The Effect of Iron Therapy on Selected Haematological Parameters and 8-km race time in Iron Deficient Bahraini Runners

By Anthony J. Brien*

ABSTRACT

Iron therapy improves exercise capacity in laboratory tested iron deficient athletes but there is a lack of documentation regarding beneficial transfer to actual competitive performance. In five iron deficient runners (serum ferritin <20 ng/ml), RBC, Hb, Hct, MCV, MCH, MCHC, RDW, PLT, SF, B_{12}, folate, submaximal HR and 8-km race time were measured during a 16 weeks training and competition period. Iron therapy consisted of 300 mg ferrous gluconate (34.8mg iron) taken three times daily (104.4mg iron) throughout the 16 weeks. The experimental design deliberately reflected the real life situation of iron deficiency detection followed by treatment prescription and the recording of the subjects' response to the treatment. Post treatment mean serum ferritin levels increased from 9.2ng/ml to 23.4 ng/ml (P<.05) and Hb increased from 13.1g/dl to 14.0g/dl, while submaximal HR and 8-km race time decreased from 162bpm to 153bpm and 20 min. 6 sec. to 27 min. 5 sec. (P<.01) respectively. The results indicate a direct relationship between an improvement in haematologic indices and an increase in physical work capacity following iron therapy.

Iron deficiency is the most common form of malnutrition worldwide[1] and is particularly prevalent in Bahrain.[2] Endurance runners are highly susceptible to a negative iron balance which causes iron stores depletion and can eventually lead to overt anaemia.[3,4] Controversy surrounds the precise aetiology(ies) of iron deficiency in these athletes but inadequate dietary intake,[5,6] reduced intestinal absorption,[3,7] gastrointestinal bleeding,[8] iron loss in urine,[4,9] iron loss in sweat,[10,11] and intravascular hemolysis due to foot impact forces,[12] have all been postulated as contributing factors.

Several studies have reported on the treatment of iron deficiency and its effects on physical work capacity in laboratory setting,[5,13] but no studies exist describing iron treatment effects on competitive performance in iron deficient male runners. The present study sought to determine:

- The significance of hypoferritinemia for competitive running performance.
- The relationship between serum ferritin and haemoglobin during an intensive endurance training period.
- A rationale for iron supplementation for iron deficient endurance runners.

METHODS

Subjects

The subjects selected (n=5) were from the national cross-country team (8) and the national modern pentathlon team (5) who were screened for iron deficiency by blood test. Iron deficiency was defined as a serum ferritin value at or below 30 ng/ml (Table 1). Three subjects from the cross country team and two from the modern pentathlon team who met these criteria agreed to participate in the study. These subjects represent the entire population of iron deficient elite endurance runners in Bahrain.

The subjects maintained a steady distance based running programme (97.6±38.8km) throughout the course of the study with the exception of subjects #3 and #4. Their schedule included two hill sessions per week, beginning at week 8 to specifically prepare for upcoming competitions. Data describing the subjects are presented in Table 2.

* Director of Research Programmes
Bahrain Sport Institute
P.O. Box 5747
Manama, Bahrain

TABLE 1
Serum Ferritin Value

Stages of iron deficiency	Serum Ferritin (nanogr. per ml.)	Haemoglobin (grams per dl.)	Bone marrow iron
normal iron storage	>60	>12.0W >14.0M	+4
prelatent	>30<60	>12.0W >14.0M	0-trace
latent	<30	>12.0W >14.0M	0
manifest anaemia	<10	<12.0W <14.0M	0

W = women; M = men. (From D. Clement, Runner's World, Dec. 1985)

TABLE 2
Description of subjects

Subject	Age (yrs)	Height (cm)	Weight (kg)	Activity (weekly)	Training (years)	Previous Best 8-km Before Study (Min. Sec.)
1	28	173.5	64	150 km run	13	25 04
2	18	179	61	80 km run	3	27 46
3	27	167	50.5	138 km run	10	26 58
4	25	188	77.5	60 km run 20 km swim 6 hr. fence	11	31 52
5	18	180	61.7	60 km run 20 km swim 6 hr. fence	2	30 47
Mean	23.2	177.5	62.9	97.6 run	7.8	28 29
SD	4.3	7.0	8.6	38.8	4.4	2 30

TABLE 3
Normal values for 12 blood parameters

Red Blood Cells (RBC)	5.4±0.9	($x10^{12}/_{l}$)
Hemoglobin (HB)	16.0±2.0	(g/dl)
Hematocrit (Hct)	47.0±5.0	(%)
Mean Cell Volume (MCV)	90±7	(fl)
Mean Cell Hemoglobin (MCH)	29.8±1.3	(pg)
Mean Cell Hemoglobin Concentration (MCHC)	33.1±1.0	(g/dl)
Red Cell Distribution Width (RDW)	11.7±1.1	
Platelets (PLT)	267±53	($x10^{9}/_{l}$)
White Cell Count (WBC)	6.5±1.8	($x10^{9}/_{l}$)
Serum Ferritin (SF)	60-230	(ng/ml)
B_{12}	200-750	(Pg/ml)
Folate	2.5-12.5	(Pg/ml)

Treatment

Subjects were instructed to take 300mg ferrous gluconate (34.8mg iron) three times a day (104.4mg elemental iron/day) for the course of the 16 weeks study.

Blood sampling

Blood was withdrawn from the antecubital vein and collected in EDTA for haematologic data and without anticoagulants for serum ferritin, vitamin B_{12} and folate determination.

Haematological analysis

The number of red blood cells (RBC), the concentration of haemoglobin (Hb), the haematocrit value (Hct), the mean corpuscular volume (MCV), the mean corpuscular haemoglobin (MCH), the mean corpuscular haemoglobin concentration (MCHC), the red cell distribution width (RDW), the platelets (PLT) and white blood cell counts (WBC) were determined by the Coulter Counter S plus 3 system. The concentration of serum ferritin (SF), vitamin B_{12} and folate were determined using a commercial radioimmunoassay reagent (Amersham, UK). Blood tests were undertaken at the start of the study and every four weeks thereafter to ascertain haematological status. Average values for these 12 blood parameters are shown in Table 3. All blood collections were analysed at the pathology laboratory of the Bahrain Defense Force Hospital.

Performance indices

Three 8-km races were run to assess competitive performance time. The first race was used as a

TABLE 4
Nutrition analyses (averaged over 3 days)

Subjects	Iron (mg)	Protein (gm)	Carbohydrates (gm)	Fat (kcal)	Calories
1	16.8	84.0	440	122	3194
2	12.6	65.5	276	118	2428
3	10.2	68.4	315	110	2523
4	10.4	71.0	284	125	2545
5	9.8	66.8	299	108	2435
Mean	11.9	71.1	322	116	2625
SD	22.3	6.7	60.1	6.6	288

baseline pre-treatment measure (week 0), while the second was run 8 weeks post-treatment, and the third 16 weeks post-treatment. The races were held at the same time of day on an undulating tarmacadam road and were open to all comers. Treadmill tests to assess submaximal heart rate were performed at week 0 pre-treatment, and at weeks 4, 8, 12 and 16 post-treatment. The treadmill test consisted of a 5 minute warm-up at a speed of 12km per hour followed by a 5 minute run at 16km per hour; with heart rate recorded at the end of the fifth minute.

Dietary intake

Nutrition information was collected from all five subjects. Dietary intake was recorded over a 3 day period and analysed to provide mean daily intakes of energy, iron, protein, carbohydrate and fat.

Statistics

Data analysis was performed by correlated t-test evaluation of the sample means. A probability (P) of less than 0.05 was considered adequate to reject the null hypothesis.

RESULTS

Environmental conditions

For the three 8-km races the temperature (22.4°C, 24.2°C and 23.8°C,) relative humidity (58%, 61% and 64%) and wind velocity (12.5, 10.6, and 13.4km/hour) were within a comparable range.

Nutrition analysis

All the subjects had dietary intakes above the recommended daily intake of 10mg iron.[14] Mean daily iron, protein, carbohydrate, fat and energy intakes are shown in Table 4.

Haematological analysis

Pre-treatment

One of the subjects demonstrated a latent iron deficiency (SF=19ng/ml), while four had severe iron deficiencies (SF<10ng/ml) two of which were hypochromic microcytic. All the subjects denied a blood disorder history and none had donated blood in the previous 12 months.

Post-treatment

No significant differences were detected in RBC, Hb, MCV, MCH, MCHC, RDW, PLT, WBC or B_{12}. Hct increased significantly (P<05) from week 0 (40.4%±3.9%) to week 8 (44.0%±4.0%)

TABLE 5
Hematological indices, mean SD of 5 subjects, before (week 0) and after (weeks 4, 8, 12, 16) iron therapy.

Week		0	4	8	12	16
RBC	($x10^{12}/_{l}$)	4.9±0.2	5.0±0.4	5.3±1.1	5.2±0.4	5.2±0.3
Hb	(g/dl)	13.1±1.7	13.6±1.1	14.0±1.1	14.1±0.9	14.2±0.8
Hct	(%)	40.4±3.9	42.2±3.5	44.0±4.0*	43.7±4.5 *	43.8±2.5*
MCV	(fl)	81.4±6.6	82.7±6.2	83.9±5.7	83.7±6.5	83.9±7.1
MCH	(pg)	27.2±2.7	27.0±2.2	26.6±2.2	27.3±2.5	27.7±1.7
MCHC	(g/dl)	33.4±0.4	32.9±0.3	31.7±1.3	32.7±0.5	33.0±0.6
RDW		13.3±1.7	13.8±1.1	14.1±2.1	13.8±1.9	14.1±2.0
PLT	($10^{9}/_{l}$)	264±46	254±37	241±29	234±31	231±35
WBC	($x10^{9}/_{l}$)	4.6±0.8	4.5±1.0	4.4±1.1	4.4±0.8	4.4±0.5
B_{12}	(Pg/ml)	5.8±2.9	7.4±2.2	8.4±1.5	8.2±0.7	8.4±2.1
Fol	(Pg/ml)	500±134	608±122**	704±43***	650±67**	586±127
SF	(ng/ml)	9.2±5.2	13.2±5.6	17.8±5.6	20.6±9.3	23.4±15.7

Levels of significance: * P<0.05, ** P<0.02, *** P<0.01

and remained elevated for the duration of the study. SF showed a significant increase ($P<.05$) only at week 16 (9.2±5.2ng/ml to 23.4±5.7ng/ml). Folate increased significantly ($P<.05$) from week 0 (500±134pg/ml) to week 4 (608±122pg/ml), reached a peak at week 8 ($P<.01$) before falling again at week 12 ($P<.05$) and showed an elevated but non-significant increase at week 16. Results of the blood analysis are presented in Table 5.

Performance indices

Iron therapy caused a mean reduction of 2 min. 1 sec. in the time to run 8-km race. Improvement was significant at 8 weeks ($P<.02$) and after 16 weeks ($P<.01$) when compared to baseline. Race times are shown in Table 6.

There was a decrease in submaximal heartrate (162-153bpm) but this was not statistically significant.

TABLE 6
8-km road race times (mins. and secs.)

Subject	Week 0	8	16	Changes
1	26.12	25.20	24.32	1.40
2	28.02	28.04*	26.51	1.11
3	27.48	27.08	26.24	1.24
4	32.05	30.17	28.52	3.13
5	31.24	30.17	28.45	2.39
Mean	29.06	28.11**	27.05***	2.01
SD	2.15	1.53	1.36	0.46

Levels of significance: ** $P<0.02$, *** $P<0.01$
* This subject ran a 10-km race the previous day.

DISCUSSION

The significant improvement in performance of our hypoferritinemic subjects, with the only intervention being iron supplementation, addresses several issues in the literature concerning iron deficiency and exercise.

The results of the study answer affirmatively Rowland et al [15] who queried if the mean increase of 74 sec. in treadmill endurance time observed in their study of non-anaemic iron deficient runners could be translated into decreased competition times. The improvement of 1 min. 40 sec. and 1 min. 11 sec. for subjects #1 and #2 disputes the contention of Magnusson et al [16] that non-anaemic hypoferritinemia does not represent true iron deficiency as these subjects exhibited 'normal' pre-treatment Hb's of 14.1g/dl and 15.6g/dl and reduced SF's of 19ng/ml and 10ng/ml respectively. The data supports the proposal of Finch et al [17] that Hb alone is not a sensitive indicator of iron status and lends credence to Parr [18] who considers any Hb below 14g/dl in females and 16g/dl in males to be suboptimal for maximum oxygen transport. Parr's hypothesis is further supported by studies of induced erythrocythemia [19,20] that demonstrated significant improvement in the competitive performance of non-anaemic trained athletes by increasing Hb approximately 1g/dl through autologous transfusion of RBCs.

The comparitively rapid recovery of endurance capacity in the present study after iron therapy challenges the generally accepted current view that severe iron deficiency results in a decrease in the iron containing enzymes necessary for the oxidative production of ATP in skeletal muscle.[21] A recent study by Celsing et al[22] corroborates this contention as their findings indicate severely depleted tissue iron stores (SF<5ng/ml) did not compromise the maximal activities of various enzymes in the human skeletal muscle. This latest evidence would appear

to vindicate the original assertion of Hahn[23] that iron enzymes are protected at all costs in the iron deficient organisms and suggests that oxygen delivery and not oxygen utilization is the major component in the impaired exercise capacity observed in iron deficiency anaemia.

Several studies on serum ferritin levels have shown it to accurately represent iron storage.[24,25] These studies have resulted in the estimation that 1ng/ml is equal to 8mg of storage iron. Runners may have false increases in SF values for a number of days following intensive racing or training,[9] but iron deficiency appears to be the only condition in which SF levels are reduced.[26] In subjects #3 and #4 SF levels decreased after week 8 (Table 7). There was no evidence of reduced haematopoeisis and 8-km times continued to improve. The SF decreases at this stage are assumed to be related to the inclusion of two intensive hill training sessions per week after week 8 in the schedule of these runners. As running intensity influences iron cost,[9,3,24] changes in training quality may alter the performance response to iron therapy. The 5% decrease in submaximal heartrate after iron therapy approximates that reported in similar moderate work capacity tests.[27,28]

The fluctuations in folate do not appear to have any clinical significance and no relation to physical performance can be found in the literature.

The mean iron intake of the subjects was 11.9mg. If we assume absorption to be 10%[29] the runners would absorb 1.9mg/day. Average iron excretion of 1mg/day coupled with sweat losses of 0.179mg/l[30] and footstrike induced intravascular hemolysis of up to 0.85ml/day of RBC,[12] provides unequivocable evidence for the negative iron balance and subsequent reduced iron stores present in our subjects.

TABLE 7
Individual SF and Hb values

Serum Ferritin (ng/ml)

Subject	Week 0	4	8	12	16	Change
1	19	23	27	38	52	33
2	10	16	20	21	23	13
3	5	9	17	15	13	8
4	5	9	15	11	6	1
5	7	9	10	18	23	16
Mean	9.2	13.2	17.8	20.6	23.4	14.2
SD	5.2	5.6	5.6	9.3	15.7	10.7

Hemoglobin (g/dl)

Subject	Week 0	4	8	12	16	Change
1	14.1	14.1	14.3	14.4	14.5	.4
2	15.6	15.6	15.8	15.6	15.5	.1
3	13.0	13.1	13.2	13.5	13.5	.5
4	10.5	12.6	14.1	14.1	14.1	3.6
5	12.6	12.7	12.7	12.9	13.1	.5
Mean	13.1	13.6	14.0	14.1	14.1	.9
SD	1.7	1.1	1.1	0.9	0.9	1.3

Subject compliance was attempted by having the coach question the runner daily as to his ingestion of the iron supplements. However, the high degree of diligence claimed by the subjects is necessarily hearsay and may have affected the results. Also, iron absorption is modified by taking the supplements with meal compositions promoting or inhibiting bioavailabity of iron, or on an empty stomach,[31] and no attempt was made to monitor these factors. Furthermore, it was assumed that the runners did not digress from the planned training programme and maintained their regular activity and dietary schedule. The issue of iron supplementation is further complicated by absorption disturbances as reported by Ehn et al[3] who found that absorption in iron deficient runners was only 16.4% compared to 30% for iron deficient controls. Similar differences comparing absorption rates of athletic

TABLE 8

Combination of foods that work together to help your body absorb the most iron

Consume a vitamin C-rich food with iron rich vegetables or meats and you will dramatically boost the iron your body absorbs.

A) One of these (in cups or other, total iron in mg)	B) Has absorbable iron (mg)	C) Combined with one of these (in cups or other)	D) Raises absorbable iron to (mg)
Dried lima beans cooked (½,2.1)	.06	Red pepper, raw (2/5)	.17
Beef hamburger (one, 3" x 5/8", 2.6)	.29	Cabbage, cooked (1½)	.36
Poached egg (medium, 1.0)	.03	Grapefruit juice (1)	.08
Dark pumpernickel bread (1 slice, 0.6)	.02	Orange juice, fresh (3/5)	.05
Dried uncooked apricots (½, 3.6)	.11	Strawberries, whole fresh (1)	.29
Dried cooked lentils (½, 2.2)	.07	Cauliflower, raw (1)	.18
Whole figs (1,1.0)	.03	Cantaloupe (1½)	.08
Lamb chop (medium, 1.0)	.11	Broccoli, cooked (3/5)	.14
Shredded wheat biscuits (2, 1.1)	.03	Papaya (1)	.09
Soybeans, cooked (½, 2.4)	.07	Tomatoes, raw (2,3" dia.)	.19

(From: J. Barone and R. Barnett, American Health, March '87).

and sedentary populations have been reported.[7] Increased intestinal transit time has been proposed as a factor for absorption discrepancy,[32] but whether this holds true for athletes is unknown.

Iron therapy of 900mg ferrous gluconate/day improved Hb concentration and 8km race time of trained runners over a 16 weeks training and competition period. However, only subject #1 who presented with a non-anaemic latent iron deficiency acquired 'normal' iron store status (SF ≃45ng/ml) as defined by Telford et al.[33] Therefore the other subjects can anticipate further performance improvements as their iron stores are repleted.

This highlights the long term process of correcting iron deficiency anaemia in concurrently exercising populations and raises the question as to whether an increased iron dosage would expedite recovery. In an equivalent iron depleted group of female runners, Clement et al[34] prescribed 209mg/day of elemental iron or twice the dosage of the present study. Comparable increase in iron status was attained in SF (7.6±4.7ng/ml to 23.1±13.3ng/ml) and Hb (11.7±1.6g/dl to 13.5±0.7g/dl). These results do not reflect a more advantageous effect from the higher dosage which is approaching the maximal safety tolerance level of 250mg[35]

Considerable caution must be taken in advocating high dosages as unwarranted iron intake can counteract the absorption of zinc due to their similar physiochemical characteristics.[36] Also, excessive iron can leave the athlete more open to infections as the toxicity of pathogenic organisms is linked with the capability by which its siderophore systems provide them with iron in serum.[37]

Based on the above considerations, the safer but slower way for the athlete to recover from a detected iron deficiency is through increasing the iron content of the diet by including foodstuffs that are rich in haem-iron and taken with vitamin C to facilitate absorption (Table 8). Accompanying the dietary changes should be a reduction in the quality and quantity of training until the recovery is complete. As this process can take many months it is imperative that the enriched iron diet is sustained as training intensity increases, in order to prevent a recurrence. Athletes susceptible to iron deficiency should be blood tested thrice yearly. Serum ferritin level is the preferred test as it is a more discriminating indicator of the body's iron stores than either haemoglobin or haematocrit.

CONCLUSION

Five iron deficient elite Bahraini runners were monitored over a 16 week period of training and competition while undergoing iron therapy. The results indicate that treatment improved serum ferritin levels and 8-km race time and suggest that oxygen delivery and not oxygen utilization is the dominant factor in reduced exercise capacity observed in iron deficient endurance runners.

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