HOW INHERENT AERBOIC CAPACITY INFLUENCES THE BONE PROPERTIES OF THE SHOULDER JOINT IN A DIET INDUCED OBESITY RAT MODEL

Abel Mebrahtu[1,2], Darsh Tripathi[2], Mauricio Delgado[2], Ruth-Anne Seerattan[2], Lauren G. Koch[3], David A. Hart[2], Raylene A. Reimer[2], Walter Herzog[1,2]
[1]Biomedical Engineering, University of Calgary, Calgary, Alberta, Canada
[2]Human Performance Laboratory, University of Calgary, Calgary, Alberta, Canada
[3]Department of Physiology and Pharmacology, The University of Toledo, Toledo, Ohio, USA
Email: abel.mebrahtu@ucalgary.ca

Summary

Subchondral bone degeneration is a key hallmark of cartilage deterioration, associated with metabolic syndrome and obesity. While aerobic capacity has been shown to protect the cardiovascular system, its ability to mitigate musculoskeletal degeneration from diet-induced obesity has not been studied. This study aimed to assess if high-capacity runner (HCR) rats, in contrast to low-capacity runner (LCR) rats, are protected from bone degeneration in an obesogenic model. N:NIH rats selectively bred for HCR and LCR were randomly assigned to a chow control and high-fat and high-sucrose (HFHS) diet group. The diet intervention lasted for 12 weeks. Rats were sacrificed, and bones of the shoulder joint were analyzed using micro-computed tomography (micro-CT). found that HCR rats had significantly different bone structure and that their bones were better protected against structural degeneration in the presence of an HFHS diet than were the bones of the LCR rats.

Introduction

Obesity and metabolic syndrome are crucial mediators in the development of a subtype of osteoarthritis (OA), called metabolic OA [1]. It has been shown that in the early stages of metabolic OA, the subchondral bone experiences bone remodelling due to the negative effects associated with systemic inflammation [1]. The subchondral bone plays a critical role in the biomechanical interface of the articular cartilage with bone because it has been reported that with OA, the stiffness and elasticity of the subchondral bone decreases, resulting in alterations of the stress distribution of the joint [2]. Furthermore, it has been shown that Type 1 muscle fibres (in contrast to Type 2 fibres) that have a high oxidative capacity are protected against fibrosis and fat infiltration in the presence of a HFHS diet [3], and that aerobic exercise is also protective of general musculoskeletal degeneration in the presence of a HFHS diet [4]. Combined, these results led to the idea that a high inherent aerobic capacity may protect bones and joints in the presence of obesity. This study was aimed at assessing if rats with a high inherent aerobic capacity achieved through selective breeding, are protected from bone degeneration in the presence of an HFHS diet compared to counterparts with a low inherent aerobic capacity.

Methods

Thirty-six male N:NIH rats selectively bred for high aerobic capacity (HCR; n=18) and low aerobic capacity (LCR; n=18) were studied. Rats were randomly placed into two dietary intervention groups: chow (n=10 HCR, n=10 LCR) and a HFHS diet (n=8 HCR, n=8 LCR). The diet intervention lasted for 12 weeks, following which the rats were sacrificed and the shoulder joints harvested. The shoulder joints underwent micro-CT imaging and analysis for bone mineral density (BMD) and trabecular thickness (Tb.Th).

Results

There were significant differences in BMD and Tb.Th ($p<0.05$) between the HCR and LCR rats for the same diet groups (Fig 1). There were also significant differences in BMD and Tb.Th between the HFHS and the chow diet for the LCR rats, but not for the HCR rats where no changes in bone parameters were found as a function of diet (Fig 1).

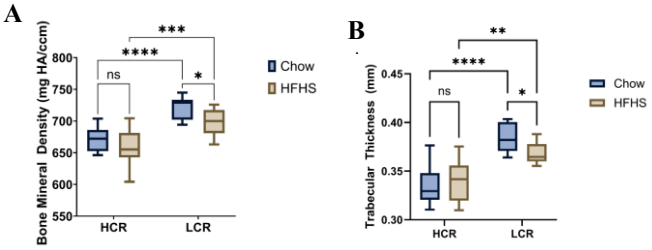

Figure 1: (A) Comparison of bone mineral density (BMD) between HCR and LCR rats and between the chow and HFHS diet. (B) Comparison of trabecular thickness (Tb.Th) between HCR and LCR rats and between the chow and HFHS diet.

Discussion and Conclusions

We conclude from the results of this study that bone structure and function are affected by selective breeding for HCR and LCR. However, bone structure and function is not overtly affected by the obesity-inducing HFHS diet in HCR animals, suggesting that the observed changes in bone structure observed in the LCR animals with the HFHS diet, are successfully prevented in HCR animals. The potential causations underlying the differences in bone structure and response to the HFHS diet between the HCR and LCR rats are currently being explored.

References

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