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Quantitation of Apolipoprotein E-Isoforms in Diabetes Mellitus

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Introduction

In very low-density lipoproteins (VLDL) an increased content of apolipoprotein (apo) E in diabetic patients (Weisweiler, Drosner and Schwandt 1982) may have particular importance because of the discussed role of apo E for the uptake of lipoproteins by the liver receptor (Sherill, Innerarity and Mahley 1980). As shown by Havel, Chao, Windler, Kotite and Guo (1980) this function of apo E is largely confined to its less acidic isoforms, especially apo E-3. We analyzed the apo E-isoforms of VLDL by isoelectric focusing in ten patients with Type 2 (non-insulin-dependent) diabetes mellitus.

Patients and Methods

We compared ten male diabetic patients (26–49 years, mean duration of diabetes \pm S.D. 3.4 ± 1.9 years, no drugs) with ten healthy control subjects (same age range, unpaired Wilcoxon rank test). Baseline recorded values of the patients (control subjects) were 14.95 ± 3.15 (5.15 ± 1.29) mmol/L 10.00 h blood glucose, 6.98 ± 2.33 (4.68 ± 0.78) mmol/L serum cholesterol, and 3.23 ± 1.36 (1.13 ± 0.43) mmol/L serum triglycerides. VLDL were isolated by ultracentrifugation after a 12-h fast and delipidated with ethanol-diethyl ether, 3:1. Analytical isoelectric focusing was carried out with 0.015 mg solubilized VLDL-apolipoproteins in 6% horizontal polyacrylamide gel layers (0.24 mm) containing 8 mol/L urea and 2% Ampholine LKB, pH 3.5–9.5, at 10°C (Görg, Postel, Westermeier, Gianazza and Righetti 1980). The stained protein bands were scanned at 570 nm.

Results

Patients and control subjects had the phenotype apo E-3/3 (Zannis, Breslow, Utermann, Mahley, Weisgraber, Havel, Goldstein, Brown, Schonfeld, Hazzard and Blum 1982). In diabetic patients, there was, nevertheless, a shift to more apo E-1 + E-1' and apo E-2, but to less apo E-3, resulting in a significantly ($p < 0.01$) lowered apo E-3/apo E-2 ratio by -18.3% (Table 1).

Discussion

The shift to more acidic isoforms has been explained by an increase of the sialic acid content of apo E (Zannis and Breslow 1982). A defect in the desialation of apo E in diabetic patients is, therefore, assumed, similar to that seen in animals on diets high in cholesterol (Guo, Ha-

Table 1 Apolipoprotein E-isoforms in Type 2 diabetes mellitus

	Diabetic subjects	Control subjects
E-1 + E-1'	21.2 \pm 9.3	19.6 \pm 7.2
E-2	32.0 \pm 11.1	28.9 \pm 10.1
E-3	46.8 \pm 13.2	51.5 \pm 15.1
E-3/E-2	1.71 \pm 0.69*	2.21 \pm 0.64

Wilcoxon rank test: Diabetic patients (n=10) vs control subjects (n=10), * $p < 0.01$. Mean \pm S.D. (% of densitometric area).

ilton, Kane, Fielding and Chen 1982). This observation can be compared with the glycosylation of low-density lipoproteins (Gonen, Baenziger, Schonfeld, Jacobson and Farrar 1981) and may be associated with the premature atherosclerosis in diabetes mellitus.

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