Surplus dietary tryptophan reduces plasma urea concentrations in endotoxaemic pigs

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Introduction

The essential amino acid tryptophan not only serves as building block for protein synthesis but also affects many physiological pathways in the body through modulation of serotonin, a neurotransmitter involved in organ and tissue functionality. Tryptophan is the immediate precursor for serotonin synthesis, and as such, dietary tryptophan is able to affect whole body serotonin production by mass action (Koopmans et al, 2006; Lallès et al, 2009). Previously we have shown that surplus dietary tryptophan exerts positive effects on stress, behavior, endocrinology, immunology and intestinal morphology in post-weaning and/or juvenile pigs (Koopmans et al, 2005 and 2012). Infection and inflammation lead to increased degradation of serotonin and increased sickness-behaviour and catabolism (Richard et al, 2009). We hypothesized that supplementation of surplus dietary tryptophan may alleviate impaired life functions during inflammation. The objective of the present study was to compare the effects of suboptimal versus surplus dietary tryptophan on performance, gut robustness and clinical health in a pig model of intraperitoneal endotoxaemia.

Materials and methods

In total, 44 crossbred piglets were weaned at 4 weeks of age (ca 8 kg BW) and housed individually over a period of 3 weeks. Three dietary L-tryptophan levels were compared: 1) sub-optimal (basal diet containing ~1.8 g TRP/kg diet), n=14; 2) optimal (basal diet plus 0.3 g L-TRP), n=16; 3) surplus (basal diet plus 5 g L-TRP), n=14 ; in control and endotoxaemic piglets. LPS from E Coli was injected in half of the pigs on days 4, 7, 11 and 14 at doses of 2, 16, 32 and 64 µg/kg, respectively and subsequently blood and gut tissue was collected from all pigs on days 18-22.

Results

LPS caused a transient fever response (+1.5 ^{o}C), reduced feed intake by ~15% and increased plasma C-reactive protein (CRP) concentrations by ~40% (all $p<0.05$) but TRP did not affect the fever response, feed intake, body weight gain nor CRP. However, TRP reduced plasma urea concentrations by ~30% ($p<0.01$) in LPS-treated but not in control piglets at identical feed intake. Furthermore, TRP increased the weight of chyme in the ileum ~2.5-fold, increased the empty weight of the duodenum by ~17%, and increased baseline body temperature by +0.6 ^{o}C (all $p<0.05$) in all piglets.

Conclusion

TRP is a bioactive amino acid which protects from whole body protein catabolism during inflammation, modulates passage time of digesta in the small intestine, increases the anabolism of the duodenum, and increases baseline body temperature.

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