Objectives: Heat shock proteins (HSPs) are supposed to be protective for cells under stress. Physical stressors such as exercise or restraint stress also induce HSP in the gut in vivo. But, it is not clear how HSP is induced in the gut epithelial cells under stress. Since commensal bacteria is capable of inducing HSP in the gut epithelial cells through Toll-Like-Receptors signaling, we hypothesized commensal bacteria is responsible for stress-induced HSP induction in vivo. We tested if commensal bacteria depletion by antibiotics treatment would affect HSP induction in restrained mice.

Methods: Male C57BL/6 mice at 10-12 weeks of age were randomly assigned to six groups: non-stressed & vehicle administered control (V-CON), non-stressed & antibiotics treated control (A-CON), stressed & vehicle administered (V-RS), stressed and antibiotics treated (A-RS), non-stressed & lipopolysaccharide (LPS) after antibiotics treatment (A-LPS-CON), stressed & LPS after antibiotics treatment (A-LPS-RS). Bacterial depletion was performed by adding cocktail of antibiotics in drinking water for 1 week. Additionally, we gave the same antibiotic cocktail solution (0.2cc/day) or the same amount of sterile water once a day through a gastric insert for 1 week. After treatment, mice in the stressed groups were restrained in a 50 ml centrifuge tube for 2 hours. Mice were sacrificed and gut HSP70 expression was quantified by ELISA and examined by immunohistochemistry.

Results: Two hours after restraint stress, gut HSP70 expression of V-RS mice were increased significantly than V-CON. Gut HSP70 expression of A-CON mice was reduced when compared to V-CON. Additionally, the level of gut HSP70 expression of A-RS mice was similar to A-CON. Reduced gut HSP70 expression by antibiotics was rescued by oral administration of LPS.

Conclusions: These results suggested that HSP70 augmentation in the intestinal epithelial tissue induced after stress depends upon commensal bacteria.

Keywords: Mice, Stress