Genetic Polymorphisms Influence Torque and Recovery After Eccentric Exercise-Induced Muscle Damage

Fernanda O. Ferreira-Schmidt[1], Liliam F. Oliveira[1], Thiago T. Matta[2], Diogo A. Tschoeke[1], and **Luciano L. Menegaldo** [1]
[1]Biomedical Engineering Program, Coppe, Federal University of Rio de Janeiro, Rio de Janeiro-RJ, Brazil
[2]Physical Education and Sports School, Federal University of Rio de Janeiro, Rio de Janeiro-RJ, Brazil
[1] Email: lmeneg@peb.ufrj.br

Summary

This study investigates the influence of genetic polymorphisms on the susceptibility to muscle damage after maximal eccentric contractions. Twenty-four healthy male participants performed eccentric elbow flexion exercises using an isokinetic dynamometer. The genetic polymorphisms rs12722 and rs4341 were associated with the production and recovery of maximum isometric elbow flexion torque. The reference genotypes produced a higher torque than the alternate alleles. These results suggest that genetic polymorphism modulates both muscle strength and recovery dynamics.

Introduction

Resisted eccentric contractions are associated with enhanced muscle strength and hypertrophy, primarily driven by exercise-induced muscle damage (EIMD). However, short-term recovery often involves decreased strength and range of motion. Our previous findings indicated substantial variability in individual responses to injury and recovery [1]. We propose that single nucleotide polymorphisms (SNPs) may underlie muscle strength and recovery differences. Understanding how genetic polymorphisms influence muscle force and its restoration after EIMD is valuable for guiding personalized training and rehabilitation strategies. Therefore, the primary objective of this study was to investigate the influence of key gene polymorphisms related to athletic performance on elbow strength and recovery following EIMD.

Methods

Twenty-four healthy men (27 ± 4 years, 76.5 ± 9.9 kg, 176.7 ± 8.9 cm) participated in the study. Inclusion required the absence of upper-extremity musculoskeletal injuries, chronic diseases, and ergogenic aid use. Participants performed maximal eccentric contractions on an isokinetic dynamometer (Biodex System 4 Pro). They were seated with the right shoulder flexed at 45°, 0° abduction, and a supinated forearm, grasping the lever with the elbow at 110° of flexion. The device extended the elbow at 30°/s to full extension (0°) while participants exerted maximal flexion torque. Ten repetitions were performed, with the lever returning passively to flexion. Maximum voluntary isometric torque was assessed pre-EIMD at 48 and 96 hours post-exercise.

DNA samples were collected with sterile swabs after a 1-hour fast. Each swab was rubbed $\tilde{3}0$ times on the inner cheek, stored in a labeled paper envelope, and sent for commercial genotyping. Data were analyzed in Python 3.9 using NumPy, Pandas, SciPy, and StatsModels. At each time (pre-EIMD, 48, and 96 hours), torque z-scores associated with the allelic variations were compared with a one-way ANOVA and Tukey post hoc test.

Results and Discussion

Pre-EIMD torque was influenced by rs12722 (COL5A1) (p=0.009), with CC genotypes producing higher torque than TT (p=0.009) and CT (p=0.026). No significant effect of rs1815739 (ACTN3) (p=0.566) was observed. In contrast, rs4341 (ACE) significantly affected pre-EIMD torque (p=0.041), with GG showing higher torque than CC (p=0.032). Torque z-scores were measured at pre-EIMD, 48h, and 96h post-exercise. The rs12722 polymorphism influenced torque recovery (Figure 1A), with CC exhibiting higher torque than CT/TT (p=0.009, 0.031, 0.021 for pre-EIMD, 48h, and 96h, respectively). The rs1815739 polymorphism showed no significant differences (Figure 1B), and rs4341 CC displayed higher torque only at pre-EIMD (Figure 1C).

These findings underscore the influence of Collagen Type V alpha 2 (rs12722) polymorphisms on muscle strength and recovery, potentially affecting RNA stability and collagen network regulation during recovery [2]. In addition, serum ACE activity modulates muscle function and metabolism, suggesting its potential role as a genetic marker of performance [3].

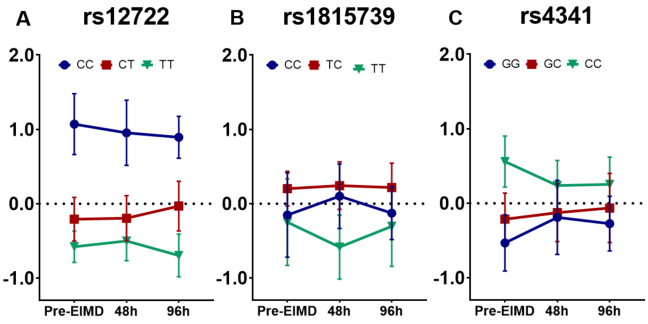

Figure 1: Torque z-score variation for rs12722, rs1815739, and rs4341 post-eccentric damage.

Conclusions

COL5A1 regulates extracellular matrix formation and was related to pre-EIMD torque and recovery. ACE, which is related to lipid metabolism, apparently influences only the baseline torque, while ACTN3, known to affect athletic performance, had no role in torque production.

Acknowledgments

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