Rapid vasodilation induced by mechanical factors in rat skeletal muscle feed arteries and arterioles

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During exercise, cardiac output redistributes to active skeletal muscles from inactive organs and to active motor units from inactive motor units within skeletal muscle. The vasomotor tone of arterioles and feed arteries plays an essential role in this redistribution of blood flow. Both vasoconstrictor and vasodilator factors work to regulate the vasomotor tone. This study focuses on the rapid vasodilation induced by mechanical compression of a soleus feed artery located outside the skeletal muscle and on the flow-induced dilation in 2A arterioles within the cremaster muscle in rats. Mechanical compression by extravascular pressure (80 mmHg) was delivered for 1 s to a soleus feed artery. Release of the pressure immediately resulted in significant vasodilation. The magnitude of vasodilation was increased in feed arteries isolated from voluntary wheel running rats. Flow-induced dilation during parallel occlusion of the arterioles of the rat cremaster muscle was examined. The arterioles dilated with a delay time of 8.8 ± 1.1 s after the onset of occlusion. Increases in diameter, WSR, velocity, and flow were significant from baseline with delay time. These results suggest that mechanical dilation of the feed arteries located outside skeletal muscle and flow-induced dilation of 2A arterioles within the skeletal muscle contribute to the rapid onset of exercise hyperemia.

Key words: mechanical vasodilation, flow-induced dilation, feed artery, 2A arterioles, exercise hyperemia

Exercise-induced angiogenesis in skeletal muscle

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Angiogenesis, the formation of new blood vessels from preexisting capillary network, is an essential process in improving endurance capability as well as in embryonic development, wound healing, and cardiovascular diseases. In 1930s, increased muscle capillarity was reported after endurance exercise training in animals. Many factors that facilitate or inhibit angiogenesis have been hitherto identified. Exercise-induced angiogenesis may involve sprouting and intussusceptive vessel growth and resulted from the coordinated responses of numerous factors and stimuli.

Vascular endothelial growth factor (VEGF) is thought to be an important regulator of capillary growth during exercise training. Expression of VEGF is increased in response to tissue hypoxia by hypoxia inducible factor (HIF) α subunits. PGC-1 and interleukin-6 are also reported to stimulate VEGF expression. Endurance exercise was shown to enhance anti-angiogenic factors, such as thrombospondin-1 and endostatin.

miRNAs may be important regulator of exercise-induced angiogenesis. miR20b expression markedly declined after acute exercise and exercise training. miR20b was shown to inhibit VEGF translation and HIFα accumulation.

Further studies are needed to clarify whether and how miRNA, undifferentiated progenitor cells, and extracellular remodelling participate in exercise-induced capillary growth.