

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

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Nina Lukač, MD

Department of Anatomy,  
Laboratory for Molecular Immunology  
Croatian Institute for Brain Research  
University of Zagreb School of Medicine  
Zagreb, Croatia



**HRZZ**

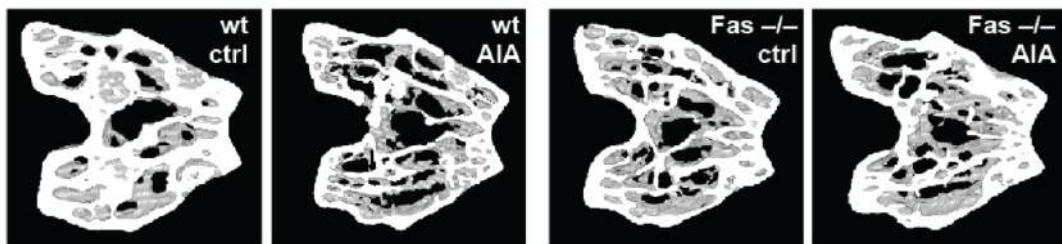
Hrvatska zaklada  
za znanost



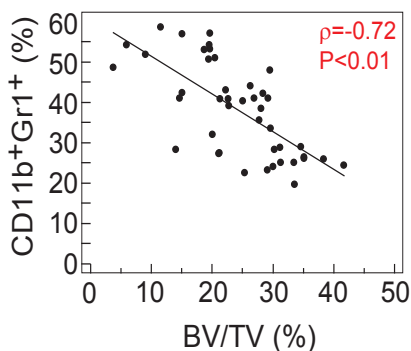
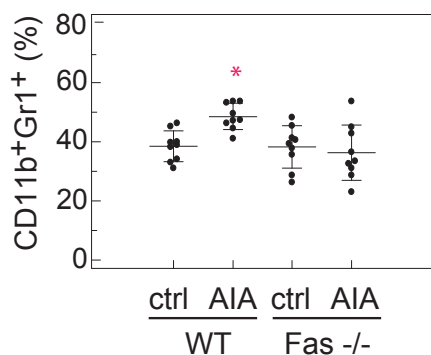
# Antigen-induced arthritis (AIA) in Fas<sup>-/-</sup> mice

Ameliorated, no subchondral bone resorption

(Lazic et al, 2018)

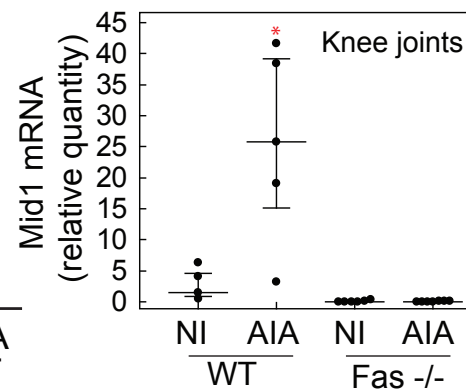
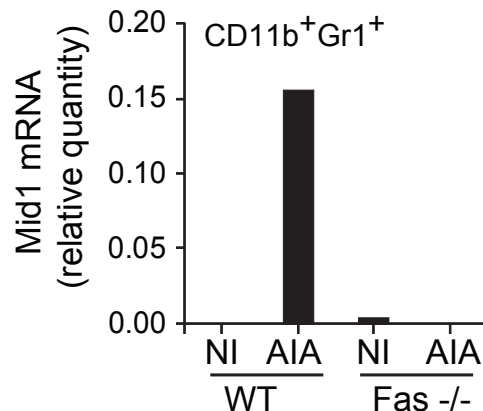
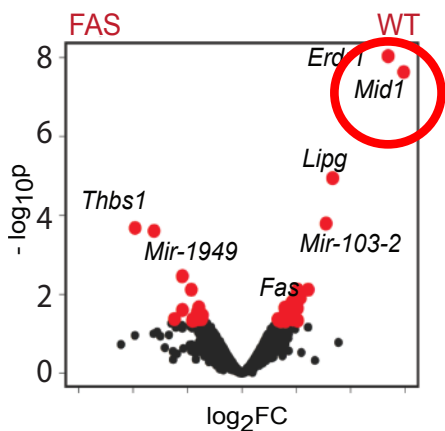


No synovial myeloid cell accumulation



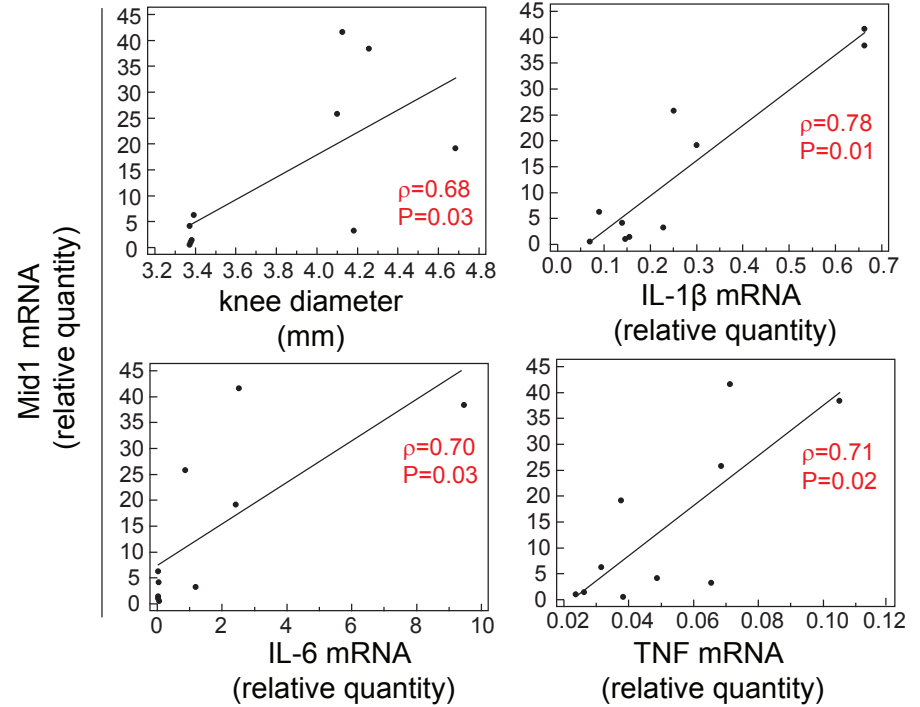
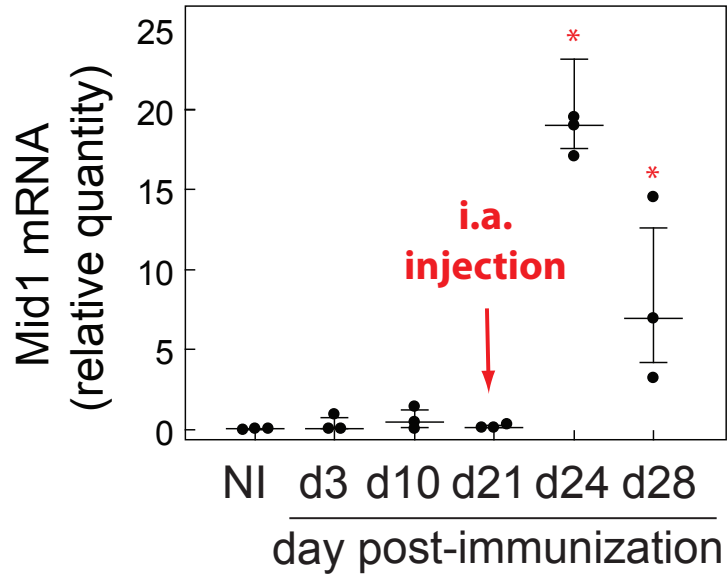
Affymetrix ST 2.0 gene chip  
& q-PCR  
(myeloid cells and bulk joint tissue)

## Mid1 upregulation in resorptive AIA of WT mice

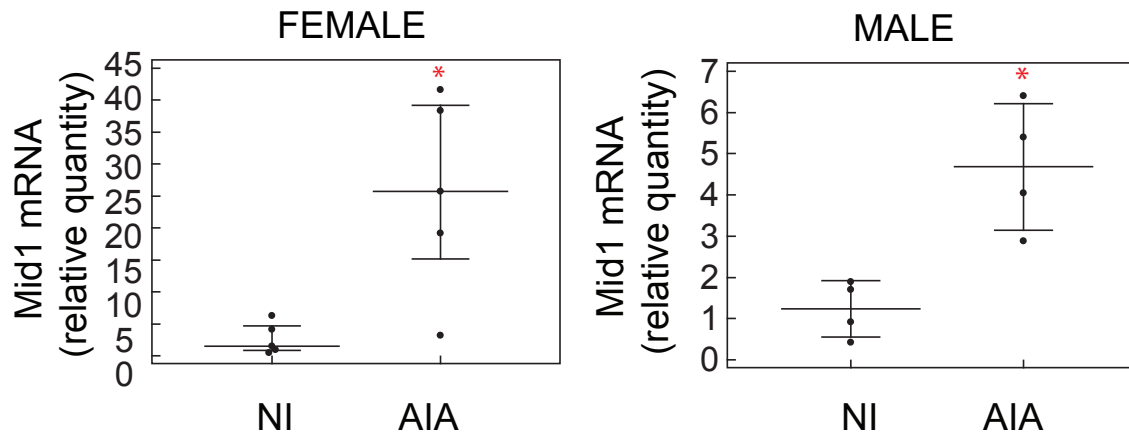


# Mid1 expression correlates with local proinflammatory cytokine expression & arthritis severity

## Mid1 upregulation early after AIA induction



## Upregulation of Mid1 is not sexually dimorphic



# Metformin (Mid1-PP2A interaction inhibition)

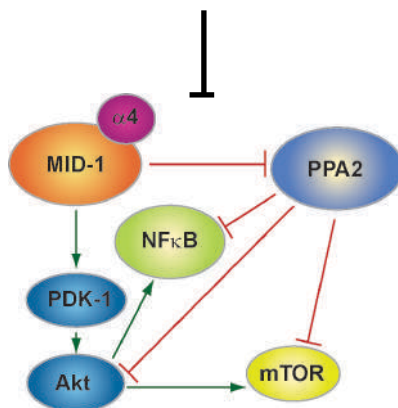
immunization

day 0 →

mBSA  
+ CFA s.c.

day 7 →

metformin  
1g/kg daily



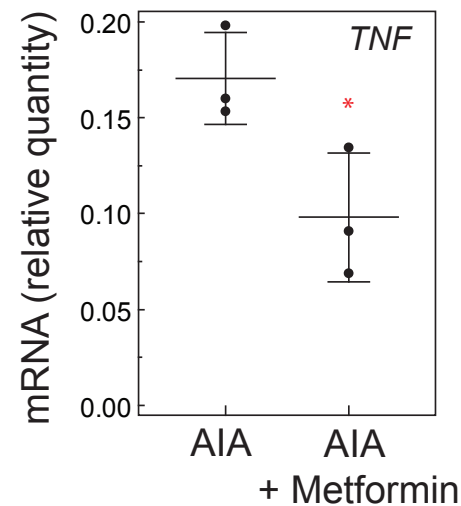
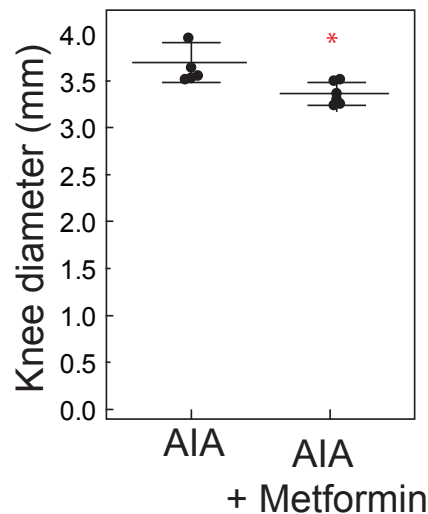
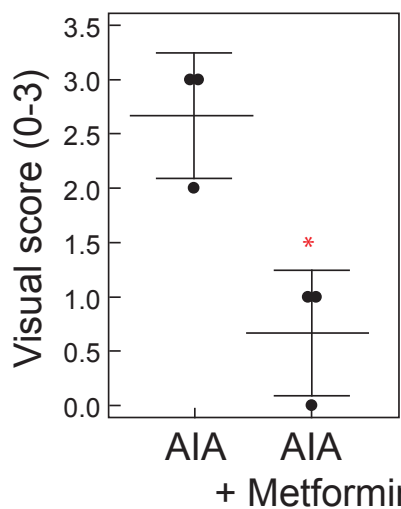
Modified according to Koehler A, 2014,  
European Journal of Cancer

arthritis  
induction  
day 21 →

mBSA/PBS i.a

arthritis  
assesment  
day 31 →

## 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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