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Biochemical Changes in a 100 km Run: Carbohydrates, Lipids, and Hormones in Serum*

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Summary. During the 100 km race in Biel, Switzerland, seven-well-trained men (age 33.3 ± 3.5 years; $\dot{V}_{O_2}$ max. 59.9 ± ml/kg) have been investigated.

Their mean running time over the 100 km distance averaged 10.41 ± 1.25 h. In contrast to almost unchanged blood glucose and lactate concentrations, blood lipids showed significant changes. Triglycerides decreased about two-fold, whereas glycerol and free fatty acids increased to extremely high concentrations (0.628 and 2.44 mmol/l respectively). Plasma insulin after the run was unaffected, whereas growth hormone, aldosterone and cortisol concentrations were significantly increased. With the exceptions of a still significantly elevated aldosterone and lactate concentration as well as a decreased triglyceride concentration all other values in the blood are restored to normal 24 h after completion of the run.

Key words: Long distance run – Heart rate – Carbohydrate and lipid metabolism – Hormones

Introduction

A continuous energy supply is needed during prolonged physical exercise. Appropriate arterial concentrations guarantee the continuous uptake of substrates by the muscle cell [25, 28]. Blood substrate concentration is regulated by neural or hormonal processes and there is a positive or negative arterio-venous difference according to changing energy demands of the muscle [22, 23, 38]. In our present study we further investigated the different variations in blood hormone and substrate concentrations occuring after heavy exercise of several hours duration. Special emphasis was given to carbohydrate and fat metabolism as well as to growth hormone, insulin, cortisol and aldosterone.

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Material and Methods

Seven trained long-distance runners participating in the 100 km race of Biel, Switzerland, were investigated for anthropometric data and performance capacity according to current methods of exercise physiology [37]. The run stared at 10 p.m. and lasted until the next morning, the mean temparature being 15–20° C. The participants were allowed free intake of food and beverages before, throughout and after the 100 km race. The heart rates of the runners were continuously recorded by the Memoportsystem manufactured by Hellige, Freiburg i. Br., FRG.

A few minutes before the start, 10 min after the race and after 24 h recovery blood samples were collected either from the ear lobe or from an antecubital vein and analyzed for glucose, lactate [20, 40], or triglycerides, glycerol, free fatty acids and cholesterol respectively [12, 24]. Plasma growth hormone, insulin, cortisol and aldosterone concentration were measured by commercially available radioimmunoassays manufactored by CEA-IRE-SORIN, Frankfurt, FRG. The plasma for the adlosterone assay was previously purified by chromatography on a silica gel column [7]. Statistical significance of paired differences in means and standard deviations was calculated by student's *t*-test ($P < 0.05$).

Results

The anthropometric data of the subjects and the results of the treadmill test are given in Table 1.

The mean running time for the 100 km was 10.4 ± 1.3 h, the range being 7.–11.9 h. Despite the food and water intake, the subjects lost 2.7 ± 1.0 kg of their body weight. The mean overall heart rate was 152 ± 5 beats per minute (Fig. 1).

The changes in blood concentrations of substrates and metabolites are shown in Fig. 2. Glucose concentration was the same before and immediately after the race (5.56 ± 0.99 mmol/l), whereas 24 h later there was a significant decrease to 4.44 ± 0.85 mmol/l. For blood lactate we observed an almost twofold concentration of 2.15 ± 0.81 mmol/l at the end of the run as well as in the recovery phase compared to the initial value (1.17 ± 0.28 mmol/l). The highest lactate concentration (3.72 mmol/l) was found in the blood of the runner with the best running time. Triglycerides showed a marked decrease from 1.24 ± 0.46 to 0.67 ± 0.19 mmol/l during the run and remained at a low level during the first 24 h of recovery. Plasma free fatty acid and glycerol concentrations were 6 and 14 times higher after the run (2.44 ± 0.36 mmol/l and 0.628 ± 0.206 mmol/l

Table 1. Anthropometric data and results of the treadmill test

Age (years)	33.3 ± 3.5
Body weight (kg)	72.7 ± 4.9
Body height (cm)	174.6 ± 3.2
Body surface area (m^2)	1.87 ± 0.1
Heart volume (ml/kg)	11.7 ± 0.9
$\dot{V}_{O_2}$ max (l/min)	4.4 ± 0.5
$\dot{V}_{O_2}$ max (ml/min · kg)	59.9 ± 4.9
Heart rate at exhaustion	183 .. ± 6.6
pH at exhaustion	7.22 ± 0.06

Means and standard deviations, $n = 7$

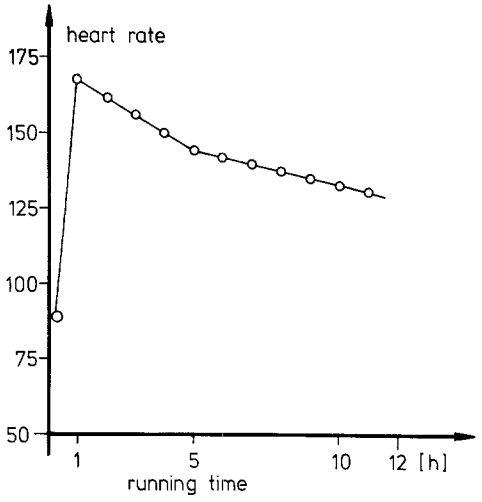

Fig. 1. Mean heart rate of the seven subjects during the 100 km run

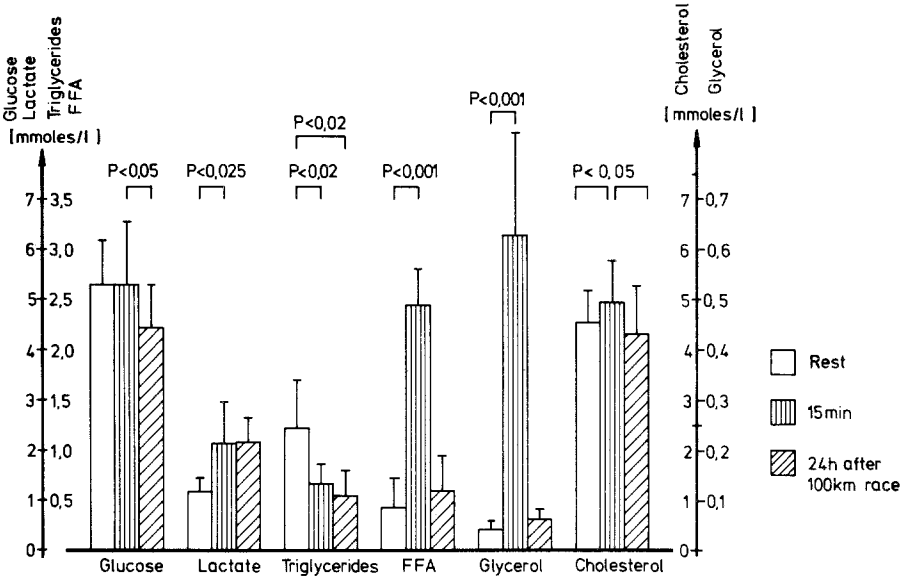

Fig. 2. Changes in blood concentrations of substrates and metabolites (means and standard deviations)

respectively), returning to the normal initial values within one day. Only slight but significant changes were found in blood cholesterol concentrations, the highest value of 5.13 ± 0.86 mmol/l being reached immediately after the race.

The plasma hormone changes are graphically represented in Fig. 3. We found a significant increase in blood concentration of human growth hormone (HGH) from 1.14 ± 1.48 ng/ml before the run to 7.24 ± 3.25 ng/ml after the run, with a total recovery on the next day (0.86 ± 0.66 ng/ml). No significant changes

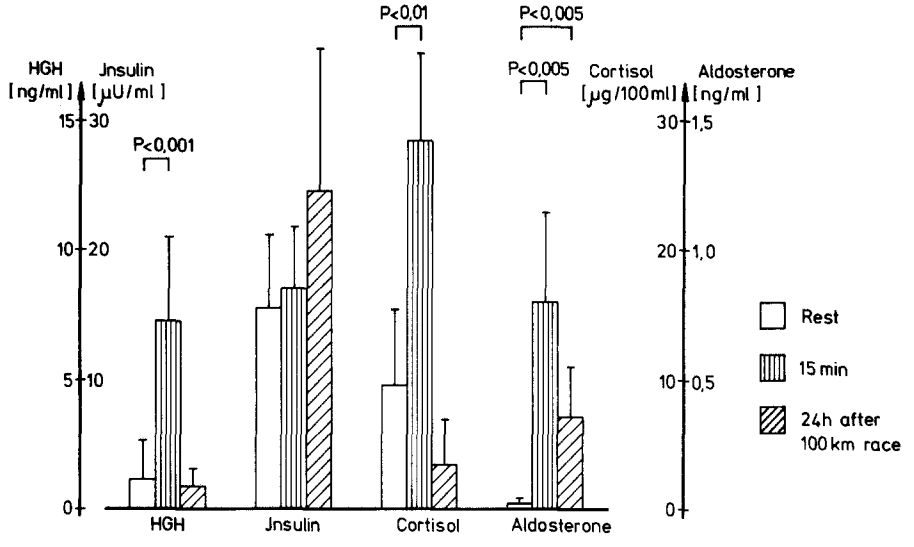

Fig. 3. Plasma hormone changes 15 min and 24 h after a 100 km run (means and standard deviations)

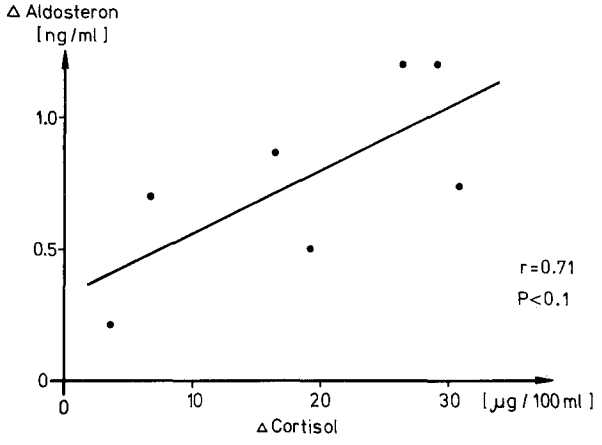

Fig. 4. Individual increments in plasma aldosterone related to plasma cortisol

in plasma insulin could be detected. For cortisol there was a significant increase from 9.59 ± 5.80 µg/100 ml before the run to 28.41 ± 6.80 µg/100 ml immediately after, and a total recovery to 3.47 ± 4.03 µg/ml on the next day. Plasma aldosterone concentration increased significantly from 26 ± 21 pg/ml to 795 ± 346 pg/ml with a still significantly elevated level of 360 ± 191 pg/ml on the next day.

To investigate whether all the observed hormonal changes would depend on a common stress factor, we calculated correlations between the individual

increments in the different hormones. The highest coefficient of correlation was found for the changes in plasma cortisol and plasma aldosterone: $r = 0.71$, $p < 0.1$ (Fig. 4). The coefficients of correlation for HGH and aldosterone on the one hand, and HGH and cortisol on the other were 0.1 and 0.48 respectively.

Discussion

Performance capacity in long-lasting exercise of high intensity depends mainly on aerobic power and substrate supply of the working muscles. The mean maximum oxygen uptake of the subjects investigated in the present study markedly exceeded the values normally found in untrained men, but it was well beyond the 80 ml of oxygen consumption per minute and kg of body weight usually attained by elite long distance runners [2]. The relative heart volume of the 100 km runners was not increased above normal values, indicating that the main adaptation in the aerobic system is to be expected in the muscle tissue, whereas the cardiac output could not have been relevantly increased [37]. The mean heart rates found to be in a range of 150–160 beats per minute indicate that the average work intensity throughout the race was about 60% of the subjects' maximum aerobic power [34]. In running competitions of 1–2 h duration the heart rate is known to be higher than 180 and 160 beats per minute respectively, the maximum heart rate for the age group studied being 190–200 beats per minute [26].

No changes in the blood glucose concentration could be observed after the 100 km race. Other authors studying the effects of submaximal or maximal exercise lasting up to 4 h found slight decreases of the arterial glucose concentration [2] and conducted on a direct relationship between the work duration and blood glucose [2]. However, it should be noted that our subjects were allowed free intake of carbohydrates thus preventing hypoglycemia. After the 100 km race no significant changes in blood insulin concentrations could be found. This result is in agreement with other studies investigating physical exercise of several hours duration [1, 35] but in contrast to the results of a decrease of insulin [18, 25, 28, 33, 37]. In short term exercise blood insulin concentration was found to either increase or decrease, these contradicting results probably being a consequence of variable work intensities [1, 19, 32]. Epinephrine secretion in very heavy exercise leads to a higher blood glucose concentration and this seems to induce an increase in insulin concentration [1]. In long lasting exercise performance capacity is strongly influenced by the muscle glycogen content which was shown to decrease by as much as 75% after 100 km races or 85 km crosscountry skiing [4, 16, 32]. However, the intracellular glycogen stores were calculated to account only for approximately 25% of the total energy output provided by carbohydrates [4]. Therefore a sufficient insulin secretion is of major importance in the maintenance of blood glucose homeostasis and energy supply to the working muscles.

At work loads requiring about 60% of an individual's maximum oxygen uptake capacity, lactate production is very low and the anaerobic threshold is not

exceeded [29]. Blood lactate concentrations of about 2 mmol/l were also observed after only 2 h of steady state running or cycle ergometer work [25, 26]. Nevertheless, there must be a higher lactate production during this type of exercise since the non-working muscles [23], the myocardium [25], the liver [3] and the kidneys [25], are continuously extracting lactate from the arterial blood.

It is a well known fact, indicated mainly by changes of the respiratory quotient, that during long-lasting exercise lipid oxidation plays a progressively more important role in energy supply [9, 10]. The increase in the plasma free fatty acid concentration during this type of work is therefore of special relevance [13, 25–28, 30]. The mechanism regulating the degradation of free fatty acids is only partly known. However, an inverse relation between glucose and fat utilisation is well established, being independent of hormonal control and described as so called glucose-fatty acidscycle [35]. The significance an elevated utilisation of free fatty acids during long-lasting exercise lies mainly in a glycogen and glucose sparing effect, blood glucose thus being available for other organs obligatorily depending on it (e.g., brain). The skeletal muscles utilize lipids as substrate, despite the fact that the yield in ATP per amount of oxygen used is 13–15% lower in the case of free fatty acids utilization compared to glucose degradation [25, 28]. The lower yield in ATP can obviously well be tolerated as the energy flow is stabilized at a level statisfying the actual needs.

The essential point for a high uptake and metabolisation of free fatty acids lies in significant increase in blood concentration to almost 2.5 mmol/l. Due to the relatively fast turnover of the free fatty acids [14, 15] an even more pronounced increase of glycerol with blood concentration exceeding 0.6 mmol/l and in individual cases even 0.8 mmol/l is well understandable (Fig. 2). As glycerol can only be metabolized by the liver but not by the muscle this increase is a proper indicator for lipolysis. As a consequence of lipolysis the triglycerides concentration in blood is also reduced to about 50% of the pre-race values. During exercise bouts of 1–2 h duration no decrease of blood triglycerides is observed, indicating a lipolysis mainly in the adipose tissue leading to increases of free fatty acids to more than 2 mmol/l and of glycerol to more than 0.5 mmol/l [25–28]. Obviously there is an even higher rate of lipolysis in the 100 km run as indicated especially by the higher plasma concentrations of glycerol. It seems that under these extreme conditions splitting of plasma triglycerides can be observed. As expected plasma cholesterol concentrations are not changed significantly after 24 h (8, 31].

The intensified lipolysis during the 100 km run has to be seen in the context of a release of growth hormone. Already after exercise of 1–2 h duration significant increases of growth hormone concentration have been observed [6, 18, 28, 33, 39], though no such changes could be found after short-lasting exercise bouts [18]. After intermittent work at very high intensity moderately elevated plasma concentrations of this hormone have been measured [18]. A strong relationship between the increase of plasma growth hormone and lipolysis during long-lasting physical exercise was found, the lipolytic effect of growth hormone appearing with a certain delay. It could be that during the 100 km run even higher growth hormone concentrations temporarily existed but that by food

intake, and especially the carbohydrates, a further increase in plasma growth hormone has been inhibited [28]. Nevertheless, the relatively small increase observed seems to be sufficient for a high rate of lipolysis.

Decreases [41], but more frequently increases of the blood cortisol concentrations have been observed after longlasting exercise [1, 18, 31, 33, 42]. We found a three-fold raise of this hormone. This finding is in agreement with other studies after Marathon races and 70 km cross-country skiing competitions [1, 11, 18, 41]. The increase in blood cortisol concentration during long-lasting exercise is important, since it induces an enhanced gluconeogenesis, inhibits the peripheral glucose utilisation [21] and promotes the degradation of fat [36].

Extreme changes were found in plasma aldosterone concentrations, which increased about 40 times. The positive correlation between the increments in aldosterone and cortisol ($r = 0.71$) is remarkable. The increase in plasma aldosterone during muscular work is promoted first by the high extracellular concentration of potassium as a consequence of glycogen degradation [3], secondly by an elevated renin-angiotensin activity as a consequence of the decreased renal blood flow [5, 17, 41] and finally by a reduced metabolisation of the hormone in the liver [41]. The latter mechanism is especially emphasised during long-lasting exercise. The very high aldosterone concentration must by means be understood as an insufficiency of the cardio-vascular system [41]. Twenty-four hours after the run the plasma aldosterone concentration remains significantly elevated, indicating that in the case of this hormone, in contrast to other, 1 day is not sufficient for a complete recovery. The high plasma aldosterone after the run certainly also reflect the immense shifts and losses of fluid (up to 2.5 l) ocurring during the 100 km run despite a continuous supply of food and beverages.

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