

IMMEDIATE RELEASE

ALPHA LIPOIC ACID



Promotes antioxidant defense & cellular health

WHAT IS IT?

Alpha Lipoic Acid (ALA) is an immediate-release dietary supplement. The softgel rapidly releases ALA to promote optimal absorption, especially when taken as directed on an empty stomach.

HOW DOES IT WORK?

ALA plays a key role in cellular energy production, promotes glucose uptake by cells, and exerts powerful antioxidant activity to support whole body health.

ALA is considered by some to be the body's universal antioxidant. Once absorbed, ALA can be converted to dihydrolipoic acid (DHLA). Together ALA and DHLA work in cell membranes and other fat-based areas of the body as well as in the blood and other water-based areas. ALA and DHLA can also regenerate other antioxidants such as vitamin C, vitamin E and glutathione, making them available to quench more harmful free radicals.¹

WHO CAN BENEFIT?

For adults who need powerful antioxidant protection, optimal cellular energy production, and healthy glucose metabolism.

PRODUCT AVAILABILITY

Bottle Size(s):
150 softgels

PRACTITIONER DISTRIBUTION

■ WholeScript™ (www.wholescript.com)



Supplement Facts

Serving Size 1 Softgel

Amount Per Softgel	% DV
Alpha Lipoic Acid	300 mg *
* Daily Value (DV) not established.	

Other Ingredients: Soybean Oil, gelatin, vegetable glycerin. Contains <2% of: Sunflower lecithin, ascorbyl palmitate, mixed tocopherols, natural caramel color, and silica.

Suggested Use: Take one (1) or two (2) softgels daily, preferably on a empty stomach, or as directed by your healthcare practitioner.

RESEARCH HIGHLIGHTS

Helps combat diabetic neuropathy

Numerous human studies report the successful use of ALA to combat oxidative stress and prevent cellular damage in people with diabetes. An oral dose of 600 mg/day for up to 5 weeks is reported to offer therapeutic effects for relief of symptoms and signs of diabetic neuropathy and is well tolerated.²

Helps combat oxidative stress in healthy people

ALA (600 mg/day for 8 weeks) has been shown to exert antioxidant effects and help reduce LDL oxidative susceptibility (a pro-atherogenic marker) in healthy adults.³

Promotes cellular uptake of glucose

ALA is reported to activate peripheral AMPK, a protein kinase involved in the regulation of muscle glucose uptake by exercise/muscle contraction. Through its action on AMPK, ALA is thought to activate cell signaling that promotes glucose uptake.¹

Supports metal detoxification

Both ALA and DHLA chelate redox-active metals in vitro and in vivo. The oxidized and reduced forms bind a number of metal ions, but with different properties depending on the metal chelated. In vitro studies show that ALA preferentially binds to copper, zinc and lead, but cannot chelate iron. DHLA forms complexes with copper, zinc, lead, mercury and iron.¹

Helps maintain optimal cellular metabolism & energy production

The metabolic role of ALA has been known for decades. ALA serves as a cofactor of several key mitochondrial enzyme complexes, including pyruvate dehydrogenase and alpha-ketoglutarate dehydrogenase. These enzymes play an essential role in generating energy from glucose.¹

Helps regenerate key antioxidants

Both ALA and DHLA are capable of scavenging a variety of reactive oxygen species (see Table). Both forms may scavenge hydroxyl radicals and hypochlorous acid, while ALA also terminates singlet oxygen. Neither species is active against hydrogen peroxide. Both, especially DHLA, can prevent protein carbonyl formation by scavenging hypochlorite. Furthermore, DHLA appears to regenerate other endogenous antioxidants (e.g., vitamins C and E) and has the property of neutralizing free radicals without itself becoming one in the process.¹

ALA/DHLA: Free Radical Scavenging Ability		
Oxidant	ALA	DHLA
Peroxynitrite	Yes	Yes
Hydroxyl radical	Yes	Yes
Peroxyl radical	Yes	Yes
Hypochlorous acid	Yes	Yes
Singlet oxygen	Yes	No
Nitric oxide	No	Yes
Superoxide	No	Yes
Hydrogen peroxide	No	No

1. Shay KP, et al. *Biochim Biophys Acta*. 2009;1790(10):1149-60.

2. McIlduff CE, et al. *Ther Clin Risk Manag*. 2011;7:377-85.

3. Marangon K, et al. *Free Radic Biol Med*. 1999;27:1114-1121.