VITAMIN C AND CANCER: 2009 UPDATE

By Bill Sardi
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"It is our opinion that the National Cancer Institute and those physicians who treat and study cancer patients have ignored far too long the discovery of the value of large doses of ascorbate (vitamin C) to patients with advanced cancer." – *Linus Pauling, Ewan Cameron,* Proceedings National Academy Science U S A, *1978 December; 75(12): 6252.*

The C in Vitamin C stands for controversy when it comes to cancer. Since Linus Pauling and Ewan Cameron showed that 10 grams of intravenous Vitamin C doubled the survival time of terminal cancer patients, which would rival most chemotherapy cancer drugs today, controversy has surrounded this essential vitamin. [*Proceedings National Academy Science U S A*, 1976 Oct; 73(10):3685-9; 1978 Sep; 75(9):4538-42]

Conventional medicine was fed up with the public clamor over Vitamin C and predictably attempted to set out to discredit it. Mayo Clinic researchers employed ten grams (10,000 mgs) oral, not intravenous, doses of Vitamin C to dispel any idea in the public's mind that Vitamin C could cure cancer. [*New England Journal Medicine*, 1985 Jan 17; 312(3):137-41; *New England Journal Medicine*, 1979 Sep 27; 301(13):687-90] When it did not cure cancer, the door was slammed on Vitamin C. End of story. Or was it?

Overlooked was the work of researchers in Japan. They analyzed terminal cancer patients at two different hospitals. Survival time was 2 to 6 times longer among terminal cancer patients with high-versus-low circulating Vitamin C levels. The quality of remaining life was also improved among Vitamin C-sufficient subjects. [*International Journal Vitamin Nutrition Research Suppl.*, **1982; 23:103-13**]

Never Say Die

Some die-hard advocates of Vitamin-C therapy persisted. Led by Hugh Riordan, M.D., researchers reported that a 70-year old male with kidney cancer (surgically-removed kidney), which later spread to his lungs (confirmed by both x-ray and CT scan), elected to forego chemotherapy and opted instead for 30 grams of intravenous vitamin C (3 times the dose used by Pauling and Cameron). Within six weeks the patient's lungs were free of any abnormalities and 3.5 years later the patient was cancer free. [*Journal Orthomolecular Medicine*, 5: 5-7, 1990]

In 2003, two cases of advanced ovarian cancer (Stage IIIC for both) were reported among patients who underwent surgery and received standard chemotherapy followed by intravenous injections of ascorbic acid at 60 grams dose, twice weekly for several months. Forty (40) months after initial diagnosis these patients presented no signs of disease and had normal tumor marker values. Researchers concluded, given that the 5-year survival is of approximately 30% in the case of advanced ovarian cancers, that these results suggested that Vitamin C may improve the efficacy of chemotherapy. [*Journal American College Nutrition*, 2003; 22:118–23]

Surprising Discovery

Then, in 2004, a rather surprising research study concluded that intravenous Vitamin C could achieve blood concentrations that are selectively toxic to cancer cells. Oral doses cannot do this. The very same National Institutes of Health researchers who mistakenly claimed oral Vitamin C produces "expensive urine" and that there is no value in consuming more than 200 milligrams per day suddenly did an about-face and reported that intravenous Vitamin C can indeed achieve cancer-toxic concentrations in the blood circulation, enough to prompt them to suggest reevaluation of intravenous Vitamin C for cancer therapy. [*Annals Internal Medicine*, 2004 Apr 6; 140(7):533-7] Despite the remarkable implications of this study, the major news media ignored it. Had the work of Linus Pauling and Ewan Cameron been vindicated?

Subsequently, three cases of cancer were reported that responded to intravenous Vitamin C and produced unexpectedly long survival times. [*Canadian Medical Assn Journal*, 2006 Mar 28; 174(7):937-42]

Door Slammed Again

With the question of whether Vitamin C is a cancer cure back on the table, some very skeptical researchers in Canada embarked upon another trial, to reinvestigate whether intravenous Vitamin C can prolong survival times among patients with advanced cancer. Four different doses of intravenous Vitamin C were administered. Vitamin C failed to demonstrate anticancer activity. [*Annals Oncology*, 2008 Nov; 19(11):1969-74] The door was slammed again!

Furthermore, the most recent study concludes that supplementation with oral Vitamin C *"offers no overall benefits in the primary prevention of total cancer incidence or cancer mortality."* [*Journal National Cancer Institute*, 2009 Jan 7; 101(1):14-23] Another recent study also concludes supplemental Vitamin C does not prevent prostate cancer. [*Journal American Medical Association*, 2009 Jan 7; 301(1):52-62] However, only 500 milligrams of Vitamin C was employed in these studies, which would only raise circulating Vitamin-C levels for a couple of hours in humans.

Pulsed, supplemental oral Vitamin C is required throughout the day to achieve optimal blood levels, as exhibited by animals that still synthesize Vitamin C naturally. Humans lost their ability to produce Vitamin C due to a genetic mutation that occurred generations ago. Humans no longer produce a liver enzyme that converts blood sugar to ascorbate (Vitamin C). [*Perspectives Biology Medicine*, 1966 Autumn; 10(1):133-4; *Medical Hypotheses*, 1979 Jun; 5(6):711-21]

In late 2008, cancer researchers at Memorial Sloan-Kettering Cancer Center in New York City reported that Vitamin C given prior to use of chemotherapy drugs reduced the therapeutic activity of the drugs. Vitamin C was demonstrated in animals to rescue cancer cells from death caused by chemotherapy drugs. [*Cancer Res.*, 2008 Oct 1; 68(19):8031-8] Another thumbs-down on Vitamin C for cancer.

All of these negative studies obtain widespread attention in the major news media.

Hope Regained

Sandwiched in between all of these recent negative reports was a study showing greater than 250 mg consumption of daily supplemental Vitamin C drastically reduces the risk for esophageal cancer. [Nutrition Cancer 2008; 60(1): 39–48]

The Sloan-Kettering study cited above is of concern, not because it was a negative study, but because it conveyed flawed logic. How could Vitamin C be deemed negatively when chemotherapy drugs do not significantly prolong survival among cancer patients, regardless of whether patients are supplementing with Vitamin C or not, and many cancer patients succumb to the toxic effects of chemotherapy drugs before they die of their cancers?!

Something Is Amiss

Back in the laboratory, National Institutes of Health researchers employed *"pharmacologic"* doses of Vitamin C in mice, in similar concentrations that have been achieved in humans, and demonstrated significant decrease in growth rates of ovarian, pancreatic, and brain tumors. The researchers concluded that intravenous Vitamin C *"may have benefits in cancers with poor prognosis and limited therapeutic options."* [*Proceedings National Academy Science U S A*, 2008 Aug 12; 105(32):11105-9]

So why does intravenous Vitamin C so convincingly stunt the growth of cancers in animals, but not humans?

Today, cancer researchers know more about the biological mechanisms behind Vitamin C's anti-cancer properties.

Researchers in Korea have demonstrated that high concentrations of Vitamin C transiently induce the formation of hydrogen peroxide, which is toxic to cancer cells. [*Cancer Letters*, 2008 Dec 22] Vitamin C in sufficiently high concentration can release iron from its transport protein, ferritin, in effect *"rusting"* cancer cells to death. [*Biochemical Pharmacology*, 76; 164-165, 2008] At high concentrations, Vitamin C selectively kills cancer cells, and not healthy cells, by functioning to promote oxidation. So high-dose Vitamin C works to thwart cancer by its ability to promote oxidation, rather than its well-known role as an oral antioxidant.

It has long been known that low dietary intake of Vitamin C is associated with cancer. Only recently have researchers demonstrated that low intake levels of dietary or supplemental Vitamin C increases instability of the human genome, resulting in shortening of the end-caps of chromosomes (called telomeres), which can lead to cancer. [*International Journal Cancer*, Oct. 22, 2008 early online]

It is of interest to note that high-dose, oral or injectable Vitamin C given to guinea pigs, which like humans do not synthesize Vitamin C as most other animals do, inhibits tumor growth (65% with injectable Vitamin C, 50% with oral Vitamin C). [*Puerto Rico Health Sciences Journal*, 2005 Jun; 24(2):145-50]

So again, why did intravenous Vitamin C work so well in animals at the National Institutes of Health laboratory but did not work effectively in humans in the Canadian trial? This goes unexplained. At issue is whether patients underwent prior chemotherapy or not.

Furthermore, contrary to what Sloan-Kettering researchers reported, Vitamin C has been shown to enhance the action of various anti-cancer drugs such as 5-Fluorouracil, bleomycin, doxorubicin, cisplatin, paclitaxel, cyclophosphamide, adriamycin, vincristin, and others. [*Biochemical Pharmacology*, 76; 164-165, 2008]

Looking Back

Nearly fifty years ago, William McCormick, a Canadian physician, observed that the generalized changes in connective tissue produced by scurvy (frank Vitamin-C deficiency) are identical to the local changes observed in the

immediate vicinity of invading cancer cells. McCormick was first to postulate that cancer is a collagen (connective tissue) disease, secondary to Vitamin C deficiency. [*Archives Pediatrics*, 1959; 76:166–71]

McCormick's hypothesis is supported by the observation that patients suffering from advanced cancer generally exhibit low concentrations of Vitamin C in blood plasma. [*Cancer*, 1952; 5:678–84] This common deficiency has, in the past, been generally correlated with low dietary intake of Vitamin C among cancer patients. [*Palliative Medicine*, 2005; 19:17–20; *British Journal Cancer*, 1993; 68:1195–8]

New Finding

However, a very recent study reveals that cancer patients exhibit profound Vitamin-C deficiency despite adequate dietary intake and that tumor cells may devour Vitamin C, thus necessitating higher intake levels to maintain adequacy. Compared to healthy subjects, 82% of cancer patients were found to be abjectly deficient and another 12% were marginally deficient. [*Biological Trace Element Research*, 2009 Jan 17 early online]

Dismay

More alarming is the recent report showing that a patient hospitalized for cancer exhibited signs of Vitamin-C deficiency (bleeding gums, hemorrhage, anemia, impaired immunity, swelling) that are generally attributed to cancer, which appeared to be induced by interleukin-2 chemotherapy. [*Clinical Experimental Dermatology*, 2008 Dec 15 early online]

Interleukin-2 is often used to treat kidney cancer, lymphoma and melanoma. Toxicity from interleukin-2 treatment is a major problem and often trials with this chemotherapy agent show improved cancer survival, but no improvement in overall survival due to the side effects of the drug.

Since 1987 it has been known that interleukin-2 therapy induces severe Vitamin-C deficiency, which produces symptoms of fever, chills, racing heart, low blood pressure, vomiting, diarrhea and fluid retention, which in turn may be avoided by provision of supplemental Vitamin C. Mean blood-plasma levels of Vitamin C are reported to drop by 80% or more after the first treatment with interleukin-2 treatment. [*Cancer Research*, 1987 Aug 1; 47(15):4208-12]

Vitamin C may counter the adverse effects of other chemotherapy drugs as well. For example, it has been shown that Vitamin C significantly prolongs the life of laboratory mice and guinea pigs by reduction of toxicity caused by adriamycin, an anti-cancer drug. [*Cancer Research*, 1982 Jan; 42(1):309-16]

This data indicts oncologists for electing to ignore pre-treatment Vitamin-C levels in cancer patients and for prescribing treatment that may induce mortal-but-avoidable side effects secondary to severe therapy-induced Vitamin-C deficiency. The horror of this fact is difficult to fathom. By ignoring Vitamin C, oncologists are delivering inhumane treatment to patients, a gross violation of medical ethics.

Conventional medicine is so averse to vitamin therapy that it ignores Vitamin-C status among cancer patients and even prescribes chemotherapy agents that induce frank Vitamin-C deficiency. There is no reason to leave 8 in 10 cancer patients in a state of utter Vitamin-C deficiency, to suffer hemorrhages, pain, nerve damage, and premature death, regardless of whether there is any evidence for a therapeutic effect of Vitamin C.

In effect, conventional medicine says, since there is no conclusive evidence to show Vitamin C is efficacious in the treatment of cancer, so doctors can continue to ignore Vitamin-C nutrition in their cancer patients to the point of letting them die of the horrible symptoms of scurvy before they succumb to cancer.

For the future, Vitamin C is likely to work far better in synergistic rather than additive fashion, when combined with bioflavonoids/polyphenols found in apple peel or onions (quercetin), grape skin (resveratrol), grape seed (proanthocyanidins), cranberry or citrus rind than Vitamin C by itself. Such a combination may reduce the need for high concentrations of Vitamin C. [*Mutation Research*, 2001 Sep 1; 480-481:85-95; *Anticancer Research*, 1999 Sep-Oct; 19(5B):4177-86; *Biochimie*, 2008 Oct; 90(10):1499-505]

In this regard, there is a growing body of evidence to show that the combination of Vitamin C plus Vitamin K literally cuts cancer cells in two in a guillotine-like action called autoschizis. Possibly the addition of Vitamin K would potentiate the action of Vitamin C to the point where oral doses could be effective in the treatment of cancer. Further study is required. [*Anticancer Research*, 2008 Sep-Oct; 28(5A):2727-32; *Pharmazie*, 2005 Oct; 60(10):765-71; *Ultrastructural Pathology*, 2005 May-Aug; 29(3-4):221-35; *Life Science*, 2004 Jul 9; 75(8):955-67; *European Journal Medicinal Chemistry*, 2003 May; 38(5):451-7; *Anticancer Research*, 2001 Sep-Oct; 21(5):3439-44]

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