Acute effects of alcohol on plasma ascorbic acid in healthy subjects[1]

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ABSTRACT The acute effects of ethanol on plasma ascorbic acid were assessed in healthy subjects. After the ingestion of 2.0 g ascorbic acid and breakfast, plasma ascorbic acid rose from a fasting concentration of 7.5 ± 0.8 ng/ml at 0900 h, to a peak of 26.9 ±m 2.0 ng/ml at 1500 h. When 35 g ethanol was ingested with ascorbic acid and breakfast, plasma ascorbic acid concentrations were significantly lower for at least 24 h. *Am. J. Clin. Nutr.* 34: 2394–2396, 1981.

KEY WORDS Alcohol, ethanol, ascorbic acid, human

Introduction

Alcohol abuse constitutes one of the principle nutritional problems of Western society (1). Data from the Australian Bureau of Census and Statistics indicated that in 1977, 18% of Australian males drank more than 40 g alcohol a day and 4% drank more than 80 g a day (2). Deficiencies of the vitamins, thiamin (3) and folic acid (4), are well recognized in alcohol abusers. However, little is known about the effect of alcohol on ascorbic acid. Dietary (5) and urinary ascorbic acid studies (6) have suggested that ascorbic acid deficiency occurs in alcoholics.

It has been demonstrated that the clearance of ethanol from the blood is proportional to the leucocyte ascorbic acid concentration (7). The activity of alcohol dehydrogenase may depend on ascorbic acid saturation.

To assess the effect of alcohol on the bioavailability of ascorbic acid, we have administered ethanol with ascorbic acid at breakfast and have measured plasma ascorbic acid concentrations.

Methods

All subjects were healthy volunteers, three males and two females, whose ages ranged from 21 to 36 yr. They were within 10% of desirable body weight (8), nonsmokers, and had an average ethanol intake of less than 20 g/day. Medications known to interfere with ascorbic acid metabolism and ascorbic acid rich foods were avoided. Each subject was studied after an overnight fast twice as designated below.

1) After a fasting blood sample (0900 h) 2 g ascorbic acid was ingested with breakfast over a 5-min period. This consisted of a buttered yeast bun and coffee with milk which was estimated to contain less than 1 mg ascorbic acid. Lunch consisted of a bread roll with salad and coffee with milk, eaten from 1230 to 1245 h; the ascorbic content was about 2 mg ascorbic acid (9). Additional blood samples were taken at 1100 and 1500 h on day 1, and fasting at 0900 h on days 2, 4, and 8.

2) Thirty-five grams ethanol in distilled water was ingested with breakfast and with 2 g ascorbic acid. Observations were made at the same times as without ethanol.

Plasma ascorbic acid was determined by the method of Attwood et al. (10). Blood alcohol concentrations were estimated with an Alcolmeter (Evidential Digital AE-D1 Lion Lab. Ltd. Pearl St., Cardiff CF 21PP).

Results

Ingestion of 2.0 g ascorbic acid supplement with breakfast caused a significant rise in plasma ascorbic acid concentration from 7.5 ± 0.8 ng/ml at 0900 h to 26.9 ± 2.0 ng/ml at 1500 h ($p < 0.001$). When 35 g ethanol was ingested with 2.0 g ascorbic acid supplement and breakfast, the fasting plasma ascorbic acid concentration of 9.9 ± 0.5/ml increased to 18.9 ± 2.8 ng/nl at 1500 h ($p < 0.01$). Plasma ascorbic acid concentrations were significantly lower with ethanol for at least 24 h. **Figure 1** illustrates these changes as a percentage of base-line.

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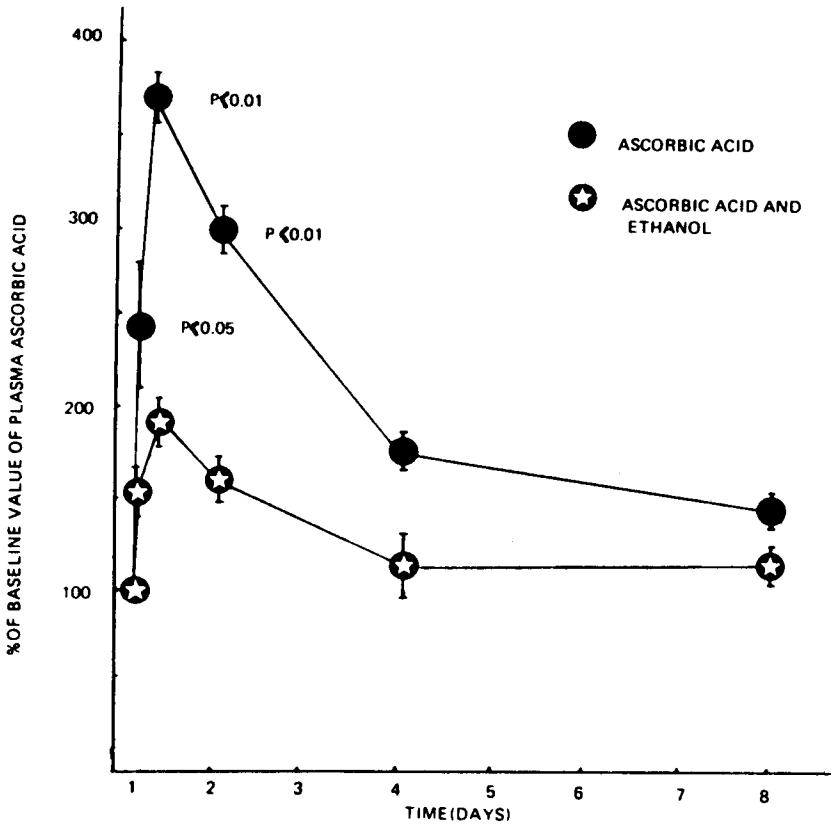

FIG. 1. The response to a 2-g dose of ascorbic acid with and without 35 g ethanol in five healthy subjects. The two responses were significantly different 2 h ($p < 0.05$), 6 and 24 h ($p < 0.01$) after the ascorbic acid dose. (Student's t test was used for paired comparisons.)

The subjects obtained a blood alcohol concentration of 0.046 ± 0.006 g/100 ml 2 h after ingestion of ethanol. Subsequent alcohol readings showed that 6 h after ethanol, blood alcohol concentrations in all subjects were zero.

Discussion

In this study, when ethanol was ingested with ascorbic acid the rise in plasma ascorbic acid was less marked. This was probably due to an impairment in absorption of ascorbic acid by ethanol, rather than an increase in the excretion, catabolism, or utilization of ascorbic acid. Ascorbic acid is absorbed by a Na^+-dependent active transport mechanism (11). Such a mechanism may be adversely affected with acute ethanol ingestion (12). Ethanol may alter the renal threshold of ascorbic acid, but with normal ascorbic acid intakes plasma concentrations do not normally reach the renal threshold (13). The increased renal acid load which results from ethanol consumption, caused by higher levels of lactic acid, uric acid, short-chain fatty acids, and ketones (14–16), might secondarily reduce the excretion of ascorbic acid and increase plasma ascorbic acid concentration. The opposite was observed. These findings indicate that ethanol may reduce the availability of ascorbic acid from food and predispose to ascorbic acid deficiency.

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