Public Abstract
First Name:Curt
Middle Name:
Last Name:Canine
Adviser's First Name:Steven
Adviser's Last Name:Segal
Co-Adviser's First Name:
Co-Adviser's Last Name:
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Title:The Control of Blood Flow in Skeletal Muscle Arterioles

Skeletal muscle arterioles dilate in response to application of acetylcholine (ACh), eliciting a conducted vasodilation (CVD) that travels along unbranched segments without decrement. CVD is known to entail cell-to-cell transmission of hyperpolarization along the endothelium via gap junction channels, a purely passive mechanism. In the present thesis I study CVD in bifurcating arteriolar networks, where the pathway for hyperpolarizing current expands compared to unbranched arterioles, to test for an active component to CVD. In a separate subset of arterioles, the effect of augmenting vasomotor tone on CVD was tested using elevated O2 or phenylephrine (PE) in the superfusion solution vs. control. Male C57BL/6 mice (n=13; 10-13 weeks old) were anesthetized with pentobarbital sodium (50mg/kg, intraperitoneal injection) and maintained at 37 °C. The cremaster was carefully exteriorized and spread onto a transparent Sylgard pedestal. The tissue was maintained at 34 °C with continuous superfusion of physiologic saline solution. Microiontophoresis of ACh evoked non-decremental CVD in both unbranched and bifurcating arterioles, supporting the role of an active component in CVD. Further, augmenting vasomotor tone with PE attenuated non-decremental CVD, whereas increasing vasomotor tone with elevated O_2 did not alter CVD. In summary, arteriolar networks in the mouse cremaster muscle exhibit robust dilation to ACh, which conducts along arterioles and across branch points without decrement, suggesting the contribution of both active and passive mechanisms. In preparations exhibiting poor spontaneous vasomotor tone, elevated O_2 can be used to improve resting tone without impacting CVD.