

# HOW MUSCLES AGE

Sarcopenia, the loss of muscle mass with age, can start as early as one's 30s, and affects a large proportion of the elderly. Fortunately, exercise can combat muscle aging, likely by reversing many of the age-related physiological changes at the root of this decline.

YOUNG  
MUSCLE

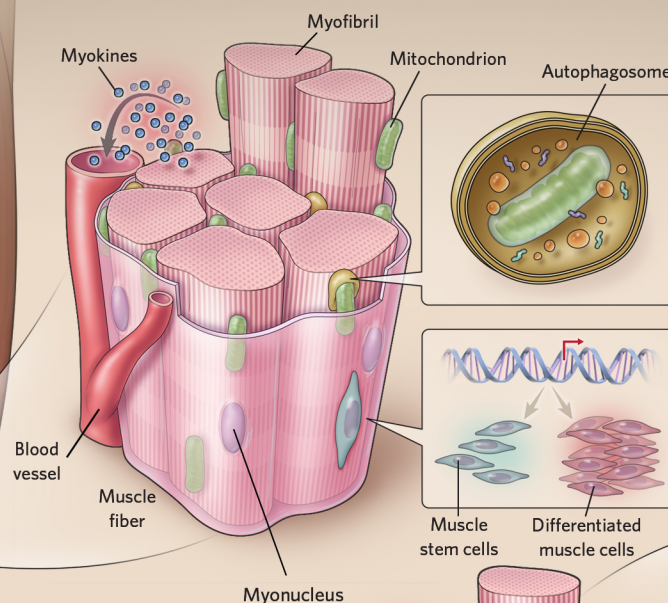
## BLOOD-BORNE FACTORS

Signaling factors known as myokines can be released into the blood directly or through excreted vesicles, and travel through the circulatory system to coordinate muscle physiology and repair. For example, apelin, which decreases with age, boosts the formation of new mitochondria, stimulates protein synthesis and autophagy, and supports the function of muscle stem cells.

OLD  
MUSCLE

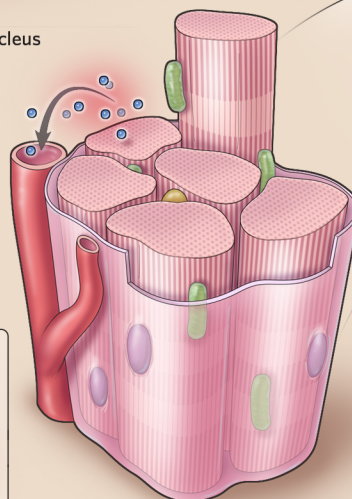
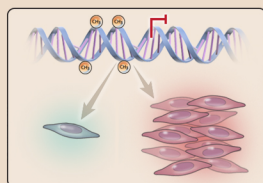
## MAINTAINING PROTEIN BALANCE

Old muscles undergo lower levels of autophagy. Combined with lower protein production, this can result in an imbalance of proteins linked to muscle aging.



## MUSCLE STEM CELLS

Muscle stem cells, or satellite cells, decrease in number as we age. In elderly-human cells DNA methylation suppresses the expression of some genes, including *sprouty 1*, an important regulator of satellite cell self-renewal.



## MITOCHONDRIA

Muscles develop abnormalities in mitochondrial morphology, number, and function with age.

**EXERCISE:** A sedentary lifestyle can induce molecular processes of muscle aging, such as decreases in the efficiency and number of mitochondria. Conversely, exercise reverses a gene expression profile consistent with mitochondrial dysfunction and restores levels of mitochondrial proteins. Exercise also increases autophagy levels and restores levels of myokines involved in muscle function.