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What the Eph(/ephrin) is going on in regenerating muscle vasculature.

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Blood vessels are like a tiny highway, acting to deliver oxygen and nutrients throughout the body. The branching of blood vessels is vital to embryonic muscle development; but how vital are they for adult muscle repair? Muscles are made up of myofibers that communicate with each other and their environment through signaling molecules. One set of signaling molecules is a family of receptor tyrosine kinases called Ephs and their ligands, ephrins. We study a specific Ephrin, EphrinB2, due to its established role in vasculogenesis during embryo development. In the absence of ephrinB2 embryos fail to develop functioning capillaries, resulting in embryo death. By removing ephrin-B2 expression only in vascular cells in adult animals, we are investigating the role of ephrinB2 in vascular regeneration, following a local injury. By scoring molecular landmarks such as embryonic myosin heavy chain (eMyHC, a marker of newly made muscle fibers) from at various time points after injury, we have compared muscle regeneration in ephrin-B2 knockout mice to what occurs in wild type mice. We found that in these mice, not only is vascular regeneration impaired but muscle regeneration was slowed significantly. We are now asking the opposite question: whether vasculature will regenerate in the absence of muscle. To accomplish this, we are using genetic methods to kill muscle stem cells (satellite cells) in adult mice. Without satellite cells muscle will not regenerate following injury. We hypothesize that the vasculature will be able to regenerate in the absence of musculature. We believe the vasculature will have a “memory” of where it was localized prior to injury, possibly due to interactions with the extracellular matrix, and thus will regrow in the same pattern/location even without muscle fibers. These experiments address basic questions in cell biology and may someday be useful in designing treatments for human injury.