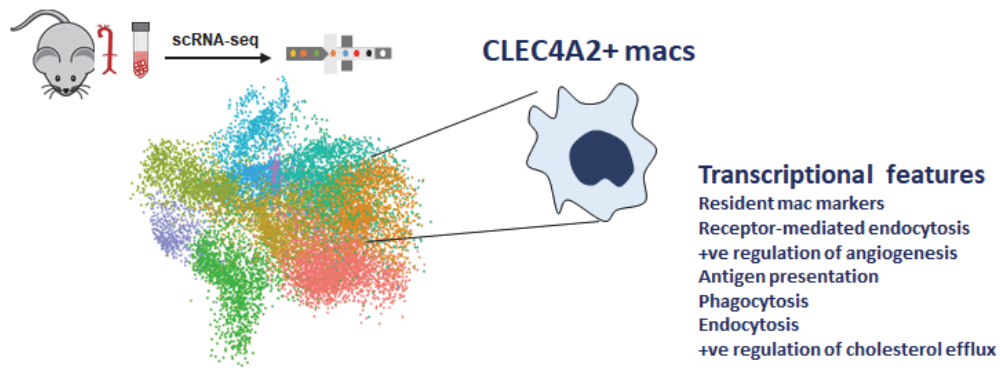
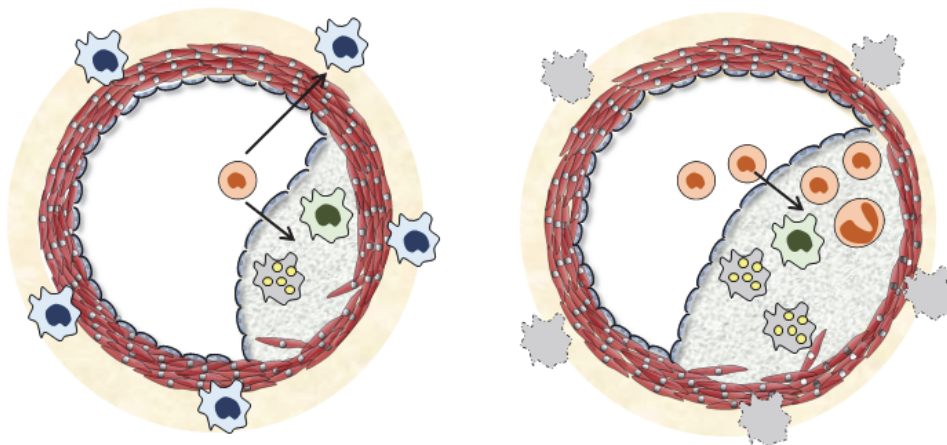


C-type lectin receptor CLEC4A2 promotes tissue adaptation of macrophages and protects against atherosclerosis. Inhye Park, Michael E Goddard, Jennifer E Cole, Natacha Zanin, Leo-Pekka Lyytikäinen, Terho Lehtimäki, Evangelos Andreacos, Marc Feldmann, Irina Udalova, Ignat Drozdov, Claudia Monaco. *Nat Commun.* 2022 Jan 11;13(1):215. doi: 10.1038/s41467-021-27862-9.

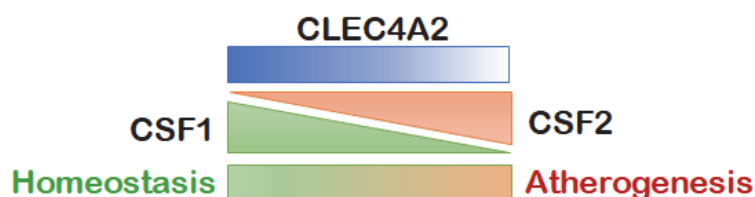
Identification of CLEC4A2+ resident vascular macrophages via scRNASeq



Loss of CLEC4A2 and DT-mediated ablation of CLEC4A2+ macs enhances atherosclerosis



CLEC4A2 licences monocytes to join the resident vascular mac pool by biasing monocyte-to-macrophage differentiation towards colony stimulating factor 1 (CSF1)



Key findings:

- This study identifies CLEC4A2+ resident vascular macrophages via scRNASeq and investigates their role in the pathogenesis of atherosclerosis.
- Loss of CLEC4A2 and diphtheria-toxin -mediated ablation of CLEC4A2+ macs enhances atherosclerosis.
- CLEC4A2 protects from atherosclerosis by limiting TLR signaling and promoting cholesterol efflux in vascular macrophages.
- CLEC4A2 as an intrinsic regulator of macrophage tissue adaptation which licenses monocytes to join the resident vascular mac pool by biasing monocyte-to-macrophage differentiation towards colony stimulating factor 1 (CSF1)
- Fostering the homeostatic functions of macrophages through the CLEC4A receptor could represent a new therapeutic strategy to combat cardiovascular disease.