Endotoxin-induced vascular endothelial cell migration is dependent on TLR4/NF-?B pathway, NAD(P)H oxidase activation, and transient receptor potential melastatin 7 calcium channel activity

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Endothelial dysfunction is decisive and leads to the development of several inflammatory diseases. Endotoxemia-derived sepsis syndrome exhibits a broad inflammation-induced endothelial dysfunction. We reported previously that the endotoxin, lipopolysaccharide (LPS), induces the conversion of endothelial cells (ECs) into activated fibroblasts, showing a myofibroblast-like protein expression profile. Enhanced migration is a hallmark of myofibroblast function. However, the mechanism involved in LPS-induced EC migration is no totally understood. Some studies have shown that the transient receptor potential melastatin 7 (TRPM7) ion channel is involved in fibroblast and tumor cell migration through the regulation of calcium influx. Furthermore, LPS modulates TRPM7 expression. However, whether TRPM7 is involved in LPS-induced EC migration remains unknown. Here, we study the participation of LPS as an inducer of EC migration and study the mechanism underlying evaluating the participation of th