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Perspective

Immunology of Vascular Endothelium and its Role in Innate Immunity

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DESCRIPTION

An effective immune response depends not only on the proper activation, regulation and function of immune cells, but also on their distribution and retention in different tissue microenvironments where they encounter different stimuli and other cell types. These activities are mediated by endothelial cells. Endothelial cells form specialized microcirculatory networks used by immune cells in both physiological and pathological functions. Endothelial cells represent a highly heterogeneous cell population that can interact with and modulate immune cell function. The microvascular endothelial cells plays an important role in in innate and adaptive immunity, inflammation, coagulation, angiogenesis, and the therapeutic implications of endothelial cell targeting in selected autoimmune and chronic inflammatory diseases.

In physiological processes Endothelial Cells (ECs) are actively involved in innate and adaptive immune responses, and then ECs have many innate immune functions that macrophages perform, including cytokine secretion, phagocytic functions, antigen presentation, pathogen-associated molecular patterning, danger-associated molecular pattern recognition, pro-inflammatory, immune potentiating and anti-inflammatory immunosuppression. Several novel receptor systems, including conditional hazard-associated molecular pattern receptors, non-pattern receptors, and homeostasis-associated molecular pattern receptors, contribute to the innate immune function of ECs. Immune metabolism and innate immune memory determine the innate immune function of ECs.

Type II activation of endothelial cells, mediated by proinflammatory cytokines such as Tumor Necrosis Factor (TNF) and Interleukin-1 (IL-1), also increases local blood flow, plasma protein leakage and recruits leukocytes. Type II activation responses depend on new gene transcription and protein translation and are slower-onset but more persistent than type I activation responses, which last hours to days. Type II activated endothelial cells spontaneously evolve from a neutrophil-recruiting phenotype to a monocyte and T-cell-recruiting phenotype. Polarizing cytokines such as interferon-γ and IL-4further modify the phenotype of activated endothelial cells to preferentially support the inflammatory response of T-helper-1 (TH1) or TH2 cell types.

In chronic inflammation, endothelial cells respond to angiogenic factors such as vascular endothelial growth factor A to form new blood vessels necessary to sustain inflammatory neotissues such as pannus in rheumatoid arthritis. Endothelial cells can also respond to lymphotoxin-β to acquire the characteristics of endothelial venules and support the development of tertiary lymphoid organs.

Endothelial cells (ECs) form the inner cellular lining of all blood and lymphatic vessels. In addition to their structural function and role in hemostasis and regulation of vascular tone (intravascular walls) and interstitial fluid drainage (intralymphatics), ECs are an important part of a functioning immune system. ECs play an active and versatile role in regulating immune responses through indirect pathways by mediating leukocyte trafficking between blood, stromal tissue, and lymphatic compartments during homeostasis and inflammation. The sequence of events driving leukocyte passage across the endothelial barrier is well characterized. However, the molecular mechanisms, signaling networks, and tissue-specific signaling pathways involved are still not clearly understood. Furthermore, increasing evidence suggests a critical role for blood and lymphatic ECs in regulating immune responses by altering antigen presentation and immune effector cell function through direct cell-cell contacts and EC-secreted soluble factors. For example, tumor-associated blood ECs actively prevents T cells from infiltrating the tumor parenchyma, whereas lymphoid ECs appear to mediate peripheral self-tolerance. It has been reported that it can directly modulate the viral immune response.

Dysfunction of ECs is associated with multiple pathologies involving inflammatory responses, ranging from autoimmune diseases to chronic infections and cancer. Therefore, detailed studies of the cellular and molecular mechanisms that mediate exchanges between ECs and the immune system are not only important for a fundamental understanding of host defense responses, but also novel therapeutics for treating EC inflammatory diseases.

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