

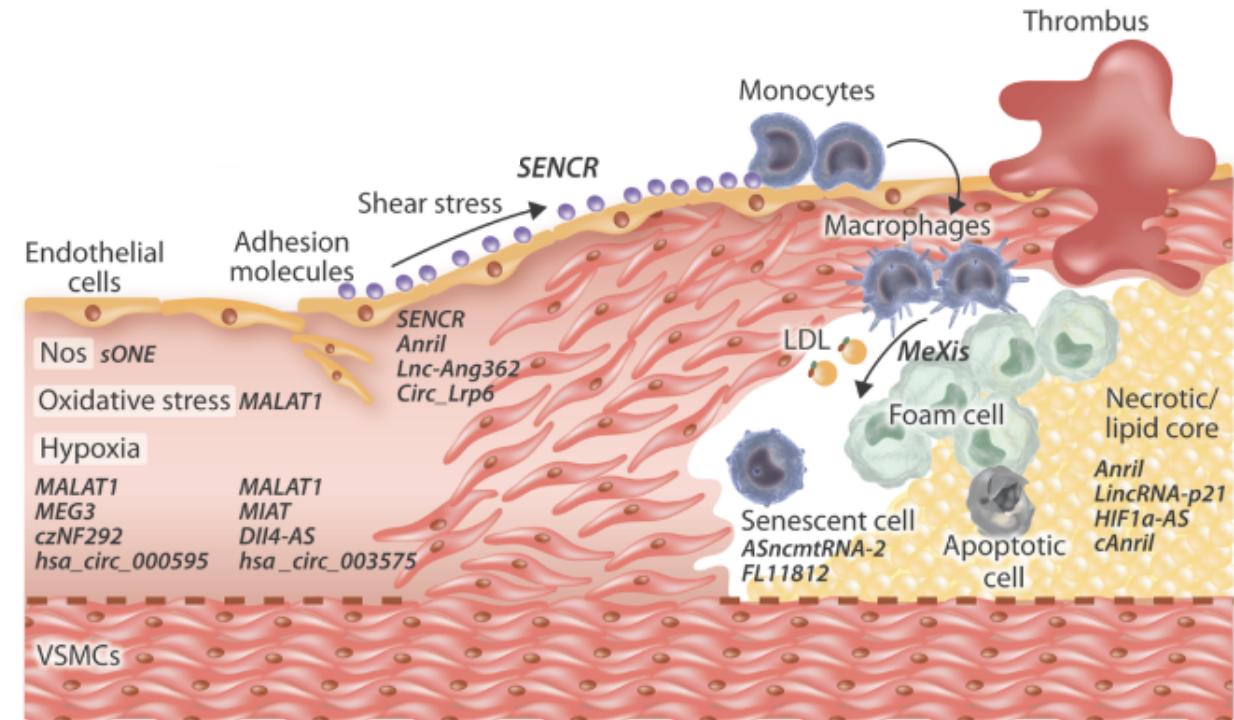
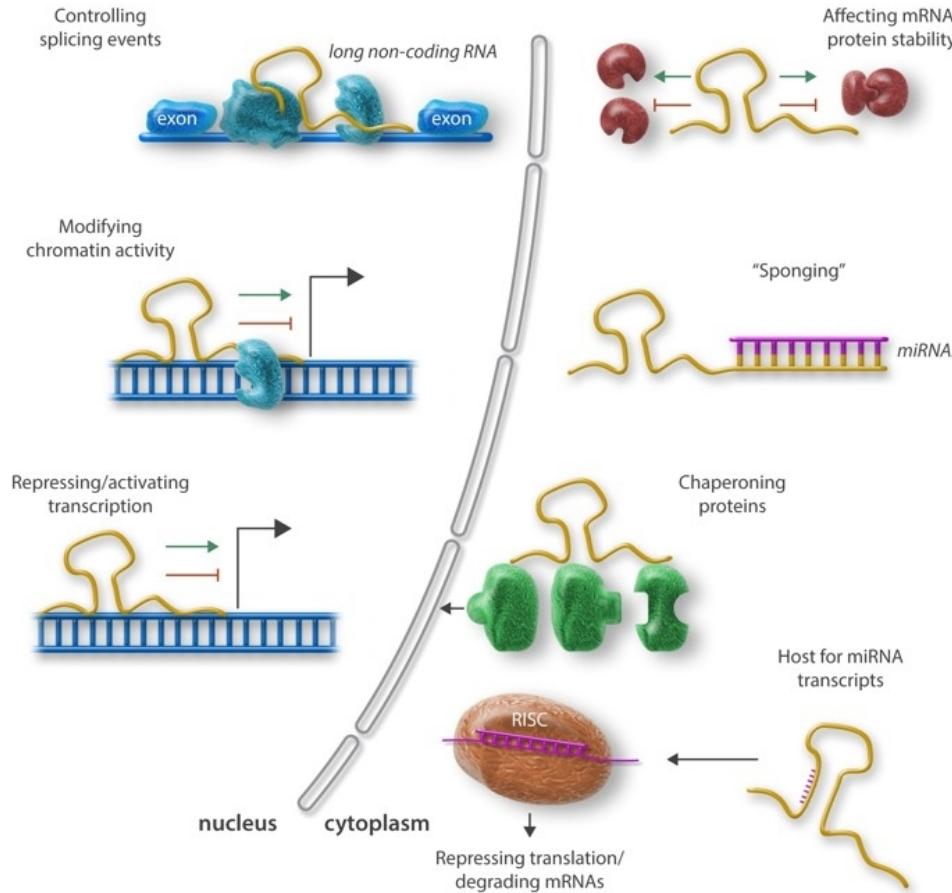
LncRNAs in atherosclerosis and plaque (de)stabilization

Lars Maegdefessel

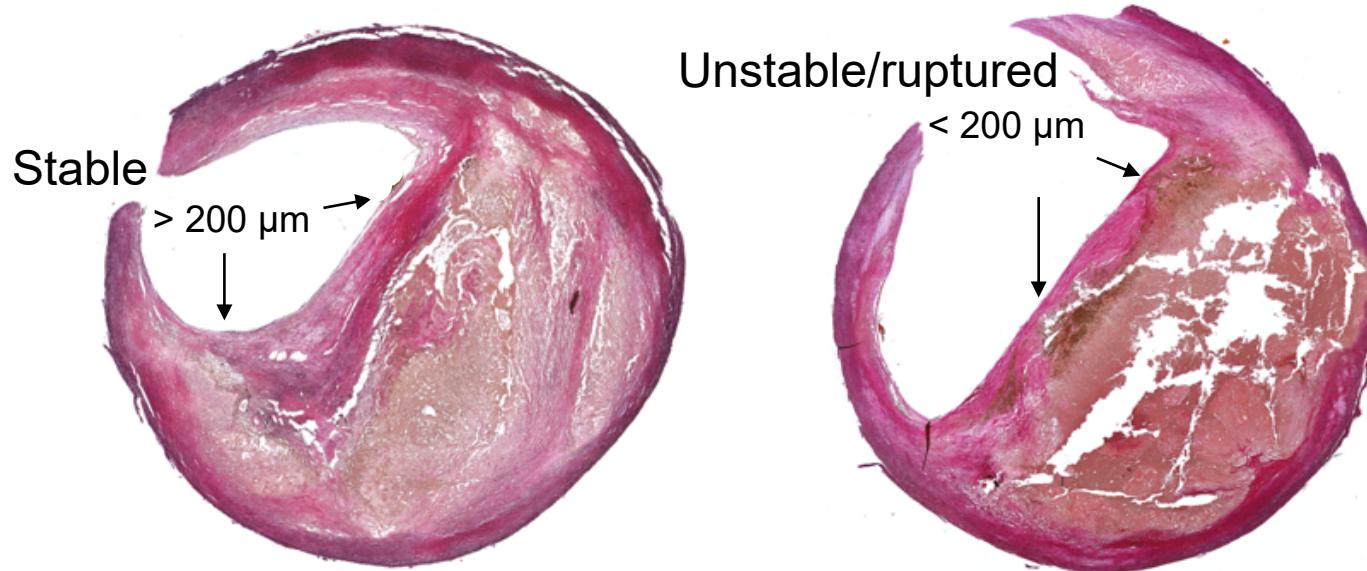
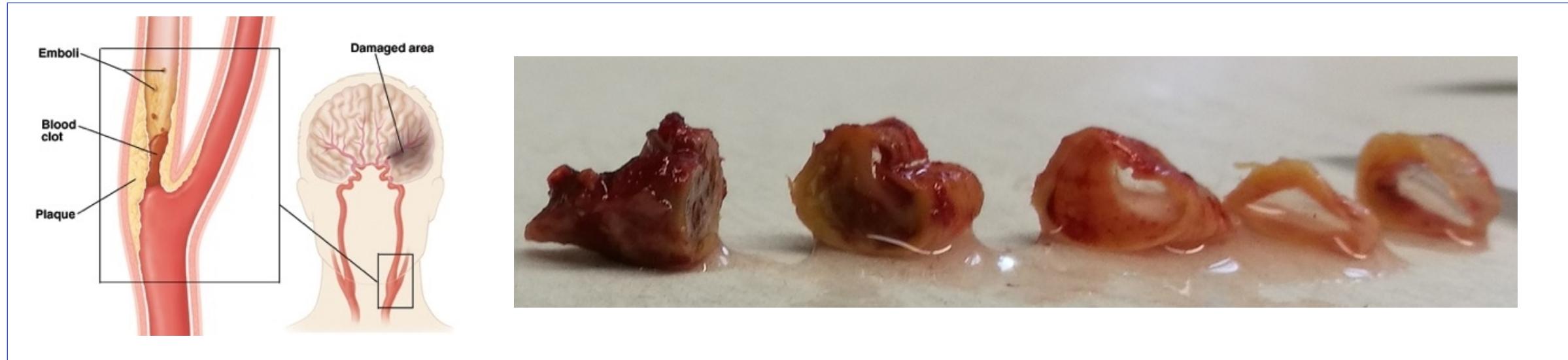
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Long non-coding RNAs in atherosclerosis



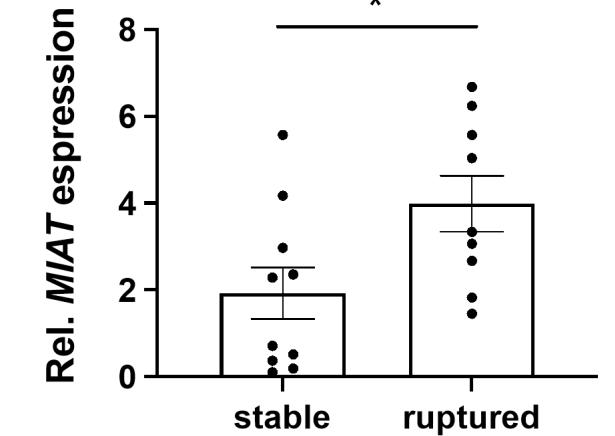
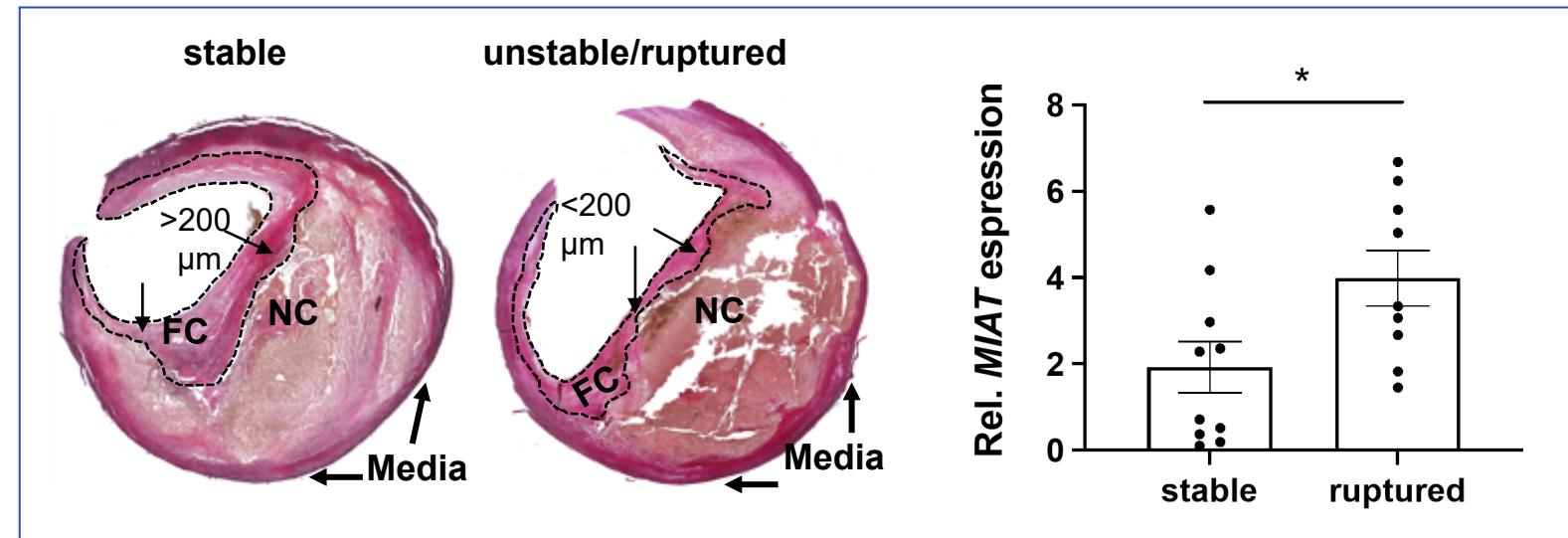
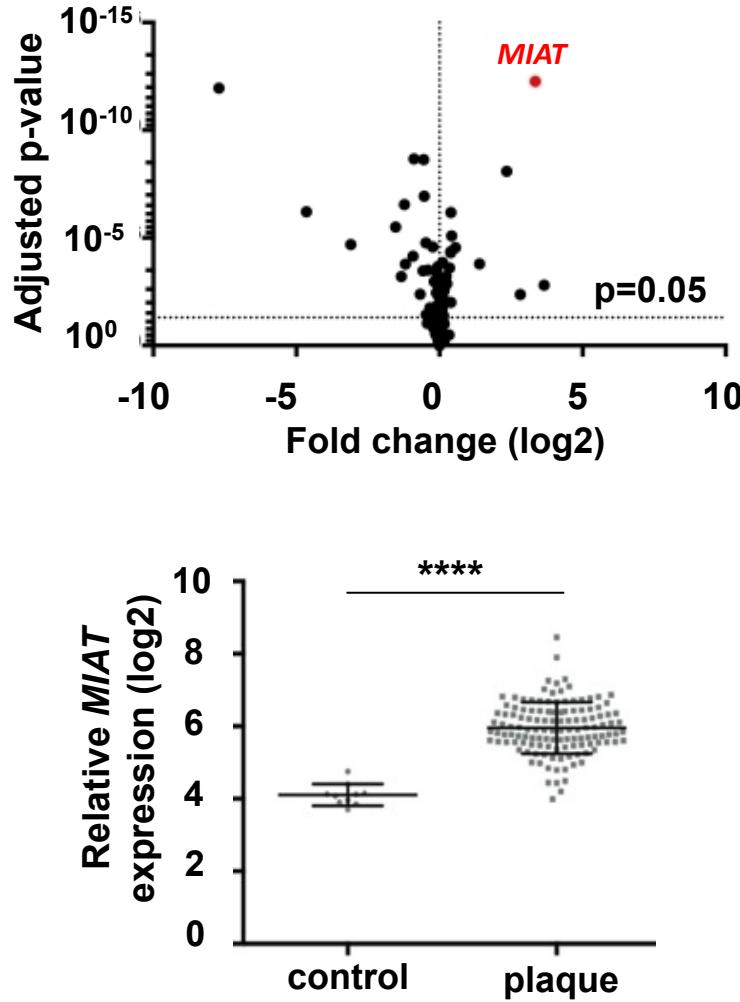
Human plaque biobank



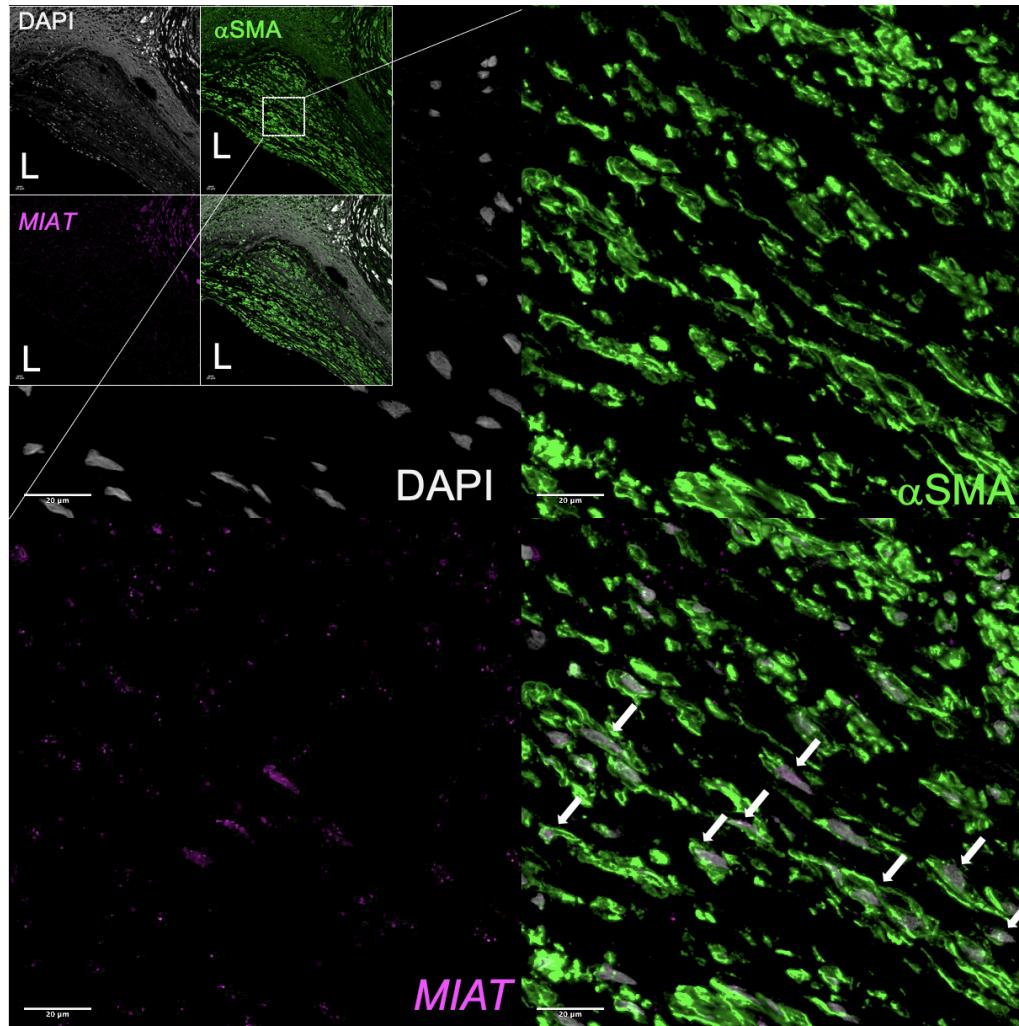
- Bulk, single cell, spatial transcriptomics
- Proteomics
- Genotyping

Clinical information:
symptoms, risk factors, imaging, follow-up

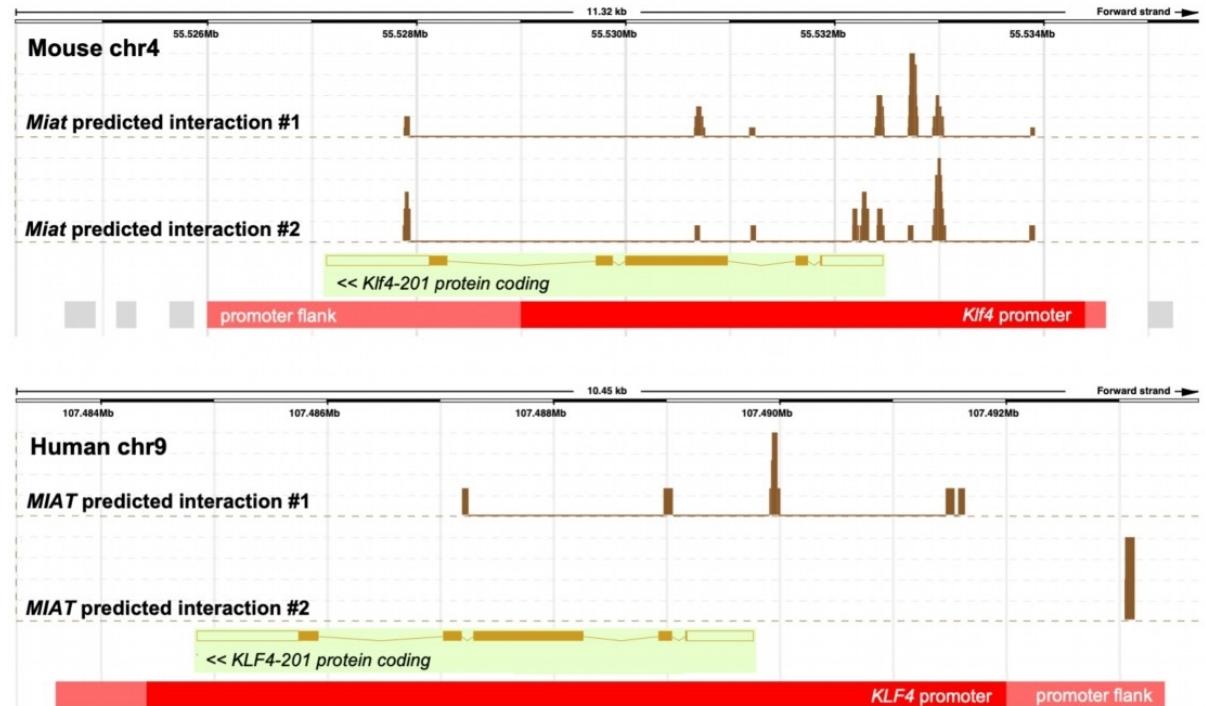
LncRNA *MIAT* is increased in advanced human plaques



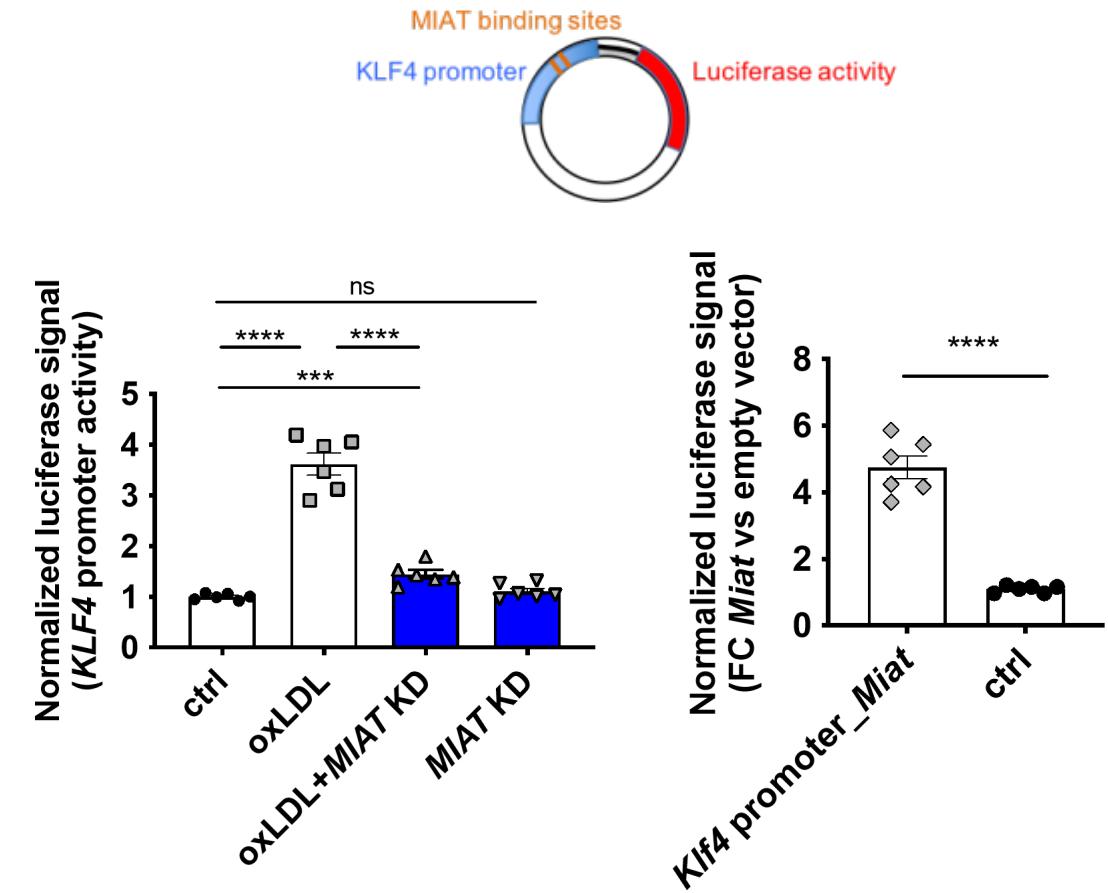
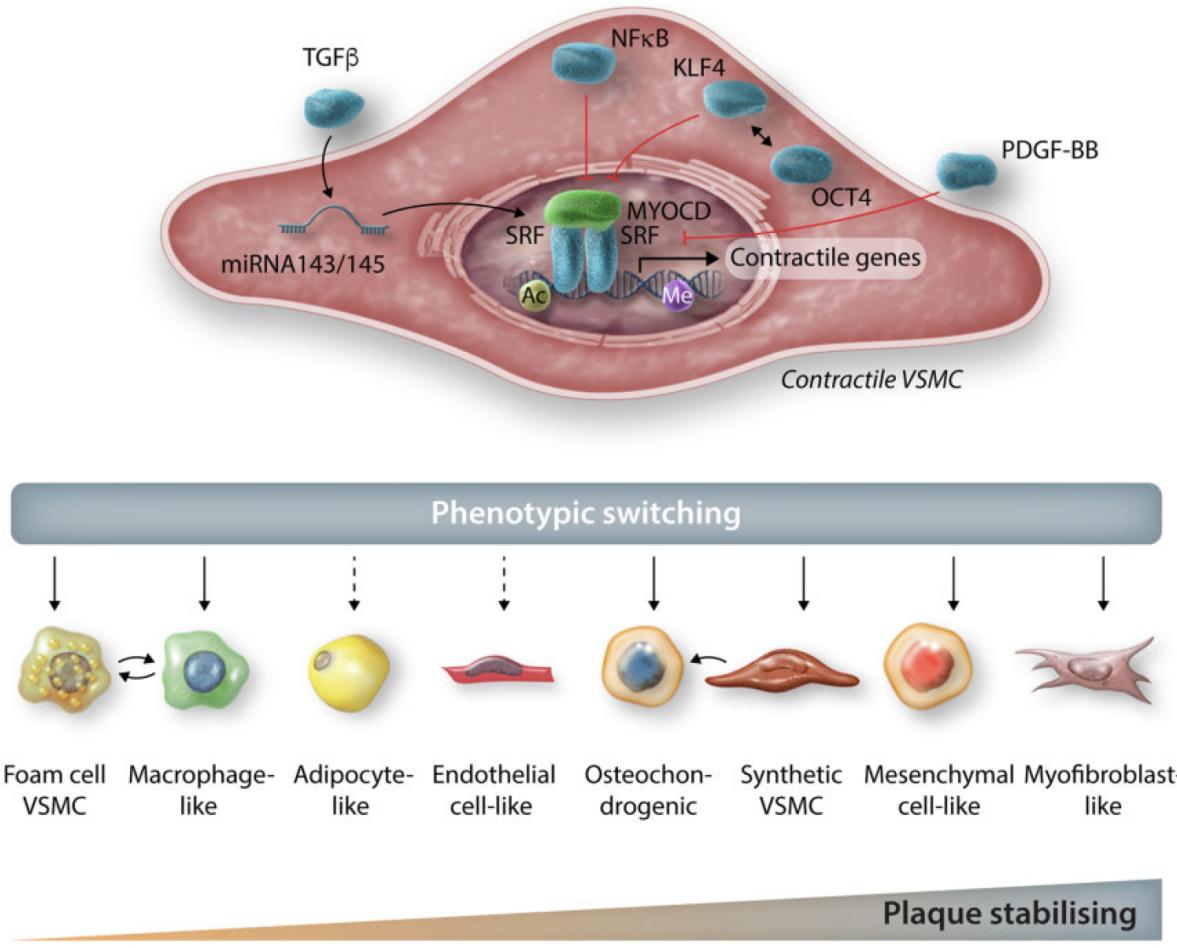
LncRNA *MIAT* is increased in advanced human plaques



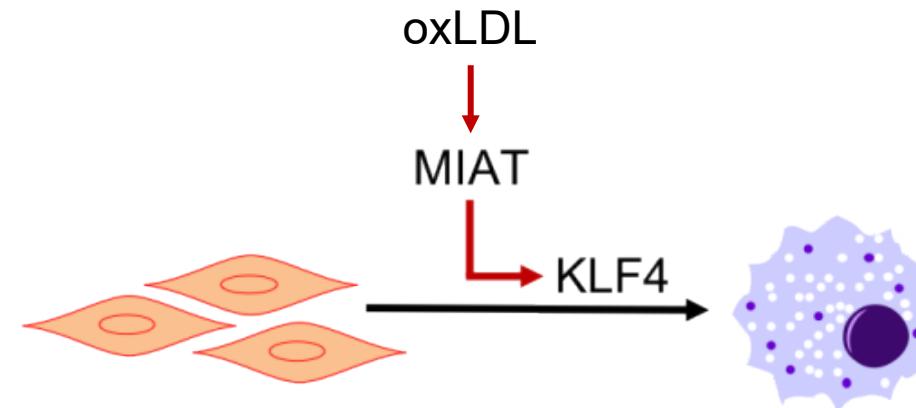
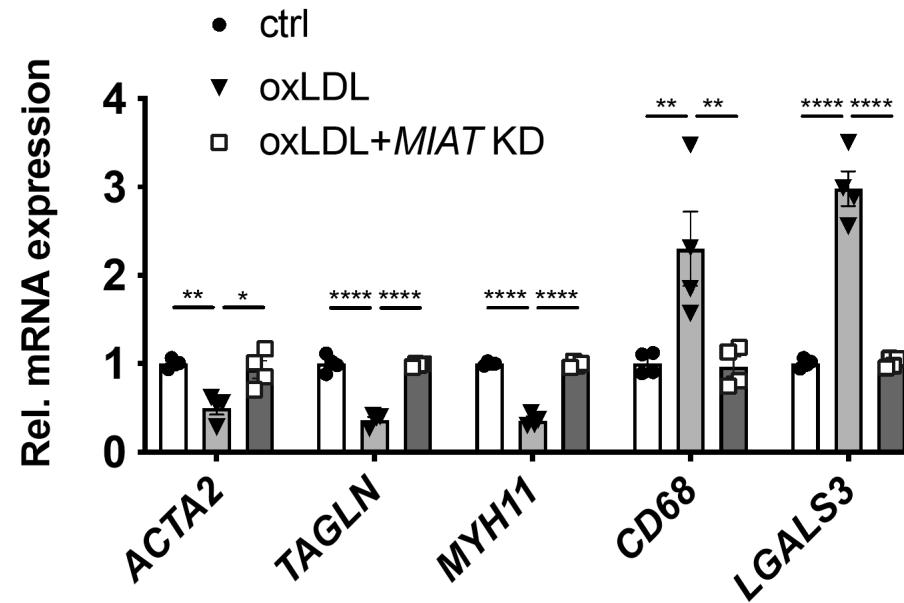
LongTarget:



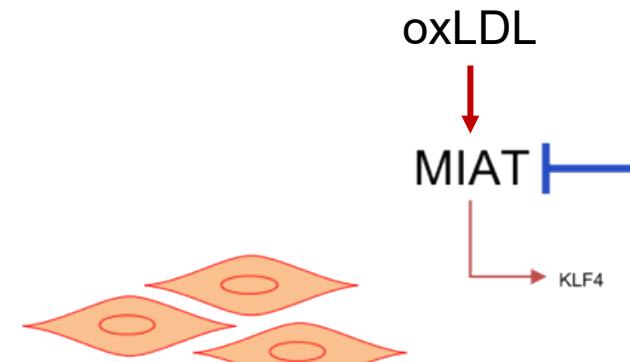
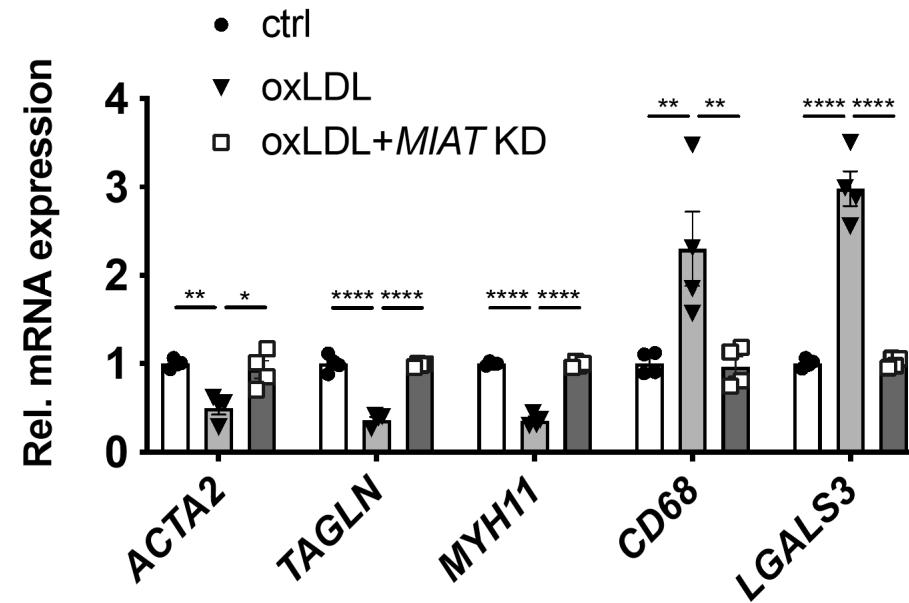
MIAT increases KLF4 in smooth muscle cells



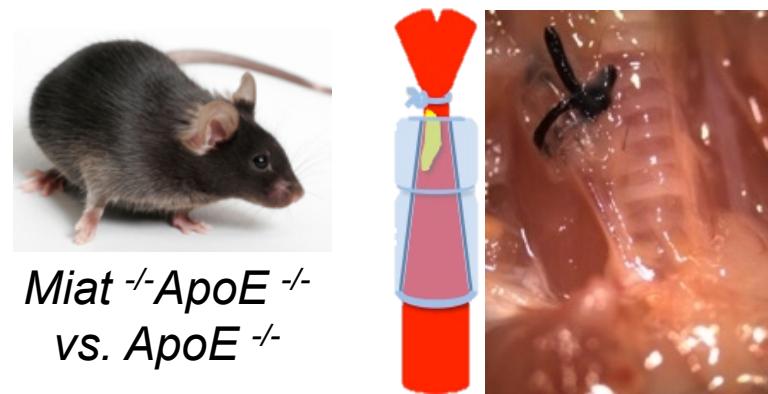
MIAT regulates SMC-macrophage transdifferentiation



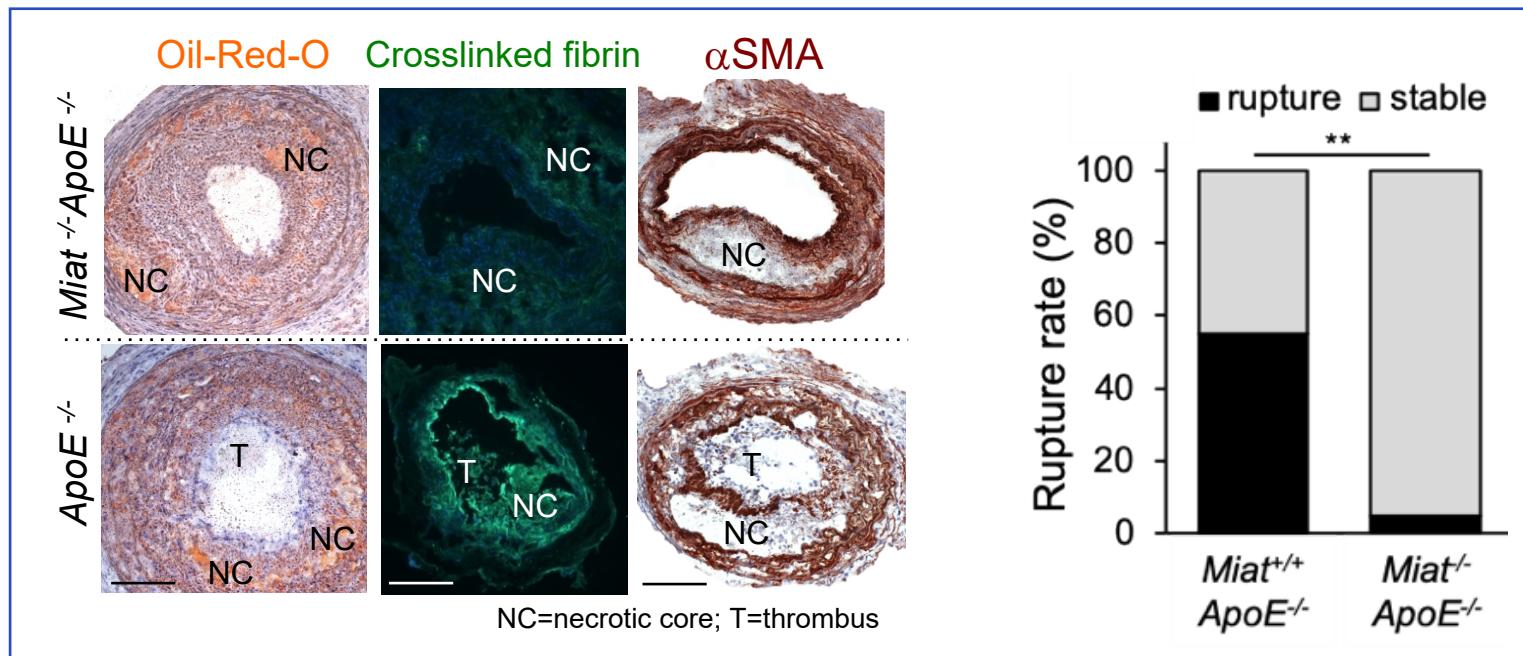
MIAT regulates SMC-macrophage transdifferentiation



Experimental *in vivo* plaque rupture



Miat^{-/-}*ApoE*^{-/-}
vs. *ApoE*^{-/-}



Summary

- LncRNAs are key regulators in atherosclerosis and plaque destabilization
- LncRNA *MIAT* triggers SMC-macrophage trans-differentiation (*via KLF4*)
- *MIAT* knockdown limits atherogenesis and enhances lesion stability

Acknowledgement



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