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Guest Editorial

Influences on antimicrobial resistance: More than bugs and drugs

The interplay of host, agent and environmental factors is often discussed in the context of infectious disease in animal populations, yet this interaction is less frequently considered in relation to the occurrence of antimicrobial resistance in such groups. Instead, when discussing antimicrobial resistance, attention is usually focussed on the relationship between antimicrobial use and resistance. Whilst antimicrobial use is undoubtedly the primary selective pressure for the emergence and persistence of antimicrobial resistance, and might be considered as a 'host' factor, the role played by other host, agent and environmental factors in the occurrence of resistance is often overlooked. The influence of such factors is demonstrated by Dr Karla Cameron-Veas and colleagues, of the Centre de Recerca en Sanitat Animal (CReSA), Barcelona, Spain, in their paper on excretion of cephalosporin resistant *Escherichia coli* in pigs treated with antimicrobial agents, published recently in *The Veterinary Journal* (Cameron-Veas et al., 2016).

In this study, the selective effect of use of antimicrobial agents was demonstrated by an increase in the numbers of cephalosporin resistant *E. coli* (CR-*E. coli*) in faeces following the use of a single injection of ceftiofur at a therapeutic dose in piglets already colonised with CR-*E.coli*. However, treatment with ceftiofur was not a significant predictor of the occurrence of resistance in the study group as a whole. Instead, farm of origin (an environmental factor) had the greatest influence on the occurrence of cephalosporin resistance, with the selective pressure of antimicrobial use only exerting an effect in those herds where CR-*E. coli* was already present. Furthermore, the study showed that the proportion of animals shedding CR-*E. coli* and the numbers of CR-*E. coli* shed decreased with animal age (a host factor).

Resistance to third generation cephalosporins in *E. coli* is mediated by a number of β-lactamases, the extended spectrum β-lactamase (ESBL) and acquired AmpC-type β-lactamases being