



Sheep CRC Postgraduate 2014 Conference Proceedings

Document ID:	SheepCRC_34_9
Title:	Pilot study on Antimicrobial resistance of Escherichia coli in piglets in Australian commercial pig herds
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Key words:	Pigs; disease in pigs;

This paper was presented at the Sheep CRC Postgraduate Conference held in 2014, as part of the presentations. The paper should be cited as:

L. van Breda, M. Ward, O. Dhungyel (2014) – *Pilot study on Antimicrobial resistance of Escherichia coli in piglets in Australian commercial pig herds*

Pilot study on Antimicrobial resistance of *Escherichia coli* in piglets in Australian commercial pig herds.

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The Australian pig industry commonly uses antimicrobials to fight diseases such as pathogenic *Escherichia coli* (*E. coli*) disease that causes severe diarrhoea in weaner and suckling piglets. Piglet diarrhoea is a cause for substantial concern as significant production losses are experienced, including reduced growth rates, high medication costs and high levels of mortality and morbidity (Fairbrother *et al.* 2005). Antimicrobial resistance (AMR) limits the effectiveness of antimicrobials used to combat bacterial diseases in pigs as well as impacting human health. AMR genes can be transferred from pigs to humans, directly via cross transmission or indirectly via transfer of mobile genetic elements (Wooldridge, 2012). Increasing and indiscriminate use of antimicrobials, can induce drug resistance and reliance on newer drugs (similar to higher importance drugs used for human health), can lead to possible emergence of resistance clones with additional virulence factors which would require immediate investigation (Smith *et al.* 2010).

The aim of this pilot study was to identify and determine virulence genes and AMR patterns of *E. coli* disease in South-eastern Australian pig herds.

Faecal samples were collected off the floor from 6 farrow-to-finish farms over a 3-month period (August–October 2013) to determine pathogenic *E. coli* virulence genes and AMR patterns. From each farm 50 samples were collected, 10 from pre-weaned and 40 from post-weaned piglets. Selective culture for haemolytic and non-haemolytic *E. coli* was performed on Sheep blood agar and also by selective enrichment in Buffered-peptone water. DNA extractions were performed using the boiling technique and PCR were performed on 16S gene for *E. coli* isolate confirmation. Twenty-five isolates were randomly selected to test for common virulence genes, fimbriae genes (F4, F18) and enterotoxin genes (STb, STa and LT) and AMR was performed using MIC testing.

All isolates were 100% resistant to Clindamycin and Tilmicosin, with strong evidence of emerging resistance to Pencillin, Sulphamethoxamine, Apramycin and Florfenicol across all 6 farms. All isolates were 100% susceptible to Enrofloxacin and Gentamicin. The most prevalent virulence genes were F18 and STb, which were present in 3 out of the 6 farms. There was also an indication of higher AMR patterns in farms where there was a presence of more virulence genes.

AMR *E. coli* strains were present at farms displaying both clinical and sub-clinical cases of diarrhoea and in both pre and post-weaned piglets. Survival of these strains in the environment could possibly lead to further spread of infection and emergence of more virulent clones, warranting further investigation.

Fairbrother, J.M. *et al.* (2005). *Anim Health Res Rev.* **6**(1): 17-39.

Smith, M.G. *et al.* (2010). *Vet Microbiol.* **145**(3-4): 299-307.

Wooldridge, M. (2012). *Rev Sci tech Off Int Epiz.* **31**(1):231-247.