

**Age-Related Changes in Skeletal Muscle Oxygen Utilization: A Review**

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**ABSTRACT**

The cardiovascular and skeletal muscle systems are intrinsically interconnected, sharing the goal of delivering oxygen to metabolically active tissue. Deficiencies within those systems that affect oxygen delivery to working tissues are a hallmark of advancing age. Oxygen delivery and utilization are reflected as muscle oxygen saturation (SmO<sub>2</sub>) and may be assessed using near-infrared resonance spectroscopy (NIRS). **PURPOSE:** This review is intended to provide an update on the current state of literature regarding age-related effects on SmO<sub>2</sub>. Furthermore, we attempt to bridge the gap between SmO<sub>2</sub> and associated underlying mechanisms affected by aging. **FINDINGS:** SmO<sub>2</sub> has been observed to be reduced by ~38% at rest, ~24% during submaximal exercise, and ~59% during maximal exercise with aging (>65 y). Furthermore, aging prolongs restoration time of SmO<sub>2</sub> back to baseline by >50% after intense exercise. Regulatory factors that contribute to reduced SmO<sub>2</sub> with age include blood flow, capillarization, endothelial cell function, nitric oxide, and mitochondrial function. These mechanisms are generally governed by reactive oxygen species (ROS) at the cellular level. However, mishandling of ROS with age ultimately leads to alterations in structure and function of the regulatory factors tasked with maintaining SmO<sub>2</sub>. **CONCLUSION:** Aging reduces SmO<sub>2</sub> at rest, during submaximal/maximal exercise, and extends restoration timeframe of SmO<sub>2</sub> following exercise. Compelling data suggests dysregulated ROS handling leads to reductions observed in O<sub>2</sub> delivery & utilization.