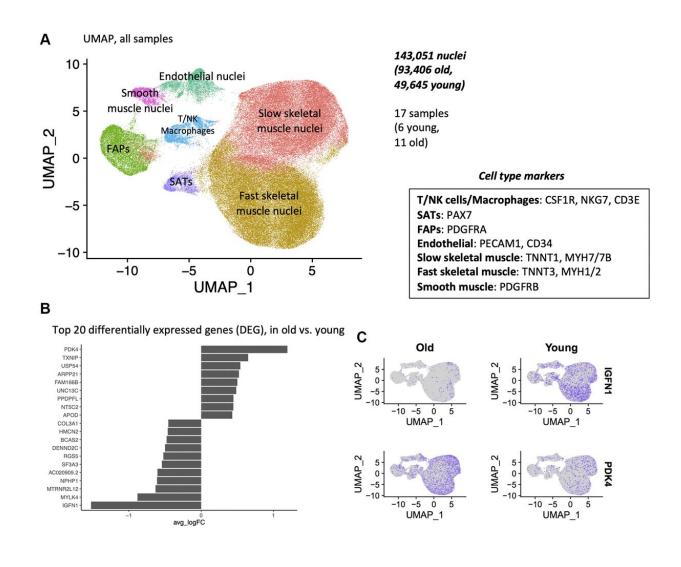


Single nuclei profiling identifies cell specific markers of skeletal muscle aging, frailty, and senescence

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Single-nuclei sequencing reveals 7 clusters of unique cell types, and differential gene expression with age. Credit: *Aging* (2022). DOI: 10.18632/aging.204435



A new research paper was published on the cover of *Aging* entitled "Single nuclei profiling identifies cell specific markers of skeletal muscle aging, frailty, and senescence."

Aging is accompanied by a loss of muscle mass and function, termed sarcopenia, which causes numerous morbidities and economic burdens in human populations. Mechanisms implicated in age-related sarcopenia or frailty include inflammation, muscle stem cell depletion, mitochondrial dysfunction, and loss of motor neurons, but whether there are key drivers of sarcopenia are not yet known.

In this new study, researchers performed transcriptome profiling on lower limb muscle biopsies from 72 young, elderly, and frail human subjects using bulk RNA-seq (N = 72) and single-nuclei RNA-seq (N = 17) to gain deeper insights into age-related muscle loss.

"This combined approach revealed changes in <u>gene expression</u> that occur with age and frailty in multiple cell types comprising mature skeletal muscle," they report.

Notably, the researchers found increased expression of the genes MYH8 and PDK4, and decreased expression of the gene IGFN1, in aged muscle. They validated several key genes changes in fixed human muscle tissue using digital spatial profiling. The researchers also identified a small population of nuclei that express CDKN1A, present only in aged samples, consistent with p21cip1-driven senescence in this subpopulation.

"Overall, our findings identify unique cellular subpopulations in aged and sarcopenic skeletal muscle, which will facilitate the development of new therapeutic strategies to combat age-related frailty," they conclude.

More information: Kevin Perez et al, Single nuclei profiling identifies



cell specific markers of skeletal muscle aging, frailty, and senescence, *Aging* (2022). DOI: 10.18632/aging.204435

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