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HEAT STRESS INDUCED CHANGES ON INTESTINAL PERMEABILITY, ENDOTOXIN TRANSLOCATION, AND INFLAMMATORY PARAMETERS IN GROWING PIGS

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Heat stress antagonizes livestock production due to increasing temperatures and frequencies of extreme climatic events. During heat stress, the gastrointestinal tract is one of the first organs affected. One of the major concerns is increased intestinal barrier dysfunction and the disruption of gut barrier function, which allows pathogens and immunogenic compounds such as endotoxins to enter the body and blood stream. In order to evaluate the effects of heat stress on gut permeability, endotoxin translocation and inflammatory biomarkers, three trials were performed.

In the first trial, animals were either kept at thermoneutral conditions (28°C) or under diurnal heat stress conditions (6 hours at 38°C; 18 hours at 32°C /day for 3 days). Pigs were euthanized and ileal permeability was assessed *ex vivo* with modified Ussing chambers. In addition, serum endotoxin concentrations were measured. In the second trial, same conditions were used to evaluate the concentrations of acute phase proteins as well as liver parameters. In the third trial, a milder heat stress was applied (6 hours at 35°C; 18 hours at 32°C /day for 3 days) and gut permeability was assessed with a non-invasive dual sugar assay.

Respiratory rate and rectal body temperature were significantly increased in pigs under heat stress conditions in all trials. Furthermore, modified Ussing chamber assays as well as the non-invasive sugar assay showed that the gut barrier was significantly impaired due to the heat stress conditions. A significant increase of endotoxin activity in the blood was observed in the first trial. In addition, there was a significant effect of heat stress on acute phase protein levels in the blood.

Results confirm that heat stress leads to increased intestinal permeability and dysfunction in young pigs. Due to this heat stress-induced disruption endotoxins were able to enter the blood flow, which further affected the immune response.

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