Ca2+, the intercellular signal in arterioles

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Vasoconstriction must be balanced with vasodilation, particularly in the arterioles that supply tissues with blood. Endothelial cells protrude through holes in the internal elastic lamina in arterioles to make contact with vascular smooth muscle cells.

Gap junctions are present at these sites where endothelial cells meet vascular smooth muscle cells. IP3 has been thought to be a signal that passes through these gap junctions to endothelial cells to mediate vasodilation.

However, Garland et al. showed that it was Ca2+, rather than IP3, that entered vascular smooth muscle cells through voltage-gated Ca2+ channels, subsequently passed through gap junctions into endothelial cells, and initiated vasodilation mediated by endothelial cells.

The magnitude of these Ca2+ signals in endothelial cells depended on IP3 receptors.

These results resolve a long-standing controversy over how vascular smooth muscle cells communicate with endothelial cells to trigger feedback vasodilation.

The study results are published in *Science Signaling*.

More information: "Voltage-dependent Ca2+ entry into smooth muscle during contraction promotes endothelium-mediated feedback vasodilation in arterioles," *Science Signaling* (2017).