

## **Iron for the Athlete**

By Peter Peeling PhD

It is well established that iron deficiency is the world's most common nutrient disorder. However, the role that iron plays in the body is fundamental to a number of key processes that sustain life, such as oxygen transport, energy production, and immune function. Clearly, such processes are also vital to athletic performance; yet iron deficiency is also a very common diagnosis amongst the athletic population.

The common symptoms of an iron deficiency include undue feelings of fatigue, lethargy and listlessness. Furthermore, as this issue progresses towards an anaemic state, the individual will begin to show haematological signs of a reduced haemoglobin concentration. Such symptoms may lead to a compromised physical work capacity and cognitive function, which will no doubt have carry-over effects to the performance of both endurance and team sport athletes. This is an important point, since it is commonly perceived that an iron deficiency is only relevant to 'long distance' athletes; however, not only do team sport players compete for a prolonged period of time (60-120 min), they also cover a lot of ground aerobically, and they need to make good decisions when on the field. As a result, optimal iron stores should be considered as an important factor for all athlete types.

Previously, it has been established that the development of an iron deficiency in athletes is multi-faceted. Well accepted mechanisms to explain the high prevalence in this group include exercise-mediated processes such as gastrointestinal bleeding, haematuria, sweating and haemolysis (impact-related red blood cell destruction). Additionally, it is suggested that the menstrual cycle of female athletes places them at a further risk of developing an iron deficiency due to the iron lost through menses, and that vegetarian athletes are also at a greater risk due to the less efficient absorption (and therefore replacement) of non-haem iron sources. No doubt, it is the culmination and combination of these mechanisms that can ultimately result in an athlete developing an iron deficiency over time.

In addition to these more established rationale, recent literature has suggested that there may also be a hormonal link to the development of an iron deficiency in athletic populations (1). Back in the early 2000s, a group of scientists (2) discovered a liver-produced peptide that appeared to influence the regulation of iron absorption in the gut. This hormone was named Heparin. Heparin functions by binding to the iron exporter channels of the body known as Ferroportin, effectively shutting them down and not allowing iron to enter the system. In fact, it is now known that individuals with the genetic iron overload disorder, haemochromatosis, actually have a deficiency in producing the Heparin hormone, hence, meaning they have a dysfunctional internal process of controlling how much iron is in their system. Further work has since shown that Heparin has a number of regulators in the body, each of which seems to increase or decrease the amount of hormone produced, dependent upon the body's iron requirement relative to the stimulus. For instance, an increase in inflammation [namely an increase in the inflammatory cytokine Interleukin-6 (IL-6)] appears to increase the circulating Heparin levels in the body. This makes sense, since the response would be to shut down the body's iron absorption in order to prevent any host infection (presumably causing the inflammation) from gaining momentum due to iron sources.

In contrast, stimuli such as altitude exposure (which causes the production of erythropoietin) appears to decrease Heparin levels in order to increase iron absorption, in an attempt to meet the haematological demand for increased erythropoiesis. So as you can see, the production of Heparin seems to be a homeostatic-driven process based on iron demand.

Although both protective and adaptive in function, the issue with this mechanism described above is that exercise causes an acute increase in one of the precursors known to increase Heparin production – Inflammation. It is well-known that exercise causes a transient inflammatory response, where amongst many

other inflammatory mediators, a large increase in the signalling cytokine IL-6 is seen. As established above, IL-6 is a key positive driver for Heparin production, and as a result, our research group looked to establish the time course of exercise effects on the inflammatory and subsequent Heparin responses in athletes. This paper, published in the International Journal of Sports Nutrition and Exercise Metabolism (3), showed that when compared to a 60 min period of seated rest, 60 min of continuous running-based activity resulted in a significant increase in IL-6 immediately post-exercise, and a significant increase in Heparin levels after 3-6 h of recovery. In a follow-up study where we had participants exercise twice in a 12 h period, it was noted that the Heparin levels had returned to baseline by 12 h after the completion of the initial exercise bout (4). When put into a practical daily training context, it would likely be typical that an athlete who trains in the morning would be consuming a lunch meal, or an athlete that trains in the afternoon would be consuming a dinner meal at approximately the same time as their body is producing a hormone that will prevent them from efficiently absorbing the iron from that meal. As a result, it is possible that if the timing of this meal regularly coincides with the peak post-exercise production of Heparin, then over-time, this hormone function may prove to be a contributor to the aforementioned mechanisms behind the prevalence of iron deficiency seen in athletes.

### **So how do we combat this issue from a nutritional or a supplementation perspective?**

Well, there are probably two ways we could approach it. Firstly, the nutrition expert may work with the athlete's training plan to prescribe oral iron supplementation (i.e., ferrous sulphate) or high iron containing meals at a time when Heparin levels should be at their lowest. This might occur prior to training sessions, 8-12 h after a training session, or periodised on rest days (i.e., consuming higher dietary iron intake or a greater amount of iron supplement on a scheduled rest day). The second approach might be to by-pass the gut entirely through intravenous or intramuscular iron supplementation. Recently, there have been some good improvements in iron status and subsequent athletic performance outcomes shown from such an approach (5), however, there are known issues with these parenteral iron preparations, which in extreme (and rare) cases may lead to anaphylaxis.

Therefore, any decision to pursue these alternative supplementation regimes should be made in consultation with a trained sports physician. Notwithstanding, it should also be mentioned that the WADA code for supplement use should be continually checked before undertaking any alternative supplementation practises, primarily as a result of the ever-changing nature of the banned substance and method of administration list.

**In conclusion**, it is clear that iron deficiency is a common issue seen in athletic populations. In addition to the well-known contributors to this issue, more contemporary literature has started to consider how hormonal regulation may play a role. Given the evidence, the time course profile of post-exercise inflammation and Heparin production recently established should be considered when determining the best iron feeding / supplementation protocol for an athlete. However, being aware of the athlete's iron needs through regular screening (i.e. every 3 months) would be a good place to start gaining an understanding of the individual's specific iron demands from training, since a 'one size fits all' approach may not be relevant here.

### **Peter Peeling PhD**

*School of Sport Science, Exercise and Health.  
The University of Western Australia.*

### **References**

Peeling, P., Dawson, B., Goodman, C., Landers, G., Trinder, D. Athletic induced iron deficiency: new insights into the role of inflammation, cytokines and hormones. Eur J Appl Physiol. 2008;103:381-91.

Park, C. H., Valore, E. V., Waring, A. J., Ganz, T. Hepsidin, a urinary antimicrobial peptide synthesized in the liver. *J Biol Chem.* 2001;276:7806-10.

Peeling, P., Dawson, B., Goodman, C., Landers, G., Wiegerinck, E. T., Swinkels, D.W., Trinder, D. Effects of exercise on hepcidin response and iron metabolism during recovery. *Int J Sport Nutr Exerc Metab.* 2009;19:583-97.

Peeling, P., Dawson, B., Goodman, C., Landers, G., Wiegerinck, E. T., Swinkels, D.W., Trinder, D. Cumulative effects of consecutive running sessions on hemolysis, inflammation and hepcidin activity. *Eur J Appl Physiol.* 2009;106:51-9.

Garvican, L. A., Lobigs, L., Telford, R., Fallon, K., Gore, C. J. Haemoglobin mass in an anaemic female endurance runner before and after iron supplementation. *Int J Sports Physiol Perform.* 2011;6:137-40.