

Asymmetric/Symmetric Division Switching of Skeletal Muscle Stem Cells in Aging

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Introduction

Satellite cell symmetric and asymmetric divisions are impaired in aged muscle, and these processes are governed by intrinsic and extrinsic complex mechanisms. This review covers many new developments in the fate of muscle stem cells in normal and ageing conditions. The balance of self-renewal and commitment divisions helps with muscle regeneration, homeostasis, ageing, and disease. Stimulating muscle regeneration in ageing could be a therapeutic target, but more research is needed to understand the numerous mechanisms that influence one another in satellite cells and their niche. We focus on the broad strokes of satellite cell divisions, the primary markers found in muscle stem cells, the ageing aspects of signalling pathways involved in symmetric/asymmetric divisions, the regenerative capacity of satellite cells and their niche alteration in senescent muscle, and genetics [1].

Description

Satellite cells, which are skeletal muscle resident stem cells, are in charge of muscle growth and repair. SCs are dormant in healthy muscle, but when activated, proliferate, and give rise to myoblast progenitors, which differentiate and fuse into multinucleated muscle fibres in response to injury or other stimuli. The asymmetric division of a SC results in the formation of one stem cell that remains in contact with the basal lamina and one differentiation competent progenitor that migrates toward and contacts the myofiber. Symmetric divisions are required for stem cell self-renewal, which replenishes the SC pool, as well as myoblast proliferation, which results in committed daughter cells for muscle regeneration [2].

The study of the asymmetric SC niche has become important due to the need to identify the factors that determine cell apicobasal polarity and orientation. Asymmetric fate is associated with apico-basal divisions perpendicular to the basal lamina. Notch3 and NUMB (biochemical regulators) as well as the PAR complex are some of the process's regulators (orients the mitotic spindle pole). Furthermore, a recent study using the zebrafish larval system demonstrated that this process occurs in vivo during muscle regeneration to generate the clonally related proliferative myoblast population required for muscle repair. WNT7a regulates the symmetric divisions, which occur in a planar orientation [3].

The ageing process involves several intrinsic and extrinsic mechanisms that influence SC behaviour. The role of muscle stem cells in sarcopenia

is still being debated. The SC pool is known to diminish with age, but a fraction survives until very old age, even with functional defects that affect their regenerative ability. Extrinsic changes appear first at the level of the environment; later, intrinsic changes accumulate (genomic and metabolic impairment), reducing the ability of the SC to activate after injury. As a result, fibrous tissue growth replaces the lost muscle fibres. Furthermore, Arpke et al. demonstrated in a comparative study on eight muscle groups in young and old mice that, unlike previous studies conducted only on locomotor muscles, self-renewal impairment with age is primarily acquired in the skeletal muscles.

The number of satellite cells decreases throughout life, resulting in functional depreciation of skeletal muscle in the elderly. Their regenerative potential is compromised, as is the maintenance of the stem cell reservoir and the production of myoblasts; however, the precise mechanism is still unknown. SC depletion causes severe muscle damage and a reduced ability to regenerate, according to mouse experiments. Many factors are implicated in the altered division pattern, including repeated regeneration, cellular stress, inflammation, extracellular matrix changes, and stem cell niche changes. Shefer et al., on the other hand, proposed that while SCs' myogenic potential is maintained with age, their population is not fully replenished throughout life [4,5].

Conclusion

Healthy skeletal muscles contribute significantly to the quality of life by allowing for independent movement and locomotion. Sedentism and muscle ageing are known to be the root causes of the emergence of very serious pathologies such as obesity and cardiovascular disease. Satellite cells play an important role in maintaining muscle mass until an advanced age in this context. Many SC mechanisms decline with age, but not all are known; some remain unknown. Given the complexity of the intrinsic and extrinsic biological processes that govern the symmetric vs. asymmetric division of SCs, as discussed in this evaluation, it is critical to keep up with the most recent research in this field. It would be interesting to investigate the behaviour of satellite cells in older people in the future.

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