

RESEARCH MONOGRAPH . COMPOUND PROFILE

R-Alpha-Lipoic Acid (R-ALA)

The R-enantiomer of alpha-lipoic acid; the naturally occurring form with mitochondrial cofactor and antioxidant activity.

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ABSTRACT

R-alpha-lipoic acid (R-ALA, (R)-thioctic acid; CAS 1200-22-2; molecular formula $C_8H_{14}O_2S_2$; molecular weight 206.32) is the R-enantiomer of alpha-lipoic acid, the form naturally synthesized in mammalian mitochondria from octanoic acid and cysteine. The compound is a mitochondrial cofactor for pyruvate dehydrogenase, alpha-ketoglutarate dehydrogenase, and branched-chain alpha-keto acid dehydrogenase. The R-enantiomer is preferentially recognized by these enzymes; racemic ALA preparations contain 50 percent inactive S-ALA. Pharmacologically, R-ALA is a redox-active compound functioning as both reduced (dihydrolipoic acid) and oxidized forms; the redox cycle scavenges multiple reactive oxygen species. Approved in Germany and other European countries for diabetic peripheral neuropathy (Thioctacid); used in many countries as a supplement. Plasma half-life is short (approximately 30 minutes); peak concentrations are higher with R-ALA versus racemic ALA at equimolar doses. Used as the canonical mitochondrial cofactor antioxidant.

1. Compound Identification

R-Alpha-Lipoic Acid (R-ALA) is described as mitochondrial cofactor / antioxidant (r-enantiomer). The R-enantiomer of alpha-lipoic acid; the naturally occurring form with mitochondrial cofactor and antioxidant activity.

1.1 Class summary

Mitochondrial cofactor / antioxidant (R-enantiomer)

1.2 Subtitle

The R-enantiomer of alpha-lipoic acid; the naturally occurring form with mitochondrial cofactor and antioxidant activity.

2. Abstract

R-alpha-lipoic acid (R-ALA, (R)-thioctic acid; CAS 1200-22-2; molecular formula $C_8H_{14}O_2S_2$; molecular weight 206.32) is the R-enantiomer of alpha-lipoic acid, the form naturally synthesized in mammalian mitochondria from octanoic acid and cysteine. The compound is a mitochondrial cofactor for pyruvate dehydrogenase, alpha-ketoglutarate dehydrogenase, and branched-chain alpha-keto acid dehydrogenase. The R-enantiomer is preferentially recognized by these enzymes; racemic ALA preparations contain 50 percent inactive S-ALA. Pharmacologically, R-ALA is a redox-active compound functioning as both reduced (dihydrolipoic acid) and oxidized forms; the redox cycle scavenges multiple reactive oxygen species. Approved in Germany and other European countries for diabetic peripheral neuropathy (Thioctacid); used in many countries as a supplement. Plasma half-life is short (approximately 30 minutes); peak concentrations are higher with R-ALA versus racemic ALA at equimolar doses. Used as the canonical mitochondrial cofactor antioxidant.

3. Mechanism of Action

Mitochondrial cofactor for keto acid dehydrogenases; redox-active antioxidant. R-enantiomer is the natural form preferentially recognized by metabolic enzymes.

The mechanistic profile described above represents the consensus position from peer-reviewed literature where available; for compounds with sparse formal characterization, this section reflects vendor-grade reports and structurally-related class pharmacology and should be interpreted with appropriate caution.

4. Pharmacokinetics and Routes of Administration

Pharmacokinetic data are summarized within the abstract for compounds with formal Phase 1 records. Compounds without published human pharmacokinetic studies are noted as such; investigators should treat the absence of data as a research limitation.

5. Reported Research Dose Ranges

Supplement 100 to 600 mg per oral administration daily.

Doses are scaled from clinical use where the compound is approved as a medicine, from Phase 2/3 trials where development advanced, or from rodent dose-response data scaled by allometric assumptions where the compound has no human clinical record. Investigators should treat dose ranges from research-chemical-grade vendors as starting points for their own pharmacokinetic and tolerability characterization rather than validated dosing recommendations.

6. Sourcing and Quality Verification

Investigators should obtain analytical confirmation of identity (mass spectrometry; for peptides, additionally amino acid analysis or N-terminal sequencing) and purity (HPLC area-percent, ideally greater than 95 percent) on every lot. Vendor certificates of analysis vary in quality; third-party analytical confirmation through independent laboratories (e.g., Janoshik Analytical, Anabolic Lab) is the gold standard for research-grade peptides and small molecules.

For compounds with established pharmaceutical-grade marketing in any jurisdiction, sourcing from the marketed product (where legal and accessible) provides the highest quality assurance.

7. Reconstitution and Handling

Peptides should be reconstituted in bacteriostatic water or sterile water for injection in the volume appropriate to the desired concentration; lyophilized peptides should be allowed to come to room temperature before reconstitution to minimize precipitation. Reconstituted peptides should be stored refrigerated and used within the timeframe specified by the manufacturer (typically 14 to 30 days for bacteriostatic water reconstitutions, shorter for sterile water).

Small molecules: storage conditions vary by compound; consult the certificate of analysis for the specific product. Most are stable at room temperature in dry, dark conditions; oxidation-sensitive compounds (e.g., catecholamine precursors) require cool storage and protection from light.

8. Stack Interaction Considerations

Investigators combining this compound with other research substances should consider receptor-level interactions (additive effects at shared targets), enzyme-level interactions (CYP-mediated drug-drug interactions where applicable), and pharmacodynamic interactions (additive sedation, additive serotonergic activity, additive hemodynamic effects).

The stack interaction section in published monographs of well-characterized compounds is necessarily incomplete for any specific combination; investigators should treat novel combinations with elevated caution and titrate gradually.

9. References

1. Packer L, et al. Alpha-lipoic acid as a biological antioxidant. Free Radic Biol Med 1995.
2. Ziegler D, et al. Treatment of symptomatic diabetic peripheral neuropathy with the antioxidant alpha-lipoic acid. Diabetologia 1995.
3. Hermann R, et al. Enantioselective pharmacokinetics and bioavailability of different racemic alpha-lipoic acid formulations. Eur J Pharm Sci 1996.

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