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Short communication

Re-challenge of pigs following recovery from proliferative enteropathy

A.M. Collins [a,*], R.J. Love [b]

[a] NSW Department of Primary Industries, Elizabeth Macarthur Agricultural Institute, PMB 8, Camden 2570, Australia
[b] University of Sydney, Faculty of Veterinary Science, PMB 3, Camden 2570, Australia

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Abstract

An experimental challenge model was developed to demonstrate *Lawsonia intracellularis* colonization and reproduction of proliferative enteropathy (PE) in naïve weaner pigs. Groups of pigs were orally dosed with between 10^{10} and 10^{5} *L. intracellularis* extracted from haemorrhagic PE affected mucosa. Pigs were monitored for clinical signs and intestinal lesions of PE and evidence of bacterial colonization by serology and faecal polymerase chain reaction (PCR). One group of challenged pigs were necropsied after 21 days to confirm the reproduction of PE. *L. intracellularis* colonization and seroconversion was delayed in pigs dosed with lower numbers of *L. intracellularis*. When faecal shedding of *L. intracellularis* ceased to be detected in all of the challenged pigs, they were re-dosed orally with approximately 10^{10} *L. intracellularis* and monitored for evidence of re-colonization and clinical disease. This study demonstrated that pigs previously challenged with *L. intracellularis* were protected from re-colonization and clinical disease on subsequent exposure 10 weeks later, regardless of the initial dose of *L. intracellularis*.
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Keywords: *Lawsonia intracellularis*; Colonization; Protection; Faecal shedding; PCR

1. Introduction

Proliferative enteropathy (PE) affects pigs of all ages across a broad range of management systems. Clinical presentations of PE vary from reduced weight gains and diarrhoea in growing pigs, to the more acute proliferative haemorrhagic enteropathy (PHE) in mainly finisher or breeding animals, with sudden death in severely affected animals (Lawson and Gebhart, 2000). The aetiologic agent of PE is *Lawsonia intracellularis*, an obligate intracellular bacterium. Both clinical presentations of PE exhibit similar histopathology with intracellular *L. intracellularis* found within the cytoplasm of proliferating immature enterocytes (Lawson and Gebhart, 2000). Control of PE is either by medication with antibiotics, or vaccination with an avirulent live *L. intracellularis* vaccine (Enterisol® Ileitis, Boehringer Ingelheim).

* Corresponding author. Tel.: +61 2 46 406 306; fax: +61 2 46 406 384.
E-mail address: alison.collins@dpi.nsw.gov.au (A.M. Collins).

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Vaccinated pigs challenged with a virulent heterologous *L. intracellularis* strain showed significant reductions in microscopic lesions of PE compared with non-vaccinated and challenged pigs (Kroll et al., 2004). However, vaccination did not prevent *L. intracellularis* colonization, as demonstrated by faecal shedding of *L. intracellularis*. Prevention of *L. intracellularis* colonization has been achieved by the continuous medication of pigs with high levels of antibiotics, but this practice carries the risk of antibiotic resistance developing in bacteria. This study aimed to determine if pigs could be protected from *L. intracellularis* colonization and disease if previously dosed with high or low numbers of *L. intracellularis* extracted from haemorrhagic PE affected mucosa.

2. Materials and methods

2.1. Experimental design

Animal experiments were performed according to the Australian Code of Practice for the Care and Use of Animals for Scientific Purposes, and approved by the Institute's Animal Ethics Committee. Large White, Landrace cross pigs were weaned at 3–4 weeks of age and transferred to a cleaned and disinfected research facility, with free access to water and a commercial weaner and grower diet without medication. Each group of pigs was housed in well-separated weaner pens, elevated off the floor, with quarantine restrictions between pens. Pigs were allowed several days to acclimatise to the accommodation prior to experimentation.

A Student's *t*-test was used to compare weekly weight gains of challenged and non-challenged pigs, or the weight gains of pigs inoculated with different numbers of *L. intracellularis* in the dose response study.

2.1.1. Demonstration of L. intracellularis colonization and disease

Sixteen pigs were randomly assigned into two groups of eight pigs containing equal numbers of males and females. The mean initial weights of Groups 1 and 2 pigs were not significantly different. Following oral dosing of Group 1 pigs with *L. intracellularis*; all pigs were monitored daily for clinical signs of disease and were weighed weekly over a 4 week period. Blood was collected for 6 consecutive weeks and individual faecal samples were collected directly from each pig two to three times per week for 4 weeks, using clean gloves for each sample. Four Group 1 pigs and four Group 2 pigs were euthanased at 21 days post dosing and intestinal sections were preserved for histopathology, including Warthin Starry silver stains. Sections of ileum, colon and mesenteric lymph nodes were also collected for the more sensitive and specific polymerase chain reaction (PCR) amplification of *L. intracellularis* DNA.

2.1.2. Dose response to protection from clinical disease and colonization

In a second study, 20 weaned pigs were randomly distributed into 4 groups of 5 pigs each (Groups 3–6). The mean initial weights were not significantly different between the groups. A seventh group of six weaned pigs (4 weeks of age) were introduced into a cleaned pen 10 weeks after the experiment began. Pigs in Groups 3–5 were dosed orally with a suspension containing decreasing numbers of *L. intracellularis* (approximately 10^{10}, 10^{7}, and 10^{5} *L. intracellularis*, respectively) and Group 6 pigs were left as undosed controls. Pigs were monitored daily for clinical signs of disease, blood was collected weekly and individual faecal samples were collected two to three times per week for 10 weeks post dosing. At day 70 post dosing, pigs in Groups 3–5 were re-dosed orally with approximately 10^{10} *L. intracellularis* and the naïve Group 7 pigs were dosed for the first time with the same inoculum. All pigs were monitored for bacterial colonization and clinical disease over 4 weeks as outlined above.

2.2. Preparation of inoculum, and oral dosing of pigs

L. intracellularis organisms were extracted from the mucosa of a naturally infected finisher pig with gross and histological lesions of PE (2189/94). This was done by a method also used for the successful *in vitro* culture of *L. intracellularis* (Collins et al., 1996). The approximate number of *L. intracellularis*/mL of inoculum were determined by indirect IFA of a known

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