Protecting the Endothelial Cell Barrier

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Editorial

Endothelial barrier dysfunction (EBD) is a major cause of various vascular diseases such as sepsis, acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) [1,2]. Current therapeutic strategies to mitigate these vascular pathologies involve administration of broad-spectrum antibiotics [3,4] and anti-cytokine antibodies [5,6] providing limited alleviation of symptoms and are clearly insufficient.

A single layer of endothelial cells (EC) lines the lumen of blood vessels forming a physical barrier between blood and tissues. This endothelial barrier regulates selective passage of various micro- and macro-molecules via either transcellular or paracellular pathways [7,8]. The paracellular pathway is regulated by a network of intercellular junction proteins and disruption of these intercellular interactions causes vascular leakage and EBD [9]. Various agonists, such as lipopolysaccharide (LPS), thrombin, cytokines, TGFβ and VEGF, bind mediated degradation of many client proteins thereby mimicking a

Therapeutic strategies targeted against EBD have involved blocking agonist-receptor interaction, signal transduction or inflammation [38]. However, combined data from previous two decades strongly suggests that pharmacological intervention of any single target is only partly successful and a more multi-targeted and broad-spectrum approach is needed. Hsp90 and HDAC inhibitors might provide an effective way to protect the endothelial cell barrier and EBD.

References


