In the first *Highlighted Topics* article featured in this issue of the *Journal of Applied Physiology*, “Aging impairs nitric oxide and prostacyclin mediation of endothelium-dependent dilation in soleus feed arteries,” Woodman et al. assessed the hypothesis that aging impairs endothelium-dependent vasodilation of feed arteries in rat soleus muscle. Such feed arteries play an integral role in the control of blood flow to this muscle during exercise. With age, the ability to increase soleus muscle blood flow during exercise declines. The primary finding of this study was that endothelium-dependent vasodilation, stimulated by increases in intraluminal flow and acetylcholine, was impaired in aged rats. In addition, these results indicated that the age-related decrement in endothelium-dependent vasodilation was due to impaired release of nitric oxide and prostacyclin by the endothelium in senescent arteries. An improved understanding of the cellular mechanisms that account for age-induced endothelial dysfunction may offer therapeutic approaches to attenuate or reverse the detrimental effects of age on skeletal muscle blood flow.

In the second article featured in this issue, “Identification of differentially expressed genes between young and old rat soleus muscle during recovery from immobilization-induced atrophy,” Pattison et al. immobilized hindlimbs of young and old rats to cause atrophy and then assessed age-related variations in gene expression. The investigators found that, whereas skeletal muscle from older rats exhibited little to no regrowth after 10 days of hindlimb immobilization, muscles of young rats returned to the precontrol size by 30 days postimmobilization. To assess candidate genes potentially responsible for the defective regrowth following atrophy in the aged rats, the investigators employed oligonucleotide microarrays, assaying 24,000 transcripts. They found that, during recovery from immobilization, young and old rats expressed 64 mRNAs differently. Real-time PCR confirmed 3 of these 64 candidates: Elfin, amphiregulin, and clusterin. Determination of the many genes playing roles in sarcopenia is essential to the development of future therapies that will offer hope to those confined to nursing homes because of physical frailty.

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