

Mid1 is a novel mediator of subchondral bone resorption in antigen-induced arthritis

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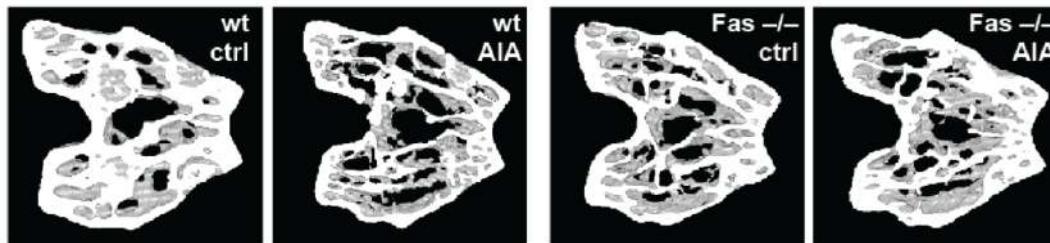
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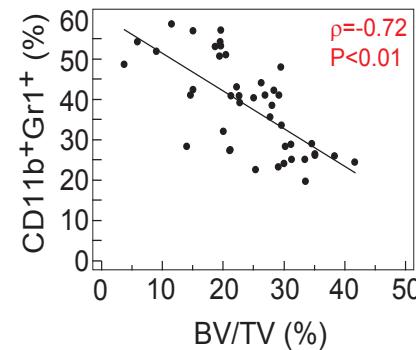
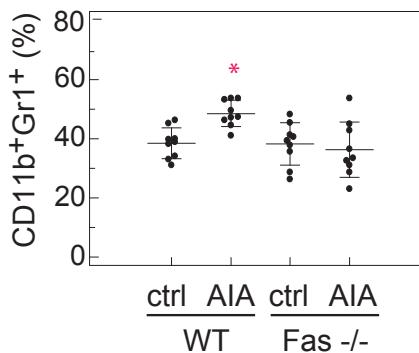
Antigen-induced arthritis (AIA) in Fas^{-/-} mice

Ameliorated, no subchondral bone resorption

(Lazic et al, 2018)

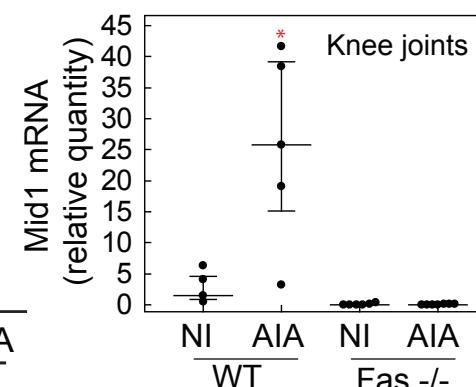
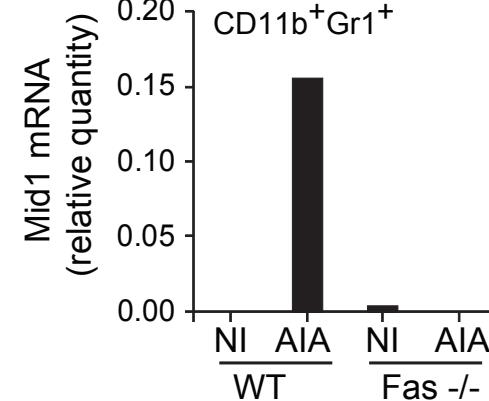
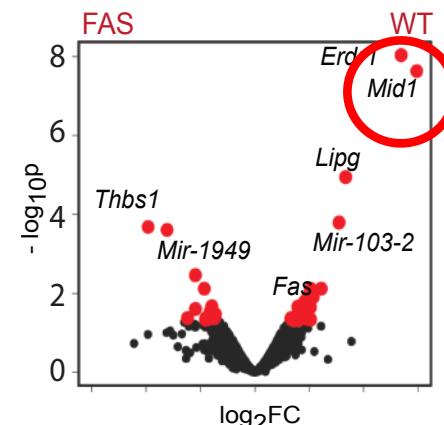


No synovial myeloid cell accumulation



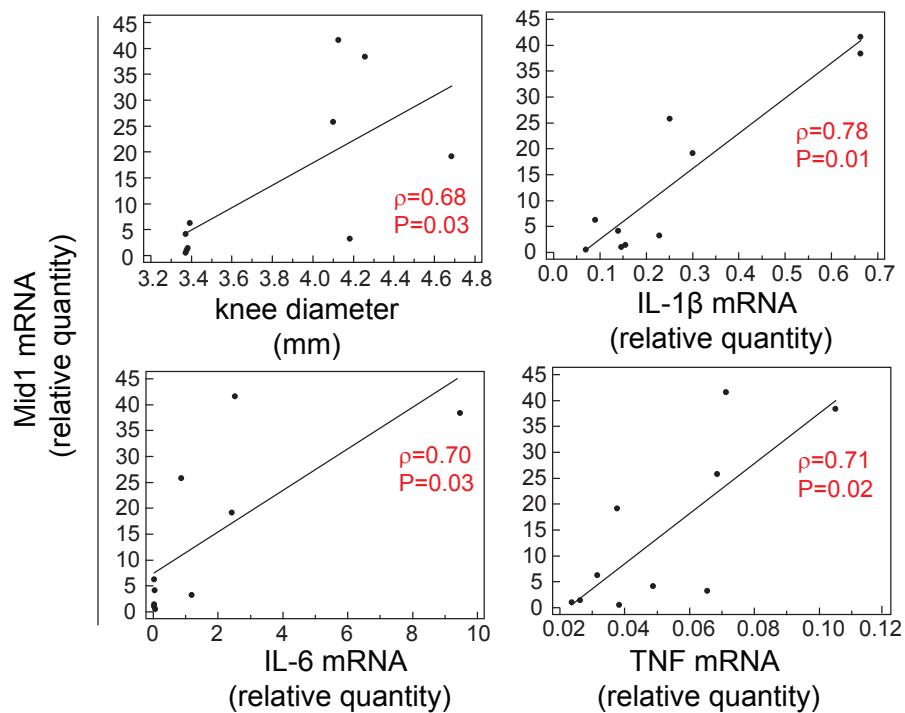
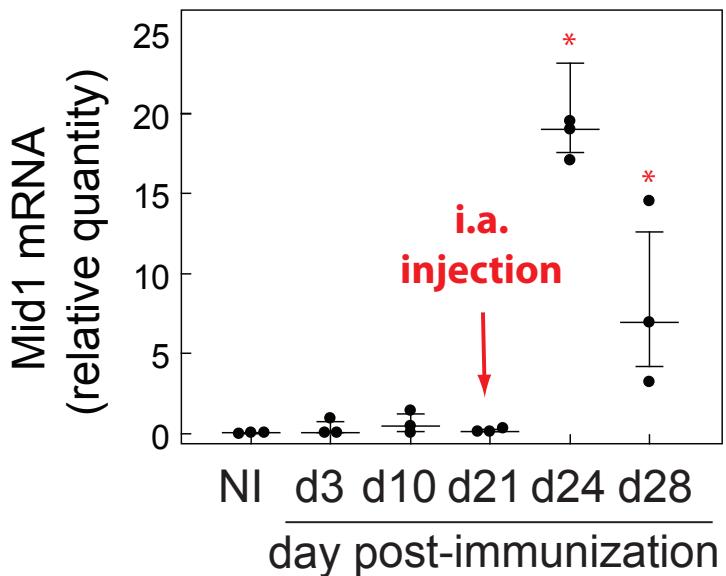
Affymetrix ST 2.0 gene chip
& q-PCR
(myeloid cells and bulk joint tissue)

Mid1 upregulation in resorative AIA of WT mice

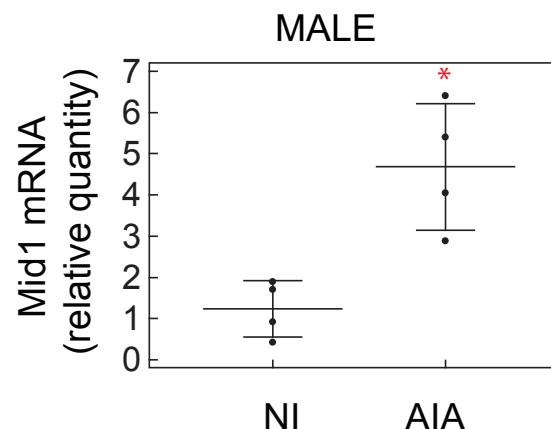
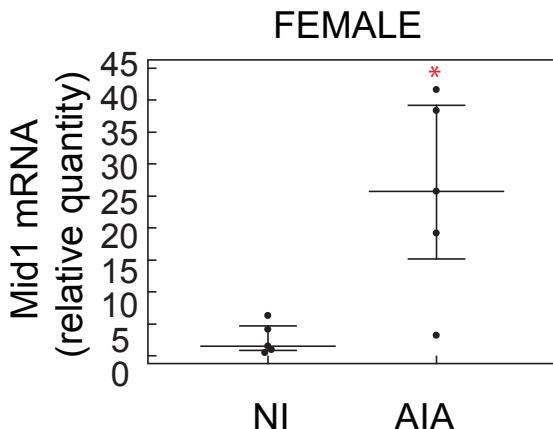


Mid1 expression correlates with local proinflammatory cytokine expression & arthritis severity

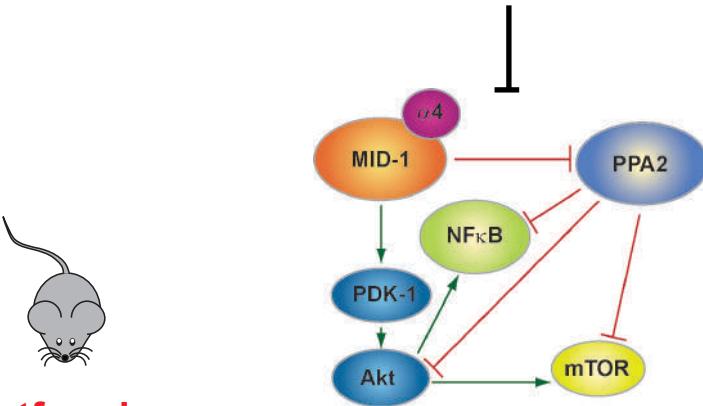
Mid1 upregulation early after AIA induction



Upregulation of Mid1 is not sexually dimorphic

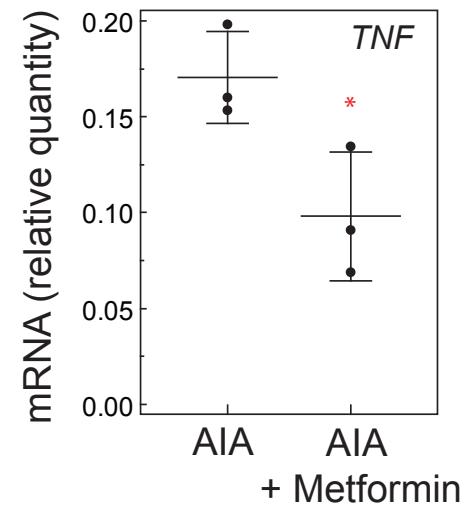
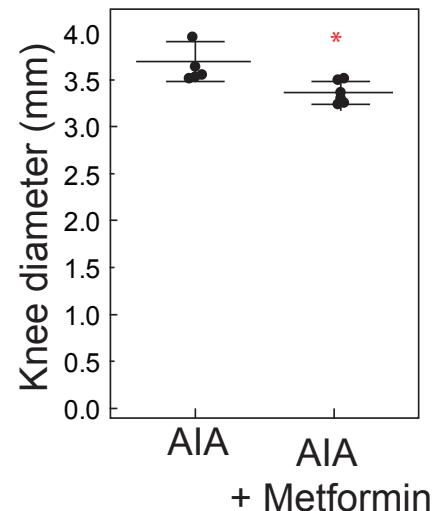
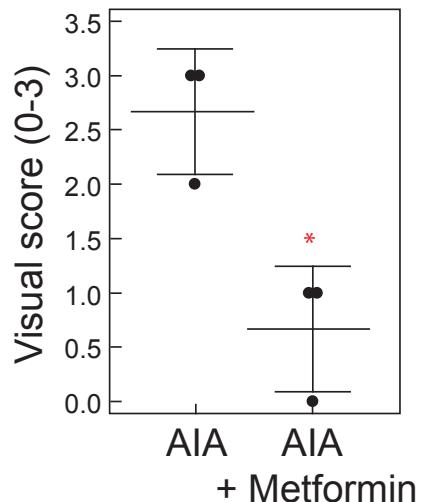


Metformin (Mid1-PP2A interaction inhibition)



Modified according to Koehler A, 2014 , European Journal of Cancer

Reduced arthritis severity and local inflammation



Conclusions

Mid1 expression is upregulated in WT mice with resorptive AIA.

Mid1 expression correlates with arthritis and inflammation severity.

Mid1 is a novel mediator of inflammation induced joint damage in AIA.

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