Plaque stabilizing and destabilizing effects in atherosclerosis

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The role of microvessels, macrophage metabolism and fibroblasts

Propositions

1. Deletion of the PDGF-B retention motif does not affect atherosclerotic microvessel density or integrity | *This thesis*
2. Deletion of the PDGF-B retention motif ameliorates plaque stability, while simultaneously driving extramedullary hematopoiesis and subsequent leukocytosis |*This thesis*
3. Partial myeloid PFKFB3 inhibition does not affect circulating lipids and plaque stability, size or burden in *Ldlr*$^{-/-}$ mice | *This thesis*
4. Myeloid *Pfkfb3* expression protects against fatty liver disease, which potentially poses an opportunity for future therapeutic therapies | *This thesis*
5. PDGFRα and DPEP1 are suitable markers for the identification of adventitial fibroblasts in healthy vascular beds | *This thesis*
6. Healthy aortic adventitia harbors three fibroblast subsets, characterized by divergent markers (CD55, CXCL14 and LOX), the proportions of which are differentially regulated by CVD risk factors aging and mild dyslipidemia | *This thesis*
7. Future treatment of atherosclerosis and fatty liver disease will benefit from the development of cell-targeted therapies
8. The field of atherosclerosis needs to reach consensus on the markers and nomenclature applied during annotation of cell clusters obtained through single-cell RNA-sequencing analysis
9. Questioning the prevailing paradigm is pivotal for scientific progress
10. Unexpected results are the rule rather than the exception, and form the sparks that ignite new avenues of exploration
11. A river carves its path through solid rock not solely due to its sheer force, but rather because of its relentless determination and persistence

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