

Short communication

Reproductive performance of gilts following an outbreak of acute proliferative enteropathy due to *Lawsonia intracellularis*

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Abstract

After a sudden outbreak of clinical porcine proliferative enteropathy (PPE) in a Croatian indoor breeding unit, the farm experienced decreased reproductive performance of *Lawsonia intracellularis* positive gilts. Conception-, farrowing-, and adjusted farrowing rates were lower ($P < 0.001$) in gilts with positive *L. intracellularis* status. The number of live born and the total born litter size were significantly lower ($P < 0.001$) in *L. intracellularis* positive gilts compared to seronegative ones. No differences were observed in numbers of stillborn and mummified pigs between the *L. intracellularis* positive and negative gilts.

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Ileitis is a common enteric disease of growing-finishing pigs and may result in poor growth rate, diarrhoea, stunting or may be manifested as sudden death or bloody diarrhoea in late finishing pigs and replacement gilts (Lawson et al., 1979). Porcine proliferative enteropathy (PPE) is caused by *Lawsonia intracellularis* (McOrist et al., 2003) and occurs in virtually all swine production systems (Stegel et al., 2000). According to McOrist and Gebhart (1999), approximately 30% of pig herds worldwide are infected by PPE. Just et al. (2001) reported a seroprevalence of up to 54% in pigs <25 weeks of age in large commercial units.

The disease has different clinical forms, namely porcine intestinal adenomatosis (PIA), currently seen in growing pigs, and proliferative haemorrhagic enteropathy (PHE) in fattening pigs and young breeding animals (McOrist et al., 2003). Epidemiological factors responsible for variation in the prevalence and severity of *L. intracellularis* infection under field conditions are incompletely understood (Just et al., 2001). Various

farm management factors such as the movement of pigs, nutritional changes, feed antibiotic usage, temperature fluctuations, pig density, pig age, facility design, sanitation, immune status, resistance, genetic susceptibility and outdoor raising may influence the development and severity of PPE (Hagen and Bilkei, 2003; Bona and Bilkei, 2003). In breeding units, replacement animals from the performance testing stations have been associated with PPE outbreaks (Bilkei, 1995). In pregnant gilts and sows acute PHE may cause abort within six days of the onset of clinical signs (Beers, 1984).

The present study was performed in a Croatian indoor breeding unit of 800 sows. The breeding females were F1 Large White X Landrace and were mated to Duroc boars. The gilts and sows received during pregnancy erysipelas, pseudorabies, leptospirosis and *Escherichia coli* vaccination. The gilts were housed in groups of 8–15. The sows were housed during gestation in groups (6–10) and during lactation in identical large farrowing crates. All reproductive phases were managed in “all-in-all-out” fashion. The annual culling rate of the breeding sows in this unit was low (32.2%). Litters were weaned at 25 ± 1.9 (SD) days of lactation. Prior to the

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PPE outbreak, the feed and water provided to the pigs did not contain antibiotics.

During February–March 2003, the farm experienced a sudden outbreak of clinical PPE (PIA [diarrhoea, stunting, dullness, apathy, unthriftiness] and PHE [bloody diarrhoea or sudden death in gilts]). PPE was confirmed at necropsy and by indirect immunofluorescence antibody (IFA) (following venipuncture, blood samples were refrigerated and shipped to a veterinary investigation laboratory, Vet Invest, Zagreb Croatia, where faecal polymerase chain reaction [PCR] tests were also conducted). Concurrently positive IFA and PCR were interpreted as acute *L. intracellularis* infection.

The outbreak was (most probably) caused by recently introduced *L. intracellularis*-IFA-positive ($n = 32$ of 93 replacement gilts) pregnant replacement animals of 301–335 days of age and average body condition of 3.02 ± 0.11 (Bilkei, 1995) from the same source. Within two weeks post infection 21/93 late pregnant gilts aborted, four died, and two were culled due to weakness, high fever and lateral recumbency. The gilts that aborted had profuse bloody diarrhoea and fever (40.6 ± 0.3 °C). The reminding 62 pregnant gilts had slight diarrhoea without blood and had no fever. Older sows (average parity 3.45 ± 0.9 , average body condition 3.27 ± 0.22 in this unit) were not affected by the disease. One pregnant gilt was culled during the study due to locomotor problems. The disease was immediately treated (February 21–March 14, 2003) with in-feed application of oxytetracycline (23 mg/kg body weight) for three weeks. The gilts reproductive performance (March–May 2003) was retrospectively evaluated.

According to Knittel et al. (1998) the sensitivity of IFA is estimated to be 0.90 and the specificity 0.99. Therefore ten percent of the sows were serologically tested for *L. intracellularis*. Blood samples were collected (February–March, 2003) from late pregnant gilts ($n = 61$) and sows of parity 2–7 (3.3 ± 1.1 , $n = 59$) refrigerated and shipped to Vet Invest. *L. intracellularis* IFA (to detect anti-LI IgG and IgM antibodies; Knittel et al., 1998) and PCR (Cooper et al., 1997) were performed. All gilts that recovered from clinical disease ($n = 61$) showed seropositivity for *L. intracellularis*. Sows of parity 2–7 (3.3 ± 1.1) showed <5% *L. intracellularis*

seropositivity, and were excluded from further evaluation. Non-infected gilts ($n = 61$) of same age, body condition and reproductive status were used as control animals (Table 1).

Infected and non-infected gilts' entry date, age at first mating, return to oestrus on day 21 post-mating, farrowing rate, litter size (total born and live born), stillbirth- and mummy rate were compared. According to previous experience in this unit (and geographic area), females inseminated during November and January had lower seasonal conception and farrowing rates. Data were evaluated during a five month period (December 30, 2002 till May 31, 2003).

A CATMOD procedure was used to analyse fertility parameters (dependant variables) namely 21-day conception rate, farrowing rate, adjusted farrowing rate (number of females farrowed, divided by number of female bred minus number of females culled or died due to non-reproductive reasons). Independent variables included farm and breeding month. The GLM procedures of SAS were used for analysis of litter size data. Dependent variables were the number of total born, number of liveborn pigs, number of stillborn pigs and the number of mummified pigs. An independent variable was the month of conception. Age at first mating (days) treated as a continuous variable, was included in the model for gilt data. Least-squares means were requested with 'Ismeans' following the model statement. Standard errors for the means and probability comparisons between means were requested using options or "stderr" and "pdiff", respectively. In the statistical model used, effects of months were observed ($P < 0.001$) and were consistent with the seasonal effect, previously experienced in this unit.

L. intracellularis status was associated ($P < 0.001$) with conception rate, farrowing rate, and adjusted farrowing rate (Table 1). Conception, farrowing, and adjusted farrowing rates were significantly lower ($P < 0.001$) in gilts with positive *L. intracellularis* status (Table 1). The number of live born and total born litter size were significantly lower ($P < 0.001$) in *L. intracellularis* positive gilts compared to seronegative ones (Table 2). No differences were observed in numbers of stillborn and mummified pigs between the *L. intracellularis* positive and negative gilts (Table 2).

Table 1

Fertility of *L. intracellularis* positive ($n = 15$ of 61 gilts) and negative ($n = 46$ of 61 gilts) (tested by immunofluorescence antibody [IFA] and polymerase chain reaction [PCR]) gilts, following a field outbreak of acute porcine proliferative enteropathy in a large indoor production unit

	<i>L. intracellularis</i> positive N/Nn = %	<i>L. intracellularis</i> negative N/Nn = %	P-value
Conception rate	301/280 = 93.0	282/272 = 96.5	0.001
Farrowing rate	301/198 = 65.8	282/218 = 77.3	0.001
Adjusted farrowing rate	301/222 = 73.8	282/236 = 83.7	0.001

N, number of gilts.

Nn, number of conceived gilts.

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