Alcohol and Postexercise Metabolic Responses in Type 2 Diabetes

Birthe M. Rasmussen, Christian Christiansen, Ole W. Rasmussen, Carsten Hansen, and Kjeld Hermansen

The objective was to investigate the impact of the combination of exercise and alcohol on the metabolic response in nonfasting and fasting type 2 diabetic subjects. In part 1, 12 untrained middle-aged type 2 diabetic subjects participated on 3 test days. On each day, they ingested a light meal (1,824 kJ) containing 48 energy percent (E%) carbohydrate, 38 E% fat, and 14 E% protein. The meal was followed by either (A) rest or (B) 30 minutes of exercise (40% of maximum O_2 consumption [VO_2max]) or (C) taken with alcohol (0.4 g/kg body weight) followed by 30 minutes of exercise (40% of VO_2max). In part 2, 11 untrained middle-aged type 2 diabetic subjects participated on 4 test days without a meal. The subjects were either (A) resting, (B) drinking alcohol (0.4 g/kg body weight), (C) exercising 30 minutes (40% of VO_2max), or (D) drinking alcohol (0.4 g/kg body weight) and exercising 30 minutes (40% of VO_2max). On each test day, regular blood samples were drawn for 4 hours for analysis of glucose, insulin, lactate, triglycerides, nonesterified fatty acid (NEFA), and ethanol. Comparing exercise and rest following a light meal (part 1, no change (7%) occurred in the plasma glucose response area (642 ± 119 *v* 724 ± 109 mmol · L^{-1} · 240 min, NS). However, it was significantly reduced (by 27%) in response to exercise and alcohol (509 ± 98 *v* 724 ± 109 mmol · L^{-1} × 240 min; $P = .03$). Similar serum insulin response areas were obtained. After exercise and alcohol, plasma lactate increased compared with the resting state (2.2 ± 0.2 *v* 1.6 ± 0.1 mmol · L^{-1}, $P = .004$) and with exercise alone (2.2 ± 0.2 *v* 1.8 ± 0.2 mmol ± L^{-1}, $P = .04$). Serum NEFAs were significantly reduced by exercise and alcohol compared with the resting state (0.50 ± 0.04 *v* 0.65 ± 0.06 mmol · L^{-1}, $P = .008$) and with exercise alone (0.50 ± 0.04 *v* 0.61 ± 0.05 mmol · L^{-1}, $P = .02$). Similar serum triglycerides were found. During the fasting state (part 2), similar plasma glucose response areas were obtained in the four situations. The insulin response area to exercise and alcohol increased significantly compared with the resting state (3,325 ± 744 *v* 882 ± 295 pmol · L^{-1} × 240 min, $P = .02$) and with exercise alone (3,325 ± 744 *v* 1,328 ± 422 pmol · L^{-1} × 240 min, $P = .007$). No difference was found compared with alcohol alone. Plasma lactate was higher after alcohol intake versus the resting state (1.9 ± 0.1 *v* 1.3 ± 0.1 mmol · L^{-1}, $P = .003$), as well as after exercise and alcohol (1.9 ± 0.1 *v* 1.3 ± 0.1 mmol · L^{-1}, $P = .01$). After exercise and alcohol serum NEFAs were significantly reduced compared with the resting state (0.43 ± 0.02 *v* 0.64 ± 0.02 mmol · L^{-1}, $P < .001$), alcohol alone (0.43 ± 0.02 *v* 0.51 ± 0.02 mmol · L^{-1}, $P < .001$), and exercise alone (0.43 ± 0.02 *v* 0.64 ± 0.02 mmol · L^{-1}, $P < .001$). Serum triglycerides were similar in the four situations. We conclude that moderate exercise with or without moderate alcohol intake does not cause acute hypoglycemia either after a light meal or in the fasting state in untrained overweight type 2 diabetic subjects.

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HYPOGLYCEMIA is a well-known and feared condition in type 1 diabetes. The risk of hypoglycemia is increased by exercise following subcutaneous insulin administration, which leads to hyperinsulinemia and impaired hepatic glucose production combined with increased glucose clearance.[1] Other factors important to the development of hypoglycemia are alcohol, which blocks gluconeogenesis through an effect on the redox state of the liver,[2] and fasting and starvation accompanied by depletion of hepatic glycogen stores.[3] How exercise and alcohol in combination with and without food intake influence plasma glucose in type 2 diabetic subjects is unknown. The potential role of exercise in type 2 diabetic subjects is primarily to increase insulin sensitivity.[4,5] Even exercise of short duration has been found to reduce the average postprandial plasma glucose level in type 2 diabetes.[6,7] Consequently, regular exercise is recommended as an integral part of the management in addition to pharmacologic treatment[8] and/or diet.[9]

Alcohol consumption accounts for 4% to 6% of the total energy intake and plays an important role in the diet in most Western countries.[10] However, our knowledge regarding the influence of alcohol on carbohydrate metabolism in type 2 diabetic subjects is limited. We have previously shown that moderate alcohol consumption has no acute influence on plasma glucose despite an alcohol-induced increase in insulin levels.[11-13] Alcohol may thus influence insulin resistance in type 2 diabetic subjects, although we have been unable to detect any acute change in insulin sensitivity as judged by the euglycemic-hyperinsulinemic clamp technique during intravenous alcohol administration.[14] Since overweight nondiabetic subjects are refractory to alcohol-induced hypoglycemia,[15-17] overweight type 2 diabetic subjects may also be less prone to develop alcohol-induced hypoglycemia.

Information on how to deal with alcohol and exercise in combination in type 2 diabetic subjects is not available. The present experiments were thus undertaken to determine whether type 2 diabetic subjects acutely develop hypoglycemia when combining exercise and alcohol intake. In part 1, type 2 diabetic subjects were given a light meal; in part 2, no meal was provided.

SUBJECTS AND METHODS

Subjects

Apart from background retinopathy, none of the subjects had any signs of diabetic complications or any history of liver disease or alcohol abuse. None were treated with β-blockers. The diabetes duration was longer than 1 year. The participants had endogenous insulin production with fasting serum C-peptide levels of 357 ± 32 pmol · L^{-1} (range, 194

From the Department of Endocrinology and Metabolism, Aarhus Amtssygehus, Aarhus University Hospital, Aarhus; and Department of Forensic Toxicology, Aarhus University, Aarhus, Denmark.

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Address reprint requests to Kjeld Hermansen, MD, DMSc, Aarhus University Hospital, Aarhus Amtssygehus, Tage-Hansens Gade 2, DK-8000 Aarhus C, Denmark.

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to 693). All subjects were recruited from our outpatient clinic and were fully informed of the experimental nature of the investigation, which had approval from the local ethics committee. Informed consent was obtained from each participant before the study began. All subjects participated in the studies with full compliance to the protocol.

Part 1 was performed in 12 untrained type 2 diabetic subjects (eight men and four women). They were aged 56 ± 3 years and overweight, with a body mass index (BMI) of 28.8 ± 1.0 kg/m^2, and had a diabetes duration of 8 ± 2 years. They were fairly well regulated, with a hemoglobin A_{1C} (HbA_{1C}) of 8.0% ± 0.4% and a fasting plasma glucose on three different occasions of 9.4 ± 0.9, 8.9 ± 0.8, and 9.1 ± 0.6 mmol · L^{-1}, respectively. Five subjects were treated with diet alone, two with sulfonylureas, and five with a combination of sulfonylureas and metformin.

Part 2 was performed in 11 untrained type 2 diabetic subjects (eight men and three women). They were aged 53 ± 3 years and overweight, with a BMI of 27.2 ± 1.0 kg/m^2, and had a diabetes duration of 7 ± 1 years. They had a HbA_{1c} of 8.1% ± 0.4% and a fasting plasma glucose in the four different situations of 9.8 ± 1.0, 9.5 ± 0.8, 9.1 ± 0.8, and 9.5 ± 0.9 mmol · L^{-1}, respectively. Three subjects were treated with diet alone, five with sulfonylureas, one with metformin, and two with a combination of sulfonylureas and metformin.

Experimental Protocol

Part 1: Exercise and alcohol with a light meal. The subjects participated on 3 study days, A, B, and C, each lasting 4 hours, with a minimum interval of 1 week. No medication was taken during the previous 12 hours. On study day A, maximum oxygen consumption (VO_2max) was estimated at the end of the study day. Subsequently, the subjects randomly participated in study days B and C. At 8 AM after an overnight fast (10 hours), the participants on each day ingested a light meal (1,824 kJ) consisting of 100 g white bread, 40 g ham, and 20 g low-fat margarine, corresponding to 48 energy percent (E%) carbohydrate, 14 E% protein, and 38 E% fat. The meal was taken with 175 mL plain water and consumed within 15 minutes (minutes 0 to 15).

On study day A, the participants rested throughout the period after the meal. On study B, they exercised from minutes 30 to 60 (40% of VO_2max, 150 to 350 kpm/min) followed by rest. On study day C, they drank ethanol (0.4 g/kg body weight) dissolved in 175 mL water with the meal (0 to 15 minutes) and exercised as on study day B. Blood samples were collected from the cubital vein every 15 minutes from 8 to 9 AM, every 30 minutes to 10 AM, and every hour to 12 noon. Analyses were performed for glucose, insulin, lactate, ethanol, nonesterified fatty acids (NEFAs), triglycerides, HbA_{1c}, and fasting C-peptide. The samples for glucose and lactate were mixed with sodium fluoride, which inhibits glycolysis, and analyzed immediately, and serum from the other samples was stored at −20°C for later analysis. The urine was collected from 8 AM to 12 noon to calculate urinary glucose loss.

A 6-minute submaximal exercise test with continuous monitoring of the heart rate was performed on a bicycle ergometer (Monark Electronic Ergometer 829 E; Monark Exercise, Varberg, Sweden). The workload was 375 to 1,125 kpm/min, and the steady-state heart rate during the last 2 minutes of work was used for calculation of VO_2max using the Åstrand protocol.[18] In a group comparable to ours, the indirect measure of VO_2max has been shown to correlate well with VO_2max determined by direct measurements, with a coefficient of variation (CV) less than 10%.[19,20]

Part 2: Exercise and alcohol without a meal. The subjects participated on 4 study days, A, B, C, and D, each lasting 4 hours, with a minimum interval of 1 week. No medication was taken during the preceding 12 hours. On study day A, VO_2max was estimated according to the method of Åstrand.[18] The subjects randomly participated in study days B, C, and D. At 8 AM after an overnight fast (10 hours), the participants on each day drank 175 mL plain water within 15 minutes (minutes 0 to 15). On study days A and B, the subjects rested throughout the period. On study days C and D, they exercised from minutes 30 to 60 (40% of VO_2max, 200 to 450 kpm/min) followed by rest. On study days B and D, ethanol (0.4 g/kg body weight) was dissolved in 175 mL water. Blood samples were taken and urine was collected as in part 1.

Analytical Methods

The serum insulin level was measured by an enzyme-linked immunosorbent assay using a commercial kit (DAKO Diagnostics, Cambridgeshire, UK), with a range of 5 to 600 pmol · L^{-1} (CV 5-9%).[21] Plasma glucose and lactate levels were measured on the test day by an oxidase method using a YSI Model 2300 Stat Plus glucose and lactate analyzer (YSI, Yellow Springs, OH; CV glucose, 0.7%; CV lactate, 1.4%).[22]

The HbA_{1c} level was measured by a commercial kit (Bio-Rad, Richmond, CA), with a range of 3.5% to 5.5% (CV 5%). The serum ethanol level was measured by gas chromatography using the headspace technique (CV 1-2%).[23] Serum triglyceride and the NEFA levels were measured by standard enzymatic colorimetric assays using commercial kits (Boehringer, Mannheim, Germany and Wako Chemicals, Neuss, Germany; CV 4% and 1.1%, respectively). C-peptide levels were measured by an enzyme-linked immunosorbent assay using a commercial kit (DAKO; CV 5.1%).[24] Urinary glucose levels were measured by the glucose oxidase-peroxidase reaction (Diabur Test 5000; Boehringer).

Statistical Methods

In part 1, the data fulfilled the assumption of normality and homogeneity of variance, and ANOVA for repeated measurements followed by Student's *t* test for paired samples were used.[25] In part 2, the data did not fulfill the assumptions and were analyzed with Friedman's two-way ANOVA followed by Wilcoxon's test for paired samples. To avoid overinterpretation, further calculations were only made when ANOVA showed a significant difference between the groups. The statistical software used was SPSS (SPSS, Chicago, IL). Results are given as the mean ± SEM. A P level less than .05 is considered statistically significant.

Plasma glucose and serum insulin responses are expressed as the incremental area above the fasting level, the area under the curve (AUC). This is calculated according to the trapezoid rule, where values below the fasting levels are ignored.[26] The fasting levels are the values measured at 8 AM. Lactate, NEFA, and triglyceride concentrations are expressed as the mean for the entire study period. Serum ethanol is expressed as the peak value. The nadir glucose is calculated as the mean of the lowest value for each subject during the 4 hours.

RESULTS

Part 1: Exercise, Alcohol, and a Light Meal

Fasting levels of glucose, insulin, lactate, NEFA, and triglycerides (not shown) were similar on the 3 test days (Fig 1). Comparing exercise and rest, no change in plasma glucose (7% ± 18%) occurred (642 ± 119 *v* 724 ± 109 mmol · L^{-1} × 240 min; NS). Exercise and alcohol suppressed plasma glucose by 27% ± 14% (509 ± 98 *v* 724 ± 109 mmol · L^{-1} × 240 min, $P = .03$). However, no difference was found between exercise and exercise and alcohol (642 ± 119 *v* 509 ± 98 mmol · L^{-1} × 240 min; NS), as well as in the nadir glucose levels in the three situations (8.7 ± 0.8 rest, 8.0 ± 0.8 exercise, and 8.2 ± 0.7 mmol · L^{-1} exercise and alcohol, respectively). Similar insulin responses were obtained in the three situations (16,547 ± 3,834, 12,340 ± 2,581, and 14,689 ± 2,799

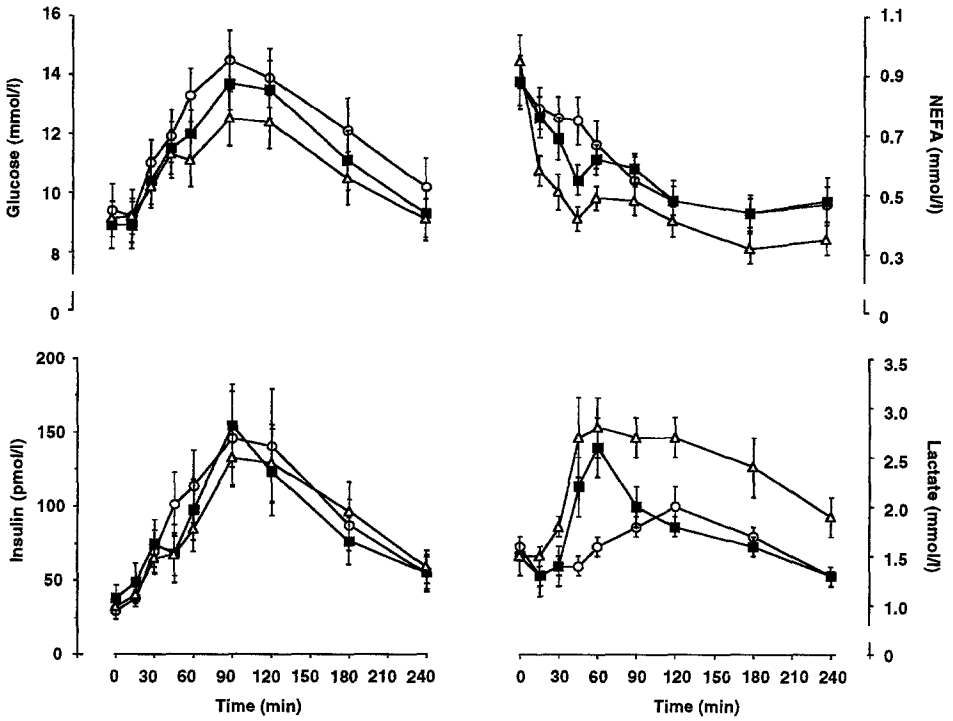

Fig 1. Postprandial profiles of glucose, insulin, lactate, and NEFA in 12 NIDDM subjects after rest (○), exercise (■), and exercise and alcohol (△). Data are the mean ± SE.

pmol · L^{-1} × 240 min, respectively). The plasma lactate response to exercise and alcohol increased significantly compared with the resting state (2.2 ± 0.2 *v* 1.6 ± 0.1 mmol · L^{-1}, $P = .004$) and with exercise alone (2.2 ± 0.2 *v* 1.8 ± 0.2 mmol · L^{-1}, $P = .04$). Serum NEFAs were significantly reduced after exercise and alcohol compared with the resting state (0.50 ± 0.04 *v* 0.65 ± 0.06 mmol · L^{-1}, $P = .008$) and with exercise alone (0.50 ± 0.04 *v* 0.61 ± 0.05 mmol · L^{-1}, $P = .02$). No differences were found in serum triglycerides (3.1 ± 0.6, 2.5 ± 0.4, and 2.5 ± 0.4 mmol · L^{-1}, respectively). Urinary glucose output was similar on the 3 study days (36 ± 15, 32 ± 16, and 35 ± 14 mmol/240 min, respectively). Serum ethanol was undetectable at fasting and peaked at 10.9 ± 1.0 mmol · L^{-1} within 30 minutes (Fig 2A). VO_2max was 24 ± 1 mL/kg/min.

Part 2: Exercise and Alcohol Without a Meal

Fasting levels of glucose, insulin, lactate, NEFA, and triglycerides (not shown) were similar on the 4 test days (Fig 3). Plasma glucose response areas were similar after rest, rest and alcohol, exercise, and exercise and alcohol (47 ± 30, 50 ± 22, 54 ± 29, and 22 ± 8 mmol · L^{-1} × 240 min. respectively). No difference was found in nadir glucose values (8.3 ± 0.7 rest, 7.8 ± 0.8 rest and alcohol, 7.8 ± 0.7 exercise, and 7.5 ± 0.7 mmol · L^{-1} exercise and alcohol, respectively). The insulin response increased to exercise and alcohol compared with the

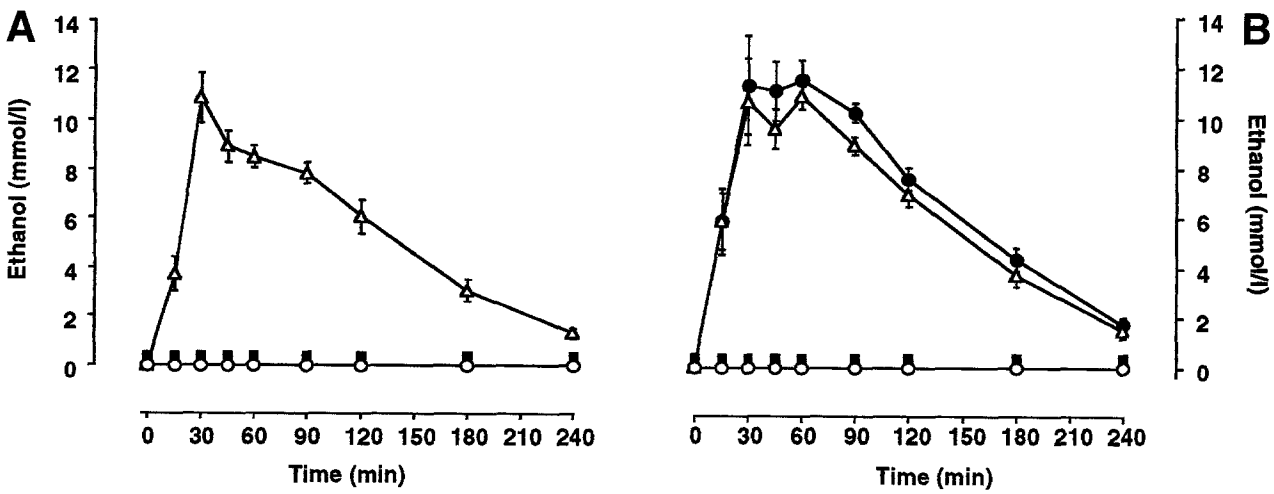

Fig 2. (A) Postprandial profiles of ethanol in 12 NIDDM subjects after rest (○), exercise (■), and exercise and alcohol (△). (B) Fasting profiles of ethanol in 11 NIDDM subjects after rest (○), rest and alcohol (●), exercise (■), and exercise and alcohol (△). Data are the mean ± SE.

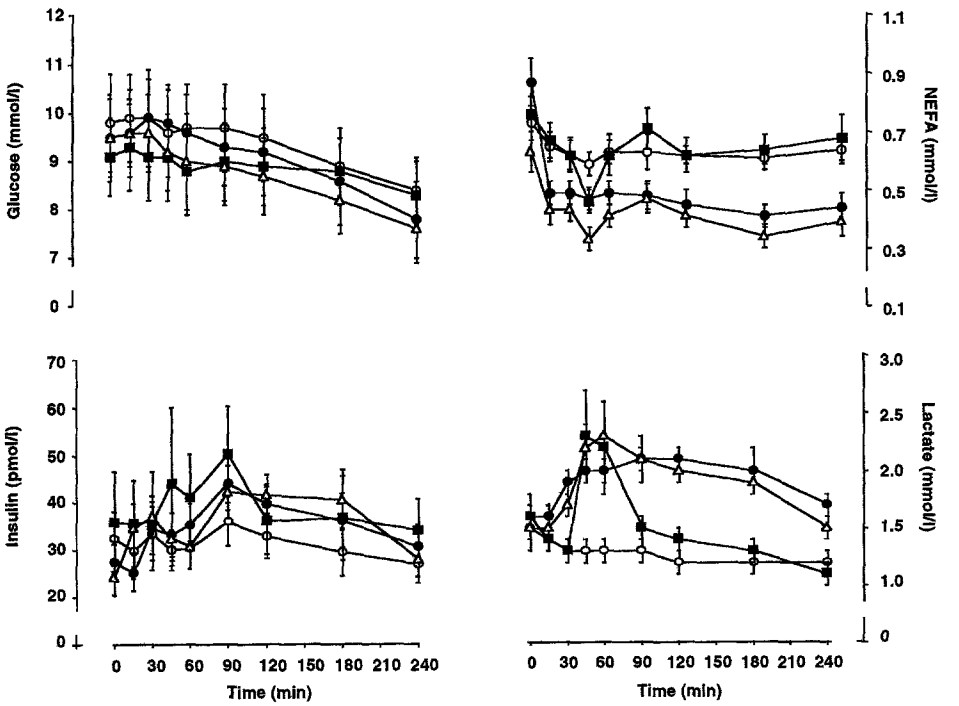

Fig 3. Fasting profiles of glucose, insulin, lactate, and NEFA in 11 NIDDM subjects after rest (○), rest and alcohol (●), exercise (■), and exercise and alcohol (△). Data are the mean ± SE.

resting state (3,325 ± 744 *v* 882 ± 295 pmol · L^{-1} × 240 min, $P = .02$) and with exercise (3,325 ± 744 *v* 1,328 ± 422 pmol · L^{-1} × 240 min, $P = .007$). No difference was found compared with alcohol alone (2,267 ± 589 pmol · L^{-1} × 240 min). Plasma lactate increased significantly after alcohol intake compared with the resting state (1.9 ± 0.1 *v* 1.3 ± 0.1 mmol · L^{-1}, $P = .003$) and after exercise and alcohol compared with the resting state (1.9 ± 0.1 *v* 1.3 ± 0.1 mmol · L^{-1}, $P = .01$). Plasma lactate was significantly lower to exercise versus alcohol (1.6 ± 0.1 *v* 1.9 ± 0.1 mmol · L^{-1}, $P = .04$) and exercise and alcohol (1.6 ± 0.1 *v* 1.9 ± 0.1 mmol · L^{-1}, $P = .03$). Serum NEFA decreased significantly after the combination of exercise and alcohol compared with the resting day (0.43 ± 0.02 *v* 0.64 ± 0.02 mmol · L^{-1}, $P < .001$), alcohol (0.43 ± 0.02 *v* 0.51 ± 0.02 mmol · L^{-1}, $P < .001$), and exercise (0.43 ± 0.02 *v* 0.64 ± 0.02 mmol · L^{-1}, $P < .001$). Serum NEFA was also reduced after alcohol compared with the resting day (0.51 ± 0.02 *v* 0.64 ± 0.02 mmol · L^{-1}, $P < .001$) and the exercise day (0.51 ± 0.02 *v* 0.64 ± 0.02 mmol · L^{-1}, $P < .001$). Serum triglycerides were similar in the four situations (2.2 ± 0.4, 2.3 ± 0.4, 2.1 ± 0.3, and 2.2 ± 0.3 mmol · L^{-1}, respectively).

Urinary glucose output was similar on the 4 study days (15 ± 10, 11 ± 7, 12 ± 7, and 15 ± 9 mmol/240 min, respectively). Serum ethanol was undetectable at fasting and attained similar peak values after alcohol alone and alcohol and exercise (11.5 ± 0.8 and 10.9 ± 0.5 mmol · L^{-1}, respectively), reached within 30 minutes (Fig 2B) VO_2max was 26 ± 2 mL/kg/min.

DISCUSSION

In the present study, acute hypoglycemia did not appear in untrained middle-aged overweight type 2 diabetic subjects in response to moderate exercise[27] with or without alcohol intake. This was found irrespective of the participants having a light meal or not together with an alcohol amount corresponding to two to three glasses of wine.

The reason we did not find any greater change in plasma glucose and serum insulin to exercise alone may be ascribed in part to the moderate intensity and/or short duration of exercise or to a preexisting prominent insulin resistance, which may counteract any potential hypoglycemic impact. The intensity of exercise must be defined relative to the individual's maximal capacity for aerobic exercise if it is to have relevance to the metabolic response. VO_2max is a measure of the highest rate of ATP generation via oxidative phosphorylation that an individual can obtain during exercise of progressively increasing intensity. VO_2max differs greatly between individuals. Our participants were overweight, middle-aged, and untrained, with a VO_2max of about 24 to 26 mL/kg/min, which is low compared with the VO_2max in highly trained young male athletes, as high as 80 mL/kg/min.[18] However, we anticipate that the exercise challenge is realistic, reflecting the actual physical capacity in this group of sedentary overweight subjects. Exercise is known to increase insulin sensitivity and to decrease plasma glucose in normal and type 2 diabetic subjects.[7,28,29] Improved insulin sensitivity to exercise disappears 3 to 5 days after the last exercise bout.[30,31] Consequently, it is unlikely that there could be any carryover effect from 1 study day to another in our investigation, with at least a 1-week interval. In the absence of carbohydrate in the intestine, plasma glucose is maintained by glycogenolysis of hepatic glycogen stores, and by hepatic gluconeogenesis during exercise. The major gluconeogenic precursors during exercise are lactate derived primarily from the breakdown of skeletal muscle glycogen, glycerol provided by hydrolysis of triglycerides, and alanine that is transaminated

to pyruvate.[32] The reason the plasma glucose level in our type 2 diabetic subjects was relatively constant despite the exercise may be the considerable hepatic glycogen stores. Thus, during mild to moderate exercise, the plasma glucose level stays remarkably constant in healthy men until liver glycogen stores are depleted.[33,34] We have previously shown that alcohol causes a dose-related increase in insulin despite unchanged glucose levels in type 2 diabetic subjects.[11] Since ethanol impairs insulin sensitivity in normal subjects,[35] the increased insulin levels in type 2 diabetic subjects (Fig 3) may be due to insulin resistance. However, we have previously been unable to detect any acute alcohol-induced deterioration of insulin sensitivity in resting type 2 diabetic subjects as estimated by the euglycemic-hyperinsulinemic clamp.[14] The initial insulin level to exercise and alcohol increases by approximately 50%, which is puzzling since the increase can be ascribed neither to alcohol nor to exercise per se. We are unable to explain this phenomenon, which may reflect an artifact with a significant impact on the calculated AUC. Consequently, the presence of increased insulin levels to exercise and alcohol should be considered with caution until further investigation.

Accelerated gluconeogenesis accounts for 90% of the increase in basal glucose production in type 2 diabetic subjects, mainly through augmented fractional extraction of lactate and other gluconeogenic precursors.[36] Alcohol inhibits gluconeogenesis via an increase in the ratio of NADH to NAD, which favors the reduction of pyruvate to lactate. This results in a decreased pyruvate availability for gluconeogenesis, and the change in the hepatic redox state also decreases gluconeogenesis from glycerol and amino acids. A maximal inhibition of gluconeogenesis of 66% has been observed in perfused rat liver at an ethanol concentration of 10 mmol $\cdot$ L^{-1}.[37] An ethanol concentration of 12 mmol $\cdot$ L^{-1} in type 2 diabetic subjects has been found to subdue gluconeogenesis from lactate by 71%, from glycerol by 65%, and from alanine by 75%.[38] The maximal ethanol values obtained in the present study of 11 to 12 mmol $\cdot$ L^{-1} correspond to levels that exert a prominent effect on gluconeogenesis. Despite a considerable alcohol-induced suppression of gluconeogenesis, Puhakainen et al[38] found no concomitant suppression of hepatic glucose output in type 2 diabetes. Therefore, one may argue that the question in the present study is whether the insulin-independent exercise-induced increase in muscle glucose uptake and/or the postexercise increase in peripheral insulin sensitivity result in an increased peripheral glucose clearance rate and subsequent hypoglycemia. Previously, we have demonstrated that an amount of alcohol similar to that in the present study taken with a light meal had no acute influence on plasma glucose in type 2 diabetic subjects.[13] In the fasting state, alcohol caused no change in either plasma glucose or serum insulin levels. This may be explained by the fact that alcohol is converted independently of insulin to acetate, which is only partly oxidized in the liver but can be utilized in the peripheral tissues without conversion to carbohydrate. Consequently, our results are consistent with the results of Puhakainen et al,[38] who suggested a regulatory mechanism in the liver maintaining a constant total hepatic glucose output despite inhibition of gluconeogenesis. The addition of ethanol to a light meal before 30 minutes of exercise reduced the postprandial glucose response by 27%. It seems unlikely that middle-aged overweight subjects should be able to exercise to an extent that would eliminate the postprandial glucose response or even cause suppression of plasma glucose levels. During fasting conditions, plasma glucose levels appeared constant irrespective of exercise, alcohol intake, or the combination of both. Although our participants did not receive oral hypoglycemic medication on the study days, residual effects of the different medical treatments may still be present and may, to some extent, have influenced the hepatic as peripheral parameters. However, our number of participants is too small to allow for analyses of the different responses among subgroups. Thus, it cannot be ruled out that concomitant oral hypoglycemic medication, a larger alcohol amount, or longer fasting period may suppress blood glucose levels. To answer these questions, additional experiments must be performed.

As expected, we found an increased lactate response to alcohol, exercise, and exercise and alcohol. This may partly be due to the impaired gluconeogenesis caused by the alcohol-induced increase in the conversion of pyruvate to lactate. During a single bout of acute exercise, insulin-mediated glucose disposal of the working muscles is enhanced in type 2 diabetic subjects.[39] However, glucose oxidation is impaired in type 2 diabetics, leading to a disproportionately high conversion of glucose to lactate, which causes an increase in plasma lactate and an acceleration of Cori cycle activity.[40]

NEFA decreased after alcohol intake, corroborating our previous studies.[11,13,14] This is a consequence of the increase in the NADH/NAD ratio favoring triglyceride accumulation in the liver. Alcohol also decreases circulating NEFA, due to the antilipolytic effect of acetate.[41] In contrast to the precise regulation of hepatic glucose output and plasma glucose levels during exercise, the lipolysis of triglycerides from the adipose tissue, the release of NEFA to the blood, and the circulating NEFA levels are matched to a lesser extent to energy demands. During the 30-minute exercise, NEFA initially decreased. This phenomenon may be explained in part by a decrease in NEFA release from the adipose tissue, due to increased NEFA reesterification secondary to the augmented levels of lactate.[42] It is also possible that vasoconstriction in adipose tissue suppresses NEFA transport from this tissue.[43]

The observation that the moderate amount of alcohol did not alter triglyceride levels is in line with our previous findings.[13,14] This does not exclude the possibility that long-term consumption or greater amounts of alcohol may elevate circulating triglycerides.

In conclusion, moderate exercise with or without the intake of a moderate amount of alcohol and with or without a light meal does not cause acute hypoglycemia in overweight sedentary type 2 diabetic subjects. However, it should be emphasized that this does not rule out the possibility that greater alcohol amounts and a prolonged fast may elicit late hypoglycemia in type 2 diabetic subjects treated with insulin or sulfonylureas, especially after more extensive and/or prolonged exercise.

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