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Nestin is a member of the fourth (IV) sequence homology class (SHC) of intermediate filament (IF) cytoskeletal proteins. Nestin expression has been reported in developing muscle, regenerating adult skeletal muscle, and in differentiating myoblasts. The satellite cells of the skeletal muscle are precursor cells that are essential in the process of skeletal muscle regeneration, repair, and hypertrophy in vertebrates. Transcription factors Pax7 and MyoD in turn control satellite cell activity during regeneration. Here, the role of nestin in muscle cells was investigated by studying the behavior of satellite cell proliferation from nestin knockout and wildtype control mice. To characterize the proliferation and behavior of satellite cells of nestin deficient mice, immuno-fluorescence labeling of the myogenic transcription factors (TFs) expressed in satellite cells and live cell imaging studies were conducted on cultured skeletal myofibers isolated from the *extensor digitorum longus* (EDL) muscles of both nestin deficient and wildtype control mice. In addition, the effect of the lack of the gene encoding nestin on the lean and fat tissue masses, as well as voluntary running performance, were investigated using *in vivo* body composition analysis and voluntary running wheel tests, respectively. Here I report that no significant differences in differentiation or in proliferation pattern of satellite cells could be observed between nestin knockout and wildtype mice. Similarly, no statistical differences in the whole body masses of fat and lean tissues and exercise performances were observed in the mice investigated here. Therefore, the deletion of the gene encoding nestin does not appear to affect the proliferation behavior of myoblasts derived from explanted myofibers, whole body masses of fat and lean tissues or muscle running performance of the mice.

KEYWORDS: Nestin, Skeletal Muscles, Satellite cells, Pax7, MyoD