Editorial Article

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Endothelial-Mesenchymal Transition (EndoMT)

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EndoMT is a complex biological process in which endothelial cells lose their specific markers and acquire a mesenchymal phenotype and express mesenchymal cell products. Endo-MT has been reported to occur in fibrosis-related disease. Current study strongly supports the reality of an EndoMT phenomenon in human disease and expands its biological role beyond fibrosis. Recent studies have demonstrated that the occurrence of EndoMT is clearly associated with inflammation and EndoMT is one of the primary drivers of vascular inflammation.

One important consequence of EndoMT is an increase in expression of leucocyte adhesion molecules by luminal endothelial cells and enhanced presence of inflammatory cells in vessel wall. Endothelial cells undergoing EndoMT express of a number of proinflammatory chemokines and cytokines and their receptors (including CCL2), leucocyte adhesion molecules (such as ICAM-1 and VCAM-1), matrix metalloproteinases (MMP2) as well as fibronectin, a proinflammatory extracellular matrix (ECM) component long linked to inflammation.

Thus, EndoMT plays an important role in leucocyte recruitment, a process underlying a number of important immune-related disease states. In addition to its proinflammatory effects, EndoMT increase expression of key genes involved in the regulation of vascular permeability, leading to an increased permeability normally observed in inflammatory. Furthermore, the emergence of cytokines (including IFNr and TNFa) leads to a profound increase in TGF-b signaling culminating in endothelial cells that, in turn, enhance EndoMT.

In summary, EndoMT plays a key role in induction of vessel wall inflammation and development and progression of immune-related diseases. Inhibition of EndoMT in mice reduces vessel wall inflammation and vascular permeability. The pro-inflammatory effects of EndoMT identify it as an important driver of immune-related diseases and show the potential of cell-type specific therapeutic intervention aimed at control of these disease. However, the role and mechanism of EndoMT in a variety of different immune-related diseases are largely unknown.