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Myeloid deletion of SIRT1 suppresses collagen-induced arthritis in mice by modulating dendritic cell maturation

Byung-Hyun Park, Youngyi Lee, and **Seong Ji Woo**
Chonbuk National University Medical School, Republic of Korea

The type III histone deacetylase SIRT1 is an enzyme critical in the modulation of immune and inflammatory responses. However, data on its role in rheumatoid arthritis (RA) are limited and controversial. To better understand how SIRT1 regulates adaptive immune responses in RA, we evaluated collagen-induced arthritis (CIA) in myeloid cell-specific SIRT1 knockout (mSIRT1 KO) mice. CIA was induced in mSIRT1 KO and wild-type (WT) mice. Arthritis severity was gauged by clinical, radiographic, and pathologic scores. Levels of cytokines and transcription factors were determined by RT-PCR and ELISA. Flow cytometry was used to quantify T cells and dendritic cells (DCs). T-cell proliferation was also investigated when co-cultured with antigen-pulsed DCs. Compared with WT counterparts, arthritis in mSIRT1 KO mice was less severe and proved less destructive to joints. Expression levels of inflammatory cytokines, matrix metalloproteinases, and ROR-γT in mSIRT1 KO mice were also reduced by comparison, paralleled by reductions in Th1 and Th17 cells and CD80- or CD86-positive DCs. In addition, impaired maturation of DCs and resultant declines in Th1/Th17 immune response were observed in mSIRT1 KO mice. In co-culture, DCs of SIRT1 KO mice were less adept at driving T-cell proliferation and Th1/Th17 immune response. Myeloid cell-specific deletion of SIRT1 appears to suppress CIA through modulation of DC maturation. Our data suggest that careful investigation of DC-specific SIRT1 downregulation is needed to weigh the therapeutic utility of agents targeting SIRT1 in RA.

Biography

Youngyi Lee has completed her master from Woosuk University. Currently, she is a PhD student of Chonbuk National University Medical School. She has an interest in the pathogenesis of rheumatoid arthritis.

bhpark@ibnu.ac.kr

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