Prevention of gut leakiness by a probiotic treatment leads to attenuated HPA response to an acute psychological stress in rats.


Abstract

BACKGROUND AND AIMS: Intestinal barrier impairment is incriminated in the pathophysiology of intestinal gut disorders associated with psychiatric comorbidity. Increased intestinal permeability associated with upload of lipopolysaccharides (LPS) translocation induces depressive symptoms. Gut microbiota and probiotics alter behavior and brain neurochemistry. Since Lactobacillus farciminis suppresses stress-induced hyperpermeability, we examined whether (i) L. farciminis affects the HPA axis stress response, (ii) stress induces changes in LPS translocation and central cytokine expression which may be reversed by L. farciminis, (iii) the prevention of "leaky" gut and LPS upload are involved in these effects.

METHODS: At the end of the following treatments female rats were submitted to a partial restraint stress (PRS) or sham-PRS: (i) oral administration of L. farciminis during 2 weeks, (ii) intraperitoneal administration of ML-7 (a specific myosin light chain kinase inhibitor), (iii) antibiotic administration in drinking water during 12 days. After PRS or sham-PRS session, we evaluated LPS levels in portal blood, plasma corticosterone and adrenocorticotropic hormone (ACTH) levels, hypothalamic corticotropin releasing factor (CRF) and pro-inflammatory cytokine mRNA expression, and colonic paracellular permeability (CPP).

RESULTS: PRS increased plasma ACTH and corticosterone; hypothalamic CRF and pro-inflammatory cytokine expression; CPP and portal blood concentration of LPS. L. farciminis and ML-7 suppressed stress-induced hyperpermeability, endotoxemia and prevented HPA axis stress response and neuroinflammation. Antibiotic reduction of luminal LPS concentration prevented HPA axis stress response and increased hypothalamic expression of pro-inflammatory cytokines.

CONCLUSION: The attenuation of the HPA axis response to stress by L. farciminis depends upon the prevention of intestinal barrier impairment and decrease of circulating LPS levels.
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