USANA Technical Bulletin

Broccoli Concentrate

Technical Background

- Cruciferous vegetables (such as cabbage and broccoli) contain indole-3-carbinol (I3C) and isothiocyanates.
- Many studies have shown I3C to be effective in helping prevent various types of cancer,[1] with special attention being given to its ability to potentially reduce the risk and/or severity of prostate cancer.[2] I3C has been shown to be effective at preventing breast and uterine cancer in rodent models by aiding in the proper metabolism of estrogens. [3, 4] One study found that I3C helps women maintain a favorable ratio of estrogens, a quality that may result in less cancer.[5] I3C also acts as an antioxidant.[6] And a recent study has shown that I3C exerts a favorable effect on platelet aggregation and adhesion.[7]
- A wide variety of isothiocyanates have been shown to help prevent cancers of various tissues,[8] including the lung, mammary gland, esophagus, liver, small intestine, colon, and bladder.[9] Recent studies have found that certain isothiocyanates may also have antibacterial and antimicrobial properties.[10]

Sources and Recommended Intake

- There is no Recommended Dietary Allowance (RDA) for cruciferous extracts.
- Dietary sources of I3C and isothiocyanates include broccoli, cabbage, cauliflower, kale, turnip greens, mustard greens, watercress, and Brussels sprouts.

Abstracts

Sarkar FH, Li Y. Indole-3-carbinol and prostate cancer. J Nutr. 2004 Dec;134(12 Suppl):3493S-3498S. Epidemiological and dietary studies have revealed an association between high dietary intake of cruciferous vegetables and decreased prostate cancer risk. Our studies have shown that indole-3-carbinol (I3C), a common phytochemical in cruciferous vegetables, and its in vivo dimeric product 3,3'-diindolylmethane (DIM) upregulate the expression of phase I and phase II enzymes, suggesting increased capacity for detoxification and inhibition of carcinogens. Studies from our laboratory and others have found that I3C can induce G1 cell-cycle arrest and apoptosis in prostate cancer cells. In addition, we found, by microarray gene expression profiling, that I3C and DIM regulate many genes that are important for the control of cell cycle, cell proliferation, signal transduction, and other cellular processes, suggesting the pleiotropic effects of I3C and DIM on prostate cancer cells. We recently found that I3C functions as an inhibitor of Akt and nuclear factor kappaB (NF-kappaB), which play important roles in cell survival and which are believed to be potential targets in cancer therapy. Studies have already shown that the inactivation of Akt and NF-kappaB is responsible for chemosensitization of chemoresistant cancer cells. Because there is no effective treatment strategy for hormone-dependent and, most importantly, hormone-independent and metastatic prostate cancer, our strategies to sensitize prostate cancer cells to a chemotherapeutic agent by I3C and DIM is a novel breakthrough that could be used for devising novel therapies for prostate cancer. In conclusion, the results from our laboratory and from others provide ample evidence for the benefit of I3C and DIM for the prevention and the treatment of prostate cancer.

References

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