

## Changes in the Fatty Acid Composition of Serum Lipid Fractions During Acute Deposition and Mobilization of Fat in Man

The key role played by adipose tissue in the energy metabolism is rendered possible by the rapid intake and output of fatty acids from cells with regard to the needs of the organism. In the initial stages of fat deposition or mobilization, of all serum lipid fractions the concentration of free fatty acids (FFA) is the first one to change<sup>1</sup>. Under conditions when the long-term mobilization of fat reserves predominates, e.g. in hyperthyroidism, in the serum FFA fractions above all an increase of the oleic acid ratio was found<sup>2</sup>. It was therefore of interest to study the fatty acid composition of serum FFA and other lipid fractions during acute deposition or mobilization of depot fat.

Twenty-five subjects, 14 women and 11 men, average age 36 years, were examined. To 11 subjects after collection of the blood specimen on fasting, 1 g glucose/kg body wt. in tepid tea was administered by mouth. The subsequent blood specimens were withdrawn 60 and 120 min after the ingestion of glucose. In the remaining 14 subjects the effect of short-term fasting was studied. Blood specimens were withdrawn following the nocturnal fast, at 08.00 h, and subsequent blood samples were collected after 4<sup>1</sup>/<sub>2</sub> h. All blood specimens were taken from the

cubital vein. The serum extraction and gas-chromatographic procedures were described previously<sup>2</sup>.

From the Table it is apparent that 2 h after ingestion of glucose in the examined subjects, the ratio of oleic acid in the FFA fraction was significantly reduced, while the changes of the remaining fatty acids were not significant. The total FFA serum concentration in these subjects on fasting was on an average 0.63 mEq/l, and 2 h after ingestion of glucose it declined to an average value of 0.40 mEq/l. In the triglyceride, phospholipid and cholesterol ester fractions no changes were found after ingestion of glucose.

Also in the second group of subjects short-term fasting caused changes in the fatty acid composition only in the serum FFA fraction. From the Table it is apparent that a significant, marked increase of the oleic acid ratio occurred, while there was a concomitant drop of the palmitic and stearic acid levels. The known increase of the total FFA serum concentration during fasting was recorded also in the investigated group and amounted on an average to 0.25 mEq/l after 4<sup>1</sup>/<sub>2</sub> h.

In the present investigation fasting was selected as a typical condition causing mobilization of depot fat, and ingestion of glucose which leads to the deposition of fat<sup>1</sup>. It was no surprise that in both these acute conditions a change in the fatty acid composition was found only in the serum FFA fraction.

It is of interest that during the deposition, as well as mobilization of fatty acids of adipose tissue cells in the investigated subjects oleic acid was utilized preferentially. In this respect, thus the short-term changes were similar to those in conditions associated with the long-term mobilisation of depot fat<sup>2</sup>. Based on investigations of the heart muscle metabolism, signs of a relatively greater metabolic turnover of monoenoic fatty acids had already been found<sup>3</sup>.

The hypothesis of a more rapid metabolic turnover of oleic acid in serum FFA (and depot fat) in man is supported also by some animal experiments. Thus an increased rate of incorporation of labelled acetate into palmitoleic and oleic acid after insulin in adipose tissue of rats was found<sup>4</sup>, and a more rapid incorporation of radioactive cholesterol into saturated and monounsaturated cholesterol esters in the rat aorta<sup>5</sup>.

It may thus be summed up that in the course of short-term deposition or mobilization of depot fat in man, marked changes were found only in the oleic acid ratio in the serum FFA fraction.

*Zusammenfassung.* Es wird gezeigt, dass die Abnahme der freien Ölsäure nach Glukoseeinnahme und deren Zunahme im Serum beim Hungern beim Entstehen atheromatöser Veränderungen eine wesentliche Rolle spielen könnte.

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Fatty acid composition of serum lipid fractions in persons after glucose ingestions and during fasting

Fatty acid	Glucose		Fasting	
	Hour 0	2	0	4 <sup>1</sup> / <sub>2</sub>
Free fatty acids				
14:0	2.42 ± 0.71	2.53 ± 0.50	2.1 ± 0.3	2.0 ± 0.2
16:0	31.20 ± 4.36	34.34 ± 5.97	31.5 ± 1.8	28.9 ± 1.3 <sup>b</sup>
16:1	5.03 ± 0.98	5.60 ± 0.79	5.9 ± 0.8	6.4 ± 0.8
18:0	16.98 ± 2.73	18.50 ± 3.23	15.6 ± 1.9	12.9 ± 1.4 <sup>b</sup>
18:1	32.05 ± 3.06	27.34 ± 2.96 <sup>a</sup>	32.9 ± 2.8	37.6 ± 1.8 <sup>b</sup>
18:2	11.17 ± 2.59	9.90 ± 2.35	10.5 ± 0.7	10.7 ± 0.7
18:3	1.45 ± 0.11	1.35 ± 0.77	1.5 ± 0.5	1.5 ± 0.3
Triglycerides				
14:0	1.87 ± 0.61	1.63 ± 0.38	1.6 ± 0.5	1.6 ± 0.4
16:0	25.13 ± 2.83	25.38 ± 3.06	26.1 ± 2.2	25.6 ± 1.7
16:1	4.85 ± 0.89	4.92 ± 0.77	5.8 ± 0.7	6.1 ± 0.8
18:0	4.60 ± 1.08	4.76 ± 1.08	4.8 ± 0.8	4.4 ± 0.8
18:1	46.66 ± 3.22	45.32 ± 4.24	45.4 ± 2.3	45.1 ± 2.4
18:2	16.03 ± 4.79	16.07 ± 3.84	15.0 ± 2.8	15.2 ± 1.6
18:3	1.51 ± 0.53	2.12 ± 1.61	1.3 ± 0.4	1.2 ± 0.3
Phospholipids				
16:0	28.88 ± 2.2	29.63 ± 2.45	29.8 ± 1.5	30.1 ± 1.0
16:1	1.98 ± 0.41	2.10 ± 0.25	1.9 ± 0.3	1.9 ± 0.3
18:0	18.55 ± 2.01	17.50 ± 1.45	16.2 ± 0.9	15.9 ± 1.0
18:1	16.33 ± 2.78	16.26 ± 2.49	15.6 ± 1.1	16.1 ± 1.0
18:2	23.40 ± 2.73	23.62 ± 2.82	23.3 ± 1.3	22.2 ± 1.4
20:3	2.38 ± 0.75	2.32 ± 0.65	2.9 ± 0.7	3.1 ± 0.8
20:4	8.78 ± 2.95	10.24 ± 0.95	10.3 ± 1.6	10.8 ± 1.8
Cholesteroesters				
14:0	1.18 ± 0.42	1.20 ± 0.33		
16:0	13.32 ± 0.65	13.46 ± 2.69	12.9 ± 0.8	13.2 ± 0.8
16:1	3.99 ± 0.97	3.82 ± 0.61	4.6 ± 0.5	4.7 ± 0.6
18:0	2.27 ± 0.78	2.05 ± 0.53	1.9 ± 0.3	1.9 ± 0.3
18:1	22.15 ± 2.61	22.92 ± 3.22	21.8 ± 1.5	22.1 ± 1.2
18:2	50.17 ± 4.15	50.96 ± 4.06	52.9 ± 1.9	52.2 ± 1.9
20:3	1.50 ± 0.48			
20:4	6.37 ± 1.31	6.05 ± 1.26	5.9 ± 0.7	5.9 ± 0.6

<sup>a</sup>  $P < 0.05$ . <sup>b</sup>  $P < 0.01$ . Statistically significant difference between values in time 0 vs. 2, resp. 4<sup>1</sup>/<sub>2</sub> h.

<sup>1</sup> V. P. DOLE, J. clin. Invest. 35, 150 (1956).

<sup>2</sup> V. FELT and P. HUŠEK, Clin. chim. Acta 23, 331 (1969).

<sup>3</sup> M. E. ROTHLIN and R. J. BING, J. clin. Invest. 40, 1380 (1961).

<sup>4</sup> A. GELLHORN, W. BENJAMIN and M. WAGNER, J. Lipid Res. 3, 314 (1962).

<sup>5</sup> V. FELT and P. BENEŠ, Biochim. biophys. Acta 176, 435 (1969).