Vitamin C Therapy

1. The RDA for Vit C is 120 mg for a nursing female. Mankind does not make Vit C so let us look at a 150 lb animal that does make Vit C. In an unstressed state it will make between 12,000 and 14,000 mg of Vit C. Doing simple math, the RDA for a nursing female is 1/100 of what this animal would have manufactured. 1%, 1%!!! – this is why I call RDA “Ridiculously Deficient Amount”.

2. Presently 99% of Vit C comes from China. Enough said. In the clinic we used to do dip stick UA’s to show patients how little Vit C was in their body and then have them take Vit C. The next time they were in the office the simple dip stick would show an abundance of Vit C in their urine. Sounds great until it was learned that this was genetically modified Vit C that the body could not effectively use it and so was dumping it into the urine!

3. We now are advocating a non-corn, non-beet (both of these are genetically modified) source of Vit C. For our cancer IV’s we definitely use this source of Vit C for its biological activity.

4. The problem with corn is that it is the #2 allergen in the USA. This potentially causes down regulation (suppression) or up regulation (auto-immune like) of the immune system. Vit C derived from corn can also still have corn residue from being genetically modified to produce its own pesticides and herbicides. Genetically modified corn fed to dairy cows in Germany killed major portions of the herd. Now it is illegal in Germany and Japan to produce products from genetically modified corn.

5. Sailors got scurvy (a Vit C deficiency disease) and their teeth fell out and would bruise with a slight blow to the skin. This was because Vit C is very involved with connective tissue formation. The difference between carcinoma in situ and a metastasis is that the basement membrane has been breached in a metastasis and the cancer is now spreading outwards. It is helpful for Vit C to establish a strong connective tissue basement membrane to potentially help stop future metastasis.

6. Vit C normally acts as an anti-oxidant when used in lower doses. In pharmacologic IV doses of 50 to 200 grams (50,000 to 200,000 mg), Vit C acts as an oxidant. These high doses release hydrogen peroxide that is detrimental to cancer cells. Normal cells have the enzyme catalase to detoxify the hydrogen peroxide and the other reactive oxygen species (ROS). Cancer cells are grossly deficient in catalase and so are not protected in a high Vit C concentration. Normal cells can easily tolerate the same dose and so this is a very effective means to target cancer cells while leaving normal cells intact.
Further Explanation of Vit C Action

Taken from the following website:

“In another study, researchers investigated the effects of dehydroascorbate on the growth of solid tumors (Krebs 2 sarcoma and Ehrlich carcinoma). Control mice with Ehrlich carcinoma had an average tumor size of more than 2 cm², whereas the subject mice, treated with injections of dehydroascorbic acid (2 mg per day about 80 mg/kg), developed no obvious tumors. In the control group, the Krebs sarcoma tumors were on average larger than 1.6 cm², yet of those in the dehydroascorbate treated group, only two of 25 mice developed detectable (small) tumors.14,15 Animal studies have shown dehydroascorbate to be an effective anticancer agent, at doses lower than those for vitamin C.16 These results were considered so unusual by an establishment accustomed to the failure of standard chemotherapy, that they were considered suspect and ignored. However, continuing research into ascorbate and dehydroascorbate as anticancer agents confirms their potential.

John Toohey has recently suggested a mechanism of action for the inhibition of cancer cells by dehydroascorbate. Toohey proposes that cancer cells synthesize homocysteine thiolactone, which reacts with dehydroascorbate to produce the toxic mercaptopropionaldehyde. Cancer cells have an increased demand for methyl groups, which leads to homocysteine formation. This methylation is combined with a high rate of protein synthesis necessary for growth. Both these processes lead to homocysteine thiolactone and a susceptibility to dehydroascorbate toxicity.

Conventional chemotherapy has had some success in Hodgkin’s disease, acute lymphocytic leukemia, testicular cancer, choriocarcinoma, retinoblastoma, and Wilm’s tumor. However, these rare forms account for less than 5% of cancers in the United States. In the majority of cancers, there is little evidence that chemotherapy extends life substantially.24 The contribution of chemotherapy to survival is approximately a 2% increase (treated versus untreated patients).25"