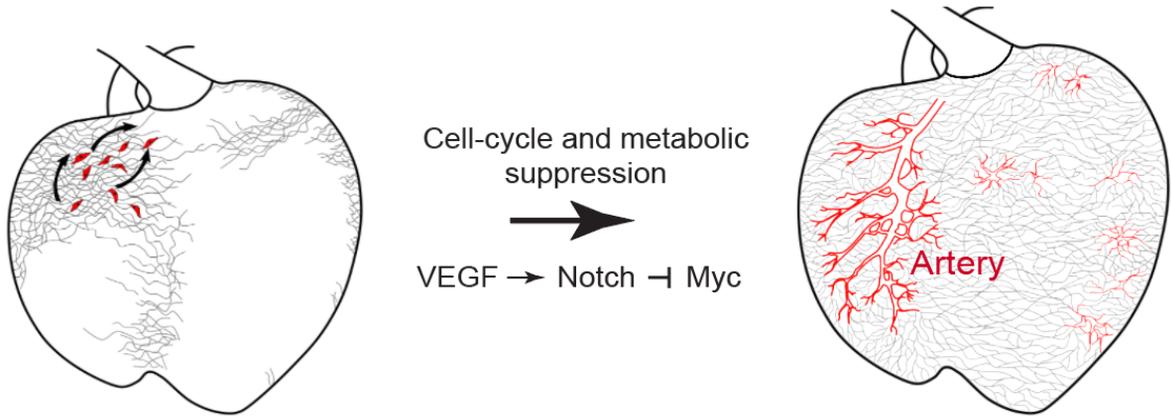


Arterialization requires the timely suppression of cell growth.

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Key findings

1. Multispectral genetic mosaics were used to map the expansion and arteriovenous fate of cells with normal or altered Notch and VEGF signalling.
2. Endothelial cells with high VEGF or Notch signalling are biased but not genetically pre-determined, and can form both arteries and veins.
3. VEGF and Notch favour the incorporation of endothelial cells into arteries by suppressing MYC-dependent metabolic and cell-cycle activities.
4. Endothelial cells lacking the Notch–RBPJ transcriptional complex regained the normal ability to form arteries when MYC function was suppressed.
5. The new mechanistic insights may enable better induction of arterialization during tissue growth, regeneration, or ischaemic cardiovascular disease.

Links to website/social media:

<https://www.cnio.es/en/investigacion/molecular-genetics-angiogenesis>

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