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Plasma-Triglycerides and Exercise: A Delicate Balance*

G. Schlierf, A. Dinsenbacher, U. Voggenreiter, B. Drews, H. Kather and M. Kohlmeier**
Medizinische Universitätsklinik Heidelberg

Summary. Alimentary lipemia was studied in 12 healthy young men with and without exercise. Three sets of experiments were performed. While continuous exercise of 90 min duration significantly reduced postprandial triglycerides by 26% (study I), this effect could not be observed when exercise was interrupted for 5 min after each 25 min (study II). Plasma free fatty acid concentrations, in the latter experiment, were significantly higher (by 311%) than during rest. When, in a third experiment continuous exercise was compared with intermittent physical activity, the latter condition significantly increased postprandial triglyceridemia, most probably due to precipitous rises of free fatty acids on each interruption of ergometry. It is concluded that in the third experiment the balance between triglyceride removal and triglyceride synthesis was shifted toward the latter. Whether exercise lowers, leaves unaltered, or raises plasma triglyceride levels may depend on subtle changes of experimental design.

Key words: Plasma triglycerides – Continuous or intermittent exercise

Nutrition [8] as well as physical exericse have been shown to exert marked influences on concentrations and patterns of plasma lipids and lipoproteins [1, 17, 20]. In addition to chronic effects of these environmental factors, there are acute influences such as alimentary lipemia following fat-containing meals [11] and its modification by intensive muscular activity [13]. While there is no dispute about increases in plasma triglyceride concentrations following meals which contain sufficient amounts of fat, reports on acute influences of physical activity on plasma triglycerides have varied: Decreases, no change, and even increases of plasma triglyceride concentrations have been described [2, 7, 9, 14, 16, 18].

Our report presents findings suggesting that continued and intermittent physical activity may have opposing effects on plasma triglycerides which may be mediated by different plasma free fatty acid (FFA) concentrations.

Material and Methods

The studies were performed using cross-over designs in 12 young (mean age, 25 years; range, 21–37 years), nonobese men, who had given their informed consent.

Exercise capacity was determined for each subject in the first study before the experiment by bicycle ergometry with 2-min stepwise increasing workloads to exhaustion. Maximum exercise capacity ranged from 270 to 360 watts.

In study I, after a 12-h fast, plasma was obtained at 0800, 0930 1100, 1230 and 1400 hours through an indwelling catheter for determination of plasma lipids and lipoproteins and free fatty acid concentrations. After the first venipuncture, the men took a standardized breakfast within 30 min containing 1188 kcal, 15 g protein, 94 g carbohydrates, and 82 g fat. It consisted of cereals, fruits, nuts, and cream. After the second blood sample at 0930 hours, the subjects exercised for 90 min on a bicycle ergometer at 40% of maximal exercise capacity, which had been determined as described above, corresponding to pulse rates ap-

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Abbreviations: FFA = Free fatty acids; VLDL = Very low-density lipoproteins

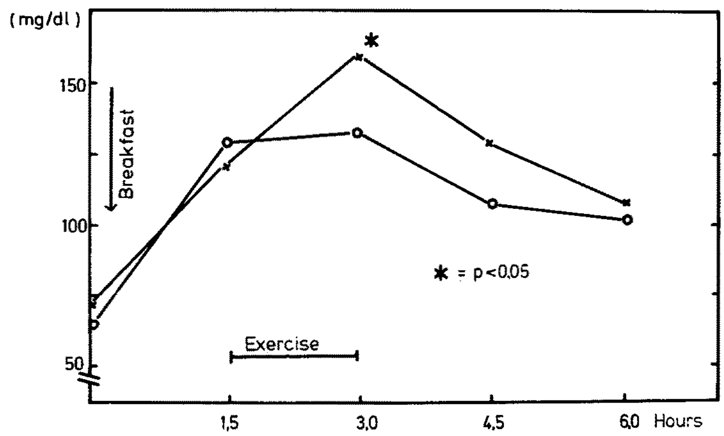

Fig. 1. Postprandial triglycerides (medians) with (o–o) and without (× – ×) exercise, corrected for free glycerol (reprinted with permission from [12])

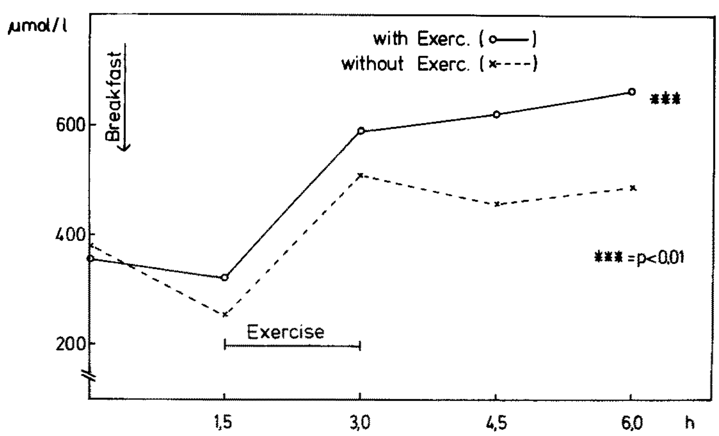

Fig. 2. Free fatty acids (medians) with (o–o) and without (× – ×) exercise (reprinted with permission from [12])

proximating 130/min on one day and continued to rest on another day after the same test meal. The sequence exercise-rest or rest-exercise was randomized. The two studies (exercise and control) in each subject were separated by at least 7 days. During the test without exercise and on "exercise days" before and after the bout of exercise, the subjects were sitting in a chair.

In study II, exercise was prolonged to 150 min. For practical reasons (avoidance of pressure sores by saddles) exercise was interrupted for 5 min each half-hour to stretch and change position. Controls were again without exercise.

In study III, uninterrupted exercise of 90 min duration (as in study I) was compared with intermittent exercise of 150 min duration (as in study I). For the purpose of this report, data were compared after 90 min of (continuous or intermittent) exercise. Behavior of FFA with intermittent exercise was observed in more detail in study III during the last 60 min of exercise.

Blood samples were immediately cooled to 4° C and triglycerides were measured after termination of each experiment according to an established enzymatic procedure.

Free fatty acids were determined by an enzymatic color procedure (Wako).

Statistical treatment of the data was done by comparison of the differences at 1100 and 0930 for the two experimental days in each subject,[1] using the Wilcoxon signed-rank test.

Results

Study I

Figure 1 shows the postprandial course of plasma triglyceride concentrations with and without exercise. There was a comparable alimentary lipemia up to 90 min following breakfast. On the day without exercise, triglycerides continued to rise for up to 3 h following breakfast, while on the exercise days, triglycerides plateaued during exercise and remained lower throughout the experiment. Similar results were obtained when triglycerides were measured in the VLDL and chylomicron fraction [12].

[1] The assistance of Dr. Scheurlen is gratefully appreciated

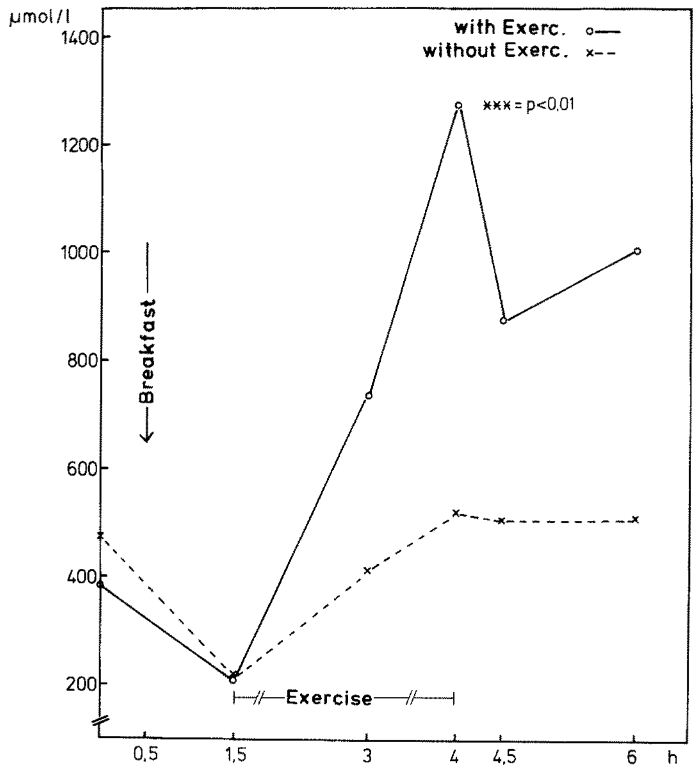

Fig. 3. Free fatty acids with (o–o) and without (×–×) intermittent exercise

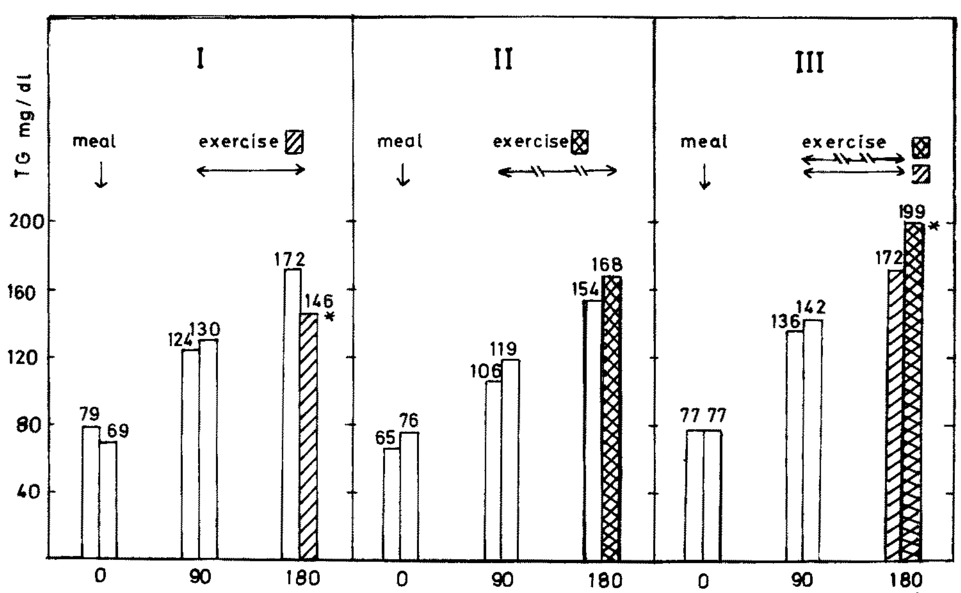

Fig. 4. Postprandial triglycerides (means) and exercise. I: rest, *open bars*; continuous exercise, *hatched bar*; II: rest, *open bars*; discontinuous exercise, *cross-hatched bar*; III: continuous exercise, *hatched bar*; discontinuous exercise, *cross-hatched bar*; difference between 180 and 90 min significant, $P<0.05$)

Figure 2 shows free fatty acid concentrations during the experiment. There was no significant difference up to the end of the exercise period, while, thereafter, free fatty acids continued to increase following cessation of exercise, yielding a significantly higher "free fatty acid area" as estimated from the sum of measurements. All results of study I have been documented in detail elsewhere [3].

In experiment II, time courses of plasma triglyceride concentrations with discontinuous exercise and without exercise were not different [15]. In

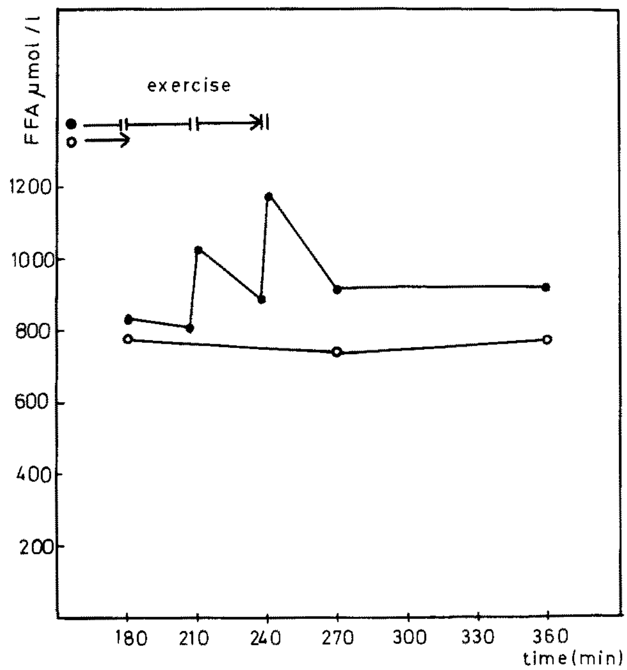

Fig. 5. Plasma free fatty acids from late phase of study III. FFA with intermittent exercise rise by approximately 200 µmol/l during the 5-min rest periods

contrast (Fig. 3) there was a marked and significant difference of plasma free fatty acid concentrations. While, without exercise, free fatty acid concentrations for up to 3 h after the meal rose by 139%, during exercise this increase amounted to 450% of the value at the start of the exercise. All results of study II have been documented in detail [15].

Figure 4 combines the data of plasma triglyceride measurements of all three experiments for up to 180 min. As shown, when intermittent and continuous exercise regimens were compared in experiment III, the former yielded significantly higher plasma triglyceride concentrations as compared to the latter. The final figure (Fig. 5) shows plasma free fatty acids as determined in experiment III in the late phase. It confirms the assumption of marked increases of plasma free fatty acids upon interruption of exercise.

Discussion

In confirmation of previous work [9], we observed in the first experiment mitigation of alimentary lipemia by 90 min of continuous, heavy exercise [12], which was probably mediated by increased lipoprotein lipase activity [6]. To our surprise, this phenomenon could not be observed when the exercise period was prolonged to 150 min, but, for practical reasons, was interrupted by four brief (5 min each) rest periods. With these conditions, free fatty acid concentrations were significantly higher than during alimentary lipemia alone (Fig. 3), quite contrary to continuous exercise. It was our hypothesis, then, that exercise- (catecholamine) induced liberation of free fatty acids from adipose tissue continued during the rest periods at accelerated rates, but utilization was suddenly interrupted [19]. Since endogenous triglyceride production is related to free fatty acid concentrations supplied to the liver [5], it was possible that by interruption of exercise the balance of triglyceride removal and triglyceride synthesis was shifted towards the latter, thereby abolishing mitigation of alimentary lipemia by exercise.

The third experiment was performed to test this hypothesis by comparing continuous, uninterrupted exercise and intermittent exercise in the same subjects. We also tried to more closely monitor free fatty acid concentrations upon interruption of exercise.

As shown in Fig. 4 there was, according to the hypothesis being tested, a significant increase of alimentary lipemia with interrupted as compared to continuous exercise of 90 min duration. We also saw that (Fig. 5) free fatty acid rose precipitously upon each interruption of ergometry due almost certainly to rapid influx from adipose tissue at a time when muscular demand had suddenly decreased.

Thus, differential effects on triglyceride removal [4] and triglyceride synthesis may be one underlying reason why effects of muscular activity on plasma lipids have differed in different studies. An interesting example of an (initial) rise of plasma triglyceride levels was presented by Wirth et al. [18], who observed students at the attempt to break the world record of continuous soccer playing. In their study, which lasted for 64 h, bouts of exercise alternated with rest periods of longer duration than in the present experiment. Plasma triglycerides were elevated at 16 h and only decreased thereafter. A quite different reason for increased alimentary lipemia with exercise was recently presented by Peden et al. [10], using β-blockade. Here, lipoprotein lipase activity was reduced by the drug yielding significantly higher postprandial triglycerides than observed in the control experiment.

In conclusion, the effects of muscular activity on triglyceride containing lipoproteins and possibly on other plasma lipoproteins may not only depend on intensity and amount of exercise, but, in a rather complicated way, on the balance between triglyceride utilization, which is directly promoted by exercise, and endogenous triglyceride production from free fatty acids, which in the present studies, were manipulated by altering their rates of liberation and utilization.

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Prof. Dr. G. Schlierf
Klinisches Institut für Herzinfarktforschung
Medizinische Universitätsklinik
Bergheimerstr. 58
D-6900 Heidelberg