

Genetic polymorphisms influence on sports injuries and muscle damage

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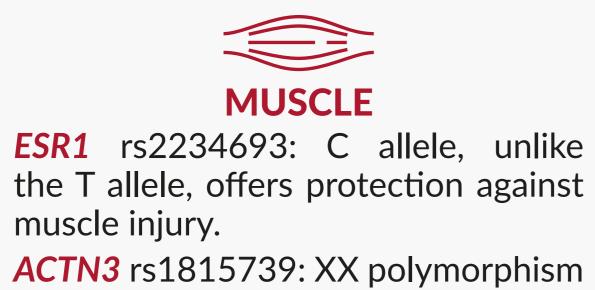
Etienne Delforge, Julien Boissiere, Sebastien Imbert, Gérard Dine, Frédéric Daussin. Genetic polymorphisms influence on sports injuries and muscle damage. ECSS, Jul 2023, Paris, France. hal-04247185

HAL Id: hal-04247185 https://hal.univ-lille.fr/hal-04247185v1

Submitted on 18 Oct 2023

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GENETIC POLYMORPHISMS INFLUENCE ON SPORTS INJURIES AND MUSCLE DAMAGE



or X allele was associated with a higher risk or incidence of non-contact muscle injury.

HGF polymorphisms: are related to the incidence or severity of noncontact muscle injury.

1. INTRODUCTION

Sports practice or physical activity has been acknowledged to be **beneficial for health.** However, it **may** also **induce injuries.** Indeed, when related to 1000 hours of practice, injury incidence during training is 3.7 in football and 3.0 in rugby union and even increases to 36.0 and 81.0 (respectively) during matches. Injuries negatively **affect** the players' **performance and health** and may have financial implications for the athletes and/or their clubs. Therefore, avoiding players' injuries became a priority and strategies are developed to limit them. Training workloads focused staff attention as their monitoring allows to maximize the training processes and performance and reduce the occurrence of injury. Internal and external outcomes are used to characterize the workload. For the same external workload, the internal responses differ for each **people** leading to different levels of fatigue or exercise-induced muscle damage.

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FIGURE 1. SPORTS INJURIES & MUSCLE DAMAGE **MMP3** polymorphisms: could affect the muscle sensibility to injury. **COL5A1** rs12722: could relate to the severity of non-contact muscle injury.

2. OBJECTIVES

While a high training load and a certain amount of muscle damage are necessary to induce adaptations and promote performance increase, excessive muscle damage favor injury occurrence. Great muscle damage inter-individual variations have been observed following the same external training load. Scientific evidence support that several factors are involved in the occurrence of injury or exercise-induced muscle damage. Besides exercise characteristics, individual risk factors, such as genetics, seem to **be** a **component** to **take into account in injury** mechanisms. Particularly, single genetic polymorphism (SNP), a variation in DNA sequence, may alter proteins structures and their function within the cell. Studies support that SNP may be considered a predisposing factor. Identifying injury-predisposing polymorphisms is of interest to improve training load prescription. This poster aims to present a review of SNP that affect muscle damage and sports injuries.









3. RESULTS & DISCUSSION

Several studies support the role of SNP in altering muscle structure and potentially compromising its integrity. Results suggest that individuals with specific genetic variations may exhibit a higher susceptibility to muscle damage or sports injuries compared to others. These polymorphisms (Figure 1), which affect the structural composition of muscle components, could make them sensitive to exercise-induced mechanical stress. Furthermore, variations in the inflammatory response and metabolic processes, such as impaired lactate transport across muscle membrane, may further weaken the muscle and increase the risk of injury. Hence, the **presence or lack** of these **SNP appears** to contribute significantly to the substantial inter-individual variability observed on sports injuries or induced muscle damage whereas the external workload exercise is similar.









METABOLISM

MCT1 rs1049434: injury incidence of AA genotype > TT in elite football. **IL6** rs1800795: associated with non-contact ACL rupture in Polish soccer players.

ACE rs4646994: DD genotype may have a protective effect against muscle damage.

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4. CONCLUSIONS

The present study supports the interest to assess genetic polymorphisms in athletes to better individualize training strategies and workload. During the last decade, several SNP involving muscles, ligaments, or tendons may affect positively or negatively the risk, incidence, and severity of injuries have been identified. Among them, ACE, ACTN3, COL5A1, IL6, MCT1, MMP3, and HGF seem to play an important role in sports injuries and muscle damage mechanisms.



