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Review

Skeletal muscle regeneration and impact of aging and nutrition

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ABSTRACT

After skeletal muscle injury a regeneration process takes place to repair muscle. Skeletal muscle recovery is a highly coordinated process involving cross-talk between immune and muscle cells. It is well known that the physiological activities of both immune cells and muscle stem cells decline with advancing age, thereby blunting the capacity of skeletal muscle to regenerate. The age-related reduction in muscle repair efficiency contributes to the development of sarcopenia, one of the most important factors of disability in elderly people. Preserving muscle regeneration capacity may slow the development of this syndrome. In this context, nutrition has drawn much attention: studies have demonstrated that nutrients such as amino acids, *n*-3 polyunsaturated fatty acids, polyphenols and vitamin D can improve skeletal muscle regeneration by targeting key functions of immune cells, muscle cells or both. Here we review the process of skeletal muscle regeneration with a special focus on the cross-talk between immune and muscle cells. We address the effect of aging on immune and skeletal muscle cells involved in muscle regeneration. Finally, the mechanisms of nutrient action on muscle regeneration are described, showing that quality of nutrition may help to preserve the capacity for skeletal muscle regeneration with age.

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1. Introduction

Skeletal muscle is a post-mitotic tissue with a very low turnover rate (Spalding et al., 2005). Even so, muscle has a strong ability to regenerate after injury. Muscle regeneration is a tightly coordinated process composed of four consecutive, interlinked phases: (i) necrosis, (ii) inflammation, (iii) activation and differentiation of satellite cells, and (iv) maturation of newly formed muscle fibers and remodeling of the regenerated muscle (Barberi et al., 2013). After activation and proliferation, satellite cells, i.e., muscle stem cells, return to their quiescent state to maintain a pool of satellite cells ready for the next regeneration process. Alternatively, satellite cells differentiate and merge to form new mature multinucleated muscle cells, i.e., myofibers, or fuse with damaged fibers (Hawke and Garry, 2001).

The regulation of proliferation and differentiation processes in satellite cells depends in part on immune cell function. During muscle regeneration, immune cells that have infiltrated in the lesion develop a pro-inflammatory phenotype. The biological function of the first immune response is to regulate the immune cells themselves, and promote migration of satellite cells to the site of injury and proliferation. In turn, muscle cells, both damaged and intact, modulate the immune response. Neutrophils and pro-inflammatory macrophages are involved in the lysis of damaged muscle cells and the phagocytosis and destruction of cell debris. Pro-inflammatory macrophages are known to stimulate the proliferation of satellite cells. The pro-inflammatory phenotype of immune cells then turns into an anti-inflammatory one. This promotes the differentiation of satellite cells, leading to the formation or the repair of myofibers. The whole process is thus highly regulated, and requires close cross-talk between immune and muscle cells during regeneration (Tidball and Villalta, 2010).

Immunosenescence is characterized by an overall alteration of immune cell functions (Shaw et al., 2013; Vasson et al., 2013). The secretion profile of immune cells is modified, resulting in an increase in circulating pro-inflammatory cytokines such as TNF-α, IL-6 or IL-1β, and leading to the development of chronic low-grade inflammation, called "inflammaging" (Franceschi and Campisi, 2014). Knowing that immune cells are in closed cross-talk with muscle cells during muscle regeneration, immunosenescence may impact the process of muscle repair. Immunosenescence have been extensively and recently reviewed elsewhere (Agarwal and Busse, 2010; Fulop et al., 2011; Montoya-Ortiz, 2013; Panda et al., 2009; Shaw et al., 2013). In this section, we review the process of skeletal muscle regeneration with a special focus on the cross-talk between immune cells and muscle cells. In addition, we point out the main characteristics of age-related change in immune function that may impact skeletal muscle regeneration.

2. Immune and muscle cell cross-talk during muscle regeneration and impact of aging

2.1. Immune cell infiltration in muscle lesion and pro-inflammatory response development

Activation of the complement system following acute muscle injury is involved in the initiation of the inflammatory response during muscle regeneration. The complement system is composed of circulating innate immunity proteins that allow a rapid immune response against an antigen. It has been shown that the complement system is rapidly activated in injured muscle, causing immune cell infiltration in the lesion site (Frenette et al., 2000).

The involvement of mast cells in the initiation of the pro-inflammatory response following muscle damage has also been demonstrated. Mast cells secrete pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α) allowing the recruitment of immune cells at the injury site (Radley and Grounds, 2006).

To our knowledge, very little is known about the changes in mast cell function with advancing age. It has been demonstrated in an ischemia-reperfusion model that the number and volume of mast cells are increased in aged compared with young rats (Harris and Rumbaut, 2001). Mast cells are known to be a source of TNF-α, thereby contributing to the low-grade inflammation observed with aging (Payne, 2006). If the number of mast cells is increased during skeletal muscle regeneration with aging, we hypothesize that the recruitment of immune cells at the lesion site could be altered, impacting the whole process of regeneration.

Besides mast cells, polynuclear neutrophils (also called neutrophils) are among the immune cells that firstly infiltrate muscle lesions. They express lymphocyte antigen 6C (Ly6C) and G (Ly6C) cell surface markers. Neutrophils are also involved in the initiation of pro-inflammatory response following muscle injury (Tidball and Villalta, 2010). The infiltration of these cells is dependent on integrin β2. In rodents, blocking integrin β2 function by targeting one of its subunits (CD11b or CD18) before muscle lesion prevents neutrophil infiltration at the site of injury (Pizza et al., 2005; Zerria et al., 2006). Neutrophils phagocyte cellular debris and secrete enzymes such as myeloperoxidase (MPO), cytokines or reactive oxygen species, which contribute to the lysis of damaged muscle fibers and the aggravation of muscle injury (Kharraz et al., 2013; Nguyen and Tidball, 2003b). The inhibition of neutrophil function results in the preservation of muscle fibers close to the injury site (Nguyen and Tidball, 2003b; Zerria et al., 2006). Although neutrophils are involved in the lysis of muscle fibers and in the phagocytosis of cell debris, their presence is required for an optimal muscle regeneration process. Hence injection of a myotoxin in the muscle of neutrophil-depleted mice results in an incomplete muscle recovery and the persistence of necrotic fibers (Teixeira et al., 2003).

With aging, a clinical study has reported that the circulating neutrophil count remains unchanged (Chatta et al., 1994), while it has been demonstrated more recently that a small decrease in neutrophil number occurs with aging (De Martinis et al., 2004). These conflicting results can be explained by the difference in the age ranges of the subjects included in the two studies: 70–80 versus 80–100 years old (Chatta et al., 1994; De Martinis et al., 2004). Among the main functions of this cell type, chemotactism, bactericidy and phagocytosis are blunted with age (Niwa et al., 1989; Wenisch et al., 2000). Thus, if the number of circulating neutrophils is modified with aging, jointly with phagocytosis and chemotactism, we suppose that the initiation of the pro-inflammatory response following muscle injury could be reduced, affecting skeletal muscle repair.

It should be noted that the implication of complement system, mast cells and polynuclear neutrophils in the initiation of immune response were demonstrated in rodent models. Effect of aging on mast cell functions implicated in muscle repair was also highlighted in animal models. Thus investigations are needed to confirm that

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