

Vinegar Ingestion at Bedtime Moderates Waking Glucose Concentrations in Adults With Well-Controlled Type 2 Diabetes

Andrea M. White, PHD and Carol S. Johnston, PHD

+ Author Affiliations

Address correspondence and reprint requests to Dr. Carol S. Johnston, 7001 E. Williams Field Rd., Mesa, AZ 85212. E-mail: carol.johnston@asu.edu

Given the importance of maintaining acceptable blood glucose concentrations, there is much interest in identifying foods and diet patterns that will help individuals with diabetes manage their condition. Based on previous data indicating that vinegar ingestion at mealtime reduces postprandial glycemia (1–4), the aim of this pilot study was to examine whether vinegar ingestion at bedtime reduces the next-morning fasting glucose concentration in individuals with type 2 diabetes.

RESEARCH DESIGN AND METHODS—

Four men and seven women (aged 40–72 years) diagnosed with type 2 diabetes (by a physician) who were not taking insulin completed the study. Participants provided a clinically determined A1C reading from a recent (<2 months) blood analysis. All participants gave written informed consent, and the study was approved by the institutional review board at Arizona State University.

Participants maintained 24-h diet records for 3 days and measured fasting glucose at 0700 h for 3 consecutive days with a calibrated glucometer before the start of the study. Participants were instructed to continue usual prescription medication use during the study. Utilizing a randomized crossover design with a 3- to 5-day washout period between treatments, participants followed a standardized meal plan for 2 days, consuming either 2 Tbsp apple cider vinegar or water at bedtime with 1 oz cheese (8 g protein, 1 g carbohydrate, and 1.5 g fat). The standardized meal plan was designed to reflect the individual's typical diet. Participants were instructed to record all foods and beverages ingested during each 2-day treatment period.

Fasting glucose was recorded with a calibrated glucometer by each participant during the trial: at baseline (day 0) and day 2 at 0700 h. These results were downloaded by the research staff from each participant's glucometer memory. A multivariate repeated-measures ANOVA test with body weight as a covariate was used to determine a significant time-by-treatment effect using SPSS (version 14 for Windows; SPSS, Chicago, IL).

RESULTS—

The duration of diabetes averaged 4.9 ± 1.0 years for the participants, and 73% of participants (8 of 11) used prescription hypoglycemic agents during the study. Before the initiation of the study, a BMI of 29.1 ± 1.2 kg/m², a typical fasting glucose of 7.6 ± 0.3 mmol/l, and an A1C of $6.7 \pm 0.2\%$ were recorded for the participants. Participants complied with the dietary protocol as indicated by the diet records maintained during the study; hence, food intake for the two treatment periods was identical within subjects. Fasting glucose was reduced 0.15 mmol/l (2%) and 0.26 mmol/l (4%) for the placebo and vinegar treatments, respectively (time-by-treatment effect, $P = 0.033$) (Fig. 1). Closer examination of the data revealed that the vinegar treatment was particularly effective for the participants with a typical fasting glucose >7.2 mmol/l; in these individuals ($n = 6$), fasting glucose was reduced 6% compared with a reduction of 0.7% in those participants with a typical fasting glucose <7.2 mmol/l ($n = 5$).

CONCLUSIONS—

These data suggest that vinegar ingestion at bedtime may favorably impact waking glucose concentrations in type 2 diabetes. The antiglycemic effect of acetic acid, the active ingredient in vinegar, has been attributed to reduced starch digestion (5) and/or delayed gastric emptying (6). Neither of these proposed mechanisms likely explains the effects noted herein; moreover, to our knowledge, this is the first report describing a hypoglycemic effect of vinegar apart from mealtime. Fushimi et al. (7,8) have published a series of trials in rats demonstrating that acetic acid alters hepatic and skeletal glucose metabolism. These

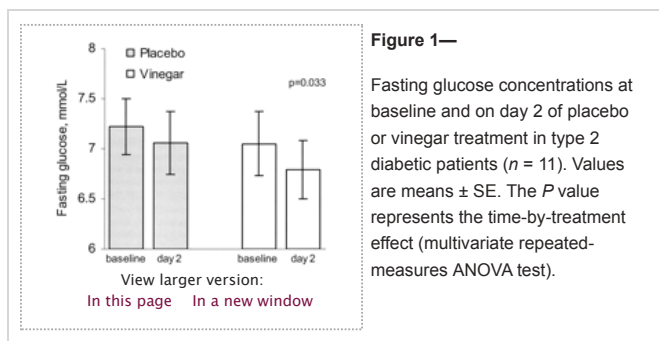
investigations show that acetic acid feeding (0.2 acetic acid/100 g diet) reduced xylulose-5-phosphate accumulation in liver and phosphofructokinase-1 activity in skeletal muscle—metabolic changes consistent with reduced glycolysis and the promotion of glycogen synthesis. Hence, acetic acid may possibly alter the glycolysis/gluconeogenic cycle in liver, which may benefit diabetic individuals with metabolic disturbances contributing to a prebreakfast rise in fasting glucose (also known as the “dawn phenomenon”) (9).

Reductions in fasting glucose of the magnitudes noted in this study (4–6%) are less than that observed in trials examining the efficacy of pharmaceutical hypoglycemic agents for inadequately controlled diabetes. In these trials, pretrial A1C values averaged 7.8–8.8%, and fasting glucose concentrations were reduced 10–15% by long-term drug therapy (10–12). In comparison, the diabetic condition of our subjects was well controlled (A1C 6.7 ± 0.2%). Notably, although 72% of our subjects regularly used hypoglycemic medications and continued their medication use during the study, the vinegar treatment significantly impacted fasting glucose. In individuals with early diabetes (A1C <6.3%) or well-controlled diabetes (A1C 6.8–7.0%), pharmaceutical interventions had a more moderate effect, reducing fasting glucose 3–6% (13,14).

The control cheese plus water treatment reduced fasting glucose 2% ($P = 0.928$), whereas the cheese plus vinegar treatment reduced fasting glucose 4% ($P = 0.046$). We cannot rule out the possibility of a synergistic effect for cheese and vinegar. Based on our previous work, we felt that the subjects needed a small amount of food to take with the vinegar for ease of application. Fasting hyperglycemia in type 2 diabetes has been related to an intrahepatic malfunction that increases overall hepatic glucose output, perhaps the inhibition of glycogen synthesis (15). In type 2 diabetes, dietary protein has a minimal effect on glycemia, even though gluconeogenesis is increased (16,17). Amino acids from cheese may provide glucogenic substrates, which in the presence of insulin convert to glycogen (18,19), a scenario that may benefit individuals with type 2 diabetes. We were unable to find research investigating a role for nighttime snacks in managing waking hyperglycemia in type 2 diabetes; our data suggest that this possibility should be explored.

Although this study is limited by the small sample size ($n = 11$), the within-subject design reduced error variance due to individual differences. Also, renal function and a detailed history of medication use were not assessed in this study and may have impacted these results.

Vinegar is widely available, it is affordable, and it is appealing as a remedy, but much more work is required to determine whether vinegar is a useful adjunct therapy for individuals with diabetes. Investigations are needed to study the mechanisms by which vinegar alters postprandial glycemia and fasting glucose and to examine the efficacy of vinegar ingestion in individuals with inadequately controlled diabetes.



Footnotes

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A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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