**Aims:** Our aim was to investigate the possible role of dexamethasone on lipopolysaccharide (LPS)-induced intestinal epithelial barrier dysfunction. Furthermore, we evaluated its ability to modulate the inflammatory response and also the expression of proteins of the intestinal tight junctions.

**Methods:** To conduct these experiments, male rats had their jugular vein cannulated for endotoxin administration, one day before the experiment. Rats were pre-treated with dexamethasone (synthetic glucocorticoid; 0.1 or 1 mg/kg, intraperitoneal) before LPS administration (1.5 mg/kg, intravenous). At 6h after endotoxemia induction, the intestinal permeability was evaluated by injecting FITC-dextran 4 kDa in the ileum, mesenteric lymph nodes were collected for microbiological analysis and also cytokines were quantified in the plasma and intestinal mucosa by ELISA technique. Additionally, the integrity of the tight junctions was determined by the expression of tight junction proteins (occludin, claudin-2, junctional adhesion molecule-A) as well by transmission electron microscopy.

**Results:** Our results demonstrated that dexamethasone administration reduces the LPS-induced permeability in the ileum and prevented the bacterial translocation to the mesenteric lymph nodes. The plasma and mucosa concentrations of TNF-α, IL-1β, IL-6, IL-10 and IFN-γ were significantly reduced in dexamethasone-treated rats. Furthermore, treatment with dexamethasone reverted the LPS-induced epithelial barrier dysfunction, increasing the expression of occludin, reducing the claudin-2 cleavage and attenuating the histological damages. The morphology of the tight junctions was also preserved by the dexamethasone administration, therefore reducing their opening induced by endotoxemia.

**Conclusion:** Together these results suggest a protective role for dexamethasone preventing the intestinal barrier dysfunction induced by systemic inflammation, possibly...
modulating the inflammatory response. Moreover, these experimental findings reinforce the Surviving Sepsis Campaign recommendations for the administration of low glucocorticoid doses in septic shock patients.