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LPS Induces Hyper-Permeability of Intestinal Epithelial Cells.

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Abstract

Necrotizing Enterocolitis (NEC) is a severe inflammatory disorder leading to high morbidity and mortality rates. A growing body of evidence demonstrate the key role of the Toll like receptor 4 (TLR4) in NEC. This membranal receptor recognizes lipopolysaccharides (LPS) from the bacterial wall and triggers an inflammatory response. The aim of the present study was to elucidate the effect of LPS on paracellular permeability known to be severely affected in NEC. IEC-18 cells were treated with LPS and the effects on morphology, paracellular permeability and their associated gene and protein expressions were measured. Our results show that LPS down regulated the expression of occludin and ZO-1 mRNAs while up regulating Cdkn1a. In addition LPS caused a significant increase in paracellular permeability and epithelial barrier damage. Finally ZO-1 protein was found to be spatially disarrayed in the intercellular junctions in response to LPS. We conclude that LPS adversely affected the functionality of the intestinal epithelial barrier suggesting a new mechanism by which bacterial infection may contribute to the development of NEC. J. Cell. Physiol. 232: 381-390, 2017.

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