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Genotypes and Phenotypes Associated with Muscular Recovery: Impact of Exercise and Diet

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Abstract

In recent years, significant advances in molecular biology has facilitated emerging knowledge pertaining to genetics in sport science research. Specific regions of DNA are known to influence genetic polymorphism(s) and partly explain individual variations in response to exercise stimuli and diet. Following exhaustive exercise, certain genetic variations or polymorphisms have been associated with muscle damage indices and may influence muscular recovery. The purpose of this narrative review is to outline the transcription factors of co-activators of associated polymorphisms that appear to play a role in muscle recovery. We also highlight the potential interaction of gene expression and the impact of macronutrients. Several genes (ACE, ACTN3, CCL 2 (C> T), COL5A1, CKM (A> G) have been implicated in various aspects of skeletal muscle remodeling. Individuals with specific genotypes experience changes in muscle damage and recovery rates following exercise. The contribution of heritability to a specific phenotype is likely dependent and the modality, intensity, and duration of exercise. Future research is warranted to explore multigenetic characteristics to provide a deeper molecular understanding of recovery, adaptation and nutritional modulation that may allow the identification of individuals with a greater genetic predisposition, or with a greater risk of developing muscle injuries.

Keywords: Genetic; Athletes; Sports Performance; Nutrients

Key Points

- This review aimed to describe genotypes and polymorphisms associated with associated with muscular recovery.
- Several genes (ACE, ACTN3, CCL 2 (C> T), COL5A1, CKM (A> G) have been implicated in various aspects of skeletal muscle recovery.
- Future research will allow the assessment of multigenetic characteristics which is required to provide a deeper molecular understanding of recovery supporting the development of guidelines.

Introduction

Muscular adaptations are multifactorial and are primarily determined by exercise stimulus and adequate nutritional support; however, there is large individual variability. As such, genetics are emerging as an important variable to predict responsiveness. Identifying key genotypes and polymorphisms is a critical factor for understanding individual acute exercise performance and physiological responses and adaptations (Ordovas et al., 2004). Presently, genes have been associated with performance, expression of metabolic proteins and enzymes, and with the activation of specific signaling pathways regulating transcription and translation in response to exercise (Eynon et al., 2011).

Exercise is well known to upregulate and signal specific proteins associated with enhancing mitochondrial biogenesis and oxidative power and capacity (Abreu et al., 2017), alter metabolic flexibility (Armstrong et al., 1991), and influence intracellular signaling and transcriptional responses (Hyldahl et al., 2014). Molecular adaptations are mediated by stimuli involved in controlling the content of mRNA and these proteins in skeletal muscle, in theory, increase exercise performance.

Some sports require greater muscular stress and eccentric overload leading to increase muscle soreness from 24 to 72 hours after a single exercise bout (Armstrong et al., 1991). Exercise intensity and type of muscular contraction are also associated with muscle injury, leading to an increased inflammatory response and activation of adjacent pathways, such as muscle protein degradation (Hyldahl et al., 2014) and changes on circulating muscle-specific proteins [i.e. creatine kinase (CK), myoglobin and α-actin] (Grobler et al., 2004; Martinez et al., 2007). Noteworthy, some polymorphisms may modify the proteins which can potentially modulate muscle function or adaptation (Baumert et al., 2016). Other polymorphisms encode key proteins in tendons, such as ACTN3 R577X SNP (Yang et al., 2003) which is a candidate to modulate recover process from strenuous exercise, due to exacerbating inflammatory responses (e.g., IL6-174) (Baumert et al., 2016). In elite athletes, the incidence of injuries is higher than general population partly due to training exposure and also by different genetic profiles (Yang et al., 2003; Nascimento et al., 2016). It is likely that the contribution of heritability to a specific phenotype depends largely on the specific sport (Nascimento et al., 2016; Aragon et al., 2017). These genetic differences may alter muscle recovery, training and responses to nutrition (Aragon et al., 2017). In athletes, body composition is often the focus of change, as it can be manipulated through total energy intake and macronutrient composition (Aragon et al., 2017; Nielsen et al 2014). Genetic variants influence the absorption, metabolism, and degradation of nutrients (El-Sohemy et al., 2017). The interactions between diet and genes related to metabolic pathways involved in both health and sports performance are becoming more widely recognized (El-Sohemy et al., 2017; Kicklighter et al., 2017).

Both aerobic and resistance exercise represent opposite ends of the exercise spectrum and elicit markedly different training responses (Lucia et al., 2010). Adaptations arising from both aerobic and resistance training are mediated by a complex interaction between genes and nutrients (Muller et al., 2009) altering signaling pathways and main transcription co-activators associated polymorphisms (Muller et al., 2009; Coffey et al., 2009) related to exercise-induced muscle damage (Coffey et al., 2009). Macronutrient imbalance may alter metabolic, physiological and functional pathways reducing performance (Longland et al., 2016; Krieger et al., 2006). Carbohydrates provide an essential fuel (Beelen et al., 2015) for brain and skeletal muscle (Goss et al., 2013; Bartolomei et al., 2017) and their consumption effects sport performance predominantly in high intensity exercise (Goss et al., 2013; Marquet et al., 2016). Proteins are essential for strength, increases and/or maintenance of lean body mass, in addition to playing an important role in immune function (Longland et al., 2016; Naclerio et al., 2016). Fat (fatty acids and glycerol), are an important source of energy, and play a critical role in the regulation of genes and in the cellular adaptation of skeletal muscles (Garaulet et al., 2011). The aim of this review was to discuss the current scientific data about genotype polymorphisms associated with exercise-induced muscle damage and the interaction with diet/macronutrients.

Transcription factors and recovery

Exercise induced skeletal muscle remodeling requires increased protein synthesis through gene expression's regulation (Roth et al., 2012). Increased cardiorespiratory fitness and/or skeletal muscle strength may improve metabolic dysfunction and prevent oxidative

stress (ref). The benefits of transcriptions are mediated, at least partially, by extensive metabolic and molecular remodeling of the skeletal muscle stimulated by physical effort (Aaltone et al., 2014; Lucia et al., 2010). Aerobic and resistance exercise induces markedly different responses, mediated by a complex interaction through signaling pathways coupled with transcription and translation regulators (Aaltone et al., 2014; Willians et al., 2008). Willianns and Folland (2008), determined the probability of occurrence of humans with the "perfect" polygenic resistance profile. The profile was obtained from the theoretically best accumulated combination of 23 genetic polymorphisms, each of which is a candidate to individually influence human variability in one or more phenotypic characteristics of associated with resistance exercise, recovery and genes related to cell differentiation (Lucia et al. ,2010; Kurosaka et al., 2012).

Skeletal muscle regeneration is a complex process, mediated by satellite cells, in which several factors are activated to regulate muscle remodeling (Kurosaka et al., 2012). Thus, the understanding of transcription factors can act at different times of gene expression, reflecting the activation and / or repression of specific signaling pathways that regulate muscle recovery (Clarkson et al., 2005). A summary of all the transcription factors is shown in table 1.

Gene	***Polymorphism***	***Population***	***Physiological effect/ Major findings***	***References***
Angiotensin gene *ACE* (I/D)	rs4646994	Physically active women	Creatine kinase and recovery from eccentric muscle lengthening	Yu JC et al. (2004)
Actin protein gene *ACTN3*	rs1815739	Physically active women and men	See above for more on *ACTN3*; some forms may predispose people to exercise-induced rhabdomyolysis (excessive muscle tissue breakdown)	Clarkson et al. (2005) Pimenta et al. (2012)
Chemokine (cell signaling) lingad and receptor genes CCL CCR	*CCL2* −3441(C>T) rs3917878 *CCL2* −289 (G>C) (rs2857656) *CCR2* −941(A>C) (rs3918358) *CCR2* 4439 (T>C) (rs1799865)	Untrained healthy young males and females Elite soccer players Healthy untrained men and women Healthy untrained men and women	Markers of exercise-induced skeletal muscle damage; soft tissue injuries	Hubal et al. (2010) Pruna et al. (2013)
Collagen repair genes COL	*COL1A1* rs1800012, *COL5A1* rs12722, rs3196378 *BstUI* RFLP *COL27A1* rs4143245, rs1249744, rs753085, rs946053 *TIMP2* rs4789932 *TNC*	Healthy trained males	Remodeling of connective tissues; variants (especially in *COL5A1*) are associated with higher rates of anterior cruciate knee ligament injury, tennis elbow, carpal tunnel and Achilles tendon tears	Kirk et al (2016) Languete et al. (2010) Stepien et al. (2013)

Creatine Kinase gene *CKM* *CKMM*	*CKM* (A>G) (rs1803285) and (rs8111989	Professional athletes, amateurs and non-practitioners	Creatine kinase as well as C-reactive protein (CRP), a marker of inflammation. Muscle-specific creatine kinase (CKMM) plays a vital role in the energy homeostasis of muscle cells	Fedotovskaya et al. (2012) Eider et al. (2015) Batavani et al. (2017)
Interleukin genes *IL*	*IL1B* −3737 (C>T) (rs4848306) *IL1B* 3954 (C>T) (rs1143634) *IL6* −174 (G>C) (rs1800795)	Healthy untrained men Healthy untrained men	Inflammatory response to exercise and muscle damage	Dennis et al. (2004) Pedersen et al. (2003) Yamin at al (2008)
Solute carrier family 30 (zync transporter) gene SLC30A8	(C> T) (rs13266634)	Untrained healthy young men and women Young adults the eccentric muscle damage	Zinc transport and insulin secretion; associated with recovery	Sprouse et al. (2014) Chimienti et al. (2004)
Superoxide dismutase 2 gene *SOD2*	(C>T) (rs4880)	Healthy men elite	Recovery from oxidative stress	Akimoto et al. (2010) Huang et al. (2000)
Myosin kinase genes *MLCK C*-49T *MLCK* 3788A	(C>T) (rs2700352) (C>A) (rs28497577)	Adults the eccentric muscle damage	Myosin muscle protein phosphorylation; may affect how well muscle fibers can tolerate mechanical force	Clarkson et al. (2005)
Tumor necrosis factor gene TNF -308	(G>A) 1800629	Moderately active young men and women	Creatine kinase (CK) response to eccentric exercise; also regulates muscles' ability to repair and grow	Yamin at al (2008) Warren et al. (2002)

Table 1: Gene polymorphism associated with exercise-induce muscle damage.

Skeletal muscle remodeling following exercise induced muscle damage

Damage induced by exercise can be divided into the initial phase, which occurs during the exercise session, and the secondary phase, which is linked to the delay in the inflammatory response (Baumert et al., 2017; Howatson et al., 2008). These phases are critical to stimulate muscle remodeling (Thiebaud, 2012; Tidball, 2005). Appropriate muscle damage caused by exercise provides a positive stimulus for muscle restructuring, hypertrophy and strength gains (Roig et al. ,2008). Evidence for myofibril remodeling, control blood pressure and inflammation associated with the ACE genotype (Yu et al., 2004). The association between ACE I / D polymorphism and elite athlete status is associated with a genotype linked with susceptibility to muscle damage and injuries (Heled et al., 2007). Yamin et al., (2007) observed different circulatory CK concentrations (i.e. markers of muscle damage) among ACE genotypes following eccentric exercise; homozygotes II had the largest CK response, while DD homozygotes had the lowest. Repeated eccentric exercise results in significantly less muscle soreness and attenuated rise in serum CK, as well as faster recovery of muscle function. R577X polymorphism of the α-actinin-3 is a gene that encodes the α-actinin-3 protein, together with the α-actinin-2 protein is an important structural component of the Z disc, which is the anchor for actin thin myofilaments and assists with maintaining the myofibriliar array (Clarkson et

al., 2005; Pimenta et al., 2012). Seto et al., (2013) provides a mechanistic explanation for the associations be-tween the ACTN3 genotype and the phenotypes of muscle size, strength, power and endurance. Recent research has suggested that α-actinin-3 can minimizes muscle damage (Yang et al., 2003). Alpha-actinin-3 can also influence oxygen consumption. For example, reduced ACTN3 can increase oxygen utilization at rest, thus enhancing metabolism and delay athletes with a CT or TT genotype (Clarkson et al., 2005). Studies in rats have found that muscle fibers without alpha-actinin 3 are more fragile and smaller, but more efficient and fatigue-tolerant representing an ideal phenotype for endurance athlete (Yang et al., 2003).

The chemokine ligand 2 (CCL2), also known as monocytic chemo-attractive protein 1 (MCP1), can be classified as a signaling factor, as it measures systemic changes induced by chronic exercise (Hubal et al., 2010). The protein 1 chemo-monocyte attractor receptor 2 (CCR2) is one of the main receptors that binds to CCL2, alongside CCL7 and CCL13. CCL2 is expressed primarily in the interstitial space between myofibers after chronic muscle exercise and is co-located with macrophages and satellite cells in the muscle (Pruna et al., 2013; Warren et al., 2005). Warren et al.,(2005), demonstrated that the test in rats for CCR2 registered regeneration was associated with inflammation and fibrotic response after injury, suggesting a strong interaction between CCL2 / CCR2 and an immune response after exercise-induced muscle injury (Hubal et al., 2010; Yahiaoui et al., 2008). Hubal et al. (2010) investigated several CCL2 / CCR2 SNPs in association with muscle damage induced by exercise in the elbow flexor muscles. After strenuous exercise, Hubal et al., (2010) associated the SNP T allele CCL2 rs3917878 (C>T) with a delayed recovery of maximum strength in males compared to higher levels of CK in females. Individuals with the C allele of SNR CCR2 (rs3918358) showed a slower recovery of strength in females, and the C allele of SNP CCR2 (rs1799865) increased pain in both sexes (Hubal et al., 2010). The significant differences between the alleles of these three SNPs occurred from 4 to 10 days after muscle damage, confirming the CCL2 / CCR2 action pattern in muscle repair and regeneration (Hubal et al. 2010).

Many athletes have more rigid or stiff tendons and ligaments that may confer an advantage in certain sports since these tissues can support more load and produce more elastic energy to generate an increase in force production; however, they may be more prone to injury (Kirk et al., 2016). Variations in the COL1A1 gene, which encodes type I collagen, are associated with several complex connective tissue disorders, in addition to dislocations and ruptures of the shoulder and anterior cruciate ligament (ACL) of the knee and / or achilles tendon (Kirk et al., 2016; Laguette et al., 2005). Tenascin-C, encoded by the TNC gene, is a glycoprotein that is also involved in wound healing, as well as in the formation of tendons, ligaments, cartilage and bones. Similar to variations in COL genes, variations in the TNC gene are linked to tendon injuries (Stepien et al., 2013). The relative proportion of different collagen subtypes in the extracellular matrix of skeletal muscle and tendon variants depends on the position and function of connective tissues in physical exercise (Brown et al., 1999; Andersen et al., 2010). Likewise, it is believed that an increase in the gene expression of collagen types I and III and laminin-β2 is based on muscle restructuring after exercise-induced damage (McHugh, 2003; Gordon et al., 2012; Mackey et al., 2011).

The function of creatine kinase (CKMM) in skeletal muscle, plays an essential role in the homeostasis of muscle cell energy. The A / G variation (rs8111989) located in the 3 ′ untranslated region of the CKM gene was considered the most relevant genetic transcription in terms of genetic testing in sport (Eider et al., 2015). Studies show that during high intensity exercise, adenosine diphosphate (ADP) accumulates in contracting muscles, triggering creatine kinase to resynthesis ATP via creatine phosphate (CrP) (i.e., CrP + ADP- H ++ Cr + ATP2-, where Cr is creatine) (Fedotovskaya et al., 2012; Eider et al., 2015). The reaction is catalyzed by the muscle isoform of creatine kinase (M-CK), one of the main enzymes in the energy supply for muscle performance (Fedotovskaya et al., 2012). Thus, the M-CK located on the surface of the endoplasmic reticulum influences the power of muscle contraction, regulating the flow of calcium ions during the phases of tension and relaxation. In addition, M-CK, together with the mitochondrial isoform of creatine phosphokinase, is involved in the transport of energy generated by oxidative phosphorylation to muscle contraction proteins (the transport of creatine phosphate) (Batavani et al., 2017; Grassi et al. , 2011). Previous studies have demonstrated that the inhibition of the CK-MM activity causes a reduction in the intensity and strength of muscle contractions resulting in an enhanced oxygen uptake by contracting muscles. This SNP may have an influence on the mRNA stability and may change the gene expression (Grassi et al., 2011).

Recent investigations have revealed that some cytokines are also expressed by skeletal muscle and are known as myokines (Pedersen et al., 2003). A functional C-G polymorphism in the 5' flanking region on the IL6 gene (rs1800795) shows that the mutant G

allele is associated with an increased creatine kinase activity following eccentric exercise (Baumert et al., 2016), suggesting that the G allele may protect skeletal muscle during powerful contractions and assist in repair, promoting adaptations after exercise (Dennis et al., 2004). Paulsen et al., (2012) reviewed the role of cytokines in the inflammation phase after exercise-induced muscle damage, particularly cytokines that are classified as proinflammatory cytokines [promoting inflammation, p. interleukin (IL) -1α, IL-1β (Paulsen et al., 2012). Some cytokines such as IL-6 can act as a pro or anti-inflammatory agent, depending on the environment (Baumert et al., 2016; Pedersen et al., 2003; Paulsen et al., 2012). Most cytokines are released from various types of cells, including muscle fibers, fibroblasts, neutrophils and macro-phages, and the expression of cytokines is determined by the modality, intensity and duration of exercise (Paulsen et al., 2012).

The SLC30A8 gene encodes a protein, the Zinc Efflux Transporter 8 protein, which is expressed primarily in pancreatic alpha and beta cells (Baumert et al., 2016). This transmembrane protein transports zinc from the cytoplasm to the insulin-secreting vesicles, where the insulin is stored as a solid hexamer attached to two Zn2 + ions in the pancreatic islets beta cells (Sprouse et al., 2014). Zn2 + ions firmly regulate insulin production and are essential for insulin storage and crystallization (Chimienti et al., 2004). Spouse et al., (2014) demonstrated that a copy of the risk allele (C allele) for the SLC30A8 variant influences an individual's response to a resistance training intervention. The SLC30A8 gene encodes a zinc transporter expressed only in the secretory vesicles of beta cells and is implicated in the final stages of insulin biosynthesis involving zinc co-crystallization (Deniro et al., 2012). Communication between skeletal muscle and beta cells is thought to contribute to healthy skeletal muscle function and mass (Pedersen et al. 2003; Deniro et al., 2012).

The MLCK's function is to phosphorylate the myosin regulatory light chain (RLC) (Clarkson et al., 2005). However, binding of Ca2 to tropomyosin-troponin is the main regulator of skeletal muscle contraction, RLC plays an important modulating role in the development of strength (Clarkson et al., 2005; Sweeney et al., 1990). However, RLC is not readily phosphorylated in type I fibers and therefore acts predominantly in type 2 fibers (Szczesna et al., 2002). It is possible that MLCK polymorphisms alter the ability to phosphorylate RLC in type 2 fibers thus decreasing the ability to withstand tension during stretching of contractions (Clarkson et al., 2005; Sweeney et al., 1990). The association of the MLCK C49T and MLCK C3788A genotypes with increase in CK and Mb may be an important clinical finding, because individuals who have the rare alleles may show an exaggerated increase in Myoglobin (Mb) in response to exercise stress (Clarkson et al., 2005; Szczesna et al., 2002).

TNF-a can be produced by activated macrophages, lymphocytes or monocytes (Bingham, 2002). The main stimulus for its production is the presence of lipo-polysaccharides that make up the membrane of gram-negative bacteria. TNF-a, when released in low concentrations act on endothelial cells promoting vasodilation and stimulating them to secrete a group of cytokines (called chemokines) that have chemotactic action in relation to leukocytes, thus promoting a local inflammatory process that makes it possible to fight infectious conditions (Warren et al., 2002). Another TNF-alternative approach is to modulate or block cytokine activity before muscle damage and then examine subsequent muscle regeneration. These effects delay the formation of new muscle fibers and increase the accumulation of lipids and necrosis in regenerating muscle tissue (Warren et al., 2002; Peterson et al. 2006). The mechanisms by which TNF-α deficiency impairs muscle regeneration are less clear, but may also involve a decline in the infiltration of neutrophils and macrophages (Peterson et al., 2006) and / or in the expression of myogenic regulatory factors (Warren et al., 2002). Few studies have reported changes in the expression of TNF-α mRNA in the muscle after exercise, and its role in hu-man skeletal muscle remains uncertain. IL-6 may be more active in regenerating tendon tissue (Costill et al., 1990). The research described above involves TNF-α and MCP-1 and their receptors in muscle regeneration after acute muscle injury (Sireman et al., 2007). They can play a different and potentially negative role in chronic diseases that involve loss of muscle mass (Peterson et al., 2006; Costill et al., 1990).

Superoxide dismutase is an antioxidant that protects cells and mitochondria from free radical damage by converting anionic superoxide into hydrogen peroxide (Huang et al., 2000). During exercise, there is a higher intracellular concentration of antioxidants within a muscle fiber that protects against the negative impact of ROS promoted by oxidative stress from exercise (Huang et al., 2000; Akimoto et al., 2010). The inhibition of superoxide dismutase causes an increase in the accumulation of superoxide free radicals, which can lead to increased damage to the mitochondrial membrane and cell apoptosis (Huang et al., 2000). The SNP Ala16Val (rs4880, C> T) of superoxide dismutase 2, mitochondrial gene (SOD2), has been associated with susceptibility to muscle damage (Schoenfeld, 2010). The

T allele is associated with a reduction in the efficiency of mitochondrial superoxide dismutase against oxidative stress (Ahmetov et al., 2014). Overall, the current evidence suggests that polymorphisms have been developing a favorable genetic profile as co-activating factors of transcription associated with the induction or repair of muscle damage, or both (Morton et al., 2009).

Macronutrients, genes and exercise-induced muscle damage recovery

Nutrients can actively act to influence the rate of transcription, through the alterations of specific signaling pathways and genetic variants relevant to exercise performance (Cousins, 1999). Optimal nutritional strategies are needed to support adaptive responses to enhance performance (Tipton, 2010; Tipton, 2013). The macronutrient composition is a critical component of any nutritional plan for optimal recovery, in addition to reducing or recovering from injury and periods of immobilization and reduced activity (Wall et al., 2015). The amount of exercise and resting energy expenditure typically dictate total energy expenditure during immobilization (Bergouignan et al 2010; Ferrando et al., 1996). An imbalance in macronutrients can result in metabolic and physiological stress and hinder recovery (Frankenfield, 2006). Carbohydrate influences the expression of genes involved in metabolic pathways. The amount of carbohydrate has a large effect on gene expression associated with cell adhesion, cycle and growth (Frankenfield, 2006; Volek et al., 2015). Carbohydrate consumption during and after exercise can affect the increased or decreased rate of muscle and liver glycogen synthesis, mainly by insulin-mediated glycogen synthase activation (Prats et al., 2009). In addition, exercise-induced muscle damage has been associated with impaired glycogen synthesis (Richter et al., 1985) and reduced glucose uptake likely due to muscle damage (Verdi, 2020) and reduced muscle insulin sensitivity. Insulin signaling influences increased blood flow and protein synthesis at rest and suppresses protein breakdown after resistance exercise, improving the net balance of muscle proteins, in particular with the delivery and availability of amino acids (Andreassen et al., 2009; Fujita et al., 2006).

Protein is essential for muscle building and repair. Most of the pleiotropic effects of proteins are mediated by the expression of the target gene (Verdi, 2020; Andreassen et al., 2009). The coding genes, such as the insulin-like growth factor system (IGF-3) are highly sensitive to nutritional status (Fujita el al. 2006). The adaptations induced by exercise are reflected by changes in protein and contractile function, as well as in mitochondrial function, intracellular signaling and transcriptional responses (Fujita el al. 2006; houp et al. 2015). This suggests that nutritional strategies are able to compensate for the anabolic resistance of physical exercise capable of increasing the rates of muscle protein synthesis, and decrease muscle loss during muscle injury or disuse (Sharples et al., 2015; Coffey et al. 2009).

Fatty acids regulate the expression of many genes, in addition to their role as an important oxidative substrate (Cluberton et al., 2005). Studies show that the actions of fatty acids in skeletal muscle have a significant effect on the expression of the gene encoding the decoupling protein 3 (UPC-3). UPC-3 in the muscle plays an important role in homeostasis and oxidation of lipids and carbohydrates (Schrauwen et al., 2001; Bonen et al., 1998). Studies provide strong evidence of the role of fatty acids in the regulation of gene expression (Macdonald, 2001) with a step in the cellular adaptation of skeletal muscles that can contribute to the prevention of injuries (Jump et al., 1999). The relative contribution of these fuels to the energy demands of skeletal muscle is associated with a complex regulation at multiple levels, including availability of substrate, hormonal concentrations, and regulation of enzymatic activities by intracellular metabolic intermediates (Cluberton et al., 2005), in addition to exercise intensity and duration (Schrauwen et al., 2001).

Conclusion

Sport performance is multifactorial, recently several genes (ACE, ACTN3, CCL 2 (C> T), COL5A1, CKM (A> G) have been implicated in various aspects of skeletal muscle remodeling. Individuals with specific genotypes experience changes in the induced muscle damage and recovery rates following exercise. The contribution of heritability to a specific phenotype is likely dependent and specific to the modality, intensity, and duration of exercise. Future research will allow the assessment of multigenetic characteristics which is required to provide a deeper molecular understanding of recovery, adaptation and nutritional modulation that may allow the identification of individuals with a greater genetic predisposition, or with a greater risk of developing muscle injuries or requiring more time to recover.

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