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Mature dendritic cells (DCs) are stimulators of T-cell immune response, whereas immature DCs support T-cell tolerance. Murine B cells can inhibit the production of IL-12 by DCs and thereby hinder the inflammatory response. Notwithstanding the importance of this modulation, only a few studies are available in humans. Here, we have developed an in vitro model of cocultures to assess its significance. We establish that human activated B cells restrained the development of monocytes into immature DCs and their differentiation into mature DCs. In addition, they decreased the density of HLA-DR from mature DCs, the expression of CD80 and CD86 coactivation molecules, the production of IL-12p70 required for antigen presentation and Th1 differentiation, and inhibited the DC-induced T-cell proliferation. These modulations were mediated by $CD19^{+}IgD^{low}CD38^{+}CD24^{low}CD27^{-}$ B cells and needed direct cell-to-cell contacts that involved CD62L for the control of CD80 and CD86 expression and a soluble factor for the control of IL-12 production. Moreover, mature DCs from patients with systemic lupus erythematosus displayed insensitivity to the regulation of IL-12. Overall, it appears that human B cells can regulate DC maturation and function and that inefficient B-cell regulation may influence an improper balance between an effector inflammatory response and tolerance induction.