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Alpha Lipoic Acid

Technical Background

- Alpha lipoic acid (also known as thioctic acid) is a vitamin-like antioxidant that is easily absorbed through the gut and transported across cell membranes, offering protection against a wide variety of free radicals both inside and outside the cell.
- Once in the body’s tissues, alpha lipoic acid can be converted to dihydrolipoic acid (DHLA), an equally potent free radical scavenger.
- Alpha lipoic acid and DHLA provide additional benefits beyond their direct antioxidant activities. Both act to regenerate (and thus prolong the activity of) other antioxidant molecules, including vitamin C, glutathione, CoQ10, and, indirectly, vitamin E. Both can act as metal chelators, binding up excess copper, iron, mercury and cadmium, thus limiting the negative impacts of these heavy metals on the body. Finally, there is evidence that alpha lipoic acid and DHLA have effects on both regulatory proteins and genes involved in normal cell growth and metabolism.
- Studies have shown that alpha lipoic acid has potential therapeutic uses in preventing and/or treating many conditions, including colon cancer, diabetes, atherosclerosis, heavy metal poisoning, neurodegenerative diseases, and HIV infection. It is also indicated as a possible therapy for cataracts and glaucoma. One study found that it may increase insulin sensitivity, making it a good candidate for diabetes and cardiovascular disease prevention. A recent study found that alpha lipoic acid may even play a role in treating multiple sclerosis.
- New research indicates that alpha-lipoic acid, along with vitamin C, improves endothelial dysfunction by decreasing oxidative stress. This may be important in the prevention of atherosclerosis.

Sources and Recommended Intake

- Alpha lipoic acid is produced naturally by most organisms, including humans. It is present in most foods, with especially high levels being found in potatoes.
- No Recommended Daily Intake has been set, nor has the issue been adequately studied. Some researchers believe that the amount needed for therapeutic antioxidant activity exceeds that produced by our bodies and consumed in a normal diet. As such, alpha lipoic acid is a strong candidate for supplementation. Maintenance doses of 10-25 mg per day have been suggested. Therapeutic doses of up to several hundred milligrams per day have been used.

Abstracts


The
antioxidant alpha-lipoic acid (ALA) has been shown to affect a variety of biological processes associated with oxidative stress including cancer. We determined in HT-29 human colon cancer cells whether ALA is able to affect apoptosis, as an important parameter disregulated in tumour development. Exposure of cells to ALA or its reduced form dihydrolipoic acid (DHLA) for 24 h dose dependently increased caspase-3-like activity and was associated with DNA-fragmentation. DHLA but not ALA was able to scavenge cytosolic O(2) (-) in HT-29 cells whereas both compounds increased O(2) (- .)-generation inside mitochondria. Increased mitochondrial O(2) (- .)-production was preceded by an increased influx of lactate or pyruvate into mitochondria and resulted in the down-regulation of the anti-apoptotic protein bcl-X(L). Mitochondrial O(2) (- .)-generation and apoptosis induced by ALA and DHLA could be prevented by the O(2) (- .)-scavenger benzoquinone. Moreover, when the lactate/pyruvate transporter was inhibited by 5-nitro-2-(3-phenylpropylamino) benzoate, ALA- and DHLA-induced mitochondrial ROS-production and apoptosis were blocked. In contrast to HT-29 cells, no apoptosis was observed in non-transformed human colonocytes in response to ALA or DHLA addition. In conclusion, our study provides evidence that ALA and DHLA can effectively induce apoptosis in human colon cancer cells by a prooxidant mechanism that is initiated by an increased uptake of oxidizable substrates into mitochondria.

References

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