Type 1 Diabetes (T1D) is a debilitating autoimmune disease that results from T-cell mediated destruction of insulin-producing β cells in the pancreas. Incidence rates of T1D are rising rapidly in the developed world for unknown reasons. It is suspected that environmental factors, especially gut microbiota (microorganisms in the lower gastrointestinal tract), play a large role in this rise. Research has shown that certain gut microbial compositions are associated with T1D onset and that antibiotics can trigger the onset of T1D. It is still unknown how gut microbiota interact with the immune system in order to alter the development of T1D. MyD88, an adaptor protein with an important function in immune cells, has been shown to be necessary for the development of T1D in mice when gut microbiota are present in these mice. MyD88 may alter the development of T1D by modulating gut microbiota, but the mechanisms by which it does this are unclear. In this study, we examined the effect of MyD88 expression in dendritic cells, antigen-presenting cells that are important for adaptive immunity, on the concentration of TGF-β and IgA and on the levels of gene expression of various antimicrobial peptides, all of which interact with gut microbiota. We found that expression of MyD88 in dendritic cells significantly increased the concentration of TGF-β in the small intestine and significantly altered the level of gene expression of two antimicrobial peptides. This research points towards future areas of study that can elucidate how MyD88 alters the development of T1D and, ultimately, towards new treatment or prevention methods for affected people.