Differential expression of Toll-like receptor 4 signaling pathway genes in Escherichia coli F18-resistant and – sensitive Meishan piglets

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Abstract

The Toll-like receptor 4 (TLR4) signaling pathway is an important inflammatory pathways associated with the progression of numerous diseases. The aim of the present study was to investigate the relationship between TLR4 signaling and resistance to Escherichia coli F18 in locally weaned Meishan piglets. Using a real-time PCR approach, expression profiles were determined for key TLR4 signaling pathway genes TLR4, MyD88, CD14, IFN-α, IL-1β and TNF-α in the spleen, thymus, lymph nodes, duodenum and jejunum of E. coli F18-resistant and -sensitive animals. TLR4 signaling pathway genes were expressed in all the immune organs and intestinal tissues, and the expression was generally higher in the spleen and lymph nodes. TLR4 transcription was higher in the spleen of sensitive piglets (p<0.05), but there was no significant difference in TLR4 mRNA levels in other tissues. Similarly, CD14 transcription was higher in lymph nodes of sensitive animals (p<0.05) but not in other tissues. IL-1β expression was higher in the spleen and in the duodenum of resistant piglets (p<0.05, p<0.01, respectively), and there were no significant differences in other tissues. There were also no significant differences in the expression of MyD88, TNF-α and IFN-α between sensitive and resistant piglets (p>0.05). These results further confirm the involvement of the TLR4 signaling pathway in resistance to E. coli F18 in Meishan weaned piglets. The resistance appeared to be mediated via downregulation of TLR4 and CD14, and upregulation of MyD88 that may promote the release of cytokines TNF-α, IL-1β, IFN-α and other inflammatory mediators which help to fight against E. coli F18 infection.

Key words: pig, TLR4, cytokine, immune response, E. coli F18

Introduction

Post-weaning diarrhea (PWD) in piglets is the most frequent acute and fatal disease affecting the swine industry at present, and the enterotoxigenic Escherichia coli (ETEC) F18 strain is the main pathogen causing the disease (Imberechts et al. 1992, Rippinger et al. 1995, Imberechts et al. 1997). The F18 fimbriae expressed by porcine toxigenic E. coli strains are comprised of 1-2 um long filaments that mediate adhesion to enterocytes and once adhered, bacteria enter the cell where they multiply and mature, releasing