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mice, truncated Notch receptors lacking the intracellular domain can induce blood vessels (Wang et al. 2013, Nature Commun. 4:2581). This observation suggests that Notch may in addition to CSL transcription factor also mediate blood vessel maturation through a Dll-independent mechanism. We hypothesize that different Notch ligands may be responsible for different functions in blood vessel development. In the current study, we will address this hypothesis by selectively perturbing Notch signaling activity in the endothelial cells via gain- and loss-of-function analyses of Delta c6a93da74d

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