

# Abstract

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## Does genetic variation in the Delta6-desaturase promoter modify the association between alpha-linolenic acid and the prevalence of metabolic syndrome?

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**BACKGROUND:** Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are associated with protection against components of the metabolic syndrome, but the role of alpha-linolenic acid (ALA), the metabolic precursor of EPA and DHA, has not been studied. The Delta(6)-desaturase enzyme converts ALA into EPA and DHA, and genetic variation in the Delta(6)-desaturase gene (FADS2) may affect this conversion.

**OBJECTIVES:** We hypothesize that high ALA is associated with a lower prevalence of the metabolic syndrome and that genetic variation in FADS2 modifies this association.

**DESIGN:** We studied 1815 Costa Rican adults. Adipose tissue ALA was used as a biomarker of intake, and metabolic syndrome was identified with the definition from the National Cholesterol Education Program, Adult Treatment Panel III. Prevalence ratios (PRs) and 95% CIs were estimated from binomial regression models, and the likelihood ratio was used to test for effect modification.

**RESULTS:** High concentrations of adipose tissue ALA were associated with lower PRs of the metabolic syndrome compared with low ALA (0.81; 95% CI: 0.66, 1.00, for the comparison between the highest and the lowest quintiles; P for trend < 0.02). Higher concentrations of adipose tissue ALA were associated with a lower PR among homozygote (0.67; 95% CI: 0.53, 0.86) and heterozygote (0.84; 95% CI: 0.72, 0.99) carriers of the FADS2 T allele, but not among homozygote carriers of the deletion variant allele (0.99; 95% CI: 0.78, 1.27; P for interaction: 0.08).

**CONCLUSIONS:** Elevated ALA concentrations in adipose tissue are associated with lower prevalence of the metabolic syndrome. A lack of association among homozygote carriers of the FADS2 deletion allele suggests that this association may be due in part to the conversion of ALA into EPA.

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