The Glucocorticoid-Induced Tumor necrosis factor Receptor GITR, a member of the tumor necrosis factor receptor superfamily, has been shown to be important in modulating immune responses in the context of T cell immunity. B lymphocytes also express GITR, but a role of GITR in humoral immunity has not been fully explored. To address this question, we performed studies to determine the kinetics of GITR expression on naïve and stimulated B cells and the capacity of B cells to develop and mount antibody responses in GITR-/- mice. Results of our studies indicate that all mature B cells express GITR on the cell surface, albeit at different levels. Expression of GITR on naïve mature B cells is upregulated by BCR signaling, but is counteracted by helper T cell-related factors and other inflammatory signals in vitro. In line with these findings, expression of GITR on germinal center and memory B cells is lower than that on naïve B cells. However, the expression of GITR is strongly upregulated in plasma cells. Despite these differences in GITR expression, the absence of GITR has no effect on T cell-dependent and T cell-independent antibody responses to model antigens in GITR-/- mice, or on B cell activation and proliferation in vitro. GITR deficiency manifests only with a slight reduction of mature B cell numbers and increased turnover of naïve B cells, suggesting that GITR slightly contributes to mature B cell homeostasis. Overall, our data indicate that GITR does not play a significant role in B cell development and antibody responses to T-dependent and independent model antigens within the context of a GITR-deficient genetic background.


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Image: School of Medicine, University of Colorado.