

Effects of one single bout of low-intensity exercise on postprandial lipaemia in type 2 diabetic men

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Fighting type 2 diabetes and its high risk of CVD, lifestyle intervention with diet and exercise is of uttermost importance. Epidemiological studies strongly suggest an inverse association between increased physical activity, moderate alcohol drinking and the incidence of both type 2 diabetes and CVD. However, alcohol is known to increase postprandial lipaemia, a risk marker of CVD, and exercise to reduce postprandial lipaemia in healthy individuals. The aim of the present study was to investigate how type 2 diabetic men respond, in the postprandial period, to a single exercise session feasible to perform on a daily basis for type 2 diabetic men. The twelve participants ingested a test meal containing 100 g butter, 50 g carbohydrate, together with 40 g alcohol, at each meal test, imitating a social meal situation. Two protocols included exercise sessions with 40 min at 40% $V_{O_{2max}}$, one where they exercised 3.5 h after, and another the afternoon before the test meal. One protocol was without any exercise. No significant effect of low-intensity exercise on postprandial lipaemia following a fat-rich meal with alcohol was seen in the middle-aged type 2 diabetic men.

Exercise: Physical activity: Diabetes mellitus: Postprandial lipaemia: Ethanol

Type 2 diabetes is characterised by insulin resistance, and is associated with a breakdown of lipid homeostasis reflected by elevated levels of circulating NEFA and triacylglycerols in the fasting and postprandial period, low HDL-cholesterol and a preponderance of small, dense LDL. All these factors contribute to an increased risk of CVD in type 2 diabetes (Fraze *et al.* 1985; Chen *et al.* 1993; Isomaa *et al.* 2001; Taskinen, 2003). Exaggerated postprandial lipaemia has been linked to arteriosclerosis (Patsch *et al.* 1992; Ryu *et al.* 1992; Boquist *et al.* 1999; Karpe, 1999). The exaggerated triacylglycerol response in type 2 diabetes seems partly to derive from an increased substrate delivery of NEFA and glycerol from the adipose tissue to the liver and an increased hepatic secretion of VLDL particles due to extrahepatic and hepatic insulin resistance (Malmstrom *et al.* 1997). In addition to fasting triacylglycerol levels, diet, current smoking and alcohol consumption have been shown to influence postprandial lipid responses (Parks, 2001; Sharrett *et al.* 2001; Thomsen *et al.* 2003).

Alcohol in moderate amounts also seems to have a beneficial effect on the risk of developing both type 2 diabetes and CVD. The Health Professionals' follow-up study showed that the regular intake of moderate alcohol amounts is associated with a reduced relative risk of developing type 2 diabetes by 50% (Conigrave *et al.* 2001). Also, drinking on at least 3 or 4 d/week is associated with a decrease in the relative risk of myocardial infarction by 37% (Mukamal *et al.* 2003). The relative risk of cardiovascular heart disease in

type 2 diabetic men who consumed alcohol was reduced by 40% compared with non-drinkers (Tanasescu *et al.* 2001).

Despite the cardioprotective effect observed in large-scale epidemiological studies, it is well known that acute alcohol intake increases postprandial lipaemia in healthy individuals (Veenstra *et al.* 1990; Pownall, 1994; van Tol *et al.* 1995; Hendriks *et al.* 1998; Fielding *et al.* 2000).

Regular physical activity is an important lifestyle factor associated with a reduced incidence of both CVD and type 2 diabetes. Bicycling to work is associated with a reduction in mortality (Andersen *et al.* 2000), and physical activity for 30 min or more on a daily basis compared with 0 min has been shown to reduce the relative risk of type 2 diabetes by 36% (Hu *et al.* 2003). Type 2 diabetic women who engaged in moderate exercise for 4 h or more/week had a relative risk of a cardiovascular event of 0.55 compared with diabetic women who exercised for less than 1 h/week (Hu *et al.* 2001). Also, the relative risk of CVD in type 2 diabetic men with low fitness was found to be twice that of fit diabetic men (Wei *et al.* 2000). Some investigators have shown that one single bout of exercise less than 24 h before a meal can reduce postprandial lipaemia (Schlierf *et al.* 1987; Tsetsonis & Hardman, 1996; Tsetsonis *et al.* 1997; Gill *et al.* 2002; Petitt *et al.* 2003). Few studies have compared how exercise influences postprandial lipaemia in response to alcohol intake (Hartung *et al.* 1993; Rasmussen *et al.* 1999; El Sayed & AL-Bayatti, 2001; Suter *et al.* 2001).

Most of the literature concerning postprandial lipaemia and exercise includes healthy individuals engaged in moderate exercise for long periods (Petitt & Cureton, 2003). Little is known about the influence of low-intensity exercise and alcohol on postprandial lipaemia in diabetic individuals, which is of relevance to describe in relation to future health recommendations regarding exercise and alcohol intake. The objective of the present study was to determine the acute effects of an exercise stimulus that would be realistic to perform on a daily basis on the postprandial responses in type 2 diabetic men when their metabolic system was challenged by alcohol and fat.

Subjects and methods

Study group

Fourteen type 2 diabetic men were included in the present study; two participants left the study, because of their job situation. The mean duration of type 2 diabetes for the participants was 3.3 (SD 2.2) years. The mean age of the twelve participants was 59.3 (SD 10.4) years, BMI 27.9 (SD 2.8) kg/m², and waist circumference 1040 (SD 59) mm. The screening level of HbA_{1c} was 7.2 (SD 1.3) %; the fasting triacylglycerol level was 2.8 (SD 2.0) mmol/l. The mean $V_{O_{2max}}$ was low (28 (SD 6) ml/kg per min). With the exception of four participants receiving sulfonylurea, the participants did not receive anti-diabetic drugs. The participants continued their regular medication during the whole study. All participants consumed alcohol on a weekly basis with a range of average weekly intake of three to thirty-five drinks (one drink containing 12 g alcohol). Three were smokers, smoking twenty cigarettes/d.

The participants were selected from outpatients of the diabetes clinic at Aarhus University Hospital, Aarhus, Denmark. The local ethics committee of Aarhus County approved the study protocol. The subjects were fully informed of the experimental nature of the study and gave their written, informed consent to participate. Inclusion criteria were: men with type 2 diabetes for more than 1 year, diagnosed by their general practitioner or at the hospital. Exclusion criteria included: uncontrolled diseases, severe heart disease (New York Heart Association classification 3 or 4) or renal insufficiency (blood creatinine over 150 µmol/l) or treatment with insulin, β-blockers or lipid-lowering medication.

Exercise

At the screening visit a sub-maximal exercise test, using the Åstrand (1960) protocol, with continuous monitoring of the heart rate was performed on a bicycle ergometer (Monark Electronic Ergometer 829 E; Monark Exercise, Varberg, Sweden). The workload, in conjunction with the resultant heart rate, was compared with the predicted relationship, adjusted for age and sex, and the $V_{O_{2max}}$ was computed. The steady-state heart rate during the last 2 min of work was used for the calculation of $V_{O_{2max}}$. In a comparable group the indirect measures of $V_{O_{2max}}$ have been shown to correlate well with $V_{O_{2max}}$ determined by direct measurements, with a CV of less than 10 % (Lindgarde & Saltin,

1981; Berntorp *et al.* 1986). In protocols B and C (see later) the participants exercised at 40 % $V_{O_{2max}}$ for 20 min followed by 20 min rest before the last 20 min of exercise. The workload during exercise ranged from 32.7 to 89.9 W. Mean workload was 59 (SD 21) W. We did not measure heart rate continuously during the exercise sessions.

Design

The study consisted of three protocols, A, B and C, each carried out on separate days with at least 1 week apart (Fig. 1). The participants took part on an outpatient basis in random order in the present cross-over study, each participant serving as his control. Each protocol started at 08.00 hours the day before a meal test and ended at 08.00 hours the morning after a meal test, i.e. a total of 48 h. During the initial 24 h of each protocol (day 1) the participants ingested food delivered by the dietitian. The level of physical activity during the initial 24 h differed between the protocols. On the second day of each protocol basal fasting blood samples and postprandial responses to a fat-rich test meal and different levels of physical activity were measured. Each protocol ended on the third morning at 08.00 hours on which final fasting blood samples were taken. In protocol A, participants rested both during day 1 and day 2 (control). In protocol B, participants rested during day one and exercised 3.5 h after the test meal. In test protocol C, participants exercised on the afternoon on day 1, and rested on day 2.

Interventions

Nutritional supplement. The participants ingested a high-carbohydrate diet delivered by the dietitian, with an energy distribution of 50 % carbohydrate, 32 % fat and 18 % protein on the first day of each protocol. The food amount corresponded to individual energy requirements estimated by the Harris–Benedict equation with adjustment for activity (Pellett, 1990). After the meal test, a meal was served at the investigation site and a food pack for the rest of the day was delivered ensuring that total energy ingested on day 2 was similar to the energy ingested on day 1; however, the energy distribution was not similar due to the high content of fat in the test meal.

Test conditions. At each meal test the same meal with alcohol was served at T = 0 min. The meal consisted of an energy-free soup with 100 g butter (80 g fat) plus 40 g alcohol and 50 g carbohydrate as white bread. The meal contained mainly saturated fatty acids (72 % of total fat). The soup was chilled briefly and then butter was added. Sliced raw leek was added to give taste. The meal test ended at T = 480 min. The morning after each meal test, the participants arrived after an 11 h fast and delivered fasting blood samples (T = 1440 min).

Exercise sessions. No exercise was performed in protocol A. Exercise in 2 × 20 min at 40 % $V_{O_{2max}}$ was performed 210 ± 21 min after T = 0 in protocol B, and in protocol C the same amount of exercise was performed during the afternoon about 16 h before T = 0.

The participants were instructed to standardise and minimise their physical activity 3 d before the protocols started.

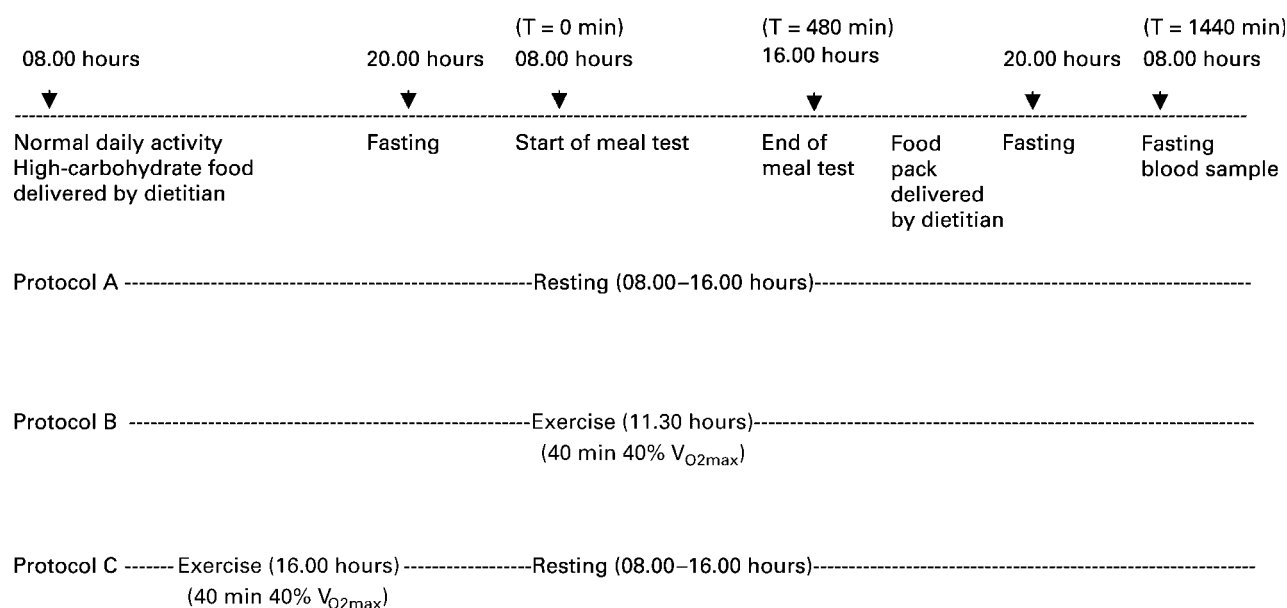


Fig. 1. Study flow chart. Test meal of soup with 100 g butter plus 40 g alcohol plus 50 g carbohydrate.

No alcohol drinking or heavy physical work was allowed 24 h preceding each protocol. No smoking or intake of medication was allowed from day 1 in the protocols. On the second day, following an 11 h fast, the subjects arrived between 07.00 and 07.30 hours using a minimum of physical activity. Subsequently a catheter was placed in an antecubital vein and basal blood samples were drawn 15 min later (baseline period). The meal test started at T = 0 min and the meal was ingested during a 20 min period. The participants were allowed to walk freely in the ward during the meal test. Blood samples were drawn every 1 h the first 4 h and thereafter at T = 360 min and T = 480 min for analysis of glucose, insulin, fatty acids, triacylglycerol, total cholesterol and HDL-cholesterol. Plasma was immediately separated by centrifugation at 2000 g for 20 min at 4°C and kept frozen at -20°C until analysed.

Bioanalysis

Plasma glucose was measured by a glucose oxidase method (CV 3.8%) (MPR 3 166 391 glucose/GOD-PAP method; Boehringer Mannheim, Mannheim, Germany) (Trinder, 1969). Serum insulin concentrations were measured by an ELISA method (CV 1.7%) (Andersen *et al.* 1993). Serum triacylglycerols (CV 2.05%), serum cholesterol (CV 1.9%), serum HDL-cholesterol (CV 3.9%) (code no. 701912, 2016630, 543004, respectively; Roche Diagnostics GmbH, Mannheim, Germany), and serum fatty acids (CV 1.1%) were measured with standard enzymic colorimetric assays by using commercial kits (code no. 994-75409; Wako Chemicals GmbH, Neuss, Germany).

Statistical analysis

Results are expressed as mean values and standard deviations of individually analysed data for the twelve participants. For paired comparisons between fasting values, area

under the curve (AUC), incremental AUC (iAUC) and one-way repeated measures of ANOVA were used. Whenever data were not normally distributed, the Friedman repeated measures ANOVA on ranks was used. In case of statistical differences between these groups, a Student–Newman–Keuls *post hoc* test was used. Significance was assessed by two-tailed tests (SigmaStat version 2.03; BMDP Statistical Software, Berkeley, CA, USA). $P < 0.05$ was considered statistically significant. The trapezoidal rule was used to calculate the AUC and iAUC for glucose, insulin, NEFA, total and HDL-cholesterol and triacylglycerol. The iAUC is calculated from the summation of the mean concentration for each time period after subtraction of the mean basal value, multiplied by the number of minutes in the time period. Values below baseline were calculated as negative. The iAUC derived expresses both the duration and the magnitude of the responses, while correcting for baseline values. Insulin sensitivity was calculated, for each of the three protocols, by the homeostasis model assessment model for the fasting values at T = 0 min and T = 1440 min (Matthews *et al.* 1985; Haffner *et al.* 1996). Insulin resistance was calculated as follows:

$$\text{Insulin resistance} = (\text{fasting insulin } (\mu\text{U/ml}) \times \text{fasting glucose (mmol/l)}) / 22.5.$$

Results

Basal values, areas under the curve and incremental areas under the curve

Twelve type 2 diabetic men completed the present study. Weight decreased slightly during the study period from 91.6 (SD 12.2) to 90.8 (SD 12.3) kg at the end of the study ($P = 0.045$).

No differences between fasting values at T = 0 for each of the three protocols (Table 1) were seen except for the

Table 1. Basal fasting glucose, insulin and lipids in twelve type 2 diabetic men for each of the protocols* (Mean values and standard deviations)

	Protocol A		Protocol B		Protocol C		P value (comparisons between protocols)
	Mean	SD	Mean	SD	Mean	SD	
Plasma glucose (mmol/l)	9.3	2.7	8.9	2.0	9.2	2.4	0.92
Serum insulin (pmol/l)	43.1	35.5	45.2	37.5	49.4	49.9	0.53
Serum triacylglycerol (mmol/l)	2.6	1.6	2.4	1.6	2.7	2.5	0.21
Serum NEFA (mmol/l)	0.7	0.2	0.7	0.2	0.7	0.2	0.57
Serum cholesterol (mmol/l)	6.0	1.0	5.7	0.7	5.9	0.9	0.25
Serum HDL-cholesterol (mmol/l)	1.07 ^a	0.29	0.88 ^b	0.36	0.97 ^{ab}	0.26	0.02

^{a,b} Mean values within a row with unlike superscript letters were significantly different ($P = 0.02$)

* In protocol A, participants did not exercise; in protocol B, participants rested the day before the meal test and exercised for 40 min at 40% V_{O2max} on the day of the meal test; in protocol C, patients exercised for 40 min at 40% V_{O2max} the day before the meal test.

lower values of HDL-cholesterol for protocol B compared with protocol A (Table 1).

Triacylglycerols. No significant differences between the three protocols were detected (Tables 1, 2 and 3). Triacylglycerol levels after 8 h were increased by about 75% compared with the fasting values (Fig. 2).

Non-esterified fatty acids. No differences between protocols were observed for NEFA (Tables 1, 2 and 3).

Cholesterol responses. Fasting HDL-cholesterol was higher at $T = 0$ in protocol A compared with protocol B (Table 1). No significant differences in AUC or iAUC for total cholesterol or HDL-cholesterol between the three protocols were detected (Tables 2 and 3).

Glucose, insulin and insulin sensitivity. No significant differences in iAUC of glucose and insulin between the three protocols were detected (Table 2). The AUC for insulin on meal test C was significantly higher than the two other days (Table 3). No significant differences in insulin sensitivity, measured by the homeostasis model assessment model, were found (Table 4).

Discussion

In the present study, we did not detect any immediate impact on postprandial lipaemia by an amount of exercise that we estimated realistic in daily living for semi-sedentary

Table 2. Incremental areas under the curve (iAUC) of glucose, insulin and lipids following a test meal in twelve type 2 diabetic men for each of the protocols*

(Mean values and standard deviations)

	Protocol A		Protocol B		Protocol C		P value (comparisons between protocols)
	Mean	SD	Mean	SD	Mean	SD	
iAUC glucose (mmol/l × 480 min)	202	535	73	555	74	609	0.42
iAUC insulin (pmol/l × 480 min)	31 563	17 079	28 640	19 417	34 513	21 626	0.56
iAUC triacylglycerol (mmol/l × 480 min)	541	332	589	537	650	443	0.21
iAUC fatty acids (mmol/l × 480 min)	-75	94	-69	92	-67	96	0.85
iAUC cholesterol (mmol/l × 480 min)	-77	72	-20	89	-24	115	0.07
iAUC HDL-cholesterol (mmol/l × 480 min)	-83	84	9	134	-32	89	0.08

* Test meal consisted of soup plus carbohydrate (50 g) plus alcohol (40 g) plus butter (100 g). In protocol A, participants did not exercise; in protocol B, participants rested the day before the meal test and exercised for 40 min at 40% V_{O2max} on the day of the meal test; in protocol C, participants exercised for 40 min at 40% V_{O2max} the day before the meal test.

Table 3. Areas under the curve (AUC) of glucose, insulin and lipids following a test meal in twelve type 2 diabetic men for each of the protocols*

(Mean values and standard deviations)

	Protocol A		Protocol B		Protocol C		P value (comparisons between protocols)
	Mean	SD	Mean	SD	Mean	SD	
AUC glucose (mmol/l × 480 min)	4658	1432	4347	1170	4493	1183	0.2
AUC insulin (pmol/l × 480 min)	51 518 ^a	31 204	50 320 ^a	35 736	58 233 ^b	42 865	0.03
AUC triacylglycerol (mmol/l × 480 min)	1803	1023	1741	1261	1932	1595	0.72
AUC fatty acids (mmol/l × 480 min)	267	76	252	66	257	88	0.52
AUC cholesterol (mmol/l × 480 min)	2783	480	2726	340	2830	480	0.72
AUC HDL-cholesterol (mmol/l × 480 min)	432	117	433	103	435	102	0.37

^{a,b} Mean values within a row with unlike superscript letters were significantly different ($P = 0.03$).

* Test meal consisted of soup plus carbohydrate (50 g) plus alcohol (40 g) plus butter (100 g). In protocol A, participants did not exercise; in protocol B, participants rested the day before the meal test and exercised for 40 min at 40% V_{O2max} on the day of the meal test; in protocol C, participants exercised for 40 min at 40% V_{O2max} the day before the meal test.

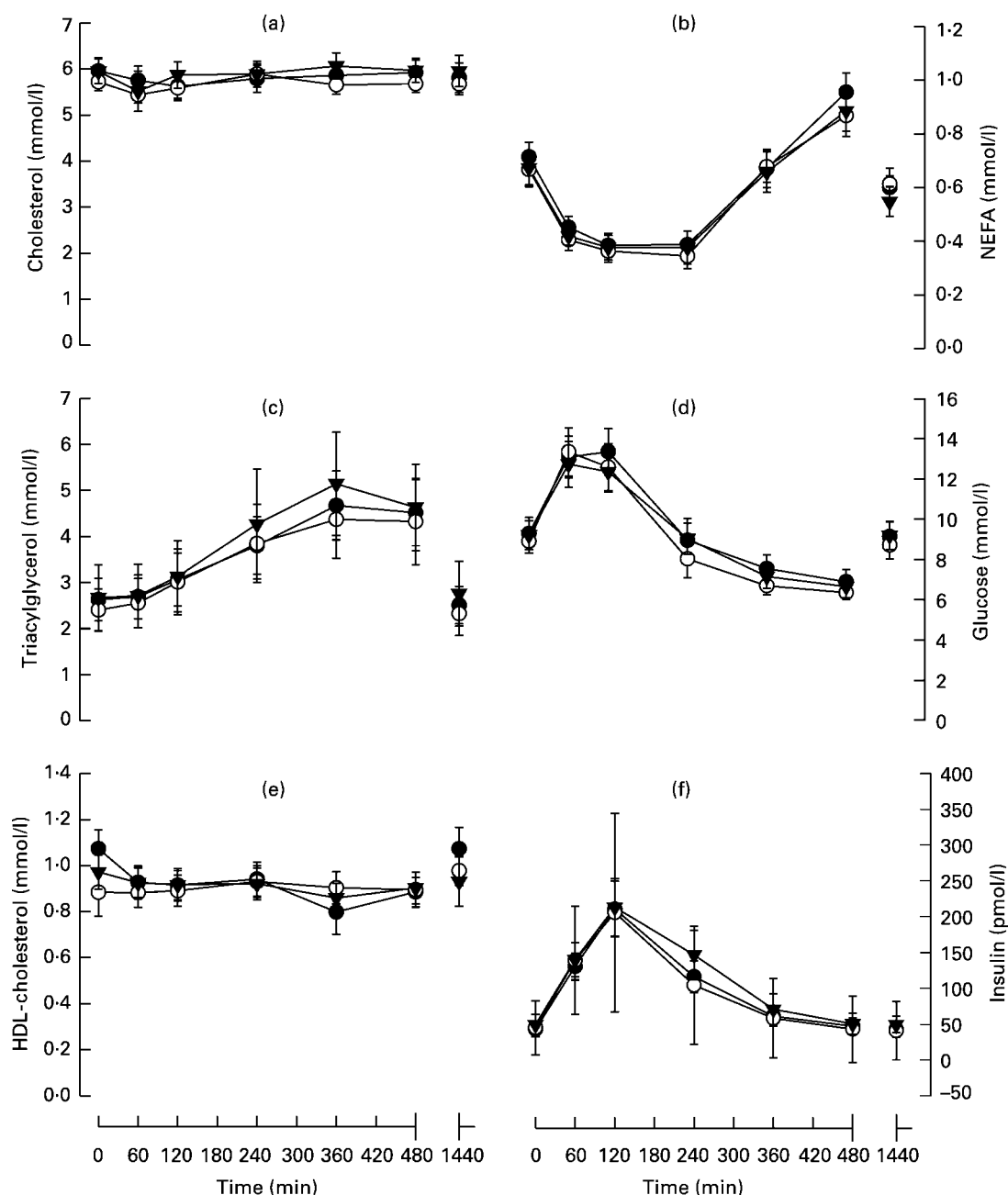


Fig. 2. Serum postprandial cholesterol (a), NEFA (b), triacylglycerols (c), plasma glucose (d), HDL-cholesterol (e) and insulin (f) in twelve type 2 diabetic men following a meal of soup with 100 g butter plus 40 g alcohol plus 50 g carbohydrate. Protocol A (●) participants did not exercise. Protocol B (○) participants rested the day before the meal test and exercised for 40 min at 40% $V_{O_{2max}}$ on the day of the meal test. Protocol C (▼) participants exercised for 40 min at 40% $V_{O_{2max}}$ the day before the meal test and rested during the meal test. Values are means, with standard errors of the mean represented by vertical bars.

type 2 diabetic men. This contrasts with previous studies on exercise and postprandial lipaemia in healthy subjects (Tsetsonis & Hardman, 1996; Gill *et al.* 2001). In the latter studies, however, a much higher exercise intensity and duration was chosen than in the present study. We decided to examine a less intensive exercise since large-scale epidemiological studies have shown that light and moderate daily exercise have a striking effect on the risk of CVD (Hu *et al.* 2001), and of type 2 diabetes (Hu *et al.* 2003). According to the present acute study, with all its limitations, the cardioprotective effect of light exercise

seems not to be mediated through a reduction in postprandial lipaemia. To be able to separate the effects of alcohol and exercise, studies using a similar amount of exercise on a meal test without alcohol should have been carried out. Although Danes do not habitually consume 40 g alcohol in the morning, it is quite common on festive occasions.

In the Insulin Resistance Atherosclerosis Study, physical activity was found to be positively associated with increased insulin sensitivity (Mayer-Davis *et al.* 1998) and one single bout of glycogen-depleting exercise in type 2 diabetic patients increased the non-oxidative glucose

Table 4. Insulin resistance using the homeostasis model assessment (HOMA) following a test meal in twelve type 2 diabetic men for each of the protocols*

(Mean values and standard deviations)

	Protocol A		Protocol B		Protocol C		P value (difference between protocols)
	Mean	SD	Mean	SD	Mean	SD	
HOMA, T=0 min	2.9	2.3	3	2.4	3.2	3.4	0.98
HOMA, T = 1440 min	2.8	2.3	2.6	2.6	3.2	2.6	0.08

* Test meal consisted of soup plus carbohydrate (50 g) plus alcohol (40 g) plus butter (100 g). In protocol A, participants did not exercise; in protocol B, participants rested the day before the meal test and exercised for 40 min at 40% $V_{O_{2max}}$ on the day of the meal test; in protocol C, participants exercised for 40 min at 40% $V_{O_{2max}}$ the day before the meal test. HOMA was calculated as mean (fasting insulin (μ U/ml) \times fasting glucose (mmol/l))/22.5.

disposal for 16 h as measured by the insulin clamp technique (Devlin *et al.* 1987). The present study did not show any effect on insulin sensitivity, measured by the homeostasis model assessment model.

AUC for insulin were higher in protocol C; however, the higher basal insulin levels in protocol C as compared with protocols A and B may explain this. Thus, when adjusted for basal levels, the iAUC did not differ between the three protocols.

Exercise for 45 min at 53% $V_{O_{2max}}$ carried out 45 min after a mixed meal significantly reduced the 4 h postprandial AUC for glucose and insulin in type 2 diabetic patients (Larsen *et al.* 1997). The explanation might be that their patients consumed a carbohydrate-rich meal and exercised 45 min after breakfast, when glucose and insulin levels were increasing, whereas our participants consumed a high-fat meal and exercised 3.5 h after the meal, when glucose and insulin levels were decreasing, and most of the ingested triacylglycerol was absorbed. It could be that exercising 45 min rather than 3.5 h after a meal more effectively reduces the postprandial glucose responses. There were two reasons for exercising our participants after 3.5 h. First, we wanted the blood ethanol to reach a level where the participants could cooperate properly in the exercise session. Second, we wanted a postprandial absorption that was similar for meal tests with and without exercise. Some studies have shown that moderate exercise increases gastric emptying (Moore *et al.* 1990), probably due to increased gastric blood flow.

It has been argued that abnormal NEFA metabolism is essential for the insulin resistance syndrome (McGarry, 2002). In lean type 2 diabetic patients (Groop *et al.* 1989), impairment of both the insulin suppression of NEFA and the turnover of NEFA was found as compared with age- and weight-matched controls.

We did not detect any differences in NEFA. In contrast, Rasmussen *et al.* (1999) found that the NEFA levels in type 2 diabetic patients were reduced by 30 min exercise at 40% $V_{O_{2max}}$ 30 min after a test meal with alcohol compared with a sedentary day without alcohol. Previously, we have found that 40 g ethanol is a suppressor of NEFA in type 2 diabetic patients (Dalgaard *et al.* 2004). The difference in both the timing of the exercise and the fact that the participants in the present study ingested ethanol on all meal tests may explain the contrasting results to the study of Rasmussen *et al.* (1999).

Regular alcohol consumption in moderate doses is associated with improved insulin sensitivity in non-diabetic

subjects (Kiechl *et al.* 1996; Lazarus *et al.* 1997; Flanagan *et al.* 2000). Evidence is conflicting regarding alcohol and insulin sensitivity in those with type 2 diabetes. Insulin sensitivity did not change in type 2 diabetic patients in response to ethanol given intravenously (Christiansen *et al.* 1996) whereas the oral intake of 12 and 24 g alcohol with carbohydrate pointed to an alcohol-induced aggravation of insulin resistance (Christiansen *et al.* 1993). It is possible that the amount of alcohol in the present study induced enhanced insulin resistance in the postprandial phase, counteracting the effect of exercise on the postprandial lipaemia. No significant effect of low-intensity exercise on postprandial lipaemia following a fat-rich meal with alcohol was seen in the middle-aged type 2 diabetic men.

Lifestyle intervention programmes have shown that both a moderate intake of alcohol and exercise have beneficial effects on the risk of developing type 2 diabetes and CVD. Evaluation of the long-term effects of regular alcohol drinking and graded exercise load on the postprandial lipaemia in type 2 diabetic individuals needs further investigation.

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