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BRIEF NOTE

Iron deficiency and aerobic endurance performance in a female club runner

Carence en fer et performance aérobie chez une coureuse de niveau compétitif

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KEYWORDS
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Summary
Objective. — This study aimed to monitor longitudinal changes in performance and aerobic adaptations as well as ferritin (Fer) and additional markers pertaining to iron status in an iron deficient female runner.
Methods. — Over 26 months, a venous blood sample was taken every 3 months, lactate profiling and VO_{2max} tests were conducted every 5—6 months. Running performance was recorded during 5 km timed runs (Parkrun). The intervention consisted of iron supplementation, increases in dietary iron intake and implementing a structured approach to training.
Results. — Fer values increased from 4 to 40 $\mu g.L^{-1}$ during the period of investigation. Running times improved from 28:02 (m:s) to 23:15. Hemoglobin increased from 9.8 $g.dL^{-1}$ to 13.7 $g.dL^{-1}$. Hematocrit ranged from 31.8 to 37.9%, peaking at 40.4%. Mean corpuscular volume and hemoglobin increased from 70 fl to 89 fl and 21.5 pg to 30.1 pg, respectively. VO_{2max} improved from 47.40 to 55.79 $mLO_2.kg.min^{-1}$.
Conclusions. — Our results demonstrate that iron supplementation, increase in dietary iron intake and structured training induced improvements in: (i) Fer and other markers of iron status, (ii) running performance and (iii) VO_{2max} of an iron deficient female club runner.

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MOTS CLÉS
Ferritine ;
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Entraînement ;
VO_{2max} ;
Lactate sanguin

Résumé

Objectif. — L'objectif de cette étude de cas longitudinale était de quantifier les améliorations de performance, les adaptations de la filière aérobie ainsi que les niveaux de ferritine (Fer) et autres marqueurs pertinents chez une coureuse à pied ayant une carence en fer.

Méthodes. — Sur une période de 26 mois, une prise de sang était effectuée tous les trois mois ; l'évaluation du profil des lactates et de VO_{2max} était faite tous les 5—6 mois. L'évolution de la performance en course à pied était mesurée lors de courses chronométrées de 5 km (Parkrun). L'intervention consistait en la prise de compléments ferriques ainsi que l'augmentation de la prise d'aliments contenant du fer et la mise en place d'une approche structurée de l'entraînement.

Résultats. — Les valeurs de Fer ont augmenté de 4 à 40 $\mu g.L^{-1}$ pendant la période étudiée. Les temps de course se sont améliorés de 28:02 (m:s) à 23:15. Les niveaux d'hémoglobine ont augmenté de 9,8 $g.dL^{-1}$ à 13,7 $g.dL^{-1}$. Ceux d'hématocrite ont augmenté de 31,8 à 37,9 %, avec un pic à 40,4 %. Le volume globulaire moyen et la teneur globulaire moyenne en hémoglobine ont augmenté de 70 fl à 89 fl et 21,5 pg à 30,1 pg, respectivement. Une amélioration de VO_{2max} a été observée de 47,40 à 55,79 $mlO_2.kg.min^{-1}$.

Conclusions. — Les résultats de la présente étude démontrent que la prise de compléments ferriques, l'augmentation de la prise d'aliments contenant naturellement du fer associées à la mise en place d'une approche structurée de l'entraînement ont induit des améliorations de : (i) Fer et autres marqueurs pertinents, (ii) performances en course à pied et (iii) VO_{2max} chez une coureuse ayant une carence en fer.

Introduction

Iron plays a vital role in physiological processes such as oxygen transport and energy production [1]. The ability to sustain a relatively high percentage of maximal oxygen uptake (VO_{2max}) over a prolonged period of time is dependent upon iron tissue concentrations. This is explained by the association between endurance performance and the activity of iron-dependent oxidative enzymes [2]. Hemoglobin (Hb) is another variable of interest as the iron-containing protein in red blood cells increases the blood's oxygen-carrying capacity and is one of the main determinant of VO_{2max} [2,3]. Therefore, a deficiency in iron and Hb affect oxygen transport and use and ultimately athletic performance during both sub-maximal and maximal intensity efforts.

Women experience iron deficiency (ID) more frequently than men partly due to a net negative iron balance [2,4]. Furthermore, iron depletion state is more prevalent in habitual female runners compared to their inactive counterparts [5]. Dietary iron treatment methods as well as iron supplementation have been shown to improve iron status and female physical performance [6,7].

Two longitudinal case studies have reported physiological adaptations to training [8,9].

Recently, Pedlar et al. case study showed that despite an ID, performance can improve over a 28-month period in an iron deficient female elite runner [10]. The aforementioned studies were conducted in elite class runners and markers of iron status other than ferritin were not investigated. The present case study considered performance data spanning a 26-month period, including a 25-month period of physiological test data and venous blood data, in a female non-elite runner (referred to as ''the athlete''). We aimed to demonstrate how increased iron levels allowed the athlete to adopt a more structured approach to training, including steady and heavy intensity sessions which had been previously impossible to implement due to fatigue, and how this consequently improved aerobic endurance performance.

Background

The athlete was a 35-year-old club runner, initially seen in May 2014 with an ongoing history of underperformance as well as low Hb and low iron. The athlete trained 4—5 times a week and participated in 5 km, 5 mile and 10 km running races as well as Parkrun events (5 km). She had not previously sought Sport Science support, nor consulted a Sport Medicine doctor and had a full time job.

The athlete wanted to improve her running performance as it had plateaued. Through informal conversations with the first author, it appeared that her training structure was not optimal. For example, the athlete would not train at varying intensities nor periodise. She reported to have been mostly training at the same running pace/speed in the years before the study. Furthermore, the athlete mentioned experiencing regular episodes of fatigue and headaches or lacking of energy. As a result, it was impossible for her to do high-intensity running intervals on a weekday. Instead, she would wait until the weekend to do so. The athlete had a history of low Hb but had never been given advice on how to treat it. The athlete reported that her diet might not be adequate and might not include sufficient iron containing foods (e.g, leafy vegetables, red meat). It was then suggested to seek medical advice from a Sport Medicine physician to further investigate the causes of these episodes of fatigue. After a first blood test showing an ID, the plan of support was initially to do lactate profiling — VO_{2max} to determine training zones and assess current level of cardiorespiratory fitness. Next we would provide training advice and implement supplementation and dietary changes to improve the lack of iron. We planned to do lactate profiling — VO_{2max} tests and blood tests at regular intervals in order to monitor progress and adjust training intensities accordingly. Thorough explanation of the protocols and assessments performed was provided to the athlete as well as all possible risks associated with her participation in the study. Following this, written informed consent was given. The study conformed to the declaration of Helsinki. A health history and Physical activity readiness (Par-Q, i.e., a screening tool) questionnaires as well as consent forms were filled before each lactate profiling and VO_{2max} tests. The athlete had no history of cardiovascular disease and did not suffer from any injury.

Methods

Lactate profiling and VO_{2max} assessments

The athlete had been instructed to arrive at the laboratory at least three hours after eating and to avoid strenuous exercise in the 48 h preceding a test session. Testing took place between 09:30 and 12:30 and temperature in the laboratory was maintained in the range of 19—20 °C. After an introduction to the laboratory and re-iteration of the protocol, baseline measurements were performed (i.e., blood pressure, heart rate and blood lactate concentrations at rest). This was followed by a 10 minute warm-up on a treadmill (Quasar, HP Cosmos, Germany). The facemask was worn half way through the warm-up to familiarise the athlete with it. The athlete was familiar with treadmill running.

Following this, a lactate profiling test was conducted where the treadmill belt's speed was increased by 1 km.h^{-1} every 3 minutes [11].

At the end of each stage, the athlete stepped on the side of the treadmill and capillary blood lactate samples were taken from her fingertip and analysed within about 15 s of collection with a hand-held portable lactate analyser (Lactate Pro 2, Akray, Japan).

The lactate profiling test was followed by a 10 minute recovery period. Then, a fast ramped protocol was conducted where the treadmill belt's speed was increased by 1 km.h^{-1} every minute until the athlete reached her maximal exercise capacity. This second part of the assessment was designed to elicit and measure VO_{2max} and its associated running speed as well as maximal heart rate. Both protocols were conducted with a treadmill slope set to 1% [12]. Breath-by-breath gas exchange measurements as well as heart rate were collected throughout the tests using a gas analyser (CPET, Cosmed, Italy). The day to day coefficient of variation in VO_{2max} determination using the laboratory's CPET unit is 2.5%. The athlete breathed through a low dead space and low resistance turbine. Gases were continuously drawn from a flow meter through a 2 m capillary line of small bore, and analysed for O_2 and CO_2 concentrations. Expiratory volume was determined by a flow meter calibrated before each test using a known volume syringe (i.e., 3 litres). Gas analysers were calibrated before each test against room air and gases of known concentrations (i.e., 5% CO_2 and 16% O_2). Respiratory gas exchange variables were averaged every 15 seconds. These physiological tests were conducted every 5—6 months.

Blood tests

Blood tests were prescribed by the second author and samples were taken in the afternoon, between 13:00 and 17:00 by a qualified nurse in the haematology department of a hospital in the Dublin area. The variables of interest were: Ferritin (Fer), Hb, Hematocrit (Hct), Mean Corpuscular Volume (MCV) and Mean Corpuscular Hemoglobin (MCH) as well as B_{12} vitamin. They were measured according to the hospital's laboratory standard operating procedures and at about three month intervals.

Intervention

Nutrition

At the initial consultation with the second author in May 2014, it was suggested to increase dietary iron intake with natural sources (e.g., red meat). Therefore, the athlete's diet included the following: Organic beef filet (twice a week), black pudding and organic leafy vegetables. Red meat was often accompanied with organic tomato sauce or organic tomatoes as these vitamin C containing foods facilitate iron absorption [2]. Two to four organic eggs were consumed per week. The athlete's caffeine intake was relatively low between May 2014 and July 2015 (one cup of coffee per day) and then close to none as the coffee drink was replaced by organic cacao powder from August 2015 onwards. This powder had high iron and vitamin C contents, 46.26 mg and 31.25 mg per 100 g, respectively.

The athlete tried different iron supplements and a 5 mg supplementation with added vitamin C was chosen as she experienced gastric discomfort when ingesting iron supplements of higher iron content. A sachet contains 25 mL of iron-rich water (nutritional information: 80 mg of Vitamin C and 5 mg of iron).

This oral iron supplementation was ingested once a day from July 2014 then twice a day from October 2014 (i.e., a daily supplementation of 10 mg of iron).

Training

The athlete trained consistently 5 times a week during the period investigated. Her training had been interrupted for 2—3 weeks over the period investigated due to colds or soreness in the lower limbs. The major challenge she experienced was to train at the prescribed intensities as, in the early stages of the study, the low levels of iron prevented her from doing so. For example, tempo runs had to be run at a slower than predicted pace as the athlete would get tired. Similarly, she would not recover as well as a non-iron deficient athlete following a 50—60 min easy run.

Between December 2014 and March 2016, a total of 15 treadmill-based training sessions were performed to provide the athlete with a better understanding of the importance of training at varying intensities (i.e., differences between easy, steady and heavy paces). Also, doing these runs on a treadmill ensured a controlled and even pace throughout the session. For example, the athlete would do 20—30 min steady runs at 10—11 km.h^{-1} and, towards the end of the study, long intervals including four repetitions of 5 min ran at 13 km.h^{-1} with 2 min recovery between the repetitions. In the early stages of the period investigated, the athlete could not complete the aforementioned sessions as fatigue would develop quickly due to the low levels of iron.

Results

As can be seen on Fig. 1, Fer values increased from 4 to 40 μg.L^{-1} during the period of investigation. Times over 5 km events (i.e., Parkruns) performed on the same course improved from 28:02 (m:s) to 23:15 between December 2013 and February 2016.

The first blood test results revealed a low Hb of 9.8 g.dL^{-1} as well as low MCV and MCH, 70 fl and 21.5 pg, respectively. Hb reached a peak of 14.1 and then plateaued to 13.7 towards the end of the study. MCV and MCH followed the same trend, their highest values being 89 fl and 30.1 pg, respectively. Between May 2014 and June 2016, Hct values ranged from 31.8 to 37.9%, peaking at 40.4% in March 2015. Vitamin B_{12} was toward the lower end of the normal range at 181 ng.L^{-1} and 151 ng.L^{-1} in August 2014 and September 2015, respectively. Thus, although not planned initially, injections were performed three times in November, December 2015 and January 2016 (see Fig. 2). These resulted in an increase in vitamin B_{12} to > 2000 ng.L^{-1} (end of January 2016). Vitamin B^{12} value decreased to 418 ng.L^{-1} in June 2016, which is well within the normal range.

Between November 2014 and February 2016, running speed at VO_{2max} improved from 14 to 16 km.h^{-1}. Absolute and relative VO_{2max} improved from 2.84 to 3.25 L.min^{-1} (+ 12.6%) and 47.40 to 55.79 mLO_2.kg.min^{-1} (+ 15%), respectively. Body mass remained stable at about 60 kg between November 2014 and October 2015 and then decreased to 58.3 kg in February 2016 (see Fig. 3) and was 58.5 kg in June 2016.

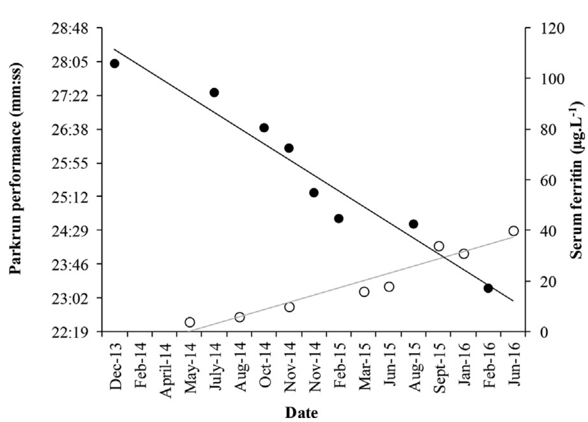

Figure 1 Serum ferritin values (open circles) and Parkrun running times (min:s; closed circles) during the period studied. Linear trend lines of best fit are shown (upper line: R^2 = 0.89 for running times and lower line: R^2 = 0.93 for Fer).

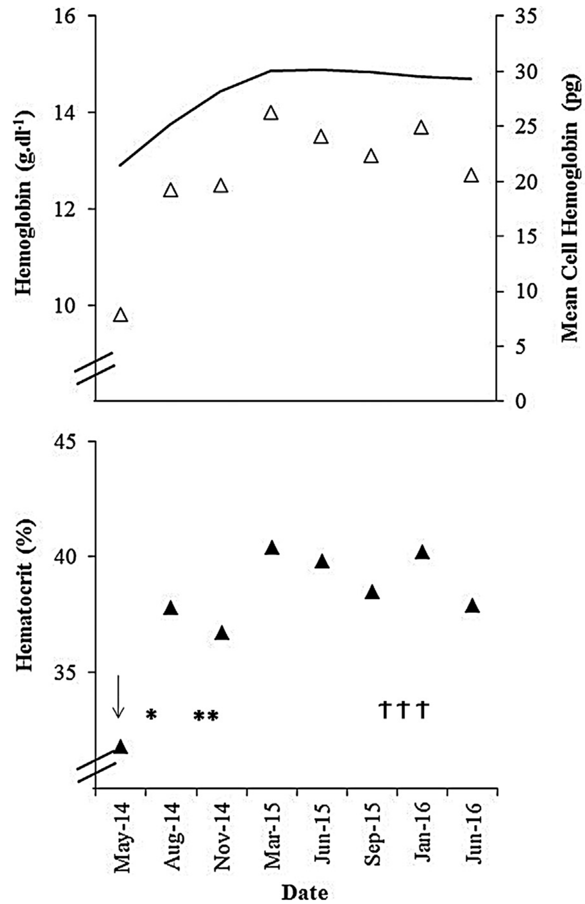

Figure 2 Hemoglobin (△), Mean Cell Hemoglobin (— ; top) and Hematocrit (▲ ; bottom) values during the period investigated. The black arrow (↓) represents the beginning of the dietary intervention, the asterisks the beginning of iron supplementation (*5 mg and **10 mg of iron daily) and each cross (†) a vitamin B^{12} injection.

The velocity at lactate threshold (i.e., lactate value of ~ 2 mmol.L^{-1}) was 10 km.h^{-1} in November 2014. It improved to 11 km.h^{-1} in March 2015, decreased to 10 km.h^{-1} in October 2015 to come back to 11 km.h^{-1} in February 2016. The greatest improvement in blood lactate concentration ([La]) was seen at 12 km.h^{-1} where it decreased between November 2014 and February 2016 from 5.9 to 2.5 mmol.L^{-1} (a 57.6% decrease). Another noticeable improvement was observed at 11 km.h^{-1} where [La] decreased from 3.5 to 1.8 mmol.L^{-1} during the period studied (see Table 1).

Discussion

The present case study aimed at monitoring longitudinal changes in performance and aerobic adaptations as well as iron and additional markers pertaining to iron status.

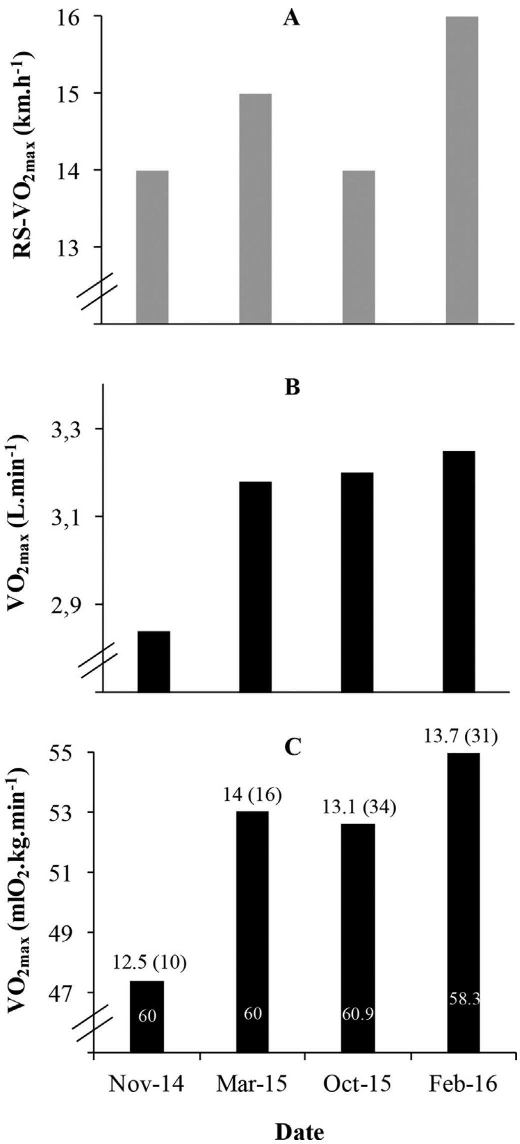

Figure 3 Treadmill running speed at VO_{2max} (A), absolute (B) and relative (C) VO_{2max}. Body mass (kg) values are inserted in the bars. Hb and (Fer) values indicated above the bars, units are $g.dL^{-1}$ and $\mu g.L^{-1}$ respectively.

Nutrition intervention

Our data shows that Fer increased throughout the study due to the combined effects of iron supplementation [13,14] and an increase in dietary iron intake (i.e., red meat) and vitamin C [2,6]. The observed linear increase in Fer is different from Pedlar et al. case study investigating an iron deficient female Olympic runner [10]. A possible explanation is that the athlete investigated herein was a recreational runner with a lower training volume than an elite athlete. Therefore, exercise-induced inflammation and hepcidin activity were lower, resulting in a constant increase in Fer during the period investigated. Further, VO_{2max} improved from 47.40 to 55.79 $mLO_2.kg.min^{-1}$. These results are in agreement with previous studies showing VO_{2max} improvements following iron supplementation [7,15,16]. Recently, an increase in VO_{2max} has been reported among participants with severe iron deficiency (i.e., ferritin levels < 12 $\mu g.L^{-1}$) after 10—12 weeks of iron supplementation [14]. Although some studies have failed to show an improvement in VO_{2max} following iron supplementation [17], it seems that increases in VO_{2max} are observed among participants with severe iron deficiency which is the case in the present study.

Training intervention

Consistent and structured training has played a role in the increase in VO_{2max}. Certainly due to Hb and Fer increases, the athlete could regularly perform interval training at the speed associated to VO_{2max} or slightly above during most of the duration of the study. This type of training has been suggested to provide an effective stimulus to improve VO_{2max} [18,19]. The athlete could not include both steady and heavy intensity runs in her training routine before the start and during the early stages of the study as her Fer and Hb levels were low (see Tables 2 and 3). ID induced fatigue prevented her from doing steady and/or heavy intensity training. Furthermore, because the athlete could train consistently with ID symptoms substantially decreasing over time, her oxygen carrying capacity improved (see Fig. 2). Banfi et al. reported that elite cyclists Hb and Hct decreased during periods of intense training and came back to their initial values following recovery [20]. Our data show that Hb and Hct increased over time (i.e., improved oxygen carrying capacity) due to

Table 1 Treadmill speeds and [La] during the period investigated.

Treadmill speed ($km.h^{-1}$)	[La] ($mmol.L^{-1}$)			
	08/11/14	31/03/15	03/10/15	19/02/16
0	1.1	1.2	1.3	1.1
8	1.8	1.3	1.6	1.2
9	1.5	1.2	1.4	1.1
10	2.2	1.3	1.8	1.4
11	3.5	2	2.4	1.8
12	5.9	3.3	3.9	2.5
13	N/A	4.8	6.4	4
14	10.2	N/A	8	N/A
15	Not attained	7.2	Not attained	N/A
16	Not attained	Not attained	Not attained	10.6

Table 2 Overview of the athlete's training progression as a function of increases in hemoglobin (Hb) and ferritin (Fer).

Blood test dates	Hb ($g.dL^{-1}$)—Fer ($\mu g.L^{-1}$)	Steady runs (LT pace)	Heavy runs (LTP pace)
May—14	9.8—4	Unable to do continuous steady runs	Unable
Aug—14	12.4—6	Unable to do continuous steady runs	6 (1 km at ~11 $km.h^{-1}$); R 3 min
Nov—14	12.5—10	25 min at 10.5 $km.h^{-1}$[a]	5 (1 km at ~12 $km.h^{-1}$); R 3 min
Mar—15	14—16	30 min at 11 $km.h^{-1}$[a]	No heavy sessions performed
Jun—15	13.5—18	30 min at 11 $km.h^{-1}$[a]	6 (1 km at ~13 $km.h^{-1}$); R 3 min
Sept—15	13.1—34	52 min at 11.5 $km.h^{-1}$[b]	6 (2 min at 13 $km.h^{-1}$); R 1 min[a]
Jan—16	13.7—31	48 min at 12.4 $km.h^{-1}$[b]	4 (5 min at 13 $km.h^{-1}$); R 2 min[a]
Jun—16	12.7—40	Participation in road races	4 (5 min at 13 $km.h^{-1}$); R 2 min[a]

LT: Lactate Threshold (i.e., pace corresponding to a [La] value of ~2 $mmol.L^{-1}$); LTP: Lactate Turnpoint (i.e., pace corresponding to a [La] value of ~4 $mmol.L^{-1}$); R: recovery between repetitions, respectively.
[a] Treadmill based sessions, fixed speed (1% slope).
[b] 10 km road races used as training sessions and ran at a pace corresponding approximately to the lactate threshold.

Table 3 Samples of two representative training weeks in November 2014 and March 2016.

	November 2014	March 2016
Monday	20 min easy run (8.9 $km.h^{-1}$)	Rest
Tuesday	30 min easy run (8.6 $km.h^{-1}$)	4 × (5 min at 13 $km.h^{-1}$); R 2 min
Wednesday	Rest	Rest
Thursday	Rest	Rest
Friday	30 min steady run (10 $km.h^{-1}$)	50 min easy run (9 $km.h^{-1}$)
Saturday	Rest	8 × 1 min high-intensity (~15.5 $km.h^{-1}$); R 1 min
Sunday	20 min steady, stationary bike	80 min easy run (9.5 $km.h^{-1}$)

R: recovery between the repetitions.

a combined effect of diet and periodised training alternating easy, moderate and severe intensity sessions followed by recovery periods. These recovery periods and a lower training volume/intensity than in elite populations might have minimised the effect of exercise-induced plasma volume expansion and led to an increase and plateau in Hb and Hct [21]. We also observed increases in MCH and MCV showing that normal cell morphology was developing.

As well as allowing the athlete to better cope with moderate to heavy intensity training, higher levels of Fer also improved ID symptoms encountered in the athlete's daily life. For example, during the period investigated, she would report being less tired at the end of the week than before the intervention. The headaches experienced before and in the early stages of the study disappeared too.

Another improvement observed is the substantial decrease in lactate concentrations at 12 $km.h^{-1}$. [La] decreased between November 2014 and February 2016 from 5.9 to 2.5 $mmol.L^{-1}$ (a 57.6% decrease). As mentioned before, the athlete could train at heavy intensities and did long intervals consisting of 4—5 repetitions at 12—13 $km.h^{-1}$ for 5 min to improve lactate buffering. This resulted in the observed decrease in [La].

As can be seen on Fig. 3 (B and C) VO_{2max} slightly plateaued or decreased between March and October 2015. A similar pattern was seen in the running speed at VO_{2max}. This occurred at the time blood results revealed a deficiency in vitamin B_{12}. The athlete was complaining of regular episodes of fatigue. Following B_{12} injections, these fatigue episodes disappeared. One limitation of this study is that we did not keep a food diary and could not verify if the athlete's vitamin B_{12} intake was 2.4 $\mu g.d^{-1}$ (i.e., B_{12} recommended dietary allowance) [22]. We can therefore speculate that the athlete may have not consumed or absorbed adequate amounts of B_{12} which resulted in a deficiency.

Conclusion

In conclusion, our results show that in this athlete, iron supplementation, increase in dietary iron intake and structured training induced improvements in: (i) iron status, (ii) Hb and Hct, (iii) VO_{2max} and (iv) running performance. Based on the present study data and athlete's feedback, it seems that performance improvements were firstly induced by the correction of ID and secondly by the athlete's ability to train consistently and do steady and heavy intensity training. This case study is a real-world example for other non-elite female athletes. Sport Science and Sport Medicine approaches were used to successfully improve the endurance performance and ID symptoms encountered in the daily life of a club runner. Further, this study and results show that a coordinated approach between Exercise Scientists, Sport Medicine doctors and coaches is effective in the management of non-elite female's training efficiency and tolerance.

The data reported herein include several markers of iron status and will be useful to practitioners. The techniques and/or protocols employed in this study could be used when working with iron deficient athletes.

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Disclosure of interest

The authors declare that they have no competing interest.

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References

[1] Buratti P, Gammella E, Rybinska I, Cairo G, Recalcati S. Recent Advances in Iron Metabolism: Relevance for Health Exercise, and Performance. Med Sci Sports Exerc 2015;47: 1596—604.
[2] Beard J, Tobin B. Iron status and exercise. Am J Clin Nutr 2000;72:594S—7S.
[3] Bassett J, Howley T. Limiting factors for maximum oxygen uptake and determinants of endurance performance. Med Sci Sports Exerc 2000;32:70—84.
[4] DellaValle DM. Iron supplementation for female athletes: effects on iron status and performance outcomes. Curr Sports Med Rep 2013;12:234—9.
[5] Pate RR, Miller BJ, Davis JM, Slentz CA, Klingshirn LA. Iron status of female runners. Int J Sport Nutr Exerc Metab 1993;3:222—31.
[6] Alaunyte I, Stojceska V, Plunkett A. Iron and the female athlete: a review of dietary treatment methods for improving iron status and exercise performance. J Int Soc Sports Nutr 2015;12:38.
[7] Pasricha SR, Low M, Thompson J, Farrell A, De Regil LM. Iron supplementation benefits physical performance in women of reproductive age: a systematic review and meta-analysis. J Nutr 2014;144:906—14.
[8] Jones AM. A five year physiological case study of an Olympic runner. Br J Sports Med 1998;32:39—43.
[9] Ingham SA, Fudge B, Pringle J. Training distribution, physiological profile, and performance for a male international 1500-m runner. Int J Sports Physiol Perform 2012;7:193—205.
[10] Pedlar CR, Whyte GP, Burden R, Moore B, Horgan G, Pollock N. A case study of an iron-deficient female olympic 1500-m runner. Int J Sports Physiol Perform 2013;8:695—9.
[11] Winter EM, Jones AM, Richard Davison RC, Bromley PD, Mercer TH. Sport and Exercise Physiology Testing Guidelines: Volume I — Sport Testing: The British Association of Sport and Exercise Sciences Guide. 1st ed. BASES Sport and Exercise Science; 2007.
[12] Jones AM, Doust JH. A 1% treadmill grade most accurately reflects the energetic cost of outdoor running. J Sports Sci 2000;14:321—7.
[13] Burden RJ, Morton K, Richards T, Whyte GP, Pedlar CR. Is iron treatment beneficial in, iron-deficient but non-anaemic (IDNA) endurance athletes? A meta-analysis. Br J Sports Med 2014;0:1—10.
[14] Wachsmuth NB, Aignier T, Völzke C, Zapf J, Schmidt WF. Monitoring recovery from iron deficiency using total hemoglobin mass. Med Sci Sports Exerc 2015;47:419—27.
[15] LaManca J, Haymes E. Effects of iron repletion on VO_{2max}, endurance, and blood lactate in women. Med Sci Sports Exerc 1993;24:1386—92.
[16] Friedmann B, Weller E, Mairbaurl H, Bartsch P. Effects of iron repletion on blood volume and performance capacity in young athletes. Med Sci Sports Exerc 2001;33:741—6.
[17] Fogelholm M, Jaakkola L, Lampisjärvi T. Effects of iron supplementation in female athletes with low serum ferritin concentration. Int J Sports Med 1992;13:158—62.
[18] Buchheit M, Laursen PB. High-intensity interval training, solutions to the programming puzzle. Part 1: Cardiopulmonary emphasis. Sports Med 2013;43:313—38.
[19] Laursen PB, Jenkins DG. The scientific basis for high-intensity interval training: optimising training programmes and maximising performance in highly trained endurance athletes. Sports Med 2002;32:53—73.
[20] Banfi G, Lundby C, Robach P, Lippi G. Seasonal variations of haematological parameters in athletes. Eur J Appl Physiol 2012;111:9—16.
[21] Mairbäurl H. Red blood cells in sports: effects of exercise and training on oxygen supply by red blood cells. Front Physiol 2013;4:332.
[22] Lukaski HC. Vitamin and mineral status: effects on physical performance. Nutrition 2004;20:632—44.