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IS ALPHA LIPOIC ACID USEFUL IN OBESITY TREATMENT? A NARRATIVE REVIEW OF CLINICAL EVIDENCE

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Abstract

Alpha-lipoic acid (ALA), also known as thioctic acid, is an organic compound naturally produced in our body and is also found in foods of plant and animal origin. Despite being relatively abundant in a variety of foods, its dietary intake is relatively small. As we age, the amount of alpha-lipoic acid synthesized in the body drops. Alpha-lipoic acid is widely available as a dietary supplement and is used by various population groups; nowadays especially young recreational athletes. While healthy individuals should be cautious of the potential intoxication and side-effects, no adverse effects were reported in people with health issues including metabolic syndrome, dyslipidemia, diabetes and insulin resistance, and polycystic ovary syndrome. Alpha-lipoic acid poses a high antioxidant potential, and its positive effects on fat and glucose metabolism and anti-inflammatory properties are well documented. Yet, clinical trials assessing the effect of alpha-lipoic acidon weight control are rather sparse. Given the extent of obesity problem globally, finding strategies which could boost weight loss in combination with energy restricted diets and increased physical activity are much needed. Pathophysiology of obesity is complex and far more than just an excessive weight, but simplified, it involves a complexity of metabolic, hormonal, inflammatory and pro-oxidative alterations which manifest as a variety of health issues. Current evidence suggests that alpha-lipoic acid alters carbohydrate and fat metabolism on the cellular level, affects insulin secretion in the pancreas, acts as antioxidant and prevent damage caused by oxidative stress and has anti-inflammatory activities. We summarize available clinical evidence on the lesser-known anti-obesity properties of alpha-lipoic acid.

Keywords: *alpha-lipoic, acid, obesity, metabolic abnormalities, clinical trials.*

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INTRODUCTION

Alpha-lipoic acid (ALA), also known as thiotic acid, is a sulphate-based organic compound that belongs to the group of antioxidant [*Tucci C et al., 2018*]. Since it is an organosulfur and a biological antioxidant, it creates covalent bonds with proteins and plays a key role in the Crebs cycle. It acts as cofactor in enzyme complexes. It is found in mitochondria where it is used as a cofactor for pyruvate dehydrogenase (PHD) and α-ketoglutarate dehydrogenase complexes, and is essential for mitochondrial metabolism [*Tucci C et al., 2018*; *Salehi B et al, 2019*]. ALA is synthesized *de novo* from mitochondrial synthesis of type II fatty acids, S-adenosylmethionine and iron-sulphur cluster. It is required for catalysis by multiple mitochondrial 2-ketoacid dehydrogenase complexes including PHD and ketoacid dehydrogenase. Many of these dehydrogenases are regulated by reactive oxygen species, mediated through the disulphide bond of the prosthetic lipoid part. For these reasons, ALA is necessary for cell growth, mitochondrial activity and coordination of metabolism [*Solmonson A et al., 2018*].

It is naturally synthesized in the human body, but it is also synthesized in plants and animals. A health organism is able to synthesize a sufficient amount of ALA, but its amount decreases considerably with age, which leads to endothelial dysfunction and should be taken exogenously, either through food or dietary supplements [*Tripathi AK et al., 2023*].

ALA exists as S and as R enantiomer (**Figure 1**), and the commercial form of ALA is a racemic mixture of these two forms [*Ghibu S. et al., 2009*]. The molecular weight of ALA is 206.318 *g/mol*. It is a stiff, pale yellow crystalline powder. The R enantiomer is one of the cofactors of mitochondrial enzymes and plays a central role in energy metabolism [*Jocelyn PC, 1967*].

ALA has a short half-life (30 minutes to 1 hour) and limited biological bioavailability (about 30%) caused by high hepatic extraction, reduced solubility and instability in the stomach [*Jocelyn PC, 1967, Salehi B et al., 2019; Teichert J et al., 2003*]. However, the bioavailability of ALA increases greatly with various innovative formulations. The R enantiomer shows better pharmacokinetic characteristics in comparison to the S enantiomer, and most importantly, has better bioavailability [*Salehi B et al., 2019*].

Age affects the bioavailability of ALA, but also its concentration in plasma. One study showed that the bioavailability and plasma concentration of ALA were significantly higher in people of 75 years of age and older, as compared to young adults aged between 18 and 45 years. No differences in bioavailability were found in men and women [*Keith DJ et al., 2012*].

OBESITY AND ITS COMORBIDITIES

Obesity is a complex chronic disease characterized by excessive fat deposits that negatively affect health and quality of life [*WHO, 2021*]. Health consequences of obesity and its pandemic rates

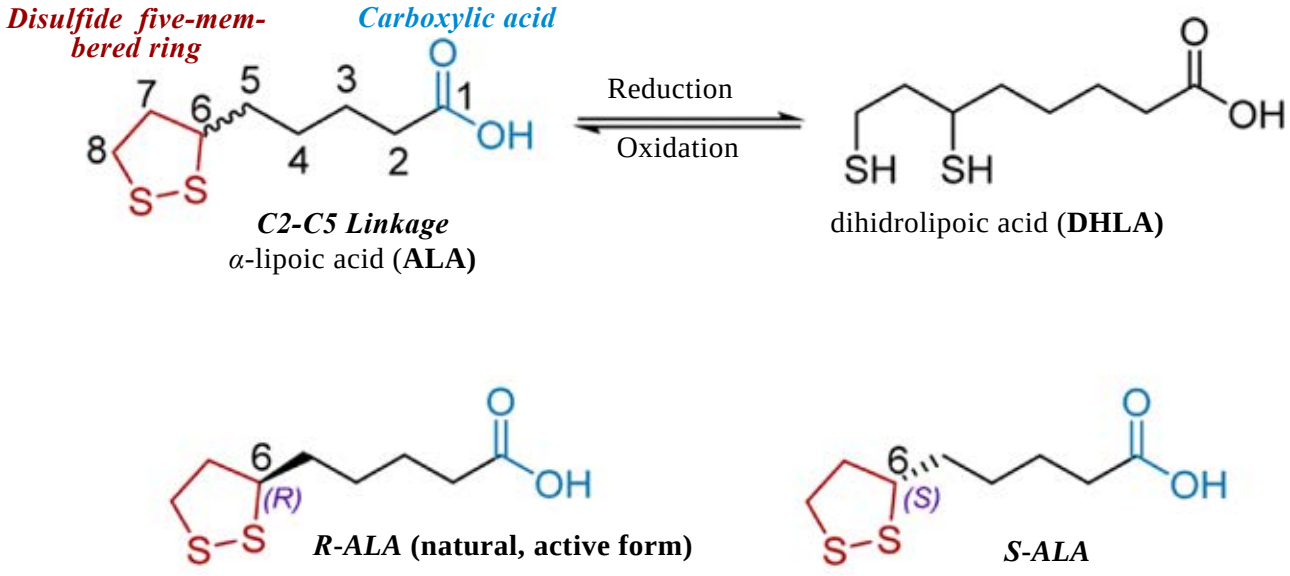

FIGURE 1. *Chemical structure of alpha-lipoic acid and its two enantiomers (prepared by the authors)*

alarmed the whole world, but despite a handful of actions, the numbers are relentless. Since year 1990, globally, adult obesity rates more than doubled, reaching 890 million in year 2022 [*WHO, 2024*]. Global obesity rates in adults vary from staggering 72.95% in American Samoa to just 2.04% in Vietnam [*NCD-RisC, 2024*]. Yet, in women, the rates are even higher [*NCD-RisC, 2024*]. Nowadays, obesity is considered a major comorbidity in gynaecology and obstetrics. Obesity and overweight put women at elevated risk of various reproductive issues, and comorbidities such as diabetes, cardiovascular disease, kidney disease and obesity-related cancers [*Flegal KM et al., 2007*]. Negative impact is visible during pregnancy too [*Simko M et al., 2019*]; only in the United States nearly 25% of women are obese at time when the pregnancy is confirmed [*Ogunwole SM et al., 2021*]. Across Europe, maternal obesity prevalence varies from 7% to 25% [*Devlieger R et al., 2016*].

Dysfunctional adipose tissue is a major factor linking obesity and other secondary chronic diseases or carcinogenesis as a result of insulin resistance, chronic inflammation and excessive secretion of adipokines [*Kruijsdijk RCM et al., 2009*]. Increased release of adipokines, free fatty acids, and inflammatory mediators cause adipocyte hypertrophy and hyperplasia [*de Ferranti S, Mozaffarian D, 2008*]. Consequently, stress on the level of endoplasmic reticulum and mitochondria induce a cascade of negative consequences in the liver, pancreatic β-cells, skeletal muscle, and heart and vascular system [*de Ferranti S, Mozaffarian D, 2008*]. As a result, metabolic syndrome, a complex entity including hypertension, dyslipidemia and insulin resistance is found in many obese individuals. In women, polycystic ovary syndrome (PCOS), which shares pathophysiology with the aforemtioned metabolic disorders, is the most common obesity comorbidity affecting reproductive health and pregnancy outcomes. Metabolic syndrome affects about 25% of world's population [*Jamali Z et al., 2024*], and PCOS prevalence is estimated between 5% and 18% globally, but half of women are either not aware of the condition or have a delayed diagnosis [*Lancet, 2022*].

Understanding the mechanism of weight loss and weight regulation is crucial for discovering effective interventional therapies for the treatment of obesity and related disorders. Researchers are increasingly focused on finding new dietary supplements that could help control and prevent this disorder [*Escote X et al., 2018*].

Anti-obesity activity of alpha-lipoic acid

Anti-obesity activity of ALA can be attributed to its fat and carbohydrate metabolism and antioxidant potential.

Firstly, ALA reduce biosynthesis of cholesterol, β oxidation of fatty acids and vascular stiffness. ALA treats central obesity by increasing adiponectin levels and mitochondrial biogenesis and can reduce food intake mainly by stimulating SIRT1 [*Najafi N et al., 2020*]. Also, ALA prevents apoptosis and degeneration of dorsal root ganglion neurons mediated by regulation of cleavage protein 2 and caspase-3, inducing the production of ATP [*Galeshkalami NS et al., 2019*].

Body weight and fat mass loss are promoted through the ability of ALA to suppress hypothalamic AMP-activated protein kinase and decrease dietary energy intake, reduce lipoprotein lipase activity, increase energy expenditure, lipolysis, and insulin sensitivity and inhibit lipogenesis [*Namazi N et al., 2018*]. ALA stimulates the utilization and storage of glucose in muscle cells by increased sensitivity to insulin via PI3K/Akt activation [*Capece U et al., 2022*].

ALA alleviates damage caused by oxidative stress associated with chronic metabolic disorders [*Nguyen H et al.,* 2025], and improves the efficiency of intrinsic antioxidant systems and restores their functionality; e.g. recycles vitamin C and vitamin E, converts NAD to NADP, NADH to NADPH). Anti-inflammatory effect of ALA is exhibited through the inhibition of NF-kappa B a protein complex that triggers various inflammatory molecules. In addition, ALA reduces the osmotic load on nerves with sorbitol, normalizes the concentration of myoinositol and taurine, and improves the ability of the myocardium for frequency variability [*Shay KP et al., 2009*; *Golbidi S et al., 2011*; *Gomes MB, Negrato CA., 2014*; *Galeshkalami NS et al., 2019*].

Clinical trials assessing the effect of alpha-lipoic acid in obesity treatment

Based on the last systematic review and meta-analysis of studies assessing the effect of ALA on weight control, evidence indicate that ALA could be considered as a valuable agent for weight loss strategies [*Vajdi M et al., 2020*]. An earlier sys-

Table 1.

Overview of the registered clinical trials assessing the effect of alpha-lipoic acid on obesity

Study title	***An Open-Label Pilot Trial of Alpha Lipoic Acid (ALA) for Weight Loss in Schizophrenia***
Registration number	NCT01355952
Participants	Men and women, age 18 to 60 years, having at least one risk factor: hypertension, dyslipidaemia or impaired fasting glucose
Study design	Open label (non-randomized)
Intervention	1800 *mg* ALA/day (600 *mg* 3 times per day) during 10 weeks
Primary outcomes	Change in body weight
Study title	***Effect of Alpha Lipoic Acid on Obesity Related Comorbidities***
Registration number	NCT00994513
Participants	Men and women, age 18 to 60 years, having at least one risk factor: hypertension, dyslipidaemia or impaired fasting glucose
Study design	Crossover, randomized (quadriple masking)
Intervention	1200 *mg* ALA/day or 1200 *mg* placebo/day during 2 months
Primary outcomes	Biomarkers of oxidative stress, insulin sensitivity, endothelial function
Study title	***Effects of Alpha Lipoic Acid Supplementation on Metabolic Syndrome Markers in Young Overweight or Obese Males***
Registration number	NCT03342599
Participants	Men, age 18-35 years
Study design	Parallel, randomized (triple masking)
Intervention	600 *mg* ALA/day or 600 *mg* placebo (cellulose starch)/day during 8 weeks
Primary outcomes	Fasting glucose, plasma inflammation biomarkers and body composition (weight, fat and muscle mass)
Study title	***The Role of R-Alpha Lipoic Acid in the Treatment of Atherosclerotic Vascular Disease***
Registration number	NCT00764270
Participants	Men and women, age 50 to 70 years, documented congestive heart disease
Study design	Crossover, randomized (quadriple masking)
Intervention	600 *mg* R-ALA/day (2 doses) or 600 *mg* placebo/day during 12 weeks followed by a 12 week washout
Primary outcomes	High sensitive C-reactive protein
Study title	***Trial of the Combination of Alpha-Lipoic Acid and Mirabegron in Women and in Men With Obesity***
Registration number	NCT05713799
Participants	Men and women, age 18 to 65 years
Study design	Crossover, randomized (single masking)
Intervention	Mirabergon (50 *mg*/day) + ALA 2.4 *g*/day or mirabergon 50 *mg*/day + placebo during 4 weeks followed by a 4-12 weeks washout
Primary outcomes	Changes in the Insulin sensitivity index
Study title	***Lipoic Acid and Prevention of Heart Disease***
Registration number	NCT00765310
Participants	Men and women, age 18 to 60 years, with elevated plasma triglycerides
Study design	Parallel, randomized (triple masking)
Intervention	600 *mg* R-ALA/day or 600 *mg* placebo/day during 12 and 24 weeks
Primary outcomes	Triglycerides
Study title	***Dose Ranging Study of the Effects of Alpha Lipoic Acid on Oxidative Stress***
Registration number	NCT00857376
Participants	Men and women, age 40 and over, diabetes or metabolic syndrome
Study design	Parallel, randomized (triple masking)
Intervention	Placebo or 600 *mg*, 1200 *mg* and 1800 *mg* ALA/day during 3 months
Primary outcomes	Biomarkers of oxidative stress
Study title	***Immunometabolic Effects of Non-drug Strategies in the Clinical Management of Obesity: Translational Study***
Registration number	NCT04436419
Participants	Women, age 18 to 75 years
Study design	Parallel, randomized (triple masking)
Intervention	Placebo or 600 *mg* R-ALA/day during 8 week full hospital programme
Primary outcomes	Variation in the prevalence of regulatory T cells, mRNA expression, redox status by measuring GSH/GSSH ratio
Study title	***Effects of Lipoic Acid and Eicosapentaenoic Acid (EPA) in Human Obesity***
Registration number	NCT01138774
Participants	Women, age 20 to 45 years
Study design	Parallel, randomized (triple masking)
Intervention	Placebo, 1.3 *g* EPA, 300 *mg* ALA or 1.3 *g* EPA + 300 *mg* ALA/day during 10 weeks along with 30% energy restricted diet
Primary outcomes	Weight change, body composition, glucose metabolism

tematic review and meta-analysis [*Namazi N et al., 2018*] found that ALA in comparison to placebo has a small but consistent effect on weight reduction and doses up to 1200 *mg/*day are well tolerated. The effect on anthropometric measures was greater in individuals with underlying health conditions in comparison to healthy individuals.

Despite being highly abundant in foods like red meat, liver, broccoli, carrots, beets, spinach and tomatoes [*Nguyen H et al., 2025*]. Given that, foods with naturally high content of ALA are consumed in small amounts and rarely (organs like kidney, liver and heart) dietary consumption of ALA is small. It is widely available as a dietary supplement, with doses varying from 100 *mg* to 600 *mg* per capsule. For healthy individuals, caution is needed for possible intoxication and adverse effects, and calculated upper safe intake is 42 *mg* ALA per person per day [*Koh EH et al., 2011*]. Favourable tolerability of ALA has been well-documented tolerability in many *in vivo* studies and in human clinical trials [*Cremer DR et al., 2006*]. Still, earlier systematic review and meta-analysis concluded that the intervention with ALA cannot be considered as a cost-effective treatment for obesity [*Namazi N et al., 2018*].

In the database of clinical trials, we conducted a search for clinical trials registered by January 31, 2025. We combined only two keywords, obesity and ALA, and got a list of 15 trials in total. The search was done in the ClinicalTrials.gov database. Out of all 15 trials, nine are completed, one is active but not recruiting, three not yet recruiting and two with unknown status. Complex interventions (ALA combined with at least one or more other active components) were used in six trials and they were excluded from the analysis. Out of the remaining nine trials (**Table 1**), one was conducted only on men, two included only women, one observed the effect of ALA on weight loss in patients with schizophrenia, two focused specifically on atherosclerosis and one assessed the effects on metabolic syndrome.

Chronic use of ALA in high doses was found to be well tolerated and improves glycaemic status and lipid profile [*Derosa G. et al., 2020*], and no significant side effects were recorded [*Huerta AE et al., 2015*; *Li N. et al., 2017*].

In women with polycystic ovary syndrome (PCOS), positive effect of ALA is primarily visible through glucose metabolism alterations, specifically reduced fasting blood glucose and improved insulin resistance. Yet, mixed findings were found for hormones (oestrogen, testosterone, luteinizing hormone etc.), varying greatly between doses (less or more than 600 *mg* ALA/day), intervention duration (less or more than 12 weeks) and whether ALA was combined with inositol, which is a common intervention modality for women with PCOS [*bu-Zaid et al., 2024*]. Non-randomized interventions study by *Genazzani AD* et al. [2018] supplemented 32 obese women with PCOS with 400 *mg* of ALA per day during 12 weeks and observed insulin resistance and metabolic profile. Metabolic profile improved in all women, especially those reporting familiar diabetes [*Genazzani AD et al., 2018*], which could have extended health benefits for these women.

After observing the effect of ALA on glucose metabolism in adults, systematic review and dose-response meta-analysis found significant reduction of insulin, HOMA-IR, but no effect was found on glucose or HbA1C. The effect of insulin was duration-dependent [*Mahmoudi-Nezhad M et al., 2021*]. In terms of inflammatory biomarkers, ALA was found to significantly reduce C-reactive protein, interleukine-6 and tumour necrosis factor-α concentrations in a linear way [*Vajdi M et al., 2023*].

Conclusion

Pandemic proportions of obesity do not show signs of stagnation. Health burden related to obesity is immense, making a point towards finding new, adjuvant options to tackle obesity. ALA has been well-researched in a number of clinical trials for various anti-obesity activities - from fat and glucose metabolism to antioxidant potential. Given its relatively small abundancy in diet, ALA dietary supplements are a worthy option to consider for people with metabolic syndrome, dyslipidemia, diabetes and insulin resistance, or conditions with an underlying inflammation. Though relatively sparse, clinical trials assessing the effect of ALA on weight control, body composition and consequent metabolic alterations show promising results.

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