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Doctoral thesis abstract

The Impact of Obesity on Skeletal Muscle Morphology, Mechanical Properties and Functional Implications

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1. BACKGROUND AND OBJECTIVES

Obesity is a growing global epidemic associated not only with metabolic and cardiovascular disorders but also with impairments in skeletal muscle and tendon integrity (Abate et al., 2016; Lui & Yung, 2021; Tallis et al., 2018; Tomlinson et al., 2016; Wearing et al., 2006). While the metabolic consequences of obesity are well described (Blüher, 2019), its impact on the structural and mechanical properties of musculotendinous tissues and their functional implications has remained underexplored. The doctoral thesis investigated how obesity alters the morphology, material properties, and function of the muscle–tendon unit (MTU), aiming to clarify the mechanisms that contribute to reduced movement efficiency, tissue fragility, and impaired functional capacity.

2. METHODS

The dissertation comprised four interrelated experimental studies using both animal and human models, following a translational and multiscale approach to investigate the mechanical, structural, and functional consequences of obesity on the MTU and respiratory system. Ethical approval was obtained for animal (Ref. No. G2-255) and human studies (Ref. No. 2023-BE10-0001), and all procedures complied with institutional and international ethical standards.

In the animal studies, male C57BL/6J mice were exposed to either an obesogenic high-fat diet or a standard control diet to establish a model of diet-induced obesity. **Study I** characterised the passive mechanical properties of isolated skeletal muscle and adipose tissue in healthy mice using *in vitro* mechanical testing, providing baseline data on tissue-specific mechanical behaviour. **Study II** employed the diet-induced obesity mouse model to investigate obesity-related structural and mechanical remodelling of skeletal muscle, tendon, and the myotendinous junction through comprehensive biomechanical testing, histological analysis, and molecular profiling.

The human studies extended these mechanistic findings to functional outcomes. Participants were sedentary men aged 30–50 years classified as class I obese (BMI: 30.0–34.9 kg/m²) or normal-weight controls (BMI: 18.5–24.9 kg/m²). **Study III** examined the effects of obesity on triceps surae MTU morphology, passive mechanical properties, muscle contractile function, and locomotor efficiency during repeated calf raise exercise. **Study IV** investigated the impact of obesity on respiratory system function, including lung capacity, respiratory muscle strength, breathing patterns, and diaphragm morphology and contractile function.

Animal experiments included *in vitro* mechanical testing of isolated muscles and tendons to assess passive mechanical properties and contraction–relaxation capacity and dynamics, combined with histological evaluation and protein expression analyses to characterise structural remodelling and

molecular signalling pathways. Human assessments employed non-invasive techniques, including ultrasonography to evaluate muscle and tendon morphology; dynamometry and surface electromyography to assess MTU mechanical properties and neuromuscular function; spirometry to measure respiratory function; indirect calorimetry to determine metabolic cost; and motion analysis to evaluate locomotor performance and efficiency.

3. RESULTS

Study I demonstrated that skeletal muscle and adipose tissues exhibit distinct passive mechanical properties. Skeletal muscle showed significantly greater modulus and lower hysteresis compared to adipose tissues ($p < 0.05$), indicating greater resistance to deformation and more efficient elastic energy storage. **Study II** showed that diet-induced obesity caused significant structural and molecular remodelling of the MTU. Obese mice exhibited increased connective tissue infiltration and elevated expression of TGF-β1 and Periostin ($p < 0.05$). These alterations were associated with increased tissue fragility and viscosity, with higher modulus in skeletal muscle but lower modulus in tendons ($p < 0.05$). Obese muscles also demonstrated reduced specific force and slower contraction–relaxation kinetics ($p < 0.05$). In humans, **Study III** demonstrated that the triceps surae MTU of individuals with obesity was characterised by increased adipose infiltration, stiffness, and hysteresis ($p < 0.05$), accompanied by reduced relative muscle strength ($p < 0.05$) and impaired calf-raise performance and movement efficiency ($p < 0.05$). **Study IV** further confirmed the detrimental effects of obesity on respiratory function, showing reduced ventilatory capacity, respiratory muscle strength, and diaphragm function ($p < 0.05$). These impairments were associated with altered breathing patterns, characterised by increased respiratory rate and reduced tidal volume for a given minute ventilation in individuals with obesity ($p < 0.05$).

4. CONCLUSIONS

Taken together, these findings provide converging evidence that obesity promotes structural and biochemical remodelling of musculotendinous tissues, including adipose infiltration, fibrotic tissue accumulation with increased collagen cross-linking and disrupted extracellular matrix organisation. Functionally, these alterations modify tissue passive mechanical behaviour, impair force production–relaxation dynamics, increase mechanical energy dissipation, and compromise tissue resilience. Consequently, individuals with obesity may experience muscle weakness, increased energy cost of movement, reduced mobility, a less efficient breathing pattern, early fatigue, and MTU fragility, all of which contribute to a decline in physical function and quality of life. The thesis underlined the importance of recognising obesity not only as a metabolic disease but also as a condition of musculoskeletal dysfunction. By elucidating the mechanical, structural, and functional consequences of obesity-induced musculotendinous remodelling, these findings provide a foundation for targeted preventive and rehabilitative strategies aimed at preserving or restoring movement efficiency and musculoskeletal health in individuals with obesity.

Keywords: Obesity, skeletal muscle, mechanobiology, muscle physiology, tendon

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