

Conquering Cancer Through Vitamin C & Other Antioxidants

The following excerpt from [Cancer: "Nutrition and Survival"](#) is an important bit of information that needs more application, and many have asked, hence this excerpt. The citations can be found in the said book.

"The toxicity of current chemotherapeutic drugs might lead us to suppose that it is hard to find agents that will kill cancer cells without poisoning the patient. This is a misconception. Numerous common substances exist, which offer the property of selectively killing cancer cells, while leaving normal cells unharmed. Indeed, taking vitamin C as an example, these substances can often be beneficial to healthy cells."

Here is a more detailed update from Dr. Hickey (PhD) end stage cancer.

"In this case oral vitamin C is given in divided doses to bowel tolerance level or the maximum the subject can otherwise tolerate. If a person cannot take high doses of standard vitamin C, or they want to get the maximum possible blood levels, they could try liposomal vitamin C. This may be easier to tolerate and allow higher oral doses.

I know one doctor's cancer protocol for IV sodium ascorbate (not vitamin C for IV!) is 75g three times a week supported by oral doses. A cycle of IV therapy lasts 5-6 weeks but the oral doses continue indefinitely. This protocol seems about right to me. The methods/techniques used for IV sodium ascorbate have been described by [Cathcart](#) and [Riordan](#) and are available from the net.

There is limited evidence on the sustained dose of alpha-lipoic acid. However, up to about 3-5g per day in divided doses (say 500mg 6 times daily) can be well tolerated with few if any side effects. R-alpha-lipoic acid absorbs more readily and is more available to the tissues."

Chris Gupta
<http://tinyurl.com/fj63k>

See also:

[The 1982 Letter On Vitamin C and Cancer](#)

[Some Cancer Resources](#)

Conquering cancer

Copyright (C) Dr Steve Hickey (2005). All rights reserved

One of the first duties of the physician is to educate the masses not to take medicine." Sir William Osler

Throughout this book, we have referred to different methods of fighting cancer. Although we have discussed these treatment methods separately, some of them work even better in combination. This exciting possibility could allow the development of alternative treatments, with the potential to overcome cancer in the majority of cases.

The microevolutionary model suggests two broad approaches to limiting cancer growth, based on population control and extinction. For population control, the aim would be to establish an environment that inhibits the division and evolution of cancer cells. A sufficiently slowgrowing tumour would have a negligible effect on a patient's expected lifespan. If growth can be limited at an early stage, so the tumour remains small, its effect on the patient's health might be inconsequential. Population control methods could allow patients to live out their normal lifespan, though this outcome is not certain. For example, when treating patients with chemotherapy, physicians consider tumour shrinkage a measure of success. In evolutionary terms, shrinkage reflects a period of negative population growth. Unfortunately, it also corresponds to a period of intense selection pressure on the remaining cancer cells; those that resist the drug are more likely to survive. At the end of the treatment, growth of these more resistant cancer cells can resume. The only way to avoid this re-growth would be to continue therapy until no more cancer cells remained. In this case, the population would be extinct.

Extinction of cancer cells is the most effective therapy and a true cure for cancer. This means keeping the cancer cells in a state of negative population growth, until they all die. This is easier said than done, since the intrinsic biological diversity of a typical cancer means that a proportion of cells are likely to be resistant to any particular therapy. Large tumours contain a variety of cell types, including normal cells, relatively benign cells, invasive cancer cells and infiltrating white blood cells.¹ Furthermore, tumours in secondary locations are even more diverse. This explains why secondary cancers (metastases) can be more difficult to destroy than primary ones.

The toxic nature of many conventional anti-cancer drugs limits their use as agents for the total extinction of cancer cells. Clearly, if the treatment is toxic to the patient, the cancer must become extinct before the patient dies, or is permanently disabled. For this reason, non-toxic therapies have an advantage in the treatment of intractable cancers.

Patients can take safe therapies indefinitely, and physicians can combine such treatments for maximal effectiveness. We will consider non-toxic therapies shortly, after a brief reiteration of the development and characteristics of cancer.

Understanding cancer

There are two main approaches to cancer, which differ with respect to the importance accorded to the biological diversity found in cancer cells. Some physicians view cancer as a single disease, with many manifestations. From this viewpoint, the diversity of cancer is secondary to its common features. Others stress the biological diversity, regarding cancer as a collection of diseases, which share common attributes, such as abnormal growth and genetic changes. Cancer cells are highly abnormal, with damaged chromosomes and genetic divergence from healthy

cells. A feature of all cancers is the abnormal growth of cells within the body. Malignant cancer is characterised by unrestrained spread to different tissue locations.

Another way of looking at cancer uses the microevolutionary model [described in this book](#). According to this theory, the different manifestations of cancer are the results of a common evolutionary process, in which selection pressures favour the development of 'species of cancer. In microevolutionary terms, the abnormal spread of cancer cells is the natural response of an independent organism – the cancer. Reproduction and spread are primary activities of biological organisms, including the single-celled creatures from which animals and plants evolved. The characteristics of living cells have evolved over billions of years, allowing them to survive, initially as single cells and, later, as multicellular organisms.

Cancer is common, because it does not have to evolve from scratch: we all possess the mechanisms for cancer within our cells. Every cell in a multicellular animal retains the biology of its single-celled ancestors. In single-celled creatures, the biological mechanisms for survival stimulate growth, movement and dispersion of cells. The cells of higher animals suppress these actions, in favour of cooperation and structural integrity. They can do this because they have evolved ways to limit the growth of individual cells. However, if something damages these controls, or removes them from a particular cell, then that cell and its descendents will proliferate, reverting to ancient patterns of division and growth. Such cells become what we call cancer.

Causes of cancer

The individual causes of cancer are manifold. Practically any insult, which leads to inflammation or an increase in oxidative stress in the body, can increase the risk of cancer. The minimum requirement for the formation of cancer appears to be an increase in erroneous cell division. Microevolutionary processes then apply selection pressure, which eventually results in the features of malignant cancer. The standard mechanisms of ecology and evolution describe this process well.

How cancer develops

Cancer is an almost inevitable result of microevolution. Whenever cells in the body divide abnormally or erroneously, they accumulate genetic damage and may become polyploid. Researchers have shown that cancer cells are [aneuploid](#), containing abnormal numbers of damaged chromosomes. [Polyploidy](#), the generation of abnormal numbers of sets of chromosomes, is considered a mechanism for the instantaneous generation of new species. Cells that become polyploid, as in cancer, are likely to behave as independent organisms, rather than members of their original species. Such abnormal cells behave in the same way as any other biological organism, reproducing and spreading, in the process known as cancer.

Antioxidant control of cancer

Antioxidants have a central role in the microevolutionary model of cancer development. [Redox](#) mechanisms occur throughout nature, being widely used for the control of cell division, differentiation and growth. Their involvement in cancer is not surprising, since they are of fundamental importance to the basic chemistry of all

living organisms. Numerous genes, enzymes and small molecules are used in controlling the cell, signalling growth, proliferation and death.

[Redox](#) mechanisms play an integral part in all aspects of cancer development. Free radicals are involved from the start of the process. Throughout its progress, increased free radical damage and internal oxidation drives the growth of cancer cells. Raised levels of oxidants initiate cell proliferation and growth. Later, [lack of oxygen restricts growth](#), until the cancer cells evolve mechanisms to stimulate the formation of blood vessels. The resulting blood supply allows the tumour to expand rapidly and spread to distant sites in the body.

Dietary advice to prevent cancer and slow its early development can be stated succinctly. A diet to prevent cancer is low in carbohydrates, with a high proportion of antioxidants. The consumption of a variety of coloured vegetables and fruit in the diet will reduce the incidence of cancer, because of the antioxidants they contain. Supplementation with a range of antioxidant [vitamins and minerals](#) will also hinder the initiation and microevolutionary development of cancer.

Anticancer agents are surprisingly common

The toxicity of current chemotherapeutic drugs might lead us to suppose that it is hard to find agents that will kill cancer cells without poisoning the patient. This is a misconception. Numerous common substances exist, which offer the property of selectively killing cancer cells, while leaving normal cells unharmed. Indeed, taking vitamin C as an example, these substances can often be beneficial to healthy cells.

In our discussion of non-toxic anti-cancer therapies, we have concentrated on redox-cycling antioxidants, such as vitamin C. Other antioxidants and nutrients, such as curcumin, may act through cell signalling, or by modification of gene expression. Cancer occurs because of the way animals have evolved from a single-celled to a multi-celled state. The success of multi-cellular organisms suggests they have evolved ways of dealing with cancer. Hence, the microevolutionary approach predicts that large numbers of agents that prevent or kill cancer cells should occur naturally. This is indeed the case: safe anticancer agents are common and relatively easy to find. However, the medical establishment is apparently unaware of them.

A diet to avoid cancer

Nutrition is one of the most important ways in which a person can alter their behaviour to minimise their risk of cancer.* The primary feature of a diet to reduce the incidence of cancer is a high intake of antioxidants. These keep the cells in a reducing environment, whereas the development of cancer requires cellular oxidation, to promote error prone division. Eating a wide range of antioxidants will provide maximal benefit. This agrees with conventional advice, which recommends a high intake of vegetables.

*We are assuming that people do not smoke or otherwise actively engage in contact with carcinogens.

However, the idea that people should increase their intake of vegetables rather than using antioxidant supplements appears to be based on magical thinking, rather than

science. Vegetables contain substances that can inhibit cancer growth and development. The identification, isolation and purification of such substances are central features of reductionist science. Once this has been done, the anticancer substances can be made available in concentrated forms, as food supplements.

The advice not to use food supplements might appear holistic or natural, but it is not good science. Historically, the chemical isolation of vitamins and other essential dietary factors represents an increase in knowledge, which has benefited the health of humanity. While there may also be advantages to eating plants that cannot be repackaged in supplement form,** this hardly represents a scientific argument for avoiding those factors that can be isolated and used for maximal benefit. Surely, the optimal approach is to eat a wide range of vegetables, while also using safe supplements. In the absence of complete data, we can suggest that the supplementation should cover a range of antioxidants in their natural forms. In particular, a dynamic flow of vitamin C,*** together with a [reduced intake of sugars and starches](#), is likely to be beneficial.

** Herbal remedies are an obvious example of the potential for the use of whole plants and their extracts.

*** Dynamic flow requires at least 2-3 grams per day, in divided doses, for a health young adult.

Supplements that kill cancer

We have described the action of vitamin C in detail. Vitamin C illustrates the basic mechanism for killing and controlling cancer cells. Taken alone, it has been shown to be completely effective in a relatively limited range of cancers; however, it potentiates the action of many other nutrients and drugs.

The cytotoxic action of vitamin C requires high doses. At low doses, cancer cells can use the ascorbate as a nutrient, whereas massive doses kill cancer cells selectively, by generating hydrogen peroxide. The optimal balance of nutrients for elimination or control of cancer has not yet been established, so we will outline a possible therapeutic regime, based on current scientific knowledge.

Oral therapy

It is possible to produce and sustain high blood plasma levels using frequent doses of oral vitamin C.¹⁵ Such ascorbate levels will kill some susceptible types of cancer cell, but not all. Evidence suggests that tumours accumulate ascorbate to higher levels than healthy tissues, which could explain the increased bowel tolerance seen in cancer patients. Cancer cells take in ascorbate preferentially, especially when glucose is restricted. This suggests that it may be possible to achieve higher ascorbate levels in tumours than previously considered possible. Orally sustained plasma levels may be high enough to allow ascorbate to be accumulated within tumour tissue, and could be cytotoxic. Use of [liposomal preparations of vitamin C](#) may increase its effectiveness, blurring the distinction between oral and intravenous therapies.

Oral therapy has practical advantages over intravenous therapy. For example, it could be self-administered by a patient attending an outpatient clinic. However, the principal advantage is that it can be sustained, in a dynamic flow.¹⁵ Patients on oral vitamin C therapy can assault the cancer with a continuous redox pressure, over months, or even years. This means the cancer cells do not have periods of respite, during which the more resistant cells can flourish. Laboratory studies of cytotoxicity generally involve short periods of exposure, often of the order of a day. The effects of continuous exposure on the viability of cancer cells have not been established, but could be far greater than intermittent treatment.

With a limited carbohydrate intake, to deny the cancer the nutrients it requires for easy growth, cancer patients on oral redox-active nutrient therapy might enjoy [similar benefits to those described by John Ely](#). Further restriction of calories and nutrients could provide additional benefits. Arthur Robinson suggests that the expected lifespan of a terminally ill cancer patient could increase by at least an order of magnitude. This means that a patient given one year to live could hope for a further ten years, or more.

We suggest the use of natural vitamin C, as L-ascorbate, for oral administration, as it is more available to cells. Liposomal preparations may enhance the effectiveness of oral vitamin C. In addition, R-alpha-lipoic acid increases the cytotoxic effectiveness of ascorbate, by a factor of more than five. Like ascorbate, it is safe for healthy cells. Oral therapies that combine ascorbic and alpha-lipoic acids move the achievable plasma levels towards the cytotoxic range.**** Sustained blood levels of L-ascorbate and R-alpha-lipoic acid will lead to accumulation of high concentrations within tumours. Experimental data suggest an intake of R-alpha-lipoic of at least 10% of the ascorbate intake - 100mg or more for each gram of vitamin C. R-alpha-lipoic acid has low toxicity, although safety data on high doses over extended periods is not available. On current evidence, a combination of vitamin C and alpha-lipoic acid is the least toxic approach to oral cancer therapy.

****A one millimolar concentration of ascorbate is at the beginning of the cytotoxic dose response curve for ascorbic acid.

Alternatively, vitamin K3' or another of the redox-cycling quinones, could be used in place of R-alpha-lipoic acid; these are also synergistic with ascorbate. The combination of vitamins C and K3 is 10 to 50 times more effective against cancer than is either agent alone.⁸⁵⁴ Vitamin K3 may be more effective than alpha-lipoic acid, but is also more toxic and requires careful administration. Once again, evidence indicates that levels sufficient to kill many types of cancer could be generated using oral doses.

In addition, it is possible to vary the administration of these treatments; for example a period with vitamin C and K3 could be followed by a period with ascorbate and alpha-lipoic acid. Periods of vitamin C and vitamin E succinate might also be employed.

Redox synergy

It is clear that vitamin C interacts with several different nutrients that create free radicals selectively within cancer cells. Vitamin K3 and alpha-lipoic acid each increase the cancer killing effectiveness of vitamin C. It is not yet known to what extent use

of multiple agents might further improve the effect. The effects of combined use of alpha-lipoic acid with vitamin K3 are not established. It is reported that alpha-lipoic acid acts as an antioxidant, inhibiting the effects of K3, but this result may apply only to lower concentrations of alpha-lipoic acid, in the absence of ascorbate.

John Ely has suggested the use of [coenzyme Q10](#) with vitamin C. Since coenzyme Q10 and vitamin K3 are chemically similar, both being quinones, they may have a similar pharmacology in large doses. The addition of vitamin E succinate to the therapy may destroy cancer cells more effectively and prevent resistance developing. An analogous approach is used for treatment of tuberculosis (TB), in which a combination of antibiotics is taken over a period of months, to prevent development of drug resistance.

Levels of oral anticancer nutrients need to be increased slowly. There are reports of cancers that are highly sensitive to the actions of vitamin C and redox cycling. Killing such a cancer quickly may produce a life-threatening shock. Having reached a maximal intake, the supplement regime should be continued indefinitely and the progress of the tumour monitored clinically and with medical imaging.

More than one approach

Viewing cancer as microevolution provides multiple approaches to fighting it. The first is to drive the population of cancer cells to extinction by sustained oxidation, generated by selective pressure from vitamin C and related substances.

If the cancer cells develop resistance to direct redox killing, it is possible to utilise an alternative approach of population growth control, by preventing oxidation within the cancer cells. For example, use of the metal binding supplement, IP6, can remove iron from the tumour, slowing growth. Combined with supplemental antioxidants and gene inhibitors, such as curcumin, cancer growth could be slowed by driving the cancer cells into a reducing state, in which cell division is impeded. This antioxidant approach makes cancer cells less oxidising, to prevent cell growth, rather than generating additional free radicals within the cancer cells. It is likely to be less effective with advanced disease.

Oral therapy for terminal cancer

We can see no reason for physicians not to recommend oral redox therapy for all terminal cancer patients, following conventional treatment. The toxicity is low; indeed, there have been reports of improved quality of life in people on ascorbate, in marked contrast to conventional therapies. Furthermore, the treatment might prolong the patient's life and, in some cases, could even cure the cancer. Such possibilities demand urgent investigation. Failure to carry out such these studies could mean that people go on suffering, or die unnecessarily early deaths.

In the case of patients that have apparently been cured by surgery, oral redox therapy could minimise the risk of recurrence, by destroying small pockets of remaining cancer. There is substantial evidence that this approach would be both non-toxic and effective. What we cannot predict is the number of patients who would benefit, since the required studies have not yet been carried out.

The combined therapy of oral redox-active nutrients and a low carbohydrate diet could be made an option for terminal cancer patients. The risk is small, provided the patient is monitored by competent medical staff. Such patients have little to lose, whereas the potential gain is huge. No one knows what proportion of terminal cancer patients would be cured, although studies are clearly essential. Neither do we know the number of "terminal" patients who might survive for years, eventually dying from an unrelated cause. However, the proportion may well be large.

Terminal patients are being written-off, without the option of a therapeutic approach that is cheap, harmless and stands a good chance of being effective. Who among medical decision makers and politicians will take responsibility for what is potentially a reckless waste of human life?

Intravenous therapy

Intravenous infusions of sodium ascorbate, in combination with vitamin K or alpha-lipoic acid, will kill cancer cells. However, intermittent treatments might cause the cancer cells to become resistant. In microevolutionary terms, if the therapy selects tumour cells for resistance to redox-induced apoptosis, then the gain in life expectancy might be limited.

One strategy to avoid such resistance would be to combine intravenous infusions with frequent large oral doses of vitamin C, to maintain high blood levels. Such a regime is needed to establish a dynamic flow of ascorbate through the body,¹⁵ using intakes close to bowel tolerance levels. This therapy might include a synergistic agent, such as alpha-lipoic acid or, perhaps, [motexafin gadolinium](#). The aim would be to keep the cancer under consistent redox pressure.

A second limitation on intravenous therapy is more practical. Such therapy requires monitoring by a physician, together with appropriate nursing care. Any intravenous treatment has associated dangers. The therapy can take several hours per session, which has an associated cost. Although the substances employed may be inexpensive, the costs of preparation, therapy and supervision may be significant. However, they would still be far lower than would those of the corresponding chemotherapeutic drugs.

Benefits of anticancer nutrition

According to the microevolutionary model, cancer occurs in multicelled organisms when cells escape the body's controls and start to behave like their single-celled ancestors. Such changes are triggered by oxidation and damage, which result in erroneous cell division. The environment in which cancer cells develop favours anaerobic cells, which use glucose as their main source of energy. This is in contrast to healthy cells, which tend to use aerobic forms of metabolism and can utilise varied food sources.

Over geological timescales, animals and plants have developed ways to stop their cells reverting to primitive forms. Hence, anticancer substances are common throughout nature. Therapies based on these nutrients take advantage of metabolic differences between cancer cells and healthy cells, to destroy the cancer cells while doing no harm to, or even helping, the healthy cells. Clinical trials are urgently needed to test such non-toxic therapies, before more people die unnecessarily.

Fundamental biological research suggests that cancer is a treatable condition. Right now, people who have been given a terminal prognosis may be able to survive in reasonable health, for a prolonged period. Although the current data is not sufficient to indicate the degree of life extension achievable, many "terminal" cancer patients might die of other causes, long before the cancer kills them. Cancer patients deserve to be offered this opportunity.