Mesenteric lymph node remodelling requires lymphotoxin beta signaling during helminth infection

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Abstract :

Inflammation-induced lymph node stromal remodelling promotes the immune cell trafficking and interaction towards driving the humoral response. Here we show that intestinal helminth infection led to an extensive expansion and remodelling of the intestinal draining mesenteric LNs (mLNs). C57BL/6J mice or lymphotoxin beta knockout mice ($LT\beta^{-/-}$) were infected with intestinal helminth: *Heligmosomoides polygyrus* (Hp) and the entire chain of the mLN was collected at 0 (naïve), and 21-day post infection. Total cellularity of the mLN from naïve or infected mice in the mLN was determined by flow cytometry. We provide evidence that lymphotoxin signaling was required to promote stromal remodelling, niches expansion and proliferation. Using immunofluorescence microscopy, we further confirm that mice lacking lymphotoxin beta ($LT\beta^{-/-}$) had fewer fibroblastic reticular cells, lymphatic vessels, and extrafollicular B cells. By flow cytometry experiments we were able to confirm that $LT\beta^{-/-}$ mice had fewer $CD11c^{+}$ dendritic cells compared to WT mice which corelated with reduced lymphatic endothelial cells expansion and associated stromal remodeling. Overall, these results highlight the role of LTβ in lymphoid expansion which regulates the DCs trafficking, B cell response and vasculature expansion towards adaptive immune response against helminth infection.