

Vitamin E Succinate, Part 2

THE PREFERRED FORM OF VITAMIN E TO COMBAT BREAST, PROSTATE AND OTHER CANCERS

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It is well-documented that women whose breast cells overexpress the ErbB2 receptor (a member of the epithelial growth-factor receptor family) on their cell surface are at higher risk for breast cancer development. Additionally, their overexpression is associated with the estrogen-receptor-negative form of the disease, which is known to be the most clinically aggressive form of this disease.^{8,9} Studies by Wang and fellow researchers demonstrated that vitamin E succinate was able to induce programmed cell death (apoptosis) in human breast cancer cells that exhibited high levels of ErbB2 expression.

A study by Yu, et al., showed that vitamin E succinate also could induce apoptosis in human breast cancer cells that are Fas-resistant.¹⁰ Fas is an immunology protein that initiates programmed cell death. Apoptosis is important in cell-mediated immune response, autoimmune tolerance and cancer control. Fas is a surface receptor that is expressed throughout the body. The Fas ligand is expressed mostly on activated T-cells, natural killer cells and microglia, but is sometimes found on other cells of the immune system.

When a cell expressing Fas encounters another cell expressing the Fas ligand, the receptor is stimulated in such a way as to transmit a message to secondary protein messengers within the cytoplasm (signal transduction) of the cell. In turn, this activates a series of events that lead to cell death (apoptosis). Certain breast cancer cells are resistant to the apoptosis-inducing effects of Fas, such as MCF-7, MDA-MB-231 and MDA-MB-435 human breast cancer cells.¹¹

The study by Yu, et al., showed that vitamin E succinate was able to convert Fas-resistant human breast cancer cells to Fas-sensitive phenotype, allowing apoptosis to proceed. These findings are important, as they suggest another mechanism through which VES may prevent breast cancer development and/or inhibit the further growth and proliferation of existing breast cancer cells.

Other Mechanisms Through Which Vitamin E Succinate May Prevent Cancer

Reports from Wu, et al., indicate that vitamin E succinate also induces apoptosis of cancer cells by first stimulating production of transforming growth factor-B, which, in turn, increases kinase activity of another protein messenger known as c-Jun N-terminal kinase (JNK), followed by phosphorylation of c-Jun. Finally, activated c-Jun triggers apoptosis. Wu, et al., have shown this apoptosis pathway to exist in human gastric cancer cells, but it might occur in many other cancer cells as well.³ Other researchers have confirmed that vitamin E succinate induces apoptosis through stimulation of the c-Jun messenger system.^{12,13} In addition, Wu, et al., report that vitamin E succinate also induces apoptosis of certain cancer cells by enhancing activity of the Fas-apoptosis system (sometimes called the Fas-death receptor, although other death receptors are also present on the cell surface).³

Summary

In regard to vitamin E supplementation, the current evidence suggests health practitioners should strongly consider recommending supplements containing vitamin E succinate (alpha-tocopherol succinate or alpha-tocopheryl succinate) as part of a high-potency multiple vitamin/mineral supplement or as a stand-alone supplement as part of a comprehensive program to reduce cancer risk. In this regard, the evidence suggests that vitamin E succinate is superior to other available forms of vitamin E and related tocotrienols. Many experts suggest a daily dose of 400 IU per day of vitamin E succinate for general health promotion and disease prevention. As an adjunct to cancer treatment, some practitioners use a total daily dose of 800-1,600 IU (Lockwood, et al.,¹⁶ recommend up to 2,500 IU of vitamin E) in conjunction with high doses of other antioxidants and natural cancer-fighting herbs and phytonutrients. Preliminary human trials using these nutritional supplements have shown impressive results in reducing cancer progression, preventing metastasis and reducing side effects of chemotherapy and radiation.¹⁴

In the review presented by Prasad and fellow researchers, the authors state, "Since it has been demonstrated that vitamin E succinate (alpha-tocopheryl succinate) in combination with dietary micronutrients (retinoic acid, vitamin C and carotenoids) is more effective in reducing the proliferation of tumor cells in culture than the individual agents and in enhancing the effect of some chemotherapeutic agents on these cells, we have recommended the use of alpha-tocopheryl succinate in combination with dietary micronutrients as an adjunct to standard therapy in the treatment of cancer." These authors have provided an excellent review of the anticancer properties of vitamin E succinate for health practitioners interested in further reading on this subject.¹⁵

Abundant evidence indicates that vitamin E succinate possesses unique biological properties that enable it to inhibit or slow the replication of human cancer cells and/or induce apoptosis of many types of cancer cells, most notably prostate and breast cancer. In this regard, vitamin E succinate has been shown to be superior to other forms of vitamin E. As cancer accounts for approximately 20 percent of all deaths in North America, this information should be of importance to health practitioners who routinely recommend multiple vitamins and/or vitamin E supplements to their patients for the purpose of reducing risk of degenerative disease and/or who advise patients on evidence-based adjunctive nutritional therapies that might be helpful to prevent recurrences or metastasis of cancer in patients who already have been affected by this disease.

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