ABSTRACT
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Dendritic cells (DC) are first in line to sense invading microbes and to deliver the signal to other immune cells. Plasmacytoid dendritic cells (pDC) are one subset of DCs and are mainly known for their ability to produce high amounts of type I interferons (IFN). The role of pDCs in bacterial infections is still poorly understood. Recent studies have shown high pDCs activation after *Citrobacter rodentium* infection in colon draining mesenteric lymph nodes (coMLN). Here we show an essential role of pDCs in regulating immune response to *Citrobacter*-infection in the colon using a specific pDC-depleted mouse model. We found that *Citrobacter*-infection had a more severe effect on pDC-depleted mice when compared to wild type mice. Deficiency of pDC during infection caused 20 % weight loss when no change in wild type mice were seen. Colon epithelium was damaged, epithelial stress genes were upregulated and overall colon length was shorter in pDC -depleted mice. T cell analysis showed that the lack of pDCs during dysbiosis blocks the induction of $Foxp3^+$ regulatory T cells in coMLN and increases IFNγ production by both CD4 (T helper) and CD8 (T cytotoxic) cells. Our results indicate that pDCs have regulative functions and in conjunction with T_{reg} cells they control inflammation in the gut during *Citrobacter*-infection.

Key words: Plasmacytoid dendritic cell, gut microbiota, *Citrobacter rodentium*, regulatory T cell