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## Short-Term Effects of Prolonged Strenuous Endurance Exercise on the Level of Haematocrit in Amateur Cyclists

### Abstract

Knowledge is sparse about the extent of potential dehydration due to prolonged strenuous cycling and its haematological acute effects on the haematocrit (Hct) in study populations credibly not taking any kind of doping. With increasing training load levels of Hct and haemoglobin (Hb) decrease in both amateurs and professionals as a long-term consequence due to expanded plasma volume (PV). On a short-term basis, however, counteracting dehydration potentially brought about by endurance exercise may cause a rise in Hct bringing competitive cyclists into conflict with the current condition regulations and Hct cut-off of 50% set by the International Cycling Union (UCI) in its fight against erythropoietin (rhEPO) doping. On the other hand adequate and sufficient fluid substitution being substantial for a successful endurance performance should prevent any pronounced Hct rises. To study the haematological acute effects of prolonged strenuous cycling we measured Hct, Hb, red blood cell (RBC) count and plasma protein in a reliably 'clean' population of 38 well-trained male amateur cyclists before, immediately after and one day after an extraordinary ultramarathon. The pre-race levels of Hct, Hb and RBC count were placed in the lower range of normal dis-

tribution and well below the Hct cut-off limit of the UCI. Immediately post-exercise the mean levels of Hct, Hb, RBC count and protein remained unchanged. One day after race, however, all four parameters significantly dropped by 3%, 6.7%, 6.5%, 9.9% respectively ( $p < 0.001$ ), indicating marked post-exercise PV expansion. The calculated percentage increase in PV was 11.9%. No evidence for coexisting exercise-induced haemolysis was found. Our study shows that in "clean, rhEPO-free" amateur cyclists who involve in strenuous marathon cycling the haematological short-term effects of extraordinary marathon cycling consist in considerable PV expansion making Hct values fall on the following day. The findings - gained from amateurs though - suggest that despite all its disadvantages the UCI Hct cut-off represents an appropriate means to discourage from excessive rhEPO doping at least as long as the available direct methods for detecting this kind of misuse are not yet applied by the international sports federations.

### Key words

Haematocrit · plasma volume · cycling · endurance sports · erythropoietin doping.

### Introduction

The aerobic energy metabolism plays the crucial role for the performance in endurance sports. Within the oxygen transport chain an enhanced red blood cell (RBC) mass improves the oxygen supply to working muscles which could increase the ath-

lete's aerobic performance capacity [6,14,18]. Both RBC mass and plasma volume (PV) are enlarged by regular endurance training taking several months to occur [8,15,28]. Due to the more pronounced PV expansion haemoglobin (Hb) concentration and haematocrit (Hct) level fall as a long-term consequence of aerobic training during the athlete's competitive season

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[7,20]. The introduction of recombinant human erythropoietin (rhEPO) in 1987 made an artificial increase in RBC mass and a correction of this “physiological Hct decrease” possible.

So far, direct methods for the detection of rhEPO misuse have not been applied by the various international sports federations [11,26]. The move by the International Cycling Union (UCI) in 1997 to exclude subjects with high Hct levels from competition for health reasons stimulated a lot of controversial discussion [1,12]. In this UCI condition regulation a Hct cut-off (>50%) and random blood testing before competition were introduced to protect athletes from health risks due to rhEPO doping [2]. It was argued that this cut-off could lead to an unacceptably high percentage of false positives partly based on natural occurrence or due to temporary dehydration as often occurs during long-term competitions [10,12,23].

On a short-term basis, the Hct level is known to be influenced in both directions by various external factors but especially by the mode, intensity and duration of exercise [15,27,28]. Whereas maximal short-term exercise generally leads to haemoconcentration, long-term exercise can cause both – haemodilution [4,9,16] and – concentration under special conditions of dehydration [3,13]. Knowledge is sparse about the extent of potential dehydration due to prolonged cycling and its haematological short-term impacts of prolonged cycling on the Hct in study populations credibly not taking any kind of doping [18,19]. We therefore sought to investigate the acute effects of marathon cycling on PV and Hct in reliably “rhEPO-free” amateur cyclists taking part in a very challenging one-day cycling race whose workload is comparable to that of professional cycling.

## Subjects and Methods

Thirty-eight male volunteers out of 1420 participants of the Ötz-taler Radmarathon held on August 29th, 1999 in Tyrol were subjects of the study. The efforts of this one-day cycling race are enormous, as illustrated by a total distance of 230 km and an altitude difference of about 5500 m, including 4 mountain passes. The workload of this competition is certainly comparable to that of the hardest mountain stages of the Tour de France or the Giro d' Italia. The race took place under dry and fine weather conditions. During the race, temperatures ranged from 14–21 °C, and humidity from 55–85%.

All 38 study participants were volunteers and experienced amateur cyclists well-prepared for the race. To the best of our knowledge they did not use any kind of doping. All athletes were considered to be healthy according to case history and prior clinical and laboratory investigations. Before the race they were instructed to maintain an adequate fluid intake *ad libitum* being rich in carbohydrates. The amounts of fluid replacement were recorded by the athletes themselves afterwards. Body weight was measured by a Tefal electronic scale immediately before, immediately after and one day after the race.

Blood specimens were taken by venopuncture of a cubital vein the day before, immediately after and one day after the competition and analysed the same day. The venopuncture was done in lying position at the same time in the morning on the day before

and after the race. On the day of competition it was performed in the afternoon immediately after the individual finish. Hb, RBC count and MCV were measured from blood with EDTA in an automated cell counter (Coulter Gene S analyser) while Hct, MCH, MCHC were calculated by using standard formulas. The performance of the instrument was monitored by the Stak-Chex Plus Retics Controls (Streck Laboratories Inc., Nebraska, USA). Plasma protein was determined by the biuret method, lactate dehydrogenase (LDH) by a standard method. The percentage changes in PV (% $\Delta$ PV) were calculated from pre- and post-exercise levels of Hct and Hb according to the equation by Strauss [21].

The changes of the various biochemical markers over the observation period were calculated by the Wilcoxon test and one sample t-test. Correlations between the markers and between them and the athletes' baseline characteristics were calculated by simple linear regression analysis described by the correlation coefficient *r* by using the SPSS software package (version 9.0). Statistical significance was assumed at a level of *p* < 0.05.

## Results

All participating athletes finished the ultramarathon successfully and without any symptoms. After competition almost all of them stated that they had been able to fulfill their personal expectations. The average racing time was 9 h 38 min. The athletes' baseline characteristics and race results are summarized in Table 1.

The pre-exercise values of Hct, Hb and RBC were placed within the lower and middle range of normal distribution. The highest individual Hct level measured was 47%, the highest Hb level 16.6 g/dL. The pre-race protein levels were rather placed in the upper range of normal distribution.

The post-race changes in the levels of Hct, Hb, RBC count and protein showed significant direct correlation. With some individual scattering the mean values of these parameters remained unchanged immediately after race. Ten cyclists showed a rise in Hct >1%, in Hb >0.4 g/L and in protein >0.5 g/L respectively, and

Table 1 Baseline characteristics and race results of the 38 athletes

|                                                         | Mean volume   | SD    | Range       |
|---------------------------------------------------------|---------------|-------|-------------|
| Age (yr)                                                | 35.1          | 7.2   | 24–52       |
| Height (cm)                                             | 179.6         | 7.1   | 164–199     |
| TBW (kg)                                                | 74.0          | 7.1   | 60.9–88.6   |
| BMI (kg/m <sup>2</sup> )                                | 22.94         | 1.77  | 19.86–28.63 |
| Training km 1999                                        | 6350          | 3265  | 1500–15000  |
| Race time (hr)                                          | 9.64          | 0.82  | 8.10–11.05  |
| Total placement                                         | –             | –     | 24–629      |
| TBW difference (kg) [% of TBW] (immediately after race) | –1.72 [–2.3%] | –1.49 | –5.1–+1.0   |
| TBW difference (kg) [% of TBW] (24 hours after race)    | –0.73 [–1.0%] | 0.94  | –3.6–+0.6   |
| Fluid replacement (L)                                   | 5.13          | 1.43  | 2.5–7.3     |

SD = standard deviation, TBW = total body weight, BMI = body mass index

7 cyclists a corresponding decline. In the remaining 21 athletes the changes of Hct, Hb, RBC count and protein were not significant post-exercise. The highest individual increase and decrease in Hct were 3.7 and 3.4%, respectively. The corresponding % $\Delta$ PV lay between -17 and +10 immediately after exercise. There was a significant correlation between the change in Hct and total body weight loss immediately after race ( $r = 0.451$ ;  $p = 0.006$ ) but no correlations were found between the changes in Hct and the racing time, age, training status or the amount of substituted fluid.

One day after the race highly significant decreases in Hct, Hb, RBC count and protein could be observed in all cyclists ( $p < 0.001$ ) (Fig. 1). The corresponding increase in % $\Delta$ PV lay between 2 - 36%. The immediate and short-term changes in the erythrocyte indices, MCV and MCHC were significant whereas they were not so for MCH. Additional laboratory parameters (i.e. LDH) indicating exercise-induced haemolysis remained negative after the marathon. The results of all parameters studied are shown as mean values  $\pm$  standard deviation (SD) in Table 2.

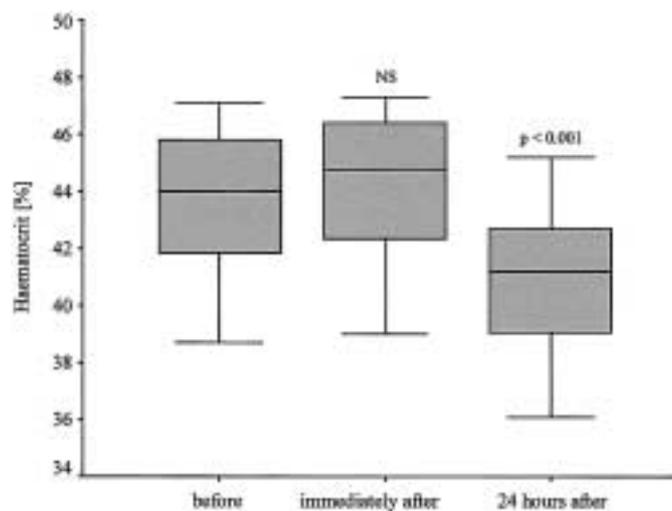


Fig. 1 Short-term changes in the level of haematocrit.

## Discussion

The major finding of our study is that the haematological short-term effects of extraordinary marathon cycling consist in considerable PV expansion making Hct values decline on the following day. The pre- and post-race Hct values of well-trained amateurs reliably not using rhEPO doping were placed in the middle range of normal distribution - well below the cut-off limit of the UCI.

The haematocrit is not a constant value but is influenced in both directions by various factors such as posture, day-time, mode of exercise and environmental conditions, i.e. temperature and humidity [15,27,28]. Diurnal variation and postural changes are reported to influence the level of Hct by 2.5 - 3%, short-term high intensity exercise by 3 - 4% [4,19,22,25,27]. Upright standing as well as maximal exercise cause fluid shifts from the intravascular space into the interstitial and intracellular compartment leading to a PV reduction and a rise in Hct. In general, acute variations in Hct are not based on changes in the RBC mass but on changes in PV due to fluid filtrations following osmotic gradients and/or increased hydrostatic pressure [19,24,25,27]. In contrast to acute exercise, periods of long-term training or repeated bouts of hour-lasting exercise as occurring during repetitive cycling competitions lead to an enlargement of PV which may be counteracted by the mechanism of dehydration during long lasting exertion [10,12,19,23,27].

There are only very few data available investigating the immediate influence of marathon cycling on Hct performed on athletes credibly not taking any kind of doping [18,19]. Recently Schmidt et al. described pronounced PV expansion (of up to 20%) during a 10-day cycling competition by observing similar Hct decreases in 4 elite cyclists averaging 2.2 - 5.2% [19]. Saris et al. investigated professional cyclists between 1980 and 1986 when rhEPO was not available [18]. They studied 34 professional cyclists from leading racing teams and found their Hct values to be normally distributed and to drop by 3% (45  $\rightarrow$  42%) in the course of the Tour de France in 1984. This Hct decline in "rhEPO-free" profes-

Table 2 Changes in parameters over the time presented as mean values ( $\pm$ SD)

|                          | Before race   | Immediately after          | One day after               |
|--------------------------|---------------|----------------------------|-----------------------------|
| Hct (40 - 52%)           | 0.44 (0.02)   | 0.44 (0.02) <sup>NS</sup>  | 0.41 (0.02)*                |
| Range                    | 0.38 - 0.47   | 0.39 - 0.47                | 0.36 - 0.45                 |
| Hb (13.3 - 17.7 g/dL)    | 14.9 (0.8)    | 15.1 (0.8) <sup>NS</sup>   | 13.9 (0.8)*                 |
| Range                    | 13.4 - 16.4   | 13.8 - 16.6                | 12.7 - 15.3                 |
| RBC (4.4 - 5.9 T/L)      | 4.89 (0.27)   | 4.95 (0.27) <sup>NS</sup>  | 4.57 (0.25)*                |
| Range                    | 4.26 - 5.48   | 4.32 - 5.24                | 3.96 - 4.94                 |
| MCH (27.0 - 32.0 pg)     | 30.46 (1.40)  | 30.51 (1.39) <sup>NS</sup> | 30.72 (1.36) <sup>NS</sup>  |
| Range                    | 26.6 - 33.2   | 26.6 - 33.1                | 26.6 - 33.1                 |
| MCV (77.0 - 96.0 fL)     | 89.49 (3.45)  | 88.93 (3.54)*              | 90.00 (3.38) <sup>NS</sup>  |
| Range                    | 80.8 - 97.0   | 80.2 - 95.7                | 80.4 - 96.0                 |
| MCHC (310 - 360 g/L)     | 340.39 (4.59) | 343.11 (4.01)*             | 341.24 (4.63) <sup>NS</sup> |
| Range                    | 331 - 351     | 333 - 351                  | 331 - 351                   |
| Protein (6.3 - 8.2 g/dL) | 8.07 (0.39)   | 8.07 (0.46) <sup>NS</sup>  | 7.27 (0.34)*                |
| Range                    | 7.28 - 8.89   | 7.21 - 9.64                | 6.50 - 7.87                 |
| LDH (120 - 240 U/L)      | 175 (27)      | 224 (33) <sup>NS</sup>     | 218 (43) <sup>NS</sup>      |
| Range                    | 133 - 246     | 168 - 316                  | 153 - 326                   |
| % $\Delta$ PV            |               | -1.5 (6.22) <sup>NS</sup>  | +11.9 (9.33) <sup>†</sup>   |
| Range                    |               | -17 - +10                  | 2 - 36                      |

\*  $p < 0.01$  (Wilcoxon signed-rank test), <sup>†</sup>  $p < 0.01$  (one sample t-test), <sup>NS</sup> = non significant

sionals strongly corresponds to the decrease of 3% (44→41%) found in the clean amateurs of our study. The corresponding increase in  $\Delta$ PV was calculated to be 11.9 which again is well comparable to that of previous data [4, 5, 17].

None of the Hct values measured in our study was above 47% before competition and 45% on the following day, respectively. Our data are further evidence that due to exercise-induced long-term PV expansion Hct values tend to be lower in a cohort of well-trained cyclists than in an untrained comparative population [18, 19]. Furthermore our findings show that “rhEPO-free” amateur cyclists exhibit physiological pre-race Hct values well below the UCI cut-off and markedly decreased post-race values on the following day - at a moment when the standardized blood testing of the UCI for the Hct controls is set in order to minimize potential influences of extraneous variables such as the state of hydration.

Adequate fluid substitution is crucial for a successful performance of long-term exercise. During the ultramarathon the athletes replaced large amounts of fluid (5.1 L) which explains the moderate immediate weight loss (1.7 kg) and why significant dehydration and coincident haemoconcentration due to prolonged fluid losses through sweat and respiration did not occur. Nevertheless, some intracellular dehydration did happen as indicated by decreased MCV and increased MCHC indices immediately post-exercise. These changes caused by a fluid shift from the intracellular erythrocyte compartment into the intravascular space were just minor and were balanced by rehydration on the following day.

In this study we investigated “clean” well-trained amateurs to reveal the real physiological effects. When exclusively studying professionals, you run the risk of obtaining falsified data influenced by rhEPO doping as several scandals have shown in the past. We are aware that any transformation of data from the amateur to the professional is difficult for several reasons, but mainly because both have their own physiological characteristics. Direct comparison would be desirable but certainly not practicable as they usually do not participate in the same competitions and professionals are generally not very pleased to take part in such studies for various reasons. Despite all differences professionals and amateurs are human beings underlying the same physiological responses to exercise. As trends observed should be identical in both groups, we consider a careful extrapolation to be justified.

Against the background of rhEPO doping our finding, which shows Hct values to decline under extreme conditions, suggests that without artificial help it is very difficult to reach a “critically high” RBC mass after strenuous marathon cycling. It does not support the criticism that the valid UCI cut-off of 50% leads to an unacceptably high number of “false positive” exclusions. As long as direct methods for the detection of rhEPO doping are not yet applied, the UCI regulation represents an appropriate means in the fight against excessive rhEPO doping.

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