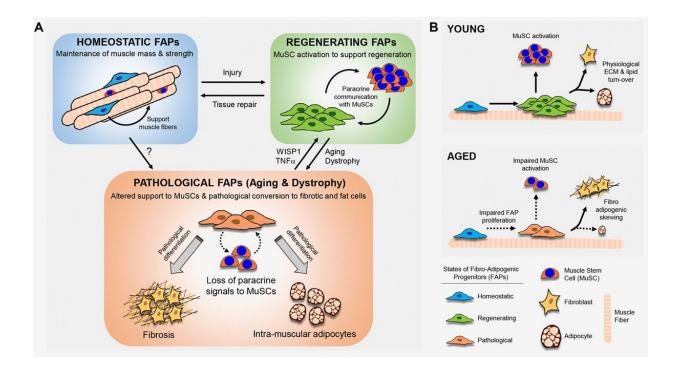


Adipogenic progenitors keep muscle stem cells young

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Fibro-adipogenic progenitors (FAPs) dynamically cross-talk with the muscle stem cell (MuSC) niche to regulate regeneration and ECM/lipid turnover in different patho-physiological cues. (A) FAPs have distinct cellular fates and functions in different patho-physiological conditions; support to muscle fibers in homeostatic conditions [5]; support to muscle stem cells during regeneration [2]; pathological differentiation to fat and fibrosis [4,6]. (B) Aging alters the support of FAPs to MuSCs and regeneration and promotes their pathological skewing to fibrosis over adipogenesis [6]. Credit: Jerome N.



In adult skeletal muscle, loss of myofiber integrity caused by mechanical injuries or diseases are repaired by resident muscle stem cells, called satellite cells, which promptly exit from quiescence after disruption of muscle architecture to expand, differentiate and drive tissue regeneration.

Dr. Jerome N. Feige from Nestlé Research, EPFL Innovation Park, Lausanne, Switzerland said, "Fibro/adipogenic progenitors constitute a population of interstitial mesenchymal <u>cells</u> in skeletal muscle which are devoid of myogenic potential, but support muscle stem cell commitment and can differentiate to the adipogenic or fibrotic lineages."

Thus, FAPs are active regulators of cellular communication in skeletal muscle niche where they directly control tissue homeostasis and regeneration by supporting Mu SCs and myofibers.

In a recent study, the author's lab investigated how aging influences the fate of FAPs and their cross-talk with Mu SCs to regulate the balance between myogenesis, adipogenesis and fibrosis in skeletal muscle.

Interestingly, aged FAPs fail to efficiently amplify following <u>muscle</u> injury and aging alters the capacity of FAPs to support Mu SC amplification and commitment.

Both in-vitro co-culture and in-vivo transplantation of young FAPs rejuvenate aged Mu SC function, but aged FAPs lose the ability to efficiently support Mu SCs.

The Feige Research team concluded, "FAPs are also likely a heterogeneous population and the clonal selection of different fates of FAPs during aging suggests a differential effect of age on distinct subpopulations.



While Tie2- expressing FAPs predominantly reside within neonatal and adult homeostatic muscles, another injury-activated subpopulation of FAPs characterized by Vcam1 expression is associated with regeneration of injured myofibers."

More information: Sara Ancel et al, Adipogenic progenitors keep muscle stem cells young, *Aging* (2019). DOI: 10.18632/aging.102304

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