Abstract

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Relationship of ascorbic acid to blood lead levels.

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CONTEXT: Some animal studies suggest that orally administered ascorbic acid may chelate lead and decrease the risk of the toxic effects of lead. However, results from several small studies in humans have yielded inconclusive evidence of a beneficial effect of ascorbic acid on lead toxicity.

OBJECTIVE: To examine the relationship between serum ascorbic acid levels and prevalence of elevated blood lead levels.

DESIGN, SETTING, AND PARTICIPANTS: Cross-sectional analysis of a probability sample of the US population enrolled in the Third National Health and Nutrition Examination Survey, 1988-1994 (4213 youths aged 6-16 years and 15365 adults aged > or =17 years) without a history of lead poisoning.

MAIN OUTCOME MEASURES: Elevated and log blood lead levels by serum ascorbic acid level.

RESULTS: A total of 22 youths (0.5%) and 57 adults (0.4%) had elevated blood lead levels (defined as > or =0.72 micromol/L [15 microg/dL]) and > or =0.97 micromol/L [20 microg/dL], respectively). After controlling for the effects of age, race, sex, income level, and dietary energy, fat, calcium, iron, and zinc intake, youths in the highest serum ascorbic acid tertile had an 89% decreased prevalence of elevated blood lead levels compared with youths in the lowest serum ascorbic acid tertile (odds ratio, 0.11; 95% confidence interval, 0.04-0.35; P for trend = .002). Adults in the highest 2 serum ascorbic acid tertiles had a 65% to 68% decreased prevalence of elevated blood lead levels compared with adults in the lowest serum ascorbic acid tertile (P for trend = .03). As a continuous predictor, serum ascorbic acid level was independently associated with decreased log blood lead levels among adults (P<.001), but not among youths (P=.14).

CONCLUSIONS: Our data suggest that high serum levels of ascorbic acid are independently associated with a decreased prevalence of elevated blood lead levels. If these associations are related causally, ascorbic acid intake may have public health implications for control of lead toxicity.

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