kills human cancer cells]. In 1998, another study showed that DIM “exerts a dose-dependent cytotoxicity [cell destruction] on human colon adenocarcinoma ..cells.”³ [i.e., DIM kills colon cancer cells]. In 2001, a study showed “potent cytostatic effects of DIM in endometrial cancer cells.”⁴ [i.e., DIM kills endometrial cancer cells].

Another 2001 study indicated that DIM inhibited key steps in the metastatic process in ... estrogen positive and ... negative cell lines... results suggest DIM may have therapeutic effects against breast tumor growth and tumor spread.”⁵ [i.e., DIM works against breast cancer growth and metastasis in estrogen positive and negative cell lines]. A 2002 study showed “DIM can induce apoptosis in breast cancer cells independent of estrogen receptor status.”⁶ [i.e., DIM kills breast cancer cells of both receptor types].

In 2003, researchers at the University of California, Berkeley found that androgen-dependent cancer... treated with a DIM solution grew 70% less than... untreated [cells].”⁷ [i.e., DIM inhibited the growth of prostate-cancer cells]. In 2003, these researchers also noted that “DIM inhibits the action of DHT, the primary androgen involved in prostate cancer.”⁷ [i.e., DIM inhibits the primary androgen which plays a key role in prostate cancer].

One of the main actions of DIM is to enhance so-called “good estrogen” and reduce so-called “bad” estrogen which both men and women express. For this reason, the impact of DIM is likely to be on these cancers: colon, prostate, breast, ovarian, cervical, endometrial, and ovarian cancer.

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5. Pomegranate

In addition to DIM as noted above, pomegranate extract has been shown to be very helpful in a prostate cancer protocol and likely estrogen-related cancers such as breast cancer, ovarian cancer, and colon cancer. Specifically, research continues to accumulate on the positive role of pomegranate as a preventive and reducer of prostate cancer. A recent study [*J of Agri Food Chem*, Vol. 55, 7732-7737, Sept 19, 2007] by researchers at UCLA showed that a key ingredient in pomegranate significantly inhibits the growth of prostate cancer cells. What's more, it concentrates to a very high degree in prostate tissue. In addition, they found that it significantly inhibited the growth of tumors grafted onto mice with impaired immune function. There is a great deal more research supporting pomegranate's role in terms of prostate cancer and now some for breast cancer as well.. Both DIM and pomegranate supplements are worth adding if you are developing an anti-prostate cancer regimen, already have prostate cancer, and/or are on a watchful waiting regimen. As noted, the research, although not as extensive, appears to apply to estrogen-related cancers such as breast cancer.

6. Vitamin D3

Activated vitamin D3 and NF-kappaB have a yin-yang relationship. They both inhibit the activity of the other. Vitamin D3 is made in the skin and converted to 25-VD3 in the liver. This form of vitamin D has little biological activity. The enzyme 25-VD3 1-alpha-hydroxylase exists in almost all cells of the body and converts 25-VD3 to its active form 1,25-VD3. NF-kappaB inhibits the activity of this enzyme, thereby blocking the synthesis of active vitamin D3 in the body [15243130].

The NF-kappaB subunit p65 (RelA) forms a complex with the vitamin D receptor (VDR) in the nucleus thereby preventing the receptor from activating the appropriate vitamin D response genes. This study also shows that the pro-inflammatory hormone TNF, via its activation of NF-kappaB, blocks vitamin D responsiveness [15211579, 11877332].

Conversely, activated vitamin D3 (1,25VD3) inhibits NF-kappaB. In fact, this form of vitamin D3 may be THE universal modulator of NF-kappaB activation. It is important to recall that inhibiting NF-kappaB is a major goal of the cytotoxic protocol as a cancer inhibiting strategy. Also note that the immune system needs NF-kappaB enhanced – which again is why you can't combine an immune-enhancing strategy with a cytotoxic strategy.

Consider the following studies:

Fibroblasts are the major cell type in the body. Vitamin D inhibits the activation of NF-kappaB in these cells [17298882]. If fibroblasts lack the vitamin D receptor, the activity of NF-kappaB increases [16507601]. The natural inhibitor of the classical (RelA) NF-kappaB activation pathway is a protein called I-kappa-B-alpha. Vitamin D increases the synthesis of this inhibitor [16455676, 15372276]. The alternative (RelB) NF-kappaB pathway is also inhibited by vitamin D. In this case, the vitamin D receptor binds the gene for RelB and inhibits its activity [16239345, 14507914]. Herein lays the problem.

There is very little vitamin D in our diets, few people take vitamin D supplements, and most importantly we are inadequately exposed to the UV sunlight rays that can make vitamin D in our skin. When you combine the lack of basic vitamin D in our bodies and the chronic inflammation, mediated by NF-kappaB, that chronically plagues us during our lifetimes, you are left with a serious physiological deficiency of activated vitamin D3.

Additional Discussion

The enzyme MN-Superoxide Dismutase protects the mitochondria from an accumulation of superoxide oxygen radicals. In the absence of this enzyme, the mitochondria will be EXTREMELY vulnerable to oxidative damage. Now, if you can inhibit MN-SOD activity AND block glutathione synthesis/mitochondrial uptake, the mitochondria are going to be damaged beyond repair. The genetic factor RelB, a member of the NF-kappaB family, activates the gene for MN-SOD in the mitochondria [16261162]. Vitamin D3, the activated form, *inhibits* the synthesis of RelB, thereby *depriving* the cancer cells of MN-SOD functioning. This is another of the ways activated vitamin D3 kills cancer cells [17604335].

7. IP-6

In a recent study at the University of Colorado Cancer Center, more specific mechanisms through which IP6 is effective were identified. Scientists had previously found that IP6 increased the activity of genes that control proteins in human prostate cancer cells lacking functional p53, the gene that provides much cancer protection. They sought to determine whether this increased activity plays a role in IP6's anti-tumor effect. They found that the two genes activated by IP6, p21 and p27, play a critical role in mediating the anti-cancer effectiveness of IP6. Following activation of the genes by IP6, they were able to halt tumor growth and promote the appropriate death of cells in a process known as apoptosis. This study was published in *Cancer Research*.

This study follows on the heels of other research from the University of Colorado that evaluated the efficacy of IP6 against prostate tumor growth and progression. Prostate cancer was induced in mice given either water containing IP6 or plain water. The researchers found that IP6 inhibited the progression of the cancer cells and strongly reduced the incidence of adenocarcinoma. This is of high significance because 95% of prostate cancers are adenocarcinomas, meaning the cancer has developed in the lining or inner surface of the gland.

The incidences of well-differentiated and poorly differentiated adenocarcinomas in the group fed IP6 were reduced by 44% and 62% respectively. Analysis of the prostate tissue showed a 3.5 fold increase in malignant cell death. This highly significant finding established for the first time that oral IP6 suppresses prostate tumor growth and progression at the stage of abnormal or uncontrolled growth.

IP6 is non-toxic and produces no side effect: IP6 is a compound found in beans, whole grains, nuts, seeds, rice and wheat bran, corn, and sesame. One-half cup of whole kernel corn contains a whopping 650 mg of IP6. It is composed of an inositol sugar molecule (one of the B vitamins), with six phosphate groups attached. Because it is a sugar molecule, it has a pleasant, sweet taste. Because it is from food and not from a drug laboratory, IP6 has no toxic effects in the body even at high doses.

Although the IP6 compound was identified many years ago, it wasn't until the late 1980's that its ability to control the rate of abnormal cell division was discovered. A scientist from the University of Maryland, Dr. Abulkalam Shamsuddin, found that IP6 was able to halt well-developed cancers. While most cancer research centered on killing cancer cells, Dr. Shamsuddin proved that IP6 could normalize the sugar production of cancerous cells, thereby altering their gene expression toward a more healthful state. This

discovery has major implications because cancer cells that are well behaved have far less negative consequences to health.

IP6 works in many ways against cancer in general: Whether from food or from therapeutic supplemental doses, IP6 works against cancer in several ways. Its ability to act as an intracellular messenger means that it is integral in many cellular activities.

Normalizing the rate of cell growth: When cancer cells lose their control mechanisms, rapid and uncontrolled division of malignant cells is often the result. As IP6 repairs the gene mutations and reestablishes control within the cells, their rate of division is slowed

Enhancing natural killer cells: Natural killer cells are white blood cells that help protect against infected or cancerous cells. Research has shown that the higher the amount of natural killer cell activity, the lower the incidence of some cancers. A healthy human produces 500 to 1000 cancer cells daily that need to be identified and disposed of by the body. Natural killer cells and natural cell programming result in the vast majority of these cells being destroyed and removed. However, when the body is under stress, including the stress produced by lack of sleep, natural killer cell population is compromised. When the body is under the ultimate stress of being forced to face a diagnosis of cancer and the terrifying toxic treatments that go with such a diagnosis, the natural killer cell population can be reduced at the time it is most needed. IP6 has been documented and proven to increase natural killer cells at such times.

Normalizing cell physiology: Because IP6 is able to restore aspects of normality to the cells, it is able to modulate how a cancer cell expresses itself and how threatening it will be. Experiments have shown that IP6 is able to normalize several aspects of cell physiology in spite of the fact that cancer cells have altered DNA. It was demonstrated in the above noted study that IP6 is able to alter gene expression to restore normality. The more a cell can return to its normal state, the more it loses its malignant characteristics.

Increasing tumor suppressor p53 gene activity: DNA contains tumor suppressor genes that inhibit pathways and processes that allow cells to become malignant. The p53 gene acts as a control to prevent genetically damaged or cancerous cells from growing and propagating. If the p53 gene becomes damaged or compromised, cancers can establish themselves more easily. IP6 has been shown to greatly increase the amount of p53 gene activity, up to seventeen times. When augmented by IP6, even the toxic treatments offered by traditional medicine become more effective due to its ability to increase p53 gene activity.

Enhancing apoptosis: Apoptosis is the orderly programmed process by which cells naturally die off. The process of apoptosis results in the removal of individual cells without affecting the surrounding cells. It is part of the growth and maintenance of healthy tissues. Cancerous cells do not want to die and are resistive to apoptosis. This is one mechanism of tumor formation. IP6 has been shown to restore cancerous cells to normality to the point where they are able to follow through with their programmed death.

Affecting angiogenesis: Angiogenesis is the process by which tumors set up their own blood supply, to assure themselves of getting the nutrients necessary to grow. Once this blood supply is set up, tumor growth spirals as more growth leads to more blood supply creation. IP6 has been shown to inhibit this process, resulting in the starvation of cancer cells.

Powerfully chelating heavy metal: Tumor cells use iron as a primary growth factor. According to researchers at Wake Forest University, iron chelators are of value in the treatment of cancer since they act

by depleting iron and limiting tumor growth. IP6 binds with iron and escorts it from the body. Because IP6 is naturally found in all human cells, it has the ability to get inside tumor cells and remove their iron.

Inhibiting metastasis: IP6 inhibits cancer cell migration and invasion by preventing the adhesion of these cells to extra-cellular matrix proteins. This limited adhesion is very important following surgery and biopsy, as these procedures can cause cancer cells to become dislodged. One reason so many breast cancer patients have lymph nodes containing cancer cells is that the squeezing of the breast by the mammography used in diagnosis can dislodge cancer cells which then migrate to the lymph nodes.

Inhibiting inflammation: One key indicator used in predicting the survival rate for a cancer patient is the level of systemic inflammation. Inflammation is like a fire going on in the body that will destroy it if not stopped. IP6 helps stop inflammation through chemical messages that halt the production of pro-inflammatory cytokines.

Inhibiting free radical production: IP6 acts as an antioxidant in the body. Antioxidants are known to protect against disease states and aging in general. Antioxidants bind with free radicals rendering them harmless. In their free state, these radicals can damage DNA and leave cells susceptible to mutations that can result in the production of cancerous cells. IP6 is significantly more potent as an antioxidant than green tea.

IP6 influences genes to halt colon cancer: In another recent study, more information about the molecular mechanisms through which IP6 acts was revealed. Scientists at the Medical University of Silesia in Poland investigated the influence of IP6 on the expression of genes encoding subunits of NFkappaB and of its inhibitor, IkappaBalpha, in human colorectal cancer cells. The results of their study suggest that IP6 primarily influences p65 and IkappaBalpha expression in colon cancer cells. The amount of activity seen was dependent on the IP6 levels and the amount of time it had been present in the cells.

Populations with diets high in IP6 have low incidences of cancers: Studies have shown that tumor regression takes place in people using 8 grams of IP6 daily for three to four weeks. This is the amount that would be found in 12 ounces of whole kernel corn. One of his studies found that after two weeks the tumors of mice treated three times a week with IP6 were 96 percent smaller than the tumors of mice that did not receive IP6. Populations with diets high in IP6 have lower incidences of cancers of the breast, colon and prostate. Laboratory experiments with IP6 have been reproduced and extended by scientists around the world, reconfirming these amazing findings. This study is from *Acta Poloniae Pharmaceutica*, November through December, 2008.

IP6 is more than a cancer treatment: In addition to the anti-cancer benefits of IP6, research is revealing its benefits in treating diabetes, depression, osteoporosis, heart disease, and kidney stones. It has recently been shown to help Parkinson's patients because of its ability to chelate excess iron and thereby reduce oxidative stress that results in neuronal degradation

Treating cancer with IP6: For anyone choosing to use IP6 as a cancer cure or a preventative there are some things to know. IP6 is present in all human cells, and considered quite safe to use. However, IP6 obtained from food is bound to protein. Before it can be absorbed by the body, it must be freed from this protein by the enzyme phytase that is present in food and naturally in the intestinal tract. The power of the phytase enzyme is damaging to IP6 and renders much of it inactive and therefore less effective when

obtained in this form. Pure IP6 from a supplement is not bound to protein and is easily absorbed intact and able to provide its complete medicinal properties.

Much of the scientific research completed with IP-6 has been done by Abulkalam Shamsuddin, a scientist at the University of Maryland School of Medicine. Several of Dr. Shamsuddin's animal and human cancer cell line studies have shown promising results with colon, prostate, liver and breast cancer. Epidemio-logical studies show that people who eat lots of foods that contain IP-6 have lower incidences of cancer of the breast, colon and prostate. Foods that are significant sources of IP-6 include rice, sesame, beans, legumes, corn and cereals [none of which are recommended if you already have cancer due to their high carb content].

Precautions: IP-6 reduces platelet activity; therefore, people with low blood cell counts, or who are taking aspirin or other blood thinning medications, may want to avoid using IP-6. Supplemental forms of IP-6 may also bind with calcium, magnesium, copper, iron and zinc and should therefore not be taken at the same time with food. This also suggests the use of a daily multi-vitamin to prevent any deficiencies.

8. Green Tea

It is known that cancer cells have signaling and cycle control pathways that let them grow out of control. Green tea was shown to inhibit cell cycle progression [Cancer Letters, 2000, June; 23(6):695-9.], while another study showed green tea causes cell cycle deregulation and death of cancer cells [*Arch BioChem Biophys* 2000 April 15;376(2):338-46¹].

Japanese researchers reported [“A receptor for green tea polyphenol EGCG,” *Nature Structural and Molecular Biology*, vol 11, 2004 using green tea concentrations, equal to about just three cups, and found that one of its polyphenols binds to the cells cancer uses to help spread it throughout the body and prevents them from prompting tumor growth. Mayo Clinic researchers reported [“VEGF receptor phosphorylation status and apoptosis is modulated by a green tea component, EGCG, in B-cell CLL.” *Blood*, March, 2004] this green tea polyphenol applied to the most common type of leukemia (B-cell CLL) inhibited new blood vessel growth needed for cancer to spread. In 8 of 10 B-Cell CLL samples, it prompted cancer cell death..

A study [*Free Radical Biology and Medicine*, April 2004;36(7):829-37] has revealed that some of the recently popular and highly praised antioxidants such as those found in red wine and grapes were not as effective cancer preventatives as tea in slowing down the progression of prostate cancer. Participants had prostate cancer and were pending prostate removal. The men drank 5 cups of black or green tea, or soda each day for 5 days prior to their surgery. The study included cutting out a piece of each man's prostate, and results revealed a decrease in the growth rate of cancer cells in the men who had consumed the tea but not the soda.

A UCLA team uncovered more about how green tea counteracts cancer [*Clinical Cancer Research* 2005, Feb 15]. Numerous prior studies have shown it induces cancer cell death and inhibits cancer's blood supply to prevent cancers ability to grow and spread. However traditional medicine often ignores such evidence if the specifics of how an ingredient works is not known [although many patented drugs are prescribed even when their mechanism of action is not fully known]. The researchers showed green tea

interrupts a process crucial in allowing cancer to become invasive and spread. As they noted, “green tea extract may keep the cancer cells confined and localized...[&] interrupts the invasive process...”

A news report [*Medical News Today*, April 20, 2005 described a study in 62 men with high-grade intraepithelial neoplasia, a pre-cancerous prostate condition. After a year of green tea polyphenols in 32 men, only 1 developed prostate cancer (3% rate) while 30% of the placebo group developed prostate cancer (9 of the 30 in the placebo group).

In another study [*Cancer Research*, Jan 15; 66(2): 1234-40, 2006], a total of 60 men were recruited with high-grade prostate intra-epithelial neoplasia, and studies have shown that 30% of such men will develop prostate cancer within 1 year of such a diagnosis of this “pre-cancerous” prostate condition. Half were given green tea catechins, and among this group, after a year only 1 developed a tumor versus the 9 expected. For the unlucky placebo group, there were indeed 9 cancers found a year later. PSA values did not change between the two groups. There was also statistically significant improvements in quality of life measures for the treated group. There were no side effects. The amount used in the trial was a net 450 mg per day.

A study [*Cancer Prevention Research* 2(7): 673-82, July 2009.] found high levels of green tea polyphenols yielded a significant reduction in unwanted growth factors associated with prostate cancer along with reduced PSA levels, suggesting possibly a slowing of the progression of the cancer. In the trial, men took the supplement for about 35 days, using 1300 mg of polyphenols daily with significant results at the end of that time. There were no reported side effects.

9. Implementing Protocol B

Here are the specifics in order to fully implement protocol B.

1. Sodium Selenite

Dose: Take 250 micrograms three times a day. Thus 750 mg total per day [this factors in the GSM trial amount].

Source: Go to www.swansonvitamins.com and enter product #TL242. This is TwinLabs 250 mcg tablet, \$5.94 for a 100. At 3 a day, the bottle will last a month.

2. Melatonin

Dose: Take 10 mg a day, at bed time. Ideally mix the melatonin in some whole milk or ½ -and -½ taking it since this will hugely increase its absorption – it is a fat soluble supplement.

Source: Swanson has a product, #SWU305, a 10 mg capsule, \$4.49 for 60 capsules. Take 1 at bed time. A bottle will last two months.

3. Flaxseed

Dose: Begin with 1-2 tablespoons per day and slowly work up to a minimum of 2 tablespoons per day.

Source: At Swanson’s site, look for product #SP029, a 14 ounce bag of ground, 100% organic, cold-milled flaxseed made by Spectrum Essentials with at least 98 mg of SDG lignans (the desired type) per 2 tablespoons. It is \$4.34 per bag and at 2 tablespoons per day, it will last a month.

Note: From the discussion on the mechanism of action of flaxseed given above, we note that if you do not have adequate stomach bacteria because you were on an antibiotic which kills these needed bacteria along with the negative bacteria targeted by the antibiotic, you won't make lignans in your stomach from flaxseed pre-lignans. You need to take a probiotic to replace these good bacteria. If you have taken one or more antibiotic regimens, consider Swanson's probiotic, product #SWL008, \$14.99 for 30 capsules [call 800-437-4148 or visit their web site at www.swansonvitamins.com]. Use up the bottle (one month's supply). There are 60 billion organisms per capsule. For a maintenance level product, Swanson's #SWU079 has 4.2 billion organisms per capsule. It is \$13.59 for 120 capsules (take 2 a day). For maintenance, taking it one month should do, and you might repeat every 6 months.

4. DIM

Dose: Men take 200 mg per day of DIM which is combined in a product with an equal amount of phosphatidylcholine to support bio-availability. Women take 100 mg per day.

Source: Virtually every product uses Indoplex as their form of DIM, which as you will see on the label is actually just 25% DIM. For example, DIM complex from Enzymatic Therapy is \$31.53 for 60 tablets of a 120 mg tablet. But actually, since it is only 25% DIM, it contains 30 mg of actual DIM. If men took 7 per day, the actual monthly cost would be over \$110 plus shipping. Even then, it is not clear that the product does in fact have the proper amount of phosphatidylcholine.

To address the lack of quality products, TRI has offered a DIM trial without charge to participants in the Formula #18 Cancer Risk Reduction Trial [administered by the GSM Corporation] except for just the shipping, packaging, and handling charge, currently \$7.95 per month [billed \$15.90 every other month].

For anyone who is reading this research contribution, we will extend the no-cost trial to anyone reading this book who may wish to enroll. You will be responsible only for the \$7.95 monthly shipping, handling, packaging, etc. charge [times 2 or \$15.90 every two months since we ship two months at a time].

Note that Swanson also carries a DIM product, but their 100 mg capsule (product #SWU119 – 30 capsules for \$5.99) is only 25% DIM so you must take 8 per day (for men). Hence a month's supply is about \$48 per month plus shipping (\$4.95). This would be an acceptable product if they also included an equal amount of phosphatidylcholine, but it does not appear so. In our formulation, men take 2 capsules per day (women take one a day). If you find an alternate DIM product, please do so since we have no special interest in being a supplier of supplements – this exception is just to address special circumstances in the marketplace and make this supplement affordable.

If there is a huge response by the readership of this book, TRI may withdraw its offer since funds are extremely limited.

5. Pomegranate

Dose: Two capsules of standardized pomegranate per day if you have an estrogen-related cancer.

Source: Swanson Vitamin Company's #SWH116 is a standardized product with 70% ellagic acid, \$4.79 for 60 capsules.

6. Vitamin D3

Dose: Take an added 4000 IU per day in oil-based gel caps. The vitamin D3 capsules recommended are ones encapsulated in oil to achieve bio-availability.

Source: Go to www.swansonvitamins.com and enter into the search box the product code CSN027. This is Carlson's 120 capsules, 4000 IU per capsule, which is \$9.79. Take 1 a day. The capsules combine D3 with cod liver oil which is critical for the bio-availability needed.

7. IP-6

Dose: Although further research must be completed before making truly definitive recommendations, we gives the following guidelines: 1-2 g of IP-6 daily for cancer prevention, 6 g daily for people with an established increased cancer risk, and 8 grams to affect a present cancer.

Source: I would suggest you consider going to swansonvitamins.com which carries a container of "IP6 & Inositol" for \$40.14 [product #ET226, which is the product Cell Forte IP-6 & Inositol by Enzymatic Therapy – Swanson has the cheapest price for it I can find, but if you are not ordering anything else from Swanson, then go to Amazon.com which has it for \$40.38 but no shipping charge]. Each "heaping teaspoon" contains 3200 mg of IP6. So take 2 heaping teaspoons per day if you need 6 grams, and take 2.5 heaping teaspoons (8 grams) if you have active cancer. At 2.5 teaspoons per day, a container will last about 24 days. Remember to take on an empty stomach since not only is this needed for absorption but also IP-6 binds with calcium and magnesium so you will lose that in the food you eat if taken together.

Some tentative research suggests that the impact of IP-6 is substantially enhanced by additional inositol. There should be 27 mg of inositol for each 100 mg of IP-6. The product above has the inositol already in it and at the correct ratio. This is a major reason I selected it since there are other slightly cheaper products but without the inositol which would then have to be added with additional supplements – a definite hassle. Although this supplement will net cost you about \$50 per month, the more I research it, the more I think it is worth adding. Note: Much of the scientific research completed with IP-6 has been done by Dr. Abulkalam Shamsuddin, a scientist at the University of Maryland School of Medicine. As a non-patentable product, it is unlikely we will see well-designed clinical trials to give further in-depth knowledge about it. Research began in 1988 but no human clinical trials have yet to be undertaken.

8. Green tea

Dose: If you have prostate cancer, consider taking 2 capsule twice a day of a capsule with a net of 300 mg of polyphenols

Source: Swanson's product #SWH099 is a 300 mg capsule, \$5.49 for 60 capsules.

Summary of Key Recommendations: Protocol B

	<u>Morning</u>	<u>Afternoon</u>	<u>Evening</u>
1. Sodium Selenite	2 tablets		1 tablets
2. Melatonin	-- --		1 capsule
3. Flaxseed	2-3 TBS. or a muffin		2-3 TBS or another muffin
4. DIM*	1 tablet		1 tablet
5. Pomegranate*	1 capsule [for men]		1 capsule
6. Vitamin D3.	1 capsule		
7. IP-6	1 teaspoon		1- 1½ teaspoons
8. Green Tea	2 capsule		2 capsule

* DIM and pomegranate only apply to estrogen-related cancers such as prostate, ovarian, endometrial, breast, and colon cancers.

Again, be sure to read and add the diet advice in Chapter 8 below

Chapter 6 – Very Aggressive and/or Advanced Cancers: Protocol C

This section will address additional interventions needed if you are also experiencing an aggressive increase in your cancer condition such as a rapidly rising PSA, rising cancer markers such as CA-15, or increases in tumor size and/or have an advanced cancer or are in remission from an advanced cancer. If you do not have evidence of an aggressive cancer and/or an advanced cancer, you may choose to omit this section at this time.

On the other hand, if the situation is serious, then as difficult as this section may be [added supplements and decidedly added costs], you very seriously want to consider it. These supplements are extremely powerful. Moreover they were quite synergistically with each other. Read the admittedly difficult descriptions of what each does. If these could be patented, they would all be chemotherapies your oncologist would be launching immediately.

But they are over-the-counter. So they will never be recognized. I note in passing that the chemo drug Tarceva acts in the same way as genistein. It however has dire side effects and huge costs – genistein, while not quite cheap, has neither. Read again section 4 of chapter 13. Then consider the options given in this protocol.

Research Support for this Aggressive and/or Advanced Cancer Protocol

We will very briefly describe just some of the research explaining why these elements have been chosen. Once you read the background, the rationale should be apparent.

We shall use the following citation form. If an eight number is given in brackets, to get the citation, enter this number in a google search along with the word PUBMED. The first hit that comes up will be the actual Abstract for the article quoted so hit that choice and you will have the Abstract along with the citation for the Abstract.

1. Genistein

To begin, the following is a good general review article on VEGF [16282508].

VEGF synthesis is induced by both low oxygen tension and growth factors. Genistein can inhibit both of these pathways. The following is a good review of HIF-1, the genetic factor that stimulates VEGF synthesis. HIF-1 is an extremely important growth factor for cancer cells since it also stimulates the synthesis of the enzymes involved in aerobic glycolysis, the main energy pathway in cancer cells. The article can be read online.[16935775].

The two worst cancers are pancreatic and GIST, gastrointestinal stromal cancer. They are largely resistant to all forms of chemotherapy or radiation therapies and are generally considered to be fatal diseases. Pancreatic cancer depends on the secretion of VEGF and epidermal growth factor for its aggressive growth. Genistein, as a general tyrosine kinase inhibitor, can inactivate the receptors for both these hormones [16807445].

The following is a tremendously exciting article which shows that genistein can inhibit BOTH HIF-1 and VEGF synthesis in pancreatic cancer cells. The article can be read online [14692041]. Genistein blocks HIF-1 and VEGF synthesis in the eye as well [15857276, 15834450]. The following study shows that

genistein can not only inhibit the synthesis of VEGF, but the tyrosine kinase activity of the VEGF membrane receptor as well.

Therefore, genistein inhibits the synthesis of new VEGF molecules while blocking the biological activity of previously synthesized VEGF molecules [17142033]. Other evidence shows that genistein can activate the gene for PTEN, the phosphatase that inhibits PI-3K/AKT signaling. This signaling pathway is fundamentally important in the growth and survival of all cancer and leukemia cells [15905199, 16673816].

PTEN inhibits the expression of the VEGF gene. This probably involves the ability of PTEN to block AKT activation [16527906]. PTEN or PI-3K inhibitors upregulate the tumor suppressor gene p53. This genetic factor blocks VEGF synthesis via the down-regulation of HIF-1 [16527906, 16867872].

In addition, the genetic inactivation of PTEN promotes the increased expression of both Ang-1 and VEGF, angiogenesis factors, and the VEGFR1 and R2 VEGF receptors [16107612]. PTEN inactivation and increased activity of the EGF receptor promotes the activation of the VEGF gene promoter. Interestingly, genistein can BOTH promote PTEN expression and inhibit the activity of the EGF receptor, thereby blocking VEGF gene activation.[16107612].

Let us address one more issue about genistein. Some physicians discourage its use.. Let's explore this issue with some science. There are two estrogen receptors in the body. The alpha or classic estrogen receptor is responsible for mammary gland growth. The beta receptor was only discovered about ten years ago. Its function or role in the body is only now becoming understood. In breast tissues, the beta receptor is considered an inhibitor of alpha mediated growth. In fact, the beta receptor apparently plays this role in many other cancers as well.

Since genistein does have a small capacity to activate the alpha receptor, physicians have discouraged women at risk for breast cancer or those who presently have breast cancer from ingesting genistein. **This is a mistake.** The alpha receptor is 10,000 to 50,000 times more sensitive to estrogen than it is to genistein. Further, genistein binds and activates the beta receptor 100 times more effectively than it does the alpha receptor [16273360, 16118406].

As suggested, the optimal treatment protocol for is to use an antagonist of the alpha receptor and an agonist or activator of the beta receptor. Genistein is a powerful anti-cancer natural medicine in general and an activator of the estrogen beta receptor. Furthermore genistein's ability to activate the alpha receptor can be blocked with melatonin, the sleep hormone. Melatonin does not block the activity of the beta receptor [16635015, 15229223]. You are taking melatonin in protocol B; be sure to do so.

In addition, melatonin is an inhibitor of aromatase activity, the enzyme that converts testosterone to estrogen, as noted already – which is a very good thing [16647824, 16080194, 15683469].

If a woman is diagnosed with an estrogen responsive cancer, she should immediately begin taking melatonin and genistein. Synthetic prescription drugs are not necessary. In addition, genistein is a general tyrosine kinase inhibitor, which means it inhibits the activity of the growth promoting EGF and HER-2 receptors [12239620].

The effective cytotoxic dose of genistein necessary to kill 75% of breast cancer cells is 10 microM, a dose that can be reached in the blood by the consumption of specific genistein products. Specific recommendations are given in the details below.

The take-home message is that the science needed to assess these matters can be quite complicated. The off-the-top-of-the-head commentary by well-meaning physicians does not serve you well. Getting good information remains an extremely challenging enterprise.

Summary

Genistein can block the activity of ALL tyrosine kinase receptors, including those for VEGF and EGF.

Genistein can also increase the synthesis of PTEN, an major tumor suppressor and inactivator of PI-3K/AKT signaling. Genistein can inhibit the activity of HIF-1, an activator of VEGF synthesis and glycolytic enzyme synthesis. In summary, genistein in the correct dose [particularly in combination with glutamine to be introduced later] can work to inhibit the growth and development of virtually all cancer including pancreatic cancer and leukemia cells.

2. Policosanol

Policosanol is a mixture of long chain alcohols isolated from sugar cane. It may reduce cholesterol levels by over 30% although recent research has called this into question. Related to our cancer cell death interest, the following study shows that Policosanol powerfully activates AMP-kinase (AMPK). [16714400]. Statin drugs directly interact with the enzyme HMG-CoA reductase, the rate controlling enzyme in the synthesis of cholesterol. Policosanol does not directly interact with HMG-CoA reductase. It activates AMPK which phosphorylates and inactivates HMG.

The following article is an excellent review of the role AMPK plays in energy metabolism. [16998529]. AMPK is activated during periods of metabolic stress. It shifts the metabolism of cells from an anabolic to a catabolic state to maintain their survival. This means the synthesis of cholesterol, fatty acids and triglycerides is inhibited. Page 657 of the article referenced above has a nice diagram showing the MANY metabolic roles of AMPK. AMPK kinase also inhibits mTOR activity, a known suppressor of autophagy. So Policosanol activates AMPK which inhibits mTOR, keeping mTOR from halting cancer cell death [18006825, 17224623].

This makes perfect sense since autophagy is also activated during metabolic stress. The primary role of autophagy is to induce the breakdown of proteins and cellular organelles in order to provide emergency nutrients for the stressed (starved) cells. If autophagy is prolonged, the cells will die of programmed death. This process is promoted by activators of AMPK signaling such as Policosanol.

There are further research results on Policosanol we shall relate below. We have discussed above the importance of inhibiting mTOR. With Policosanol, we now have two different ways to inhibit mTOR activity. Policosanol directly activates AMP kinase, the inhibitor of mTOR signaling. Glutamine inhibits

mTOR by an entirely different pathway, probably via its ability to inhibit PI-3K/AKT signaling, a known activator of mTOR.

We know that mTOR inhibits the activation of autophagy, **and** it also promotes aerobic glycolysis, the signature metabolic pathway in cancer cells. This makes the mTOR pathway a fundamental target in the treatment of cancer and leukemia! [16288290].

HIF-1 is a master regulator of cancer cell metabolism and growth via its ability to stimulate the synthesis of VEGF and the enzymes involved in aerobic glycolysis. The PI-3K/AKT pathway activates mTOR and shifts the metabolism of cells from oxidative phosphorylation to glycolysis. mTOR activates the activity of HIF-1, the master regulator of cellular metabolism. When mTOR is inhibited, aerobic glycolysis is blocked and cancer cells revert to a normal oxidative metabolism. Inhibiting mTOR is a fundamental goal [17502379, 15156201].

A recent study found that one of the IKK enzymes, activators of NF-kappaB, directly associates with mTOR and increases its activity [17616684]. This shows that glutamine can inhibit mTOR activity in two different ways. First, it inactivates PI-3K/AKT signaling, a known activator of mTOR, and second it inhibits IKK activity via its ability to stimulate hsp70 synthesis. Direct AMP kinase activators, inhibitors of mTOR, such as Policosanol are truly synergistic with glutamine in the treatment of cancer and leukemias.

3. Feverfew (parthenolide)

Feverfew is a source for the active ingredient parthenolide. It is well established that parthenolide inhibits both NF-kappaB and STAT3 signaling. A paper was published this year demonstrating that parthenolide stimulates oxidative stress. This stress inhibits JAK1 kinase activity which results in an inhibition of STAT3 activation [17385713].

How does parthenolide promote oxidative stress? According to the following three papers, parthenolide reduces the level of glutathione in cells. This results in the gradual build-up of ROS, reactive oxygen radicals in the cells. Apoptosis follows [17275679, 12151389, 15142672].

In order to substantially inhibit glutathione synthesis, 50 microM of parthenolide must enter the body. Based on my calculations, we only need 62 milligrams of parthenolide a day to meet this goal. Unfortunately, parthenolide is completely insoluble in water. Very little of this product is going to enter the body in capsule form. This is why I recommend the product be dissolved in warm ½ and ½ or cream before ingestion. We know this technique dissolves the parthenolide and gets it into the body. But we still do not know how much parthenolide enters the lymph and blood.

Parthenolide also inhibits protein kinase C zeta signaling. This membrane bound enzyme is involved in many aspects of growth control. The inhibition of PKCzeta signaling inhibits the activation of the p38 stress enzyme. P38 activation is involved in the breakdown of both bone and muscle. We will discuss this issue in depth in future essays [16051639].

One of the roles of PKCzeta in the body is to activate HIF-1a, hypoxia inducible factor, by promoting its association with p300 [14744756]. HIF-1 is one of the most important enzymes in cancer cells. It

promotes cancer cell survival during periods of low oxygen by stimulating the synthesis of VEGF, the angiogenesis factor. HIF-1 also stimulates the synthesis of enzymes involved in the glycolytic energy pathway, the energy pathway utilized by cancer cells [17404504, 17330811].

In summary, parthenolide inhibits cancer and leukemia cell growth and survival by many different pathways. It inhibits NF-kappaB, and PKCzeta activity, and it reduces the level of glutathione in the cell, thereby generating oxidative stress. The increased ROS block JAK1 activity which blocks the activation of STAT3. We are only now beginning to understand the diverse biological roles that parthenolide plays in the body. It is truly a natural medicine wonder compound.

A recently published article answers a lot of questions about parthenolide. The structure of the parthenolide molecule allows it to interact with specific proteins that produce superoxide anion. This occurs inside and outside the mitochondria. Moreover, the induction of superoxide anion production occurs at very low doses of parthenolide [17275679].

Consider the following. Parthenolide specifically blocks NF-kappaB activation. This results in a reduced synthesis of anti-oxidant enzymes, such as Mn superoxide dismutase. MnSOD is a mitochondrial enzyme that degrades superoxide anion. Now we find that parthenolide can directly increase the level of superoxide anion in the mitochondria. The combination of increased superoxide synthesis and reduced degradation will rapidly destroy the integrity of the mitochondria.

5 microM parthenolide substantially depletes the glutathione stores in the cells over 6 hours. Keep in mind that glutathione cannot be made in the mitochondria. It must be pumped into this organelle. This uptake process is blocked by glutamic acid, a breakdown product of glutamine [to be introduced shortly]. Parthenolide and glutamine are clearly synergistic, at least with respect to reducing the glutathione content of the cancer cells, leading to their death. Also keep in mind that parthenolide, via the induction of oxidative stress, blocks JAK1/STAT3 signaling.

4. Niacinamide

Niacinamide is critically involved in inhibiting cancer cell growth and survival. This is the story.

It begins with SIRT1. SIRT1 is a so-called longevity factor which inhibits the death of cells, including cancer cells. This deacetylase shuttles back and forth between the cytoplasm and the nucleus. In the cytoplasm, it deacetylates and inhibits various anti-growth transcription factors such as p53, FOXO and PPARgamma. [17197703].

In the nucleus, SIRT1 binds to histones and deacetylates them. Deacetylation means that SIRT1 removes an acetyl group from these proteins. This inactivates or activates the protein depending on the particular protein. In cancer, many anti-growth genes are inactivated by the methylation of their DNA. A massive scientific literature has been published on this topic. This is step one. In step two, the methylated DNA appears to interact with histone deacetylase proteins. These proteins remove acetyl groups from histones, thereby inactivating their activity. Histones are necessary for the activation of specific genes.

Many histone deacetylase inhibitors have been developed and are in clinical trials. The objective of these drugs is to reactivate silent genes by blocking the activity of histone deacetylase enzymes. Most histone deacetylase inhibitors CANNOT reactivate genes if the gene DNA remains methylated. Other histone deacetylase inhibitors can inhibit histone deacetylase enzymes while promoting the demethylation of gene DNA. Unfortunately, these multi-purpose inhibitors tend to be a tad toxic. [17276411].

The following article is one of the most important studies ever published on the topic of gene regulation. The title of the article speaks for itself [16596166].

This study shows that gene methylation, per se, is NOT enough to silence genes. The hypermethylated DNA apparently attracts the class 3 histone deacetylase SIRT1 to the gene and this interaction is critically important in gene silencing. If SIRT1 activity is silenced, methylated DNA does NOT silence genes. This is a remarkable observation. The activity of type 1 and 2 histone deacetylase enzymes is now irrelevant. They cannot contribute to the silencing of genes if SIRT1 is not bound to the methylated DNA.

In the nucleus, SIRT1 promotes DNA repair by the deacetylation of a major repair enzyme. If we are trying to kill cancer cells, we do not want their DNA repaired. [17334224].

Nicotinamide (niacinamide) is the NATURAL inhibitor of SIRT1 activity. As such, it is a powerful anti-cancer compound and a type 3 histone deacetylase inhibitor. Now we consider POLY(ADP-Ribose) Polymerase (PARP-1). PARP-1 is involved in a variety of pathological conditions. In addition, PARP-1 is absolutely necessary for NF-kappaB activation. The following review article, which speaks for itself, can be read online [12223530].

The key observation is that niacinamide is the natural inhibitor of PARP-1.

5a. Glutamine

Large supplemental doses of glutamine can kill cancer and leukemia cells in many different ways. Let us explain by first looking at TNF signaling. TNF or tumor necrosis factor is THE most potent anti-cancer factor ever identified. Unfortunately, it is also toxic to normal cells. With one minor exception, the FDA has prohibited its use against cancers. TNF activates two different pathways.

The apoptosis pathway is highly dependent on the induction of oxidative stress, especially in the mitochondria. The PI-3K/AKT pathway promotes a powerful resistance to apoptosis. Clearly, the resistance pathway neutralizes the anti-cancer potential of TNF. We have found that glutamine can BOTH promote apoptosis while blocking the resistance pathway to TNF signaling.

Consider the resistance pathway. First, oral glutamine inhibits PI-3K/AKT signaling in experimental breast cancer. The AKT enzyme promotes aerobic glycolysis and cellular survival in cancer cells [14621121].

Second, glutamine promotes the synthesis of hsp70 and other heat shock proteins. Hsp70 blocks IKK activity and inhibits NF-kappaB activation. This blocks the resistance pathway and enhances TNF cytotoxicity [17030616, 15198984]. In addition to NF-kappaB-activated survival genes by glutamine, there are also the powerful angiogenesis factor IL-8, a NF-kappaB dependent gene. We can conclude that glutamine blocks two MAJOR anti-apoptosis pathways. Now consider the apoptosis pathway.

Glutamine, converted in cells to glutamate, blocks the uptake of the anti-oxidant glutathione into the mitochondria. The lack of mitochondrial glutathione enhances TNF induced tumor cytotoxicity. [11522449, 11063916].

But there is more to the story. The enzyme caspase 3 is the final mediator of TNF-induced apoptosis. When glutamate depletes GSH from cancer cells, the caspase 3 gene becomes activated. At the same time, gene expression of the anti-apoptosis BCL-2 gene declines [15609127].

The cystine/glutamate antiporter exchanges intracellular glutamate for extracellular cystine. Cystine is the direct precursor of cysteine, a critical amino acid for the production of glutathione. If the extracellular glutamate concentration becomes elevated, such as by the administration of large supplemental doses of glutamine (in the liver, about 65% of an oral glutamine dose is converted to glutamate), it blocks the activity of the cystine/glutamate antiporter thereby depriving cells of cysteine. This results in a rapid depletion of glutathione [19178660].

Therefore, *glutamate both depletes the cells of glutathione and inhibits the uptake of glutathione into the mitochondria*. The result is oxidative stress and apoptosis [cancer cell death] since cancer cell growth and survival is highly dependent on the maintenance of high levels of intracellular glutathione. While glutamine, via glutamate, can deplete glutathione in cancer cells, it enhances glutathione synthesis in normal cells. In fact, the enhanced glutathione levels in normal cells can block carcinogenesis by toxic chemicals such as DMBA [18313901].

There is more. Oral glutamine, via its ability to deplete cellular glutathione and create oxidative stress, induces the synthesis of p53, the major tumor suppressor in the body. In addition, glutathione depletion inhibits the synthesis of c-myc, a tumor gene that is activated in up to 90% of all cancers [16898871, 18221043].

We have been discussing autophagy. Autophagy is now our preferred method of cancer and leukemia mediated cell death. The mTOR pathway is the natural inhibitor of autophagy. Glutamine inhibits the mTOR pathway which is another valuable payoff for glutamine. [NOTE: To guide the reader, the supplement Policosanol discussed below also inhibits mTOR which is why it is also in the protocol]. There is yet added important research about glutamine that the reader may find of interest. It is well established that the migration of macrophages into tumors promotes cancer cell growth and angiogenesis. Macrophages secrete many pro-inflammatory immune hormones in addition to enzymes like MMP9 which break down the intercellular matrix. In addition, macrophages secrete VEGF, IL-8, macrophage chemotactic protein and other pro-growth factors. The following paper identified the genetic factors NF-kappaB and AP-1 as being responsible for the synthesis and secretion of these factors [17924976].

It is known that high dose glutamine can inhibit the activation of NF-kappaB [16317391]. Glutamine stimulates the synthesis of HSP70 which inhibits NF-kappaB activation [17234954]. The following two papers show that glutamine deficiencies, which are very , very common during illnesses of all kinds, promote the synthesis of both VEGF and IL-8. Apparently, when certain key amino acids become depleted, it causes oxidative endoplasmic reticulum stress in cells. This stress promotes the synthesis of angiogenic factors such as VEGF and IL-8 so more blood vessels can be directed into the tumor mass. This brings in oxygen, glucose and amino acids to enhance cancer cell survival [15256456, 14738568].

The increased synthesis of IL-8 is due to the activation of NF-kappaB and AP-1. It is not known how low glutamine increases VEGF synthesis. In the first paragraph, I cited a study which showed that NF-kappaB

and AP-1 promoted macrophage activation in tumors. This means that high glutamine doses could neutralize this macrophage activation and the production of growth promoting angiogenesis factors. Although VEGF gets all the attention, the angiogenesis factor IL-8 is equally important. This factor promotes angiogenesis, the migration of macrophages and neutrophils into tumors, and is a growth factor itself. The progression of many cancers, including pancreatic, breast, ovarian and melanoma is highly correlated with increased IL-8 synthesis. The secretion of VEGF by these cancer cells and invading macrophages may be of much less clinical significance [11544106].

Interestingly, taxol, the toxic chemo drug that is the standard treatment for ovarian and other cancers, stimulates the secretion of IL-8. Although taxol is toxic to these cells, the secretion of IL-8 *reduces* the drug's overall efficacy [15823106]. Taxol increases the level of NF-kappaB and AP-1, which *promotes* cancer cell survival and IL-8 gene activation [10823417].

The Role of Other Amino Acids

Autophagy is a reversible survival pathway that is usually induced by starvation. As a result, the presence or absence of certain amino acids profoundly affects its activity. The mTOR biochemical pathway is known to promote protein synthesis, especially in muscle. Therefore, it makes biological sense that mTOR is also a direct inhibitor of autophagy, an initiator of cellular breakdown. Some amino acids act as metabolic sensors in the body. In many respects, they act like hormones by virtue of their ability to turn specific biochemical pathways on and off. Four notable amino acid sensors are leucine, arginine, glutamine and isoleucine.

Both arginine and leucine *activate* the mTOR anti-autophagy pathway. Both are to be avoided [15010853, 18056791, 17623010]. Glutamine, on the other hand, *inhibits* the mTOR pathway and is an antagonist to leucine [17978888, 15567168]. Leucine *activates* mTOR signaling partly by its ability to inhibit the AMP kinase [17178807]. Glutamine *inhibits* mTOR signaling by inhibiting the PI-3K/AKT pathway [14621121]. The AKT pathway DIRECTLY activates mTOR [17786026].

The excessive consumption of leucine and arginine block cancer cell death by autophagy. Glutamine does the opposite. So nuts which contain a highest concentration of arginine are to be avoided on this protocol.

5b. Theanine

There is yet one more issue. Glutamine will form some glutamate in the body. Cancer cells will take in the glutamate and then exchange it for the amino acid cystine which is then converted to cysteine inside the cell and helps form glutathione. Glutathione neutralizes free radicals but we do not want this in our cytotoxic protocol which is why we are avoiding anti-oxidants. Our protocol is creating free radicals to kill the cancer. However there is a solution to this unfortunate side effect of taking glutamine (which creates some glutamate).

The supplement theanine blocks the uptake of glutamate and cystine into cancer cells. This reduces the formation of glutathione which keeps the cancer cell sensitive to death by free radicals. Since theanine also keeps cancer cells from pumping out chemo drugs, theanine also enhances the chemo-therapeutic effectiveness of cancer drugs such as doxorubicin. Drugs to use while taking chemo, however, is the focus of another small book by itself and is not further addressed in this volume.

Broadly speaking, since there are other sources of glutamate in your diet, theanine should be used if you are taking glutamine and even if not since it reduces glutamate even in normal cells. Either way, theanine reduces the stability of cancer cells by depriving them of the glutathione they need to survive. However in our protocol, we are combining it with the glutamine since we want to reduce the side effects of the glutamate that is formed as well as add power to the impact of the glutamine.

The reason we want to take the theanine only at bed time (besides the fact that it makes you sleepy) is that glutamate is a daytime neurotransmitter which stimulates the awake phase during the day of brain function. We want to maintain a careful balance.

Alert: We are aware of several negative issues with glutamine. The first is that some of the glutamine is converted to glutamate as we noted above which is not good for you in excess amounts. After pondering the downside of glutamine, versus the upside, I believe that if you must invoke Protocol C, then it is much better to get the benefit of glutamine and simply not worry at this point in time about some of the long-term possible residual effects it can have. Survival is the foremost consideration at this point in time. In addition, the companion of theanine to the glutamine will eliminate an important amount of the side product of glutamate to reduce the problem to manageable proportions. Take note that the cancer killing mode of both glutamine and feverfew identified above is neutralized by mega-doses of anti-oxidants. Here is where the rubber meets the road. Protocol C is neutralized by large doses of antioxidants although, again, regular low doses such as found in typical daily multi-vitamins and a regular diet including fruits and vegetables will not create any undue contradictions.

6. Curcumin

The argument for curcumin is the same for Meriva – which is curcumin – but much more is much better for serious situations. We are suggesting 5-10 grams twice a day. You will best buy this in bulk to get this regimen and you will need to mix it with heavy cream or coconut oil to achieve absorption. Also take bioperine with it which multiplies its absorption.

Implementing Protocol C

[Doses and Purchase of Your Supplements]

We will identify dose levels and at least one suitable source for each of the supplements identified.

1. Genistein

Dose: You will be taking 8 capsules a day. Do this for 3 weeks, then rest for 1 week (no genistein). Then repeat 3 weeks on, 1 week off, consistently.

Source: Life Extension's change of product has necessitated some further research. Use the Super-Absorbable Soy from LEF, #01649, and take 8 a day for 3 weeks, then rest a week. So a bottle will last one week, and you will use 3 bottles a month [the fourth week you rest]. If you order 4 bottles, and are a member of LEF, then the 3 of the 4 bottles will come to about \$60 a month [depending on the added shipping fees]. Also the genistein and the curcumin have a synergy effect, so be certain to continue this and the curcumin.

*In the journal Nutrition and Cancer, around Sept. 2011, it was reported that genistein reduced PSA by 7.8% in a matter of weeks, in a trial done in an Oslo hospital by the University of Oslo. The placebo arm **increased** 4.4% in a comparable period of time.*

Now research shows that one needs more than 100 mg to get the full impact and more works even better. Hence the 8 capsules recommended which will deliver 224 mg of genistein daily. Do not take less since too little genistein is counter-productive.

2. Policosanol.

Dose: Take 20 mg at night.

Source: The Policosanol used in the primary study which guides this protocol was purchased in a health food store by the researchers. The pills were ground with a mortar and pestle and extracted with alcohol. The extracted alcohols were used in the experimental studies. This is very fortuitous. Using mass spectroscopy, the authors determined the exact nature of the alcohols present in the sugar cane extracts. Octacosanol (60%) and triacontanol (19%) are the major alcohols found in the supplement pills. ONLY triacontanol is able to substantially active the AMPK pathway as desired. There are many Policosanol products for sale. Most of these products are inadequate because they contain excessive concentrations of octacosanol. This analysis is according to Life Extension Foundation which we currently believe.

Currently there are ONLY two acceptable commercial Policosanol products that can be used to treat cancer. The Policosanol used by the scientists was made by Source Naturals. Many companies sell this product. Do not purchase this product combined with CoQ10 or anything else. CoQ10 protects the mitochondria against oxidative stress. We want the mitochondria of cancer cells to be oxidatively stressed. Previously the other source of quality Policosanol was made by LEF. It is their product number 561. LEF can be located at www.lef.org Now a Source Natural Policosanol can be found at www.vitacost.com, a 20 mg capsule, \$14 for 60 which will obviously last 60 days. The product from LEF is a 10 mg capsule, which is \$24 for 60, so 60 days' worth would be \$48. Clearly the vitacost strategy is wiser.

3. Feverfew

Dose. Each daily dose is 4.5 grams of bulk extract, 2¼ grams twice a day along with some half-and-half or cream.

Source. For now, there is Swanson's SWH147, a 500 mg capsule so you will take 3 capsules three times a day. If you can tolerate it, another 2 capsules daily would be even better, but this begins to push to envelop of compliance, I know.

4. Niacinamide

Dose. The minimum dose is 3 grams per day.

Source. Go to iherb.com and type in niacinamide into the search box. Select Source Natural brand, 1500 mg time-released product [make sure it is the time-released version]. It is \$9.29 for 100 tablets. Start with one a day. Then move to 2 a day. Even with a time-released version, you may experience some flushing. This is annoying but not dangerous in any way.

5a. L-Glutamine

Dose: Take a daily dose to 0.6 grams per kilo [2.2 pounds] of body weight. An average 150 pound person is 68 kilos ($150 \div 2.2 = 68.18$) and would take $.6 \times 68 = 41$ grams per day. Mix the glutamine in some juice or other beverage.

Source: The supplier Bulk Nutrition sells 1 kilogram (1000 grams) of glutamine for about \$25. They are located at www.bulknutrition.com/?products_id=2460 [this takes you directly to L-glutamine]. At 40 grams a day, 1000 grams will last you 50 days.

5b. Theanine

Dose: Take 200 mg at night right before bed time since this supplement will make you sleepy.

Source: Swanson has product SWU110, a 100 mg capsule, \$6.89 for 60 capsules. Take 2 at bed time.

6. Curcumin

Dose: Take 5-10 grams twice a day. Mix it with heavy cream or coconut oil. Take a capsule of bioperine with each serving [for example Swanson's SWU308 or Vitacost.com has a product from Source Natural] since this is needed to enhance absorption.

Source: Search the web for bulk curcumin suppliers.

Some Issues with Additional Supplements. You must not use any *large doses* of supplements such as vitamin C, E, NAC (N-acetyl cysteine), and alpha lipoic acid if you add Protocol C to your list. Protocol C is relying on oxidation through its mechanisms of action [Protocol A and B essentially do not]. So if you add Protocol C, avoid any high level uses of antioxidants and avoid very high anti-oxidant fruit juices and foods. The reason as noted is that the cytotoxic Protocol C will cause cell death with targeted free radicals, so anti-oxidants will be counter-indicated at this time. A multi-vitamin is fine. Don't get to fanatical about this point. Just avoid mega doses or any large amounts of added anti-oxidant supplements such as added vitamin E, CoQ10, etc. Some naturally occur in your diet. This is not a problem.

Nuts contain a highest concentration of arginine and are counter-indicated on this protocol C. Also berries and berry juice as very high antioxidants are to be avoided as much as possible. To repeat, as discussed above, this is because antioxidants will protect the cancer cells from death [but are excellent as cancer prevention if you had *NO* cancer]. Also quercetin, ellagic acid, and possibly other flavonoids are to be avoided by anyone undergoing this cancer protocol C. Whatever anti-cancer properties possessed by quercetin is completely over-shadowed by its induction of glutathione synthesis [in other words,

anti-oxidant activity]. A regular healthy diet is not of concern – it is the added doses of anti-oxidants at high levels.

Note that a carefully constructed combination of key supplements including some with antioxidant effects can be prove additionally useful within a immune-boosting context for Protocol A and B – we are just avoiding the major anti-oxidant strategy since it is counter-indicated with a cytotoxic strategy as given in protocol C above. This conflict is often missed by those who construct their own Protocol. We have in fact developed such an additional carefully constructed immune support formulation but it is not a part of the cytotoxic Protocol we have developed above.

If you wish information about this, contact the author directly about that design. There is nothing proprietary or hidden – it is just not part of this book’s focus.

Summary of Protocol C

	<u>Morning</u>	<u>Evening</u>
1. Genistein	2 capsules	3 capsule
2. Policosanol		1 capsule
3. Feverfew	2¼ grams	2¼ grams
4. Niacinamide	1 capsules	1 capsules
5a. Glutamine	about 20 grams	about 20 grams
5b. Theanine		2 capsules
6. Curcumin	5-10 grams	5-10 grams
	[take each curcumin serving with bioperine to enhance absorption]	

Chapter 7 – Selecting the Protocols Right For You

The three protocols provided above address another of situations and circumstances. Let us begin with each protocol and its intended uses.

Protocol A

As was discussed, all the elements of this protocol focus on the prevention of metastasis which is your first and foremost enemy. If you are still alive, then just preventing the cancer from gaining any further ground is sufficient to keep you alive and reasonably well thereafter. You will not die from a tumor as long as it is held in check. As science grows ever more sophisticated, they will find cancer to some degree in everyone. This will lead to an exponential explosion in needless treatment.

In any case, the body can tolerate a tumor that remains in place. Protocol A is intended to accomplish precisely this. As you re-read the material provided in that section, you will see there is a basis of optimism that the elements of protocol A may accomplish this. In truth, its success really depends on how aggressive the cancer is and your own state. Stress makes a tumor more aggressive – although to be more accurate, the forces that keep it in check are reduced when you are under stress. Another unspoken treatment is to try to stay as stress-free as possible. This is always good advice.

From what I have indicated, if you have or have had active cancer diagnosed, you should consider Protocol A as an ongoing regimen. If your cancer was “modest” in its nature, such as skin cancer, many forms of DCIS, or low-level prostate cancer where you are simply on watchful waiting, you can omit the isoleucine in that Protocol and even the Super CitraMax. However if you had any serious cancer from higher stages of Breast Cancer, ovarian cancer, brain cancer, cancer of other critical organs such as kidney cancer, any head and neck cancer, pancreatic cancer, etc., then you want to do all of Protocol A.

Protocol B

Protocol B is also intended for virtually all cancers other than simple skin cancers. If you have had essentially any non-skin cancer diagnosed, you want to follow this protocol. It is the core cytotoxic regimen comprising cancer cell killers. Although every supplement in this list might potentially have some degree of antioxidant activity, it is not that action that brings the supplement into this listing. The supplements work on specific pathways that should remain inhibited, once you have had cancer. So they are all cytotoxic (cancer cell killers).

The alternative strategy might have been strictly an immune-enhancing strategy to boost your own immune system so that it will defend you. In my experience, once you have had cancer, there is evidence on the table that your immune system is not up to the task. This does not mean you do not want a healthy immune system aiding you. In fact you do. It means that you do not want to excessively engage that activity. Why not? Simply because you cannot do both at the same time. Beyond a reasonable and moderate level, boosting your immune system will work counter to the cytotoxic mechanisms that Protocol B employs. In effect, a huge increase in free-radical fighters will protect the cancers from Protocol B which will help kill the cancer with a build-up of free radicals or by weakening the cancer – which will be prevented by a huge increase of anti-oxidants. You need not shun them. A mixture of

some at a reasonable level all taken together will prove valuable. But you cannot engage in any mega-doses.

For most cancer patients, they will take Protocols A and B for the long haul.

Protocol C

This protocol is engaged when things are at a rather serious degree. You may have advanced stage cancer. You may already have some metastasis. You may have had chemotherapy and radiation and they are about to prescribe more. Experience has shown that if you have had three rounds of different chemotherapy regimens, they are just playing with you. If you have had a serious cancer at the onset, or even have achieved a declared “remission” over one, you are at great risk. There is no remission. There is only the window of opportunity in my assessment to undertake a serious attack. This is protocol C.

There are currently six elements in Protocol C. Take each one and take it seriously. I recognize this will be a challenging regimen as well as a significant expense for some. You have limited choice although when there is no money for these supplements, there is just no money. Roughly speaking, they are of all equal importance. Our experience has been that you need to triangulate your strategy – that is, use multiple supplements at once rather than lots of just one. Do your best.

Chapter 8 – Cancers Demanding The *Total 10-Yards*: U.S. and Foreign Clinics

This section is somewhat complicated because it involves discussing actions outside of the protocol itself. Specifically, there are a number of therapies not offered at virtually any oncology center in the US that nonetheless are very powerful. These may include the use of cesium, insulin potentiation therapy, the use of Caressing which is just a high-powered ginseng-based supplement, hyperthermia, sonodynamic therapy, etc. Many of these therapies have usefulness but simply are typically unavailable since they are not part of the US standard of care. The patient much further down the line or close to the end of the line could seriously consider visiting one of these clinics for these advanced therapies, assuming their finances permitted. But don't wait until the last days – then even these options may be too little too late.

Dr. Ralph Moss had a report available some time ago on Mexican clinics. He has mentioned the Hope4Cancer Institute in Mexico. The Health Sciences Institute did an article on cesium therapy indicating how you could even implement this yourself. We had begun a formal trial at UCLA using Careseng while our Chief Medical Officer, Dr. Jerome Block, was still alive. Dr. Moss introduces in one of his recent newsletters sonodynamic therapy in which ultrasound waves are used to kill cancer cells. He noted that this is being offered at the Indiana Center for Advanced Medicine in Indianapolis as well as the Hope4Cancer Institute in Mexico. A recent book, *Cancer Breakthrough USA*, identified the Reno Integrative Medical Center here in the USA as offering some of these innovative approaches [Editor's Note: I have no personal knowledge about any of these Medical Centers. In addition, just as Dr. Moss has also clearly stated, any mention of these institutions by either of us does not constitute in any an endorsement on our parts but are simply provided by way of informational purposes]. Dr. Moss is now doing an update on Mexican clinics which will be available soon.

In summary, the tools in the tool kit your standard oncologist offers even at the very best of cancer centers are still a fraction of the *bone fide* tools that have been shown effective to address advanced cancers, stage IV cancers, etc. – at least for some specific patients in specific settings.

The overall point of this section is to alert you that there are quite a number of legitimate possibilities that have turned cancer around for patients for whom the US establishment had given up hope or for whom the options they could provide (typically more chemo) would not nor ever suffice. So do not rule this option out as fanciful or extreme. Dr. Moss' own book, *Cancer Therapy*, first issued over 18 years ago, discusses many of these very possible therapies among the approximately 100 he describes, such as cesium, ginseng, hyperthermia, etc. as well as some of the specific supplements now being recommended in this protocol [e.g., selenium, DIM, green tea, curcumin]. Some of these ideas have been advanced and refined in the last two decades since his book came out so that they are ready for prime time.

In my professional opinion, many of these tools may prove to be effective for some patients who otherwise might have fatalistically given up hope. The main problem is the high cost of clinics if one wishes to have these tools provided. Although TRI does not provide any services itself, we continue to collect information about their availability. For a few select tools or strategies we think are likely worthwhile, we can direct individuals to web sources that purport to enable you to initiate some of these strategies yourself at relatively low cost [e.g., cesium therapy]. We have no ties, affiliations, or financial connections to any of these sources but simply accumulate information we can pass along. In short,

standard US oncology care is not the best cancer care available. If things get ominous, consider seeking useful tools, even for end-stage cancer, which are available – which may have life-saving implications.

Chapter 9 – Dietary Issues: Do’s and Don’t’s

There are a number of general dietary issues we will address that affect your cancer status.

1. Omit Olive Oil (if you have overt cancer)

The following is a brilliant piece of analysis by Dr. Stephen Martin who posted it in a public blog. He has since passed away, but I am confident he would want his ideas disseminated in this manner – without charge and freely available to the public.

His citation approach is to give an 8 digit number which you are instructed to enter into a Google search along with the word PubMed. The first item that will come up on the Google search will be the Abstract for the citation. Just click on the first hit to obtain the citation directly.

Before giving you his exposition, I will give you the summary so that you will know where he is going. So let me summarize the problem with olive oil. Definitive research shows olive oil activates a factor that is called PEA3. The first research report indicated that PEA3 inhibited the Her-2/neu gene which would impede cancer, so olive oil might impede cancer.

But as it turns out, later research showed that actually PEA3 does NOT inhibit the Her-2/neu gene, as first thought. What is more, PEA3 may have cancer promotion actions. Further research showed that PEA3 is required for Her-2/neu cancer development so it is particularly bad for breast cancer as well as estrogen-related cancers — such as *prostate* cancer. So olive oil activating PEA3 is very negative news, *if* you already have cancer. Thus olive oil is good if you do not have cancer but likely a cancer *promoter* if you have cancer. Things never are so simple.

So do not use olive oil [at least avoid it when possible] if you have or suspect you may have overt cancer. Following this section, I will identify your best substitute oil. Here is Dr. Martin’s full explanation and documentation of this discovery about olive oil.

Olive Oil Can Promote Cancer Cell Development by Dr. Stephen Martin

Oleic acid, the dominant fatty acid in olive oil, promotes cancer cell development.

Let me make this very clear. Oleic acid does not cause cancer. It simply promotes the further development of already established cancers. Oleic acid, a mono unsaturated fatty acid, stimulates cellular growth in both direct and indirect ways.

1. It activates the free fatty acid receptor GPR40 which stimulates the PI-3K/AKT signaling pathway. This pathway is activated in all cancer cells. The PI-3K/AKT pathway is inhibited by the saturated fat palmitic acid. .

2. Oleic blocks the cytotoxic ability of palmitic acid by redirecting PA from its free form back to re-esterification into triglycerides (fat molecules). This neutralizes PA's ability to make the death lipid ceramide or kill cancer cells directly. In a previous Blog, I explained that PA could directly damage the endoplasmic reticulum and the mitochondria by a variety of different methods. Ceramide, made from PA,

is VERY anti-cell growth. Oleic promotes cellular growth while blocking cell death pathways, a frightening combination.

Palmitic is a well-known cytotoxic agent against cancer cells. We naturally think of saturated fat as bad, while unsaturated fats like olive oil oleic acid as good. At least with respect to cancer, we had better rethink this dogma immediately. Pork fat contains equal amounts of oleic acid and PA. However, if the ratio of oleic acid to PA in our diets falls to 1:10, which means one oleic acid molecule to ten PA molecules, the oleic acid will completely block the killing ability of PA against cancer cells. In reality, we have much more oleic acid in our diets than PA. If you have cancer, this is not good news [1103797, 2805375, 5695516].

3. Olive Oil and Breast Cancer

Olive oil consists of 55-85% oleic acid. In the previous Blog essay, I cited evidence that oleic acid promotes cancer cell growth. I know this idea is controversial, but facts are facts. The Mediterranean Diet of fresh fruits, vegetables, and red wine has been touted as a preventative for both heart disease and some cancers. The health benefits of this diet, even in the presence of perhaps excessive amounts of saturated fat intake (lots of butter), are not in question. I am not writing about the prevention of cancer. That subject can wait for another time. Right now I am interested in what promotes cancer cell development AFTER the cancer cells begin to develop.

In 2005, a paper was published that claimed oleic acid suppressed Her-2/neu expression in cancer cell lines. Her-2/neu is a membrane epidermal growth factor receptor that is over-expressed in about 30% of all advanced breast and ovarian cancers. The presence of this receptor is a bad diagnostic sign. The monoclonal antibody drug Herceptin is used to turn off this receptor. Herceptin must be injected weekly and is only approved as a co-treatment with chemotherapy drugs. It kills cancer cells poorly on its own. Herceptin is one of those drugs presently bankrupting the American health care system. It cost \$40,000 a year, and is, in my opinion, completely expendable.

Anyway, this article, published by scientists at Northwestern University, received a massive amount of press. If you read these news releases, you'd swear that olive oil, or at least oleic acid, was a cure for breast cancer. Nothing could be further from the truth [5642702].

This year the same authors published another paper showing that oleic acid activated the PEA3 transcription factor. This transcription factor is thought to inhibit the expression of the Her-2/neu gene. [6406575]. The same authors also claim that gamma-linolenic acid, an omega 6 oil found concentrated in Borage oil, also inhibits Her-2/neu via PEA3. [6264182].

PEA3 was found to suppress HER-2/neu over-expression some years ago. Unfortunately, there is serious doubt as to whether this data is correct. [0655108].

These studies were conducted in cell lines grown in tissue culture plates. This is common practice in science. Unfortunately, the data accumulated in one culture condition often cannot be duplicated in other culture conditions. All cell lines are by definition mutated or they wouldn't grow in tissue culture in the first place. They are not representative of so-called primary human breast cancer cells removed from a human lesion.

The next study, which could not replicate the data in the studies mentioned above, was quite different. This study analyzed breast cancer tumor samples taken from 130 women after surgery and preserved in

liquid nitrogen. The women did not have chemotherapy or radiation therapy prior to surgery. The samples were collected from 1977 to 1989. This study is incredibly exciting because the women were followed up from 1 to 15 years.

The tumor samples were analyzed using real time quantitative PCR, the most sensitive scientific method available for detecting the presence of mRNA for specific molecules. The authors tested over 30 genes, including PEA3 and all four epidermal growth factor receptors, including Her-2/neu. The authors found a negative relationship between PEA3 expression and the presence of the estrogen alpha receptor. This means that PEA3 expression might be responsible for down-regulating the normal estrogen receptor. This is a bad diagnostic sign.

In contrast to the studies mentioned previously, the authors found a positive relationship between PEA3 and Her-2/neu expression. Clearly, PEA3 is not inhibiting the expression of the Her-2/neu gene in primary breast cancer tissues (the real world of cancer).

PEA3 definitely plays a role in breast cancer growth, but it is not a prognostic factor. This means that PEA3 activation, and its ability to activate genes such as MMP2, a protease that degrades the extracellular matrix, thereby allowing cancer cells to migrate, promotes cancer cell aggressiveness but it doesn't cause cancer in any way.

This thorough study strongly argues that PEA3 is not, in any way, a tumor suppressor gene as suggested by others [4633660]. The following study shows that PEA3 is REQUIRED for Her2/neu mammary cancer development. In transgenic mice over expressing the Her2/neu gene, PEA3 was found to be necessary for Her2/neu cancer development [1719215].

PEA3 is over expressed in human breast cancers and in almost ALL Her2/neu over expressing variants. [1467448]. PEA3 expression is generally associated with cancer cell growth and development. It is NOT a tumor suppressor molecule [6322223, [1519038, [2684413, 2898592, [4604892].

The next study is one of my favorites. It shows that Her2/neu over expression activates the over expression of the Cox-2 gene. The Cox-2 enzyme is over expressed in virtually all cancers. Its production of prostaglandins such as PGE2 promotes cancer cell growth, metastasis and angiogenesis. It is bad news. Interestingly, the Cox-2 gene has a DNA binding site for PEA3. If this site is mutated, Her2/neu can no longer stimulate the synthesis of the Cox-2 enzyme. Clearly Her2/neu utilizes the PEA3 biochemical pathway to promote cancer cell growth [1901151].

If the oleic acid in olive oil and other foods does indeed activate PEA3 activity, this puts oleic acid, an 18.1 omega 9 mono unsaturated oil, in the category of a cancer promoter (but not a carcinogen).

2. Use Coconut Oil

As I will discuss below, the oil to use is coconut oil. Since many are not aware that this is the most healthiest of oils, I will give the complete explanation even though it is somewhat long. Once you read the article, that should convince you for life.

Coconut Oil

The medical and scientific communities are now fairly united in the opinion that **hydrogenated vegetable and seed oils should be avoided**. These unsaturated fats, artificially manipulated into saturated fats, are also called trans fats, and no doubt you've heard about them lately. Some cities and states have now outlawed their use. There is no controversy anymore regarding the health dangers of these artificially saturated fats. These are the same damaged trans fats that have been touted as "healthy" and "heart-friendly" for the last 60 years by the vegetable and seed oil interests!

But the truth finally came out. Trans fat was rebuked, debunked, and revealed as the true enemy to good health that it has always been, regardless of what the seed- and vegetable oil shills told the American public for the last half century. Unfortunately, this rightful vilification of hydrogenated saturated fats has created a lot of confusion regarding naturally occurring saturated fats, including coconut oil. If one form of saturated fat is bad for you, the argument goes, then all saturated fat must be bad. This is not true.

The Truth about Coconut Oil

Back in the 1930's, a dentist named Dr. Weston Price traveled throughout the South Pacific, examining traditional diets and their effect on dental and overall health. He found that those eating diets high in coconut products were healthy and trim, despite the high fat concentration in their diet, and that heart disease was virtually non-existent. Similarly, in 1981, researchers studied populations of two Polynesian atolls. Coconut was the chief source of caloric energy in both groups. The results, published in the *Amer. Journal of Clinical Nutrition*, demonstrated both populations exhibited positive vascular health.

In fact, no evidence exists that the naturally occurring high saturated fat intake had any kind of harmful effect in these populations. Based on 60 years of negative public policy towards naturally occurring saturated fats, you would expect these cultures to be rife with clogged arteries, obesity and heart disease. It may be surprising to realize that the naturally occurring saturated fat in coconut oil actually has some amazing health benefits, such as: promoting your heart health; promoting weight loss, when needed; supporting your immune system health; supporting a healthy metabolism [6]; providing you with an immediate energy source; keeping your skin healthy and youthful looking; and supporting the proper functioning of your thyroid gland.

Coconut Oil's Secret Ingredient

50 percent of the fat content in coconut oil is a fat rarely found in nature called lauric acid. Lauric acid a "miracle" ingredient because of its unique health promoting properties. Your body converts lauric acid into monolaurin, which has anti-viral, anti-bacterial and anti-protozoa properties. Monolaurin is a monoglyceride which can actually destroy lipid coated viruses such as: HIV, herpes, measles, influenza virus, and various pathogenic bacteria, protozoa such as giardia lamblia.

Lauric acid is a powerful virus and gram-negative bacteria destroyer, and coconut oil contains the most lauric acid of any substance on earth! Capric acid, another coconut fatty acid present in smaller amounts, has also been added to the list of coconut's antimicrobial components. This is one of the key reasons you should consider consuming coconut oil, because there aren't many sources of monolaurin in our diet. But the health benefits of coconut oil don't stop there.

The Benefits of Medium-Chain Fatty Acids

Coconut oil is about 2/3 medium-chain fatty acids (MCFAs), also called medium-chain triglycerides or MCTs. These types of fatty acids produce a whole host of health benefits. Coconut oil is nature's richest source of these healthy MCFAs.

By contrast, most common vegetable or seed oils are comprised of long chain fatty acids (LCFAs), also known as long-chain triglycerides or LCTs. These long-chain fatty acids are *not as healthy for you* as the MCFAs found in coconut oil. LCFAs are difficult for your body to break down -- they require special enzymes for digestion. LCFAs put more strain on your pancreas, liver and your entire digestive system. LCFAs are predominantly stored in your body as fat. LCFAs can be deposited within your arteries in lipid forms such as cholesterol.

In contrast to LCFAs, the MCFAs found in coconut oil have many health benefits, including the following beneficial qualities. MCFAs are smaller. They permeate cell membranes easily, and do not require special enzymes to be utilized effectively by your body. MCFAs are easily digested, thus putting less strain on your digestive system. MCFAs are sent directly to your liver, where they are immediately converted into energy rather than being stored as fat. MCFAs stimulate your body's metabolism, leading to weight loss.

Coconut Oil Helps Fight Diabetes

Your body sends medium-chain fatty acids directly to your liver to use as energy. This makes coconut oil a powerful source of instant energy to your body, a function usually served in the diet by simple carbohydrates. But although coconut oil and simple carbohydrates share the ability to deliver quick energy to your body, they differ in one crucial respect. Coconut oil does not produce an insulin spike in your bloodstream.

Indeed, coconut oil acts on your body like a carbohydrate, without any of the debilitating insulin-related effects associated with long-term high carbohydrate consumption! Diabetics and those with pre-diabetes conditions (an exploding health epidemic in America), should immediately realize the benefit of a fast acting energy source that doesn't produce an insulin spike in your body. Coconut oil added to the diets of diabetics and pre-diabetics has been shown to help stabilize weight gain, which can dramatically decrease your likelihood of getting adult onset type-2 Diabetes.

Coconut Oil, Friend to Athletes and Dieters

If you live in the US, you have an almost 70% chance of being overweight. One of the best benefits of coconut oil lies in its ability to help stimulate your metabolism. Back in the 1940s, farmers found out about this effect by accident when they tried using inexpensive coconut oil to fatten their livestock. However coconut oil made the animals lean, active and hungry. Many animal and human research studies have demonstrated that replacing LCFAs with MCFAs results in both decreased body weight and reduced fat deposition. In fact, the ability of MCFAs to be easily digested, to help stimulate the metabolism and be turned into energy has entered the sports arena. Several studies have now shown that MCFAs can enhance physical or athletic performance.

Additionally, research has demonstrated that, due to its metabolic effect, coconut oil increases the activity of the thyroid. A sluggish thyroid is one reason why some people are unable to lose weight, no matter what they do. Besides weight loss, there are other advantages to boosting your metabolic rate. Your healing process accelerates. Cell regeneration increases to replace old cells, and your immune system functions better overall.

Coconut Oil and Your Heart

Heart disease is the number one cause of death in the U.S. And heart disease is often a silent killer. The first sign of cardiovascular disease is commonly a heart attack, and sadly, over one third of heart attacks are fatal. And despite the propaganda, the truth is this: it is UNSATURATED fats that are primarily involved in heart disease, not the naturally occurring saturated fats, as you have been led to believe. Plus, the polyunsaturated fats in vegetable and seed oils encourage the formation of blood clots by increasing platelet stickiness. Coconut oil helps to promote normal platelet function.

Coconut Oil in Your Kitchen

Extra-virgin olive oil is a monounsaturated fat that works great as a salad dressing. However, it should not be used for cooking. Due to its chemical structure, heat makes it susceptible to oxidative damage. And polyunsaturated fats, which include common vegetable oils such as corn, soy, safflower, sunflower and canola, are absolutely **the worst oils to use in cooking.** These omega-6 oils are highly susceptible to heat damage because of their double bonds. Discard these vegetable oils in your cabinets. Why?

Reason # 1:

Most people believe that frying creates trans-fat. That is not the major problem, in my opinion. Although some are created, they are relatively minor. There are FAR more toxic chemicals produced by frying omega-6 oils than trans-fat. Frying destroys the antioxidants in oil and as a result oxidizes the oil. This causes cross-linking, cyclization, double-bond shifts, fragmentation and polymerization of oils that cause far more damage than trans-fat.

Reason # 2:

Most of the vegetable oils are GMO. This would include over 90 percent of the soy, corn and canola oils.

Reason # 3:

Vegetable oils contribute to the overabundance of damaged omega-6 fats in your diet, which creates an imbalance in the ratio of omega-6 to omega-3. Excessive consumption of damaged omega-6 fats contributes to many health concerns. They are all highly processed and consumed in amounts that are about 100 times more than our ancestors did a century ago. This causes them to distort the sensitive omega-6/omega-3 ratio which controls many delicate biochemical pathways which results in accelerating many chronic degenerative diseases.

Only coconut oil is stable enough to resist mild heat-induced damage, while it also helps you promote heart health and even supports weight loss and thyroid function. So, to cook, use coconut oil instead of butter, olive oil, vegetable oil, margarine, or any other type of oil.

Coconut Oil Safety

The medium-chain fats in coconut oil are considered so nutritious that they are used in baby formulas, in hospitals to feed the critically ill, those on tube feeding, and those with digestive problems. Coconut oil

has even been used successfully by doctors in treating aluminum poisoning. Coconut oil is exceptionally helpful for pregnant women, nursing moms, the elderly, those concerned about digestive health, athletes (even weekend warriors), and those of you who just want to enhance your overall health.

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Note: This information on coconut oil was derived from Dr. Joe Mercola's web site. Dr. Mercola is the founder of the world's most visited natural health web site. You can obtain additional information from his excellent web site at www.mercola.com

As for obtaining coconut oil, I obtain mine from vitacost.com. They have a very high quality product, item #NUV 2200052, \$20.49 for 54 ounces. It is an extra-virgin coconut oil, not refined, not deodorized or bleached. It is organic and so is free of GMO's, hexane and pesticides. They press their coconuts within 2 hours of chopping. There is a fix fee for shipping of \$4.99 for any quantity ordered.

3. More on Cooking Oils. I have already addressed this issue above. Let me add another point. Another issue with fats and cooking oils is that fats and oil yield a compound called arachidonic acid (AA) which generates an enzyme that stimulates prostate cancer cells. The following is a list of oils and their AA content: Safflower oil (74%), corn (58%), cottonseed (52%), soybean (51%), and peanut (52%). The healthiest oil you should consume is coconut oil. It has direct anti-cancer benefits and most alternative health care practitioners have noted that it is the likely most healthy oil to use.

4. Meat Fats. Now the next biggest issue in the diet prostate cancer world is arachidonic acid (**AA**) from meat fats. Meat fat does contain AA as does many other fats and such things as French fries [since they are fried in those other AA fats] or cheese made from some fat. So eating lean meat is well worthwhile. Some physicians [who quite frankly are not paying a lot of attention] generalize to not eating much meat. But if the meat is lean, then the AA issue is minimized. Now here is the really good news!!

The omega 3 fatty acids EPA and DHA inhibit [i.e., block] AA and its resulting enzyme creation which stimulates prostate cancer cells. This is really good news. So when you do wind up having some fries or some fatty meat, take a fish oil capsule afterwards as an antidote. One product is Swanson's Fish Oil, #SWE026, \$6.39 for 100 softgels. Take one after that salmon dinner (yes, there is a fair amount of fat in the salmon) or those fries or any "lapse" that occurs now and then to keep life interesting. If you really indulged, take two.

If you don't have much in the way of fatty foods or cheeses, just use it on an "as needed" basis but if there is a regular ingestion of lots of animal fat, then consider just taking two capsules daily. As a useful precaution (since AA can sneak up on you in many ways), consider a regular addition of at least one a day if you can add it to your regimen. **Note:** The broader issue of fats and cancers is treated far more extensively below.

5. Sugars and Carbs. This issue may be the next most important one and we have already addressed it above. The key points bear repeating. Most of us simply eat too much sugar. So that part is easy — cut the sugar whether in pastry, donuts, sweets, etc. At least cut way back. The more aggressive your cancer, the more you need to eliminate sugar and carbs [which as we have said, become sugar shortly after eating them].

Now we all know that we are supposed to eat fruits and vegetables. True, they are healthy. But many fruits are very high in sugar. We want to focus on the low-sugar-content fruits. It does not matter that fruits are good for us and must be healthy. Get a list of low fructose (sugar) fruits and eat accordingly. Now comes the hard part.

All carbs — all of them — are converted to sugar shortly after you eat them. This point simply bears repeating (and repeating): carbs turn more or less immediately into sugar as soon as we eat them. Potatoes, that healthy brown rice, those healthy fiber-rich beans, pasta, whole wheat brown bread, etc. They all convert to sugar — right after eating them — and then you just ate your body weight in sugar. Many otherwise healthy vegetables and foods are simply sugar — waiting to happen.

So you must become very carb conscious. A huge number of "low(er) fat foods" such as low-fat mayo might seem healthy, but in fact the mayo has much higher carb content than just regular mayo. In fact, most low(er) fat foods have much higher carb content [partly it is because they have put in the unhealthy soy oil or just sugar to add back taste]. Regular cream cheese is better for you than low fat cream cheese. It has lower carbs, and hence lower net sugar— and if your AA ingestion level is rising due to any fat, you can add a fish oil capsule later. So lower your carbs significantly to lower your sugar.

6. Calcium. There has been some issue that calcium causes prostate and/r other cancer or dairy increases it. The key is to be sure you have enough vitamin D3 daily (5000 IU daily) since when this is

taken, the negative effects of calcium or dairy are eliminated. Most physicians seemed to have missed this follow-up news. In addition, the research on calcium's effect itself has been updated. Here are the essential results.

Park et al. have just reported the results of the truly vast NIH-AARP Diet and Health Study, which included data from more than 490,000 older men and women (aged between 50 and 71 when the study started in the 1990s) over an average 7-year follow-up period. The results of the study clearly show that, in older Americans, a dietary calcium intake level of up to 1300 mg/day *in the form of dairy and other foods* has a significant impact on risk for cancers of certain types — but *not* on risk for prostate cancer.

It has long been hypothesized that dairy foods and calcium intakes play roles that differ among individual cancer sites, but the evidence has been limited and inconsistent. Park et al. examined dairy food and calcium intakes in relation to total cancer as well as cancer at individual sites using a food frequency questionnaire. Incident cancer cases were identified through linkage with state cancer registries. The results of the study showed that: During an average of 7 years of follow-up, there were 36,965 and 16,605 cancer cases in men and women, respectively.

Calcium intake was not related to total cancer in men but was associated with total cancer in women: the risk *decreased* up to approximately 1300 mg/day, above which no further risk reduction was observed.

In both men and women, dairy food and calcium intakes were inversely associated with cancers of the digestive system.

Decreased risk was particularly pronounced with colorectal cancer.

Supplemental calcium intake was also inversely associated with colorectal cancer risk.

The authors conclude, “Our study suggests that calcium intake is associated with a *lower risk of total cancer* and cancers of the digestive system, especially colorectal cancer. It was noted that although some previous studies have linked diets high in calcium with prostate cancer, the current study found no such risk.

7. Butter — Not Margarine. Stay away from the chemical factories such as margarine. The toxic impact on your body of these phony chemically composed products is far worse than any (incorrect) worry about polyunsaturated fats or the like. If the label of anything reads like a chemical text, avoid it. Again, fats in general are treated below.

8. Fish — Generally Not. Fish used to be healthy — until it became laden with mercury. Do not eat any large fish (such as swordfish). Do not eat tuna. Avoid farm-raised fish to the largest measure (since they are extremely toxin-laden). Quite frankly, although a fish-lover myself, I hardly eat any fish any more. Wild-caught salmon could be worthwhile. Fish infrequently is not a terrible idea. But unfortunately it is no longer a very safe food — and the situation is only getting worse. Update: It has gotten worse – many national advisors essentially no longer eat fish.

9. Organic Meat. If I had the money, I would only eat organically raised beef and chicken. The regular stuff is filled with antibiotics, growth hormones, fed genetically modified soy rinsed in hexane,

and so forth. Ultimately you are ingesting things that are toxic and can only facilitate problems such as cancer. For those whose budgets allow it (and if there is availability), organic meat is a wise investment.

10. Tea and Pure Water. Tea is terribly healthy for you. That is one reason we include green tea supplements on the supplement list. Drinking it daily is also a very worthwhile idea. In addition, buy some sort of water filter and filter your drinking water. It might just be a water pitcher filter or a top-of-the-sink product. But filter your water — again there are just too many toxic substances in even the better water systems. Those toxins are ultimately carcinogenic.

By the way, tea especially black tea is high in aluminum. Lemon in tea multiplies the aluminum toxicity up to seven-fold. Hence white tea (lowest in aluminum) or green tea are more healthy – but in any case, definitely omit the lemon.

11. Consume Added B6 for Prostate cancer

This recommendation relies in part of the following study which I will reprint for you below.

Summary: In a population-based, prospective study involving 525 men diagnosed with prostate cancer between 1989 and 1994, who were followed up with for a median 6.4 years, dietary intake of vitamin B6 was found to be inversely associated with prostate cancer-specific death (HR=0.71), particularly in men with a diagnosis of localized-stage disease (HR=0.71), but not advanced-state disease (HR=1.04), when comparing subjects in the highest versus the lowest quartiles of vitamin B6 intake. No significant associations were found between intakes of folate, riboflavin, vitamin B12, and methionine and prostate cancer survival.

The authors conclude, "A high vitamin B-6 intake may improve prostate cancer survival among men with a diagnosis of localized-stage disease." [Citation: "One-carbon metabolism-related nutrients and prostate cancer survival," Kasperzyk JL, Fall K, et al, **Am J Clin Nutr**, 2009; 90(3): 561-9.]

As for foods that contain B6, here are some examples: Potato, baked, with skin 1 medium 0.70 mg, Banana 1 medium 0.68 mg, Salmon 3 ounces 0.48 mg, Chicken, light meat without skin 3 ounces 0.46 mg, Spinach, cooked 1 cup 0.44 mg, Avocado ½ medium 0.42 mg, Turkey, without skin 3 ounces 0.39 mg, Hamburger 3 ounces 0.39 mg, Fish 3 ounces 0.29 mg, Collard Greens ½ cup 0.17 mg, Brown Rice ½ cup 0.13 mg, and Green Peas ½ cup 0.11 mg.

You can easily obtain your recommended daily allowance from food sources, but if you are experiencing overt prostate cancer, I would suggest you include a single B-complex supplement addition. For example, consider Swanson's SW057, a B-complex supplement with 50 mg of B6 plus other B vitamins. It is 3.99 for 100 capsules. I would take one a day. The reason for the B-complex rather than just B6 is that this keeps your overall B vitamins in balance with each other.

12. Reduce Selected Amino Acids (primarily those in nuts)

The bottom line is this: The excessive consumption of arginine **blocks cancer cell death** (via the process of autophagy) while glutamine does the opposite. So nuts which contain a highest concentration of arginine are to be avoided on this protocol. Here is the extended science and documentation behind this observation:

Some amino acids act as metabolic sensors in the body. In many respects, they act like hormones by virtue of their ability to turn specific biochemical pathways on and off. Four notable amino acid sensors are leucine, arginine, glutamine and isoleucine. Remember that cancer is inhibited by inhibiting the mTOR pathway. So the following conclusions arise:

Both arginine and leucine *activate* the mTOR anti-autophagy pathway. Both are to be avoided. [15010853, 18056791, 17623010].

Glutamine, on the other hand, *inhibits* the mTOR pathway and is an antagonist to leucine [17978888, 15567168].

Leucine *activates* mTOR signaling partly by its ability to inhibit the AMP kinase [17178807].

Glutamine *inhibits* mTOR signaling by inhibiting the PI-3K/AKT pathway [14621121].

The AKT pathway DIRECTLY activates mTOR [17786026]. We see from the above that in particular, nuts, which contain a highest concentration of arginine, are to be avoided on this protocol.

13. Fats and Cancer

This is a more complex and misunderstood area of research. We provide our assessment below.

Saturated fat and cancer are widely believed to be closely linked. Many doctors, nutritionists and health authorities believe that a high intake of saturated fat increases the risks of cancer – just as they believe that saturated fat increases the risks of heart disease. As closer examination of the disease patterns tells us, however, that there is no positive relationship between saturated fat and cancer. Just as in the case of heart disease, the same situation applies.

Until early last century, people ate large amounts of saturated fat and used it as their main form of cooking oil – pork lard in China, butter in Europe, ghee in India, coconut oil in the tropics. Yet cancer was rare. The rate of cancer began to rise sharply only in recent decades – when the consumption of saturated fat actually fell. Thus, we need to seriously re-consider the idea that saturated fat and cancer are closely related.

The McGovern Committee report

The idea that saturated fat causes cancer began to form in the 1950s, when Ancel Keys stated that saturated fat raised cholesterol levels and caused heart disease. As we saw in the research on causes of coronary heart disease, this idea is badly mistaken. However, many scientists embraced this idea and, by the 1970s, saturated fat acquired a strong reputation as the “bad fat” – even though it is medically known that saturated fat is necessary for many body functions. In 1977, the US Senate Select Committee on Nutrition and Human Needs, chaired by Senator George McGovern, released its Dietary Goals for the United States.

The Dietary Goals stated categorically that "the over-consumption of fat, generally, and saturated fat in particular. . . have been related to six of the ten leading causes of death. . ." in the United States. The McGovern Committee report claimed that a high fat diet caused cancer. The report said saturated fat and cancer – particularly breast cancer and colon cancer – were closely linked. It urged for Americans to substitute polyunsaturates for saturated fat from animal sources – use margarine and corn oil instead of butter, lard and tallow. The rest of the world followed America's example.

Testimony of Dr Fred Kummerow

Among the scientists who testified before the McGovern Committee was Dr Fred Kummerow of the University of Illinois. He disagreed with the view that there were links between saturated fat and heart disease, as well as links between saturated fat and cancer. Fred Kummerow pointed out that the real harm was caused by trans fat in products like margarine. He also warned against soft drinks, which contained large amounts of sugar. And, Fred Kummerow pleaded for a return to traditional foods rich in saturated fats. However, the testimony of Fred Kummerow was buried in the voluminous McGovern Committee report. His views and the scientific evidence he presented were largely ignored.

Saturated fat and cancer Mary Enig's research

At the time the McGovern Committee released its Dietary Goals for the United States, Mary Enig was a graduate student at the University of Maryland. Mary Enig, who was familiar with the research of Fred Kummerow, noted that the McGovern Committee report claiming a strong link between saturated fat and cancer contradicted many real life situations:

In America, the consumption of saturated fat had been declining steadily since the turn of the century, yet the cancer rate was rising sharply.

Greece had the same level of dietary fat intake as Israel, but only one-fourth the breast cancer rate.

Spain had a slightly higher dietary fat intake than France or Italy, but only one-third the mortality rate from breast cancer.

Puerto Rico, with a high animal fat intake, had a very low rate of breast and colon cancer.

The Netherlands and Finland both have the same level of animal fat intake – about 100 grams per person per day. But the Netherlands has twice the rate of both breast and colon cancer. The difference was that people in the Netherlands consumed 53 grams of vegetable fat per person compared to 13 grams in Finland.

This seems to suggest that there is a stronger link between vegetable oil, or polyunsaturated fat and cancer, than between saturated fat and cancer.

A study in Cali, Columbia found a fourfold excess risk for colon cancer in the higher economic classes, which used less animal fat than the lower economic classes.

A study on Seventh-Day Adventist physicians, who avoid meat found they had a significantly higher rate of colon cancer than non-Seventh Day Adventist physicians.

Mary Enig analyzed the US Department of Agriculture data that the McGovern Committee had used and she reached an opposite conclusion:

There is a strong positive correlation between total fat / vegetable fat and cancer. There is a strong negative correlation or no correlation between animal fat or saturated fat and cancer deaths. In other words, Mary Enig found that the use of vegetable oils seemed to predispose to cancer and animal fats seemed to protect against cancer.

Saturated fat and cancer Harvard University Research

More recent research by Prof Walter Willett at the Harvard School of Public Health provides further evidence that there is no real connection between a high fat diet and cancer. Nor is there any link between saturated fat and cancer. Walter Willett's research at Harvard includes the Nurses Study II, which monitors the long-term health condition of nearly 116,000 women, and the Health Professionals Study, which monitors the long-term health condition of 52,000 men.

These studies reveal that the type fat has a far greater effect on cancer risks than the amount of fat. In other words, quality matters more than quantity. The website of the Harvard University School of Public Health reports:

Fat and breast cancer

Initially, international comparisons showed higher breast cancer rates in countries with higher per capita fat intake. But as more detailed studies were performed over the next couple of decades, *the apparent link between total fat intake and breast cancer has faded.*

Fat and colon cancer

As with breast cancer, international comparisons initially suggested an association between total dietary fat intake and colon cancer risk. But later studies contradicted these earlier findings and revealed instead an association that was weak at best.

However, Harvard researchers found that although fat and saturated fat intake did not seem to increase colon cancer risk, high consumption of red meat appears to increase colon cancer risks.

Fat and prostate Cancer

Here, the evidence is contradictory. There is some evidence that diets high in animal fat and saturated fat increase prostate cancer risk. However, some studies have also shown no association, while others have implicated unsaturated fats [Note from Evans – when I looked in depth at the studies, the saturated fats list included, for example, ice cream and cheese spreads – both of which are either high in sugar or nitrates and trans-fats – now known to be cancer instigators. That is, the definition of saturated fats in the prostate research was quite naive; also see the comment about trans-fat below].

Fat and other Cancers

In the Nurses' Health Study, Harvard researchers found that a high intake of trans fats increased the risk for non-Hodgkin's lymphoma and that a high saturated fat intake increased the risk for endometrial cancer.

Saturated fat and cancer

Although the Harvard website did not specially mention a lack of correlation between saturated fat and cancer, its findings can be taken to include saturated fat as part of overall fat intake. One of the problems with earlier scientific studies on saturated fat and cancer is that the researchers often do not take into account trans fats. Even Walter Willett and his team of Harvard researchers failed to make this distinction until the 1990s. Once they took trans fats into account, they discovered that trans fats are often the real culprits in causing heart disease, cancer, diabetes, obesity and other modern, degenerative diseases.

So before the 1990s, Walter Willett found, like most other researchers, that saturated fat “caused” heart disease and cancer. But in the early 1990s, his researchers contacted Mary Enig for data on trans fats. Working with the refined data, Walter Willett confirmed, in the Nurses Health Study II, that nurses with higher rates of cancer were those who consumed more margarine and vegetable shortenings – ***not*** those who ate butter, eggs, cheese and meat. In other words, he no longer found any link between saturated fat and cancer, but now found a strong link between trans fats and cancer. This correlation between trans fat and cancer was never published, but was reported at the Baltimore Data Bank Conference in 1992.

Unfortunately, even many present day research studies continue not to make any distinction between saturated fat and trans fat. And so we continue to get reports every now and then about links between saturated fat and cancer, heart disease and various other diseases. As more researchers work with better and more accurate data, a clearer picture will emerge showing that, just as there was no link between saturated fat and cancer a hundred years ago, there is no link today.

The problem is not saturated fat – which even protects against cancer in certain cases. In fact, even trans fats that occur naturally – in the milk and meat of cows and other grass-fed animals – are known to protect against cancer. The real problem is with artificial trans fats, and other types of artificial foods [e.g., see my note about cheese spreads above]. What I believe we may conclude is that non-trans-fat is not implicated in cancer.

Chapter 10 – Cancer Pain: A Natural Option

This section deals with the problem of cancer pain, often arising from a metastatic condition. There is one supplement about which you should be aware that can prove somewhat beneficial. The supplement is called Bromelain which I will explain below.

Bromelain is usually sold as a digestive aid and indeed it is. However it had been discovered that when taken on an entirely and totally empty stomach, it acts as a pain reliever. Big time. Now it is not like a double shot of morphine – it is more like a double-strength Advil. So what's the point?

Bromelain is simply highly condensed extract from pineapple. Unless you are allergic to pineapple, you can take it without worry. And there is the beauty of it. You are limited to the number of Advil you can take. Ibuprofen will give you ulcers and you must pace yourself. But the Bromelain is just pineapple. You can take it with Advil or ibuprofen or anything. There is no worry about “doubling up.” It is like a free extra double-strength Advil but you don't have to count it on your total Advil or ibuprofen limits.

I had one prostate cancer client who had contacted me due to his metastasis throughout his body. I recommended Bromelain and he was able to halt all other pain medication other than the Bromelain. Thus he was cogent to his dying day. Still, Bromelain is just helpful for most, not a total pain panacea.

There are two keys to using bromelain. You need to take the full-strength version. This will usually say something like “2040 GDU per capsule” – or higher. This is the one with the needed intensity since there are cheaper weaker versions with 240 GDU's per capsule but this does not have enough power. The cheapest I can find is Swanson's product SWU235, \$15.99 for 100 capsules. Use as you would a double-strength Advil, but without the worry of its impact on your stomach lining.

The second key principle was noted above - it **MUST** be on an entirely empty stomach. If you have food in your stomach, the Bromelain will combine and help your digestion. This is all well and good – but you will not get ANY pain relief. It must be on an empty stomach. Now when you wake up in the morning, your stomach is empty. However keep in mind that you can be digesting food two or more hours after you have eaten. Take this into account so that you don't waste this supplement. Note that if you can find any Bromelain anywhere on the web with at least 2000 GDU per capsule that is cheaper, buy it. There is nothing whatsoever about the Swanson product other than the fact that it was the least expensive I can find so far. Bromelain is bromelain so don't feel you have only one supplier.

Chapter 11 – Osteoporosis: A Natural Approach

Some of the drugs you may be taking for your cancer can possibly lead to osteoporosis. Some of you may take added calcium thinking this will help you. You are mistaken. Let me explain why. The body does not use calcium to build bones – without other co-factors – since these other elements are required to enable the body to build bone. You must take calcium with its co-factors for bone building or you are wasting your time.

You want to take the following supplements since it takes all seven to maintain maximum bone integrity. As noted, just taking calcium does no good and even is counter-productive. We have identified possible products from the Swanson Vitamin Company (we have no connection with them) which is at 800-437-4148 or find them at www.swansonvitamins.com — but any comparable product is fine (e.g. Puritan Pride, Vitamin Shoppe, etc.).

1. Calcium. First, take about 1200 mg of calcium per day, being absolutely certain you take the calcium with food in your stomach. One product to consider is Swanson's CalciBoost, which has 600 mg of calcium per capsule, and one would take 2 a day. It is item #SW795, \$3.59 for 120 capsules which will last two months. [Swanson is at www.swansonvitamins.com or call 800-437-4148]. If your bones are in good shape, you actually can just take about 600 mg a day and still maintain bone integrity. The 1200 mg of calcium is your total daily target and many people consume in their food about 600 mg already. You do not want to take excessive calcium. If you take the rest of the supplements itemized below, 1200 total mg of calcium **including** what is in your diet is likely optimal while more than that will begin to be counter-productive.

2. Magnesium. Second, magnesium should be in the ratio of at least one part magnesium to two to three parts calcium to facilitate the incorporation of calcium into the bone structure. The magnesium can be as high as a ratio of one-to-one to calcium which researchers are coming to believe is the better choice. Target about 400 mg to 600 mg of magnesium per day (or even 800 mg per day if you are taking 1200 mg of calcium). Swanson has a product, #SN120, 425 mg of magnesium malate per capsule, \$8.61 for 180 capsules. Take one a day (up to 2 a day).

3. Vitamin D3. The third thing you want to take is vitamin D3 [Note that you will be taking this for other reasons in one of the earlier protocols but it is repeated here for completeness]. Calcium cannot be absorbed in the intestine unless there is vitamin D3 present (plus the magnesium above). Most Americans are vitamin D3 deficient. Order from Swanson's their 2000 IU vitamin D3 capsule, #SW1210, \$6.39 for 250 capsules -- take 2 a day. The new recommendation (in the year 2000) from the Institute of Medicine to NIH is for the amount of 2000 IU as a daily tolerable limit which adults can take every day without any side effects, so it is not too high, and most valuable. The newest recommendation from the Institute of Medicine based on the advice from the world's leading experts on vitamin D3 was revised to 2 a day (4000 IU per day), but NIH has yet to act on this recommendation. Cutting edge research [*American Journal of Clinical Nutrition*, Vol. 87, No. 6, pp. 1952-1958, June 2008] demonstrated a needed amount of 3800 IU daily for those with no deficiencies. Thus the 4000 IU level is an extremely good target with world-class evidence and expertise behind it. I note that

nationally-published researchers are taking a total of 5000 IU daily and recommending it in their newsletters.

4. Boron. Add boron to your regimen. Look at Swanson's boron, a 3 mg capsule, item #SW599 for \$3.79 for 250 capsules. Take one a day. Note: If you are taking a Multi-vitamin and Mineral daily supplement with 2-3 mg of boron, you need not add any more.

5. Copper. Ideally you would add 2 mg of copper to your regimen. Swanson has product #SW223, a 2 mg tablet, 300 for \$2.39 — take one a day. Note: If you are taking a Multi-vitamin and Mineral daily supplement, with 1-2 mg of copper, you need not add any more.

6. Vitamin K2. Ideally you would add vitamin K2 (this is not the common vitamin K in supplements which is also called K1). K2 is very useful in bone development. One would want to add 100-200 mcg per day. Note that you need the "other" form of vitamin K, called K2 or sometimes K-7. For example, there is Swanson's product JR091, a 90 mcg tablet for \$12.97 for 60 softgels. Note that the other types of vitamin K (vitamin K1) will not aid bone development as the K2. Take two a day.

7. Strontium. This element has been extensively shown to aid in building the bone matrix which adds to your bone density. The proper osteoporosis prevention regimen must include it. Take about 700 mg per day. Swanson has a product, #SW1195, which is a 340 mg capsule of strontium, \$7.49 for 60 capsules. One would take 2 a day. Note — do not take strontium at the same times as you take the calcium since they both compete for the same binding sites. If taken together, the calcium will knock out the strontium. So take both capsules together at a time when you are not taking your calcium.

I have had many clients eliminate their osteoporosis [as diagnosed with bone density scans] by using this protocol. It is widely known and the evidence is extremely strong in its favor. This one is a no-brainer.

Chapter 12 – Radiation: Protecting Yourself

If you are undergoing radiation, there are some simple things you can do that will facilitate the radiation while protecting you from collateral damage. These three steps are itemized below.

1. Green tea Polyphenols. To construct a plan, recognize that x-rays create a barrage of free radicals and can do DNA gene damage. To fight this cellular attack, first, take green tea polyphenols which have been shown to block whole-body radiation.¹

Consider Swanson's product NWF240, an intense green tea polyphenol product, \$13.76 for 180 capsules [Swanson is at swansonvitamins.com or 800-437-4148 or also for example Puritan's Pride at puritan.com which is another reliable supplier. We have no connection with these or any other suppliers]. Take one a day two weeks before your radiation exposure all the way through two weeks afterwards.

2. Meriva. The second key product to reduce the inflammation caused by radiation is curcumin. Remarkably, curcumin protects normal cells from radiation but enhances the death of tumor cells.² It has also demonstrated increased survival in animals exposed to high dose radiation.³ I suggest Swanson's Meriva, an intense curcumin supplement. This is Swanson's product SWU493, \$9.99 for 60 capsules. Take one twice a day with some food also two weeks before, during, and after radiation exposure.

Question – since we noted above that green tea and curcumin inhibit each other, should you take both? Although I am not certain, I believe that the mechanism of action of the green tea for radiation is different than its use in the cytotoxic protocol so that there would be no conflict. Also the mechanism of action for the curcumin seems to be different. Hence the prospects for conflict again are reduced. Thus I believe taking both for radiation protection will be a suitable choice.

3. Ginkgo Biloba. The third product to take is ginkgo biloba. Ginkgo has been shown to protect organs from radiation damage as well as protect cells such as after radioactive iodine treatment.⁴ Its effect is so pronounced that it was useful in treating workers at the Chernobyl nuclear plant explosion long after their original exposure.⁵ Adding more evidence for ginkgo, in one very important study⁶, researchers divided 25 patients into two groups getting radiation. One subgroup received 120 mg of ginkgo biloba daily and the other a placebo. The ginkgo prevented the expected DNA damage without interfering with the radiation. Swanson has product #NWF309, a standardized 120 mg capsule, \$9.89 for 100. Also take 2, two weeks before and after exposure.

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Chapter 13 – Getting Good Advice About Cancer: Other Sources

1. Personal Consultations from Dr. Ralph Moss

Dr. Ralph Moss offers written personal consultation/report for people with cancer, providing detailed information, up to 400 pages, on promising alternative treatments and their sources. The fee is \$300. Follow-up written questions and answers are included. Call 1-800-980-1234 or 1-814-238-3367, 9AM-5PM EST, Monday-Friday. He also has a website: www.cancerdecisions.com

Dr. Ralph Moss has written the book, *Questioning Chemotherapy*, which documents the ineffectiveness of chemotherapy in treating most cancers. On November 19, 1977, he was fired for telling the public the truth. At a press conference on November 18th, he and the Second Opinion working group released a well-documented 48-page report that stated the top officials of the Memorial Sloan-Kettering Cancer Center had lied about the results of a study performed at the center regarding "Laetrile" – (a natural, alternative cancer treatment).

Dr. Moss has gained credibility by writing eight books, including his most recent work, *Cancer Therapy: The Independent Consumer's Guide to Non-Toxic Treatment*. He also wrote *The Cancer Industry*, a documented research work telling of the enormous financial and political corruption in the "cancer establishment". He indicates that the motivating forces in cancer research and treatment are often power and money, and not the cure of cancer patients. He also writes, *The Cancer Chronicles*, a newsletter reporting on new cancer treatments and preventive measures.

Dr. Moss' work documents the ineffectiveness of chemotherapy on most forms of cancer. However, he is fair in pointing out that there are the following exceptions: Acute Lymphocytic leukemia, Hodgkin's disease, and nonseminomatous testicular cancer. Also, a few very rare forms of cancer, including choriocarcinoma, Wilm's tumor, and retinoblastoma. But all of these account for only 2% to 4% of all cancers occurring in the United States. This leaves some 96% to 98% of other cancers, in which chemotherapy doesn't eliminate the disease.

The vast majority of cancers, such as breast, colon, and lung cancer are barely touched by chemotherapy. However, there is another category where chemotherapy has a relatively minor effect -- The most "successful" of these is in Stage 3 ovarian cancer, where chemotherapy appears to extend life by perhaps eighteen months, and small-cell lung cancer in which chemotherapy might offer six more months.

Effective cancer treatment is a matter of definition. The FDA defines an "effective" drug as one which achieves a 50% or more reduction in tumor size for 28 days. In the vast majority of cases there is absolutely no correlation between shrinking tumors for 28 days and the cure of the cancer or extension of life. When the cancer patient hears the doctor say "effective," he or she thinks, and logically so, that "effective" means it cures cancer. But all it means is temporary tumor shrinkage. Chemotherapy usually doesn't cure cancer or extend life, and it really does not improve the quality of the life either. Doctors frequently make this claim though. There are thousands of studies that were reviewed by Dr. Moss as part of the research for his book -- and there is not one single good study documenting this claim.

What patients consider "good quality of life" seems to differ from what the doctors consider. To most it is just common sense that a drug that makes you throw up, and lose your hair, and wrecks your immune system is not improving your quality of life. Chemotherapy can give you life-threatening mouth sores. People can slough the entire lining of the intestines! One longer-term effect is particularly tragic: people who've had chemotherapy no longer respond to nutritional or immunologically-based approaches to their cancers. And since chemotherapy doesn't cure 96% to 98% of all cancers anyway...People who take chemotherapy have sadly lost their chance of finding another sort of cure.

It's especially telling that in a number of surveys most chemotherapists have said they would not take chemotherapy themselves or recommend it for their families. Chemotherapy drugs are the most toxic substances ever put deliberately into the human body. They are known poisons, they are designed poisons. The whole thing began with experiments with "mustard gas," the horrible chemical-warfare agents from World War I. Dr. Moss' position on chemotherapy is supported by many major students of the study of cancer treatment.

Following are some examples: Dr. John Bailer is the chief of epidemiology at McGill University in Montreal and was formerly the editor of the Journal of the National Cancer Institute. In 1986 the New England Journal of Medicine published an article by Dr. Bailer and Dr. Elaine Smith, a colleague from the University of Iowa. Bailer and Smith wrote: "Some 35 years of intense and growing efforts to improve the treatment of cancer have not had much overall effect on the most fundamental measure of clinical outcome - death. The effort to control cancer has failed so far to obtain its objectives.

Dr. John Cairns, a professor of microbiology at Harvard, published his view in Scientific American in 1985, "that basically the war on cancer was a failure and that chemotherapy was not getting very far with the vast majority of cancers." As far back as 1975, Nobel Laureate James Watson of DNA fame was quoted in the New York Times saying that the American public had been "sold a nasty bill of goods about cancer."

In 1991, Dr. Albert Braverman, Professor of Hematology and Oncology at the State University of New York, Brooklyn, published an article in Lancet titled "Medical Oncology in the 1990s," in which he wrote: "The time has come to cut back on the clinical investigation of new chemotherapeutic regimens for cancer and to cast a critical eye on the way chemotherapeutic treatment is now being administered." Dr. Braverman says that there is no solid tumor incurable in 1976 that is curable today.

Dr. Moss confirms this and claims that the greatest breakthrough in the objective study of chemotherapy came from a biostatistician at the University of Heidelberg, Dr. Ulrich Abel. His critique focused on whether chemotherapy effectively prolonged survival in advanced epithelial cancer. His answer was that it is not effective. He summarized and extended his findings and concluded that chemotherapy overall is ineffective. A recent search turned up exactly zero reviews of his work in American journals, even though it was published in 1990. The belief is that this is not because his work was unimportant -- but because it's irrefutable.

With the extensive documentation in Dr. Moss' book, and all the statistics developed by the experts, why is chemotherapy still pushed by the large majority of oncologists? Dr. Moss feels that "there's a tremendous conflict going on in the minds of honest, sensitive, caring oncologists." They're in a very difficult position because they've been trained to give these drugs. And they've devoted many years to reaching a very high level of expertise in the knowledge of poisonous, deadly compounds. They're really

in a bind, because they went into oncology to help the cancer patient, yet the tools they've been given don't work. And they see what happens to physicians who "step out of line" and treat cancer with alternative means.

Armed raids, loss of licensure, professional smearing and ostracism are some of the consequences. These could all be related to the quotation in the book made by Dr. Lundberg, editor of the *Journal of the American Medical Association*. At a recent National Institute of Health meeting, he said of chemotherapy: "[It's] a marvelous opportunity for rampant deceit. So much money is there to be made that ethical principles can be overrun sometimes in a stampede to get at physicians and prescribers." You never heard that on the evening news.

The economics of cancer treatment are astounding. Cancer treatment is close to \$100 billion annually (\$100,000,000,000). The chemotherapy part of that by 1995 will be up to \$8.5 billion. Looking from another angle: the Bristol Myers company owns patents on twelve of the nearly forty "FDA-approved" chemotherapeutic drugs. The president, past president, chairman of the board, and a couple of the directors of Bristol Myers all hold positions on the board at Memorial Sloan-Kettering Cancer Center.

Dr. Moss' book details the failures (and very few successes) for chemotherapy with more than fifty types of cancer, includes a complete description of the major chemotherapy drugs, and has a section about questions to ask your doctor. All of Dr. Moss' books and Cancer Chronicles newsletters are available from Equinox Press, 1-800-929-WELL or 718-636-4433.

We are obviously losing ground with conventional cancer treatment, because the death rates keep going up. The reason for this is because conventional treatment is based on a faulty standard: That the body must be purged of cancer by aggressive and toxic methods such as surgery chemotherapy and radiation therapy. This, of course, seemed reasonable back in 1894 when William Halsted, M.D. did the first radical mastectomy, but it has proven to be so wrong over the last 50 years that continuing to adhere to it constitutes more fraud than honest mistake. However, this standard still dominates conventional cancer therapy, and until that changes, we will continue to lose ground with cancer.

2. A Report From Dr. Whitaker

Dr. Whitaker, a firm believer in Dr. Moss' work and alternative cancer therapy goes on to give some of his personal views. What is lost in the unemotional statistic of 500,000 cancer deaths per year is how those people died. Dr. Whitaker goes on to say more about the treatment of cancer:

In my opinion, conventional cancer therapy is so toxic and dehumanizing that I fear it far more than I fear death from cancer. We know that conventional therapy doesn't work -- if it did, you would not fear cancer any more than you fear pneumonia. It is the utter lack of certainty as to the outcome of conventional treatment that virtually screams for more freedom of choice in the area of cancer therapy. Yet most so-called alternative therapies regardless of potential or proven benefit, are outlawed, which forces patients to submit to the failures that we know don't work, because there's no other choice.

Because cancer treatment is such a sensitive issue, I need to set some ground rules before I tell you what I would do if I had cancer. What follows is what I personally would do. It is not a recommendation for you, and should not be considered as such. It is not even what my wife would do (that would be her decision), nor is it what my young son would do (that would be the joint decision of my wife and myself). The choices to be made in treating cancer are not easy ones, because there is so little certainty of cure in any of them. The course that someone chooses to take is very personal, and reflects not only that person's knowledge of the options, but also his/her beliefs.

Yet, because we are strongly influenced by our natural fear of death, we lineup for conventional cancer therapy, not so much believing that it will work, but hoping that it will not fail. If expensive, debilitating procedures to eliminate acne scars had the same failure rate as cancer treatment, they would be abandoned. It is only because cancer is so often fatal that conventional approaches were not abandoned long ago. We continue to use them not because they work, but because those who perform them have so vigorously eliminated any other choice.

My Imaginary Cancer Scenario (by Dr. Whitaker)

Though I would approach my own dilemma with hopes of total cure, I would be the first to admit that, regardless of the course I took, the chances of that are small. Consequently, my choices of cancer therapy are a mix of science and philosophy. They are as much a reflection of how I would struggle for survival as of how I would wish to die if the struggle failed. For the purposes of this discussion, let us assume that I have just been diagnosed with cancer of the lung, and a particularly virulent one. (Please understand that I do not have cancer, nor do I smoke.) Before going into what I would do and why, let me say what I wouldn't do, and why.

I Wouldn't Take A Passive Role

If I am going to fight for my life, I want to do just that. I am always perplexed by the news stories of some celebrity, doped to the gills with heinous poison, "courageously battling for his life." What does this mean? The celebrity, who simply accepts conventional cancer therapy, is no more "courageous" than a laboratory mouse. This is not to say that what the celebrity is doing is wrong, only that it is the very opposite of a willful act of courage.

Taking a passive role with today's conventional therapy is terribly dangerous. Recently Jackie Kennedy, after a "courageous fight," succumbed to non-Hodgkin's lymphoma - or did she? Her early demise, attributed to the cancer, was a shock to cancer specialists worldwide, and brought into question the real

cause of her death. She had been given an unproved protocol of very high-dose chemotherapy. The drugs alone could easily have caused her death - and this would not be unusual. There are numerous cases of iatrogenic (doctor-induced) deaths from chemotherapy.

I'd Actively Fight For My Life

On the other hand, the cancer patient who says, "no, thanks" to chemotherapy recommended by large cancer treatment centers, and takes off to Grand Bahamas Island to receive Immuno-Augmentative Therapy (IAT); or to Houston, Texas, to receive antineoplastons from Dr. Stanislaw Burzynski; or who heads to the public library to make a battle plan, has begun fighting and is acting courageously.

Whether I win or lose, that is the course I would take. What have I got to lose? Conventional treatment is toxic and simply doesn't work, so I would throw my lot with something safe that might work, and folks, a lot of approaches fit that description. I also believe patients who seek alternative therapies are more optimistic. They have only one worry - the cancer- not the cancer and the therapy!

And Now. Here's What I Would Do (by Dr. Whitaker)

I'd turn my back on 50 years of institutionalized expertise, because it follows the wrong paradigm. Everything that is done in medicine or in any other discipline fits some paradigm. The paradigm I use for cancer is that it is a systemic problem in which the normal control mechanisms of your body are altered. Your immune system likely bears the largest burden for this control; thus, all techniques that enhance it are promising. Those that damage it are not.

I would switch to a mostly vegetarian diet. I'd also take the Nutritional Supplements "Green foods," such as GREENS+ (800/643-1210) or Green Magma (from Healthy Directions; 800/722-8008, ext. 572). These supplements include the phyto-chemicals, antioxidants, vitamins, and minerals required for optimal health. I would enhance that basic program with the following:

Vitamin C - 10,000 mg per day in divided doses. Ewan Cameron, a Scottish physician, did a study in which 100 cancer patients were given 10,000 mg of vitamin C for the rest of their lives, while control patients were not. The patients on vitamin C lived much longer than the age-matched controls. The Mayo Clinic did two studies on vitamin C, and in both studies found that vitamin C did not help. However, both studies were set up in a manner that almost guaranteed failure. Frankly, I think that this was done intentionally to generate negative publicity for this non-toxic approach.

There are other things to take And I would be in a constant search for effective, non-toxic therapies. One place to begin that search is with Ralph Moss, Ph.D. He is probably the most knowledgeable writer in the world on alternative therapies for cancer, and has recently published a 530-page book, *Cancer Therapy, The Independent Consumer's Guide to Nontoxic Treatment and Prevention*. (Equinox Press, New York, NY, 1995). In addition, Dr. Moss offers a report service called *Healing Choices*, which ascertains, through a questionnaire, the type and severity of cancer, and suggests alternatives. This costs \$250, and it is well worth it. If I had cancer, I would start here for more information. You can get more information by sending a large SASE to The Cancer Chronicles, 2 Lincoln Square, Suite 31A, New York, NY 10023, or by calling Melissa Wolf at 718/636-4433.

Note: A subscription to Dr. Whitaker's monthly newsletter which covers alternative medicine can be obtained by going to dr.whitaker.com which is consistently filled with excellent commentary.

3. People Against Cancer

Another source of information is People Against Cancer, which provides a comprehensive counseling service called the Alternative Therapy Program. It includes a review of your medical records by a network of doctors using alternative therapies. It also costs \$250. People Against Cancer can be reached at 515/972-4444. Their Internet address is: <http://www.dodgenet.com/nocancer>. It is not a reasonable belief to think that conventional cancer experts offer the best approaches for most cancers. There is just too much evidence to the contrary.

4. Your Worst Sources

In some ways, this section hidden here in the depth of this volume may be the most important. The reason is because I am sorry to report that the worst source of information if you have cancer is your oncologist. There are many reasons why this is so.

First, your oncologist is constrained to give you what is called the standard of care. There are guidelines that the aggregate of oncologists have determined are the proper approaches for every type and stage and variation of cancer. Oh, there is some wiggle room but nothing worth writing home about. It violates the Standard of care for them to do otherwise. This creates havoc.

For one thing, nothing in this volume is part of the standard of care. So for all intents and purposes, there is nothing here for them to consider. Where they may think it might not harm you, they might tell you, essentially, “whatever.” Since anything might affect what they do, or they fear that anything might affect it, the only safe thing to protect their own legal self is to just negate anything until they are completely through. Maybe by then you are in hospice care, preparing for death. Then, do what you will. Maybe you are in “remission,” and Job Accomplished, so you can now do what you will. Otherwise, they are usually against it.

But this is hardly the reason they make the ranks for the worst source. For that you will need more information. Oncologists provide what the FDA has approved. That is more or less 100% valid. The FDA approves things that the drug companies have tested and which past the tests. It would appear so far, so good. However it must be understood that the drug companies are aq business – which is fine – and their business is to make money, also fine. Their business is not to cure cancer. Their business is to make money.

Now the rules are set so that if any chemotherapy drug can show an utter modicum of variance, extending your life for example by 2 days after the six weeks and \$100,000 of chemo, then it is an approvable drug. In my example, this is at least some extension. However the drug companies won the right from the FDA to pass an even lower test. This test is to show tumor regression or shrinking for 30 days. This so-called marker must surely be great – and the drug is hopefully approved, especially if not too many patients died. Is it really the case that tumor regression for a month or more is a net gain?

The simple answer is no. The tumor might have been beaten back temporarily, but the remaining cells are now completely impervious to the chemotherapy, and so the tumor, now stronger than ever, comes back with a vengeance. Hence many times tumor regression has nothing to do with extended life. You have paid a very high, in more ways than one, and many have in the end absolutely nothing to show for it.

I hear from patients all the time that their tumor regressed – and now they have hope – when in fact they have not actually gained a day of added life.

This is still not the reason why oncologists are the worst source of information. One of the facts of life is that oncologists make a small fortune off the chemotherapy drugs you get. When you walk into an oncologist's office, you are a \$100,000 net profit, maybe a \$250,000 meal ticket, perhaps \$500,000 ticket to ride. That chemo drug you get in his or her office is marked up innumerable times from the wholesale to the retail cost. Every series of chemo rounds is worth a new BMW to the oncologist. Now we all know they are wonderful people and we know how much you love your oncologist (as so many patients tell me). But the conflict of interest here is large enough for five Mac trucks to dive through.

They have convinced themselves that the very next round – the silver bullet perhaps – will be your salvation, but there is the unfortunate “reward” that by you trying it, they are rewarded handsomely. I once asked a very dear colleague why in the world he offered chemo in certain situations, knowing it was useless. *Well, he said, if I didn't do it, the next oncologist would promise a possible silver bullet – and he would do it and get the revenue. So I might as well do it – and I have more compassion than the others.* Actually he did. But an oncologist's advice is tainted 10 ways to Sunday.

The situation gets even worse. If they don't play ball and follow the rules, they fear getting their hides sued to kingdom come. This is not all that unlikely if the Standards of Care have not been carefully followed, no matter what. Now this tenacious adherence can put the oncologist between a rock and a hard place. For example, you will read in Chapter 14 regarding chemotherapy (especially in sections 9 and 10) that you can get a service to pre-test innumerable drugs against your tumor to find the ones (or even better, the combinations) that work best. Every tumor is different, with a different assortment of genes expressed. One drug that may work for one breast cancer may be the worst possible choice for another.

Oncologists generally abhor this service and very few work with it. Why? The main problem is that if the recommendation is different from the prescribed Standard of Care, they are in quite the dilemma. Go with what science indicates is the best, and violate the Standard of Care. Go with the standard of Care, and if the patient dies, they at least can't be faulted. Oncologists have children to support, tuition to pay at private schools, car payment, etc. So which would you choose? Nope, you're wrong. This service is hardly ever invoked. Now at least you can see why.

As for section 10 of Chapter 14, research shows that the most popular although admitted most dangerous chemo drug, adriamycin, used for breast cancer, will not work in about 92% of the cases. You need a certain gene configuration, with which the drug cannot work. The gene is known and can be tested. So the next question is, are oncologists testing for the gene to spare 92% of women the agony of a drug called within the “brotherhood” as the Red Death? Nope. It is still a Standard of Care. It may take years before this is changed. So women will get it until someone covers oncologists for choosing something else. These are the challenges your oncologist confronts.

There is one last point to make. There are a vast number of therapies used in Europe with a long history of success. Hyperthermia is one of them. So why do these other therapies remain outside the U.S.A. although Duke University just imported a hyperthermia machine to do experiments. The answer is that

any treatment, any treatment at all, must have FDA approval before it is legitimate. And before it will be reimbursed. So just who is interested in spending the tens if not hundred's of millions to validate some of these therapies.

The reader might think that surely there is a government agency that does this. No. Surely the drug companies want to cure cancer. No, as explained they are a business with a goal to increase shareholders' value. How about medical Schools? Yes medical schools have researchers and there are limited monies looking at new ideas, such as Duke I mentioned. These ultimately must feed into a business that thinks the therapies will prove substantially helpful, will convince oncologists to use it (very difficult), and finally, some significant profit will be made. It is just the way of our world – and it reduces the likelihood of such new innovations to enter our marketplace.

In summary, the oncologist is constrained with one of the most limited tool kits of any medical group in the world. However, to their great luck, their reimbursement for using these limited tools are spectacular. Your oncologist stands between you and the fear of death. You want to believe in him. In most cases, he or she even wants to help you, cure you, be your savior. This is fine. However you are likely to trust someone whose agenda is radically different from yours. Such is the challenge you face, and in part, is one reason I wrote this book.

Before you end this section, re-read Dr. Moss' commentary in section 1 of this chapter. Then you will see why Dr. Whitaker in section 2 says you must take over the intervention, you must arm yourself, you must educate yourself. I end this entire book with the warning about entering it willingly (I won't spoil the exact quote). Here I will note that you may fail to heed these warnings also at your own great risk.

Chapter 14 – Chemotherapy and Radiation: Various Issues

Below we will comment on a number of issues all pertaining to chemotherapy and/or radiation

1. General Limitations of Chemotherapy and Radiation

This section will prepare the reader for why alternative strategies such as described in the discussion above may be required, based on common US standards of care. We begin with the following news story posted February 24, 2010:

Feb. 24--Max Wicha is coming to Pittsburgh today to deliver a startling message:

Standard cancer treatments not only often fail to eradicate cancer, but can make it worse. “

That argument isn't coming from a fringe proponent of alternative medicine, but from the founder of the University of Michigan's Comprehensive Cancer Center and a pioneer in research on why cancers recur and spread to other parts of the body.

The reason breast cancer and other malignancies often return aggressively after treatment is that when tumor cells die under assault from chemotherapy and radiation, they give off substances that can reactivate a special set of master cells known as cancer stem cells, Dr. Wicha said in an interview Tuesday. Dr. Wicha's lab has found that inflammatory molecules secreted by dying tumor cells can hook up with the stem cells and cause them in effect to come out of hibernation.

The existence of cancer stem cells is still controversial in some quarters, Dr. Wicha acknowledged, but is gaining traction. In the last two months alone, researchers around the nation have published studies on cancer stem cells in breast, ovarian, prostate and brain cancer. Adult stem cells exist in most tissues, and go into action to repair damage from wounds or infections. In cancer, they can mutate and no longer obey normal bodily signals to stop growing, Dr. Wicha said.

He and other researchers say that even when chemotherapy and radiation cause tumors to shrink dramatically, these stem cells can stay alive, living under the radar until they are once again spurred into action. They also believe stem cells are probably the ones that break away from an original tumor and cause cancer to spread elsewhere in the body.

Chemo and radiation kill off the fastest-growing cells in the body, which applies to most cancer cells, but the cancer stem cells that create those rapidly dividing tumor cells actually grow much more slowly themselves, and are less susceptible to those therapies, he said. One tactic to address this problem is to kill off both types of cancer cells at once, Dr. Wicha said.

A recent experimental trial with advanced breast cancer patients at the University of Michigan, Baylor University in Texas and the Dana-Farber Cancer Institute at Harvard University used standard chemotherapy along with a substance designed to block one of the biochemical pathways of stem cells.

The approach killed off more than 90 percent of the cancer stem cells, Dr. Wicha said, and researchers now hope to expand the treatment to a much larger group of patients. Ultimately, he hopes cancer treatments can avoid general chemo altogether, with its debilitating side effects, and just use targeted

therapies against the stem cells. There is still a long road ahead, he said, and "my feeling is, to really knock these stem cells out, we're probably going to have to use multiple inhibitors."

2. Specific Limitations of Chemotherapy

The common treatments such as chemotherapy and radiation will kill some but not all the cancer cells while creating new cancer cells. There is good evidence that surgery and biopsies can and often will aid and facilitate your cancers. We have pointed out above that the more logical strategy is to halt the cancer process where it stands now — and then proceed with your life with the cancer condition simply held at bay. Those who must rid themselves of the vile cancer at any cost are more likely to do themselves more harm than good. Often they kill themselves in the process of their war on cancer. This point requires a new understanding of cancer as a condition that can be simply held at bay your whole life without harm or additional threat. You have been taught to kill any detected cancer at any cost — that cost may more frequently be your life.

The cancer patient may assume that the addition of chemotherapy and radiation may be of benefit to their well-being since it is medically-approved. We should not confuse what has been approved for treatment with that which may be useful treatment. The point of this section is to provide added evidence that the presumption that chemotherapy has some remarkable benefits is exaggerated. Consider this study:

Citation: Clinical Oncology ® College of Radiology). 2004 Dec;16(8):549-60. “The contribution of cytotoxic chemotherapy to 5-year survival in adult malignancies.”

Morgan G, Ward R, Barton M. , Department of Radiation Oncology, Northern Sydney Cancer Centre, Royal North Shore Hospital, Sydney, NSW, Australia. gmorgan1@bigpond.net.au

AIMS: The debate on the funding and availability of cytotoxic [chemotherapy] drugs raises questions about the contribution of curative or adjuvant cytotoxic chemotherapy to survival in adult cancer patients.

MATERIALS AND METHODS: We undertook a literature search for randomized clinical trials reporting a 5-year survival benefit attributable solely to cytotoxic chemotherapy in adult malignancies. The total number of newly diagnosed cancer patients for 22 major adult malignancies was determined from cancer registry data in Australia and from the Surveillance Epidemiology and End Results data in the USA for 1998. For each malignancy, the absolute number to benefit was the product of (a) the total number of persons with that malignancy; (b) the proportion or subgroup(s) of that malignancy showing a benefit; and © the percentage increase in 5-year survival due solely to cytotoxic chemotherapy. The overall contribution was the sum total of the absolute numbers showing a 5-year survival benefit expressed as a percentage of the total number for the 22 malignancies.

RESULTS: The overall contribution of curative and adjuvant cytotoxic chemotherapy to 5-year survival in adults was estimated to be 2.3% in Australia and 2.1% in the USA.

CONCLUSION: As the 5-year relative survival rate for cancer in Australia is now over 60%, it is clear that cytotoxic chemotherapy only makes a minor contribution to cancer survival. To justify the continued funding and availability of drugs used in cytotoxic chemotherapy, a rigorous evaluation of the cost-effectiveness and impact on quality of life is urgently required.

So what this and many similar independent studies have repeatedly shown is that essentially chemotherapy provides 5-year survival for 2.1% of cancer cases in the USA. We argue you must take matters into your own hands if you are to possibly improve these odds.

3. Chemotherapy and One Cancer Treatment: Use of Vitamin C

The study below found that vitamin C, one of conventional medicine's ignored vitamins, may actually sensitize cancer cells to chemotherapy drugs. It might actually make chemotherapy drugs more effective.

In the study, patients took vitamin C along with the common chemotherapy drugs 5-FU and cisplatin. The researchers found that vitamin C altered the DNA of the cancer, which made it more sensitive to chemotherapy. They also found that vitamin C actually improves the cancer-fighting ability of the drugs.

Said one physician, "In my experience, I've seen vitamin C protect patients against the devastating effects of chemotherapy. I've even seen it make the chemo work better. And I've found that it will help prevent many patients from losing their hair. If you or anyone you know is using chemotherapy, this is life-saving information. Make sure the oncologist sees the article below. The cancer you kill, and the hair you save, might be your own."

Abdellatif, et al. "Vitamin C enhances chemosensitization of esophageal cancer cells in vitro." *J Chemother*, 2005;17(5):539-549.

4. Avastin

Avastin was given a preliminary approval for breast cancer based on early studies that did not include survival data. The advisory committee agreed unanimously that two new trials failed to confirm the impression of benefit of the earlier studies. In particular, use of Avastin failed to prolong overall survival in the women who received it. In one study, the drug delayed tumor worsening by one month. In simple terms, Dr. Patrick Loehrer, Sr. of Indiana University (and a panel member) said, "It just delays by another visit before they get the news that their tumor progressed." An FDA panel had voted in December 2007 by a 5-4 vote to recommend against approval. But the FDA commissioner of the time, Andrew C. von Eschenbach, MD, then overruled his own panel (something that's almost never done) and approved the drug....a decision that turned out to be worth around \$500-700 million per year to Genentech.

However, FDA almost never revokes approval once it is granted. Thus, a government report has shown that of the 90 drugs given accelerated approval not one had been removed from the market and in fact the agency sometimes did not make sure that follow-up studies were done. It will be an interesting test to see how the new FDA commissioner, Margaret A. Hamburg, MD, reacts to this nearly unanimous panel recommendation.

Update: The drug has not been removed – just restricted in its use. Oncologists are still free to use it "off-label" and many will.

5. Avodart

If your doctor recommends a drug called Avodart to help lower your risk of prostate cancer, he's going to offer some evidence that might seem convincing. But before you rush into anything, consider the following: Avodart is designed to treat enlarged prostate (also known as BPH, or benign prostatic hyperplasia). In a recent *New England Journal of Medicine* study, about 3,300 men at high risk of prostate cancer took Avodart for four years. About 3,420 men--also at high risk--took a placebo. Compared to placebo, the relative risk of any level of prostate cancer was reduced by nearly 23% in the Avodart group.

So based purely on that number, your doctor might encourage you to take Avodart if you're at high risk of prostate cancer. And keep in mind that every man between the ages of 50 and 75 is considered high risk. But let's look a little deeper before you fill that prescription...

In years one through four, nearly all subjects underwent a needle biopsy. Among 3,299 men in the Avodart group, 220 tumors were detected with moderate to high Gleason scores (the system that rates the aggressiveness of tumors)

Among 3,407 men in the placebo group, 233 tumors were detected with moderate to high Gleason scores. During years three and four, 12 tumors with high Gleason scores were discovered in the Avodart group, but only one in the placebo group. So when it comes to aggressive tumors, Avodart doesn't appear to reduce risk at all.

And then there were the side effects: Sexual dysfunction was significantly higher in the Avodart group, and--even more important to know--subjects in that group were nearly twice as likely to experience heart failure compared to placebo.

At this point, any man who's still on the fence about using this expensive drug to prevent prostate cancer should consider a second opinion from an experienced urologist. Someone like Patrick Walsh, M.D. Dr. Walsh is currently a Distinguished Service Professor of Urology at Baltimore's Johns Hopkins School of Medicine where he served as Urologist-in-Chief for 30 years. In other words, when it comes to man-plumbing, Dr. Walsh knows his stuff.

An editorial by Dr. Walsh appears in the same *NEJM* issue as the Avodart study. And to get the full gist, you'll need to know that "dutasteride" is the generic name for Avodart, and "finasteride" is the generic name for Proscar, another popular BPH drug.

Dr. Walsh: "Dutasteride and finasteride do not prevent prostate cancer but merely temporarily shrink tumors that have a low potential for being lethal, and they do not reduce the risk of a positive biopsy in patients who have an elevated PSA level." And just last year, in the journal *Prostate Cancer Discovery*, Dr. Walsh had this to say about finasteride: "Men will believe that it prevents cancer, will be pleased that their PSA levels fall, and will not understand the potential danger they're in--of undiagnosed high-grade disease."

In a recent interview with *Medscape Oncology*, Dr. Walsh said his 2009 comments about finasteride also apply to dutasteride. Speaking specifically about the Avodart study, he said the results showed, "there was a 23% reduction in low-grade tumors that the patients would never have known they had. Does this

sound like an indication to take a pill with sexual side effects that costs \$4 a day?" If Dr. Walsh is unimpressed with the Avodart study results, there's no reason you or your doctor should be impressed.

6. Natural Aromatase Inhibitor For Estrogen-Related Cancers

The information presented here is essentially contained within the detailed information about genistein as given in Protocol C. However for those who might have missed the information at the end of the genistein section, we shall briefly review it again since it is very important.

There are two estrogen receptors in the body. The alpha or classic estrogen receptor is responsible for mammary gland growth. The beta receptor was only discovered about ten years ago. Its function or role in the body is only now becoming understood. In breast tissues, the beta receptor is considered an inhibitor of alpha mediated growth. In fact, the beta receptor apparently plays this role in **many other cancers** as well. That is why this discussion bears on other estrogen-related cancers too such as ovarian, endometrial, cervical, prostate, and colon cancer.

Since genistein does have a small capacity to activate the alpha receptor, physicians have discouraged women at risk for breast cancer or those who presently have breast cancer from ingesting genistein. This is a mistake. The alpha receptor is 10,000 to 50,000 times more sensitive to estrogen than it is to genistein. Further, genistein binds and activates the beta receptor 100 times more effectively than it does the alpha receptor. [16273360, 16118406].

As suggested, the optimal treatment protocol for breast cancer is to use an antagonist of the alpha receptor and an agonist or activator of the beta receptor. Genistein is a powerful anti-cancer natural medicine in general and an activator of the estrogen beta receptor. Furthermore genistein's ability to activate the alpha receptor can be blocked with melatonin, the sleep hormone. Melatonin does not block the activity of the beta receptor [16635015, 15229223].

In addition, melatonin is an inhibitor of aromatase activity, the enzyme that converts testosterone to estrogen [16647824, 16080194, 15683469].

If a woman is diagnosed with an estrogen responsive cancer, she should immediately begin taking melatonin and genistein. Synthetic prescription drugs are not necessary. In addition, genistein is a general tyrosine kinase inhibitor, which means it inhibits the activity of the growth promoting EGF and HER-2 receptors [12239620].

The effective cytotoxic dose of genistein necessary to kill 75% of breast cancer cells is 10 microM, a dose that can be reached in the blood by the consumption of specific genistein products. Specific recommendations are given in the details of our more comprehensive protocols.

7. Article by Ralph Moss, Ph.D. -- Update on Chemotherapy – [Feb. 1, 2009] from *Cancer Decisions*

Medicare has broadened its payment for new anticancer drugs, according to a recent article in the *New York Times* (1/26/09). At first sight, this might seem like a good development for cancer patients. Medicare will now pay for more drugs! But, as the story makes clear, the change actually raises serious questions about the overuse and promotion of dubious drug combinations that have become popular in recent years.

As background, the Food and Drug Administration (FDA) has approved a variety of new drugs over the past few years that, at best, are minimally effective. Sometimes, they confer a few months of extra survival; oftentimes there is no proof that they do even that. They also may carry an increased risk of serious side effects, and in almost every case they are very expensive.

According to the *Times* authors, "for many such uses there is scant clinical evidence that the drugs are effective, despite costing as much as \$10,000 a month." Medicare approval may therefore waste money and needlessly expose patients to the adverse effect of drugs that might hurt them, and make their final months more miserable. New drugs are approved by FDA only for specific indications, and they cannot be assumed to work in other circumstances.

For a dozen or so years, however, industry has succeeded in chipping away at these FDA barriers. Now the dam has broken. Medicare has in effect over-ridden the carefully constructed FDA barriers and agreed to pay for a much wider degree of "off-label" use. These uses are sanctified by various guidelines, called compendia, that are often written - according to the *Times* - with the eager input of drug company representatives.

"We have very little faith that those indications that make it into the compendia are safe, let alone effective," said Dr. Allan M. Korn, the chief medical officer for the Blue Cross and Blue Shield Association. Medicare is now providing "carte blanche in treatment for cancers," according to Steven Findlay, a health policy analyst for Consumers Union. He also said that such coverage encourages doctors to use patients as guinea pigs for unproved therapies. But skeptical views like this are overwhelmed by a chorus of cheers coming from Big Pharma and its many friends in the oncology community.

Political Implications

What will this change mean in practice? For example, Avastin (bevacizumab) - at \$100,000 per year one of the most expensive of the newer drugs - will now be covered by Medicare for ovarian cancer. But does Avastin actually work for ovarian cancer, i.e., does it increase the patient's overall survival? No one can say, since randomized controlled trials (RCT), formally required for FDA approval, have never been done.

But the new Medicare ruling "makes it easier to give drug after drug," said Andrew Berchuck, MD, director of gynecologic oncology at Duke University, "and keep the fantasy alive." Keeps the fantasy alive! The "fantasy" in this case is the idea that oncologists actually have effective pharmaceutical solutions to intractable ovarian cancer, when they do not.

Implications for CAM

Oncologists have hailed the change as a way of generating new knowledge about what works and what doesn't. But I suspect that, on the contrary, these new rules will further reduce the motivation to do rigorous research. Who now will bother if you can get compensation based just on a drug industry-influenced compendium? This also has serious implications for complementary and alternative medicine (CAM).

On the one hand, I realize it may make it easier for the few CAM practitioners, who utilize these drugs in off-label ways, to get compensated for doing so. But, on the other hand, the rules will further decrease the attractiveness of alternative treatments, since now patients will perceive their conventional doctors as the source of a wider array of potential treatments (without evidence that they can actually increase survival).

What I find particularly disturbing is that the double standard has just gotten much wider. CAM treatments that have a considerable amount of data behind them still have little chance of being accepted by FDA or paid for by Medicare. They are expected to jump through all the hoops of a lengthy testing process, including prohibitively expensive randomized clinical trials.

This is virtually impossible for them to do, but Big Pharma - which has the money to do such trials - now gets a free pass. At the same time, the combined forces of FDA, Medicare, ASCO, and their friends, continue to prevent accurate information on these competitive treatments from reaching the ears of oncologists. Take, for example, ASCO's guidelines on exhibiting at their massive annual meeting:

"Dietary supplements that (I) make any claim to diagnose, mitigate, treat, cure, or prevent any disease, specific class of disease, disease symptom, or abnormal medical condition; or (ii) *claim an effect similar to that of an approved drug*, biological product, or medical device must not be exhibited" (emphasis added).

Is it any wonder, then, that rank-and-file oncologists remain ignorant or scornful of the effects of less toxic or botanical compounds, and eagerly turn to the products of Big Pharma. In addition, although not mentioned in the current Times article, readers should be aware of several excellent articles by the same authors on the so-called "chemotherapy concession." This means, essentially, that oncologists are allowed to sell chemotherapy in their offices at a profit, a privilege granted to few other medical doctors. They may also receive rebates from these same companies when they prescribe a high volume of their products.

CAM treatments are not paid for by Medicare, even when they have extensive testing behind them or have been approved as legal treatments in other countries. The high regulatory barrier remains in place for nutritional, non-toxic or unconventional approaches. But the products of Big Pharma, once they have gained a foothold in a single indication (often in the absence of proof of life-prolongation) are now eligible for compensation for many other indications. This is a move that primarily benefits Big Pharma and profit-oriented oncologists, and not cancer patients in search of more effective treatments.

--Ralph W. Moss, Ph.D.

8. Chemo and Low White Blood Cells

Dr. Jonathan Wright has noted that keeping a normal white blood cell count is one of the very basic necessities for good health. Your body uses white blood cells to fight off viruses, bacteria, and all sorts of potentially harmful foreign invaders. If your levels fall, you're much more prone to illness. Chemotherapy and radiation are probably the most well-known offenders causing low white cell counts, but levels can fall for any number of reasons. But researchers have found that lithium can increase white cell numbers again in people whose levels fell due to radiation and/or chemotherapy (and even if the person continues those treatments).

Lithium achieves these effects by stimulating the stem cells in bone marrow, which then turn into platelets and white blood cells. I've observed that low dose lithium (5- 10 milligrams twice daily) will also usually raise a low count to normal even if radiation and chemotherapy aren't the culprits. Lithium can be obtained at these low doses from over-the-counter supplements via the web. For example, the Vitamin Shoppe at www.vitaminshoppe.com sells a lithium orotate product made by General Research Labs. These pills each contain 5.8 mg of elemental lithium. Taking 1-2 pills twice a day, depending on the severity of the condition, yields a possible dose range as targeted. One might do this for a month and then assess your white count again.

9. Regarding Chemotherapy Usefulness – The Original Abstract

This article has been widely quoted, as it showed that chemo's contribution to 5-year survival in the U.S. was 2.1%. This is an extraordinarily low rate of usefulness and should be factored in by anyone considering this option. The Abstract of the original article is given below.

Clin Oncol ® Coll Radiol). 2004 Dec;16(8):549-60: The contribution of cytotoxic chemotherapy to 5-year survival in adult malignancies.

Morgan G, Ward R, Barton M. , Department of Radiation Oncology, Northern Sydney Cancer Centre, Royal North Shore Hospital, Sydney, NSW, Australia. gmorgan1@bigpond.net.au

AIMS: The debate on the funding and availability of cytotoxic drugs raises questions about the contribution of curative or adjuvant cytotoxic chemotherapy to survival in adult cancer patients.

MATERIALS AND METHODS: We undertook a literature search for randomized clinical trials reporting a 5-year survival benefit attributable solely to cytotoxic chemotherapy in adult malignancies. The total number of newly diagnosed cancer patients for 22 major adult malignancies was determined from cancer registry data in Australia and from the Surveillance Epidemiology and End Results data in the USA for 1998. For each malignancy, the absolute number to benefit was the product of (a) the total number of persons with that malignancy; (b) the proportion or subgroup(s) of that malignancy showing a benefit; and © the percentage increase in 5-year survival due solely to cytotoxic chemotherapy. The overall contribution was the sum total of the absolute numbers showing a 5-year survival benefit expressed as a percentage of the total number for the 22 malignancies.

RESULTS: **The overall contribution of curative and adjuvant cytotoxic chemotherapy to 5-year survival in adults was estimated to be 2.3% in Australia and 2.1% in the USA.**

CONCLUSION: As the 5-year relative survival rate for cancer in Australia is now over 60%, it is clear that cytotoxic chemotherapy only makes a minor contribution to cancer survival. To justify the continued funding and availability of drugs used in cytotoxic chemotherapy, a rigorous evaluation of the cost-effectiveness and impact on quality of life is urgently required.

10. Adriamycin for Breast Cancer

In a analysis of 18,000 women with breast cancer in 47 randomized trials [see *The Lancet* 358(9278): 277-286, 2001], the advantage of chemotherapy was explicitly quantified. With a follow-up extending 10 years, for older women (50-69), chemotherapy added just 2.9 months to overall survival. This reviewer was struck by the remarkably negligible benefit that chemo added by the 10th year for all the agony and cost of chemo. Also if you survived another 10 years, added deaths from chemo's cardiotoxic properties might ultimately wash away this slim gain. I wondered why there was so little net benefit. Additional research answered this question.

The chief science reporter for NBC reported on a private session held at the meeting of the American Society of Clinical Oncology this June, 2007. There Dr. Dennis Slamon, Chief of Oncology at UCLA, revealed that current research indicates that the main chemotherapy regimen for breast cancer almost certainly provides no benefit whatsoever to 92% of breast cancer patients. His research showed that only 8% of all women with breast cancer, specifically those who over-express a specific gene called Topoll-2,

stand to benefit from the typical anthracycline-based chemotherapy for breast cancer since these drugs work by targeting Topoll-2.

As the report indicated, the revelation that 92% of women given breast-cancer anthracycline-based chemotherapy can “expect to derive absolutely no benefit (and potentially considerable harm” due to its cardiotoxic, heart-harming actions) was viewed as momentous, coming from such an unimpeachable source such as Dr. Slamon. Thus the lack of improved survival from chemo described above is explained. Since most women (92%) are not getting any benefit, the benefits for just 8% of the women has to be divided over the whole population, thus making the “average” net gain negligible. If you know someone who has just been diagnosed with breast cancer, the smartest strategy is to attempt to get a Topoll-2 gene test to see if the chemo will do any good whatsoever. As for getting the test, it is not commercially available as yet, and even if it were, it would not likely be reimbursable by your insurance. From my past experience as head of a research office for several different national gene testing companies, I would call Johns Hopkins or Mayo Clinic to see if they are doing this test in a research environment (I have no inside information — these are just research leaders in this area). In the commercial world, I would call Myriad Corporation, a national leader in gene testing, to see if they had any information.

Although Dr. Slamon's work has not yet been published in any medical journal, Bazell considered it newsworthy enough to publish at the MSNBC Web site. Aside from the Annie Appleseed Project (www.annieappleseedproject.org), almost no one on the Internet has seemingly noticed this important report.

11. The Downside to Chemotherapy in Advanced Cancer Stages

[– Source: *Times* Online November 12, 2008]

Another study, The National Confidential Enquiry into Patient Outcome and Death (NCEPOD), found that more than four in 10 patients who received chemotherapy toward the end of life experienced potentially fatal effects. And after reviewing data from over 600 cancer patients who died within 30 days of receiving treatment, it was found that chemotherapy hastened or caused death in 27 percent of those cases.

Doctors have been urged to be more cautious in offering cancer treatment to terminally-ill patients as chemotherapy can often do more harm than good, a study suggests. The National Confidential Enquiry into Patient Outcome and Death (NCEPOD) found that more than four in ten patients who received chemotherapy towards the end of life suffered potentially fatal effects from the drugs, and treatment was “inappropriate” in nearly a fifth of cases. In a study of more than 600 cancer patients who died within 30 days of receiving treatment, chemotherapy probably caused or hastened death in 27 percent of cases, the inquiry found. In only 35 percent of these cases was care judged to have been good by the inquiry’s advisors, with 49 percent having room for improvement and 8 per cent receiving less than satisfactory care.

12. Chemotherapy – Risk of Off -label Uses

"We know there is a high demand for new treatments in oncology," said the leader of the new *Journal of Clinical Oncology* research, Dr. Jeffrey Peppercorn of Duke University Medical Center. He's found that

more than eight out of ten cancer doctors prescribe medications that are still in testing. Dr. Peppercorn's new study appeared in the *Journal of Clinical Oncology*[*JCO*] (online, Oct. 25, 2010).

It focuses on drugs that are actually FDA-approved -- but not for the use the cancer doctor wants to try. This is called "off-label" prescription and it's perfectly legal. Once a drug is approved for a given application a doctor can pretty much prescribe it for anything he or she wants. In theory, doctors are only supposed to use the drugs in the approved way but there's no legal restriction.

If the manufacturer conducts successful trials of the new application, it can become an approved "on-label" use. In the absence of FDA trials, the new application remains off-label. The *JCO* study looked at off-label use of drugs that were in trials aimed at getting them approved for some new application.

"People need to realize that because the trials have not been completed, there is a great deal that is not known about the treatments," said Dr. Otis Brawley, the chief medical officer of the American Cancer Society. Dr. Brawley was not involved in the study.

Dr. Peppercorn's current *JCO* study finds that very few off-label drugs survive the testing stage and win FDA approval for the new application. His team looked at 172 trials of such drugs and found that two-thirds were associated with severe or life-threatening side effects. Dr. Peppercorn concludes, "In most cases, we should refrain from using experimental drugs outside of clinical trials."

Dr. Steven Joffe, of the Dana-Farber Cancer Institute in Boston, says, "I perfectly understand why a patient with a poor prognosis might want to have access to these drugs and why doctors would give them the drugs. They may think they know that it works. But the fact is, somebody is running a study to find out if it does. Almost by definition it hasn't been proved safe and effective."

In short, always ask if the chemotherapy about to be used is "off-label." If so, the odds are further stacked against you, its usefulness even more unlikely [see the articles above], and the prospects for harm ever higher.

13. Colon Cancer – A Natural Chemotherapy to Halt Remission

A new food supplement from Israel targets colon and rectal cancer, as well as ulcerative colitis and other bowel diseases. The product, which has yet to hit the world market, is called Coltect. It is a combination of green tea polyphenols, curcumin powder from the turmeric root and the trace mineral selenium. Its effects were described at a recent oncology meeting and it is the subject of two clinical trials.

Results were presented at the 2010 American Society for Clinical Oncology (ASCO) Gastrointestinal Cancers Symposium. The authors, from Tel Aviv Sourasky Medical Center, tried Coltect alone or combined with a common drug, 5-aminosalicylic acid (5-ASA), in cell line and animal models of colon cancer.

Depending on the dose, there was up to an 83 percent inhibition of cancer cell growth using Coltect. In the animal model, the combination of Coltect and the drug 5-ASA reduced the number of precancerous lesions from 66.5 in the control group to 20 in the group that received both agents. The authors concluded that **Coltect "can be administered as a chemopreventive regimen to prevent" colorectal cancer.**

While waiting for Colcortol to hit the world market, one might consider taking a combination of green tea polyphenols, turmeric (with its key ingredient, curcumin) and Brazil nuts (a good source of selenium—use the kind that you have to shell yourself).

As for 5-ASA, it is not available without a prescription. But it is a derivative of salicylic acid and is chemically similar to aspirin. A 2003 journal article concluded: “Preclinical, observational, and clinical data consistently show that non-steroidal anti-inflammatory drugs (NSAIDs) — particularly aspirin — reduce colorectal carcinogenesis” (Hawk and Vine, 2003). So you might ask your doctor about taking a baby aspirin (81 mg) along with the anticancer food components.

Colon cancer afflicts over 100,000 Americans each year. Perhaps some of these cases could be prevented by the judicious use of anticancer foods.

Reference: Hawk ET, Viner JL. Aspirin: still learning about the wonder drug. *Gut*. 2003; 52(11): 1535-1536

NOTE: All of the above are part of the Protocols identified in the basic document. The Meriva [item #1] in Protocol A contains the curcumin, the green tea is in Protocol B, and salicylic acid in the form of baby aspirin can be added to Protocol B. So for colon cancer patients, using protocol A and B and adding baby aspirin makes a great deal of sense.

14. Tarceva – A Standard Therapy for Lung Cancer – Does it Work?

[essay by Dr. Ralph Moss]

Prominent oncologists are now urging that the 'targeted' drugs Avastin (bevacizumab) and Tarceva (erlotinib) be routinely added to standard chemotherapeutic regimens for non-small cell lung cancer (NSCLC). According to David Ettinger, MD, professor of oncology and medicine at Johns Hopkins University School of Medicine, Baltimore, Md., the addition of these drugs represents "the most important change" in the treatment of this disease (Susman 2006).

Although neither drug offers patients a cure, Dr. Ettinger told United Press International, both agents allegedly prolong survival. "A few years ago, the idea that we could have a two-year survival for patients with stage 4 non-small cell lung cancer was not in the picture," Ettinger declared at the 11th annual conference of the National Comprehensive Cancer Network (NCCN) in Hollywood, Fla. "But now we are seeing that 22 percent of these patients are surviving two years and longer." (The NCCN is a consortium of 19 major cancer hospitals, including both Johns Hopkins and Memorial Sloan-Kettering. It has been writing algorithms for the treatment of various forms of cancer since 1996. The guidelines are used as a standard of care in government demonstration projects and have gained national and international acceptance.)

The UPI article gave full exposure to Dr. Ettinger's views but failed to mention the fact that he himself has performed contract work for OSI, the developer of Tarceva, and has been a consultant to Genentech, the American manufacturer of both Tarceva and Avastin. In the same article, Mark Kris, MD, chief of the thoracic oncology service at Memorial Sloan-Kettering Cancer Center in New York, echoed Dr. Ettinger's sentiments. He claimed that the use of the targeted therapies extends overall survival in NSCLC by about four months compared to treatments that do not include these agents. He waxed enthusiastic about the future of such therapies.

"I can see the time that we will be able to treat advanced lung cancer much the way we treat people with diabetes – as a chronic disease," he said. The UPI article failed to point out that, like Dr. Ettinger, Dr. Kris

has also received research funding as well as consulting fees from Genentech and other companies in the targeted therapy field.

Many of the positive assessments of Tarceva are based on a study carried out by the National Cancer Institute of Canada Clinical Trials Group. This study followed 731 patients who had already failed first-line or second-line chemotherapy, usually including one of the platinum-based drug regimens (including cisplatin, carboplatin, etc.). These patients were then given either 150 milligrams per day of Tarceva or an inert placebo (look-alike) pill.

The overall response rate was less than 8.9 percent in the Tarceva group and the median duration of this response was 7.9 months. For the NSCLC patients as a whole, the overall survival rate was 6.7 months for those receiving Tarceva vs. 4.7 months for those who got placebo, i.e., an absolute gain of two months. Five percent of the patients discontinued Tarceva because of excess toxicity (Shepherd 2005). The study reported that 31.2 percent of patients receiving Tarceva were alive at one year vs. 21.5 percent in the placebo arm of the trial. This is the source of the claim that Tarceva extends survival by "several months."

At the Tarceva.com Web site, the manufacturer, Genentech, Inc, indulges in a now very familiar piece of statistical sleight of hand, presenting the survival gain for Tarceva in relative (as opposed to absolute) terms. Thus a two-month absolute survival gain is expressed as a "42.5 percent improvement" in survival! That sounds a lot more impressive than a mere eight weeks survival benefit. By expressing the advantage as a relative survival percentage increase, the manufacturer is able to project a misleadingly rosy impression of what the drug actually does. Often overlooked is the fact that, a few months later, a larger trial, named the TRIBUTE study, based at M.D. Anderson Cancer Center, Houston, came up with essentially negative conclusions on the value of Tarceva.

In the TRIBUTE trial, Tarceva was combined with chemotherapy to determine if it could improve the outcome for patients with advanced (stage IIIB or IV) NSCLC. Experimental subjects with good performance status who had not been previously treated received six cycles of the standard drugs carboplatin and paclitaxel along with 150 milligrams per day of Tarceva. This was followed by single-agent maintenance therapy of Tarceva. A comparison group received the identical chemotherapy with only a placebo (look-alike) pill instead of Tarceva.

The median overall survival time for patients treated with Tarceva was 10.6 months vs. 10.5 months for those getting placebo. In other words, Tarceva had NO impact on survival in this patient population. Nor was there any difference in the overall response rate, nor in the median time to progression. Patients taking Tarceva experienced an increased incidence of the extensive rash and diarrhea that is characteristic of the drug.

In a subgroup analysis, those relatively few patients who reported never having smoked experienced some improvement in overall survival after taking Tarceva (22.5 v 10.1 months for placebo). No other pre-specified factors were associated with an increase in overall survival with Tarceva. Thus, among lifelong non-smokers Tarceva might add about one year of extra survival.

The authors concluded that Tarceva plus standard chemotherapy "did not confer a survival advantage over carboplatin and paclitaxel alone in patients with previously untreated advanced NSCLC." However, "never smokers" treated with Tarceva and chemotherapy "seemed to experience an improvement in

survival" and will hopefully be the subject of further investigation in future randomized trials. There are certain mutations of epidermal growth factor receptor (EGFR) that are common in nonsmokers with lung cancer compared to smokers who develop what is ostensibly the same diagnosis. Thus nearly 50 percent of non-smokers in one Sloan-Kettering study had these EGFR mutations, while just 5% of the smokers in the study did (Pao 2004).

It is difficult to reconcile the results of two phase III clinical trials. In the 2005 Canadian study, patients who received Tarceva lived approximately two months longer than those who did not receive the drug (Shepherd 2005). In the larger M.D. Anderson study, however, there was no survival advantage whatsoever in the general patient population, although lifelong non-smokers did seem to benefit (Herbst 2005). Each trial featured a somewhat dissimilar drug regimen and a different patient population. So the divergent results were probably due to differences in the way that patients were selected for the two studies.

According to an article by Robert L. Comis, MD, of Drexel University, Philadelphia, in the magazine *Oncologist*, there are no clear cut factors that predict either objective response, time to progression, or survival with Tarceva (or for a similar drug, Iressa) in NSCLC. In particular, patients who are female, lifetime non-smokers, and have adenocarcinoma, particularly if they are of Asian descent, might derive the most benefit. But Comis emphasizes that "the vast majority of lung cancer patients do not fit into the categories of patients that might derive benefit from [Tarceva] erlotinib-based interventions for advanced lung cancer and less advanced disease" (Comis 2005).

Meanwhile, Genentech may run into consumer resistance to its strategy of charging whatever the market will bear for these drugs. A study in Canada showed that lung cancer patients, while accepting the general utility of such treatments, are only willing to pay around \$100 per month in Canadian dollars (equal to around US \$90) for such a drug. In reality, the cost of Tarceva alone is US \$90 per day (not per month!), or over \$30,000 per year. Avastin will cost much more still - around \$100,000 per year for lung cancer patients.

--References:

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15. Tarceva – A Possible Alternative Choice

Tarceva is a small molecule designed to target the human epidermal growth factor receptor (HER1) pathway, which is one of the factors critical to cell growth in non-small cell lung and pancreatic cancers. HER1, also known as EGFR, is a component of the HER signaling pathway, which plays a role in the formation and growth of non-small cell lung and pancreatic cancers. Tarceva is designed to inhibit the tyrosine kinase activity of the HER1 signaling pathway inside the cell.

Now go back and take a look at the supplement genistein in Protocol C. What do we find? We showed that in the study you can access below, genistein can not only inhibit the synthesis of VEGF, but the tyrosine kinase activity of the VEGF membrane receptor as well. Therefore, genistein inhibits the synthesis of new VEGF molecules while blocking the biological activity of previously synthesized VEGF molecules [17142033].

So Genistein can block the activity of ALL tyrosine kinase receptors, including those for VEGF and EGF. Also genistein can also increase the synthesis of PTEN, an major tumor suppressor and inactivator of PI-3K/AKT signaling. Moreover genistein can inhibit the activity of HIF-1, an activator of VEGF synthesis and glycolytic enzyme synthesis. Therefore, in summary, genistein [particularly in combination with glutamine], in the correct dose, can work together to inhibit the growth and development of virtually all cancer. It might be argued that tarceva is simply a poor substitute for genistein [although it is patented and genistein cannot be]. I myself would take genistein if I had lung cancer.

16. Tamoxifen: Killer “Cure”

The breast cancer drug Tamoxifen might well be marketed with the slogan, "If it doesn't kill you, it will cure you"— except that would be an overstatement. The truth is that it probably *won't* cure you of anything, but may very well kill you, as yet one more study makes clear. According to that study, just published in *Cancer Research* no less, long-term use of tamoxifen ups the risk of getting aggressive cancer in the other breast by 440 percent. In spite of this finding, researchers insist that women should keep taking the drug because, they say, the benefits outweigh the risks. Then again, they're not the ones at risk.

Tamoxifen has been used for over 25 years both to treat breast cancer, as well as to prevent it. Most breast cancers spread when exposed to estrogen, and since tamoxifen inhibits estrogen receptivity, it has been the standard treatment, although recently aromatase inhibitors, a new class of drugs, have taken precedence. Tamoxifen also has frequently been prescribed to high-risk women who don't have breast cancer as a prophylactic measure to stave off tumors. And, it's been used to treat osteoporosis, bipolar disorder, and prostate cancer.

But problems with Tamoxifen have been surfacing for years, and now this study shows the drug causes an even more deadly version of the disease it supposedly cures. The tumors negatively associated with tamoxifen use do not feed on estrogen, no drugs on the market have been effective in treating them, and the prognosis for cancers featuring these tumors is worse than for estrogen-dependent cancers.

The study followed 1100 women aged 40-79 who received treatment for estrogen-receptive breast cancer between 1990 and 2005. Those who took tamoxifen were 60 percent less likely to develop estrogen-dependent breast cancer in the other breast compared to those not taking the drug. But those

women who took the drug for more than five years, as already mentioned, had a hugely increased risk of developing estrogen-negative tumors. The study did *not* include women who took the drug long-term to prevent breast cancer in the first place. If it had included that group, the risk most likely would reflect far higher numbers.

So, you might conclude, since the drug does seem to provide significant short-term protection, you can safely take it for the recommended five years (that's the standard regimen) and then stop, thus reaping the benefits and avoiding the risks. Doctors point out that in the study, the overall numbers of women developing the aggressive breast cancers remained small (an interesting double standard as we'll discuss in a moment). The tumors appeared in only 14 of the 358 women treated for longer than five years. But, two troublesome key points make those arguments look lame. First, tamoxifen causes life-threatening problems other than breast cancers. Also, studies have found that while tamoxifen may prevent estrogen-dependent breast tumors, it does very little, if anything, to prolong life expectancy, at least when used as a cancer preventative. Let's examine these issues in a little more detail.

First, the other health problems associated with Tamoxifen include blood clots, strokes, uterine cancer (about double the risk), ovarian cancer, liver cancer, gastrointestinal cancers, and cataracts among them, as well as the usual chemotherapy discomforts — nausea, vomiting, headaches and so on. "Any sort of treatment has risks and benefits, and the benefits for tamoxifen are very clear, particularly with respect to reducing mortality," says lead researcher Christopher Li, MD, of the Fred Hutchinson Cancer Research Center in Seattle. But are they? A huge study of 13,000 women by the National Cancer Institute back in the 1990's found that while tamoxifen did indeed cut "the incidence" of breast cancer by 30-50 percent in the high-risk group of women who took it as a preventative, seven years into the follow-up, women in the no-tamoxifen control group had fewer deaths from breast cancer than those in the tamoxifen group — marginally fewer, but fewer none-the-less. Please read this last sentence again.

And, as I've written before, those statistics touting the huge percentage reductions in new tumors from tamoxifen's effects do not hold up on closer inspection. About ten years ago, newspapers cited studies proving the efficacy of tamoxifen that consistently read something like "The National Cancer Institute's Breast Cancer Prevention Trial reported that there was a 49 percent decrease in the incidence of breast cancer in women who took tamoxifen for four to five years." That's stunning. If your doctor told you that using tamoxifen cut your chances of getting breast cancer by 49%, would there be any question in your mind on whether or not to use it? But if you look past the statistics, the truth is that according to the study, your odds of getting breast cancer without using tamoxifen were only 1.3%. With tamoxifen it dropped to .68%. And yes, that could be represented as a 49% difference between the two numbers. But the reality is we're talking about a difference of just a little over one-half of one-percent (0.64%) in real terms.

As Sidney Wolfe, M. D. noted, the likelihood of getting endometrial cancer is increased when taking Tamoxifen by nearly one percent, so as he published, it's a wash. You will lose as much as you gain — not counting the newer research presented at the beginning of this article.

Bottom line for this researcher, when my mother got breast cancer, and after her operation, I recommended against Tamoxifen and she was never sorry. She died many years later of pneumonia.

Although I discussed this point under melatonin, let me repeat some crucial information again in case you have forgotten. Just as the drug Tamoxifen inhibits estrogen binding to its receptor so that cells are not

encouraged to grow (which is why it is prescribed), melatonin does the same thing. Tamoxifen also kills estrogen-insensitive breast cancer cells by a pathway called JNK. This pathway is activated by virtually all chemotherapeutic drugs and radiation therapies. Melatonin does the same thing. Also melatonin inactivates the aromatase pathway just as aromatase inhibitors do (although Tamoxifen doesn't) [15683469]..

So all in all, melatonin is extremely valuable, and your author would personally choose it over Tamoxifen any day. Unlike Tamoxifen, melatonin does not increase your chances of endometrial cancer while Tamoxifen does. Why didn't this cheap, effective supplement get caught up into clinical practice? For that answer, you can re-read section 4 of Chapter 13 again if you wish.

17. Chemotherapy Selection

We next come to useful tools to select chemotherapy if you elect to have it. Tests known as chemo sensitivity and resistance assays, or CSRA's, test different chemotherapy drugs directly against a sample of tumor to identify which is the most effective. In contrast, currently oncologists select chemo based on its overall performance in clinical trials, but even the best drugs may fail to help between 30% and 60% of patients. It is impossible to predict the outcome of a particular chemo agent for any individual due to genetic and other differences among cancers and individuals.

There are two types of CSRA's — one determines whether a drug stops a cancer from growing, and the other determines whether a drug kills the cancer outright (cell death method). The cell death method is the much better of the two. These tests can cost \$2000 or more and are often not covered by insurance. The patient must also have enough tumor so that a large enough sample can be tested.

This author can not imagine undergoing chemo without first doing a CSRA if it can be feasibly done, although many oncologists are opposed to CSRA's. Firms doing CSRA's include Wiesenthal Cancer Group (Huntington Beach, CA), Oncotech (Tustin, CA), Rational Therapeutics (Long Beach, CA), NuOncology Labs (Virginia Beach, VA), Impath (multiple locations), Anticancer, Inc. (San Diego, CA) and Precision Therapeutics (Pittsburgh).

Some studies suggest your chemotherapy may be up to 10 times more effective if it is the properly selected one [or ones]. This should not really come to anyone's surprise. So far I have not personally found an oncologist particularly interested in this crucial testing. It is my opinion that if the results show another choice or combination from your oncologist's choice, three things can happen. We can see an ego conflict – “who is in charge here” – or the oncologist has a reimbursement problem if the choices differs from a standard of care – or the oncologist has a worried “legal” problem if he or she will not be doing the typical standard of care.

All of the above are not your problems. Your life may depend on finding a good answer.

18. Reducing Chemotherapy Side Effects

Unfortunately, there are times when cancer patients must take chemotherapy. If you're in this position, what could be such a powerful protector without any cost or medical intervention? It's fasting! According to a somewhat recent study¹, fasting for two days before you have chemo will protect your health cells from the poison.

In the study, researchers starved a line of yeast cells and found them 1,000 times more resistant to oxidative stress or chemotherapy drugs. And they also found that healthy cells starved of nutrients survived the ravages of chemotherapy far better than cancer cells so treated.

That was in a test tube. What about in animals? Well, the researchers then tried the same thing on living mice. They gave an unusually high dose of etoposide (80 mg/kg) to mice they starved for 48 hours. In humans, that is twice what is considered to be a very high dose of the drug.

The high dose killed 43% of the mice except one that didn't fast. The starved mice all survived and regained their lost weight within four days. An even higher dose killed all of the well-fed mice from a different genetic strain, but none of the starved mice. And again the mice that fasted regained their weight.

We know that fasting prolongs life. It may be that fasting induces normal cells to go into a hibernation mode. Cancer cells don't have an "off" switch. In the "hibernation mode" the cells' metabolism slows so much that the chemo doesn't do a lot of damage. The cancer cells don't get that protection.

Fasting isn't pleasant. For some people it's very hard to do. And it can increase nausea in some people. However, these usually are far less severe than the chemo. So if you can do it, fasting might be a terrific way to protect yourself from this toxic drug. I might note that if you can't fast for two days, even some fasting [perhaps one day] might still be of value.

Reference 1: *Proc Natl Acad Sci U S A. 2008 Mar 31.*

Chapter 15 – An Important Stand-Alone Treatment: Low Dose Naltrexone

In Brief: Although prospective, controlled clinical trials on LDN in the treatment of cancer are yet to be accomplished, as of March 2004 clinical "off-label" use of this medication by Dr. Bihari in some 450 patients with cancer, almost all of whom had failed to respond to standard treatments, suggests that more than 60% of patients with cancer may significantly benefit from LDN.

Of the 354 patients with whom Dr. Bihari had regular follow-up, 86 have shown objective signs of significant tumor shrinkage, at least a 75% reduction. 125 patients have stabilized and/or are moving toward remission. Dr. Bihari's results sharply contrast to prior usual cancer treatment outcomes: either a cancer-induced death or a total cure. LDN therapy presents a viable third alternative, the possible long-term stabilization and/or gradual reduction of tumor mass volume.

Thus, with LDN, cancer can, in some cases, become a manageable chronic disease. Patients have the possibility of living free of symptoms, without, in many cases, the crippling side-effects of chemotherapy and radiation treatment.

The following is crucial – as you read further, this report notes that the usefulness of LDN is greatly or possibly totally diminished for those who have had previous chemotherapy. In our own limited experience, this has been precisely the case. LDN did not help those to whom we directed it when they had already had extensive chemotherapy. They also had stage IV terminal pancreatic cancer so this of course confounds the matter too. However it is my belief that LDN is far more likely of greater potential benefit when it is started before rounds of chemotherapy have been undertaken.

How It Works

Low dose naltrexone might exert its effects on tumor growth through a mix of three possible mechanisms: By inducing increases of met-enkephalin (an endorphin produced in large amounts in the adrenal medulla) and beta endorphin in the blood stream;

By inducing an increase in the number and density of opiate receptors on the tumor cell membranes, thereby making them more responsive to the growth-inhibiting effects of the already-present levels of endorphins, which induce apoptosis (cell death) in the cancer cells; and

By increasing the natural killer (NK) cell numbers and NK cell activity and lymphocyte activated CD8 numbers, which are quite responsive to increased levels of endorphins.¹ (abstract)

Cancers that are reported by Dr. Bihari to apparently respond to LDN:

Bladder Cancer

Breast Cancer

Carcinoid

Colon & Rectal Cancer

Glioblastoma

Liver Cancer

Lung Cancer (Non-Small Cell)

Lymphocytic Leukemia (chronic)

Lymphoma (Hodgkin's and Non-Hodgkin's)
Malignant Melanoma
Multiple Myeloma
Neuroblastoma
Ovarian Cancer
Pancreatic Cancer
Prostate Cancer (untreated)
Renal Cell Carcinoma
Throat Cancer
Uterine Cancer

What the Future Holds

If the results of trials of low dose naltrexone in certain cancers are positive, the drug could eventually become an additional mainstay of cancer treatment ? adjunctive with chemotherapy, radiation, and other cancer cell growth inhibitor receptor agonists ? or even a replacement for current therapies, as primary treatment for those cancers that show little response to standard therapies.

Recent Developments: As of March 2004 —

Since February 1999, Dr. Bihari has begun treatment of some 450 cancer patients with LDN. Since many of these patients, particularly those seen before October 2000, were seen only once in consultation with medical follow-up by their oncologists, Dr. Bihari is missing up-to-date follow-up data on 96 patients.

As of March 2004, of the remaining 354 patients, 84 have died, all but 4 of cancer-related causes. Most of these deaths have occurred in the first 8 to 12 weeks on LDN. For the most part, these were patients who were quite ill when first seen, and had exhausted all other treatment possibilities. Of the remaining 270 patients, 220 have been on LDN for six months or longer. Of these, 86 have shown significant movement toward remission, identified for this purpose as a reduction of at least 75% in tumor mass and tumor-related symptoms. Of the other 134 patients, 9 have continued to show tumor progression, whereas the other 125 have stabilized and/or are moving toward remission but do not yet meet the 75% reduction criterion.

Among those who have shown significant movement toward remission, most had never received chemotherapy. The apparent remissions:

- 2 children with neuroblastoma
- 6 patients with non-Hodgkin's lymphoma
- 3 with Hodgkin's disease
- 5 with pancreatic cancer metastatic to the liver
- 5 with multiple myeloma
- 1 with carcinoid
- 4 with breast cancer metastatic to bone
- 4 with ovarian cancer
- 18 with non-small cell cancer of the lung
- 1 with small cell cancer of the lung
- 5 with prostate cancer (no prior hormone-blocking therapy)

(Although recently-diagnosed prostate cancer patients who have not received other therapies appear to do well on LDN, patients with prostate cancer who have already been treated with hormone-related therapies, including testosterone-blocking drugs and PC-SPEs, have not responded to LDN.)

An overview of these results must assume the basic statistical principle that the patients with no follow-up contact have not done as well as those who have maintained continual medical contact with Dr. Bihari. Measured in terms of disease stabilization and/or movement toward remission, and assuming that patients in continual follow-up are twice as likely to have had a good outcome thus far, it appears that over one-half of all cancer patients whom Dr. Bihari has started on LDN have done well.

Taking into account the relatively large number of patients who were in advanced stages of disease when first seen by Dr. Bihari, and that some patients in the "not followed up" and "LDN < 6 mos" groups will likely have positive outcomes, it appears possible that more than 60% of patients with cancer may significantly benefit from LDN. This is underscored by Dr. Bihari's observation that better outcomes tend to be seen when treatment with LDN is begun in earlier stages of the disease.

Of interest, there is a negligible rate of relapse in patients who are started on LDN after or during successful initial treatment with surgery (e.g., for breast cancer) or with chemotherapy (e.g., for Hodgkin's disease or non-Hodgkin's lymphoma). It will clearly require extensive study of LDN in prospective, controlled clinical trials to determine which cancers respond best and which other therapies are complementary to or synergistic with LDN.

Other Developments

LDN Alone in the Treatment of Cancer. Dr. Bihari now has 88 patients with cancer in complete or partial remission whose improvement appears to be clearly attributable to LDN alone. In contrast, the vast majority of patients who consult with him for cancer tend to be on other concurrent treatments as well, which obviously interferes with drawing conclusions about LDN's role in their improvement. The successful LDN-only group includes five breast cancer patients, one patient who had widespread metastatic renal cell carcinoma, three with Hodgkin's disease and six with non-Hodgkin's lymphoma. Other such cases, some now on LDN for as long as four years, include a score of patients with non-small cell lung cancer, as well as patients with ovarian cancer, uterine cancer, pancreatic cancer (treated early), untreated prostate cancer, colon cancer, malignant melanoma, throat cancer, primary liver cancer, chronic lymphocytic leukemia, multiple myeloma and some others.

NCI Examining LDN Cancer Cases. In June 2002 an oncologist and an oncology physician's assistant from the National Cancer Institute reviewed some 30 charts of cancer patients at Dr. Bihari's office. About half were chosen as appearing to have responded to LDN without question. With patients' permission, copies of these were sent to the NCI for further data collection on its part for consideration for NCI's Best Case Series.

Noteworthy Cases: As of June 2004 —

Lung Cancer. C., a 61 year old woman, previously a heavy smoker, was found to have a lesion in the right upper lobe of the lung in 1999 and a supraclavicular node in April 2001. Biopsy showed that the node was metastatic from the lung tumor. In August 2001 an MRI of the chest showed supraclavicular clusters of nodes and stellate-shaped lesions in the apex of the right upper lobe. She then started taking

low dose naltrexone. She began getting quarterly C-T scans of the chest, which have shown no change over the following 40 months. The C-T scan interval was changed to every 6 months. Her most recent C-T scan in the spring of 2004 continues to show no change from the August 2001 films.

Malignant Melanoma. L. is a 53 year old woman with metastatic malignant melanoma whom Dr. Bihari first saw in August 2000. Her primary skin lesion had been removed from the lower back in late 1976. A lump in the left groin was biopsy positive in December 1977. It appeared to respond to treatment with BCG in a clinical trial in January 1978. She was disease free for 20 years until a cancerous lesion appeared near the site of the original primary. It was removed surgically. She started a melanoma vaccine trial in April 1999 but developed two new skin lesions on the low back over the next six months. In February 2000 a bone scan showed a lesion in the left sixth thoracic rib, with growth evident on a repeat bone scan in April 2000, which also showed further lesions in the left sacrum and the L5 vertebra. She began taking low dose naltrexone in August 2000. She showed no growth of these three bone lesions and no appearance of new lesions over a forty month period since that time. She has remained on naltrexone only.

Esophageal Cancer. Reverend X is a patient at Johns Hopkins Hospital where he received most of his medical care. He first developed problems with digestion and some pain in the mid-chest area with swallowing in April 2002. An upper GI exam in May 2002 showed narrowing and irregularity of the lower esophagus. In June 2002, a C-T scan of the chest, abdomen and pelvis showed a 2cm thickening of the lower esophagus extending into the upper stomach. Also seen were five enlarged nodes in the chest and five in the abdomen. Rev X refused chemotherapy and began low dose naltrexone in August 2002. In the following months his difficulty in swallowing has significantly decreased and his weight has stabilized. He notes an improved sense of well being. He has had no therapy but low dose naltrexone.

Renal Cell Carcinoma. R., a 41-year-old man from Toronto with renal cell carcinoma, with metastatic lesions in his liver and lungs, contacted Dr. Bihari about 36 months ago. His oncologists told him there was no effective therapy available, and he said he was anxious to try treatment with LDN. There was no further contact with the patient until early 2002 when his wife called to thank Dr. Bihari. She said that he was doing quite well and that there had been complete clearing of the metastatic lesions as demonstrated by chest and abdominal CT scans.

Throat Cancer. D., a 54-year-old man who had cancer of the tonsillar area in his throat along with two large metastatic lesions easily visible in his neck, had refused the extensive head and neck surgery proposed by his physicians. They held out little hope for him. Thirty months ago, Dr. Bihari prescribed LDN. The patient's most recent contact with Dr. Bihari was in May 2004 when he was examined. The primary tumor had decreased by one-third in size and the two neck masses had regressed by about 50%. The patient had received no radiation or chemotherapy but had tried unproven alternative treatments obtained in Mexico.

Non-Hodgkin's Lymphoma. B., a 75-year-old woman, was diagnosed with non-Hodgkin's lymphoma in January 1999 by a biopsy of an enlarged lymph node in the side of her neck. CT scans showed enlarged nodes in her chest and abdomen, as well as an enlarged spleen. Bone marrow biopsy showed "10% involvement". Her oncologist recommended a wait and watch approach. She started LDN in July 1999. In January 2000, CT of the chest showed an approximately 50% decrease in the size of all the involved nodes. Repeat CT of the chest in November 2000 showed an 80% decrease in total tumor mass.

Prostate Cancer. M. is a 59-year-old man with prostate cancer, diagnosed with a biopsy and CT scan in September 1999. With no treatment other than low dose naltrexone, after 4 months on LDN his PSA dropped from 6.3 to 3.4. A special ultrasound, performed after 6 months on LDN, showed a 65% shrinkage of the tumor. His PSA remained stable over the following 16 months when he became ill and died of what may have been a cerebrovascular accident.

Pancreatic Cancer. D. was an 82-year-old woman with pancreatic cancer, treated with surgical removal in April 1999. Scans showed that a tumor mass had reappeared in the pancreatic area in August 1999, and two metastatic lesions were noted in the liver at the same time. She started low dose naltrexone in September 1999 and stopped taking gemcytabine at that time after a short course of four weeks. Some four months thereafter, an MRI demonstrated disappearance of the primary tumor that had previously re-grown, and the liver metastases had cleared entirely. Two months later, D. had a heart attack and died.

Carcinoid. C. is a 53-year-old woman with carcinoid, a malignancy that generally arises in the appendix or small intestine and spreads to the bones and throughout the abdominal cavity. She started LDN in June 1999. At that time, she had considerable abdominal swelling, diarrhea two to three times a day, frequent episodes of flushing due to the tumor, poor energy and appetite, and significant metastatic spread to numerous bones. No other treatment for the cancer was administered; none was available. By December 1999, much of the cancer-induced swelling of the abdomen had receded, the diarrhea had completely stopped, the flushing had stopped, and the pain in her right elbow, due to a bony metastasis, had markedly decreased. Follow up in February 2001 indicated that she still had some of the above symptoms and, though clinically stable, was not showing further movement towards remission. A telephone follow-up call in April 2004 indicated that she was experiencing only minimal symptoms.

Multiple Myeloma. W. is a 72-year-old man with multiple myeloma, diagnosed in the summer of 1998 when a medical workup for severe back pain (that occurred while playing golf) revealed fractures of three vertebrae. Tumor was present in several other bones, blood counts were low, and a bone marrow biopsy showed 20% replacement of normal marrow with myeloma cells. His serum paraproteins were very high, as they often are in people with myeloma, at 12.6 and with no response to high dose chemotherapy. He started LDN in January 1999 and continued intermittent chemotherapy until October 1999.

Since then, he had no chemotherapy but remained on LDN daily. There was a gradual normalization of all of his blood counts, as well as a drop in his abnormal serum proteins from 12.6 to a normal level of 1.4. Bone scans showed continued slow healing of affected bones, and two bone marrow biopsies showed no sign of myeloma. He had deferred plans for a high-dose chemotherapy with stem cell transplant procedure which had been earlier, and had decided to "watch and wait" while continuing nightly LDN. He was back to playing golf and tennis regularly, but there has been no contact since early 2003.

Hodgkin's Disease. H., a 36-year-old RN with Hodgkin's disease, was diagnosed in October 1991 with fevers, multiple infections (including toxoplasmosis of the brain), and a positive lymph node biopsy. She had a brief remission of several months following treatment with antibiotics and chemotherapy. She refused repeat chemotherapy when tumor activity resumed, and she remained ill with fevers and many gradually growing tumor masses (externally and internally) over the next four years. She started LDN in June 1997. No other therapy was provided.

By October 1997, her fevers had cleared, all of her external enlarged lymph nodes had shrunk to normal, and all of the enlarged nodes seen in the spring of 1997 on CT scans were gone. She was determined by her oncologist to be in remission. Since that time, she has moved, gotten married, and not returned repeated phone calls. A long term friend reported that she continues to do well except for some persistent memory loss (due to brain lesions associated with her toxoplasmosis). She has stayed on LDN since and, as of the last phone contact in October 2003, had had no sign of relapse.

Non-Hodgkin's lymphoma. J., a 48-year-old man, had a CT scan in January 1999 because of low back pain after an auto accident. In addition to a bulging disc in his spine, the CT scan showed many enlarged abdominal lymph nodes. Biopsies of nodes in two locations were diagnostic of a non-Hodgkin's lymphoma. The patient refused chemotherapy and treated himself with antioxidants and multiple nutritional supplements. He added low dose naltrexone in October 1999. A repeat CT scan in late January 2000 showed a significant reduction in the size of the pathological nodes, each being reduced in size by about one-third. A more recent CT scan in early August 2003 showed further shrinkage of the enlarged nodes, which were reduced to less than 50% of their original size. The reduction of tumor mass occurred in the absence of chemotherapy or other standard treatments, with low dose naltrexone his only pharmacologic therapeutic agent.

Breast Cancer. M. is a 41-year-old patient with breast cancer, diagnosed and treated elsewhere in 1998, whose course was complicated by a recurrence involving metastasis to the hip. Outpatient hospice services were sought. Her walking was so badly impaired that she had to be assisted by her friends on her first office visit to Dr. Bihari in June 2000 ? at which time she began LDN. She revisited his office in mid-October and reported that she not only was able to return to work but also was well enough to play tennis again. Repeat bone scan in October 2000 showed a 40% reduction in metastatic tumor mass. She then enrolled in an experimental chemotherapy trial at a major cancer treatment center in New York in December of 2001 and died of liver failure on the fourth day of the trial.

Non-small Cell Lung Cancer. M. is a patient in his late 50's who first visited Dr. Bihari in June 2000. A chronic cigarette smoker, he was told in May 2000 that he had metastatic non-small cell lung cancer. Many abnormal opaque areas had been seen on his chest x-ray, and a biopsy performed on a sizable mass in his right neck had confirmed the diagnosis. He had refused chemotherapy. On examination, he had a 3cm x 4cm x 2cm mass in his right neck. He was started on LDN in mid-June 2000 and, at the beginning of November, revisited Dr. Bihari for the first time. At that time, the patient reported that energy was better and his appetite was good. He had regained 15 pounds, and had returned to working full time. The volume of the neck mass appeared to have decreased by 50%. An MRI exam in November 2000 showed 80% shrinkage of the right neck mass and 20% shrinkage of the masses in both lungs. As of April 2004, the mass in his right neck remained halved in size, with no further growth of his pulmonary lesions.

Ovarian Carcinoma. V., a 49-year-old woman, first visited Dr. Bihari in early September 2000. She had a five-year history of ovarian carcinoma, with a persistently growing tumor despite repeated courses of chemotherapy and multiple debulking surgery. There was recent increased involvement of the descending colon with the disappearance of formed stools, and she was now experiencing vomiting. Hospitalization was under consideration. She had lost 15 pounds in the two weeks prior to her visit. She was started on LDN at that time, in addition to her existing low-dose Taxol therapy, and within ten days the signs of large bowel obstruction had disappeared.

In four weeks, a repeat CA 125 revealed that this tumor marker had dropped from 1600 to 87. Within the first week of November 2000, it was reported down to 42, and her gynecologic oncologist told her that, on abdominal-pelvic examination, he found no masses. She had regained some 25 pounds and felt ?wonderful?. A repeat MRI showed no visible masses. In March 2001, the CA 125 had risen to 52, then 70, with no return of symptoms or of palpable masses on abdominal and pelvic exams. However, in October 2001 the abdominal masses recurred despite LDN and she died of metastatic cancer 4 months later.

Background

Before it was first used to treat cancer, LDN had been in use in the treatment of HIV/AIDS. A double-blinded placebo-controlled trial in 1986 showed significant immune system protection from HIV in a group of patients given the active drug. The development of LDN was based on several biological facts. One was the fact that naltrexone, which had been licensed in 1984 as an adjunct in treating heroin addiction, has the ability to induce increases in the endorphin levels in the body. Another was the fact that endorphins are the primary supervisors or (homeostatic) regulators of the immune system, representing 90% of immune system hormonal control. Ninety percent of the day's endorphins are produced by the pituitary and adrenal glands between 2a.m. and 4a.m.

Dr. Bihari and his colleagues then showed that endorphin blood levels averaged less than 25% of normal in people with AIDS. These facts all provided the background for the discovery of the value of LDN in HIV/AIDS. The nocturnal production of endorphins allowed Dr. Bihari and his colleagues to experiment with small doses of naltrexone taken at bedtime in order to jump-start endorphin production. They found that LDN increased endorphin production when taken at bedtime in doses of 1.5mg to 4.5mg. Doses lower than 1.5mg had no effect on endorphin production. Doses higher than 4.5mg produced no more of an endorphin boost, but did block endorphins for significantly longer, thereby reducing the benefit of increased endorphin levels.

During the course of the placebo-controlled trial of LDN in people with AIDS in 1986, a friend of Dr. Bihari's (M.B.) called him when she discovered that she was experiencing an exacerbation of non-Hodgkin's lymphoma which had gone into remission five years earlier after treatment with chemotherapy. Because of her awareness of the decreased likelihood of a long-term remission with a second round of chemotherapy, she called to ask if his AIDS drug might help her cancer.

A recently published study of human lymphoma transplanted into mice suggested that it might. In this study, all of the mice in an untreated group died of lymphoma. A second group of mice was pre-treated with a single injection of beta-endorphin before the lymphoma transplant. Half of this second group did not get ill with lymphoma. The other half of these mice did, but with a much more slowly growing tumor and a much prolonged life span compared with that of the non-pre-treated group.

Dr. Bihari agreed to treat M.B. with LDN, and used the three golf-ball-sized tumors in her groin as markers of response. All three shrank and disappeared over the next six months. M.B. stayed on LDN and had no further exacerbations of her malignancy. She died six years later in her mid-seventies from her third heart attack. Several months later, Dr. Bihari, while in Paris to present the LDN AIDS results at an International AIDS Conference, met a woman (C.P.) in her early forties who was quite ill with metastatic malignant melanoma. This had spread from a malignant mole on her arm to her brain, which showed four metastases on C-T scan. Her speech was slurred, her balance and handwriting impaired, and she suffered from headache and recent memory impairment. Her oncologist in Paris said the malignancy was

untreatable, and believed that she had perhaps three to six months of life remaining. On his return to New York, Dr. Bihari shipped LDN to C.P.'s daughter, who started the patient on it. Nine months later, with all neurological signs and symptoms having cleared, C.P. had a repeat C-T scan, showing no residual tumor.

C.P. remained on LDN for the succeeding 12 years, stopping it without her family's knowledge in late 1999. Until that time, she had remained in complete remission, without any recurrence of her malignancy. Eight or nine months after stopping LDN she developed nodules under her skin and began to cough up blood. A C-T scan of the chest showed multiple metastatic lesions. Biopsy of one of the subcutaneous nodules confirmed recurrence of malignant melanoma. Dr. Bihari shipped LDN to the patient's family and she resumed it in early 2000. Eight months later, the nodules in the skin had cleared and a repeat C-T scan of the chest showed no residual tumor. She appears to be, once again, in remission.

Over the years encompassed by these two cases, 1986 to 1999, Dr. Bihari focused his research energy on the study of LDN's effect on immune function and on immunological approaches to the treatment of HIV/AIDS. In 1999, however, conversations with three small pharmaceutical companies revealed some interest in the development of LDN, with a goal of getting FDA approval for immune-related diseases including cancer. With this development possibility, Dr. Bihari decided to revisit the potential value of treating cancer with LDN.

Dr. Bihari began an informal private-practice-based evaluation of the effects of LDN with a variety of types of cancer in February 1999. He had seen positive results with a small but growing number of patients with cancer during the preceding 14 years, while developing the drug as an immune modulator for HIV/AIDS. The drug was compounded by pharmacists in 3mg capsules and taken once a day at bedtime. Most patients have recently had their LDN dose increased to 4.5mg daily. It is nontoxic and has no side effects. Its only interaction with other drugs is with narcotics (such as morphine, Demerol and Percocet), which it briefly blocks.

Mechanisms

The mechanisms involved in the apparent beneficial effect of LDN on cancer have three main elements. The first is the effect of LDN, when taken late at night, in inducing a sharp increase in pituitary and adrenal production of beta-endorphin and metenkephalin, respectively, in the pre-dawn hours, when 90% of the day's manufacture of these hormones occurs.

Most studies have shown that naltrexone induces a two to three-fold increase in production of metenkephalin, the endorphin that most specifically activates delta-opioid receptors, the primary endorphin-related anti-growth factor on cancer cells. The low dose of naltrexone, which in higher doses would block endorphin and enkephalin action on the receptor, is gone from the body in about three or four hours ? whereas the elevated levels of endorphins and enkephalins persist all day.

The second step involved in the anti-cancer effect of these hormones results from *direct* activation of opioid receptors of cancer cells by the increased endorphins. If this activation occurs while the cell is dividing, it dies. In fact, relatively small concentrations of metenkephalin, when added to human pancreatic cancer cells or human colon cancer cells growing in the test tube, have been shown to kill both. The apparent mechanism of cell killing is called apoptosis (programmed cell death). This appears to be one of the mechanisms by which endorphins and enkephalins combat cancer.

A third element, which may play a major role in controlling cancer, involves the cells of the immune system, which is regulated/orchestrated to a great extent by endorphins. In particular, endorphins raise the circulating levels of natural killer cells and lymphocyte-activated CD-8 cells, the two immunological cell types that prevent cancer by killing cancer cells as they arise.

It should be emphasized that Dr. Bihari's patients were all treated in a private practice setting without the scientific rigor of a prospective clinical trial. This precludes any scientific claims about the drug's efficacy in treating any of the above-mentioned types of cancer. The results thus far do, however, raise the possibility that the manipulation of opioid receptors on cancer cells as anti-growth factors through the use of endorphins and endorphin-inducing opioid antagonists may eventually prove to have considerable merit, particularly in view of the many years of published, supportive laboratory research findings.

Those cancer cells that have opioid receptors on their cell membranes, and that may, therefore, respond to LDN, include all of those that arise from the gastrointestinal tract. This includes the mouth, esophagus, liver, pancreas, stomach, small intestine, colon and rectum. Lymph glands and the spleen have large numbers of opioid receptors, suggesting that Hodgkin's disease, non-Hodgkin's lymphoma, multiple myeloma and lymphocytic leukemia should respond to LDN. Other malignancies with sizable numbers of opioid receptors on their cell membranes include breast cancer, neuroblastoma, prostate cancer, malignant melanoma, renal cell carcinoma, glioblastoma, astrocytoma, endometrial cancer & small cell and large cell cancers of the lung.

Research History

Ian Zagon, Ph.D., whose research group has done much of the basic animal work in the area of cancer treatment and endorphins, showed in 1981 in a mouse neuroblastoma model that very small doses (0.1 mg./kg) of naltrexone, given once a day, inhibit tumor growth, prolong survival in those mice that develop tumors and protect some mice from developing tumors altogether.^{2, 3}

Zagon had hypothesized that the small daily doses of naltrexone work to enhance the endorphin-related protective effect against cancer in mice by increasing the number and density of opiate receptors on tumor cells. He hypothesized as well that the increase in endorphins known to be induced by naltrexone might play a part in the protective effect of the small daily dose by working directly on the tumors' opiate receptors.⁴

In 1996 and 1997, Zagon and his co-workers, reported on laboratory research using specially-bred mice that had no immune system (so-called "nude mice"). They transplanted, in separate experiments, human colon cancer and human pancreatic cancer into the animals and compared the growth of the cancer between those mice that received daily injections of metenkephalin and a control group that received placebo. In each experiment, metenkephalin acted as a negative regulator of tumorigenesis and was significantly able to suppress tumor appearance and growth in the treated group.⁵ (abstract)

Of especial importance, in 1996 the same group of researchers demonstrated that by utilizing LDN to induce an intermittent blockade of opioid receptors in similar laboratory animals (nude mice), the growth of inoculated human colon cancer was markedly retarded. "At the time (10 days) when all control mice had tumors, 80% of the mice in the 0.1 mg/kg NTX group had no signs of neoplasia." When measurements of metenkephalin plasma levels were made, the group that received LDN had metenkephalin levels that were elevated 2.5-fold compared with the control group. The researchers concluded that the results suggested "that daily intermittent opioid receptor blockade with NTX [low dose

naltrexone] provokes the interaction of opioids and receptors in the interval following drug availability, with opioids serving to inhibit tumorigenicity of human colon cancer".⁶ (abstract)

New findings by Zagon and colleagues at The Pennsylvania State University in Hershey were published in the December 1999 issue of the journal *Brain Research*. They had identified the specific cell receptor for one of the endorphins, metenkephalin (the levels of which are increased by LDN). Zagon stated that the opioids act as growth inhibitors, as well as neurotransmitters, and that this feature has implications for cancer treatment. Metenkephalin is found in all tissues, and appears to be associated with cells undergoing renewal or proliferation. Zagon's group was described as having mounted Phase I trials using metenkephalin in an attempt to control the growth of pancreatic cancer in humans. Pancreatic tumors appear to have low levels of the metenkephalin receptor. Low peptide [metenkephalin] or [opioid] receptor levels may exist in cancer cells in general since they want to stimulate their own growth, Zagon said.⁷ (abstract)

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Dr Julian Whitaker says: LDN requires a prescription and can only be obtained through compounding pharmacies. (Regular pharmacies typically carry only 50 mg capsules.) The optimal dose of LDN is 4.5 mg at bedtime. Some people have vivid dreams when they first begin using LDN. If this is an issue for you, start with 1.5–3 mg and build up over two months. Do not take LDN if you use narcotic drugs—it blocks their effects and causes withdrawal symptoms. LDN may be started three days after narcotics are completely out of your system. To learn more, visit Dr. Gluck’s Web site, lowdosenaltrexone.org, and search the Internet for LDN.

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Dr. Bernard Bihari, 29 W. 15th Street, New York, NY 10011 212-929-4196

Chapter 16 – Surgery: Potential for Metastasis

A number of newsletters have reported the result published in an extremely prestigious journal [1] of a significant study from the highly regarded John Wayne Cancer Institute in California. They studied 663 women known to have breast cancer. Half had a biopsy, and the other half had surgery removing the tumor. The study found that women who had any kind of needle biopsy were 50% more likely to have cancer in their sentinel nodes than women who just underwent surgical removal of the tumor.

The report stated that “*manipulation of an intact tumor by fine needle aspiration or large-needle core biopsy is associated with an increase in the incidence of sentinel node metastases, perhaps due in part to the mechanical disruption of the tumor by the needle.*”

Up to now, modern medical experts had uniformly assumed these biopsies were totally safe (but this historically was not always so). In 1940, the influential *NY Medical Record* said, “One who harpoons ... a tumor ..especially an encapsulated tumor ... will almost inevitably destroy his patient’s chances of recovery....”

In 1974, the American Cancer Society textbook on cancer stated, that biopsies “may contribute to the spread of cancer in some cases ...” By 1991, their concern was not so explicit, stating that the disadvantage of the larger core needle biopsy is “seeding of the needle track with tumor cells.”

Yet the John Wayne report is not the first to cast a modern stone. In 2004, an article entitled “Useless and dangerous — fine needle aspiration of hepatic colorectal metastases” appeared in the prestigious *British Medical Journal* [2].

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Follow-up to Surgery and Cancer.

In a report in September¹ [2005], researchers uncovered evidence that taking out a breast tumor triggers the release of substances (perhaps as part of the wound healing process or perhaps in response to the absence of the tumor) which enables cancer cells to grow that had been previously lying dormant in other parts of the body.

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Chapter 17 – Breast Cancer: Radiation and Survival Benefits

A standard treatment for early-stage breast cancer is to remove the tumor via lumpectomy and then follow that with radiation therapy and the drug, tamoxifen. But a report presented at the 2010 annual meeting of the American Society of Clinical Oncology (ASCO) has called this approach into question. Researchers at Massachusetts General Hospital, Boston, studied women over the age of 70 who had estrogen receptor positive (ER+) tumors that were removed by lumpectomy. The subjects were randomly assigned to receive either tamoxifen alone or tamoxifen *plus* radiation therapy.

After more than 10 years, the women who received just the tamoxifen fared about the same as those who also received radiation. Although radiation resulted in fewer recurrences in the affected breast, the chance of being free from distant metastases was 95% with tamoxifen alone vs. 93% for tamoxifen plus radiation. The 10-year breast-cancer-specific survival was 98% with tamoxifen alone vs. 96% with radiation. The overall survival was 63% with tamoxifen alone vs. 61% with radiation added, i.e., survival was slightly higher when women did *not* receive radiation.

The authors themselves concluded that “the addition of radiation does not impact survival, distant disease free survival, breast cancer specific survival or breast conservation” (Hughes 2010).

The Web site Breastcancer.org states that “these results shouldn’t be used to make treatment decisions for women younger than 70.” Nonetheless many readers are bound to wonder whether radiation is worthwhile for women under the age of 70. That wasn’t addressed in this study. Radiation’s main purpose after breast surgery is to prevent recurrences, and it improved the odds by 2%. However, its impact on survival is not as demonstrated as some people suppose. Even the authoritative Perez and Brady textbook refers to “the lack of survival benefit associated with breast irradiation....” Needless to say, a lot of questions remain about the actual survival benefit of radiation therapy, including some indications for which it is now commonly used.

References

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Further followup with citations:

1. Dr. Bernard Fisher found no survival benefits from adding radiation-therapy to surgery for breast cancer in 1989 after 8 year follow-up and confirmed this in 2002 after 20 years follow-up.

2. Similarly the Early Breast Cancer Trialists' Collaborative Group in the UK found no survival benefits from adding Radiotherapy to Surgery in Early Breast Cancer in 1995. All of these studies were in the NEJM.

3. Richard Evans on his website at least 15 years ago was aware of the lack of survival benefit from adding radiotherapy to surgery for breast cancer.

4. The fact that radiotherapy causes long-term harm when used for breast cancer has also been known since 1994 when Jack Cusick published his paper in J Clin Oncol.

5. In 1993 Barry Brown writing in the JNCI found that treatments for breast cancer (including radiotherapy) increased the deaths from other causes by 9 percent.

All the most recent trial confirms is that what has been known for at last 15 years for women aged below 70 years also applies to women older than 70.

Chapter 18: -- Sulforaphane: Important Supplement Update

There is an important advancement in treating cancer, particularly prostate cancer. Our own research had previously documented that one of the main components in broccoli can target cancer cells through multiple mechanisms. New research puts this choice over the bar for its use among supplements addressing cancer in general and prostate cancer in particular.

Specifically researchers have shown¹ that this element called sulforaphane *selectively targets both benign hyperplasia prostate cells as well as cancerous prostate cells while leaving the normal prostate cells unaffected*. So this compound selectively kills just cancer cells with a specific affinity for prostate cancer. This compound inhibits HDAC enzymes which otherwise permit cancer to flourish and also helps turn on important genes that were silenced by cancer besides restoring normal cellular function that might have been altered by cancer. This is remarkable. [Note: this action is different from DIM found in broccoli, and you still need to take DIM for its benefits. This product does not substitute for DIM]. Your Editor notes this is not just the first time such results have been achieved with this supplement. It is the accumulated research that causes us to strongly suggest this supplement not only for prostate, breast, and colon cancer patients but other cancers also.

A capsule with a 20-to-1 broccoli sprout concentrate with guaranteed levels of sulforaphane (30 mg per capsule) can be found at the Swanson's web site [www.swansonvitamins.com or call 800-437-4148] It is product #JR0508 – a product by Jarrow – for \$18.7 for 60 capsules. Based on past consultation from my former colleague, Dr. Stephen Martin, I am suggesting 1-2 capsules a day if you have cancer, depending on your budget, particularly prostate or breast cancer. If you do not have cancer, take 1 daily as a strong cancer preventive.

Some of you may be eating cruciferous vegetables such as broccoli or cabbage, kale, cauliflower or even broccoli sprouts. Broccoli sprouts permit a recovery of about 74% sulforaphane while broccoli powder alone for example yields 19%. The sulforaphane is stored in the vegetables and must be released by an enzyme called myrosinase. Without the myrosinase, you cannot obtain the crucial sulforaphane. Now here is the clincher: heat destroys this needed enzyme so if you cook any of these vegetables, you get essentially none of the sulforaphane. Moreover capsules that contain cruciferous vegetables which sound healthy most likely do not contain the needed enzyme. So you do not get this crucial benefit.

To emphasize this point, an important study² found no correlation at all between consumption of cooked cruciferous vegetables and bladder cancer but found that individuals who ate one or more servings of *raw* broccoli per month had less than half the risk of their bladder cancer returning compared to those who ate broccoli less often. In short, you are losing most of the cancer-fighting power of your broccoli if it is cooked. Now you can do the following. If you are eating cooked broccoli, just take a capsule of the supplement noted above to get your enzyme to then take full benefit of the cooked broccoli. Just one would do. If a capsule is not handy, eat some raw broccoli with your cooked vegetable and you get the enzymes to process the cooked vegetable.

While we are strongly recommending the addition of this supplement for cancer prevention or cancer control, we note other research³ showed that sulforaphane also inhibited a variety of bacterial and fungal

pathogens as an added benefit. What's more, there are some few but interesting reports of the sulforaphane improving urination among men with enlarged prostates who have tried innumerable other strategies without much success. This would not be unexpected since sulforaphane is also suppose to shrink somewhat an enlarged prostate.

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Chapter 19: Immune Phase Protocol

In this immune phase, we are attempting to activate the innate immune system against the cancer cells. The innate immune system consists of gamma delta T cells, NK or natural killer cells, certain CD8 T cells, activated macrophages and neutrophils. Properly activated, these cells can non-genetically kill any cancer cell. With very few exceptions, cancers and leukemias are sufficiently reactive to be recognized and killed by the immune system *if their secretion of immuno-suppressive factors can be blocked*. That is the essential problem: all cancers and most leukemias secrete factors that block their destruction by the innate and adaptive immune systems. This is why cancer vaccines work in a culture dish, but not in the body. Therefore, our number one goal in this part of the protocol is to inhibit the synthesis and/or release of these blocking factors while at the same time also enhancing the immune system.

With cancer present, what we specifically uncovered was the fact that as a result of cancer, the Cox-2 enzyme and its product PGE2 activate all four known immuno-suppressive pathways. These pathways prevent the immune system from finding the cancer. So in short, cancer is using PGE2 to keep the immune system from finding it. However we find that selenium in our Protocol B specifically inhibits PGE2. So we definitely will want the selenium from Protocol B elected in any immune plan since clearly the inhibition of Cox-2 synthesis and activity is our number one priority in this Immune Protocol.

Our second priority is to block STAT3 activation, which we found to be another critically important immuno-suppressive factor which cancer generates to keep from being detected. What we also discovered was that the synthesis/activation of STAT3 can be blocked by curcumin and feverfew [parthenolide]. Fortunately both are in our Protocol as curcumin or Meriva in Protocol A and as feverfew in Protocol C. So again, these two need to be pulled forward for the Immune Protocol to work.

At the same time, we alert you that some useful factors that improve the immune system will impede the cancer cell death we wish to achieve in the first part [cytotoxic] of the protocol. Specifically, recall that some supplements in the first part work by creating a free radical storm to kill cancer cells but if you take antioxidants which halt these storms, even if they help your immune system, then you halt the cell death. Innumerable researchers and cancer advisors seem to have missed this point. In short, some supplements will completely inhibit other ones. To give an important example, we thought of adding lactoferrin to this part of the protocol since it will enhance your immune system. However it significantly reduces oxidative stress, and by so doing, will inhibit some of the supplements in the first three protocols. It took many hours to document its value, then even more hours to uncover its mechanism of action, and thus in the end to disqualify it.

However – it is not as simple as avoiding **any** antioxidants. Far from it. Some so-called antioxidants are in fact *immune modulators* – meaning that they can be both oxidants AND antioxidants just depending on the cellular circumstances.

For example, vitamin C is an antioxidant when we need it to be – and an oxidant when this aspect is needed. So we must use the research literature to identify the right choices – not just “common sense” which may not give the right answer at all. Below we identify additional immune system activators we

believe are safe to take with the first part of the Protocols [Protocol A, B, and C] which will further enhance your anti-cancer impact.

As important, and discussed above, we identified the key factors that permit cancer cells to hide their existence – and then identified supplements which will prevent this cloaking. To summarize, there was curcumin as the product Meriva In Protocol A. Another was selenium in Protocol B. Finally there was feverfew in Protocol C. These three will work to halt the cancer cloaking. So to make this immune protocol effective, you will need to include them if you are to add a powerful immune support component.

One obvious question arises: who should add this non-contradictory immune component? This question has taken more time than might be obvious at first. Weighing the issues of compliance and cost, nonetheless I believe that since cancer is known to be systemic – affecting the whole body no matter which place or organ first exhibits it – then it will take a systemic strategy to find all the cancer cells. Only the immune system can undertake this difficult seek-and-destroy mission.

The next question might be: then why not just take the immune protocol alone? The answer comes from our past experience. If cancer is well-established enough, which is the case if you have any staged cancer, then the immune system approach is not likely strong enough on its own to eliminate the cancer. Still – it is probably “close.” But our personal experience has been that the added power of some [or a great deal] of cytotoxic cancer cell killing from the first Protocols A, B, and C most likely must be added to the immune strategy. I will try to approximate the various levels or cancer states that will correspond to the Protocol combinations.

A. No Cancer – Elevated Risk

If you are at elevated risk for cancer, but have not had any detected, then actually this case has been specifically addressed by our Cancer Prevention Trial which is not actually discussed in these documents. The reason is that there is some cost to participate in the trial which we have sponsored for more than 10 years now and we do not wish to introduce the issue within the Cancer *Control* Book. You will need to contact Steven Evans at sevans@gsm-usa.com to learn more about this trial or go to the web site www.protocols4health.com to read about this trial

B. Cancer Which Allegedly is Completely Gone

For those who have had cancer other than a nearly irrelevant simple skin cancer, you should take Protocol A to prevent metastasis from any lurking cancer cells, some or all of Protocol B, since this will address precisely those lurking cancer cells, and ideally the Immune Protocol to attempt to get your immune system to operate at a higher level to prevent cancer cells from arising in the future. Some may wish to add the Cancer Prevention Trial Protocol noted above but that is not discussed in this document.

C. Cancer Which is Under Watchful Waiting

In this scenario, you want Protocols A, B, the Immune Protocol and possibly some selections from Protocol C depending on your circumstances [if you are watching Prostate cancer and it remains steady, no need to add from Protocol C but if it begins to expand, then some of C must be added].

D. If You Have Ongoing Aggressive Cancer

The Protocols A, B, C, and the Immune Protocol are essentially what you need to be taking.

Generally I will try to help each individual fine-tune their Protocols.

Disclaimer: Please note that this effort in providing personalized information has been ongoing and happening and as of this writing, there are probably nearly 140-160 people in the queue waiting right now. Many of you call and expect a return call within a few days while this is not at all possible. “It won’t take long – surely you can just call me.” My great apologies – but even at 14-16 hours a day, nearly 7 days a week, it is not possible right now to return calls. The smartest thing to do is email me at sevans@gsm-usa.com and summarize your cancer history, and there is at least a possible chance I can at least identify Protocol entries for you within a reasonable period of time.

The Immune Boosting Protocol

1. Original “Aspirin” as white willow bark

In a March 2012 study in *Lancet Oncology*, Peter M. Rothwell of the John Radcliffe Hospital, Oxford, showed that regular use of aspirin was associated with a **38 percent reduction** in the incidence of colorectal cancer. There were similar reductions in the risk of esophageal, stomach, bile duct and breast cancer as well (Rothwell 2012).

An important finding was that “regular use of aspirin was associated with a reduced proportion of cancers with distant metastasis....” (*ibid.*) Rothwell examined the medical records of 17,000+ people involved in clinical trials of aspirin for the prevention of cardiovascular events. Of these, about 1,000 developed new solid tumors during the 6.5 years follow-up period. But those who received aspirin were 36 percent less likely to have distant metastases of their cancers. This protective effect was even greater in patients with adenocarcinomas (i.e., solid tumors formed from glandular structures), where there was a one-half reduction in metastatic disease.

We are recommending the original aspirin, white willow bark which is the herbal from which aspirin was derived – for safety reasons. Take 500 mg twice a day. Swanson has product SWH148, a 500 mg capsule, \$6.99 for 120 capsules.

2. L-theanine from Tea

This amino acid, made only in tea plants, activates gamma delta T cells. Actually, ethylamine, a break-down product of theanine activates these cells. Note that tea is already in our protocol B. So whether you take some or all of Protocol B, take the green tea recommendation.

3. Vitamin A

Take vitamin A as a 5,000 IU a day capsule. We uncovered a number of things about vitamin A. First, there is a vast amount of literature documenting its value. Due to time constraints, I will not itemize this substantial body of material. Trust me. We also found that beta carotene which is very typically one way vitamin A is provided [from which the body can make vitamin A] is an antioxidant while vitamin A itself is not. So we must use vitamin A, not the beta carotene form. The other thing we found is that past a certain point, vitamin A begins to negate the value of vitamin D. So there is a “sweet” spot to shoot for – 5000 IU.

Select the Swanson product SW004, which contains 5000 IU of the proper form of A and 400 IU of D3 which is a very small amount and may be safely added to your regimen without worry. The product is \$3.89 for 250 capsules. If you are using the multi-vitamin product that is part of the Cancer Prevention Trial sponsored by the Genetic Services Management [GSM] group, you are already taking this.

4. Vitamin B6

In Chapter 9 [section 11], we note that B6 is particularly valuable for prostate cancer. Here we are focusing on the fact that vitamin B6 increases immune functioning. Consider Swanson's product SW022, a 100 mg capsule. \$4.99 for 250 capsules. Take two capsules twice a day. Even if you are taking the multi-vitamin from GSM, add this nonetheless.

5. Beta Glucans

Beta glucans are tumor cell killers in its own right besides being a major immune system booster. The literature is extremely extensive but I will give one example. Clinical trials in China using traditional chemo yielded a 10% effective rate while adding beta glucans yielded over an 80% cure rate. Extraordinary!. It is now the standard combination treatment in China [not in the US of course]. I suggest Swanson's SWU187, a beta glucans product called BetaRight. It is \$15.99 for 60 capsules. Take at the minimum one capsule twice a day. If your budget allowed it, two capsules twice a day would be ideal [but expensive].

5b. Vitamin C

It has also been discovered that beta glucans are not absorbed very well but its absorption is significantly multiplied if vitamin C is taken at the same time. So with each serving of beta glucans, add 1000 mg of vitamin C. Consider Swanson's SW1054, a 1000 mg capsule [not tablet], \$2.89 for 90 capsules.

6-8 Meriva, Selenium, and Feverfew

Be sure to incorporate the Meriva, selenium, and feverfew from Protocols A, B, and C to properly "activate" this immune section as discussed above.

Historical Note: My late and brilliant colleague Dr. Stephen Martin was seeking the holy grail of a combination cytotoxic and immune boosting protocol his whole life. After I estimate about 2400 hours of work on his part, he did not believe it was possible. After his untimely death, I have added another 1600 hours or more of research time, and building on his brilliant efforts, I believe the additions above might have met his approval for a combination Protocol that provides the benefits of both perspectives. I am deeply indebted to his contributions to this protocol research.

Summary of the Immune Boosting Protocol

1. White willow bark	500 mg capsule	Twice a day
2. Green Tea	300 mg capsule	Two capsules twice a day
3. Vitamin A*	5000 IU capsule	One capsule a day
4. Vitamin B6	100 mg capsule	Two capsules twice a day
5a. Beta Glucans	250 mg capsule	One capsule twice a day
5b. Vitamin C**	1000 mg capsule	Take one with each beta glucans dose
6. Meriva	500 mg capsule	Take 2-4 capsules daily
7. Sodium Selenite	250 mcg tablet	Take one tablet three times a day
8. Feverfew	4.5 grams	Take 3 capsules 3 times a day of the 500 mg capsule

* [not from beta-carotene]

** The purpose of the vitamin C is to enhance the absorption of the beta glucans

Appendix A – About the Author: Steven Evans and the TRI Background

Since I have created this protocol, one question may be: Who the hell is Steven Evans? The short answer is– nobody. All the research I have assembled above is based on the published peer-reviewed medical literature. That is why I took the effort to give the basis for the choices. It wouldn't matter if I had 10 Ph.D.'s and five medical degrees. If there is no basis, there is just unsubstantiated opinion. I prefer to recognize that I am just a graduate of Hartsfield Elementary School and everything after that was "secondary." Still, I recognize that some people feel better if they know the information collector is not a homeless bum with a Commodore 64 computer. So I will give some background, but be certain – this does not imply that the information is any better than the sources upon which it is built.

1. Steven Evans – Brief and Abbreviated Resume.

Steven Evans has been actively engaged in health-sciences-related research, development, and its practical implementation for over 39 years, including the fields of clinical practice, hereditary cancer, genetics, genetic counseling support systems, health sciences education, and nutritional supplements (particularly related to cancer prevention). As a contributor to these fields, he has disseminated and discussed his research and analysis in over 100 journal articles and/or professional presentations, monographs, chapters, books, and/or technical papers. He also holds a number of patents within the health field.

His other research areas have included non-numeric simulations, expert systems, neural networks, and knowledge discovery/data mining methodologies, particularly as they apply to health care, as well as hereditary cancer syndromes and nutritional supplement research.

In 1975, Evans founded and headed the Office of Instructional Science Research within Creighton University's Health Sciences Center, in Omaha, Nebraska to implement a number of his theories and applications. He was Director of Instructional Science Research for the Creighton University Health Sciences Center including the Schools of Medicine, Pharmacy, and Nursing for 14 years during which he intensively studied all the instructional content of these programs of study. He has also provided contributed services to the Hereditary Cancer Institute within the Creighton School of Medicine for 25 years.

As co-founder of Genetic Systems Management (GSM), Inc. in 1990, Evans implemented a hereditary cancer-detection system in conjunction with the Hereditary Cancer Institute (HCI) within Creighton's Medical School. This approach was distributed to major cancer centers nationwide.

Evans later designed and launched an international clinical trial [still ongoing] initiative focusing on cancer prevention and the prevention of cancer recurrences through the use of over-the-counter supplements [For an interim report on progress on this trial, see Evans, S. "Interim Clinical Study Results for A Supplement for Cancer Risk Reduction." *Townsend Letter for Doctors & Patients*, July, 2005].

Later Evans founded the non-profit [and registered 501(C)(3)] Therapeutic Research Institute (in 2001) to identify alternative medicine approaches to all diseases. Evans is Editor-in-Chief of a nutraceutical information newsletter distributed nationally and internationally by TRI. He is the designer and Trial Administrator for a number of prior institutional-review-board-approved clinical trials. Evans is

currently providing consultation regarding his research and alternative medicine protocol developments to clients in the US, Canada, Europe, and China.

You may contact him at sevans@gsm-usa.com but note the following rules. He sometimes receive over 300 emails or more each day. You must put in the subject line "TRI Cancer Protocol" or it may possibly be ignored. Also be aware that of all the cadre of colleagues who undertook with him to provide no-fee consultation to the best of our ability without any biasing affiliations, he is the only surviving member. He takes as much time as is needed with every individual. So it sometimes takes a while to get back to inquiries – you will have to be patient. Very patient.

For those who simply cannot get enough background information, his last 10 publication are appended below along with some books and chapters on a variety of medical topics. Of course remember that past successes are no indication of future performance, as they say in the investment world.

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8. Block, J.B. and Evans, S. "Clinical Evidence Supporting Cancer Risk Reduction with Antioxidants and Implications for Diet and Supplementation," *The Journal of the American Nutraceutical Association*, Vol. 3, No. 3, fall, 2000, pp 6-16.
9. Evans, S., et al. "Automated Detection of Hereditary Syndromes Using Data Mining," *Computers and Biomedical Research*, Vol. 30, No.5, 1997, pp. 337-348.

10. Evans, S, Lynch, HT, and Fusaro, RM. "Clinical Results Using Informatics to Evaluate Hereditary Cancer Risk," in Gardner, RM, ed. *Proceedings of the Nineteenth Annual Symposium on Computer Applications in Medical Care*. New Orleans: Hanley & Belfus, October, 1995 pp 834-838.
Another 80 or so available by special request.

BOOKS

PACE: An Expert Nursing System, New York: Springer-Verlag, 1996 (invited volume for Springer's *Computers and Medicine Series*)

CONTRIBUTED BOOK CHAPTERS

1. Evans, S. "Software Applications for Nursing: Education and Decision-Support Systems," Geisow, M. J. and A. N. Barrett, *Microcomputers in Medicine*, Elsevier Biomed Press, Amsterdam, 1986.
2. Evans, S. "Decision Support Systems for the Outpatient Office: Two Examples of Medical Expert Systems," Zimmer, J., ed., *Primary Care*, v. 12, no. 3, W. B. Saunders, 1985, pp. 445-413.
4. Evans, S. "Computer Literacy in Family Medicine," Zimmer, J., ed., *Primary Care*, Vol. 12, No. 3, W. B. Saunders, 1985, pp. 403-413.

PATENTS ISSUED

1. "Interpretative Systems for Systematic Learning and Support for Decision-Making.
2. "Methods for Identifying Human Disease Patterns."
3. "Process to Modulate Disease Risk with Doses of a Nutraceutical"

2. The Therapeutics Research Institute (TRI)

The mission of the Therapeutics Research Institute (TRI) is to accrue useful information regarding approaches to all diseases and dysfunctions and to disseminate this information world-wide. We do not charge any fees for these consultations. We do not receive any funding from any sources (which otherwise might bias our research) other than private contributors who may elect to provide funding for our efforts. No contributions are ever used for salaries for research personnel who provide all their efforts on a no-fee basis.

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Please pay particular note that all of the information presented above is as accurate as we have been able to make it at the time of this publication but new and updated information that might contradict this presentation can arise at any time.

Also all of the above is presented for educational purposes only. There are no health care providers as part of TRI who are advising you regarding your own specific health care choices. You must check with your own health care provider(s) or any other consultants for actual health care actions that may be best for you. None of the information above is intended as specific medical advice, consultation, or instruction for individuals nor substitutes for professional medical advice. This document does not present information which is alleged or intended to diagnose, treat, prevent or cure any diseases. Moreover the information in his book is not intended to replace a one-on-one relationship with a qualified health care professional and is not intended as medical advice. It is intended as a sharing of knowledge and information from the research and experience of Steven Evans who encourages you to make your own health care decisions based upon your research and in partnership with a qualified health care professional. In other words, as Count Dracula said, enter at your own free will.

Appendix B – Summary of All Protocols: Protocols A, B, and C

Summary of Key Recommendations: Protocol A

	Morning	Afternoon	Evening
1. Meriva	2 capsule		2 capsule
2. Isoleucine	5 grams		5 grams
3. Cimetidine (Tagamet)*	400 mg tablet		400 mg table
4. Super CitraMax	3 capsules		3 capsules
5. Nattokinase	1 capsule	1 capsule	1 capsule

* Tagamet used just for a total of 7-10 days

Summary of Key Recommendations: Protocol B

	Morning	Afternoon	Evening
1. Sodium Selenite	2 tablets		1 tablets
2. Melatonin	-- --		1 capsule
3. Flaxseed	2-3 TBS. or a muffin		2-3 TBS or another muffin
4. DIM*	1 tablet		1 tablet
5. Pomegranate*	1 capsule [for men]		1 capsule
6. Vitamin D3.	1 capsule		
7. IP-6	1 teaspoon		1- 1½ teaspoons
8. Green Tea	2 capsule		2 capsule

* DIM and pomegranate only apply to estrogen-related cancers such as prostate, ovarian, endometrial, breast, and colon cancers.

Summary of Key Recommendations: Protocol C

	Morning	Evening
1. Genistein	2 capsules	3 capsule
2. Policosanol		1 capsule
3. Feverfew	2¼ grams	2¼ grams
4. Niacinamide	1 capsules	1 capsule
5. Curcumin	5-10 grams	5-10 grams

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