Iron supplementation and iron status in exercising young women

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The effect of moderate aerobic exercise training and iron supplementation on iron status was studied in college-age women. Thirteen sedentary women, randomly assigned to a placebo group or an iron treatment group (50 mg iron/day as $FeSO_4$), exercised at least three days per week at 70–80% of maximal heart rate for 12 weeks. Increases in maximal oxygen consumption in both groups indicated improved cardiovascular fitness. Venous blood samples were obtained for hemoglobin, hematocrit, serum iron, total iron binding capacity, transferrin saturation, and ferritin determinations at weeks 0, 4, 8, and 12. Analysis of covariance using initial baseline values as the covariate showed that ferritin levels between groups were significantly different ($P < 0.01$), suggesting compromised iron stores in association with moderate exercise. Iron supplementation was beneficial in maintaining or improving the iron stores of moderately exercising women.

Keywords: iron; iron supplementation; exercise

Introduction

Iron deficiency is considered the most common nutritional deficiency in the United States. The latest assessment of the U.S. population demonstrates that iron deficiency is most prevalent among children, adolescents, and women between the ages of 18 and 44.[1] During the childbearing years, women are at greater risk of becoming iron deficient due to menstruation, inadequate dietary iron intake, and pregnancy. Evidence suggests that exercise may be another factor affecting iron status in women. The incidence of iron deficiency among athletes has been reported to be higher than among less active individuals[2-4] while several studies demonstrate the detrimental effect of strenuous physical activity on iron stores.[5-7] However, most of these studies did not evaluate the role of dietary iron independently of exercise on iron status. Iron plays a role in exercise performance in two important ways. First, as a component of hemoglobin in the red blood cell, iron aids in the delivery of oxygen to the working muscles. Second, as a component of myoglobin in muscle, and cytochromes in the mitochondria, it is involved in oxygen storage and energy production via oxidative metabolism. Iron deficiency, whether or not it manifests itself as anemia, may decrease physical work capacity, and therefore, can be detrimental to performance.[8-13]

The iron status of well-trained athletes and individuals involved in strenuous physical activity such as marathon running has been well-studied. Frequently mentioned causes of iron deficiency in these individuals include iron loss due to hemolysis,[14,15] sweating,[16] gastrointestinal bleeding,[17,18] and hematuria.[19] With more health professionals advocating exercise for people of all ages and more women becoming physically active on a regular basis, the question arises as to what effect milder forms of exercise have upon the iron status of previously sedentary women. Several studies indicate that moderate aerobic exercise is detrimental to iron stores.[20,21] The purpose of the present study is to further examine the effect of improved fitness, as assessed by maximal oxygen uptake ($\dot{V}O_2$ max), resulting from moderate aerobic exercise intervention and the benefit of iron supplementation on the iron status of women.

Methods and materials

Thirteen Caucasian women, aged 18–25 years, were recruited from the campus of Purdue University, West

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Lafayette, Indiana, to participate in a 12-week individualized aerobic exercise program. Information concerning medical history, oral contraceptive use, regularity of menstrual cycles, and physical activity was obtained via a pre-experimental health questionnaire. Only sedentary subjects were selected who did not regularly participate in an exercise program. Capillary blood samples were obtained to screen for anemia and to ensure that hematocrit values were within normal limits. All procedures in the investigation were approved by the institutional Human Research Review Board and subjects gave their informed consent.

Individuals were randomly assigned to an iron treatment ($n = 7$) or placebo group ($n = 6$). Iron supplements contained 50 mg iron in the form of controlled release ferrous sulphate, Feosol® Capsule (SmithKline Consumer Products, Philadelphia, PA). The placebo consisted of lactose granules (SmithKline Consumer Products, Philadelphia, PA). Capsules were taken by both groups once per day after the evening meal.

Subjects participated in one or more of the following forms of exercise: jogging, bicycling, swimming, or aerobic dance sessions held at a campus recreational facility. Exercise was carried out in accordance with American College of Sports Medicine recommendations (i.e., frequency of 3–5 days/week, duration of 15–60 min per session, and an intensity of 60–90% of maximum heart rate reserve or 50–85% of $\dot{V}O_2$ max).[22] Compliance with the exercise prescription was assessed by having subjects document the type, intensity, duration and frequency of activity in exercise journals and submit them to the investigators on a weekly basis.

Fasting venous blood samples were collected at baseline (twice within one week) and at 4-week intervals during the exercise program. For each blood draw, a total of 15 ml of blood was collected into EDTA-containing lavender top and iron-free navy blue top Vacutainer tubes (Becton-Dickinson, Rutherford, NY). To control for diurnal variation, blood was sampled between 7:30 a.m. and 10:00 a.m. Whole blood was used to determine hemoglobin[23] and hematocrit[24] while serum recovered after centrifugation was frozen in plasic tubes and later assayed for iron,[24] total iron binding capacity (TIBC),[24] and serum ferritin (ICN Micromedics Systems, Inc., Horsham, PA) by radioimmunoassay. Percent transferrin saturation (TS) was calculated as the ratio of Fe/TIBC × 100. Serum albumin was measured to monitor any changes in blood volume.

Detailed written and verbal instructions on keeping a daily food record were given using food models. Subjects completed 3-day dietary records prior to the start of the study and again during the twelfth week of exercise. Computrition nutritional analysis software package (Chatsworth, CA) was used to determine total iron, total protein, ascorbic acid, and total calories.

Age, height, body weight, and percent body fat data were collected on each subject at baseline and again at week 12. Body density was determined by hydrostatic weighing with residual lung volume measured using an O_2 dilution technique.[25] Body density was converted to percent body fat using the Siri equation.[26]

$\dot{V}O_2$ max consumption tests[27] to assess cardiovascular fitness were conducted at baseline and after 12 weeks of exercise. To control for the increase in energy expenditure that occurs post-ovulation, oxygen consumption tests were performed between days 3 and 11 of the menstrual cycle.[28]

An analysis of covariance with baseline values as the covariate was used on hematologic parameters to determine differences between iron treatment and placebo at week 12. A multivariate analysis of covariance with baseline values as the covariate was used to determine significant differences between groups on iron status parameters across time. A two sample t test determined dietary intake differences between groups while a paired t test determined differences in pre- and post-dietary intake, percent body fat measures, $\dot{V}O_2$ max measures, and percent change in ferritin levels within groups. In all analyses, results were considered significant if $P < 0.05$.

Results

Table 1 represents age, height, weight, and percent body fat data for subjects receiving iron supplements ($n = 7$) and placebo ($n = 6$). Both groups demonstrated significant decreases in body fat indicating subject compliance with the exercise regimen. Subjects also lost weight but the difference did not reach statistical significance.

Mean $\dot{V}O_2$ max levels increased for both placebo and iron supplement groups (*Table 2*). The iron treat-

Table 1 Descriptive characteristics of subjects in iron treatment and placebo groups

	Iron	Placebo
Age (yr)	20.6 ± 2.4	21.2 ± 2.5
Height (cm)	163.6 ± 6.4	164.3 ± 3.3
Weight (kg)		
pre	59.8 ± 3.7	62.8 ± 7.3
post	58.9 ± 5.5	62.0 ± 8.0
Body fat (%)		
pre	20.8 ± 2.4	20.7 ± 6.1
post	18.9 ± 1.7[a]	19.0 ± 5.0[b]

Note: Descriptive characteristics denotes $\bar{X} \pm SD$.
[a,b] denotes a significant decrease ($P < 0.05$) from pre to post.

Table 2 Mean $\dot{V}O_2$ max values in placebo and iron treatment groups

	Baseline	Week 12	Mean % improvement from baseline to week 12
Placebo	27.8 ± 1.0	29.3 ± 1.7	5.4
Iron	33.8 ± 2.0	38.0 ± 2.0	13.5[a]

[a] $P < 0.05$.

Table 3 Iron status parameters and albumin in iron treatment and placebo groups at baseline, week 4, week 8, and week 12

	Time			
	Baseline	Week 4	Week 8	Week 12
Hemoglobin (g/dl)				
Placebo	14.0 ± 0.6	13.1 ± 0.6[a]	13.4 ± 0.6[a]	13.8 ± 0.6
Iron	13.8 ± 0.2	14.0 ± 0.3[a]	14.1 ± 0.4[a]	13.9 ± 0.3
Hematocrit (%)				
Placebo	41.6 ± 1.5	41.7 ± 1.8	41.6 ± 2.1	42.4 ± 1.6
Iron	41.7 ± 0.8	42.6 ± 0.6	45.1 ± 0.9	43.2 ± 0.8
Serum Fe (μg/dl)				
Placebo	105 ± 16	104 ± 16	129 ± 12	112 ± 15
Iron	113 ± 12	142 ± 14	146 ± 24	126 ± 19
TIBC (μg±dl)				
Placebo	312 ± 28	316 ± 9	322 ± 54	355 ± 48
Iron	394 ± 41	447 ± 63	423 ± 56	412 ± 56
Transferrin Sat. (%)				
Placebo	30 ± 3	33 ± 5	41 ± 4	34 ± 6
Iron	32 ± 6	33 ± 4	38 ± 8	36 ± 5
Serum Ferritin (μg/dl)				
Placebo	26 ± 5	21 ± 3	20 ± 4	17 ± 6[b]
Iron	29 ± 8	35 ± 8	36 ± 8	42 ± 9[b]
Albumin (g/dl)				
Placebo	4.0 ± 0.2	4.0 ± 0.3	4.1 ± 0.2	4.0 ± 0.3
Iron	4.0 ± 0.1	4.1 ± 0.1	4.2 ± 0.1	4.1 ± 0.1

Note: Iron status parameters reflect $\bar{X}$ ± SEM.
[a] Denotes significant difference between groups at the same time as assessed by MANOVA.
[b] Denotes significant difference between groups from baseline to week 12 as assessed by ANCOVA.

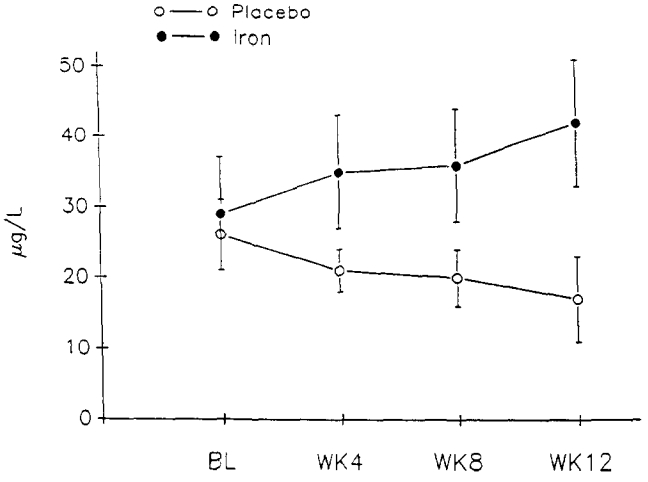

Figure 1 Changes in mean (± SEM) serum ferritin in iron treatment and placebo groups during twelve weeks of exercise.

ment group demonstrated a significant mean improvement of 13.5% ($P = 0.03$) in $\dot{V}O_2$ max from baseline to week 12 compared to a nonsignificant increase of 5.4% ($P = 0.36$) in the placebo group. The change in $\dot{V}O_2$ max was not significantly different between groups. Improvements in $\dot{V}O_2$ max of 12–20% have been reported in exercise programs varying in length from 4 weeks to 8 months.[29] Examination of exercise journals revealed that 91.6% of the subjects in the iron treatment group complied with the exercise protocol of 3 or more sessions per week. Compliance in the placebo group averaged 85.7%. Women in the placebo group tended to have shorter exercise sessions than those in the iron treatment group. These differences most likely account for the lower percent improvement in the placebo group.

Table 4 Analyses of baseline and week 12 dietary records

	Baseline	Week 12
Total iron (mg)		
Placebo	7.9 ± 2.3 (5.6–10.6)	8.9 ± 4.0 (4.1–13.2)
Iron	8.8 ± 3.6 (4.2–14.1)	65.5 ± 7.4[a] (59.3–77.7)
Ascorbic acid (mg)		
Placebo	61 ± 34 (18–88)	68 ± 49 (24–131)
Iron	106 ± 86 (25–227)	121 ± 57 (45–197)
Protein (g)		
Placebo	41 ± 8 (30–45)	53 ± 21[b] (35–76)
Iron	53 ± 15 (31–68)	66 ± 21[b] (41–90)
Energy (kcal)		
Placebo	1123 ± 171 (894–1283)	1680 ± 838[b] (880–2445)
Iron	1427 ± 346 (935–1726)	1861 ± 721[b] (1103–2709)

Note: X ± S.D. Ranges are given in parentheses.
[a] Includes 50 mg supplement; significantly different from placebo group ($P < 0.05$).
[b] Significant increase ($P < 0.05$) from baseline to week 12 for all subjects.

No significant differences were observed in hematocrit, serum iron, TIBC, TS, and albumin levels between groups at baseline, weeks 4, 8, and 12 (*Table*

3). Hemoglobin concentration differed significantly between groups at weeks 4 and 8. Mean hemoglobin level in the placebo group dropped from baseline to week 4 and then rose gradually at weeks 8 and 12, while mean hemoglobin value in the iron treatment group remained fairly constant.

Baseline ferritin values in both groups were within the range reported for women having normal iron status.[30,32] Increased ferritin levels in the iron treatment group contrasted significantly with the decrease observed in the placebo group at week 12 (*Figure 1*).

No significant differences in ascorbic acid, protein, or energy intake between groups at baseline and week 12 were observed (*Table 4*). When supplemental iron was not included in the analysis, there was no difference in total iron consumption between groups. Both groups demonstrated significant increases in protein and energy intake from baseline to week 12.

Discussion

Bothwell et al.[30] described the three stages of iron deficiency anemia. Stage one, called iron depletion, is characterized by decreased iron stores mainly in the bone marrow, liver, and spleen. This occurs when iron loss exceeds iron absorption over a period of time. Serum ferritin is the most useful test for detecting iron depletion and a high correlation between serum ferritin levels and iron stores has been demonstrated.[31-33] Iron depletion is characterized by serum ferritin concentrations of < 12 μg/l.

After iron stores are depleted, hemoglobin synthesis depends upon iron absorbed from the diet. However, if available dietary iron is inadequate, iron supply to the marrow for red blood cell production is reduced. During this stage, the synthesis of transferrin is increased resulting in a decreased transferrin saturation. A TS of $< 16\%$ and a serum ferritin of < 12 μg/l is indicative of stage two called iron deficient erythropoiesis. In the final stage, iron deficiency anemia, there is a decrease in total body iron and subsequently a decrease in circulating hemoglobin levels. This phase is characterized by serum ferritin < 12 μg/l, TS $< 16\%$, and hemoglobin < 12g/dl.

After 12 weeks of exercise, subjects in the placebo group experienced a decrease in iron stores as indicated by a $30.0 \pm 16.8\%$ decline in mean serum ferritin values. The incidence of iron depletion went from 1 of 6 subjects at baseline to 3 of 6 subjects at week 12. Blum et al.[21] reported a similar decline in serum ferritin when women participated in a 35-minute aerobic class for 13 weeks; however, the incidence of iron depletion remained unchanged. Iron depletion was present in 2 of 7 subjects in the treatment group prior to taking iron supplements and was eliminated by the twelfth week.

The average mean decrease in serum ferritin of 9 μg/L in the placebo group represents a decrease in storage iron of 70–90 mg. Blum et al.[21] proposed that iron stores decrease in response to moderate exercise in order to meet the increased need for iron-containing compounds. It is possible that the cytochromal activity and/or mass increased with exercise,[34] but this would not likely account for the entire decrease in storage iron. Furthermore, the increase in lean body mass calculated from the body composition and weight measurements was 0.4 kg for both the placebo group and the iron supplemented group in this study. Therefore, an overall increase in lean body mass is not a plausible explanation for the observed decrease in plasma ferritin with exercise. Nor can an increase in total blood volume explain the ferritin decrease since serum albumin concentration did not change.

The decline in serum ferritin could result from a decrease in iron absorption or balance due to exercise and/or bioavailability of dietary iron. However, these parameters were not measured in this study. A decrease in iron balance would indicate a decrease in iron absorption with exercise and/or an increased loss of iron due to gastrointestinal bleeding or sweat losses. It is unlikely that the moderate exercise prescribed in this study would produce such results. Even profuse sweating would only result in μg quantities of losses.[16] Hallberg and Magnusson[35,36] proposed that in elite runners, a shift in red cell catabolism from the reticuloendothelial system to hepatocytes occurs which could result in a decrease in serum ferritin. Whether mild exercise could lead to sufficient red cell hemolysis to precipitate such a shift requires investigation. Although anemia did not manifest itself after 12 weeks, it is not known what effect a moderate exercise program of longer duration would have had upon these women. In contrast to the placebo group, mean serum ferritin increased from baseline to week 12 in the iron treatment group indicating that iron supply was adequate and increased needs due to exercise were met.

Tests performed to detect iron deficiency erythropoiesis including serum iron, TIBC, and TS remained within normal limits throughout the study. This was expected given the moderate intensity and duration of the exercise. Physical activity most commonly associated with the incidence of iron deficiency is of a greater intensity, performed more frequently and continued over longer periods of time.[5-7,37,38]

Hemoglobin and hematocrit are generally the last parameters to be affected by a chronic decline in iron status. Although subjects in the placebo group demonstrated a decline in hemoglobin concentration from baseline to week 4, values gradually increased again at weeks 8 and 12. Since albumin levels remained unchanged, the decrease in hemoglobin was not due to a hemo-dilution effect. Hemoglobin concentration remained well above 12.0 g/dl and iron deficiency anemia did not occur in any of the women at any time during the study. No significant difference in hematocrit values between groups was detected.

Within the limitations associated with the use of 3-day diet records, no striking differences in diet were observed between the two groups, except for total iron intake due to supplementation. Energy and protein intake increased from baseline to 12 weeks in both groups which could reflect an increase in energy de-

mand with exercise. The decrease in body weight despite an increase in calorie intake supports an increase in energy demand. Total iron intake for all subjects at baseline averaged only 8.4 mg or 56% of the Recommended Dietary Allowances (R.D.A.). Average food iron intake increased to 12.2 mg at week 12 but remained below the recommended level of 15 mg.[39] A dietary intake of 10–12 mg has been reported to be typical for American women aged 12–50.[40] Intake of ascorbic acid, a known enhancer of iron absorption, was quite variable although mean values were at or well above the R.D.A.

In agreement with the findings of Blum et al.,[22] a 12-week moderate exercise program decreased iron stores in previously untrained women. In contrast to the findings of Blum et al.,[22] however, the results of the present study suggest that exercise can increase the incidence of iron depletion particularly in women with low intakes of bioavailable iron. These results give merit to future research directed toward quantifying the relationship between exercise intensity and duration and iron depletion and the protective effect of adequate dietary iron using larger numbers of women. If this effect of moderate exercise on iron stores is confirmed in additional experiments, then women who embark on a moderate exercise program will need to be made aware of the importance of obtaining adequate bioavailable iron.

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