Effect of alcoholic beverages on postprandial glycemia and insulinemia in lean, young, healthy adults1–3

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ABSTRACT

Background: Ethanol’s ability to inhibit gluconeogenesis might reduce postprandial glycemia in realistic meal settings.

Objective: The objective was to explore the effect of 3 types of alcoholic beverages consumed alone, with a meal, or 1 h before a meal on postprandial glycemia in healthy subjects.

Design: In study 1, isonenergetic (1000 kJ) servings of beer, white wine, and gin were compared with a 1000-kJ portion of white bread. In study 2, the same servings were compared with water as an accompaniment to a bread meal. In study 3, 20-g alcohol portions were served as a premeal drink. Fingertip capillary blood samples were taken at regular intervals over 2–3 h.

Results: In study 1, the mean (±SE) glucose scores for beer (58 ± 11), wine (7 ± 3), and gin (10 ± 5) were significantly lower (P < 0.001) than those for bread (= 100). In study 2, meals consumed with beer (84 ± 1; P = 0.03), wine (63 ± 6; P < 0.001), and gin (80 ± 12; P = 0.007) produced less glycemia than did the meal consumed with water (= 100). In study 3, all 3 beverages reduced the postprandial glycemic response to the subsequent meal (67 ± 5, 75 ± 6, and 78 ± 4 with the beer, wine, and gin trials, respectively; P < 0.003).

Conclusion: In realistic settings, alcoholic beverage consumption lowers postprandial glycemia by 16–37%, which represents an unrecognized mechanism by which alcohol may reduce the risk of chronic disease. Am J Clin Nutr 2007;85:1545–51.

KEY WORDS Alcohol, glucose, insulin, postprandial hyperglycemia

INTRODUCTION

Large prospective studies and meta-analyses have directly linked light-to-moderate alcohol intake with a lower risk of coronary heart disease and type 2 diabetes (1–4). Several mechanisms have been suggested to underlie the apparent protective effect, including ethanol’s ability to raise the HDL concentration and reduce coagulatory activity. Moderate consumption (1–3 drinks/d) has also been associated with lower fasting glucose and insulin concentrations (5, 6), improved glucose tolerance (6, 7), and lower insulin resistance estimated indirectly by homeostasis modeling assessment (6). Some evidence is conflicting. Alcoholic beverages have decreased glucose utilization in some studies (8, 9), yet increased insulin sensitivity in others (6, 10, 11). Ethanol consumption is also known to directly inhibit gluconeogenesis (12, 13), which theoretically might contribute to reductions in postprandial glycemia. In one study, ethanol administered every hour in the afternoon before a glucose load at 1700 resulted in substantially reduced blood glucose concentrations (14). Alcohol consumption is a recognized risk factor for hypoglycemia in individuals with type 1 diabetes (15, 16), but the effect of alcoholic beverages on postprandial glycemia in healthy subjects (9) and individuals with type 2 diabetes (17) is thought to be negligible. In this context, however, most studies have used forearm venous sampling to detect changes in plasma glucose. It is now recognized that rapid changes in blood glucose concentrations after a meal may be identified more readily at fingertip sites than at the forearm (18). For this reason, it is possible that modest, but clinically important, effects of alcohol on postprandial glycemia may have gone undetected.

The aim of the present studies was to investigate the acute effects of realistic portions of 3 different alcoholic beverages (not alcohol per se) consumed alone, before, or with a carbohydrate meal on postigestive plasma glucose and insulin responses. We hypothesized that alcohol’s ability to acutely inhibit gluconeogenesis and enhance insulin sensitivity might reduce postprandial glycemia on one hand, but that other components in the beverages, such as the carbohydrates in beer, might increase postprandial responses on the other.

SUBJECTS AND METHODS

Study design

Postprandial responses to beer, white wine, and gin were studied alone (study 1), with a carbohydrate-containing meal (study 2), and 1 h before a meal (study 3). In all 3 studies, each subject underwent 5 tests: 3 with the test drinks and 2 with the reference meal. All tests were given in random order, on separate days ≥3 d apart. Subjects were tested at the same time of the day under similar conditions and acted as their own controls. They were instructed to maintain regular activity patterns, to abstain from...
alcohol on the previous day, and to avoid legumes in the prior evening meal. The energy, macronutrient, and alcohol contents of the test foods and reference meals in each study are shown in Table 1. Nutrient composition was calculated by using FOODWORKS Professional Edition version 3.0 (Xyris Software, Brisbane, Australia), which is based on the Australian food-composition tables and manufacturers’ data.

**Subjects**

Healthy, nonsmoking volunteers were recruited by advertisement from the University of Sydney student population. Exclusion criteria included a family history of type 2 diabetes or a recent history of gastrointestinal disease. In study 1, there were 10 subjects (7 men, 3 women) with a mean (±SEM) age of 29 ± 3 y (range: 21–46 y) and a BMI (in kg/m²) of 25 ± 1 (range: 20 ± 27). In study 2, a second set of 10 subjects (5 men, 5 women) with a mean (±SEM) age of 23 ± 1 y (range: 19–26 y) and a BMI of 22 ± 1 (range: 18 ± 25) was studied. In study 3, there were 18 subjects (8 men, 10 women) with a mean (±SEM) age of 22 ± 2 y (range: 22–32 y) and a BMI of 22.3 ± 2.5 (range: 20 ± 23). The study protocol was approved by the institutional human ethics committee, and subjects gave written informed consent.

**Study 1**

The 3 test foods consisted of 1000-kJ portions of beer (Budweiser regular, 5% wt:vol alcohol; Anheuser-Busch, St Louis, MO), white wine (Columbard Chardonnay, 11% wt:vol alcohol; Yalumba Winery, Angaston, South Australia), and gin (Beefeater London distilled dry gin, 40% wt:vol alcohol; James Burrough, London, United Kingdom), corresponding to 33, 39, and 47 g alcohol, respectively. We chose energy equivalence, rather than carbohydrate or alcohol equivalence, because it represented a “level playing field” for the comparison of the postprandial effect of all foods, irrespective of nutrient composition, on glycemia and insulinemia. The white wine and beer were consumed with 250 mL water and the gin with 400 mL water. The reference meal consisted of a 1000-kJ portion of white bread (Tip Top Sunblest Sandwich; Tip Top Bakeries, Chatswood, NSW, Australia) with 250 mL water. On each test day, subjects attended the Human Nutrition Unit at 0800 after a 10-h overnight fast. On arrival, a breakfast consisting of cereal, fruit, and milk was consumed in self-selected amounts held constant within each subject. Exactly 4 h after breakfast, the test beverage or reference food was consumed within 25 min. Fingerprick blood samples (0.5 mL) were collected from warmed hands with an automatic lancet (Autoclix Boehringer Mannheim Australia, Castle Hill, New South Wales) at 0 min (the start of the test meal) and 15, 30, 45, 60, 90, and 120 min after the test food.

**Study 2**

The 3 test meals consisted of 1000-kJ portions of beer, white wine, or gin as in study 1 but were consumed together with a sandwich consisting of 2000 kJ white bread (Tip Top Sunblest Sandwich) and 280 kJ margarine (Meadow Lea Original; Meadow Lea Foods, Macquarie Park, NSW, Australia). The reference meal, tested twice, consisted of the sandwich and 250 mL water. The study design was identical to study 1, except that additional blood samples were taken at 150 and 180 min to allow for the higher total energy content of the meal.

**Study 3**

The 3 predinner drinks consisted of 2 standard drinks (two 10-g alcohol portions) of beer (Toohey’s New Lager; Tooheys Pty Ltd, Lidcombe, NSW, Australia), white wine (Sauvignon Blanc; Yalumba Winery), and gin (Gordon’s Special London Dry Gin); the latter was mixed with low-energy, artificially sweetened “diet” tonic water (Cadbury Schweppes, Sydney, NSW, Australia). In the reference trial, 250 mL water was consumed in lieu of the alcoholic beverages. At 0800 on the test day,
after a 10–12 h fast, the subjects consumed a prepackaged breakfast consisting of cereal (Just Right, 45 g; Kellogg’s, Battle Creek, MI), whole milk (250 mL, Dairy Farmer’s Ltd, Homebush Bay, NSW, Australia), toasted white bread (Tip-Top, 35 g), and orange juice (250 mL, Just Juice; National Foods Ltd, Melbourne, Victoria, Australia). At 1100, the subjects consumed the predinner beverage and, 1 h later, consumed a standard lunch meal consisting of a 50-g carbohydrate portion of mashed potato (Table 1) within 12 min. Fingerprick blood samples were taken at baseline (the start of the meal) and 15, 30, 45, 60, 90, and 120 min after the meal.

Blood samples (≈0.8 mL) were collected into Eppendorf tubes previously coated with heparin (10 IU heparin sodium salt; Sigma Chemical Co, St Louis, MO) and centrifuged at 12 500 × g for 1 min. Plasma was pipetted into chilled tubes and stored at −20 °C until assayed (<1 mo). Plasma glucose concentrations were measured in duplicate with a Cobas Fara automatic spectrophotometric analyzer (Roche Diagnostica, Basel, Switzerland) per the hexokinase/glucose-6-phosphate dehydrogenase method (Unimate 5 Gluc HK; Roche Diagnostic Systems, Frenchs Forest, New South Wales). The mean intra- and interassay CVs were both <3%. Plasma insulin was measured with a commercial antibody coated tube radioimmunoassay kit (Coat-a-Count; Diagnostic Products Corporation, Los Angeles, CA). The mean intraassay CV was 3%, and the mean interassay CV was 3.5% in study 1 and study 2 and 6.3% and 6.6%, respectively, in study 3.

Data treatment and statistical analysis
Cumulative changes in plasma glucose and insulin responses were quantified as the incremental area under the 120-min (study 1 and study 3) or 180-min (study 2) area under the curves (AUCs) and were calculated by using the trapezoidal rule with fasting concentrations representing the baseline truncated at zero. Any area below the baseline was ignored. For each of interpretations, glucose scores were calculated by using the following equation:

\[
\text{Glucose score} = \frac{\text{area under the 120-min (or 180-min) glucose response curve for the test food}}{\text{average area under the 120-min (or 180-min) glucose response curve for the reference meals}} \times 100 \ (1)
\]

In study 2, the AUC was determined over 180 min (in lieu of 120 min) to allow for the higher total energy content of the meals in comparison with study 1 and study 3. Insulin scores were calculated by using the same equation using the corresponding plasma insulin AUC values.

For each study separately, the glucose and insulin AUC data were analyzed by using a general linear model with drink as a fixed factor and subject as a random factor. When drink was

![FIGURE 1. Study 1. Mean (±SE) changes in plasma glucose and insulin and incremental areas under the curve (AUCs) in lean healthy subjects (n = 10) after consumption of a 1000-kJ portion of white bread, beer, wine, or gin. *Significantly different from white bread, \( P < 0.001 \) (2-factor ANOVA followed by pairwise comparisons with Bonferroni adjustment).](image)
significant, multiple comparisons were undertaken by using a Bonferroni adjustment for multiple tests. The effect of sex was examined in study 2 and study 3 by using an extended model that added sex and drink-by-sex interaction as fixed factors. Finally, the overall effect of sex was investigated across all studies by including study as a fixed factor. Statistical analyses were carried out using STATVIEW Student Software (Abacus Concepts Inc, Berkley, CA) and the STATISTICAL PACKAGE FOR THE SOCIAL SCIENCES (SPSS, version 12; SPSS Inc, Chicago, IL), and (adjusted) \( P < 0.05 \) were considered significant.

**RESULTS**

The protocols in all studies were well tolerated, except in one subject, in study 2, who consumed only 75% of the gin portion. Within each study, fasting glucose and insulin concentrations did not differ significantly, averaging 5.2 ± 0.1 mmol/L and 40.9 ± 8.7 pmol/L, respectively, in study 1 and 5.4 ± 0.1 mmol/L and 33.6 ± 8.6 pmol/L, respectively, in study 2. In Study 3, the mean baseline glucose and insulin concentrations at the start of the meal were 5.1 ± 0.1 mmol/L and 48.9 ± 6.0 pmol/L, respectively. There were no significant differences between water and alcohol trials at this time point.

**Study 1**

Mean plasma glucose and insulin response curves in response to the alcoholic beverages and white bread are shown in Figure 1. Taking the reference food as 100, the glucose scores for beer (58 ± 11), white wine (7 ± 3), and gin (10 ± 5) were all significantly lower than the reference food (\( P < 0.001 \) in each case). Beer produced significantly higher scores than did white wine and gin (\( P < 0.003 \) and 0.007, respectively). Similarly, insulin scores for beer (27 ± 5), white wine (4 ± 2), and gin (1 ± 1) were all significantly lower than the reference food (\( P < 0.001 \) in each case). Two hours after each type of alcoholic beverage, the mean insulin concentration, but not the glucose concentration, was significantly lower than fasting concentrations (\( P < 0.05 \)).

**Study 2**

Mean plasma glucose and insulin response curves in response to the 3 test meals and the average reference meal are shown in Figure 2. Relative to the white bread and water trial (= 100), glucose scores for beer, wine, and gin were 84 ± 11, 63 ± 6, and 80 ± 12, respectively, with the white wine and gin meals producing significantly lower glucose AUCs than the reference meal (\( P = 0.001 \) and 0.015, respectively). The beer meal showed only marginal significance (\( P = 0.07 \)). Reductions in glycemia in the wine trial appeared to be more pronounced in the men (50 ± 3) than in the women (77 ± 6), but the effect of sex was not statistically significant overall (\( P = 0.22 \)). Insulin scores for the 3 alcohol-containing test meals were not significantly different from those for the reference meal or each other (Figure 2).
Study 3

Mean plasma glucose and insulin response curves after the standard lunch are shown in Figure 3. In each case, glucose scores (67 ± 5, 75 ± 6, and 78 ± 4 for the beer, wine and gin trials, respectively) were significantly lower than those in the water trial (P < 0.001 for both beer and wine; P = 0.003 for gin).

The mean peak glucose concentration was also significantly lower (8.3 ± 0.2, 8.5 ± 0.2 and 8.6 ± 0.2 in the beer, wine and gin trials respectively) than in the water trial (9.3 ± 0.2; P < 0.001). Conversely, insulin scores tended to be higher (111 ± 7, 114 ± 11, and 138 ± 11 for beer, wine, and gin, respectively), but the difference was significant only in the gin trial (P = 0.007).

There were no differences in peak insulin concentrations. At 120 min after the start of the meal, glucose and insulin concentrations were similar and the alcohol trials did not differ from the water trial (Figure 3). There were no significant sex differences, and adding sex to the model left the results essentially unchanged.

When all 3 studies were combined, sex was not significant for the glucose AUC (P = 0.13) and was only marginally significant for the insulin AUC (P = 0.046).

DISCUSSION

The findings of these 3 studies suggest that, under realistic conditions, moderate quantities of beer, wine, and gin reduce postprandial glycemia by up to 37% in lean healthy subjects. This applies when isoenergetic portions (1000 kJ, or 240 calories) are consumed as an accompaniment to a high-carbohydrate meal or as the equivalent of 2 predinner drinks (20 g alcohol). Surprisingly, beer was effective in reducing glycemia, despite its having a higher carbohydrate content than wine or gin. By themselves, the alcoholic beverages produced much less glycemia than did an isoenergetic portion of a starchy food. The physiologic basis of these findings is likely to be ethanol’s ability to acutely inhibit gluconeogenesis and hepatic glucose output (12). Reducing postprandial hyperglycemia may therefore be an additional mechanism, hitherto unrecognized, through which moderate alcohol consumption may improve glucose homeostasis and consequently lower the risk of chronic disease.

Relatively few studies have systematically investigated the acute metabolic effects of alcoholic beverages (not alcohol per se) on postprandial glycemia. Moreover, to our knowledge, none has specifically studied the effect of predinner drinks consumed in the hour or so before a meal. Although suppression of hepatic glucose output would largely explain the findings (13), beer, wine, and spirits contain components other than ethanol, which means that additional effects are possible. Standard beers contain a small amount of rapidly digested carbohydrate as small-molecular-weight dextrins (3–5% wt:vol, representing the breakdown products of starch digestion by the enzymes in germinated grains) that could potentially contribute to postprandial glycemia and insulinemia. The relative glycemic potency of carbohydrates in different foods can be compared by using GI tables (19), but alcoholic beverages contain too little carbohydrate to be
tested by standard methodology. Even in the case of beer, a 25-g or 50-g carbohydrate portion represents an unrealistically large amount for consumption within a 12 min period (ie, ≈750 and 1500 mL, respectively). Using a nonstandard 10-g carbohydrate portion size for testing, our group found that the GI of beer (Toohey New Draft, 4.6% wt:vol alcohol) was 66 on the glucose = 100 scale (Sydney University’s Glycemic Index Research Service, unpublished observations, 2003). Surprisingly, however, when consumed with or before a carbohydrate meal, we found that beer tended to reduce, rather than increase, postprandial glycemia. This implies that alcohol inhibits hepatic glucose output quickly, counteracting the glycemic effect of glucose absorption from the gut and thereby reducing the overall glycemic response. Prior carbohydrate and alcohol consumption probably “primes” the liver (inhibits gluconeogenesis), which reduces the hepatic glucose output and up-regulates the enzymes involved in glucose uptake.

In type 1 diabetic individuals, alcohol consumption, specifically gin, is known to predispose individuals to hypoglycemia (20, 21). This is corroborated by the findings of study 3, in which all 3 alcoholic beverages produced lower peak glucose concentrations than did water. None of the concentrations, however, were indicative of “true” hypoglycemia, defined as a plasma glucose concentration <3.0 mmol/L in these young healthy subjects. Interestingly, all the beverages tended to result in higher insulin concentrations, but only gin was statistically significant (38%). It is probable that the stepwise increase in plasma insulinemia, although not statistically significant, was related to the stepwise reduction in glycemia. This implies that the increased insulin response is effective in driving faster glucose uptake into the tissues. Last, although there are differences in alcohol metabolism between men and women (22), we found no significant effect of sex. Because the number of subjects of each sex was small, further studies with larger numbers of men and women are needed.

Pure alcohol acutely improves insulin sensitivity, as demonstrated by the euglycemic, hyperinsulinemic clamp technique (15, 23, 24) and the homeostatic model (6). In study 2, wine produced the greatest reduction in the postprandial glycemic response, despite contributing less alcohol than the gin (39 g compared with 47 g). This suggests the possibility that wine contains substances other than alcohol that are physiologically relevant. Wine’s acidity may slow down gastric emptying (25), and specific components may inhibit α-amylase or α-glucosidase activity and, therefore, the rate of starch digestion. Interestingly, a recent prospective observational study of ≈37,000 adults found wine consumption, but not beer or spirit consumption, to be associated with significantly lower risk of type 2 diabetes (19).

We conclude that alcoholic beverages consumed alone, with or before a carbohydrate-containing meal, are capable of reducing peak blood glucose concentrations or the overall postprandial glycemic response in young, lean, healthy subjects. In contrast, insulin concentrations were largely unchanged, which suggests an acute enhancement of glucose metabolism. These effects may provide a hitherto unrecognized benefit of moderate alcohol consumption for cardiovascular health.

The authors’ responsibilities were as follows—JCB-M: designed the study, obtained funding, and drafted the manuscript; KM, CM, and VL: recruited subjects, managed the testing sessions, and performed data entry; MB, FA, and PP: interpreted the data and performed the statistical analysis. All authors reviewed and commented on the manuscript. JCB-M serves on the board of directors of Glycemic Index Limited, a not-for-profit company that manages the Glycemic Index Symbol food labeling program in Australia (Internet: www.gisymbol.com.au); is the director of a glycemic index testing service at the University of Sydney (Internet: www.glycemincdex.com); and is the coauthor of a series of books under the general title The New Glucose Revolution (published by Marlowe and Co in North America), which explains the theory and practice of the glycemic index to the lay public. None of the other authors declared a conflict of interest.

REFERENCES
Erratum


On page 977, Table 3, an error exists in the third sentence of footnote 1. The sentence should read as follows: “Does not include 48 mg phosphorus, 10 μg vitamin D, 100 mg magnesium, or 10 μg vitamin K from multivitamin-minerals or salt from shaker.”

Erratum


The second author’s last name was misspelled. The correct spelling is “Fatema.”

Erratum


An incorrect e-mail address was provided for Angela M Zivkovic. The correct address is as follows: amzivkovic@ucdavis.edu.

Erratum


On page 211, footnote 2 to Table 1 is incorrect. It should read as follows: “± SD (all such values).”