Title	**Lactobacillus rhamnosus GG modulates intestinal barrier function and inflammation in BALB/C mice following dietary exposure to deoxynivalenol and zearalenone through changes in gut**
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LACTOBACILLUS RHAMNOSUS GG MODULATES INTESTINAL BARRIER FUNCTION AND INFLAMMATION IN BALB/C MICE FOLLOWING DIETARY EXPOSURE TO DEOXYNIVALENOL AND ZEARALENONE THROUGH CHANGES IN GUT MICROBIOTA

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Deoxynivalenol (DON) and zearalenone are mycotoxins produced by *Fusarium* species, which naturally co-occur in foods and feeds. The gastrointestinal tract represents the first barrier met by exogenous food/feed compounds. The purpose of the present study was to investigate the ability of *Lactobacillus rhamnosus* GG (LGG) to improve intestinal barrier functions and ameliorate inflammation in Balb/c mice (6 weeks old) fed diets containing mycotoxin mixtures (i.e. DON and ZEA) through modulation of intestinal bacterial compositions. An exposure regimen which stimulated the human exposure experience was designed. Two different protocols that vary the time-points of oral administration of LGG (1 x 10^8 CFU per day) were used to determine whether it could prevent and treat unwanted effects induced by DON (12 μg/g) and ZEA (0.5 μg/g). Chronic ingestion of DON and ZEA induced histological changes, reduced several tight junction protein gene expression such as claudin (Cldn)-1, Cldn-3, Cldn-4, Cldn-5, β-catenin (Ctnnb-1) and occludin (Ocln) in different intestinal segments accompanied by increases in plasma D-lactate and endotoxin levels. At the end of the experiment, plasma cytokine (TNF-α, IL-1β, IL-6, IFN-γ and IL-8) and serum immunoglobulin levels (IgA, IgG and IgM) were also assessed by ELISA and some of them were significantly up-regulated. Supplementation of LGG before and after DON and ZEA exposure in mice can improve, in certain extent, the intestinal barrier functions in mice following mycotoxin exposure, as shown by increased tight junction protein expression, improvement of the local intestinal immune function as well as inhibition of inflammatory responses in the intestine. These changes may be associated with changes in gut microbiota composition.