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EFFECT OF ACARBOSE ON THE INCREASED PLASMA CONCENTRATION OF URIC ACID INDUCED BY SUCROSE INGESTION

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□ Sucrose is converted fructose and glucose, which may increase plasma uric acid concentration (pUA) through increased purine degradation and/or decreased uric acid (UA) excretion. To investigate effects of acarbose, an inhibitor of alpha-glucosidase, on the increased pUA from sucrose administration, we measured pUA and urinary UA excretion in 6 healthy subjects before and after administering sucrose, with and without co-administration of acarbose. Sucrose raised pUA by 10% ($p < 0.01$). However, excretion and fractional clearance of UA were unchanged. Sucrose and acarbose coadministration also increased pUA, but less than did sucrose alone (sucrose: 4.9 to 5.4 mg/dl; sucrose + acarbose, 4.7 to 4.9 mg/dl, $p < 0.05$) without changes in urinary excretion and fractional clearance of UA. Acarbose appears to attenuate the rise in pUA by sucrose ingestion by inhibiting sucrose absorption.

Keywords Sucrose; uric acid; alpha-glucosidase inhibitor; purine degradation

INTRODUCTION

Large amounts of sucrose ingestion have been shown to cause an increased concentration of plasma uric acid through enhanced purine degradation.^[1] Alpha-glucosidase inhibitors reduce or delay carbohydrate digestion,^[2,3] and thereby are expected to suppress the uric acid raising-effect of sucrose by retarding fructose and glucose absorption. We studied whether oral administration of an alpha-glucosidase inhibitor, acarbose, could alleviate the uric acid raising-effect of sucrose.

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SUBJECTS AND METHODS

Six healthy men were included in this study. In Experiment 1, urine was collected every hour for a total of three times (periods 1, 2, and 3), and blood was drawn at the mid-point of each one-hour collection period. After the period 1 urine collection, 1.5 g/kg body weight of sucrose was administered. Two weeks later, the second experiment (Experiment 2) was performed with the same protocol, except that acarbose (100 mg) was administered at the beginning of sucrose ingestion. The concentrations of hypoxanthine, xanthine, and uric acid in plasma and urine were measured. Plasma glucose, insulin, and lactate were also measured. Data are reported as the mean \pm SD and a student's *t* test was employed to estimate the observed differences of the two groups. A *p* value less than 0.05 was considered significant.

RESULTS

Experiment 1

Sucrose increased the plasma concentration of uric acid from 4.9 ± 1.0 mg/dL (period 1) to 5.3 ± 1.2 mg/dL (period 2, $p < 0.01$), and 5.4 ± 1.1 mg/dL (period 3, $p < 0.01$), whereas sucrose did not significantly increase the plasma concentrations of hypoxanthine and xanthine. Sucrose did not significantly affect the urinary excretion and fractional clearances of purine bases. The plasma glucose level was increased by 40% ($p < 0.01$) at period 2 as compared to that at period 1. The concentrations of plasma insulin and blood lactate were also significantly increased at period 2 and at period 3, as compared with that at period 1.

Experiment 2

Sucrose plus acarbose increased the plasma concentration of uric acid from 4.7 ± 1.3 mg/dl (period 1) to 4.9 ± 1.4 mg/dl (period 2, $p < 0.01$) and 4.9 ± 1.4 mg/dl (period 3, $p < 0.05$), whereas sucrose plus acarbose did not significantly increase the plasma concentration of hypoxanthine and xanthine. The increase in plasma concentration of uric acid was blunted by the pre-administration of acarbose (period 2–period 1; $p < 0.05$, period 3–period 1; $p < 0.01$). Sucrose plus acarbose did not significantly affect the urinary excretion or the fractional excretion of purine bases. The plasma glucose level was increased by 14% ($p < 0.01$) at period 2 as compared to that at period 1. The plasma insulin concentration was also increased by 3.1 fold ($p < 0.01$) at period 2 and 3.8 fold ($p < 0.01$) at period 3, as compared with that at period 1. Acarbose suppressed the increase in the levels of plasma glucose and serum insulin induced by sucrose ingestion.

TABLE 1 Increase in plasma uric acid concentration after sucrose and sucrose plus acarbose ingestion

Uric acid (mg/dL)	Period 2–Period 1	Period 3–Period 1
Sucrose	0.45 ± 0.31**	0.57 ± 0.20*
Sucrose + acarbose	0.17 ± 0.08**	0.17 ± 0.12*

Data are reported as the mean ± SD.

*and** denote $p < 0.01$ and $p < 0.05$.

DISCUSSION

This study demonstrates that acarbose alleviates the uric acid-raising effect of sucrose. However, levels of plasma insulin and blood lactate concentrations increased by sucrose ingestion were not sufficient to decrease uric acid clearance. In contrast, it was demonstrated that sucrose ingestion increased purine degradation, as indicated by the increased plasma and urinary excretion of oxypurines after allopurinol pre-treatment.^[1] In Experiment 1, we confirmed the results of this previous study, and in Experiment 2, we showed that acarbose suppressed the uric acid-raising effect of sucrose, but neither increased fractional uric acid clearance nor decreased urinary uric acid excretion. Therefore, the alleviating effect of acarbose on the rise in uric acid concentration by sucrose ingestion may be due to its inhibitory action on sucrose absorption at the small intestine leading to the delayed conversion to fructose, rather than to a direct effect on purine metabolism. From these results, acarbose is expected to prevent the development of hyperuricemia caused by excessive intake of sucrose, as well as the preferable effect of preventing or delaying conversion of impaired glucose tolerance to overt diabetes.^[4]

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