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INFLAMATORY RESPONSE OF JEJUNAL EXPLANTS FROM PIGLETS EXPOSED TO LPS, DEOXYNIVALENOL, AND LPS + DEOXYNIVALENOL

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The resident gut microbiome is one of the most abundant sources of bacterial lipopolysaccharides (LPS) in animals, which are potent inflammatory inducers and may negatively affect. Deoxynivalenol (DON) is a trichothecene mycotoxin that selectively target tissue with a high mitotic index such as intestinal tissue, inducing inflammatory signaling. DON naturally occur in cereals used in swine production. Once the gastrointestinal tract represents the first barrier met by compounds such as DON and LPS, we investigated the effect of DON, LPS, and DON+LPS on COX-2 and TNF-α immunohistochemistry staining in jejunal explants from piglets. The jejunum of three piglets was segmented into four parts (2 cm^2), washed with PBS and antibiotics, and randomly assigned to one of the treatments. The jejunal explants were maintained in Dulbecco's Modified Eagle Medium (DMEM) and exposed according to the following four treatments; T1 - control; T2 - 2µg/mL *E. coli* LPS; T3 – 46 µM DON; T4 – T2 + T3. After 1 h of explants exposure, the segments were washed with PBS, fixed in 10% neutral buffered formalin solution, serially exposed to graded alcohol concentration, and embedded in paraffin. The tissue microarray technique (TMA) was applied on the paraffin blocked tissue and sections were examined for immunohistochemical staining of COX-2 and TNF-α. Comparisons were assessed using the Student t -test with unequal variance (P<0.05). COX-2 immunohistochemistry staining shown greater (P<0.05) percentage of marked area promoted by DON (3.85±1.33), LPS (1.68±0.23), and DON+LPS (7.34±4.36) exposure compared to control (0.88±0.52). DON (17.75±4.89), LPS (9.57±1.37), and DON + LPS (14.02±2.22) exposure compared to control (3.29±1.93) also increased TNF-α percentage. LPS+DON combination potentiated (P<0.05) the % COX-2 (7.34±4.36 vs. 1.68±0.23 LPS) and % TNF-α (14.02±2.22 vs. 9.57±1.37 LPS) compared to LPS. DON, and LPS are potent inflammatory inducers and its co-exposure can amplify inflammatory response.