**Involvement of IRF4 dependent dendritic cells in T cell dependent colitis**

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Inflammatory Bowel Disease (IBD) is a chronic non-curable inflammatory disease of the intestine that affects as many as 1.4 million persons in the United States and 2.2 million persons in Europe. IBD results from abnormal immune response to bacterial components of the commensal microflora in genetically susceptible individuals and pathogenic CD4+ T cells, which accumulate in the inflamed mucosa, are believed to be key drivers of the disease. While dendritic cells (DCs) are important in the priming of intestinal adaptive immunity and tolerance their role in the initiation and perpetuation of chronic intestinal inflammation remains unclear. In the current study we used the CD45RBhi T cell transfer model of colitis to determine the role of IRF4 dependent DCs in intestinal inflammation. In this model naïve CD4+ T cells when transferred into RAG-/- mice, proliferate and expand in response to bacterial derived luminal antigen, localize to the intestinal mucosa and induce colitis. Adoptive transfer of naïve T cells into CD11cCre.IRF4fl/fl.RAG-1-/- mice resulted in reduced monocyte recruitment to the intestine and mesenteric lymph nodes (MLN) compared to Cre- controls. Inflammatory cytokines including IFNγ, TNFα and IL-6 also were reduced in the serum and intestinal tissues of these mice. Additionally CD11cCre.IRF4fl/fl.RAG-1-/- mice displayed significantly reduced numbers of CD4+ T cells in intestinal draining mesenteric lymph nodes and spleen but not the colonic lamina propria. Collectively these results suggest an important role for *Irf4* dependent DCs in T cell driven colitis.