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ORIGINAL ARTICLE

Iron excess in recreational marathon runners

S Mettler[1,2] and MB Zimmermann[3,4]

[1]*Physiology and Behavior Group, Department of Agricultural and Food Sciences, ETH Zurich, Zurich, Switzerland;* [2]*Swiss Federal Institute of Sport Magglingen, Magglingen, Switzerland;* [3]*Laboratory for Human Nutrition, Department of Agricultural and Food Sciences, ETH Zurich, Zurich, Switzerland and* [4]*Division of Human Nutrition, Wageningen University, Wageningen, The Netherlands*

Background/Objectives: Iron deficiency and anemia may impair athletic performance, and iron supplements are commonly consumed by athletes. However, iron overload should be avoided because of the possible long-term adverse health effects.
Methods: We investigated the iron status of 170 male and female recreational runners participating in the Zürich marathon. Iron deficiency was defined either as a plasma ferritin (PF) concentration $<15\,\mu g/l$ (iron depletion) or as the ratio of the concentrations of transferrin receptor (sTfR) to PF (sTfR:log(PF) index) of $\geqslant 4.5$ (functional iron deficiency).
Results: After excluding subjects with elevated C-reactive protein concentrations, iron overload was defined as PF $>200\,\mu g/l$. Iron depletion was found in only 2 out of 127 men (1.6% of the male study population) and in 12 out of 43 (28.0%) women. Functional iron deficiency was found in 5 (3.9%) and 11 (25.5%) male and female athletes, respectively. Body iron stores, calculated from the sTfR/PF ratio, were significantly higher ($P<0.001$) among male compared with female marathon runners. Median PF among males was $104\,\mu g/l$, and the upper limit of the PF distribution in males was $628\,\mu g/l$. Iron overload was found in 19 out of 127 (15.0%) men but only 2 out of 43 in women (4.7%). Gender (male sex), but not age, was a predictor of higher PF ($P<0.001$).
Conclusions: Iron depletion was present in 28% of female runners but in $<2\%$ of males, whereas one in six male runners had signs of iron overload. Although iron supplements are widely used by athletes in an effort to increase performance, our findings indicate excess body iron may be common in male recreational runners and suggest supplements should only be used if tests of iron status indicate deficiency.
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Keywords: iron; exercise; performance; ferritin

Introduction

As a functional component of iron-containing proteins, including hemoglobin (Hb), myoglobin and cytochromes, iron is a key element required for the delivery of oxygen to tissues and the processing of oxygen at the cellular level (Lukaski, 2004). Inadequate iron status may impair Hb and red blood cell production, causing anemia and impairing physical performance (Schumann *et al*., 2007). Low iron stores in the absence of anemia may also adversely affect performance (Brownlie *et al*., 2002, 2004), although not all studies agree (Newhouse *et al*., 1989; Eichner, 2000). A low plasma ferritin (PF) is an indicator of low iron stores, (Baynes, 1996; Eichner, 2000), and if negative iron balance continues, the soluble transferrin receptor (sTfR) will increase as low iron supply to the bone marrow causes ineffective erythropoesis (Baynes, 1996). Calculation of body iron based on the PF/TfR ratio (Cook *et al*., 2003) allows assessment over the entire range of iron status.

Among iron-depleted subjects, only subjects with elevated sTfR increased their performance after iron supplementation (Hinton *et al*., 2000; Brownlie *et al*., 2002, 2004). PF is the recommended method for monitoring of an athlete's iron status, but there is currently no agreement on the PF level at which supplementation is recommended, nor is there consensus on the optimal plasma concentration (Rodenberg and Gustafson, 2007). As it is an acute-phase protein, use of PF alone to define iron status in elite endurance athletes, who may be subjected to chronic training-induced inflammation, is problematic unless controlled for by measurement of C-reactive protein (CRP) (Zimmermann and

Correspondence: Dr MB Zimmermann, LFV E 19.2, Schmelzbergstrasse 7, ETH Zurich, Zurich CH-8092, Switzerland.
E-mail: michael.zimmermann@ilw.agrl.ethz.ch
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Hurrell, 2007). Therefore, the sTfR and sTfR:PF index have been suggested as more sensitive indicators of functional iron deficiency in athletes than PF (Baynes, 1996; Sinclair and Hinton, 2005; Suedekum and Dimeff, 2005), because, in contrast to PF, the sTfR is not an acute-phase protein and is not influenced by inflammation (Ferguson *et al.*, 1992) or previous training loads (Malczewska *et al.*, 2000a).

As the adverse effects of iron deficiency on physical performance are well recognized, iron supplements and multivitamin/mineral supplements containing iron are commonly consumed by both competitive and recreational athletes (Zoller and Vogel, 2004; Rodenberg and Gustafson, 2007). Iron excess may be pro-oxidative and has been linked to several chronic diseases, although the data are not conclusive (Salonen *et al.*, 1992; Schumann *et al.*, 2007). Excessive iron intake may have adverse effects on the intestinal, vascular and cellular level (Schumann *et al.*, 2007). Iron overload has been reported in competitive athletes (Deugnier *et al.*, 2002; Zotter *et al.*, 2004), but there are few data on iron excess in recreational athletes. Thus, the purpose of this study was to examine iron status in recreational marathon runners using both PF and sTfR, and specifically, to determine the prevalence of iron excess in this group.

Methods

Subjects in this study were originally recruited for a study on fluid intake and hyponatremia at the 2006 Zurich marathon (Mettler *et al.*, 2008). They were recruited by an announcement on the official marathon website and at the runners' exposition the 2 days before the race. All registered runners who were $\geqslant 18$ years of age and had an expected finishing time within the official time limit of 5 h were eligible. The study was approved by the ethical committee of the ETH Zurich and written informed consent was obtained from all subjects.

At enrollment, age, sex and height were recorded by questionnaire and weight was measured on the race day as previously described (Mettler *et al.*, 2008). A blood sample was collected by venipuncture from an antecubital vein with the subject in supine position at the start area before the start of the marathon. Blood was collected into heparin and EDTA-containing tubes (S-Monovette, Sarstedt, Nümbrecht, Germany). In all, 177 recreational marathon runners were enrolled. In three subjects, a complete blood sample was not obtained. Thus, 174 subjects (130 men and 44 women) were included in this study.

Laboratory analysis

Hemoglobin, hematocrit and mean corpuscular volume were analyzed using an automated hematology analyzer (Sysmex XE-2100, Sysmex Corporation, Kobe, Japan) using control material supplied by the manufacturer. The remaining blood was placed on ice and then centrifuged within 20 min of venipuncture at 4 °C for 12 min at 1800 *g* (Omnifuge 2.0 RS, Heraeus Sepatech, Osterode, Germany). Plasma was aliquoted and immediately frozen at −20 °C, and then transferred within 24 h to storage at −80 °C until analysis.

At the Human Nutrition Laboratory at the ETH Zürich, PF and CRP were analyzed on an IMMULITE auto immunoanalyzer (DPC Bühlmann GmbH, Aschwil, Germany) from EDTA plasma. sTfR was analyzed using enzyme-linked immunosorbent assay (Ramco Laboratories, Stafford, TX, USA) from EDTA plasma. Plasma iron and total iron-binding capacity (TIBC) was analyzed by colorimetry (Fielding, 1980) using heparin plasma. All samples were run in duplicate. Transferrin saturation was calculated as: plasma iron/TIBC*100.

Statistical analysis

Data analyses were carried out using SAS for windows (version 8.2, SAS institute Inc., Cary, NC, USA). Anemia was defined as Hb <120 and 130 g/l for women and men, respectively (WHO, 2001; Sinclair and Hinton, 2005). Iron deficiency was defined as either: (1) a PF $<15\,\mu$g/l, indicating iron depletion (WHO, 2001); or (2) an sTfR:log(PF) index of $\geqslant 4.5$, indicating functional iron deficiency (Sinclair and Hinton, 2005). Iron overload was defined as PF $>200\,\mu$g/l (Salonen *et al.*, 1992; WHO, 2001). Body iron (mg/l) was calculated as $-(\log(\text{sTfR}\cdot\text{PF}^{-1})-2.8229)\cdot 0.1207^{-1}$ as described by Cook *et al.* (2003). Subjects with CRP >5 mg/l were excluded from statistical analysis, because high CRP values indicate an acute-phase response, which may elevate PF (Baynes, 1996). To identify associations among variables, linear regression analysis was performed. PF values were not normally distributed and were log transformed before statistical analysis. Results are presented as mean ± s.d. or as median (min, max) in case of non-normally distributed values. *P*-values of <0.05 were considered statistically significant.

Results

Four subjects (three men, one women) had CRP values >5 mg/l (the values were 9, 18, 32 and 46 mg/l, respectively) and were excluded from the data analysis. The Hb, PF, sTfR and sTfR:log(PF) index values of these four subjects were within the normal range, except for elevated PF concentrations in two of the men (224 and 250 μg/l). The characteristics and hematological data of the remaining 170 subjects (127 men, 43 women) are given in Table 1. Prevalence of iron deficiency anemia, iron deficiency without anemia, normal iron status and iron overload among the male and female marathon runners are shown in Figure 1. Only 2 out of 127 men (1.6%) had a low PF indicating iron depletion, and one of these men was also anemic. Among women, 12 out of 43 (28.0%) had low PF; of these, six were anemic (Figure 1). The man who was anemic and four of the six anemic women had abnormally low transferrin saturation ($<16\%$) (Zimmermann and Hurrell, 2007). Using the sTfR:log(PF) index, four men (3.1%) and five

Table 1 Age, body mass index, hematological and iron status in recreational runners in the Zurich, Switzerland marathon

	Women (n = *43*)	*Men* (n = *127*)
Age (years)	39 ± 9	43 ± 9
Body mass index (kg/m^2)	20.6 ± 1.6	23.7 ± 2.1
Hemoglobin (g/l)	133 ± 12	149 ± 7
Hematocrit	0.403 ± 0.028	0.439 ± 0.019
Mean corpuscular volume (fl)	87.6 ± 4.6	86.4 ± 3.7
Transferrin saturation (%)	33 ± 13	34 ± 11
Plasma iron (μg/ml)	1.41 ± 0.50	1.37 ± 0.39
Total iron-binding capacity (μg/ml)	4.38 ± 0.74	4.13 ± 0.58
Body iron (mg/kg)	6.5 (−5.8, 14.7)	10.0 (−2.9, 16.3)
Ferritin (μg/l)	40 (4, 267)	104 (6, 628)
sTfR (mg/l)	5.28 ± 2.67	4.33 ± 1.29
sTfR:log(ferritin) index	4.65 ± 4.74	2.32 ± 1.26
Pre-race sodium[a] (mmol/l)	139 ± 2	140 ± 2
Number of previous marathons	2.3 ± 2.8	6.4 ± 8.5
Time to complete marathon (h:min)	4:05 ± 0:23	3:40 ± 0:32

Abbreviation: sTfR, transferrin receptor.
[a]Pre-race sodium and hydration status was normal in all subjects (Mettler *et al.*, 2008).
Values are means ± s.d. or median (min, max).

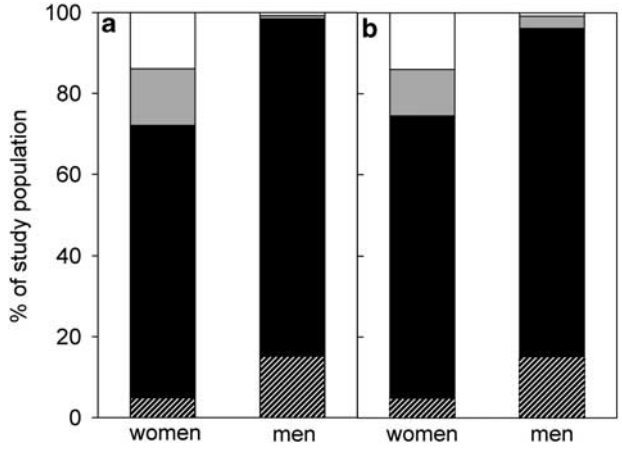

Figure 1 Prevalence of normal iron status (black), nonanemic iron depletion (**a**) or functional iron deficiency without anemia (**b**) (gray), iron deficiency anemia (white), and iron overload (striped) among male ($n = 127$) and female ($n = 43$) athletes. Anemia was defined as hemoglobin <120 and 130 g/l for women and men, respectively (WHO, 2001; Sinclair and Hinton, 2005). Iron deficiency was defined as either: (**a**) a PF <15 μg/l, indicating iron depletion (WHO, 2001); or (**b**) an sTfR:log(PF) index of ⩾4.5, indicating functional iron deficiency (Sinclair and Hinton, 2005). Iron overload was defined as PF >200 μg/l (Salonen *et al.*, 1992; WHO, 2001).

women (11.6%) were classified as functionally iron deficient without anemia. One man (with anemia) and eight women (18.6%) had a negative body iron (Figure 2). In subjects with iron depletion, PF ($P < 0.001$, standardized coefficient = 0.86) and body iron stores ($P < 0.001$, standardized coefficient = 0.89) were predictors of Hb.

Controlling for age, body iron stores were significantly higher among male compared with female marathon runners ($P < 0.001$); median body iron stores was 10 mg/kg in males

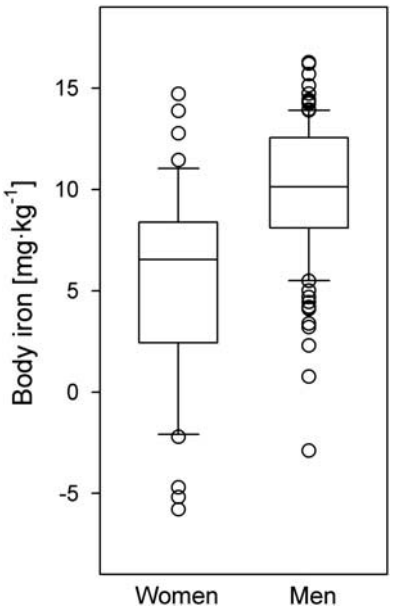

Figure 2 Distribution of body iron stores, calculated from the sTfR:PF ratio (Cook *et al.*, 2003), among male and female marathon runners. Body iron stores were significantly higher ($P < 0.001$) among men.

(Figure 2). Median PF among males was 104 μg/l, and the upper limit of the PF distribution in males was 628 μg/l (Table 1). Iron overload was found in 19 out of 127 (15.0%) male athletes, but only in 2 out of 43 women (4.7%) (Figure 1). Gender (male sex), but not age, was a predictor of higher PF ($P < 0.001$) and Hb ($P < 0.001$). Time to complete the marathon was significantly associated with gender (males had shorter race times), but not with PF, body iron, Hb or age.

Discussion

There is still debate as to whether endurance athletes are at higher risk of low iron stores due to an imbalance between absorption of dietary iron and exercise-induced iron loss (Nielsen and Nachtigall, 1998). Body turnover of radioactive iron is ca. 20% greater in athletes than in nonathletes (Ehn *et al.*, 1980). The prevalence of iron deficiency and anemia is generally higher in athletes than in healthy sedentary individuals (Newhouse and Clement, 1988), and in most studies, runners were the most affected group (Beard and Tobin, 2000). But not all studies agree (Schumacher *et al.*, 2002), and some studies have reported better iron status in athletes compared with sedentary controls (Malczewska *et al.*, 2000b). Nevertheless, most studies examining iron status in athletes have focused on iron deficiency rather than iron excess (Schumacher *et al.*, 2002; Dubnov and Constantini, 2004; Sinclair and Hinton, 2005). This focus on iron deficiency and performance may result in a lay misperception that iron deficiency is widespread, and thus, contributes to overuse of iron supplements by athletes

(Deugnier *et al*., 2002; Zoller and Vogel, 2004; Zotter *et al*., 2004), and/or their trainers and coaches (Rodenberg and Gustafson, 2007). Iron supplement abuse and iron overload has been reported in elite cyclists (Deugnier *et al*., 2002; Zotter *et al*., 2004). Our findings suggest that this may also be occurring in recreational endurance athletes.

Although we did not collect data on iron supplement use or iron intakes in our subjects, other studies have highlighted the frequent use of iron supplements by athletes. Iron supplements are used regularly by athletes due to the widespread belief that they increase the number of red blood cells and thus performance (Zotter *et al*., 2004). The most popular mineral supplements used by athletes are iron supplements (Colombani and Mannhart, 2003; Huang *et al*., 2006).

But unnecessary consumption of iron may result in iron overload, and hyperferritinemia in elite athletes is usually due to excessive iron supplementation (Deugnier *et al*., 2002). Professional endurance athletes have PF concentrations two- to threefold higher than those of matched sedentary individuals (Lippi *et al*., 2005). In a 2002 study, 27% of PF values among professional road cyclists were $>300\,\mu g/l$ (Zotter *et al*., 2004).

The long-term consequences of iatrogenic iron overload are unclear, but excess iron may react directly with unsaturated fatty acids and generate free radicals (McCord, 1998). Excessive iron intake has the potential to cause harm on the intestinal, vascular and cellular level (Schumann *et al*., 2007). High iron stores are associated with oxidative stress and DNA damage (Tuomainen *et al*., 2007) and a PF $>200\,\mu g/l$ is associated with increased risk of myocardial infarction (Salonen *et al*., 1992; Klipstein-Grobusch *et al*., 1999). Athletes with hereditary hemochromatosis are at high risk of organ damage due to iron overload and iron supplements in this group are contraindicated (Pietrangelo, 2004; Zoller and Vogel, 2004).

In agreement with other studies, we found a higher prevalence of iron deficiency and iron deficiency anemia in female than in male athletes (Nielsen and Nachtigall, 1998; Sinclair and Hinton, 2005). Among both women and men in our study, but particularly in men, the prevalence of iron deficiency was generally lower than in other studies (Dubnov and Constantini, 2004; Merkel *et al*., 2005; Sinclair and Hinton, 2005). This may be, at least, partially due to the older age of our subjects; most other studies included adolescent athletes (Dubnov and Constantini, 2004; Merkel *et al*., 2005) whose iron requirements are higher due to growth (Eichner, 2000). Age was not a significant predictor of iron status in our study, but we included only adult athletes. Another potential explanation for our lower prevalence of iron deficiency compared with previous studies (Dubnov and Constantini, 2004; Merkel *et al*., 2005; Sinclair and Hinton, 2005) is that, because our study population was originally recruited for a study of fluid balance and hyponatremia, there was likely no selection bias for abnormal iron status.

Among women, the prevalence of iron deficiency was nearly identical when using the sTfR together with PF (sTfR:log(PF) index) compared with using PF alone, but the subjects falling below the cutoffs were not always the same. Three women with PF $<15\,\mu g/l$ (13.8, 13.9 and 14.2 μg/l) had a low sTfR (3.2, 3.7 and 4.8 mg/l) and a low sTfR:log(PF) index (2.8, 3.2 and 4.1), whereas two women had an increased sTfR:log(PF) index (5.0 and 5.6), but a normal PF (19.1 and 15.3 μg/l). There is debate on the PF cutoff for iron deficiency in athletes and there are conflicting results about the effect on performance of iron deficiency without anemia (Nielsen and Nachtigall, 1998; Eichner, 2000; Hinton *et al*., 2000; Brownlie *et al*., 2002, 2004; Rodenberg and Gustafson, 2007). An advantage of using the sTfR:log(PF) index is that, unlike PF alone, it is sensitive to a wider range of body iron status, as it includes a measure of iron-deficient erythropoesis. In iron-depleted athletes without anemia, only those with elevated sTfR values showed impaired performance that was responsive to iron supplementation (Baynes, 1996; Hinton *et al*., 2000; Brownlie *et al*., 2004). Moreover, sTfR may decrease in athletes with an accompanying improvement in body iron, even though PF decreases (Petersen *et al*., 2006). In our study, three women and no men had PF $<15\,\mu g/l$, but had normal sTfR. This suggests these athletes with low iron stores were maintaining normal rates of erythopoesis. In contrast, three male and two female subjects had PF between 15 and 40 μg/l, but had elevated sTfR suggesting impaired erythropoesis, a condition that has been associated with impaired athletic performance (Hinton *et al*., 2000; Brownlie *et al*., 2002, 2004). Thus, the sTfR:log(PF) index may be preferable to PF alone to assess iron status and the need for supplementation in athletes (Petersen *et al*., 2006).

Because of time constraints at the start of the marathon, we did not ask the subjects to fill out a questionnaire. Thus, the interpretation of our results is limited by the lack of information on other potential contributors to iron status in our study population, including dietary iron intake, vegetarianism, menstrual blood loss and oral contraceptive use in females, and use of drugs that affect iron metabolism. Further studies in other endurance athletes from other countries and settings are needed to confirm our findings. But to prevent iatrogenic iron overload, iron supplementation by recreational athletes should be done only after monitoring of iron status. Iron supplementation is generally not indicated for male recreational athletes. Apart from supplements, iron nutrition can also be improved by simple dietary changes. These include regular consumption of modest amounts of lean meat (Lyle *et al*., 1992; Baech *et al*., 2003), addition of vitamin C (an iron absorption enhancer)-rich foods to meals containing non-heme iron and avoidance of tea and coffee consumption (these are high in iron absorption-inhibiting polyphenols) with meals (Thankachan *et al*., 2008).

Conflict of interest

The authors declare no conflict of interest.

Acknowledgements

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