Effects on Blood Pressure, Glucose, and Lipid Levels of a High-Monounsaturated Fat Diet Compared With a High-Carbohydrate Diet in NIDDM Subjects

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OBJECTIVE — To compare the influence on blood pressure, glucose, and lipid levels of a diet rich in monounsaturated fatty acids with an isocaloric, high-carbohydrate diet in 15 NIDDM subjects.

RESEARCH DESIGN AND METHODS — A crossover design with diet interventions and wash-out periods of 3 wk was applied. The patients were randomly assigned to a 3-wk treatment with a high-carbohydrate diet containing 50% of energy as carbohydrate and 30% of energy as fat (10% of energy as monounsaturated fatty acids) or an isocaloric diet with 30% of energy as carbohydrate and 50% of energy as fat (30% of energy as monounsaturated fatty acids). On the last day of the two diets, 24-h ambulatory blood pressure was measured and day profiles of glucose, hormones, and lipids were performed to a test menu rich in carbohydrates.

RESULTS — The diet rich in monounsaturated fat reduced daytime systolic (131 ± 3 vs. 137 ± 3 mmHg, $P < 0.04$) and 24-h systolic blood pressure (126 ± 8 vs. 130 ± 10 mmHg, $P < 0.03$) as well as daytime diastolic (78 ± 2 vs. 84 ± 2 mmHg, $P < 0.02$) and diurnal diastolic blood pressure (75 ± 6 vs. 78 ± 5 mmHg, $P < 0.03$) as compared with the high-carbohydrate diet. Evidence of lowered blood glucose levels on the high-monounsaturated diet compared with the high-carbohydrate diet were found with lower fasting blood glucose (6.1 ± 0.3 vs. 6.8 ± 0.5 mM, $P < 0.05$), lower average blood glucose levels (7.4 ± 0.5 vs. 8.2 ± 0.6 mM, $P < 0.04$), and peak blood glucose responses (9.9 ± 0.6 vs. 11.3 ± 0.7 mM, $P < 0.02$). The two diets had the same impact on lipid levels.

CONCLUSIONS — A diet rich in monounsaturated fat has beneficial effects on blood pressure and glucose metabolism, whereas no adverse effects on lipid composition in NIDDM subjects is detected.

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NIDDM, non-insulin-dependent diabetes mellitus; CHO, carbohydrate; TG, triglyceride; MUFA, monounsaturated fatty acids; BP, blood pressure; sBP, systolic blood pressure; dBP, diastolic blood pressure; FFA, free fatty acid; GH, growth hormone; RIA, radioimmunoassay; ANOVA, analysis of variance; LDL, low-density lipoprotein; HDL, high-density lipoprotein; FBG, fasting blood glucose.

Current dietary recommendations for diabetic subjects prescribe reducing total fat and saturated fat and replacing the fat with complex CHOs (1–3). However, the advantages of substituting a high-CHO, low-fat diet for a low-CHO, high-fat diet in NIDDM subjects have been questioned (4–8). Thus, an increased intake of CHO has been found to increase TG levels (5,8), which in NIDDM is strongly related to the development of cardiovascular complications (9,10). Recent studies of NIDDM indicate that diets rich in MUFA did not elicit deleterious effects on the CHO and lipid metabolism (7) or even had beneficial effects (6). This is especially noteworthy considering the data from epidemiological studies in the Mediterranean area showing a lower incidence of CVD (11) and a favorable influence on BP in areas where people consume high levels of MUFA in their diets (12). Although NIDDM and hypertension are often associated, little is known about the effects of MUFA on the BP.

The primary question under consideration was whether a high intake of MUFA would alter the BP in NIDDM patients. In this study we compare the effects of two diets—one rich in MUFA and the other rich in complex CHO—on clinical and 24-h ambulatory BP, CHO, lipid, and lipoprotein levels.

RESEARCH DESIGN AND METHODS

— Fifteen NIDDM subjects (10 men, 5 women) participated in the study (Table 1). None of the subjects had diabetic complications except background retinopathy, 8 were treated with diet alone, and 7 received additional oral antidiabetic drugs (7 sulfonylureas and 4 sulfonylureas and metformin). They took the prescribed medicine throughout the study, and no change in dosage was allowed. Oral antidiabetic drugs were the only medical drugs taken. The study had approval from the local ethical committee of Aarhus County.

Table 1—*Clinical characteristics of 5 women and 10 men with NIDDM*

Age (yr)	Diabetes duration (yr)	BMI (kg/m^2)	Body weight (kg)	FBG(mM) Before high-CHO diet	FBG(mM) Before high-MUFA diet	HbA_{1c} (%) Before high-CHO diet	HbA_{1c} (%) Before high-MUFA diet
57 ± 2	6 ± 1	27.0 ± 1.1	80.5 ± 3.8	8.4 ± 0.9	8.4 ± 0.6	7.0 ± 0.4	7.1 ± 0.3

Data are means ± SE.

Experimental protocol

A crossover study was conducted on outpatients. After a 2-wk run-in period, 7 participants were randomly allocated to a 3-wk treatment with a high-CHO diet and 8 patients with a high-MUFA diet. After a 3-wk wash-out period, the participants received the alternative diet for 3 wk. The calorie content of the diets was calculated individually according to the 4 days' diet records in the run-in period and was adjusted weekly throughout the study to maintain constant body weight in the study periods. For each patient, the two diets were isocaloric with a mean energy content of 8.9 ± 0.6 MJ/day. Fructosamine, HbA_{1c}, and lipoproteins were measured before and on the last day of the two diet periods. Clinical auscultatory and oscillometric BPs were measured on the last day of the two diets; the 24-h ambulatory BP equipment was also applied at this time. Furthermore, the patients collected a 24-h urine sample.

24-h ambulatory BP. Ambulatory BP was measured by means of a portable automatic monitor (SpaceLabs model 90202, Redmond, WA), which has been recommended for clinical use (13) and previously evaluated in a NIDDM population (14). This was conducted on one of the last 3 days during the 3-wk treatment with the high-CHO and the high-MUFA diets. The equipment measures BP by oscillometry and was programmed for cuff insufflation every 20 min from 0600 to 2400 and every hour during the night. The average of three oscillometric and auscultatory measurements are termed the clinic oscillometric and the clinic auscultatory BPs, respectively. The auscultatory measurements were recorded with a random zero sphygmomanometer (Hawskley, Lancing, UK) and a normal size cuff (120 × 225 mm) using Korotkoff V as diastolic pressure.

Day profiles. To evaluate the long-term effects on glucose and fat metabolism of the two diets we measured day profiles of glucose, insulin, glucagon, TGs, FFAs, and GH during a high-CHO test menu (8–16 h) of 3720 KJ (2 meals and 2 snacks). This was conducted at the last day during the 3-wk treatment with the high-CHO and high-MUFA diet.

During the high-MUFA diet period we also looked at the metabolic effects of an isoenergetic test diet (3720 KJ) with a high-MUFA content being ingested as 2 meals and 2 snacks between 0800 and 1600. This was conducted 3 days preceding the high-CHO test diet (see above), and the results were compared. Samples for blood glucose analysis were collected every 15 min from 0730 until 0900, every 30 min from 0900 to 1000, and then hourly until 1600.

The samples for glucose, insulin, glucagon, GH, and FFAs were stored at −20°C until assayed. A 24-h urine sample was collected concomitantly with the 24-h BP measurement and was analyzed for glucose, carbamide, potassium, calcium, and creatinine.

Dietary interventions

Before the study began and during the last week of the diet interventions, the participants weighed and recorded their foods for 3 working days and 1 weekend day to estimate the energy intake and composition. The food records were coded by the dietician and the nutrient content was calculated by the computer program Dankost (Dansk Catering Center, Herlev, Germany) (15). The nutrient composition is given in Table 2. The prescribed high-CHO diet provided 50% of energy as CHO and 30% as fat (10% MUFA); the prescribed high-MUFA diet provided 30% as CHO and 50% as fat (30% MUFA).

The MUFA diet was based on the patient's food records, and CHOs (bread, potatoes, rice) were reduced and exchanged with MUFA. No butter and margarine were allowed during the MUFA diet, and olive oil (cold pressed virgin olive oil, Elanthy, Piraeus, Greece) was used as the main MUFA source in the high-MUFA diet (provided by Cervera A/S, Rodding, Denmark). Special rolls and meat dishes containing 5–6 g and 30 g of olive oil were prepared and supplied frozen. Apart from olive oil 10–20 g of almonds or nuts were allowed daily, but no avocados were used. Olive oil contributed 29% of total energy intake during the high-MUFA diet period. The high-CHO diet was enriched mostly by bread, potatoes, and rice. Apart from the food, subjects continued their habitual eating pattern and activities of daily living.

The participants visited the dietician every week and were weighed without shoes, jackets, and sweaters. Their food scheme was reviewed and the calorie intake adjusted if body weight dif-

Table 2—*Composition of the habitual and the intervention diets of 15 NIDDM subjects according to dietary records*

	Diet		
	Habitual	High-CHO diet	High-MUFA diet
Protein (% of energy)	16 ± 1	17 ± 1	14 ± 1
Fat (% of energy) total	40 ± 2	32 ± 2	50 ± 1
Saturated fat	15 ± 1	11 ± 1	10 ± 0
Monounsaturated fat	14 ± 1	11 ± 1	30 ± 1
Polyunsaturated fat	8 ± 1	7 ± 1	7 ± 0
Glycerol	3 ± 0	3 ± 0	3 ± 0
CHO (% of energy) total	40 ± 2	49 ± 2	36 ± 1
Simple sugars	10 ± 1	9 ± 1	8 ± 1
Polysaccharides	29 ± 2	39 ± 1	28 ± 1
Alcohol (% of energy)	4 ± 1	3 ± 1	1 ± 1
Cholesterol (mg)	354 ± 29	256 ± 33	202 ± 26
Fiber (g)	27 ± 3	33 ± 4	32 ± 2

Data are means ± SE.

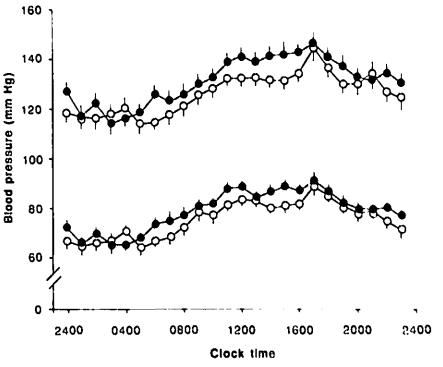

Figure 1—*Diurnal BP after 3 wk treatment with a high-CHO diet (●——●) and a high-MUFA diet (○——○) in 15 NIDDM subjects. Data are means ± SE.*

fered more than 500 g from the weight at the beginning of the study.

Analytical methods

Plasma and urinary glucose levels were measured by a glucose oxidase method. Serum insulin, glucagon, and GH levels were determined by a specific RIA (16). HbA_{1c} was measured by a commercial kit (Bio-Rad, Richmond, CA), (normal 3.5–5.5%). FFAs were determined by a standard enzymatic calorimetric assay method using a commercial kit (Boehringer-Mannheim, Mannheim, Germany). TGs and lipoproteins were measured on Enichom Chem 1 analyzer. Fructosamine was measured by microdetermination (17).

Statistical analysis

The incremental areas over the 480-min observation period were calculated geometrically as the incremental areas above fasting levels (18). The results are expressed as means ± SE. Student's *t* test for paired data was used for statistical analysis of ambulatory BP measurements, whereas multiple comparisons between response areas and mean values of the rest of the results were made by ANOVA (BMDP Statistical Software, Barkeley, CA). $P < 0.05$ was considered statistically significant.

RESULTS — All subjects consumed the meals completely during the 3-day profiles in the outpatient clinic. The fasting glucose and HbA_{1c} levels were similar before the two diet periods (Table 1). On both diets the patients had a minor weight reduction averaging 0.9 kg on the high-CHO diet versus 0.8 kg on the high-MUFA diet. The high-MUFA diet compared with the high-CHO diet induced significantly lower FBG levels (6.1 ± 0.3 vs. 6.8 ± 0.3 mM, $P < 0.05$) and 24-h glucosuria (38 ± 13 vs. 15 ± 7 g, $P < 0.01$). Mean fructosamine concentration decreased significantly during the high-MUFA diet from 353 ± 10 to 334 ± 10 μM, $P < 0.05$, but was constant during the high-CHO diet (323 ± 10 vs. 323 ± 9 μM). No differences were seen in fructosamine, fasting insulin, FFA, or TG levels on the last day of the two diets.

24-h ambulatory BP

Figure 1 shows 24-h profiles of BP measurements after 3 wk on the two diets. Table 3 gives the mean values of clinical auscultatory and oscillometric measurements. The clinical auscultatory BP did not exceed 171 mmHg in sBP and 97 mmHg in dBP in any of the patients.

The clinical oscillometric and auscultatory BPs were comparable with respect to sBP. The dBP was significantly overestimated by the monitor by 4 mmHg ($P < 0.05$).

Although no difference was found in the clinical BPs (Table 3), the patients at the end of the high-MUFA diet showed uniformly reduced 24-h ambulatory BP. As compared with the high-CHO diet, the high-MUFA diet reduced 24-h sBP (126 ± 8 vs. 130 ± 10 mmHg, $P < 0.03$) and dBP (75 ± 6 vs. 78 ± 5 mmHg, respectively, $P < 0.01$). As seen in Table 3, the effect of the high-MUFA diet on BP was mainly present during the daytime as no change was found when BP measurements during the night were analyzed separately. No carryover or time effect was found on the BP measurements during the two diets.

24-h urine collection

The 24-h excretion of electrolytes and albumin measured concomitantly with diurnal BP were similar on the high-CHO and the high-MUFA diets accounting: 130 ± 15 vs. 128 ± 11 mmol (so-

Table 3—*Average values of the clinical and 24-h ambulatory BP recordings in 15 NIDDM subjects at the end of a high-CHO diet and a high-MUFA diet*

Measurements	High-CHO diet	High-MUFA diet	*P* values
Auscultatory clinic sBP (mmHg)	139 ± 5	137 ± 4	0.65
Oscillatory clinic sBP (mmHg)	141 ± 4	138 ± 3	0.44
Ausculatory clinic dBP (mmHg)	82 ± 2	80 ± 2	0.35
Oscillatory clinic dBP (mmHg)	86 ± 2	84 ± 2	0.44
24-h sBP (mmHg)	130 ± 3	126 ± 2	0.03*
24-h dBP (mmHg)	78 ± 1	75 ± 2	0.01*
Daytime sBP (mmHg)	137 ± 3	131 ± 3	0.04*
Daytime dBP (mmHg)	84 ± 2	78 ± 2	0.02*
Night sBP (mmHg)	118 ± 3	115 ± 2	0.20
Night dBP (mmHg)	66 ± 1	66 ± 1	0.42
24-h pulse rate (beats/min)	73 ± 2	74 ± 2	0.81
Daytime pulse rate (beats/min)	78 ± 3	78 ± 3	0.79

Data are means ± SE.
*Indicates statistically significant differences.

dium), 66 ± 6 vs. 71 ± 5 mmol (potassium), and 12 ± 1 vs. 13 ± 1 mmol (calcium); 14.0 ± 7.7 vs. 7.7 ± 2.3 μg/min (albumin), respectively.

Day profiles

Figure 2 shows day profiles of blood glucose, insulin, glucagon, TG, FFA, and GH levels obtained during the high-CHO test menu served the last day of the two diets. It can be seen that the high-CHO test menu induced both a higher average blood glucose value (8.2 ± 0.6 vs. 7.4 ± 0.5 mM, $P < 0.03$) and peak blood glucose concentrations (11.3 ± 0.7 vs 9.9 ± 0.6 mM, $P < 0.01$) at the last day of the high-CHO diet rather than at the last day on the high-MUFA diet. No difference in mean values of insulin, glucagon, TGs, FFA, and GH were found. Similar blood glucose areas (above basal) were seen during the 8-h observation periods (776 ± 129 vs. 722 ± 135 mM × 480 min, respectively). No differences were present in insulin or glucagon response areas during the two diets.

Figure 3 illustrates the comparison of the day profiles to the high-MUFA test menu after 3 wk on the MUFA diet with the isocaloric test menu to high-CHO test menu after 3 wk on the high-CHO diet. The average and peak blood glucose levels were lower on the high-MUFA test menu than on the high-CHO test menu (7.0 ± 0.4 vs. 8.2 ± 0.6 mM and 8.9 ± 0.5 vs. 11.3 ± 0.7 mM, $P < 0.01$), whereas average glucagon, TG, and GH levels were similar. Mean insulin levels were lower in the high-MUFA test menu compared with the high-CHO test menu (173 ± 26 vs. 205 ± 28 pM, $P < 0.05$), whereas mean FFA levels were higher (0.44 ± 0.02 vs. 0.33 ± 0.02 mM, $P < 0.01$). Mean incremental area of blood glucose to the high-MUFA test menu was subdued as compared with the high-CHO test menu

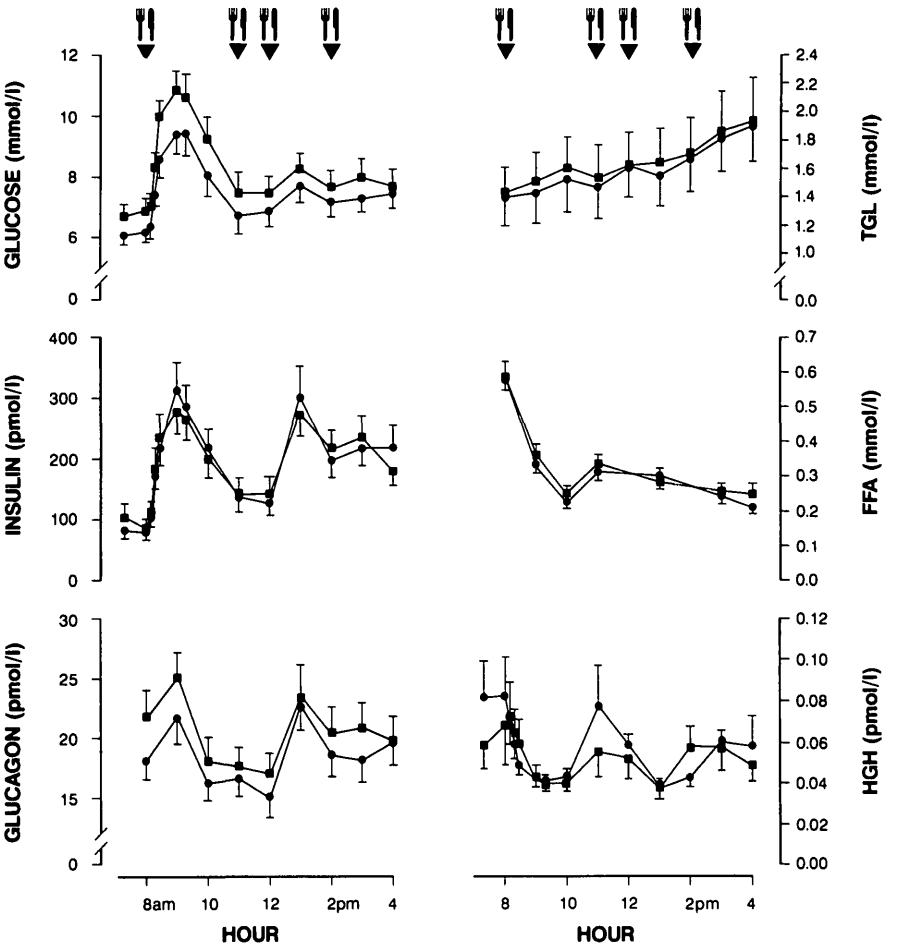

Figure 2—*Day profiles of blood glucose, insulin, glucagon, TG, FFA, and GH in 15 NIDDM diabetic subjects to a high-CHO test menu (3720 KJ) after 3 wk on a high-CHO diet (■——■) and a high-MUFA diet (●——●) period. Data are means ± SE.*

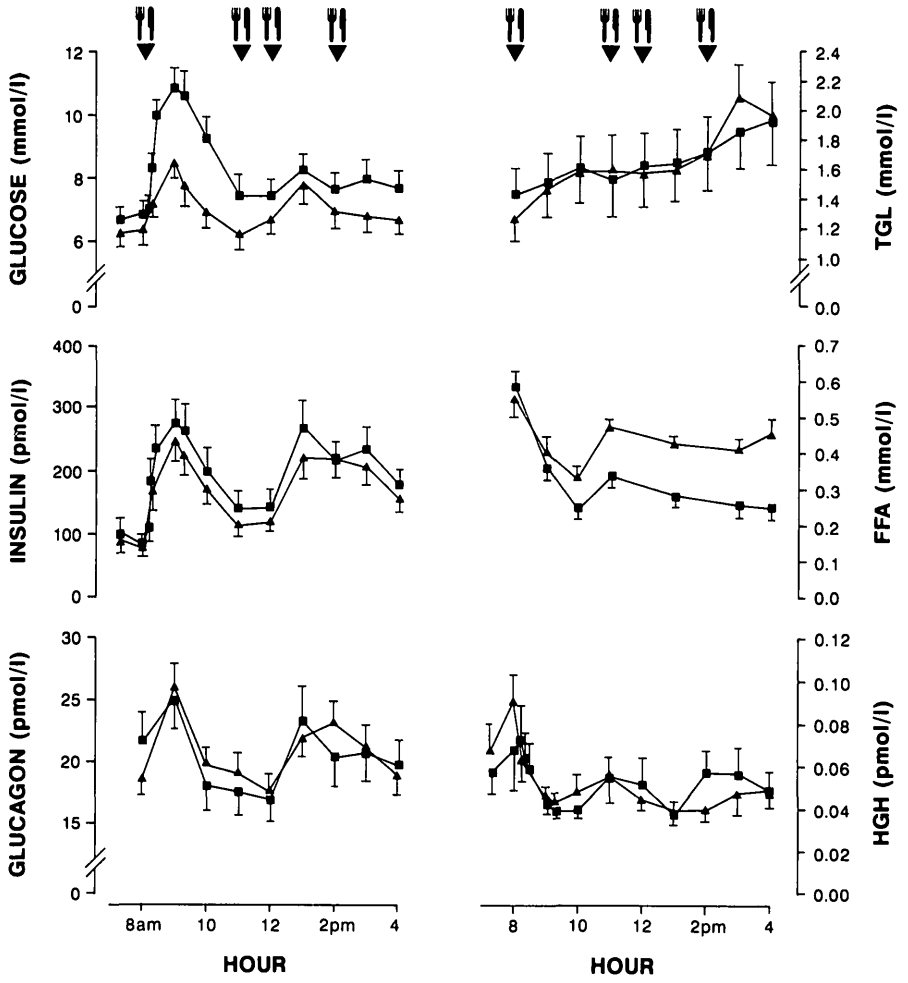

Figure 3—*Day profiles of blood glucose, insulin, glucagon, TG, FFA, and GH in 15 NIDDM diabetic subjects to CHO-rich test menu (■——■) after 3 wk on a high-CHO diet and an isocaloric (3720 KJ) MUFA-rich test menu diet (▲——▲) after a 3-wk high-MUFA diet period. Data are means ± SE.*

after the 480-min period (515 ± 150 vs. 776 ± 129 mM × 480 min, $P < 0.01$), whereas similar insulin response areas were found.

Lipids

The high-CHO and high-MUFA diets had a similar impact on lipid and lipoprotein metabolism. After 3 wk on these diets no differences were observed in fasting levels of TG (1.6 ± 0.2 vs. 1.6 ± 0.2 mM), total cholesterol (5.7 ± 0.3 vs. 5.4 ± 0.2 mM), LDL cholesterol (3.8 ± 0.3 vs. 3.5 ± 0.2 mM), HDL cholesterol (1.2 ± 0.1 vs. 1.2 ± 0.1 mM), and LDL/HDL ratio (3.2 ± 0.2 vs. 3.0 ± 0.2).

CONCLUSIONS— In this study a high-MUFA diet significantly reduced daytime as well as 24-h sBP and dBP compared with a high-CHO diet in 15 normotensive NIDDM subjects. Assuming no inherent diet effect on heart rate, the similarity of ambulatory heart rate during the two periods (Table 3) indicates that the reduced BP during the high-MUFA diet treatment period could not be ascribed to diminished physical activity.

The study was rigorously conducted according to the study plan. Despite this, a small weight reduction appeared during both 3-wk experimental periods, which might be caused by an underreporting of their estimated energy intake. Because the weight reduction was similar after the two diet periods, it cannot, however, explain the difference observed in various parameters. In the high-CHO diet period 49% of energy was CHO and 31% of energy was fat (11% of that was MUFA), whereas in the high-MUFA diet period 50% of energy was fat (30% of that was MUFA), and 36% of energy was CHO. The higher CHO content during the high-MUFA diet compared with the prescribed diet (36 vs. 30% of energy, respectively) is probably attributable to the high intake of homemade rolls with olive oil that the patients were advised to take if they felt hunger. The lower sBP and dBP during the high-MUFA diet were apparently not caused by weight loss or reduction in the sodium, potassium, or calcium intake since weight changes and the 24-h urinary excretion of sodium, potassium, and calcium were similar at the two diet periods. Reduction in diurnal BP induced by a high-MUFA diet has not been demonstrated previously in NIDDM subjects. The mechanism behind the BP reduction is not known. Although similar heart rates were observed at the end of the two 3-wk diet periods this does not entirely disclose the possibility that a high-CHO diet causes higher BP because of a stimulation of the sympathetic nervous system. Recent studies have demonstrated that an increase in the saturated fat content of the Mediterranean diet in middle-aged men also increased the sBP inversely to the decrease in the P/S ratio (12). Furthermore, salt-sensitive (sodium-sensitive) rat diets high in saturated fat (palm oil) induced higher BP than diets high in MUFA (olive oil) (19).

An automated ambulatory BP re-

cording model was used in our study to minimize measurement bias. Any systematic bias between the groups is unlikely because the reading and recording of BP was conducted by technicians who were unaware of the subjects' diets. Thus, the lower 24-h ambulatory BP of the high-MUFA diet compared with the high-CHO diet represents a true reduction. The difference in results between clinical and ambulatory BP measurements may appear puzzling, however, no normal relationship has been observed between clinical and ambulatory BP. Clinical BP is influenced by situational factors and is much more variable than ambulatory BP. In some patients clinical BP is higher than the ambulatory BP (the white coat effect), and in others the opposite is true (20). Even in the same patient no fixed relationship was found between the difference in clinical and ambulatory BP measured on the two occasions (14). Although clinical and ambulatory BP usually correlate statistically in a group of patients, large individual differences between the two values may be present (14).

During the high-MUFA diet period the day profiles to the high-MUFA test menu were found to induce lower blood glucose and insulin values than to the isocaloric high-CHO test menu during the high-CHO diet period (Fig. 3). One might claim that this is not surprising since the MUFA test menu contains about 33% less CHO than the high-CHO test menu. In other words, what we have observed (Fig. 3) might simply express differences in acute effects. To see if this is true, we have also looked at the impact of the high-CHO test menu at the last day of both 3-wk diet periods (Fig. 2). Improved glycemic control was reflected in lower FBG levels, reduced postprandial peak blood glucose levels, suppressed average glucose levels, and lower 24-h glucosuria that were observed at the end of the high-MUFA diet compared with the high-CHO diet when given a high-CHO test menu. The difference in blood glucose values following the high-CHO test menu served on the last day of the 2 diets apparently cannot be explained by changes in insulin, glucagon, or GH levels. Most recently Garg et al. (21) in NIDDM patients showed identical insulin sensitivity on a high-CHO diet and a high-MUFA diet. Furthermore, Bonanome et al. (7) found neither improved glucose homeostasis nor changes in lipoprotein concentrations after a 2-mo intervention period with a high MUFA diet versus a high-CHO diet in NIDDM subjects. A CHO-induced hypertriglyceridemia may be a potential disadvantage of a high-CHO diet (5,8,21). However, in this study similar fasting and mean levels of TG were observed during day profiles (Figs. 2 and 3). In contrast, Garg et al. found significantly lower serum TG levels on the last day of the high-MUFA diet compared with the high-CHO diet in insulin treated (6) and orally treated (21) NIDDM subjects. This might be explained by a better glycemic control in our patients compared with that found in the study groups of Garg et al. (6,21).

The increased FFA levels after the high-MUFA test menu compared with the high-CHO test menu is probably caused by the postabsorbtive lipemia, as long-chain unsaturated fatty acids are absorbed as triacylglycerols that are hydrolyzed to FFA and glycerol by the lipoprotein lipase. A high FFA level has been proposed as a candidate to elicit insulin insensitivity in NIDDM (8). The increased FFA level to the MUFA-rich menu did not, however, appear to provoke this effect because lower blood glucose and insulin levels occurred concomitantly.

We found a similar influence of a MUFA-rich diet compared with a CHO-rich diet on cholesterol and lipoprotein levels; these results corroborated with the data of Bonanome et al. (7). In contrast, Garg et al. found improved lipoprotein composition after a high-MUFA diet compared with a high-CHO diet in insulin treated (6) and orally treated (20) NIDDM subjects.

In summary, these data demonstrate that in NIDDM a high-MUFA diet causes a slight but significant reduction in BP and improved glycemic control without exerting adverse effects on lipid and lipoprotein levels. Consequently, a MUFA-rich diet might prove to possess therapeutical effects in diabetic subjects with hypertension and/or incipient or overt nephropathy.

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