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Urinary albumin excretion rate and 24-h ambulatory blood pressure in NIDDM with microalbuminuria: effects of a monounsaturated-enriched diet

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Summary Previous studies have shown that unsaturated fat-enriched diets may have a beneficial effect on blood pressure in non-insulin-dependent diabetic (NIDDM) patients, whereas little is known about the effects on albuminuria. In a 3-week cross-over design we compared the effects of a currently recommended high-carbohydrate diet (50 % carbohydrate, 30 % fat [10 % monounsaturated fat]) vs a diet rich in monounsaturated fat (30 % carbohydrate, 50 % fat [30 % monounsaturated fat]) on urinary albumin excretion rate, 24-h ambulatory blood pressure and metabolic control in ten NIDDM patients with persistent microalbuminuria. The 24-h ambulatory blood pressure was similar before and after both the high-carbohydrate diet (mean ± SD: 145/78 ± 25/10 vs 143/79 ± 19/10 mmHg (NS) and the monounsaturated fat diet: 140/78 ± 16/8 vs 143/79 ± 15/8 mmHg (NS). No changes were observed in day or nighttime blood pressures. Urinary albumin excretion rate was unaffected after 3 weeks' treatment by the diets: from (geometric mean ×/÷ tolerance factor) 32.4 ×/÷ 2.1 to 36.0 ×/÷ 1.9 μg/min (NS) vs from 34.2 ×/÷ 1.9 to 32.1 ×/÷ 2.1 μg/min (NS). Fasting plasma glucose, serum fructosamine and HbA_{1c} as well as lipid and lipoprotein concentrations were stable during both diets. Compared to the high-carbohydrate diet a reduction in the LDL/HDL cholesterol ratio was observed during the monounsaturated fat diet ($p < 0.03$). In conclusion, compared to a high-carbohydrate diet, 3 weeks' treatment with a monounsaturated fat diet did not affect the levels of 24-h ambulatory blood pressure or albuminuria in microalbuminuric NIDDM patients. Moreover, glycaemic control and lipoprotein levels were unchanged, although a potential beneficial effect on the LDL/HDL-cholesterol ratio was noted. Monounsaturated fat represents an alternative in the diets of NIDDM patients especially when caloric intake is not a concern. [Diabetologia (1995) 38: 1069–1075]

Key words Non-insulin-dependent diabetes mellitus; microalbuminuria, blood pressure, monounsaturated fat diet, olive oil, diet, metabolic control.

In non-insulin-dependent diabetes mellitus (NIDDM) a slight elevation in urinary albumin excretion rate (UAE), so-called microalbuminuria (defined as UAE between 20 and 200 μg/min) is associated with an increased incidence of cardiovascular morbidity and mortality [1–4]. Microalbuminuria may be present at the time of diagnosis of NIDDM [5–8] and is often associated with atherogenic risk factors, such as an abnormal lipoprotein metabolism, hyperglycaemia, obesity and hypertension [9–12]. Moreover, progression to overt clinical proteinuria (UAE > 200 μg/min) is more likely to occur in microalbuminuric patients than in patients with normoalbuminuria (UAE < 20 μg/min) [1].

Recent reports have demonstrated that an improvement of glycaemic control in newly-diagnosed

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Abbreviations: UAE, Urinary albumin excretion rate; NIDDM, non-insulin-dependent diabetes mellitus; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; ANOVA, analysis of variance; CHO, carbohydrate; CI, confidence interval.

NIDDM reduces and partly normalizes UAE [5, 6, 13]. Furthermore, it has become increasingly clear that not only a restriction of the total caloric intake (in combination with a weight reduction), but also dietary composition affects metabolic parameters and may improve cardiovascular risk factors in some patients. The currently advised dietary composition of NIDDM patients suggests that saturated fat should be reduced through a high-carbohydrate (CHO), low-fat diet including 50 % of energy from complex CHO, 30 % energy from fat and about 20 % from protein [14–16]. Moreover, unprocessed vegetables (i.e. fibre) should be implemented as a CHO source [17, 18].

Epidemiological studies from the Mediterranean area show a lower incidence of cardiovascular disease and a beneficial influence on the blood pressure in areas where people consume high amounts of monounsaturated fatty acids (MUFA) [19–21]. Moreover, intervention studies in both non-diabetic subjects and NIDDM patients have indicated that a high amount of CHO may cause untoward effects on metabolic risk factors [22–24]. Conversely, replacement of a high-CHO/low-fat diet by low-CHO/high-fat diet including high amounts of unsaturated fat, and especially monounsaturated fat, may favourably influence glucose metabolism (including insulin sensitivity), lipoproteins and blood pressure [24–29]. In two recent studies a high-MUFA diet was found to be associated with a reduction in diurnal blood pressure in normotensive NIDDM subjects with normal UAE (< 20 μg/min) as compared to a CHO diet [29] or a diet rich in polyunsaturated fat [30]. The influence of dietary fat on albuminuria in diabetic patients has not received much attention [31, 32] and the effect of a monounsaturated fat diet has so far not been examined.

Whether a MUFA diet in NIDDM patients with microalbuminuria affects metabolic parameters, suppresses the blood pressure and alters UAE is unknown. Thus, in a cross-over design we compared the impact of 3 weeks' treatment with a MUFA-enriched diet with a standard CHO diet, on metabolic control, 24-h ambulatory blood pressure and UAE in a group of NIDDM patients with persistent microalbuminuria.

Subjects and methods

Ten NIDDM patients (seven men and three women) with persistent microalbuminuria were randomly selected from the outpatient clinics. Additional criteria for inclusion were: age 50–75 years, diabetes duration greater than 1 year, and normal serum creatinine level. Patients with diabetic nephropathy, neuropathy or retinopathy (except background retinopathy) as well as patients taking hypolipidaemic agents were not included. Informed consent was obtained from each patient before taking part in the study, as well as ethical committee approval.

Study design. A total dietary registration of caloric intake for 4 days was performed in each patient and reviewed by a dietician. Based on food models and standard tables of food composition, individual isocaloric dietary schemes for a high-CHO-/low-fat diet and a low-CHO/high-MUFA diet were computed [25], aiming at providing the patients with a weight-maintaining diet. Patients were then assigned to either a CHO diet or a diet rich in MUFA for 3 weeks. This was followed by a wash-out period of 3 weeks after which the patients were switched to the opposite diets for an additional 3 weeks. The energy composition of the prescribed CHO diet was: 50 % CHO, 30 % fat, of which 10 % were MUFA. The MUFA-rich diet contained: 30 % carbohydrate and 50 % fat, of which 30 % was MUFA. The amount of dietary fibre was 25 g/day in both diets. As a source of MUFA cold-pressed virgin olive oil (Elanthy, Piraeus, Greece) was used. Four patients started on the CHO diet and six patients started on the MUFA diet. Special rolls and meat dishes containing 8 g and 30 g of olive oil, respectively, were provided frozen from the clinic together with olive oil. During the dietary intervention the patients visited the dietician once a week for reviewing of food schemes and weighing. Caloric intake adjustments were performed if body weight differed more than 500 g from the initial value. Registrations of caloric intake for 4 days were repeated at the end of both diet periods and used for computation of the actual energetic composition of the diets. Hypoglycaemic and antihypertensive drugs as well as insulin dosages were maintained constant throughout the study.

Before and after both diet periods, fasting blood samples were drawn in addition to measurements of 24-h blood pressure and UAE. Plasma glucose was measured by a glucose oxidase technique, serum fructosamine by a spectrophotometric technique [33], and HbA_{1c} by HPLC [34]. Serum insulin was determined by radioimmunoassay as described by Ørskov et al. [35] with minor modifications (normal level of fasting insulin in 42 healthy subjects (23 men, 19 women; age: 37 (21–53) (mean (range)) years; body mass index (BMI): 24.2 (16.8–29.6) kg/m^2) was 6 (4–7) mU/l) (median 25th–75th percentile)). Plasma C-peptide was assessed by a radioimmunoassay kit (Immunonuclear Corp., Stillwater, Minn., USA). Measurements of serum cholesterol by continuous-flow analysis [36], serum triglycerides by an enzymatic technique [37], serum (HDL)-cholesterol by a dextran sulphate-Mg^{2+} precipitation procedure [38] and serum creatinine by a modified Jaffe's reaction [39] were all adapted to the Technicon CHEM 1 (R) analyzer (Bayer $^{A}/_{S}$, Tarrytown, NY, USA). LDL-cholesterol and VLDL-cholesterol levels were calculated using the formula of Friedewald et al. [40]. Serum non-esterified free fatty acids were determined by a colorimetric method (Wako Chemicals, Neuss, Germany). UAE was measured by radioimmunoassay [41] and assessed as the mean of three 24-h collections. BMI was calculated as weight (kg)/height(m)^2 and used as an index of overall obesity.

Diurnal ambulatory blood pressure was measured by a portable automatic blood pressure monitor (SpaceLabs model 90202, Redmond, Wash., USA) using oscillometry [42]. The device was programmed to measure blood pressure every 20 min between 06.00 and 24.00 hours and every hour during the night. Patients recorded actual time for going to bed and rising in the morning for accurate appraisal of day and night blood pressures.

Statistical analysis

Values are given as mean ± SD or median (25th–75th percentiles). UAE was log-transformed due to the positively-skewed

Table 1. Clinical characteristics of NIDDM patients studied

Sex (men/women)	7/3
Age (years)	66 (40–71)
Diabetes duration (years)	11 ± 6
Fasting plasma glucose level (mmol/l)	8.6 ± 2.5
HbA_{1c} (%)	8.1 ± 1.5
Body mass index (kg/m^2)	27.9 ± 2.9
Serum creatinine (μmol/l)	96 ± 25
Diabetes treatment (d/o/i)	0/7/3
Antihypertensive treatment (yes/no)	3/7

Mean ± SD, median (range). d, Diet; o, oral hypoglycaemic agents; i, insulin

Table 2. Recorded energetic composition during 3 weeks of dietary intervention

	CHO diet	MUFA diet
Energy (kJ)	6037 ± 1785	7203 ± 1599[a]
Carbohydrate (%)	47.9 ± 3.7	34.2 ± 4.0
Protein (%)	17.5 ± 3.5	13.4 ± 2.7[b]
Fat (%)	29.9 ± 3.8	50.3 ± 6.3
PUFA (%)	7.7 ± 2.4	6.7 ± 1.1
MUFA (%)	9.0 ± 1.4	30.3 ± 4.5
SAT (%)	10.2 ± 1.8	10.0 ± 1.2
Alcohol (%)	1.5 (0.0–8.0)	1.0 (0.0–4.0)

Mean ± SD or median (25^{th}–75^{th} percentile)
[a] $p < 0.001$; [b] $p < 0.03$ vs CHO diet

Table 3. Urinary excretion rates before and after 3 weeks of dietary intervention

	CHO diet		MUFA diet	
	Before	After	Before	After
Sodium (mmol/day)	158 (145–184)	169 (127–177)	162 (126–203)	149 (132–173)
Potassium (mmol/day)	72 ± 23	70 ± 21	70 ± 24	71 ± 18
Creatinine (mmol/day)	11.0 ± 3.8	10.8 ± 3.8	10.4 ± 2.9	10.7 ± 3.5

Mean ± SD or median (25^{th}–75^{th} percentile)
Before vs After and CHO vs MUFA (ANOVA): NS

distribution and is presented as the geometric mean ×/÷ antilog SD. Where appropriate, 95 % confidence intervals (CI) are indicated. Baseline and endpoint differences between treatments as well as within-treatment changes were analysed using Student's *t*-test for paired comparisons or Wilcoxon's test for matched pairs. Between-treatment comparisons of overall dietary effects were analysed by repeated-measures analysis of variance (ANOVA). Correlations were evaluated using Pearson's *r* or Spearman's rho. A *p*-value less than 0.05 was considered statistically significant.

Power analysis. A mean change in 24-h systolic blood pressure of 9 mm Hg (expected SD of the mean change: 9 mm Hg) or in 24-h diastolic blood pressure of 4 mm Hg (expected SD of the mean change: 9 mm Hg) holds a statistical power of 80.4 % ($\alpha = 0.05$) in a paired *t*-test. Similarly, expecting a mean reduction in albuminuria of 20 % (UAE-final/UAE-baseline = 0.80), calculation of power on the log-transformed figures (mean change − 0.097) [expected SD of the mean change: log(1.200)] holds a power of 93 %.

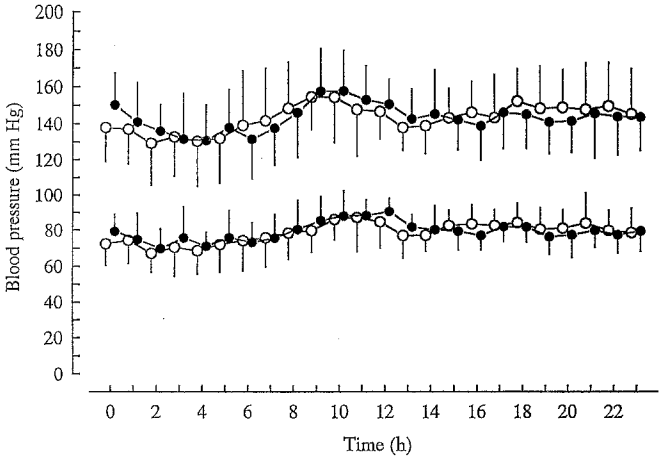

Fig. 1. Diurnal systolic and diastolic blood pressures after 3 weeks treatment with a high carbohydrate/low fat diet (○) and a low carbohydrate/high MUFA diet (●). No difference between the groups (ANOVA)

Results

Table 1 shows the clinical characteristics of the patients. Table 2 outlines the results of the energy composition of the diets as calculated from the individual registrations of food intake conducted at the end of each treatment period. The recorded protein content was slightly higher in the CHO diet than in the MUFA diet (62.1 ± 9.3 vs 56.4 ± 12.4 g/day, $p < 0.03$). Sodium and potassium intake, as estimated from the urinary electrolyte excretion rates were similar (Table 3). Both diets were well-tolerated and without significant adverse clinical effects. A slight decrease in body weight was noticed during both treatments (CHO: from 81.8 ± 12.0 to 81.0 ± 12.2 kg, $p < 0.02$ vs MUFA: from 82.4 ± 12.2 to 81.6 ± 11.8 kg, $p < 0.05$).

Figure 1 shows the 24-h blood pressure profile as recorded at the end of each treatment period. Twenty-four-hour ambulatory blood pressure was similar before and after the CHO diet (from 145/78 ± 25/10 to 143/79 ± 19/10, NS; mean difference (95 % CI) − 2 (− 10–7)/1 (− 2– 4) mm Hg) as well as after the MUFA diet (from 140/78 ± 16/8 to 143/79 ± 15/8 mm Hg), NS; mean difference (95 % CI) 4 (− 3–10) /2 (− 1– 5) mm Hg. Moreover, blood pressure changes were comparable during the two diets both for the systolic (mean difference (95 % CI) − 5 (− 17–6) mm Hg and the diastolic blood pressure − 1 (− 5–3)) mm Hg. Furthermore, no changes were found in either day or night-time blood pressures (Table 4).

UAE remained stable both during the CHO diet (from 32.4 ×/÷ 2.1 to 36.0 ×/÷ 1.9 μg/min, NS; relative rise: 1.11 (0.87–1.41) (mean ratio (95 % CI)) as well as during MUFA (from 34.2 ×/÷ 1.9 to 32.1 ×/÷ 2.1 μg/min, NS; relative rise: 0.94 (0.78–1.12) (mean ratio (95 % CI)). Moreover, no significant difference in the relative rise in UAE was found between the CHO and the MUFA diets (CHO ratio/

Table 4. Day- and night-time values during 24-h blood pressure and heart rate recordings

	CHO diet		MUFA diet	
	Before	After	Before	After
Systolic (diurnal)	145 ± 25	143 ± 19	140 ± 16	143 ± 15
Systolic (daytime)	148 ± 25	148 ± 19	143 ± 17	146 ± 16
Systolic (night-time)	138 ± 25	133 ± 21	131 ± 17	134 ± 17
Diastolic (diurnal)	78 ± 10	79 ± 10	78 ± 8	79 ± 8
Diastolic (daytime)	81 ± 11	82 ± 10	80 ± 8	81 ± 8
Diastolic (night-time)	72 ± 11	71 ± 11	71 ± 10	73 ± 9
Heart rate (diurnal)	76 ± 9	78 ± 9	79 ± 11	79 ± 11
Heart rate (daytime)	79 ± 10	83 ± 12	81 ± 11	81 ± 13
Heart rate (night-time)	68 ± 6	70 ± 10	70 ± 6	70 ± 7

Mean ± SD
All values NS

MUFA ratio: 1.18 (0.87–1.60) (mean ratio (95 % CI)). As seen from Table 5 glycaemic control and plasma lipoprotein concentrations were unaltered during both diets. Compared to the CHO diet a significant reduction in LDL/HDL cholesterol ratio was found during the MUFA diet ($p < 0.03$ (ANOVA)). No significant correlations were found between changes in blood pressure, UAE or metabolic parameters.

Discussion

In a cross-over study in NIDDM patients with persistent microalbuminuria we found no differing effects on 24-h ambulatory blood pressure or UAE after 3 weeks' treatment with either a MUFA-rich or a standard CHO diet. Glycaemic control and lipoproteins were unaffected during treatment, although a reduction in the LDL/HDL cholesterol ratio (thought to be an atherogenic risk marker) was noticed during the MUFA diet.

We used a 3 weeks' intervention design in order to compare the results with our previous study of normoalbuminuric NIDDM patients [29]. Other studies have demonstrated that a 3-week dietary intervention is sufficient to detect changes in metabolic control and lipoprotein concentrations [25]; and during recent years similar studies of NIDDM patients have also shown effects on metabolic control, lipids, blood pressure and insulin sensitivity [22, 23, 26, 27, 29, 43].

The difference in total energy consumption (Table 2) during the two dietary periods may have been

Table 5. Glycaemic control and lipoproteins during 3 weeks of dietary intervention

	CHO diet			MUFA diet			Between treatment
	Before	After	difference (95 % CI)	Before	After	difference (95 % CI)	difference (95 % CI)
Plasma glucose (mmol/l)	8.5 ± 2.5	8.7 ± 3.3	0.2 (–0.6–1.0)	7.7 ± 2.8	7.7 ± 2.7	0 (–1.2–1.2)	0.2 (–1.0–2.0)
Fructosamine (mmol/l)	350 ± 62	331 ± 76	–19 (–59–22)	349 ± 68	339 ± 73	–10 (–37–17)	–9 (–35–18)
HbA_{1c} (%)	7.9 ± 1.5	7.9 ± 1.5	0 (–0.2–0.2)	8.0 ± 1.5	8.0 ± 1.4	0 (–0.3–0.2)	0 (–0.4–0.4)
Insulin (mU/l)	13 (10–19)	13 (7–38)	1 (–6–28)	16 (12–17)	16 (12–21)	1 (–1–14)	0 (–6–22)
C-peptide (ng/ml)	1.7 ± 0.5	1.9 ± 0.9	0.2 (–0.2–0.6)	1.8 ± 0.7	1.7 ± 0.6	–0.1 (–0.3–0.2)	0.3 (0.0–0.6)
Total cholesterol (mmol/l)	6.0 (5.1–6.4)	6.2 (5.2–6.5)	–0.1 (–0.5–0.7)	5.7 (5.3–6.6)	5.9 (5.4–6.1)	–0.4 (–0.6–0.3)	0.3 (–0.2–0.8)
HDL-cholesterol (mmol/l)	1.45 ± 0.4	1.40 ± 0.4	–0.06 (0.19–0.08)	1.38 ± 0.3	1.41 ± 0.3	0.04 (–0.04–0.12)	–0.09 (–0.24–0.06)
VLDL-cholesterol (mmol/l)	0.72 (0.69–0.99)	0.78 (0.59–1.18)	–0.03 (–0.28–0.25)	0.78 (0.65–0.86)	0.80 (0.71–0.93)	0.06 (–0.14–0.14)	0.07 (–0.32–0.45)
LDL-cholesterol (mmol/l)	3.7 (2.8–4.2)	3.4 (3.3–4.3)	0 (–0.3–0.4)	3.4 (3.0–4.3)	3.5 (3.1–3.8)	–0.4 (–0.6–0.2)	0.3 (–0.1–0.8)
LDL/HDL cholesterol	2.71 ± 0.9	2.77 ± 0.9	0.06 (–0.24–0.36)	2.95 ± 1.1	2.64 ± 0.8	–0.31 (–0.68–0.06)	0.37 (0.05–0.69)[a]
Triglycerides (mmol/l)	1.60 (1.54–2.19)	1.74 (1.32–2.62)	–0.05 (–0.63–0.55)	1.74 (1.44–1.91)	1.78 (1.57–2.07)	0.12 (–0.31–0.31)	0.16 (–0.69–1.00)
NEFA (mmol/l)	0.94 ± 0.53	1.02 ± 0.44	0.07 (–0.05–0.19)	0.85 ± 0.43	0.98 ± 0.39	0.13 (–0.10–0.37)	–0.06 (–0.35–0.23)

Values are mean ± SD, median (25^{th}–75^{th} percentile), mean or median difference (95 % confidence intervals of difference); Serum insulin only for non-insulin treated patients ($n = 7$). [a] $p < 0.03$ change from baseline during CHO vs during MUFA (ANOVA)

slightly overestimated, since the patients were monitored on a weekly basis and since the changes in body weight were small and similar (< 1 kg) during both diets. A small weight reduction is common during this type of dietary intervention, as also seen in our patients, and is probably associated with the well-known tendency to underreporting (10–15 %) of energy content during food registration. The question arises whether this difference in energy and/or protein content could explain why no difference in blood pressure was detected as previously demonstrated in normoalbuminuric NIDDM patients [29]. To the authors' knowledge, no such evidence is presently available in the literature. From a theoretical point of view, a further increase in energy content in the CHO diet would also increase the amount of MUFA, making the difference in MUFA between the two diets smaller, and the amount of saturated fat would be increased. Theoretically, an increase in MUFA content would, if anything, favour a decrease in blood pressure, while increasing the amount of saturated fat would, if anything, favour an increase [44, 45]. No studies evaluating the effect of dietary protein restriction on blood pressure or albuminuria in NIDDM patients with normo- or microalbuminuria have been published. In insulin-dependent diabetic patients protein restriction is known to produce minor reductions in renal plasma flow, glomerular filtration rate, albuminuria and blood pressure [46]. However, the observed difference in the protein content in our study is very small compared to what is used in most clinical trials. Moreover, the protein content was already slightly higher during the CHO diet. Thus, increasing the energy content in the CHO diet would merely add to the difference in protein content.

It has long been recognized that a complementary exchange of dietary carbohydrate for unsaturated fat may influence blood pressure in both non-diabetic and diabetic subjects [44, 47, 48]. Recently, we found a significant reduction in day-time and 24-h systolic and diastolic blood pressure in 15 normoalbuminuric NIDDM patients treated with a MUFA diet as compared with a standard CHO diet for 3 weeks [29]. Using the same design, however, we found no changes in blood pressure in microalbuminuric patients. The mechanism behind the blood pressure-lowering potency is so far unknown. Sacks et al. [49] observed no effects of MUFA on erythrocyte sodium-lithium countertransport activity, which is associated with essential hypertension. Studies in young Sprague-Dawley rats have documented that low-fat diets markedly reduce the receptor-mediated vessel reactivity, and particularly MUFA was observed to reduce the non-receptor-mediated contractile response [50]. Other studies in NIDDM patients have suggested an increased insulin sensitivity and a reduction in hyperinsulinaemia following the exchange of a CHO diet for a MUFA-rich diet [26, 27]. In the latter study of mildly diabetic patients, however, only a slight tendency towards an improvement was observed. Hyperinsulinaemia may be a pathogenetic determinant in arterial hypertension [51–53], and it is possible that the blood pressure-lowering effect of MUFA may be mediated via an increased sensitivity to insulin action and a reduction in the concomitant hyperinsulinaemia. In fact, some reports have suggested that NIDDM patients with microalbuminuria, in general, may be more insulin-resistant, than are their normoalbuminuric counterparts [54–56]. As seen in Table 5, no change was observed in insulin or C-peptide levels during either diet. This would, to some extent, offer an explanation for the lack of changes in both blood pressure, UAE and metabolic parameters in our patients, and the question arises whether a sufficient influence on the hyperinsulinaemia was achieved to actually elicit a suppressive action on the blood pressure and modify other parameters associated with the insulin-resistance syndrome [57, 58]. In this context it is worth noticing that no other intervention modalities, except blood pressure reduction (especially systolic), have appeared to be effective in reducing microalbuminuria in NIDDM [59]. The long-term consequences are, however, not known, and different antihypertensive drugs may not cause similar effects on the level of albuminuria.

The LDL/HDL cholesterol ratio was significantly lowered during the MUFA diet as compared to the CHO diet. This is in accordance with other short-term studies in NIDDM showing that MUFA-rich diets may lower the total cholesterol to HDL-cholesterol ratio [24, 27, 28]. Moreover, a decrease in HDL-cholesterol has been described during a high-carbohydrate diet, but this is usually omitted or even increased during the high-MUFA diet [22, 27, 28]. Some studies, but not all, also describe minor reductions in VLDL-cholesterol and serum triglyceride concentrations [24, 27, 28, 43]. We found no changes in individual lipoprotein fractions. Furthermore, we observed no adverse effects on glycaemic control during the high-carbohydrate diet, probably because the composition of this diet was similar to the patients' usual diets. Plasma glucose and serum insulin concentrations are usually unaffected or slightly lowered during MUFA diets, whereas CHO diets may be associated with adverse effects on glycaemic control [25]. We did not measure day profile excursions in plasma glucose, serum insulin or triglycerides following a CHO or MUFA test meal, and it is important to realize that even though fasting levels of glucose metabolism and lipids are unchanged, significant differences may be obtained postprandially [24, 28, 29, 43]. In our previous study of normoalbuminuric NIDDM patients we found higher day-long glucose levels but similar insulin and triglyceride levels during the high-CHO diet as compared to the high-

MUFA diet [29]. In another study Garg et al. [60] found similar glucose and insulin day-long profiles but increased insulin sensitivity in NIDDM patients following a 3-week high-MUFA diet as compared to a high-CHO diet.

The different actions on blood pressure during a low-CHO/high-MUFA and a high-CHO/low-MUFA diet, as previously described in normoalbuminuric NIDDM patients [29] were not found in our microalbuminuric patients. In contrast to a number of studies glycaemic control and lipids were unaffected in the present study during both diets and no alterations in the microalbuminuria was observed. In conclusion, this and other studies suggest that MUFA represents an alternative in the diets of NIDDM subjects [60]. However, microalbuminuric patients may not be as sensitive to the effects of a change in dietary composition as are normoalbuminuric subjects. It is hypothesized that alterations in parameters associated with the insulin-resistance syndrome are not as easily modified in microalbuminuric patients, perhaps due to the prevalence of a further reduced insulin sensitivity, which is generally encountered in this group of patients. Whether a longer treatment with a MUFA-rich diet is necessary in NIDDM patients before any beneficial effects on cardiovascular risk markers can be unveiled remains to be elucidated.

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