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Anaemia and Iron Deficiency in Athletes

Practical Recommendations for Treatment

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Abstract

Trained athletes frequently experience low levels of blood haemoglobin (13 to 14 g/100ml in men and 12 g/100ml in women) plus low haematocrit and low ferritin levels. These parameters define the concept of ‘sports anaemia’. Low iron levels may be due to mechanical haemolysis, intestinal bleeding, haematuria,

sweating, low iron intake or poor intestinal absorption. The resulting decrease in blood gas transport and muscle enzyme activity impairs performance.

The concept of sports anaemia can be criticised. Simply measuring the blood levels does not take into account the haemodilution that occurs in athletes because of training. The lack of these measurements makes it difficult to diagnose anaemia or evaluate any treatment. Anaemia is treated by preventing decreased iron stores through a balanced food intake or iron supplements. Self-medications must be discouraged because of intolerance, risk of overdose and many other drug interactions.

1. Anaemia and Iron Deficiency in Athletes

1.1 Anaemia

Athletes trained for endurance often have low blood haemoglobin levels. It may even be below the normal range (i.e. 13 to 14 g/100ml in men and 12 g/100ml in women) and associated with low haematocrit values.[1-5] Early studies showing these low haemoglobin and haematocrit values gave rise to the concept of 'sports anaemia' to describe this phenomenon.[4,5]

But this altered haematological status does not occur in all trained athletes.[2,6-9] Stewart et al.[10] found only some blood haemoglobin values close to normal values in 101 members of the 1972 Australian Olympic team. De Wijn et al.[11] found that only 5 to 6% of the athletes on the Dutch national teams had haemoglobin values below 14 g/100ml (men) and 12 g/100ml (women), and that only 2 of them experienced true anaemia. These results were confirmed by Weight et al.,[8] who identified only 3 cases of anaemia among 120 male and female endurance runners, and by Balaban et al.,[12] who found 4 anaemic athletes among 72 highly trained endurance runners. Furthermore, 37 of the female runners had higher haemoglobin levels than their sedentary counterparts.[12]

However, these studies did not take into consideration the haemodilution that takes place over the 48 hours following every bout of endurance exercise, and which can persist for as long as a week.[13-15] This haemodilution is a permanent feature of athletes in training when that training requires high energy outputs, especially among those athletes involved in endurance sports. Highly trained endurance athletes can have haematocrits of 40 to 42% without any reduction in their circulating haemoglobin.[16-18]

Training increases the plasma volume and stimulates erythropoiesis, but these adaptations are regulated by independent mechanisms.[16] The increase in plasma volume is regulated by changes in osmotic pressure, which depend on hormonal and protein responses to short and long term exercise. Erythropoiesis depends on the production and release of erythropoietin, which is regulated by the oxygen content of the blood vessels irrigating the kidneys. Plasma volume expansion due to training occurs more rapidly and to a greater extent than the increase in red cell volume. It seems to be responsible for the pseudo-anaemic status of highly trained athletes, who have apparently low haemoglobin levels and normal body iron stocks.[17-21] Several studies[22-24] have shown that periods of intense training are coupled with reduced haemoglobin and haematocrit values in highly trained swimmers, while the less intense training periods preceding major competitions (taper periods) result in increases in these haematological variables. These results are in keeping with those of Pelliccia and Di Nucci,[25] who reported normal haematological values in swimmers participating in the European Championships.

1.2 Iron Deficiency

A diagram of the body's iron stores is shown in figure 1. Ferritin levels are an indicator of iron stores,[26] and 1μg of ferritin is equivalent to an iron store of 8mg.[27] Iron is distributed within the body

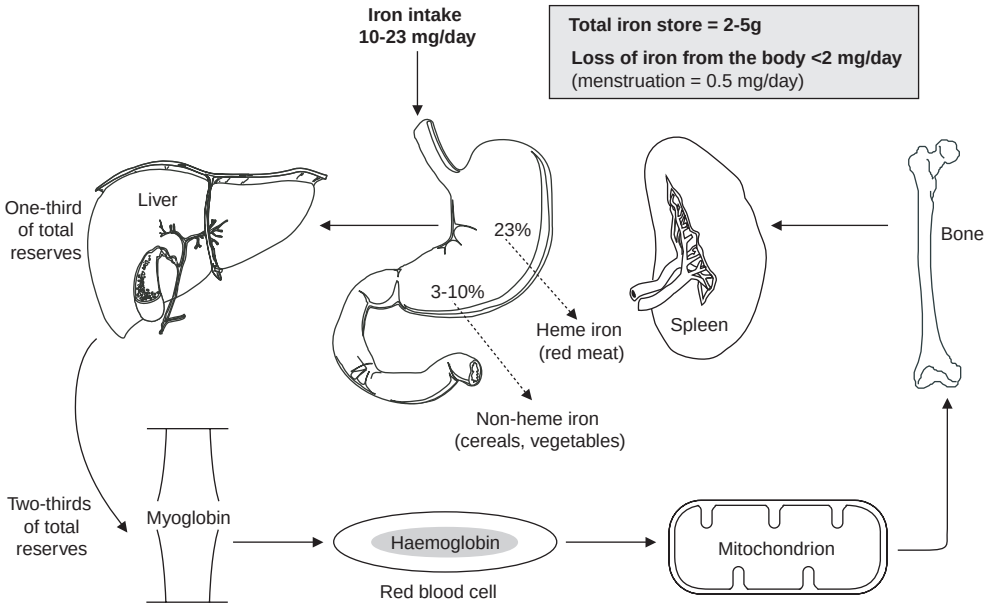

Fig. 1. Body iron stores. ⋯> indicates 23% of heme iron and 3 to 10% of non-heme iron ingested are absorbed from the gastrointestinal tract.

via transferrin in the plasma. Over 95% of the serum iron is bound by transferrin. In iron deficiency, transferrin levels increase from 330 μg/100ml to more than 500 μg/100ml, while the degree of saturation of transferrin decreases.[26,28]

Bainton and Finch[29] demonstrated that a saturation of less than 16% is inadequate to meet basal erythropoietic needs and that transferrin saturation is a better predictor of iron deficiency than serum iron. An individual with a transferrin saturation of 50% but a serum iron concentration of 40 μg/L may thus have an adequate iron supply for erythropoiesis, while another individual with a serum iron of 40 μg/L, but a saturation of 10%, may not.[26] Serum iron levels show a circadian variation with a morning peak and an evening nadir and a considerable day-to-day variation due to the variable release of the iron to the plasma by the reticuloendothelial system. Indeed, the plasma iron pool is small (about 3mg) in comparison with the daily plasma iron turnover (about 35mg).[26]

Although highly trained athletes usually have normal absolute levels of haemoglobin, they often experience iron deficiency, generally latent, that implies no decrease in haemoglobin. De Wijn et al.[11] reported that 7% of male and 17.5% of female athletes studied had a transferrin saturation index below 20%, but very few were anaemic. Subsequently, a series of studies on the iron reserves of highly trained athletes were published and have been summarised in several review articles.[1-3,20,30-36]

Clement et al.[37] studied elite Canadian endurance runners, and found that 29% of the men and 82% of the women had extremely low ferritin values (<25 μg/L), even though their blood haemoglobin and serum iron levels were normal. Wishnitzer et al.[38] reported normal haematological values along with a complete or almost complete depletion of the bone marrow haemosiderin stocks in 12 international level middle-distance and long-distance runners. Nachtigall et al.[39] recently reported ferritin values below 35 μg/L in 23 of 45 endurance runners.

Kaiser et al.[40] showed that this decrease in ferritin in athletes involved in endurance running occurred only during the initial 6 months of training, while the apparently low red cell count remained throughout the whole 20-month training period. However, the red cell count of the female athletes was not modified, although 28 to 56% of them had ferritin values below 20 μg/L at some time in the follow-up period. Thus, the subnormal ferritin values that occur in athletes are generally of no clinical significance.

These observations have led sports physicians and scientists to monitor the circulating ferritin levels of athletes as part of their testing programmes. Candau et al.[41] in cross-country skiers, Roberts and Smith[42] in swimmers and Banister and Hamilton in runners,[43] showed that the ferritin values decrease as the training load increases, suggesting that ferritin could be a useful marker of training tolerance.

Thus, only a small number of athletes are at risk of having subnormal circulating ferritin levels, and only a minority have ferritin values that can be considered as pathological. This index of reduced body iron stores may or may not be associated with low haemoglobin values. Lastly, runners seem to be the endurance athletes at greatest risk of iron store depletion.

2. Factors Affecting the Haematological Status of Athletes

2.1 Gastrointestinal Bleeding

There have been numerous reports of gastrointestinal bleeding in athletes.[1,3,31,39,44,45] Healthy volunteers experiencing no gastrointestinal pathology and completely asymptomatic under resting conditions may experience gastrointestinal bleeding following endurance running training sessions or after a marathon. The bleeding generally involves the upper part of the intestinal tract and may not be apparent. It has been detected using isotopically labelled iron.[39] Blood loss can reach several millilitres/day. Its frequency in runners, which is 8 to 83% depending on the study,[31] seems to be linked to the intensity of exercise, the distance covered[39] and the degree of dehydration.[46] However, truly pathological manifestations are rare. Thompson et al.[47] reported a case of death due to gastrointestinal bleeding following the Rhode Island marathon. Papaioannides et al.[48] reported a few cases of haematesis. Bleeding diarrhoea, on the other hand, is more frequent, as it affected 1 to 2% of the runners questioned.[46,49,50]

Gastrointestinal bleeding seems to be independent of clinical manifestations such as abdominal pain or diarrhoea.[51] Hidden bleeding is responsible for the loss of 1 to 7ml of blood/day, corresponding to a loss of 0.5 to 2mg of iron/day and a level of 1 to 3mg haemoglobin/gram of stool.[39,45,52] Most studies suggest that gastrointestinal bleeding after endurance exercise lasts no longer than 72 hours. However, a daily loss of 7 to 10ml of blood is enough to induce a negative iron balance.[3] The factors responsible for gastrointestinal bleeding include temporary ischaemia of the gastrointestinal tract, which is linked to the intensity and duration of exercise, exercise stress-associated gastric acid secretions, organ shock and intakes of pharmacological agents such as anti-inflammatory drugs or aspirin (acetylsalicylic acid). The injuries are reversible and manifest as a temporary ischaemia: the mucosa appears purple with disseminated ulcerations, and glandular necrosis, increased gastric secretions, and a reduced thickness of the polysaccharide layer protecting the walls.[44]

2.2 Haematuria

Exercise can produce haematuria. Indeed, haematuria was reported in 17 to 90% of runners studied after a marathon run.[31,53-55] Reversion to normal urine within 24 to 72 hours has been proven by repeat urinalysis. Such reversibility is useful in eliminating other disease processes.[56] If training is carried out too frequently, the temporary decrease of haemoglobin may become permanent[57] and athletes may develop an anaemia with or without complaining of discoloured urine.[58]

Indeed, haematuria is most often microscopic, although it can sometimes be macroscopic. It is

commonly accepted that >3 red blood cells per high-power microscopic field or 1000 red blood cells per millilitre of urine is abnormal.[59] Haemolysis is marked by a decrease in plasma haptoglobin. If the haemolysis is slight, no haemoglobin is lost in the urine.[60] Haptoglobin binds free haemoglobin, preventing its urinary excretion. The haptoglobin-haemoglobin complex is then recovered by the hepatocytes, and the iron recycled.

Low haptoglobin levels have been observed following training sessions on a cycle ergometer. They have mostly been found at the beginning of the training programme, and appear to be due to the destruction of older red blood cells.[17,18] Guglielmini et al.[61] have shown that intravascular haemolysis is associated with a reduced erythropoiesis in professional cyclists riding an average of 30 000 km/year, resulting in low circulating haemoglobin levels even though their iron stores are not diminished.

The causes of haematuria may be categorised according to the site of injury (kidney or bladder) and the particular sport with which it occurs: contact versus noncontact; football or boxing versus running or jumping.[62] Many explanations have been directed toward a potential cause:[59] foot-strike haemolysis, renal ischaemia, hypoxic damage to the kidney, release of a haemolysing factor, bladder and/or kidney trauma, nonsteroidal anti-inflammatory drugs, dehydration, increased circulation rate and body temperature, myoglobinuria release, muscle tissue damage and the peroxidation of red blood cells (for a complete review of these mechanisms see Cianfloco[56] and Jones and Newhouse[59]). Bladder injury appears to involve impact of the flaccid posterior vesicle wall against the trigone and may be facilitated by an empty bladder.[62]

Renal blood flow and glomerular filtration are diminished in proportional to the duration and intensity of the exercise as well as the subsequent dehydration. This decrease results from constriction of renal blood vessels to redistribute blood flow to skeletal muscle. As a result, hypoxic damage occurs to the nephron with a subsequent increase in glomerular permeability and excretion of erythrocytes into the urine. Renal vasoconstriction is more pronounced in the efferent glomerular arteriole which leads to increased filtration pressure, and filtration fraction enabling the passage of red blood cells into the urine.[56,59]

Athletes with proven sports haematuria may continue to exercise, but should be encouraged to maintain adequate hydration by drinking copious fluids, voiding just before running may not be recommended.[56,59]

2.3 Sweating

The exact quantification of sweat iron is difficult. Indeed, the iron content of sweat varied from different body sites, and from person to person.[63] To avoid contamination of the skin, sweat measurements need to be collected in great quantity[39,64] and over a long time exposure. Indeed, iron levels in sweat are higher at the beginning of sweat production than at later phases of a long distance run.[56,59,64]

Sweat, free of peeling cells, contains an average 300 to 400μg iron/litre.[1,3,31] Seiler et al.[64] found higher values after several hours of running (470 to 530μg iron/litre) using a radio iron labelling method. Sweat loss can reach 2 to 3 L/h or more during prolonged exercise in moderate thermal environments (above 10°C) and thus may result in daily iron losses of 1 to 2mg, which is close to the amount absorbed from the diet. Increased iron elimination associated with an intestinal malabsorption might be the explanation for the suboptimal iron situation found in long distance runners who train extremely hard.[65] Nachtigall et al.,[39] using the radio iron labelling method, showed that under intensive training or race conditions the iron loss in sweat did not derive from the haemoglobin pool.

2.4 Menstruation

Iron deficiency occurs more frequently in women athletes,[1,3,31,40,66,67] and 20 to 47% of Canadian and American female athletes had ferritin values below 12 μg/L, compared with only 2 to 13% of their male counterparts.[1] Iron loss is much greater during the

menstrual cycle (0.5 to 0.6 mg/day), corresponding to a median loss of 30ml of blood per cycle.[26]

An iron deficiency is almost inevitable when menstrual bleeding is heavy (>60ml)[31] and/or the menstrual cycle lasts more than 5 days. Menstrual bleeding may be excessive in athletes using an intrauterine device.[36] Many women are unaware that their menstrual blood losses are excessive. Indeed, 40% of the women of one study who lost more than 80 ml/period considered their menstruation to be moderate or even scanty.[68]

Rowland and Kelleher[66] found an inverse relationship between the amount of menstrual flow and serum ferritin levels. They also found poor dietary intakes of iron averaging 43% of the recommended dietary allowance. These data were confirmed by other authors.[34,36,45,52,67,69,70]

Thus, in women, daily iron requirements are greater than in males, but their energy intakes are lower and therefore so are their iron intakes.[26,71] Hence, the greater frequency of anaemia among women is not surprising.

2.5 Training

Training itself can also be responsible for the apparent changes in the iron stores.[41] These authors demonstrated that ferritin levels of some athletes decreased or increased too rapidly to be attributed only to haemolysis, bleeding or sweating over a 33-week training period. Banister and Hamilton[43] showed that serum iron and transferrin saturation varied in phase with fatigue and training load. When training increases, transferrin becomes saturated and limits the iron release from the mucosal cells of the intestine. On the contrary, the iron release is facilitated by a low degree of plasma transferrin saturation. Thus, the efficiency of iron supplementation is questionable during heavy training and concomitant high fatigue. Indeed, transferrin saturation is very high at this time and ineffective in promoting absorption of dietary iron. This could explain the lack of any relationship often found between iron intake and circulating ferritin.[72,73] It could also explain why rest is a good treatment for sports anaemia.

3. Functional Consequences

3.1 The 3 Stages of Anaemia

Healthy people (fig. 1) have 2 to 5g of stored iron,[1,30,74] two-thirds of which is in the haemoglobin, myoglobin and certain mitochondrial enzymes (cytochromes). The remaining one-third is in bones, liver and spleen.[1] The loss of iron can be divided into 3 stages (fig. 2) according to their impact on red cell synthesis.[26] Stage 1 is 'iron depletion' in which there is an isolated decrease in serum ferritin levels. Stage 2 is 'iron deficient erythropoiesis'. In this stage, the supply of iron to the erythroid marrow is inadequate, serum ferritin levels are low (<10 to 20%), transferrin saturation is decreased (<16%) and the total iron binding capacity is increased (>390 μg/100ml). The anaemia of iron deficient erythropoiesis may be too mild to be detected by some arbitrary value for haemoglobin which is used to separate normal from anaemic state. Stage 3 is 'iron deficient anaemia', in which haemoglobin levels are subnormal. The anaemia state is defined by 2 criteria: decrease of the iron stores and decrease in haemoglobin levels.[20] Thus, an isolated decrease in ferritin does not indicate an anaemia, but it does indicate a risk of anaemia soon if the iron stores continue to be depleted.

The functional consequence of iron depletion is anaemia (stage 3). Anaemia impairs blood gas transport (O_2 and CO_2) and limits work capacity. It may be responsible for fatigue, weakness, dizziness and sensitivity to cold. The cardiovascular adaptations to anaemia mask most of the problems when the person is at rest. During exercise, when the energy demand is maximum, the first symptom of anaemia is a drop in performance or a plateau. The decrease in performance is proportional to the loss of haemoglobin. Even a small decrease of 1 to 2 g/100ml causes a 20% decrease in performance,[75] which can be restored by blood transfusion.[76] The lack of haemoglobin could also lead to decreases in intellectual performance.[77] Iron deficient erythropoiesis without objective anaemia (stage 2) may induce clinical manifestations, muscle and hormonal dysfunction, and altered resistance to infection.[26]

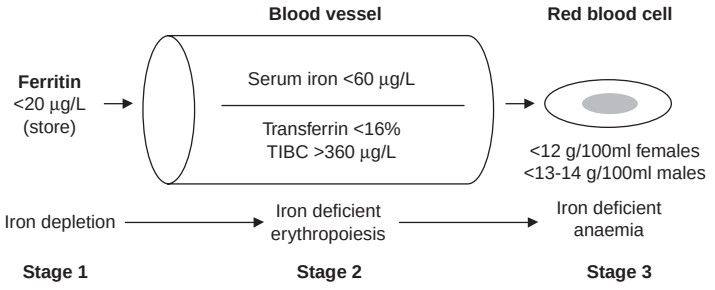

Fig. 2. Stages of iron deficiency as defined by Bothweel et al.[26] **TIBC** = total iron binding capacity.

3.2 Erythropoiesis

A moderate exercise haemolysis leads to a physiological adaptation, with an increase in the erythropoietin levels some days after exercise.[78] This gives rise to younger, larger red cells, so that the mean volume of the red cells is increased together with the number of the reticulocytes.[17,18,41,42,79] The proportion of younger red cells can be quantified by densitometry or by measuring the ratio of 2 red blood cell cytoskeletal proteins, the phosphoproteins 4.1a and 4.1b.[80] Although the younger cells are relatively low in haemoglobin, they are better for O_2 transport. They are more deformable and have higher 2,3-diphosphoglycerate levels.[32,79,81] They are in part responsible for the increase in the haemoglobin P50 that occurs after training.[18,21,81] These adaptations might lead to a decrease in the erythropoietin secretion and hence a decrease in red cell production,[74] but this was not confirmed by Weight et al.[21] Instead, they found that the circulating erythropoietin levels of 135 athletes was similar to that of 40 sedentary people.

3.3 Muscle Enzymes

Many redox enzymes, such as the mitochondrial cytochromes, contain iron. Sideropenia could thus cause a decrease in aerobic metabolism, without any anaemia being apparent.[2,3] Ohira et al.[82] showed that there are beneficial effects of iron treatment that cannot be related to changes in haemoglobin. They showed that iron supplementation was more significant to increase work capacity in iron deficient groups than in normal iron groups even when both groups had similar haemoglobin levels (i.e. 11.9 versus 11.8 g/100ml). This could explain the disappearance of asthenia after preventive iron supplementation. But not all the studies are in agreement on this.[22,76]

4. Practical Recommendations for Treating Anaemia and Iron Deficiency in Athletes

An algorithm for guiding treatment decisions is shown in figure 3. Treatment is designed to prevent the loss of iron stores and to prevent a real anaemia and/or a physical asthenia. The effectiveness of treatment is judged by clinical (increased physical performance, disappearance of the asthenia) and laboratory criteria (increased iron stores and haemoglobin levels). The haemodilution that often occurs in the athletes makes the diagnosis and supervision of treatment difficult. It is often only after testing that the need for preventive and/or curative treatment is established. Unfortunately, a placebo effect can never be ruled out.

4.1 Prevention

A decrease in iron stores can naturally be prevented by a combination of a balanced diet and planned training, with the most intensive training periods being balanced by rest periods.

4.1.1 Diet

About 10 to 15% of the iron intake is absorbed by the intestinal mucosa.[31] The daily loss of iron

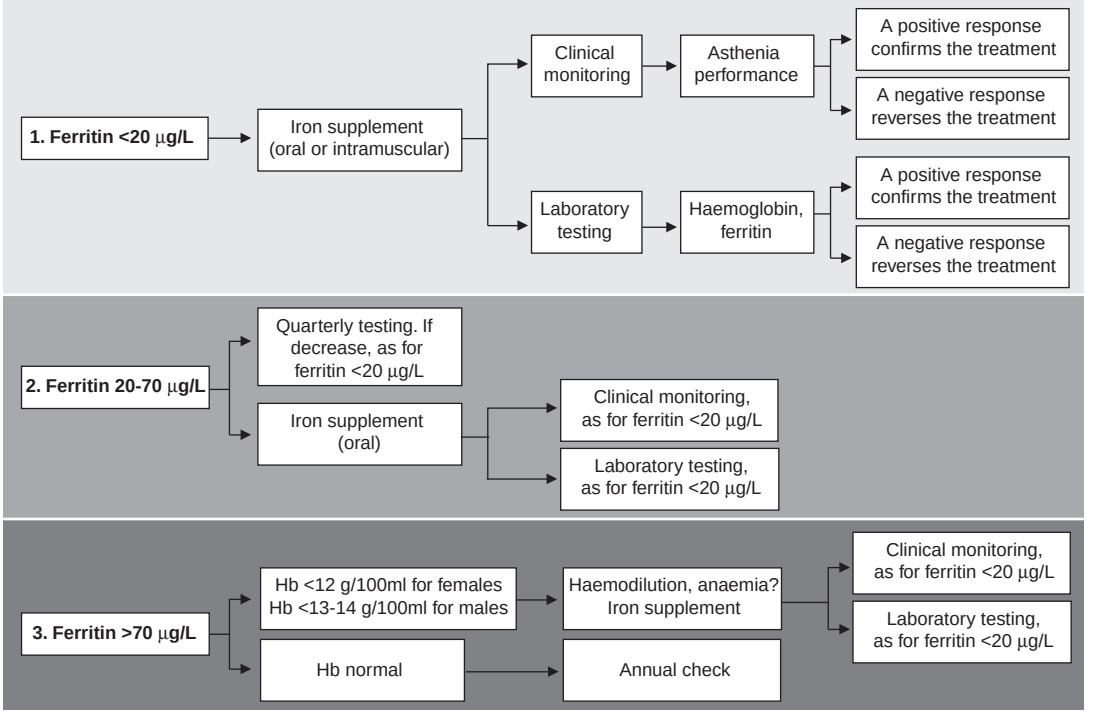

Fig. 3. Treatment algorithm based on plasma ferritin and haemoglobin levels (C reactive protein is considered normal). **Hb** = haemoglobin.

is less than 2mg for men and 2.3mg for women. Thus, the normal intake is 10 to 23 mg/day for men and women.[31] The best sources of iron are those containing heme iron: red meat, liver, kidney, heart. This kind of iron is better absorbed that the others (about 23% of the intake). Shiraki et al.[83] found a daily intake of 2g of animal protein per kilogram of bodyweight prevented a major iron deficit. Non-heme iron, the other kind of food iron, is abundant in peas, nuts, bread, cereals, leafy vegetables, eggs, dried fruit and wine. Intestinal absorption is less efficient (3 to 8%), but is helped by the presence of vitamin C,[84] chicken meat or fish. Non-heme iron absorption is inhibited by calcium, phosphate, phytates, bran, polyphenols (found in tea) and antacids. A diet rich in fibre can also reduce iron availability,[45] as does excess tea[85] or indigestion medicines such as magnesium, aluminium and calcium salts and oxides,[86] or endogenous bile secretion.[87]

The average, western diet contains 5 to 6mg of iron per 4000kJ of energy. As such, it is difficult to rebuild iron stores, especially those of the women whose daily requirement is over 20mg.[1,30,31] Growing adolescents also have daily requirements that are not satisfied by this intake.[33] In some sports, such as gymnastics, marathon running, or ballet dancing, athletes tend to reduce their food intakes to keep their bodyweight down, increasing the risk of an insufficient iron intake.

4.1.2 Moderate Altitude

Erythropoiesis is stimulated by living at a moderate altitude.[88-92] Living high and training low is used by athletes to improve sea level performance.[93,94] Indeed, well trained competitive runners living at an altitude between 2500 to 3000 metres above sea level maximise acclimatisation while minimising acute mountain sickness and muscle wasting. They increase red blood mass volume, haemoglobin and oxygen

carrying capacity of the blood and aerobic power after return to sea level. On average, 4 weeks of living at an altitude of 2500 metres above sea level increases red blood cell mass volume by 10% and maximal oxygen uptake by 5% above normal levels.[94] Training low, while living high, also allows athletes to maintain higher intensity of training and to improve running performance at sea level in direct proportion to the increase in maximal oxygen uptake.[94] Indeed, hypoxia at higher altitude limits training intensity, which in elite athletes may result in relative deconditioning.[93]

Erythropoietin secretion decreases after return to a low altitude. Wolski et al.[92] suggests that there may be a risk of a reactional anaemia 1 or 2 months after descent. This opinion is supported by lower haemoglobin levels found 2 months after descent from moderate altitude training by Haymes et al.[95] However, it may also be the result of an expansion of the plasma volume.[95]

For successful altitude training, adequate iron stores are necessary. Indeed, failure of red cell volume increase was found after 4 weeks altitude exposure at 2500m in athletes beginning training with pre-existing iron deficiency.[96]

4.1.3 Iron Supplementation

People at risk of an iron deficiency (serum ferritin below 20 μg/L and degree of saturation of transferrin below 16%), can be given an iron supplement in a fasting state (e.g. 100mg of ferrous sulphate per day, 10 to 20% of which is absorbed by the intestine for 20 consecutive days per month). Athletes with normal ferritin levels should not take iron supplements.

4.1.4 Treatment Monitoring

Treatment is monitored by measuring plasma ferritin levels and the degree of saturation of transferrin at the end of the main training periods. This criteria is not valid in people with inflammatory syndrome,[95] quantified by the C reactive protein levels. Inflammation increases ferritin production without increasing the iron stores in the liver or bones.[28] Inflammation diminishes also the transferrin iron binding capacity and the total serum iron.[26] Ferritin level criteria is not valid after an ultra-marathon as it remains artificially elevated for 1 to 2 weeks.[13] Thus, a high level of plasma ferritin can be erroneously reassuring. This can be avoided by measuring the ferritin levels in the red cells, where levels are more stable.[6]

The appearance of reticulocytes in the circulation is a good indication of the efficiency of the iron supplementation. A small increase occurs after 4 to 7 days of treatment. If there is no clinical response to a 1-month course of iron supplementation, there is no point in continuing the treatment.[97]

The red cell count, haematocrit or haemoglobin levels are not sufficient to monitor the treatment in athletes because of the haemodilution that occurs in exercise or training. This haemodilution can vary greatly, up to 25% within a few days period (Chatard, unpublished data). These variations can be easily observed day-by-day by measurement of the haematocrit or the plasmatic volume following the Blue Evans Technique.[98] These variations were shown to depend on the training load.[14,15,99] Thus, to interpret correctly the blood sample measurements, a stable training period is needed as far as possible from the competition period.

4.2 Treatment

4.2.1 Iron Supplements

Iron supplements were first used to treat anaemia in France in 1832.[1,97] Nowadays, iron supplements are given as soon as ferritin levels drop below 12 to 20 μg/L[1,3,30] for 1 to 3 months.[33] Ascorbic acid (vitamin C) supplements can also be given. Some authors have also used iron supplements for patients with higher ferritin levels (30 to 70 μg/L),[30] since the ferritin levels can be elevated because of training, inflammation or other diseases, without any link to the iron reserves.

Iron deficiency increases intestinal iron absorption,[97] and absorption can reach 50% of the intake.[35] However, this positive adaptation is sometimes missing in trained athletes.[2,31] This explains the variable effect of iron supplementation from one person to another.[89] Telford et al.[72] reported a ferritin increase of 10 μg/L in people given a 300 mg/day iron supplement for 15 weeks, while

LaManca and Haymes[73] observed a ferritin increase of 8 μg/L after only 8 weeks on an iron supplement of 100 mg/day. An intramuscular injection of iron can be given if the oral treatment is inadequate, as injection is more efficient than oral supplement.

4.2.2 Special Precautions

Iron intake may not be well tolerated, as it can cause intestinal cramps and black stools when the dosage is too high (over 200 mg/day) or the iron is consumed with an empty stomach.[33,77,100] Liquid iron can stain the dental enamel but this can be avoided by using a straw.[77,100] The absorption of iron salts is decreased by the ingestion of magnesium, aluminium or calcium salts, oxides and hydroxides. The intestinal absorption of iron competes with the absorption of some other minerals, and may cause copper or zinc deficiencies.[1,2] It also decreases the absorption of certain antibiotics (e.g. tetracyclines and fluoroquinolones), penicillamine, bisphosphonates and thyroxine.[77,100,101] Iron salts should therefore be taken 2 hours before other medicines.[86]

The percentage of athletes who take iron supplements for periods that may exceed 6 months is high, up to 94% according Burke and Read.[33] This type of supplementation without any medical supervision should be avoided. Excess iron is toxic for the liver and ferritin levels higher than 200 μg/L are associated with increased coronary risk.[102] The intramuscular injections of iron can also be dangerous because of the possibility of anaphylactic shock, facilitated by oral ingestion of iron salts, which can be fatal.[30,33,77,86,100]

4.2.3 Treatment Monitoring

The same procedures as were used to monitor preventive measures are used to monitor treatment, but more attention is paid to the haemoglobin levels. Iron supplementation should be discontinued if haemoglobin levels do not increase. Magazanik et al.,[103] Lukaski et al.,[71] Celsing et al.[76] and LaManca and Haymes[73] found that the increase attributable to iron supplementation is proportional the increase in haemoglobin levels. But Fogelholm et al.[104] and Klingshirn et al.[105] did not find this relationship.

5. Conclusion

Regular, intense physical activity is often associated with low iron stores as measured by plasma ferritin levels. In normal training conditions, with no infection or inflammation syndrome, as long as ferritin levels are above 20 to 30 μg/L, and the degree of saturation of the transferrin above 16%, iron supplements are not necessary. A supplement can be given when ferritin levels and the degree of saturation are below these values. In other conditions, supplementation may be considered. A positive clinical or laboratory response indicates there was a deficit that required treatment.

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