When synoviocytes and BMCs were cultured with 1,25(OH)2D3 and RANKL and the decoy receptor for RANKL, OPG, were quantified by ELISA in the supernatant of the cultures. At harvest, the synoviocytes and BMCs were cultured separately and their number was counted. The time points at which bone destruction observed in RA were measured. Our aim was to examine whether a co-culture system of human synoviocytes and mouse BMCs might be developed. Through this analysis, we found that ATO prevented activated naïve CD4+T-cell differentiation into Th1 cell and reduced cytokine production by activated Th17 cells by downregulating their signature transcription factors, STAT3 and orphan nuclear receptor (RORγt). Notably, ATO reduced Th17 cells frequency while increased Treg cells frequency under specific polarizing conditions from treatment-naïve RA patients by transfecting siRNA STAT3 and lentivirus STAT3 to verify the mechanism of ATO on Th17/Treg balance in vitro. Collagen-induced arthritis (CIA) model was constructed to detect the clinical score, histopathological change, bone destruction, Th17/Treg proportion and joint tissues immunohistochemistry.

**References**


**Acknowledgement:** National Science Foundation of China (NO. 81723291) and (NO. 81771748), 81771749

**Disclosure of Interests:** None declared

**DOI:** 10.1136/annrheumdis-2019-eular.6330

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