

Possible effect of decreased insulin resistance on ferritin levels after Nordic Walking training

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Dear editor

We read with interest the article by Kortas et al¹ entitled “Effect of Nordic Walking training on iron metabolism in elderly women.” In their study, the authors investigated the effect of Nordic Walking (NW) training on serum ferritin, C-reactive protein (CRP), hepcidin, hemojuvelin, and vitamin D, which have been stated as components of the inflammatory system. In this study, while NW training significantly reduced iron stores and increased hemojuvelin and tended to reduce CRP levels, it did not affect hepcidin levels. The authors also observed a significant weight loss after training. The authors concluded NW training has pro-healthy effects manifested by decreased inflammation and a drop in iron stores.

It is well known that obesity is associated with insulin resistance and physical exercise and weight loss reduce insulin resistance. Several studies indicate a positive correlation between iron levels and insulin resistance.^{2,3} There is also a relationship between insulin resistance and CRP levels.⁴ The possibility of decreased insulin resistance with weight loss after training can also contribute to the decline in CRP and ferritin levels in this study. The authors mentioned that they did not observe the anticipated increase in hepcidin levels after exercise. As the authors stated, hepcidin levels increase in response to both inflammation and exercise. Because the mean body mass index of the study population is in the overweight range, these subjects might have insulin resistance and associated inflammation. Thus, the anticipated increase in hepcidin levels with exercise might have been attenuated via decreased inflammation in this study. This two-way interaction may account for the unchanged hepcidin levels. The authors did not report insulin levels in this study and did not discuss the probable effect of insulin resistance on the relationship between ferritin and exercise. We suggest that consideration of these factors may facilitate interpretation of the findings of this study. We also suggest that insulin resistance would better be assessed in future studies in this field.

Disclosure

The authors report no conflict of interest in this communication.

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Dear editor

We are really pleased to hear that you are interested in our article and we believe that a very important issue has been raised. It is suggested that the effects of lack of exercise on blood hepcidin may be due to reduced inflammation and increased insulin sensitivity. In our opinion, there are studies which do and do not support this suggestion. There is evidence for a positive correlation between body iron stores and insulin resistance. However, there are two important questions to be answered: 1) Can accumulated iron be responsible for induction of insulin resistance? 2) Can impairment of insulin signaling lead to iron accumulation? There is some evidence that the answers to these questions are positive. For example, reduction of body iron stores by phlebotomy leads to an increased insulin sensitivity.¹ Our observation is that a decrease in body iron stores after the training should be considered a positive change and we agree that it would be interesting to associate these data with parameters of glucose metabolism. In addition, elevated hepcidin concentrations and the association between diabetes and obesity with ferritin and CRP levels were observed.² Which supports the claim. Conversely, there is evidence that insulin can depress

hepcidin biosynthesis.³ And insulin resistance is associated with low blood hepcidin levels in diabetics.⁴ This might explain why there is iron accumulation in type 2 diabetic patients. The reason for these results is difficult to speculate. Anti-inflammatory effects of exercise are well documented.⁵ In fact, we observed some decrease in CRP concentration after 32 weeks of training, but it is too early to speculate if this was responsible for the observed changes in iron metabolism. We checked the correlation between weight and body mass index versus CRP, hepcidin, hemojuvelin, and ferritin. We found out that only correlation between the level of ferritin and weight was significant (at baseline: $r=0.46$, $P=0.01$; after training: $r=0.47$; $P=0.01$). It is important to note that there were training-induced increases in hepcidin in 16 of our subjects, whereas in the remaining 21 a decrease was observed. The reason for this differing response to exercise is not clear to us. Thus, we should consider that other factors may be involved. In summary, we agree that the effects of exercise on iron metabolism can be mediated by changes in insulin sensitivity and some modification of inflammation. We would like to thank Ilhan et al for their valuable suggestions, which will all be addressed in our forthcoming project.

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Disclosure

The authors report no conflict of interest in this communication.

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