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Consequences of 6 weeks of strength training on red cell O_2 transport and iron status

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Summary. Effects of endurance training on O_2 transport and on iron status are well documented in the literature. Only a few data are available concerning the consequences of strenuous anaerobic muscular exercise on red cell function. This study was performed to test the influence of strength training alone on parameters of red cell O_2 transport and iron status. Twelve healthy untrained males participated in a strength-training programme of 2-h sessions four times a week lasting 6 weeks. After 6 weeks a small but significant reduction of haemoglobin (Hb; $-5.4\ g \cdot l^{-1}$) was found ($p<0.05$). Mean red cell volume did not change, but a pronounced decrease of mean cell Hb concentration (from $329.2\ g \cdot l^{-1}$, SE 2.5 to $309.8\ g \cdot l^{-1}$, SE 1.2; $p<0.001$) and mean corpuscular Hb (from 29.6 pg, SE 0.4 to 27.7 pg, SE 0.3; $p<0.01$) was observed. Serum ferritin decreased significantly by 35% ($p<0.01$); transferrin, serum iron and iron saturation of transferrin were unaltered. Serum haptoglobin concentration was diminished significantly by 30.5% ($p<0.01$). The reticulocyte count had already increased after 3 weeks of training ($p<0.05$) and remained elevated during the following weeks. Strength training had no significant influence on the O_2 partial pressure at which Hb under standard conditions was 50% saturated, red cell 2,3-diphosphoglycerate and ATP concentration as well as on erythrocytic glutamate-oxalacetate transaminase activity. The data demonstrate that mechanical stress of red cells due to the activation of large muscle masses led to increased intravascular haemolysis, accompanied by a slightly elevated erythropoiesis, which had no detectable influence on Hb-O_2 affinity. Training caused an initial depletion of body iron stores (prelatent iron deficiency). Although Hb had decreased by the end of the training phase a true "sports anaemia" could not be detected.

Key words: Training - Haemolysis - Iron status - O_2 dissociation curve - 2,3-Diphosphoglycerate

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Introduction

Effects of endurance training on red cell O_2 transport and on iron status are well documented. Changes due to long-lasting physical training are an increase in blood volume arising from increases in plasma volume (Röcker et al. 1976), total red cell mass and total amount of haemoglobin (HB) (Kjellberg et al. 1949; Holmgren et al. 1969; Brotherhood et al. 1975; Remes et al. 1979). In most cases the increase in plasma volume exceeds that of total red cell mass, causing a reduction in blood haemoglobin concentration ([Hb]) and packed cell volume (PCV). Low PCV and Hb values as a result of physical training are termed in the literature as "sports anaemia". Besides haemodilution, other reasons for low [Hb] in physically active persons are a reduced production of Hb as a consequence of protein (Yoshimura et al. 1980) or iron deficiency (Hunding et al. 1981; Clement and Sawchuk 1984) and an enhanced rate of red cell destruction with accompanying haemolysis.

As described in middle- and long-distance runners (Dufaux et al. 1981; Hunding et al. 1981) the reason for haemolysis may be a mechanical compression of mainly old red cells in capillaries of the sole of the foot and stress on their membranes due to increased blood flow and blood pressure, both disturbing the red cell membrane integrity. Haemolysis, by reducing O_2 transport capacity, acts as a stimulus for erythropoiesis. This leads to a higher proportion of young red cells in the circulation, which exhibit higher concentrations of 2,3-diphosphoglycerate (2,3-DPG) and a lower affinity of Hb for O_2, thus facilitating O_2 uncoupling in the periphery. These phenomena have only been described as effects of endurance sports such as long-distance running (Rand et al. 1973; Böning et al. 1975; Brodthagen et al. 1985), cycling (Veicsteinas et al. 1984) and swimming (Hasibeder et al. 1987).

If mechanical stress is one of the main reason for erythrocyte destruction, activation of large muscle masses as during strength training should also develop haemolysis with its consequences. The aim of this study

was to investigate the influence of 6 weeks of strength training alone on the following parameters, i.e. red cell oxygen transport, iron status and physical work capacity on untrained individuals.

Methods

Subjects. Twelve healthy, non-smoking males participated in this study (mean age 23 years, SD 2; mean height 181 cm, SD 2; mean mass 73 kg, SD 7.4). After the subjects had been familiarized with the test procedures and the test protocol, their written consent was obtained.

Training programme. All volunteers participated in a strength-training programme which lasted 6 weeks. Training was undertaken in at least 2-h sessions four times a week. Before each training session they underwent a warm-up cycle of about 20 min. Strength training consisted of different elements, including exercises for improving maximal strength as well as muscular endurance. For this the repetition maximum method was applied; load was chosen in such a way that the participants could perform between 12 and 16 repetitions. This corresponded to about 50% of their maximal load. In each training session at least 10 drills which exercised the main muscle groups of the body were carried out. To exclude the possibility of influences from aerobic training overlapping with those of strength training, the individuals were asked not to engage in endurance sports like running or cycling during the 6-week period of training.

Physiological tests. To evaluate the effect of strength training on maximal strength and muscular power an exercise test was performed with the Cybex II plus-dynamometer (Lumex, Ronkonkoma, NY, USA) according to standard methods (Davies 1985) before and after the 6-week training period. To represent training induced changes the power of 25 repetitions of knee extension and flexion was measured on both sides.

In addition a two-step spiro-ergometric cycle test on a mechanically braked cycle ergometer (Monark-Crescent, Varburg, Sweden) was performed before and after the training period. A first exercise intensity of 2 W·kg body mass^{-1} for 4 min was chosen to reach a steady state. At a second exercise intensity of 4W·kg body mass^{-1} the participants were subjected to exercise until exhaustion. During the tests parameters of ventilation (body temperature, pressure, and saturation), oxygen consumption ($\dot{V}_{O_2}$) (standard temperature and pressure, dry; Ergo-Oxyscreen, Jaeger, Würzburg, FRG) and heart rates (ECG, Siemens Elema, Solna, Sweden) were recorded. Blood lactate concentration ([la_b]), using a test kit from Boehringer (Mannheim, FRG), was determined from blood samples collected from the hyperaemic ear lobe at rest, at the end of the first exercise intensity as well as upon exhaustion.

Haematological tests. Before and after 3 and 6 weeks of training venous blood samples from an antecubital vein were collected between 0800 hours and 1000 hours after overnight fasting. The sample was taken after 15 min rest with the subject in the supine position. Immediately afterwards an aliquot of the blood was deproteinized with ice-cold 0.6 N perchloric acid. In these samples the concentration of 2,3-DPG was measured at 30° C with an UV-test kit (No. 35-UV) from Sigma (St. Louis, Mo., USA); ATP concentrations were determined with test kits from Boehringer (Mannheim, FRG). For the spectrophotometric determination of both 2,3-DPG and ATP a DU-6 spectrophotometer (Beckman, Irvine, Calif., USA) was used.

As a measure of the O_2 affinity of Hb the O_2 partial pressure at 50% saturation under standard conditions (P_{50st}, pH = 7.4, PCO_2 = 40 mmHg, Bohr coefficient = 0.48) was determined using the microaequilibration technique of Astrup et al. (1956). Further details have been described in an earlier publication (Mairbäurl et al. 1986).

The [Hb] and PCV were determined by standard haematological methods. Red cell counts (RBC) were determined with a Coulter Counter, model DN (Coulter, Harpenden, UK); reticulocytes were counted in blood smears after 10-min staining with new methylene blue (Sigma, St. Louis, Mo., USA); at least 1000 RBC were counted per smear. Mean cellular haemoglobin concentration (MCHC), mean corpuscular haemoglobin (MCH) and mean red cell volume (MCV) were calculated according to standard formulae.

The activity of red cell enzyme glutamate-oxalacetate transaminase (GOT) was determined spectrophotometrically in diluted freeze-thaw haemolysates of washed erythroctes. Contamination with white blood cells was reduced below 300 per millions of erythrocytes by filtration through a mixture of α-cellulose and microcrystalline cellulose (Sigma, St. Louis, Mo., USA) according to Beutler (1975).

Serum iron was measured photometrically by the ferrozin method; serum transferrin determination was carried out turbidimetrically with antibodies from Kallestad Laboratories (Houston, Tex., USA) and serum ferritin was evaluated by radioimmunoassay (Becton and Dickinson, Orangeburg, NY, USA). Iron saturation of transferrin was calculated as $\left(\frac{[\text{Iron}] \times 100}{\text{transferrin} \times 0.26}\right)$.

Serum haptoglobin was determined with Nor-partigen immunodiffusion-plates (Behring, Vienna, Austria) using the method of Mancini et al. (1965).

Statistical evaluation. Parameter-free analysis of variance for coupled samples according to Friedman was used to discern overall effects, followed by the Wilcoxon signed rank test to locate specific effects. The data presented in the text and tables are mean values and SD; data in the figures are mean values and SE. The level of significance was $p < 0.05$.

Results

Exercise tests

After 6 weeks of training maximal power in the Cybex test had increased for all conditions measured. Power of the knee extension increased in the right knee from 245 W, SD 44 to 287 W, SD 49 ($p < 0.01$) and in the left knee from 225 W, SD 43 to 262 W, SD 50 ($p < 0.01$). The power of the knee flexion was elevated from 119 W, SD 26 to 159 W, SD 38 at the right side ($p < 0.01$) and from 97 W, SD 35 to 125 W, SD 37 ($p < 0.01$) at the left side.

Table 1. Heart rates (HR), oxygen consumption ($\dot{V}_{O_2}$), ventilation ($\dot{V}_E$), blood lactate concentration (la_b) at Rest (R), during submaximal (S) and maximal (M) exercise before (0) and after (6) the 6 weeks of training ($n = 12$). * $p < 0.05$, 0 versus 6

Condition	HR ($l \cdot min^{-1}$)		$\dot{V}_{O_2}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)		$\dot{V}_E$ ($l \cdot min^{-1}$)		[la_b] ($mmol \cdot l^{-1}$)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
R0	78	14	4.8	1.4	7.4	1.9	1.2	0.5
R6	77	13	4.6	1.0	6.9	1.4	0.9	0.3
S0	158	10	33.5	4.7	58.5	8.6	3.9	1.3
S6	150	8	32.0	4.3	60.1	7.0	3.4	1.1*
M0	192	7	50.4	5.1	141.2	25.6	12.9	1.8
M6	192	8	51.9	5.4	154.4	26.9*	12.4	1.9

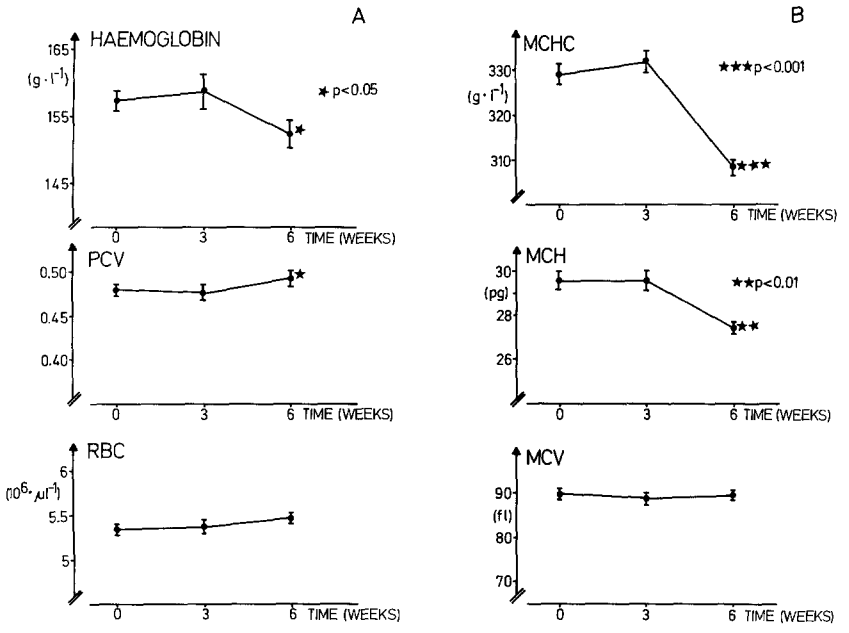

Fig. 1A, B. Haemoglobin concentration, packed cell volume and red blood count (*RBC*) (**A**) as well as erythrocytic indices *MCHC* (mean corpuscular haemoglobin concentration), *MCH* (mean corpuscular haemoglobin) and *MCV* (mean red cell volume) (**B**) before (0), after 3 weeks and after 6 weeks of strength training ($n = 12$). Values are plotted as means and SE. * $p < 0.05$, 0 vs 6 weeks; ** $p < 0.01$, 0 vs 6 weeks; *** $p < 0.001$, 0 vs 6 weeks

Since body mass did not change during training exercise intensity in the two-step cycle ergometer test remained unaltered (146 W and 293 W, respectively). The duration of exercise on the 4 $W \cdot kg^{-1}$ exercise intensity was 197 s, SD 81 before and 300 s, SD 98 after training ($p < 0.01$). Heart rate and $\dot{V}_{O_2}$ at rest, at submaximal exercise were similar before and after training (Table 1). Submaximal ventilation was the same before and after training, but at exhaustion ventilation had elevated significantly by 9.4% in the post-training tests. The [la_b] at rest and during maximal work was not affected by training. In contrast, a significantly lower submaximal [la_b] was found after training.

Red cell O_2 transport and iron status

In Fig. 1A [Hb], PCV and RBC before, in the middle and at the end of training are shown. After 6 weeks a small but significant reduction in [Hb] from 157.6 $g \cdot l^{-1}$, SE 1.5 to 152.2 $g \cdot l^{-1}$, SE 2.0 was found. PCV had increased significantly from 0.479, SE 0.006 to 0.493, SE 0.006 accompanied by a slight, but not significant elevation in RBC ($5.32 \times 10^6 \cdot \mu l^{-1}$, SE 0.07 vs $5.49 \times 10^6 \cdot \mu l^{-1}$, SE 0.07. Red cell indices are presented in Fig. 1B. While MCV did not change, a pronounced decrease of both MCHC (329.2 $g \cdot l^{-1}$, SE 2.5 vs 309.8 $g \cdot l^{-1}$, SE 1.2; $p < 0.001$) and MCH (29.6 pg, SE 0.4 vs 27.7 pg, SE 0.3; $p < 0.01$) was observed at the end of the 6 weeks.

Table 2 shows the influence of strength training on iron status. Serum ferritin decreased significantly by 35% after training. Serum transferrin, serum iron and iron saturation of transferrin exhibited no alteration.

Table 2. Iron status before, after 3 weeks and after 6 weeks of strength training ($n = 12$)

	Before		3 weeks		6 weeks	
	Mean	SD	Mean	SD	Mean	SD
Ferritin ($\mu g \cdot l^{-1}$)	74.8	53.9	57.8	45.0	49.3	38.2**
Transferrin ($mg \cdot dl^{-1}$)	280.3	36	271.2	45.9	280.5	34.1
SE-fe ($\mu mol \cdot l^{-1}$)	17.3	6.3	16.3	4.3	15.3	6.5
Fe-Sat (%)	35.6	15.6	34.5	12.9	30.5	15.2

SE-fe = Serum iron; Fe-Sat = iron saturation of transferrin
** $p < 0.01$, before vs 6 weeks

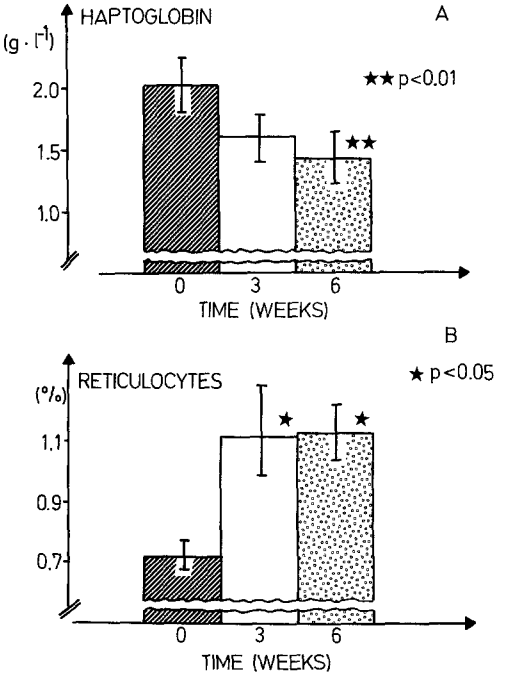

Fig. 2A, B. Serum haptoglobin concentration (**A**) and reticulocyte counts (RBC) (**B**) at the beginning (0), after 3 and after 6 weeks of strength training ($n = 12$). Values are plotted as means and SE. * $p < 0.05$, 0 vs 3 and 6 weeks; ** $p < 0.01$, 0 vs 6 weeks

Serum haptoglobin concentration was unchanged after 3 weeks of training, but it was diminished significantly by 30.5% after the 6 weeks of training (Fig. 2A). The reticulocyte count increased from 0.72%, SE 0.06 to 1.08%, SE 0.16 after 3 weeks and to 1.11%, SE 0.11 after 6 weeks ($p < 0.05$) (Fig. 2B).

Strength training had no significant influence on P_{50st} (mean = 3.62 kPa, SD 0.09), erythrocyte 2,3-DPG (mean = 13.6 $\mu mol \cdot gHb^{-1}$, SD 0.9) and ATP concentration (mean = 598.1 $\mu mol \cdot l^{-1}$, SD 71.5) or on red cell GOT activity (mean = 3.31 $IU \cdot gHb^{-1}$, SD 0.13).

Discussion

The 6 weeks of strength training were sufficient to increase maximal power during the Cybex dynamometer tests. The increment in maximal power was about 17% for knee extension and about 31% for knee flexion. So the training response of the knee flexors was significantly greater than that of the knee extensors ($p < 0.05$).

All participants could exercise at the second stage of the cycle ergometer test for a longer time after training (+51%). Heart rate and $\dot{V}_{O_2}$ upon exhaustion were unchanged in the post-training test, so the improvement in duration of exercise may be explained by an amelioration of muscle coordination.

The [Hb] did not change after 3 weeks of training, but at the end of the strength training phase a significant decrease in [Hb] was observed (Fig. 1A). The reduction of 5.4 $g \cdot l^{-1}$, although statistically significant, is too small to interpret as "sports anaemia". This small decrement in [Hb] after training is in contrast to the pronounced depression either in highly endurance trained athletes described by Hunding et al. (1981) and Pate (1983) or in untrained subjects observed within the first days after the beginning of a regular training programme (Yoshimura et al. 1980; Lindemann 1978). This early detection of suboptimal [Hb] of non-athletes is mainly explained by haemodilution, resulting from an increase in plasma volume without an accompanying elevation of red cell mass (Convertino et al. 1980; Schmidt et al. 1988). Convertino et al. (1980) found plasma hypervolaemia after 8 days of cycle ergometer training, which was explained by the high levels of renin activity and vasopressin during exercise and a progressive increase in plasma albumin content. The increment in plasma protein mass and plasma protein concentration seems to be an early response to physical training, reaching its plateau after 2 weeks (Schmidt et al. 1988). Thereafter, the increase in plasma volume tends to over-ride the plasma protein concentration. In our study the decrease of [Hb] was not a rapid response to strength training. Thus, since PCV and RBC tended to increase at the end of the training phase, it seems obvious that reasons other than haemodilution were responsible for [Hb] reduction.

As well as for other reasons low [Hb] are observed as a consequence of iron deficiency. Serum ferritin, transferrin, serum iron and iron saturation of transferrin were unchanged after 3 weeks of training. After 6 weeks a significant decrease of ferritin of 25.5 $\mu g \cdot l^{-1}$ was observed (Table 2). Although statistically not significant serum iron and iron saturation of transferrin tended to decrease as the training phase progressed. Serum ferritin concentration serves as a parameter for evaluating the size of body iron stores (Lipschitz et al. 1974) and in healthy people has been shown to be directly proportional to the body iron stores. The critical ferritin concentration indicating the beginning of iron depletion is about 60 $\mu g \cdot l^{-1}$ (Heinrich et al. 1977). Levels below 20 $\mu g \cdot l^{-1}$ are associated with the absence of iron in the bone marrow. In our study five subjects had serum ferritin values below 30 $\mu g \cdot l^{-1}$, which points to a pronounced reduction in their iron stores. In addition, in three of these subjects subnormal serum iron concentrations (< 10 $\mu mol \cdot l^{-1}$) were also found. Since both MCHC and MCH were significantly reduced at the end of the training phase (Fig. 1B) and since these parameters are reduced with iron deficiency, the situation of a low serum ferritin concentration and hypochromia with [Hb] within the normal range can be interpreted as a depletion of iron stores and prelatent iron deficiency, respectively (Cook 1982). The possibility of body iron depletion and low serum iron is a well documented phenomenon in endurance training (Hunding et al. 1981). Besides an inadequate nutritional iron intake other possible explanations are:

1. An increased rate of intravascular haemolysis and accelerated destruction of erythrocytes leading to a stimulation of erythropoiesis and to an increased iron requirement (Lindemann 1978; Yoshimura et al. 1980)
2. An increased iron demand from myoglobin and several iron-containing respiratory enzymes
3. A depressed intestinal iron absorption (Ehn et al. 1980)
4. An accelerated iron elimination and loss of iron due to profuse sweating (Paulev et al. 1983), faecal loss (Stewart et al. 1984), haemoglobinuria and haematuria (Siegel et al. 1979).

In contrast to our finding of a gradual development of prevalent iron deficiency, Maganzanik et al. (1988) reported a very rapid depletion of serum iron concentrations and low ferritin values in non-athletes following only 2 weeks of intensive physical exercise. An explanation of these differences could be that during the latter study more intensive training was performed by using aerobic as well as anaerobic elements and also that females were studied, who are more susceptible for iron depletion. To the best of our knowledge, this study is the first report of a prelatent iron deficiency after several weeks of exclusively anaerobic strength training.

Normally, consequences of iron deficiency, even without anaemia, are low levels of physical work capacity and excess lactate formation (Finch et al. 1979; Nilson et al. 1981). Since, in our study, endurance time was improved after the training period, maximal [la_b] remained unchanged and submaximal [la_b] was even reduced, the degree of body iron depletion was not sufficient to induce excess lactate formation and influence work capacity negatively.

Another result of strength training was a reduction in plasma haptoglobin concentration. In the middle of the training period haptoglobin was slightly reduced, but at the end of 6 weeks a pronounced decrease of 31% was observed. In general it is accepted that reduced haptoglobin values indicate chronic intravascular haemolysis. Haemolysis is an often described phenomenon in long-distance runners mainly thought to be the result of traumatic compression of old red cells in capillaries of the sole of the foot (Hunding et al. 1981; Lindemann 1978). Since in this study the diminished haptoglobin concentration cannot be due to red cell haemolysis primarily in plantar vessels, the activation of large muscle masses in strength training seems to lead to haemolysis in capillaries of different skeletal muscles. Moreover, other factors such as high levels of adrenalin and lysolecithin, which increase in plasma during strenuous muscular exercise and are able to alter membrane integrity, should be taken into consideration as possible "haemolytic agents" (Yoshimura et al. 1980). Red cell haemolysis during strength training activated erythropoiesis as indicated by significantly elevated reticulocyte counts after 3 and 6 weeks training (Fig. 2B). Although this increase was statistically significant, the overall activation of red cell production was moderate, as indicated by the reticulocyte counts, which were within the normal range.

As described in long-distance runners (Rand et al. 1973; Böning et al. 1975; Brodthagen et al. 1985), swimmers (Hasibeder et al. 1987) and cyclists (Veicsteinas et al. 1984), endurance-trained athletes exhibit higher concentrations of red cell 2,3-DPG concentration and a reduced Hb-O_2 affinity (i.e. high P_{50} values). In our study strength training did not influence 2,3-DPG and Hb-O_2 affinity. Also red cell GOT activity, known as a very sensitive indicator of red cell age (Fisher and Walter 1971), remained unaltered, indicating that mean red cell age did not change. At present it is difficult to explain the unchanged O_2 binding properties after strength training. Katz et al. (1984) demonstrated that high-intensity anaerobic interval training for 8 weeks had no significant effect on erythrocyte 2,3-DPG levels. So it might be that anaerobic training is less efficient in affecting red cell 2,3-DPG and Hb-O_2 affinity than predominantly aerobic types of exercise.

In summary, the data demonstrate that mechanical stresses on red cells induced through contraction of large muscle masses led to increased intravascular haemolysis and slightly elevated erythropoiesis, which had no detectable influence on O_2 binding properties of Hb. In addition, a depletion of body iron stores began to develop. Although haemoglobin decreased at the end of training a pronounced "sports anaemia" was not observed.

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