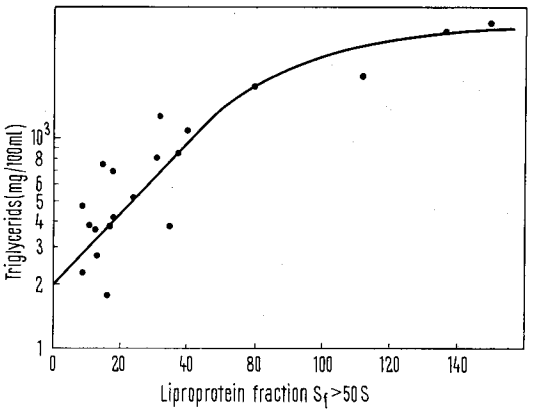
Investigations on the Role of Low Density Lipoproteins in the Pathogenesis of the Essential Carbohydrate-Induced Hypertriglyceridemia

SAILER, SANDHOFER and BRAUNSTEINER[1] proposed a disturbance of transport as a probable reason for the essential carbohydrate-induced hypertriglyceridemia (ECH). In order to gain a better insight into the pathogenesis of the metabolic disorder, we examined the lipoprotein and the lipid pattern of persons with ECH and compared the results with those in normal persons.

Materials and methods. We analyzed the sera of 21 persons with ECH and 12 normal persons. The triglyceride were determined using the method of CARLSON and WADSTRÖM[2], the free fatty acids according to DUNCOMBE[3].

The lipoproteins in the serum were gained quantitatively by preparative ultracentrifugation according to GATTO, LINDGREN and NICHOLS[4]. These lipoproteins were separated in the flotation classes by analytical ultracentrifugation. (The flotation experiments were carried out in an analytical ultracentrifuge type Phywe U60L and MOM G 120.) The lipoproteins were divided into 3 groups: $S_f < 10$ S, S_f 20–40 S and $S_f > 50$ S. Since it was impossible to establish the same initial concentration for all sera, we used the 'middle component' as a standard for the evaluation of real relations and referred the concentrations of the other fractions to this value.

This concept of a disorder in the interaction between lipid and protein components of the lipoprotein molecules may also be supported by the appearance of fractions within the range of elevated flotation classes in some pathological sera[8].



Plot of triglycerid value as a function of LDL concentration.

Average values of lipoproteins and lipid fractions in the case of normal and pathological lipid metabolism

	S_f (0–10 S) (mg%)	(rel.%)	S_f (15–40 S) (mg%)	(rel.%)	$S_f > 50$ S (mg%)	(rel.%)	S_f max.	Triglyceride (mg/100 ml)	Free fatty acid (μÄq)	Cholesterin (mg/100 ml)
Essential hypertriglyceridemia	290	9.0	495	17.0	3584	74.0	700	1036	383	374
Normal persons (n = 12)	300 ± 31	31.5	495 ± 50	51.9	158 ± 16	16.6	80–150	65 ± 9	140 ± 30	197 ± 10

Results. The Table gives a survey of the flotation classes and the lipid fractions of the sera from patients with ECH and normal persons. Characteristic – especially with persons showing high triglycerid values – is the appearance of components with extremely elevated flotation coefficients, which could not be found in normal sera.

Comparing the average values of patients with ECH with those of normal persons, a considerable elevation of both the absolute and relative low density lipoproteins (LDL) concentration was observed. Yet the absolute values of the LDL show no significant differences. The correlative aspect of the serum lipid fractions and the lipoproteid classes shows only a relation between LDL concentration and the logarithm of the amount of neutral fatty acids.

Discussion. These results lead, corresponding to FURMAN, ALANPOVIC and GUSTAVSON[5], to the conclusion that the ECH is a disease which is linked to a disturbance within the LDL. In addition we may say that, according to observations of FREDRICKSON[6] and JONG and MARSH[7], the LDL fraction has its own apoprotein which can be separated antigen-analytically and chemically from the apoprotein. Also the relation between LDL concentration and the amount of triglycerides demonstrates that the disturbance occurring in ECH is limited to the LDL.

The appearance of higher concentrations of quickly wandering flotation classes can be interpreted only by the assumption that in the case of ECH gigantic molecules or aggregates of molecules containing abnormal high lipoprotein-components are produced.

Zusammenfassung. Die mit physikochemischen Untersuchungen nachweisbaren LDL-Anomalien bei der essentiellen Hypertriglyceridämie lassen den Schluss zu, dass Störungen der LDL-Kinetik bei der Pathogenese dieser Krankheit eine wichtige Rolle spielen.

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