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Letter to the Editor

Re: Bone: an acute buffer of plasma sodium during exhaustive exercise?

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Dear Editor(s)

We write to you with regards to the recently published original manuscript, *"Bone: An Acute Buffer of Plasma Sodium During Exhaustive Exercise?"* [1]. The authors, Hew-Butler, Stuempfle and Hoffman have presented an interesting hypothesis which contradicts current thinking on explanations for low bone density frequently observed in competitive endurance athletes. At present, the current theory for bone loss in endurance athletes is based on an uncoupling of bone turnover due to an energy deficit, whereby there is a reduction in collagen synthesis which coincides with reductions in circulating oestradiol, triiodothyronine (T3) and insulin-like growth factor (IGF) 1 [2, 3]. Energy conservation is thought to be the primary reason for hypo-oestrogenism and reduced bone density in athletes who are either intentionally or unintentionally energy deficient, and there is strong evidence to support this. This evidence has accumulated over the last 3 decades, from the initial observations of low bone density in amenorrheic female runners, to observations also in male endurance runners [4-5]. The research progressed to uncover associations of low body weight, body fat, high volume training, energy deficient diets, with low bone density, and to well-controlled studies where energy deficit has been experimentally induced and a reduction in bone formation markers reported [2, 3].

In contrast to the above, Hew-Butler et al. [1] propose that bone loss in endurance athletes may arise from the dissolution of bone mineral during periods of sodium loss, whereby sodium is extracted from bone to restore equilibrium in plasma sodium levels. The authors state, *"Over time, we speculate that a decrease in bone sodium stores (cumulative sweat sodium losses) may potentially manifest as decreased lumbar spine bone mineral density as a transient homeostatic response to protect [Na +] p levels during chronic training and competition [1 – 5]"* [1]. Their hypothesis is based upon observations of acute changes in dual energy X-ray absorptiometry (DXA) - measured bone mineral content (BMC), in a group of 6 ultra endurance athletes participating in a 161km mountain footrace, which correlated with changes in plasma sodium pre and post race. DXA is not a tool that is used for the assessment of changes in bone over a short time period, given that common thought is that at least one bone modelling cycle is required to enable a detectable change in bone mass in response to a given stimuli [6]. One normal bone remodelling cycle takes approximately 3-4 months. Machine precision error should also be considered more fully. The reported %CV in this study was based on just one subject, whereas, a df 30 is recommended. Determining true BMC change in this study is unfortunately not possible, and is particularly problematic at the spine due to interference from the rib cage and sternum (when derived from a total body scan). We also note discrepancies in loss/gain between skeletal sites, for example, gains in BMC at the left and right rib. This may reflect difficulties with reproducibility of regional measurements from a total body scan. Therefore the proposal that DXA may be used for the assessment of acute bone mineral change is at present difficult to ascertain.

To evaluate fully bone as an acute buffer of plasma sodium, assessment of both calcium and sodium gains of both losses from within BMC would be needed. In addition, changes in body mass and sodium loss in sweat would be essential to delineate the concurrent relationship between gains in sodium from exogenous sources (for example fluid and dietary intake), plasma sodium concentration and putative bone sodium turnover during exhaustive exercise. We recommend that future research is directed by a precise measurement of fluid and

sodium balance, and utilises bone biochemistry as a measurement of dynamic, short term changes in skeletal status. The study by Noakes at al. [7] referenced within the paper, demonstrates the large degree of inter-participant variability of sweat loss and the regulation of plasma sodium, thus this must be quantified in a study of this type. In short term, experimental studies of bone metabolism, endurance cycling induces a short, rapid increase in bone resorption [8]. Indeed, should bone act as a buffer for plasma sodium, it would be expected that the mechanism would be through increased bone resorption. Therefore, the proposed theory of bone as an acute buffer for plasma sodium is certainly plausible, but to prove this is a scientific challenge.

References

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