Does a high inherent aerobic capacity protect muscle morphology and mechanical properties from an obesogenic diet?

Mauricio Delgado[1,2,3], Darsh Tripathi[1,2], Barbod Shiri[1], Ignacio Cordero[3], Giorgio Poletto[1], Venus Joumaa[1], Ruth Seerattan[1], Raylene A. Reimer[1,2], David A. Hart[1,2], Lauren G. Koch[4], and Walter Herzog[1,2]
[1]Human Performance Laboratory, University of Calgary, AB, Calgary, Canada.
[2]McCaig Institute for Bone and Joint Health, University of Calgary, AB, Calgary, Canada.
[3]Departamento de Kinesiología, Escuela de Ciencias de la Salud, Pontificia Universidad Católica de Chile, Santiago, Chile.
[4]Department of Physiology and Pharmacology, The University of Toledo, Toledo, Ohio, USA.
Email: mauricio.delgado@ucalgary.ca

Summary

This study was aimed at examining if inherent high aerobic capacity protects against musculoskeletal degeneration caused by a high-fat/high-sucrose (HFS) diet. Thirty-five rats bred for high (HCR) or low (LCR) running capacity were fed either a HFS or a standard chow diet for 12 weeks. LCR-HFS rats, compared to LCR-Chow rats, showed increased fibrosis and macrophage content in the vastus lateralis, but not in the soleus, muscle. No diet-induced differences were observed in HCR rats. These results suggest that high aerobic capacity prevents muscle-specific changes in fibrosis and inflammation in obesogenic conditions in HCR but not in LCR rats.

Introduction

Obesity, and its related health issues, often lead to reduced mobility, sedentary lifestyle, and musculoskeletal degeneration [1]. Obesity has also been associated with changes in muscle integrity, fat infiltration and fibrosis, which have been linked to muscle inflammation and decreased force capacity in Sprague Dawley rats [2]. We found that rat muscles comprised of primarily slow twitch fibers with a high aerobic capacity were protected, while primarily fast twitch muscles with a low aerobic capacity degenerated within days of exposure to a high fat/high sucrose (HFS) diet [2]. Also, rats exposed to aerobic exercise preserved musculoskeletal health in the presence of a HFS diet [3]. Aerobic capacity has also been shown to be crucial in regulating metabolic disruption in the presence of an obesity-inducing diet [4]. However, the hypothesis that high inherent aerobic capacity prevents or mitigates musculoskeletal degeneration in the presence of a HFS diet has not been tested systematically. The purpose of this study was to investigate the effect of a HFS diet on the structural integrity and mechanical properties of skeletal muscles in rats with a high or a low inherent aerobic capacity.

Methods

Animals: Thirty-five 25-week old N:NIH male rats selectively bred for either high running capacity (HCR) or low running capacity (LCR) were used in this study (4). Animals were fed a HFS diet or a standard chow diet (CD) for 12 weeks, resulting in four experimental groups (HCR-Chow, n=10; HCR-HFS, n=9; LCR-Chow, n=8; LCR-HFS, n=8). Animals were sacrificed and the vastus lateralis (VL) and soleus (Sol) muscles harvested. *Muscle integrity:* Muscle structural integrity was assessed using histological analysis. 10 µm mid-belly muscle sections were used to determine the amount of fibrosis using Picro Sirius Red, the amount of fat infiltration using Oil Red-O staining, and the presence of inflammatory macrophages using CD68 enzyme histochemistry staining.

Mechanical properties: Skinned single muscle fibers (HCR-Chow, n=20; HCR-HFS, n=18; LCR-Chow, n=16; LCR-HFS, n=16) were isolated and tested for their passive force and maximal isometric force at sarcomere lengths of 2.4-3.2µm.

Results and Discussion

The collagen (p=0.03) and macrophage content (p=0.001) was increased in VL of the LCR-HFS compared to the LCR-Chow group rats (Fig 1). There was an increase (p= 0.02) in fat infiltration in the VL of the HCR-HFS compared to the HCR-Chow group. There were no differences in Sol fibrosis, macrophage number, or fat infiltration between diet groups. There was no difference between diet and aerobic capacity groups in active and passive forces for VL and Sol fibers when normalized to cross-sectional area.

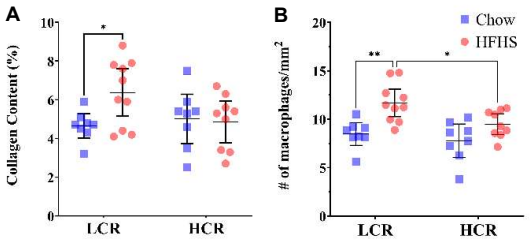

Figure 1: Collagen (A) and macrophage (B) content in the VL muscle of the tested groups (*p < .05, **p < .01)

It has been shown that macrophage infiltration in muscles increases with local inflammation, contributing to increases in collagen content [5]. Our results suggest the presence of fibrosis in VL may be explained by the local inflammation due to the HFHS diet only in the LCR group, suggesting that the high aerobic capacity may reduce the impact of a HFS diet on fast twitch muscles. The lack of mechanical changes in both muscles shows that the detrimental effects of twelve weeks of HFHS diet do not affect the fiber level. Further investigation is required to elucidate the mechanism behind the progression of muscle fibrosis and the potential protective effect of the intrinsic high aerobic capacity.

Conclusions

We conclude from the results of this study that high inherent aerobic capacity may protect against muscle fibrosis in the presence of a HFS diet.

References

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