The Real Story of Vitamin C and Cancer

Steve Hickey Ph.D.;[1] Hilary Roberts, Ph.D.

Introduction

In the last couple of weeks, vitamin C and cancer has become a hot news topic. For people who have followed this matter, the media's sudden interest comes as something of a surprise: the evidence that vitamin C is a selective anticancer agent has been known for decades. This story is important, as it illustrates how the head-in-the-sand conventional view (that nutritional supplements are useless) can lead to restrictive legislation, reduced health, and limited approaches to the treatment of disease.

The recent news story arose from a study by researchers at the US National Institutes of Health (NIH).[1] The NIH experiment showed that, when injected into mice, vitamin C could slow the growth of tumours. The NIH paper presents its findings as new, ignoring the long history of research into vitamin C and cancer. Far from being novel, many of the findings reported in this paper have been recognized for decades. What is strange, however, is that the media suddenly decided to report a story they had ignored for so long.

A History

One strand of this story begins with the work of an old friend, Dr. Reginald Holman. In 1957, Holman published a paper in Nature about how hydrogen peroxide (the chemical Marilyn Monroe reportedly used on her hair) destroyed or slowed the growth of tumours in mice.[2] Reg Holman met with some hostility from the medical profession, which slowed his research and clinical work over the following half century. Nevertheless, scientists have known that hydrogen peroxide kills cancer cells for over fifty years.

1. FCET, Staffordhire University, Beaconside, Stafford, England, ST16 9DG. s.hickey@staffs.ac.uk

In 1969, when man first walked on the moon, researchers found that vitamin C would selectively kill cancer cells without harming normal cells.[3] That finding meant that vitamin C was like an antibiotic for cancer: potentially a near perfect anticancer drug. Before 1970, it was known that vitamin C was an example of a new class of anticancer substances. However, the medical research establishment largely ignored these scientific results.

In the 1970s, some members of the public and pioneering doctors experimented with high doses of vitamin C to treat cancer. By 1976, double Nobel Prize winner Linus Pauling and Scottish surgeon Ewan Cameron reported clinical trials, showing an unparalleled increase in survival times in terminal cancer patients treated with vitamin C.[4] However, by this time Pauling was considered a quack, having claimed that vitamin C could prevent or cure the common cold, so these apparently amazing findings made little impact.

Cameron and Pauling published a second report in 1978.[5] The Mayo Clinic responded with a study that suggested vitamin C had no effect, which the medical profession readily accepted, perhaps because it confirmed existing prejudices. However, despite the Mayo Clinic study being "considered definitive,"[1] it was highly criticized from the start. In particular, it used relatively low oral doses for short periods, rather than the lifetime combination of high oral and intravenous (IV) doses in the Pauling and Cameron study. The Mayo Clinic refused to provide Pauling with their data so he could check it. When we emailed the Mayo Clinic with a similar request, we received no reply.

If Cameron and Pauling's work, back in the 1970s, had been just a single study,

it would have been interesting and suggestive. Such a large increase in survival time demands a proper scientific follow-up and, indeed, other studies soon backed up the findings. Japanese researchers found similar survival times,[6] apparently confirming Pauling's early results. Subsequently, Dr. Abram Hoffer, working in Canada, provided more evidence that vitamin C could enable cancer patients to live much longer. We have analyzed these results and found them to be statistically valid. They are not explicable by placebo effect or by a simple biased selection of long-lived patients. Moreover, over the last three decades, a large number of clinical and anecdotal patient reports support the claims.

A long time before the NIH's mouse experiment, Pauling also studied the effects of vitamin C on cancer in mice. He worked with Dr. Art Robinson but, unfortunately, the two researchers fell out over their interpretations of the results. Robinson left the Linus Pauling Institute (which he had helped establish) and completed the experiment alone. It was eventually published in 1994.[7] The results were outstanding: mice with cancer that were given high dose vitamin C in the diet, or fed a diet of raw vegetables, lived up to 20 times longer than controls. Translated into human terms, this might mean that a person with one year to live might get an extra 20. Importantly, Robinson and Pauling had been inspired to do this experiment by claims from cancer sufferers in the popular literature.

Doctors Hugh Riordan, Ron Hunninghake, Jim Jackson, Jorge Miranda-Massari, Michael Gonzalez and others in the Center for the Improvement of Human Functioning, Inc., did the core research on vitamin C and cancer. They repeated and extended the early work, which had showed vitamin C would selectively kill cancer cells. They have years of experience of treating cancer patients with high dose

vitamin C. Their work is consistent with results from independent researchers and doctors worldwide.[8]

The authors of this article recently reviewed the literature on vitamin C and cancer, in our book *Cancer: Nutrition and Survival*.[8] We found solid evidence that vitamin C, in high enough doses, acts as a selective anticancer drug. In healthy tissues, vitamin C is an antioxidant, while in cancer it acts as an oxidant generating free radicals and killing the abnormal cells. Furthermore, an understanding of its action provides insight into the cancer development process. Oxidants, such as hydrogen peroxide, are able to make cells grow and divide erroneously. So, as the cells divide, they form a population of varying cells that compete with each other for survival. It was immediately clear that oxidation could explain how cancer starts; following which Darwin's theory of evolution takes over. Given enough time, cells divide and the "fittest" are selected. In this context, the fittest to survive are those cells that grow rapidly to form an invasive cancer. Cancer is not a mysterious disease but is a result of straightforward biological processes.

This microevolutionary model for cancer makes highly specific predictions. One is that high dose vitamin C should prevent cancer and even higher doses should kill cancer cells. The model also predicts that there could be thousands of selective anticancer drugs. Animals, and especially plants, will contain these substances, because they evolved in the presence of cancer and had to develop ways to control it. If such predictions are correct, we should find a multitude of safe anticancer agents in food. Checking against medical databases, we immediately found numerous examples, such as curcumin from turmeric, alpha-lipoic acid, and vitamin D_3. Everywhere we looked, we found substances with the predicted properties. Unfortunately, many

are the very supplements the Alliance for Natural Health (ANH) is trying to protect from being banned!

To conclude our history, the NIH paper was essentially a repeat of previous animal experiments. Despite this, the NIH authors appear not to have referenced many of the scientists who did the original work on vitamin C and hydrogen peroxide in cancer. Instead, they present their work as standing alone, in an informational vacuum: with the exception of the Cameron and Pauling clinical trial, the original scientists' work is not mentioned in the NIH text. Wrongly, a reader might gain the impression that the NIH's work was fundamentally original, rather than repeating the work of others. This might mislead the media into ascribing credit for the work on vitamin C and cancer to the NIH, which would be unfair to the real pioneers of this subject.

Intravenous or Oral?

Dr. Mark Levine of the NIH claims that "When you eat foods containing more than 200 milligrams of vitamin C a day –for example, 2 oranges and a serving of broccoli–your body prevents blood levels of ascorbate from exceeding a narrow range."[9] This statement is demonstrably false (the NIH's own data refutes it) and is an artefact of the way the NIH group interpret their experiments.

In their mouse paper, the NIH used intravenous vitamin C, rather than oral. To be more accurate, the NIH used intravenous ascorbate. Sodium ascorbate is normally used for injection, as vitamin C (ascorbic acid) can cause local inflammation at the injection site. The results they obtained are suggestive of a response, but do not show the same large effects reported by Robinson. Robinson fed his mice dietary vitamin C, in very high doses. Thus, the NIH's suggestion that only intravenous vitamin C is useful as an anticancer agent does not appear to fit

the animal data. Likewise, the idea that only intravenous vitamin C is effective against cancer does not fit the clinical data. Abram Hoffer, for example, used oral doses and obtained essentially the same results as Cameron and Pauling.

The NIH's insistence that the body has "tight controls," which prevent oral vitamin C from functioning as an anticancer agent, is wrong. In our book *Ascorbate: The Science of Vitamin C,* we have shown that the NIH claims for blood "saturation" at a low level (70 μ M/L) are incorrect.[10] The NIH authors never admitted this error, despite a long email correspondence between Hickey and Levine. However, they have changed the wording they use, from "saturated" to "tight controls," and increased the level by about three times (to 200 μM/L). It would appear that they are holding onto an outdated idea about how vitamin C acts in the body. As an alternative, we have proposed a dynamic flow model, in which, at high doses, vitamin C flows through the body, providing antioxidant support, potentially preventing cancer growth and killing cancer cells.[11]

Dynamic Flow

Dr. Mark Levine claims:

"Clinical and pharmacokinetic studies conducted in the past 12 years showed that oral ascorbate levels in plasma and tissue are tightly controlled. In the case series, ascorbate was given orally and intravenously, but in the trials ascorbate was just given orally. It was not realized at the time that only injected ascorbate might deliver the concentrations needed to see an anti-tumor effect."[9]

As we have explained, there is no evidence for such tight control. The suggestion that the legendary scientist, Dr. Linus Pauling, or consultant surgeon, Ewan Cameron, did not know the difference between oral and intravenous administration[12] is bizarre and, again,

demonstrably incorrect.[8] The difference between oral and intravenous vitamin C is, however, more complex than suggested by the NIH. Contrary to their conclusions, it is not clear that intravenous vitamin C necessarily provides an advantage over oral supplements in the treatment of cancer. There is a fair case for suggesting that high dose oral administration could be more effective.

At low intakes, the body prevents vitamin C from being lost through the urine; if this were not the case, we would all be at risk of acute scurvy. The body tries to retain a minimum of about 70 µM/L of vitamin C in blood plasma. This level can be maintained with an intake as low as 200 mg a day. At higher doses, the body can afford to let some vitamin C escape in urine. This saves energy, which the kidneys would otherwise use to keep pumping the vitamin C molecules back into the blood. If dietary vitamin C is in plentiful supply, there is no need for our bodies to retain it all. So, at high doses, vitamin C flows through the body, being taken in from the gut and excreted in the urine. With such high intakes, the body has a reserve that it can call upon in times of need.

A single 5 gram dose of vitamin C can generate blood levels of about 250 µM/L; this is above the NIH paper's claimed maximum of 200 µM/L. Moreover, repeated large doses can sustain these levels. We have achieved vitamin C plasma levels above 400 µM/L, following a single dose of oral liposomal vitamin C.[13] It seems that the claimed "tight control" concept will need revising again soon.

People vary in their responses to vitamin C. In some people, a single 2 gram oral dose of vitamin C may have a laxative effect. Our collaborator, Dr. Robert Cathcart, described this as the bowel tolerance level. Strangely, bowel tolerance has been observed to increase dramatically when a person is ill, say with the flu. A person with a laxative effect at, say, 2 grams, may be able to tolerate 100 times more if they become ill. This increased bowel tolerance also occurs in cancer sufferers. It suggests that at times of stress or illness, the body absorbs extra vitamin C. When promoting intravenous vitamin C, the NIH authors have not considered the possibility of such increased bowel tolerance to oral doses.

To achieve the maximum blood plasma levels possible with oral vitamin C, a typical healthy person may need a total intake of about 20 grams, spread throughout the day (say 3 or 4 grams every four hours). However, cancer patients may require far more. Such massive intakes result in consistently high blood levels, which tumour tissues absorb, and which then generate the hydrogen peroxide that kills the cancer cells.

Other possible mechanisms for how vitamin C kills cancer cells[14] are not covered by the NIH study. The NIH base their work on laboratory studies of mice, in which vitamin C kills cancer cells over the course of, perhaps, a couple of hours. Lower levels of vitamin C may simply take longer to kill the cells, which is a standard dose response relationship. Sustained oral doses can increase plasma vitamin C consistently, over periods measured in months or years: this may, in the end, be more effective that the short, sharp shock of intravenous therapy. Sustained levels also reduce the likelihood of tumours developing resistance to the therapy (analogous to bacterial resistance to antibiotics.)

Redox synergy

When combined with alpha-lipoic acid, selenium, vitamin K3, or a range of other supplements, vitamin C is a far more powerful anticancer agent than when used alone. Experimental data from Riordan and others shows that the cancer destroying effect of such combinations is much higher. We have described some of

these combinations in a recent book "*The Cancer Breakthrough*".[15] Strong scientific reasons suggest that such combinations, given orally, could provide cancer sufferers with a large increase in lifespan and increased quality of life.

Just as your doctor advises you to take a whole course of antibiotics continuously, until all infection is gone, vitamin C based redox therapy needs to be continuous. Like bacterial infections, cancers can rapidly become resistant to intermittent treatments. Typically, intravenous ascorbate is given at intervals, whereas oral ascorbate can maintain blood levels continuously and indefinitely. This is a valid medical reason to prefer an oral regime. Also, patients prefer the oral route, as they have greater control, lower cost, and are more involved in their treatment.

People often ask us what we would do, if we developed the disease. In the event that one of us developed a malignancy, we would opt for a vitamin C based redox therapy as our primary approach to treatment. This would be based on oral intakes: we would consider intravenous ascorbate only as an adjunct. We might use liposomal vitamin C to sustain blood levels at 400-500 µ M/L, together with alpha-lipoic acid, selenium, and other synergistic nutrients.[15] While we realize malignant cancer would place us at high risk of death, we would expect to live a greatly extended life. While the assessment of increased longevity could be inaccurate (the data is not definitive), the risks are small and the potential benefits substantial.

Conclusions

Mark Levine claims that the "NIH's unique translational environment, where researchers can pursue intellectual high-risk, out-of-the-box thinking with high potential payoff, enabled us to pursue this work."[9]

However, the recent NIH study, while interesting, adds little to the studies it replicates. More interesting is the lack of historical perspective, which may detract from the people, such as Hugh Riordan, Abram Hoffer, or Linus Pauling, who deserve the credit for carrying out original research, despite conventional medicine actively suppressing their work. The ground breaking work of doctors such as those in the British Society for Ecological Medicine, who have risked their careers to provide vitamin C based treatments for cancer and other conditions should be recognized. These pioneering doctors are often well aware of the scientific evidence and should not be described as "complementary" or "alternative". Perhaps, one day, the media will realize the true story of vitamin C and cancer, and patients will have the opportunity to benefit.

The Alliance for Natural Health is defending our right to supplements. Over the last century, we have benefited from a large increase in life expectancy and freedom from many diseases. Much of that benefit has arisen directly from nutrition.[16] We need access to supplements, which provide the possibility of disease prevention without significant risk. If this basic right is removed by Codex Alimentarius, or similar legislation—for example, the draconian regulatory measures the natural health sector is facing in Europe—even pioneering doctors will find it difficult to progress the nutritional treatment of disease. The health of most of us will suffer. We will get more illnesses, more often, and options for medical treatment of major killers, such as cancer, heart disease, and stroke, will decline.

References

1. Chen Q, Espey MG, Sun AY, et al: Pharmacologic doses of ascorbate act as a prooxidant and decrease growth of aggressive tumor xenografts in mice, *PNAS,* 2008; 105(32): 11105–11109.
2. Holman RA: A method of destroying a malignant rat tumour in vivo, *Nature,* 1957; 179(4568):1033.

3. Benade L, Howard T, Burk D: Synergistic killing of Ehrlich ascites carcinoma cells by ascorbate and 3-amino-1, 2, 4, -triazole, *Oncology,* 1969; 23: 33–43.
4. Cameron E, Pauling L: Supplemental ascorbate in the supportive treatment of cancer: Prolongation of survival times in terminal human cancer. *Proc Natl Acad Sci USA,* 1976; 73: 3685–3689.
5. Cameron E, Pauling L: Supplemental ascorbate in the supportive treatment of cancer: Reevaluation of prolongation of survival times in terminal human cancer, *Proc Natl Acad Sci USA,* 1978; 75: 4538–4542.
6. Murata A, Morishige F, Yamaguchi H: Prolongation of survival times of terminal cancer patients by administration of large doses of ascorbate, *Int J Vit Nutr Res, Suppl,* 1982; 23: 101-113.
7. Robinson AR, Hunsberger A, Westall FC: Suppression of squamous cell carcinoma in hairless mice by dietary nutrient variation, *Mech Aging Devel,* 1994; 76: 201-214.
8. Hickey S, Roberts H: *Cancer: Nutrition and Survival,* 2005; Lulu Press.
9. NIH News: *Vitamin C Injections Slow Tumor Growth in Mice,* Embargoed for Release, Monday, August 4, 5:00 p.m. EDT. 2008.
10. Hickey S, Roberts H: *Ascorbate: the Science of Vitamin C,* 2004, Lulu Press.
11. Hickey S, Roberts H, Cathcart RF: Dynamic flow, *J Orthomol Med,* 2005; 20(4), 237-244.
12. Padayatty SJ, Levine M: Reevaluation of ascorbate in cancer treatment: Emerging evidence, open minds and serendipity, *J Am Coll Nutr,* 2000; 19(4): 423-425.
13. Hickey S, Roberts H, Miller NJ: (2008) Pharmacokinetics of oral ascorbate liposomes, *JNEM,* 2008, (in press).
14. Toohey JI: Dehydroascorbic acid as an anti-cancer agent, *Canc Lett,* 2008; 263: 164–169.
15. Hickey S, Roberts HJ: *The Cancer Breakthrough,* 2007, Lulu press.
16. Wootton D: *Bad Medicine,* 2007, Oxford University Press.