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## Comment on "B Cell Depletion Enhances T Regulatory Cell Activity Essential in the Suppression of Arthritis"

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## Comment on “B Cell Depletion Enhances T Regulatory Cell Activity Essential in the Suppression of Arthritis”

We read the interesting article by Hamel et al. (1), which provides encouraging data on the role of B cells in arthritis. We suggest that increasing T regulatory cells (Tregs) reveals a novel mechanism of B cell depletion in rheumatoid arthritis (RA) therapy.

B cells play a pivotal role in the pathogenesis of RA through autoantibody production, chemokine/cytokine secretion, and inhibiting autoreactive T cell responses (2). Although a series of treatment regimens that affect B cells was developed (3), the exact mechanism for the efficacy of B cell-depletion therapy remains unclear. The depletion of B cells with a decrease in autoantibodies in RA does not necessarily correlate with the clinical outcome, indicating that mechanisms other than producing Abs are in operation. Recently, Yeo et al. (4) identified B cells as the main source of receptor activator of NF- $\kappa$ B ligand, which contributes to osteoclast activation and bone destruction. Interestingly, Hamel et al. (1) first correlated B cell depletion with the improvement of Tregs, which can inhibit osteoclast formation and activation (5), and with other regulatory mechanisms. The authors found that during proteoglycan-induced arthritis (PGIA), B cell depletion strikingly increased Tregs and decreased the se-

verity of arthritis. In addition, both Tregs and B cell depletion restored proteoglycan-induced arthritis, although the resurgent autoantibody response was absent.

Collectively, B cell depletion could be used for controlling arthritis by improving Tregs. Better insights into the role of B cell depletion in Treg-mediated osteoclast suppression could result in innovative therapies for RA and other bone-destructive diseases.

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