Glycemic effects of moderate alcohol intake among patients with type 2 diabetes: 
A Multi-center, randomized clinical intervention trial

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Received for publication 11 June 2007 and accepted in revised form 8 September 2007.

Additional information for this article can be found in an online appendix at http://care.diabetesjournals.org.
Abstract

Introduction- In a randomized controlled trial, we assessed the effect of daily moderate alcohol intake on glycemic control in the fasting and postprandial states in patients with type 2 diabetes who previously had abstained from alcohol.

Methods- We randomized 109 alcohol abstaining patients (41–74 yrs old) with established type 2 diabetes to receive 150ml wine (13g alcohol) or non-alcoholic diet beer (control) each day during a three-month multi-center trial. The beverages were consumed during dinner. Diet and alcohol consumption were monitored.

Results- During the intervention, 17% of participants (12% from the alcohol group) dropped out, leaving 91 who completed the trial. Within the alcohol group, fasting plasma glucose (FPG) decreased from 139.6+/-41 to 118.0+/-32.5mg/dL after 3 months compared to 136.7+/-15.4 to 138.6+/-27.8mg/dL in the controls (Pv=0.015). However, alcohol consumption had no effect on 2-hour postprandial glucose levels (difference=18.5 in the control vs. 17.7mg/dL in the alcohol groups, Pv=0.97). Patients in the alcohol group with higher baseline HbA1c levels had greater reductions in FPG (age-adjusted correlation=-0.57, Pv<0.001). No significant changes were observed in the levels of bilirubin, alkaline phosphatase, ALT, or AST, and no notable adverse effects were reported. Participants in the alcohol group reported an improvement in the ability to fall asleep (Pv<0.001).

Conclusions- Among previous alcohol abstainers with type 2 diabetes, initiation of moderate daily alcohol consumption reduced FPG but not postprandial glucose. Patients with higher HbA1c may benefit more from the favorable glycemic effect of alcohol. Further intervention studies are needed to confirm the long-term effect of moderate alcohol intake.

ClinicalTrials.gov Identifier: NCT00295334
As summarized in a recent editorial, proving the beneficial effect of moderate alcohol intake awaits results of randomized controlled intervention trials. In observational studies, moderate alcohol intake is associated with lower incidence of type 2 diabetes, with an apparent J-shape association. Also, a recent meta-analysis of patients with type 2 diabetes suggests that moderate alcohol consumption is associated with a lower risk of mortality and coronary heart disease (CHD). Successful long-term control of hyperglycemia decreases diabetic complications, and is therefore a major goal in diabetes management. Ethanol metabolism increases the hepatic cytosolic NADH/NAD+ ratio that inhibit gluconeogenesis, a process that is elevated in type 2 diabetes, particularly when impairment of glucose homeostasis is advanced. The decline in hepatic glucose production can provoke hypoglycemia when alcohol is ingested in the fasting state. Since ethanol does not appear to directly affect insulin secretion or glucose disposal, a hypoglycemic effect of ethanol is likely to be highly dependent on nutritional state. Several small short-term studies, of 5-20 patients with type 2 diabetes, reported a decrease in plasma glucose concentrations with moderate alcohol consumption. However, other studies found no effect of alcohol on glycemic control.

Inhibition of hepatic glucose production is the major therapeutic effect of established anti-diabetic medications, such as metformin, so the potential impact of moderate alcohol consumption on glycemic control in diabetics remains intriguing, but unproven.

We therefore conducted a three-month multi-center randomized controlled intervention study of alcohol (150 ml wine; 13g alcohol/day) or a control nonalcoholic beer among 109 alcohol-abstainers with type 2 diabetes, and assessed the effect on fasting and postprandial glycemia.

Research Design and Methods

Study population

We enrolled patients from three diabetes units in academic medical centers in Israel (Hadassah Hebrew University Medical Center, Jerusalem; Wolfson Medical Center, Holon; and Soroka University Medical Center, Beer-Sheva). Inclusion criteria were: 1) Established diagnosis of type 2 diabetes, 2) Male or female alcohol abstainers (not more than 1 drink/week), 3) Age between 40–75 yrs, and 4) Clinically stable, with no history of stroke or myocardial infarction (MI) or major surgery within the previous 3 months. Exclusion criteria were: 1) More than 2 insulin injections/day or insulin pump therapy, 2) Triglycerides > 500 mg/dL, 3) HbA1c > 10%, 4) Serum creatinine > 2 mg/dl, 5) Liver dysfunction (greater than 2-fold elevation of ALT or AST), 6)
Evidence of severe diabetes complications (such as proliferative retinopathy or overt nephropathy), 7) Autonomic neuropathy manifested as postural hypotension or hypoglycemia unawareness, 8) Use of drugs that might significantly interact with alcohol such as sedatives, antihistamines, or anti-coagulants, 9) Presence of active cancer, or chemotherapy within the past 3 years, 10) Major illness that may require hospitalization, 11) A high potential for addictive behavior based on physician’s assessment or personal or family history of addiction, alcoholism, or alcohol abuse, 12) Pregnant or lactating women, or 13) Participation in another trial with active intervention.

The study was coordinated by the International Center for Health and Nutrition, Ben-Gurion University, Beer-Sheva, and was independently approved by the institutional review boards of each of the three medical centers. All volunteers gave written informed consent and did not receive compensation for their participation.

**Study design**

We screened 201 patients with type 2 diabetes, of whom 126 were eligible. Of these, we randomized 109 and 91 completed the study ([Online Appendix Figure 1](http://care.diabetesjournal.org)). The randomization design used a 2:1 ratio (intervention:control), to obtain better estimates of any adverse effects of the alcohol intervention. Participants met with the nurse study coordinator in the diabetes center on 8 occasions during the trial and with the physicians and the dietitians at weeks 1, 7, and 12 ([Online Appendix Table 1](http://care.diabetesjournal.org)). Three months after the end of the study, we interviewed participants who completed the alcohol arm by telephone to assess voluntary continuation of alcohol consumption, as well as adverse effects.

**Intervention**

All participants received individual dietary counseling by registered dietitians trained to work with type 2 diabetes patients. Each dietitian reinforced identical nutritional strategies to achieve glycemic control in both study groups, but did not specifically try to promote weight loss. Reinforcement of dietary counseling for both groups was based on the American Diabetes Association (ADA) recommendations for patients with type 2 diabetes which include 45–60% calories from carbohydrates, up to 30% from fat (with restriction of saturated fat to <7% of total calories and minimization of trans fat), and 15 to 20% from protein. Caloric intake was calculated according to age, gender, and level of physical activity. Based on these calculations, patients were instructed to consume an isocaloric diet. To compensate for the calories in the assigned beverages, the alcohol group was instructed to reduce carbohydrates by 100 kcal, but not at dinner, to decrease the likelihood of alcohol-induced
hypoglycemia. The control group was instructed to deduct 30 kcal from carbohydrates. Participants completed three-day food diaries and drink pattern questionnaires before each visit to enable the dietitians to monitor adherence to the diet and alcohol intake. Patients assigned to consume alcohol were instructed to start drinking gradually (over a 2 week period) 150 cc of wine (13% alcohol, 13g) that we provided, using a standard measured glass, during dinner. The patients could choose either dry red (Merlot) or white (Sauvignon Blanc) wine; 75% chose red wine. Participants randomized to the control group were instructed to drink 150 cc of the non-alcoholic diet malt beer we provided, using the same standard measured glass, during dinner. Every other week, the study coordinator provided either three bottles of wine (750 ml each) or two bottles of non-alcoholic diet malt beer (1.5 L each), after the return of empty bottles from the previous fortnight.

**Blood and clinical measurements**

Baseline and 12-week blood samples were drawn in the morning, after an 8-hour fast. All biochemical determinations were performed in the central laboratories of the medical centers using Olympus analytical equipment and reagents. Low Density Lipoprotein cholesterol (LDL-c) was calculated by Friedewald formula. HbA1c was determined using COBAS INTEGRA reagents and analytical equipment. A value below 5.8% is considered normal. Blood pressure was measured sitting, following 5 minutes of rest, using an Omron M41 digital apparatus. Waist circumference was measured half way between the last rib and the iliac crest. The patients were instructed to measure glucose, pre- and 2 hours post-prandial at dinnertime, three times weekly using their own self-glucose monitoring device. The glucometers used were Accutrend Sensor (Roche Diagnostics), Elite (Bayer Diagnostics) or Freestyle (Thera Sense, Alameda, CA).

**Statistical Analysis**

We used chi-square analyses to determine differences between categorical variables, and paired t-tests to compare changes in measurements within the two groups. In the main analyses, we compared the mean of the individual changes, from baseline to 12 weeks, in the two arms of the trial. We also calculated age-adjusted correlations and performed interaction tests between the intervention groups and strata of gender, median BMI, and median age. The levels of individual post-meal glucose represent an average of three reports, taken 2 hours after dinner in the same week. We compared the proportion of positive responders in both groups to the following question: “Do you think that, since the beginning of this study, the addition of the drink to your dinner was associated with an increase in the following symptoms/ adverse effects?” Statistical analyses were
performed using SPSS software (version 14.0).

**Results**

The randomized patients, 61 men and 48 women, ranged in age from 41 to 74 years, had an average FPG level of 144.5 mg/dL, HbA1c of 7.39%, blood pressure of 133.7/76.5 mm/Hg, and BMI of 30.1 kg/m². These characteristics were similarly distributed between the randomized groups, as were other parameters such as duration of the disease, smoking status, physical activity, and regular consumption of nutritional supplements, waist circumference, and years of education (Online Appendix Table 2). After randomization, but before the intervention began, 12 participants withdrew from the trial, 5 (7%) from the alcohol group and 7 (21%) from the control group. During the intervention, four additional participants withdrew from the alcohol group and two from the control group. The 18 patients who withdrew from the study (12% of the intervention and 26% of the control groups) were generally similar to the 91 who completed the study, but they had significantly higher baseline levels of FPG (167 vs. 140 mg/dL) and were younger (Online Appendix Table 3).

The individual changes in FPG and 2 hours post-meal glucose between baseline and at the end of the trial are shown in Figure 1. The alcohol group experienced a significant 9.2% decrease in FPG levels, dropping from 139.6+/−41 at baseline to 118.0+/−32.5 mg/dL after 3 months (Pv < 0.001), whereas there was no material change in FPG levels in the control group [136.7+/−15.4 at baseline and 138.6+/−27.8 mg/dL at week 12 (Pv = 0.783)]. The difference between the groups was significant (Pv = 0.015). The postprandial values represent an average of three self-measurements that were taken after dinner, at baseline, and during weeks 11–12. We observed nonsignificant increases in the 2 hour post-meal glucose levels of similar magnitude in both groups (18.5 in the control vs. 17.7 in the alcohol groups, P for difference = 0.97). Within the alcohol group, but not among controls (Figure 2), we found a significant inverse correlation between baseline levels of HbA1c and changes of FPG levels (age-adjusted correlation= -0.567, Pv < 0.001), suggesting that patients with type 2 diabetes with higher baseline HbA1c levels had greater reduction in FPG after moderate alcohol consumption. We found no modification of the alcohol effect by gender, age, BMI, or specific medical center (data not shown), though the statistical power to observe such interactions was limited.

We observed significant decreases (Table 2) in levels of HbA1C, LDL-c, and waist circumference in the alcohol group, and an unexpected significant reduction in HDL-c in the control group after 12 weeks compared to baseline levels. However, none of these changes...
differed significantly between the two groups. We found no significant changes in weight, blood pressure, or TGs among patients in either group, and no material changes in levels of bilirubin, alkaline phosphatase, ALT, or AST.

We elicited reports of symptoms (Online Appendix Figure 2) that participants attributed to the intervention. In the alcohol group, one woman dropped out because of gastric pain and 5% reported episodes of hypoglycemia, headaches, or muscle weakness, symptoms that were not reported in the control group. No other adverse effects were reported. Participants in the alcohol group (8%), but none in the control group, reported increased sexual desire. The only item that differed significantly was improved ability to fall asleep in the alcohol group as compared to controls (P < 0.001).

Three months after the study ended, 61% of the participants in the alcohol group reported that they thought that the alcohol was beneficial to them and 49% reported continuing to drink alcohol in moderation (frequency ranging from 1 drink a week to 1 drink a day). None reported an increase of the quantity of alcohol consumed.

Discussion

In the present randomized trial among patients with type 2 diabetes who previously abstained from alcohol, we showed that moderate alcohol consumption significantly decreased fasting, but not postprandial, glucose levels. Those with higher baseline HbA1c levels appeared to benefit more. Initiating moderate daily alcohol consumption in type 2 diabetic abstainers over age 40 caused no notable adverse effects or changes in liver function biomarkers during the three-month intervention.

Several limitations of the study warrant consideration. Neither participants nor the diabetes clinic staff could be blinded to the intervention (alcohol versus control non-alcoholic beverage), and though adherence was good, the dropout rates were not negligible. However, participants who dropped out had a generally similar clinical profile to those completing the study, and, in fact, two-thirds of the dropouts occurred immediately after the randomization, before the intervention began, and rates were higher among controls, suggesting that adverse effects of alcohol caused few if any dropouts. The 3-month intervention period, though longer than most alcohol trials, could not capture all the possible long-term adverse or beneficial effects of alcohol, limiting our ability to draw conclusions about the long-term risks and benefits. We believe our inclusion criteria (particularly age > 40 and screening for past addictive behavior) largely limited the danger of promoting alcohol addictive behavior. In a telephone interview three months after the end of the trial, all participants reported alcohol consumption of 1 drink a day or
less. The alcohol dose of 13 g/day may be a less than optimal to achieve maximal effects in patients with type 2 diabetes. Red and white wine presumably contain different amounts of polyphenols, possibly confounding the effects of the alcohol per se. Finally, we assayed fasting and post-meal glucose levels and HbA1C, but have no data on levels of insulin and glucagon, degree of insulin resistance, or hepatic glucose output. This limits our ability to dissect out the relevant importance of mechanisms mediating the alcohol-induced decrease in FPG, and the differential effects on FPG and PPG.

There are several strengths to this study: The number of participants is larger than most other alcohol intervention trials and adherence to the intervention protocols was high. Most importantly, the randomized trial design permitted assessment of the independent effect of initiating moderate alcohol consumption in abstainers. Nutritional counseling to both groups of participants was adjusted for the added calories, but did not introduce new dietary instructions aimed at promoting weight loss.

Moderate alcohol consumption has been associated with lower risk of cardiovascular disease and type 2 diabetes. The apparent beneficial effects for cardiovascular disease are likely mediated via effects on lipid metabolism, coagulation, fibrinolysis, and insulin sensitivity. We have previously shown that among over 700 men with type 2 diabetes, moderate alcohol intake was associated with decreasing levels of inflammatory biomarkers, (sTNFR-2, sICAM-1, fibrinogen) as well as elevated circulating levels of adiponectin. Prospective studies found an inverse relationship between alcohol consumption and diabetes incidence, with moderate drinkers having a 43% to 46% reduction in risk for diabetes compared with nondrinkers. In addition, alcohol is linked to lower cardiovascular risk among patients with type 2 diabetes.

In a recent meta-analysis of cohort studies among patients with diabetes, alcohol consumers had a 21–36% lower total mortality rate, and a 25–66% lower rate of total and fatal CHD than abstainers. The magnitude of these associations is stronger than in the general population.

Although beneficial effects of moderate alcohol consumption have been strongly suggested by observational studies, data from randomized trials of alcohol, especially among patients with type 2 diabetes, are sparse. In a randomized controlled crossover trial of 63 healthy postmenopausal women over 8 weeks, consumption of 30 g/d of alcohol (2 drinks per day) reduced insulin and triglyceride concentrations and improved insulin sensitivity in these nondiabetic women, but fasting glucose concentrations were not materially affected. In a trial
among patients with diabetes after a first myocardial infarction, red wine, taken with meals, significantly reduced oxidative stress and pro-inflammatory cytokines.

The major glycemic effect in our trial was a decrease in fasting, but not post-meal, plasma glucose levels. The mechanisms for this likely involve enhanced insulin secretion and the well-documented effect of alcohol metabolism, which, by increasing the hepatic cytosolic NADH/NAD+ ratio, inhibits gluconeogenesis, a process largely controlling fasting, rather than post-meal, glycemias. The non-significant increase of post-prandial glucose levels could be a consequence of an increase consumption of simple carbohydrates in the evening meal. The contribution of increased flux through the gluconeogenesis pathway to hyperglycemia is a characteristic of dysregulated glucose homeostasis in diabetes. Thus, it is plausible that patients with higher HbA1c have elevated gluconeogenic flux, and hence, exhibit more pronounced fasting hypoglycemic effect when started on moderate alcohol consumption. In the post-myocardial infarction trial cited earlier, fasting glucose levels were lower in the wine consumers, but levels of HbA1c did not differ, consistent with our findings showing a differential effect of wine on fasting versus post-prandial glucose (which was not measured in that trial).

In our study, patients in the alcohol group significantly reduced their waist circumference, LDL-c, and HbA1c levels, but these changes were not statistically significant when compared to the change in these parameters in the placebo group. Intriguingly, we observed that diabetics consuming 13 grams of alcohol daily for 3 months showed no increase in HDL. The likely explanations for this observation are related to the alcohol dose, duration, or to unique characteristics of the study population. Significant increase in HDL could be observed in healthy men as early as 17 days after initiating 40 g/day of alcohol. Alternatively, it is possible that the HDL elevating effect of alcohol is less readily detectable among diabetics, particularly when also treated with glucose and lipid-lowering medications. This notion is supported by observations made during a previously mentioned trial among post-MI diabetics, in which a significant increase in HDL was observed only after 9 months of alcohol intake (Marfella R, personal communication). Thus, in the diabetic population, alcohol apparently exerts a more rapid glucose-lowering effect, whereas the elevation in HDL requires more prolonged intervention. In doses shown in epidemiology to confer CVD and glycemic benefits, not all metabolic changes attributed to alcohol can be captured within 3 months in patients with type 2 diabetes.
Moderate alcohol and diabetes- randomized trial

Longer intervention studies are needed to determine the long-term efficacy and safety of initiating moderate alcohol intake among abstainers with type 2 diabetes, with assessment of clinical or intermediate outcomes.

Acknowledgments
We thank Tishbi Wines, Israel, and Admiral Wine Imports, US, for providing the wine for this study. We thank the following physicians, dietitians, nurses, and researchers for their valuable contribution to the study: Dr. Joseph Glassman, Dr. Mariella Glant, Orit Shemesh and Eti Abutbul of Hadassah Medical Center; Dr. Lea Chananshvili, Dr. Gila Dovinski, Dr. Lisy Ludmila, Naomi Mor, Tami Uzer, and Naomi Mevorach of Wolfson Medical Center; Dr. Tatiana Shuster, Dr. Natalya Shapiro, Dr. Idit Liberty, Dr. Max Mayzlus, and Shula Witkow of Soroka University Medical Center; Prof. Shimon Weitzman, Prof. Yaakov Henkin, Rachel Golan, and Osnat Tanji-Rozental of Ben-Gurion University.
References
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**Table 1. Levels of measurements before and after the 12-week alcohol intervention (n=91).**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Alcohol group</th>
<th>Control group</th>
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<tbody>
<tr>
<td></td>
<td>MEAN (SD)</td>
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<tr>
<td></td>
<td>Time 0</td>
<td>Time 12</td>
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<tr>
<td>Fasting plasma glucose, mg/dL</td>
<td>139.57 (41.04)</td>
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<td>2 hours post-meal glucose, mg/dL</td>
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<td>83.66 (15.54)</td>
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<td>Waist, cm</td>
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<td>7.07* (0.91)</td>
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<td>LDL-C, mg/dL</td>
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<td>85.11† (28.31)</td>
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<td>Bilirubin , mg/dL</td>
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1. p < 0.05
2. p < 0.01
3. p < 0.001
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<td>(3.79)</td>
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*Pv < 0.05, †Pv < 0.01 within group difference, paired t-test, as compared to Time 0
‡Pv < 0.05 between group differences, group t-test of deltas
Figure legends

Figure 1. Individual changes in fasting plasma glucose and 2 hours post-meal glucose after 12 weeks of moderate alcohol intervention among patients with type 2 diabetes

Figure 2. Correlations between baseline levels of HbA1c and change of fasting plasma glucose levels after 12 weeks of moderate alcohol intervention among patients with type 2 diabetes
**Figure 1**

The vertical lines indicate the mean value ± SD

**Fasting plasma glucose (FPG)**
- **Baseline** Control: 136.7 mg/dL, Alcohol: 138.6 mg/dL
- **Wk 12** Control: 139.6 mg/dL, Alcohol: 138.0 mg/dL

Delta = 1.92

Pv = 0.015

**2 Hour post-meal glucose (PMG)**
- **Baseline** Control: 135.6 mg/dL, Alcohol: 150.0 mg/dL
- **Wk 12** Control: 128.8 mg/dL, Alcohol: 146.5 mg/dL

Delta = 18.46

Pv = 0.966

Delta = 17.68
Figure 2.

Delta of FPG, mg/dL

HbA1c, %

*r* = -0.57
p<0.001

* Age adjusted correlation among the alcohol group