In vitro analyses of autoreactive B cells and pDCs clearly demonstrate a critical role for both TLR7 and TLR9 in the detection of endogenous nucleic acid ligands and activation of autoreactive B cells and dendritic cell subsets. Ligands for TLR7 and TLR9 are bound by surface receptors, either the BCR or an Fc?R, and then delivered to endolysomal compartments where TLR7 or TLR9 are subsequently engaged, leading to immune activation. However, in vivo analyses of autoimmune-prone mice have revealed a more complex interplay between TLR9- and TLR7-driven activation of key effector populations and the severity of systemic autoimmune diseases such as SLE. It is clear that TLR7/BCR co-engagement promotes B cell activation, autoantibody production, and clinical disease. By contrast, TLR9 has a biphasic role and appears to initially limit the activation of B cells reactive to DNA or DNA-binding proteins, either by contributing to the production of protective antibodies or by directly constraining B cell responses to endogenous ligands. Nevertheless, TLR9 is still required for the full-scale activation of B cells with similar DNA/DNA-binding autoanigen reactivities.

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