Eur J Appl Physiol (1982) 48: 407–414

European Journal of
Applied
Physiology
and Occupational Physiology

The Influence of Moderate Prolonged Exercise and a Low Carbohydrate Diet on Ethanol Elimination and on Metabolism*

P. M. Schürch, J. Radimsky, R. Iffland, and W. Hollmann

Institut für Kreislaufforschung und Sportmedizin
(Leiter u. Lehrstuhl f. Kardiologie u. Sportmedizin: Prof. Dr. W. Hollmann),
Deutsche Sporthochschule Köln, Carl-Diem-Weg, D-5000 Köln 41, Federal Republic of Germany
Institut für gerichtliche Medizin (Direktor: Prof. Dr. M. Staak) der Universität zu Köln,
D-5000 Köln 30

Summary. The influence of physical exercise together with a carbohydrate reduced diet on ethanol elimination has been examined in eight healthy subjects.

Three alcohol load-tests took place 10 h after the last meal. The alcohol dose amounted to 0.66 g/kg body weight. Two of the tests were followed by cycle ergometer exercise at 50% of the maximum individual oxygen uptake. This exercise began 120 min after alcohol ingestion and lasted 90 min. Between these two work tests there was an interval of 3 days. During these 3 days the subjects received carbohydrate reduced diet. The determination of alcohol took place every half-hour; serum glucose, triglycerides, glycerol and free fatty acids were measured before and after physical exercise.
The essential findings were:
1. Ethanol elimination increased by about 43% with normal food, and by 25% with a carbohydrate reduced diet during physical exercise. This finding has no pathological significance.
2. The combination of alcohol, carbohydrate deficiency and physical exercise induced an enhanced mobilization and oxidation of fat. Some subjects developed hypoglycemic serum glucose levels.
3. The muscles are not able to utilize ethanol directly or indirectly. The increased ethanol elimination seen in physical activity is probably due to enhanced enzyme activity in the liver induced by a rise in body temperature. Additionally a certain loss of alcohol through perspiration and exhalation is possible.

Key words: Physical exercise – Carbohydrate reduced diet – Ethanol elimination – Metabolism

* Supported by the Bundesinstitut für Sportwissenschaften Köln
Offprint requests to: Dr. P. Schürch, Forschungsinstitut ETS, CH-2532 Magglingen, Switzerland

0301-5548/82/0048/0407/$ 01.60

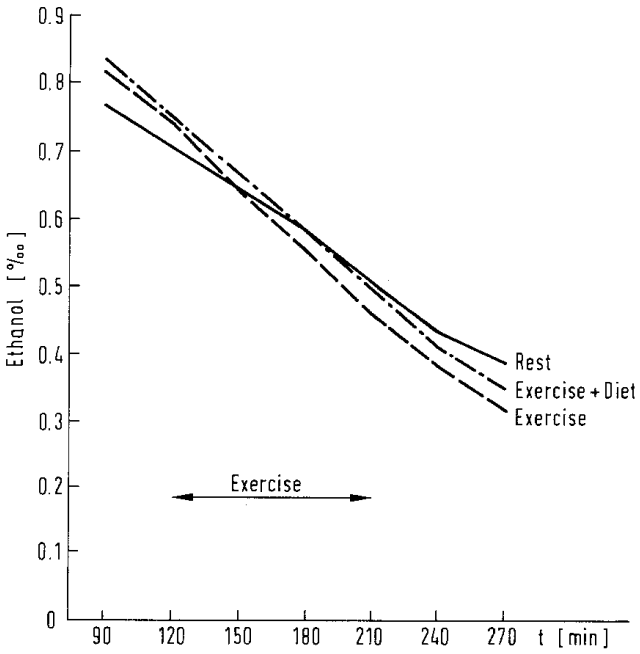


Fig. 1. Mean values of blood alcohol in *rest,* exercise with normal food diet (*exercise*) and exercise after carbohydrate reduced diet (*exercise + diet*)

Various experiments to enhance the elimination of blood alcohol have been performed during the last decades. Studies with drugs and physical exercise are a part of these investigations. Some results show an accelerated ethanol elimination [4, 5], others produced no change [9, 12]. It is an interesting fact that children with glycogenosis type I (glucose-6-phosphate deficiency) and low serum glucose levels show a high elimination rate of ethyl alcohol [19].

The following points were of special interest in our investigation: what is the influence of physical exercise on alcohol elimination provided the exercise is of a long and intensive enough duration? Is it possible in regard to alcohol reduction to simulate glucogenosis type I? Of what influence are physical exercise, alcohol and carbohydrate deficiency?

Methods

Eight healthy sports students took part in these experiments. Their particulars were: age 24 ± 1.7 years, height 178 ± 4.7 cm, weight 72.7 ± 4.3 kg. $\dot{V}O_2$ max 55.3 ± 4.3 (ml/min/kg). Their endurance conditions differed. Three times each student had to consume a drink containing 40% alcohol. The experiment started in the morning at least 10 h after the last meal. Each time the alcohol dose amounted to 0.66 g/kg body weight.

"Test I" was an examination during rest, "Test II" and "Test III" were experiments with physical exercise. "Test III" always took place 3 days after "Test II". During these 3 days the students received a low carbohydrate diet.

We use a cycle ergometer for the working experiments. The work load amounted to 50% of the individual maximum oxygen uptake. Exercise began 120 min after a drink had been taken and lasted 90 min.

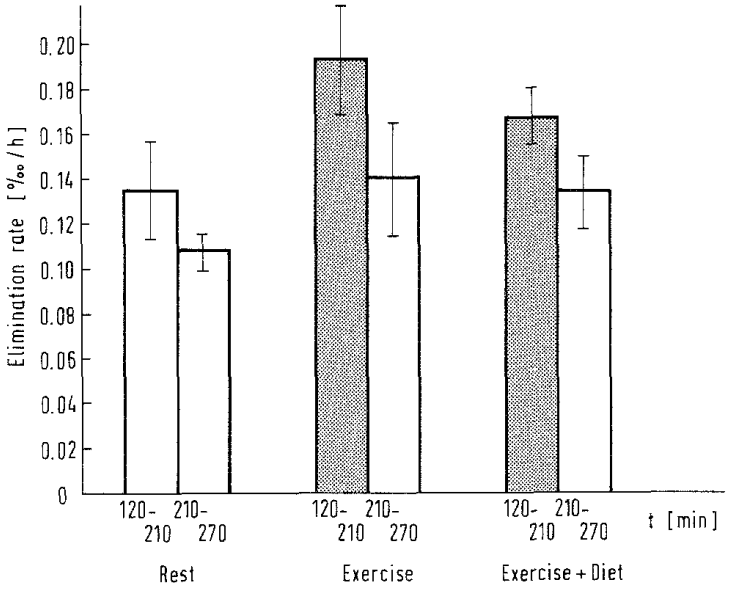


Fig. 2. Rate of alcohol elimination per hour during rest and exercise in *rest*, exercise with normal food diet (*exercise*) and exercise after carbohydrate reduced diet (*exercise + diet*)

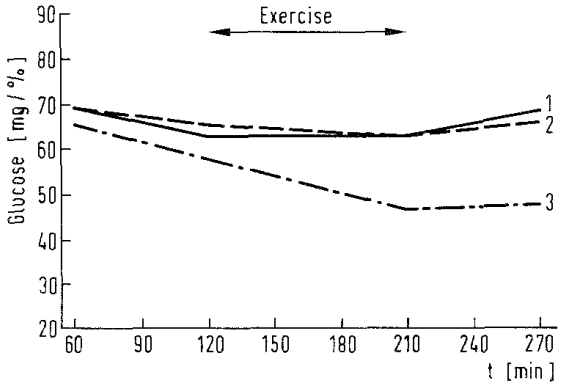


Fig. 3. Mean blood glucose values and statistical analysis in rest (*1*), exercise with normal food diet (*2*) and exercise after carbohydrate reduced diet (*3*)

60 min	II	III
I	–	–
II		–

120 min	II	III
I	–	–
II		–

210 min	II	III
I	–	–
II		–

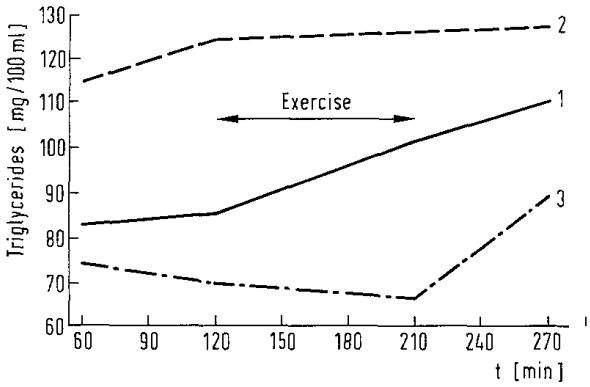
270 min	II	III
I	–	+
II		–

$- p > 0.05$; $+ p < 0.05$

Beginning 60 min after a drink, alcohol concentration was measured in venous blood at 30-min intervals. Serum glucose, triglycerides, glycerol and free fatty acids were measured after 60, 120, 210, and 270 min. Ethanol and free fatty acids were determined by gaschromatography; serum glucose, triglycerides and glycerol by special test combinations (Boehringer GmbH, Mannheim, FRG).

Results

1. In contrast to the period of rest (Test I) there was an enhanced ethanol elimination during physical exercise in Test II ($p < 0.001$) and Test III ($p < 0.01$). A comparison of the alcohol elimination results showed that a carbohydrate reduced diet produced slightly lower values than a normal diet.



60 min	II	III
I	+	−
II		++

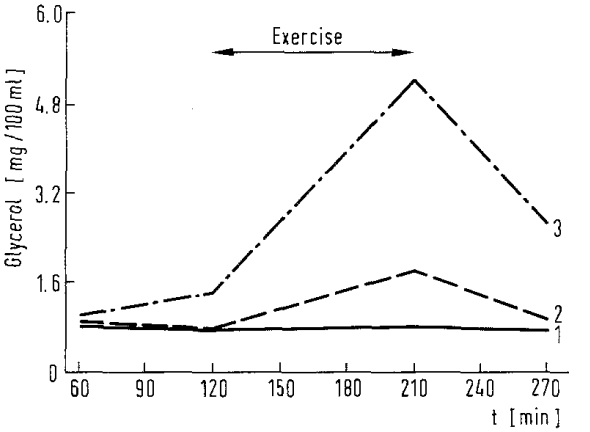
120 min	II	III
I	+	−
II		++

210 min	II	III
I	−	+
II		++

270 min	II	III
I	−	+
II		−

$-\ p > 0.05$; $+\ p < 0.05$; $++\ p < 0.01$

Fig. 4. Mean triglyceride values and statistical analysis in rest (*1*), exercise with normal food diet (*2*) and exercise after carbohydrate reduced diet (*3*)



60 min	II	III
I	−	−
II		+

120 min	II	III
I	−	−
II		−

210 min	II	III
I	+++	+++
II		+++

270 min	II	III
I	−	+
II		+

$-\ p > 0.05$; $+\ p < 0.05$; $++\ p < 0.01$; $+++\ p < 0.001$

Fig. 5. Mean glycerol values and statistical analysis in rest (*1*), exercise with normal food diet (*2*) and exercise after carbohydrate reduced diet (*3*)

During the period after the physical exercise tests faster alcohol elimination than during Test I was observed (Fig. 1).

It was also shown that the physical exercise induced an enhanced ethanol elimination greater than the one found during the period after physical exercise ($p < 0.01$). The slower elimination during the period 210–270 min in the resting experiment was not significant (Fig. 2).

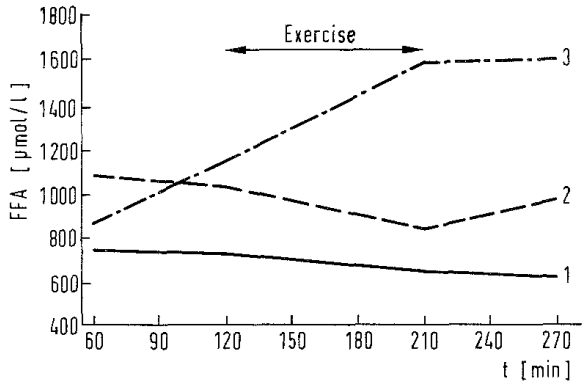


Fig. 6. Mean values of free fatty acids (all differences $p > 0.05$) in rest (*1*), exercise with normal food diet (*2*) and exercise after carbohydrate reduced diet (*3*)

2. In contrast to the other two experiments, serum glucose decreased when a carbohydrate reduced diet had been given. Due to the large standard deviation the differences remained insignificant (Fig. 3).
3. After physical exercise in Test III lower values of serum triglycerides were observed than in Test II (Fig. 4).
4. During rest the serum glycerol values remained constant. However, during physical exercise, we discovered a significant increase. The additional distinctive increase in Test III was very high (Fig. 5).
5. Due to the large standard deviation, no significant difference could be found in free fatty acids. During rest and Test II free fatty acids tended to decrease, whereas in Test III there was a tendency to rise (Fig. 6).

Discussion

Physical exercise of sufficient duration and intensity enhances elimination of blood alcohol [4, 5]. A carbohydrate reduced diet had no additional effect. The reason that some authors have not found changes in ethanol elimination is probably due to physical exercise lacking enough intensity or duration. The elimination rates of our subjects were within the range of published values [16]. In addition, the increased elimination rates during physical exercise were in the highest range of values contained in available literature and therefore no pathological effect could be found. The enhanced ethanol elimination leads to several hypotheses:
1. Physical exercise accelerates alcohol oxidation in the muscles directly or indirectly.
2. Physical work increases body temperature [18]. This increased body temperature increases metabolic rate as well as an increased oxidation of alcohol.
3. In addition alcohol is eliminated through the skin and by exhalation.

The hypotheses are discussed in the following section.

It is known that fructose ingestion increases alcohol reduction [16]. Fructose leads to an increased reoxidation of cytoplasmatic $NADH_2$, which seems to be a limiting factor for ethanol oxidation [16]. This statement is proved by the atypical alcohol dehydrogenase, with five-fold higher activity in vitro and nearly normal alcohol elimination rates in vivo [17].

An accelerated ethanol oxidation is also found in people accustomed to alcohol [10]. A "microsomal ethanol oxidizing system" is oxidizing ehtanol in addition to ADH. Such an adaptation needs higher doses of alcohol over several weeks. Our subjects were not accustomed to alcohol. Moreover half of them passed Test I after the working tests.

Very high ethanol elimination rates have been described by Zuppinger et al. [19] in patients with glycogenosis type I (glucose-6-phosphatase deficiency). This enzyme deficiency causes a disturbance of carbohydrate metabolism. The liver is not capable of delivering glucose to the blood. We tried to simulate this disturbance of carbohydrate metabolism. Muscular activity [6] and alcohol [16] induced a glycogen depletion in muscles and liver. The carbohydrate-reduced diet kept glycogen stores at a low level. During prolonged physical exercise, oxidation of free fatty acids usually increases with sinking glycogen reserves [8]. But on the other hand some authors discovered that β-oxidation of free fatty acids is reduced by alcohol, at least in the liver [16]. If alcohol is inhibiting oxidation of free fatty acids in the muscles as well, there would be the theoretical possibility that acetate originating from ethanol could be used as fuel for muscular work and could produce an enhanced elimination. A direct oxidation of ethanol by the muscles has not been demonstrated [16]. Since the metabolism responds to carbohydrate reduction with increased fat mobilization both in the presence and absence of alcohol, a major indirect utilization of ethyl alcohol by the muscles appears to be excluded.

Results from physical exercise coupled with a carbohydrate reduced diet showed that ethanol elimination was slightly decreased as against the results of exercise together with a normal diet. A number of authors found similar results at rest [16].

There is a possibility that increased body temperature induced by physical exercise provokes an enhanced ethanol oxidation by increased enzyme activity in the liver. Nevertheless, liver blood flow is reduced during the most intensive work loads. Moreover, further alcohol could be eliminated by additional loss through perspiration or exhalation. A significant loss of ethanol in the urine can be excluded.

The small reduction in serum glucose level at rest and in Test II can be induced by alcohol alone [16] or by prolonged physical exercise with or without the influence of ethanol [2]. Mostly constant glucose levels exist in endurance trained athletes during physical exercise lasting for several hours [3]. A falling serum glucose is a sign of reduced reserves of liver glycogen. In three not endurance-trained subjects we found values below 30 mg/100 ml. Also, without advanced glycogen exhaustion, prolonged physical exercise in combination with alcohol may produce hypoglycemia [7]. Alcohol reduces glucose mobilization of the liver during physical exercise [7]. The role of alcohol seems to be

predominant, since similar work tests with carbohydrate reduced diets alone did not provoke hypoglycemia [15], even though after exhaustive prolonged exercise and carbohydrate restriction extremely low glucose values are possible [1].

Results on triglycerides are contradictory. This concerns the influence of alcohol [16] as well as of physical activity. In contradiction to findings of decreased neutral fats during exercise [15] there are reports of unchanged triglyceride levels [8]. Neutral fat values during rest are reduced by low carbohydrate diets [14]. During exercise, falling or rising triglyceride levels are possible [15].

Fat oxidation can be assessed by serum glycerol and free fatty acids. Intensity and duration of exercise, state of training and – due to their short stability – blood tests show very different reactions of the free fatty acids when they are made at different time intervals [8]. On the other hand, the glycerol level rises during prolonged exercise [8] and is a good indicator of metabolized triglycerides. The development of glycerol proves that the metabolism is able to increase fat mobilization at times of extreme carbohydrate deficiency despite ethanol influence. This mechanism does not perform so well in non endurance-trained athletes, and so low serum glucose levels are possible. Endurance training induces an enhanced fat mobilization [11] and glucose saving [13]. This is the reason why we did not discover any glucose reduction in our well trained subjects, and no correlation between glucose- and glycerol levels was found.

Thus we can summarize our conclusions as follows: Prolonged physical exercise produces an enhanced ethanol elimination if the intensity and duration of exercise are sufficient. But this finding has hardly any pathological meaning. Prior to the tests almost no influence of diet could be found. The reasons for the enhanced elimination of blood alcohol are probably to be found in the elevated body temperature caused by physical exercise and in a supplementary loss of alcohol by perspiration and exhalation. The muscles are not able to utilize ethanol either directly or indirectly. Even after alcohol consumption, fat mobilization and oxidation increase during physical exercise and glycogen deficiency. Fat metabolism can be activated sufficiently only in well trained endurance athletes. In non-trained subjects physical exercise together with low glycogen stores can provoke hypoglycemia.

Acknowledgement. We wish to thank Mrs. B. Krüger, Mrs. E. Schürch, Mr. R. Föhrenbach, and Mr. R. Krischer for their technical help.

References

1. Bergström J, Hultman E (1967) A study of the glycogen metabolism during exercise in man. Scand J Clin Lab Invest 19: 218–227
2. Christensen EH, Hansen O (1939) Arbeitsfähigkeit und Ernährung. Scand Arch Physiol 81: 160–171
3. Gollnick PD, Januzzo CD, Williams C, Hill TR (1969) Effect of prolonged severe exercise on the ultrastructure of human skeletal muscle. Int Z Angew Physiol 27: 257–265
4. Hebbelink M (1959) Influence of muscular work on the blood alcohol concentration in man. Arch Int Pharmacol 119: 521–523

5. Hecksteden W, Fehler W (1942) Über den Einfluß körperlicher Arbeit auf die Geschwindigkeit der Umsetzung von Alkohol im menschlichen Körper. Dtsch Z Gerichtl Med 36: 311–318
6. Hultman E (1967) Physiological role of muscle glycogen in man, with special reference to exercise. Circ Res [Suppl 1] 21/22: 99–114
7. Juhlin-Dannfeldt A, Ahlborg G, Hagenfeldt L, Jorfeldt L, Fehlig P (1977) Influence ot ethanol on splanchnic and skeletal muscle substrate turnover during prolonged exercise in man. Am J Physiol 233: 195–202
8. Keul J, Doll G, Keppler D (1969) Muskelstoffwechsel. Barth, München
9. Krauland W, Mallach HJ, Mellerowicz H, Miller J (1965) Über das Verhalten des Blutalkoholspiegels unter dem Einfluß körperlicher Arbeit. Blutalkohol 3: 63–75
10. Misra PS, Lefèvre A, Ishi H, Rubin E, Lieber CS (1971) Increase of ethanol, meprobamate and phenobarbital metabolism after chronic ethanol administration in man and in rats. Am J Med 51: 346–351
11. Molé TE, Holloszy JO (1970) Exercise induced incrase in the capacity of skeletal muscle to oxidize palmitate. Proc Soc Exp Biol Med 134: 789–792
12. Newman HW, Yee J (1947) Effect of electrically induced convulsion on rate of alcohol metabolism in man. Proc Soc Exp Biol Med 65: 122–123
13. Paul P, Holmes WL (1975) Free fatty acid and glucose metabolism during increased energy expenditure and after training. Med Sci Sports 3: 176–184
14. Rabast U, Kasper H, Schönborn J (1975) Zur Frage der kohlenhydratarmen, relativ fettreichen Diät in der Adipositastherapie. Med Klin 70: 653–657
15. Schürch PM, Reinke A, Hollmann W (1979) Kohlenhydratarme Diät und Metabolismus. Med Klin 1279–1285
16. Wallgren H, Barry H (1970) Actions of alcohol. Elsevier, Amsterdam
17. von Wartburg JP, Schürch PM (1968) Atypical human liver alcohol dehydrogenase. Ann. NY Acad Sci 151: 936–946
18. Wyndham CH, Strydom NB (1972) Körperliche Arbeit bei hoher Temperatur. In: Hollmann W (Hrsg) Zentrale Themen der Sportmedizin. Springer, Berlin Heidelberg New York
19. Zuppinger K, Papenberg J, Schürch P, von Wartburg JP, Colombo JP, Rossi E (1967) Vermehrte Alkoholoxidation bei der Glykogenose Typ I. Schweiz Med Wochenschr 97: 1110–1117

Accepted October 26, 1981