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Lymphocytes constantly survey the lymph nodes in search for potential infection by a pathogen. They enter the afferent lymphatic vessel that serves as a conduit to transport the motile lymphocytes to the draining lymph node. Lymphatic vessels (LVs) are present in most vascularized tissues. They are traditionally regarded as passive conduits for soluble antigens and leukocytes. Afferent LVs begin as blind ended capillaries, which give rise to collecting vessels that merge and connect with draining lymph nodes (dLNs). Initial lymphatic capillaries are composed of Lymphatic Endothelial Cells (LECs) connected by discontinuous cell junctions, which join to form larger collecting lymphatic vessels, and ultimately feed into the LN subcapsular sinus. Within the LN, LECs are localized to the subcapsular, cortical, and medullary sinuses, where they interact with incoming and exiting leukocytes. LECs, and in general LN stromal cells, have emerged in the recent years as active players in the immune response. In support to this, studies have shown that the immune response generated during inflammation and under pathologic conditions is accompanied by modeling of the LVs and generation of new lymphatics, a process known as lymphangiogenesis. These facts strongly suggest that LECs and stromal LN cells in general. are not inert players but rather are part of the immune response by organizing immune cells movement, exchanging information and supplying survival factors. The purpose of this research topic is to review the role of the LECs during immune homeostasis and cancer.

Considering the critical role of lymphangiogenesis in many pathologies like chronic and acute inflammation, autoimmunity, wound healing, graft rejection, and tumor metastasis, it is important to understand the molecular mechanisms that govern the cross talks between the LECs and immune cells during homeostasis and inflammation.