confirm several targets of HOXD10, -D11, and –D13 by qPCR, e.g. NR4A1, ROR2, LIF, ATF3.

Figure 1. Comparison of the genes which were differentially expressed after HOXD10-11-13 silencing

Conclusion: The expression of HOXD10, -D11 and –D13 in synovial fibroblasts and tissues strikingly overlaps with prediction sites for RA. Silencing experiments suggested that these embryonic HOX transcription factors have a crucial role in regulating fibroblast functions and might shape a joint specific environment that modulates the development and course of RA in specific joints.

REFERENCES:


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Adaptive immunity (T cells and B cells) in rheumatic diseases and Innate immunity in rheumatic diseases

OP0022
RHO EXPRESSION FACILITATES T CELL MIGRATION TO LYMPH NODES IN RESPONSE INFLAMMATION

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Background: Deficiency in geranylgeranyltansferase type I (GGTase-I) results in accumulation of active Rho family proteins Rhoa, Rac1 and Cdc42, responsible for cell communication and migration. We reported that mice with GGTase-I deficient macrophages (GLC mice) develop a spontaneous and age-dependent arthritis, reproducing pathology of RA [1].

Objectives: We study how GGTase-I deficiency in Ma changes T cell phenotype to facilitate their translocation to joints and the development of arthritis.