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Effect of *Ascaris suum* infection on performance of fattening pigs

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ABSTRACT

Scientific investigations of production losses in pigs due to roundworms are rarely conducted in commercial farms, despite the fact that this information is needed to decide whether anthelmintic treatment is cost-effective or not. Therefore, the aim of our study was to compare performance in fattening pigs treated or not treated for *Ascaris suum* infections. Two Danish pig farms producing fatteners and showing *A. suum*-induced liver white spot prevalences of 10–33% were selected for the study. In each farm, pens with fattening pigs were randomly assigned to either treatment with an anthelmintic (Flubenol®, Janssen Animal Health), or a placebo. Pigs were treated by administering anthelmintic or placebo mixed in the feed for five consecutive days (5 mg/kg body weight) on day 2–6 and day 36–40 after introduction to the finishing unit. Fecal egg excretion before first shipment, liver lesion scores (white spots), lean meat percentage at slaughter, average daily gain, and feed conversion were recorded weekly per pen and evaluated for the entire fattening period (30–100 kg). *A. suum* egg excretion was detected in none of the 57 pens where pigs were treated with anthelmintic compared to 18.2% of 57 pens in the placebo group. Pen floor fecal sampling underestimated the prevalence of *A. suum* in the fattening units compared to individual rectal sampling; only 22% of pens where *A. suum* was present were diagnosed correctly by both sampling methods. The prevalence of white spots did not differ significantly between pigs treated with anthelmintic (26.7%) and pigs treated with placebo (23.7%), but considerable variation between batches and over time was observed. Liver lesion scores (none, few, moderate, many) were not significantly influenced by de-worming twice during the fattening period. There were no significant differences in average daily gain, feed conversion and lean meat percentage between pigs treated with anthelmintic or placebo. This lack of effect may be explained in part by a rather low infection intensity, as measured by fecal egg counts, but in contrast, white spot numbers were moderate to high. A possible negative influence of other disease, mainly diarrhea due to *Lawsonia intracellularis*, on performance could not be excluded. Treatment with flubendazole twice during fattening prevented *A. suum* egg excretion but did not reduce the occurrence of liver white spots. To improve performance significantly, repeated treatments over several consecutive rounds might be necessary.

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1. Introduction

Infections with the parasitic roundworm *Ascaris suum* are common in pigs worldwide (Nansen and Roepstorff, 1999). In Denmark, *A. suum* is by far the most prevalent helminth species in pig production, infecting the majority of herds (Roepstorff and Jorsal, 1989; Roepstorff et al., 1998). Infection with *A. suum* may affect all age groups of pigs but housing and management factors often determine which age group shows the highest prevalence. In Denmark and other Nordic countries it has been observed that in conventionally managed herds with standard hygiene, solid floors and use of bedding material, *A. suum* occurs most frequently in weaners and fattening pigs (Roepstorff et al., 1998). In contrast, in more intensively managed pig herds with good hygiene and slatted floors, *A. suum* is most prevalent in sows and boars (Roepstorff, 1997).

A. suum infections rarely cause clinical disease in pigs (Hale et al., 1985; Eriksen et al., 1992). The acute phase of infection is characterized by frequent coughing, caused by migrating larvae in the lungs (Urquhart et al., 1996). Although the result may be loss of appetite and depression, these transient clinical signs of larval migration do not seem to affect productivity significantly (Hale et al., 1985). Instead it has been suggested that migrating *A. suum* larvae in the lungs may exacerbate lung infections caused by other pathogens (Flesjå and Ulvesæter, 1980; Adedeji et al., 1989; Nilsson et al., 1991; Bouwkamp et al., 2006).

Pigs harbouring patent infections, even with large numbers of adult worms, normally appear clinically healthy. However, it has been suggested that reduced performance in fattening pigs infected with *A. suum* may be due to patent infections, i.e. the presence of large, adult worms in the small intestine (Stephenson et al., 1980; Hale et al., 1985). A correlation between the number of adult worms present and reduced weight gain has been reported by several authors (Stephenson et al., 1980; Forsum et al., 1981; Hale et al., 1985). Experimental studies with three dose levels of *A. suum* have shown linear depression of both daily gain and feed efficiency compared to uninfected control pigs (Hale et al., 1985). In contrast, no difference in daily gain and feed efficiency was observed in pigs experimentally infected with *A. suum*, but naturally exposed pigs showed significantly reduced weight gains (Urban et al., 1989).

Economic losses due to *A. suum* infection in pigs are mainly incurred as (1) condemnation of livers at slaughter, (2) decreased daily gain and feed conversion efficiency, and (3) cost of anthelmintic treatment (Stewart and Hale, 1988). However, estimating the economic impact of *A. suum* infection on pig performance is not an easy task. Based on low-level experimental *A. suum* infections, Stewart and Hale (1988) estimated the economic loss to pig producers in the US from increased feed to gain ratio to US$ 155 million annually. More recent cost estimates are provided by Stewart (2001), who calculated an estimated loss of US$ 17.5 million due to liver condemnation at slaughter as well as an estimated loss of US$ 60.1 million for extra feed to finish pigs for slaughter.

Production losses in pigs due to *A. suum* worms have mainly been documented in controlled experiments. However, simultaneous comparisons of performance in pigs with and without worms have not been carried out in commercial pig farms, i.e. under natural exposure conditions, as this type of study is difficult to conduct. For example, comparing the performance of finisher pigs with and without *A. suum* infection in a naturally contaminated farm environment is almost impossible to achieve because all pigs will be exposed to infection, and it is not physically possible to remove infection from e.g. 50% of pens in a pig house. Therefore, most studies published to date deal with improving herd health and production following deworming of pigs, aiming to show that performance will improve with time and using the entire herd as its own (pre-treatment) control (Kanora et al., 2004; Jacobs et al., 2006). From a practical perspective, the latter approach is very relevant. However, the use of historical controls is doubtful from a scientific point of view, because many other factors that are not controlled for, might influence pig productivity during the treatment period. Therefore, we chose to conduct a clinical trial with a treatment group and a control group in each herd and each stable.

The aim of our study was to compare performance in finisher pigs infected with *A. suum* and treated with anthelmintic or placebo under commercial farm conditions.

2. Materials and methods

2.1. Study farms

The study was conducted between 1 May 2004 and 1 June 2005 in two Danish pig farms situated in northern (Farm A) and southern Jutland (Farm B).

Farm A was a conventional farrow-to-finish herd with free-range (i.e. outdoor) sows and confined (i.e. indoor) weaners and fattening pigs. Slaughter pigs were raised in four fattening units with all in/all out management. Each unit consisted of 14 pens containing 28 pigs each, all of which were fed restrictively with liquid feed. Pre-study examination of fattening pigs revealed 23% of fecal samples positive for *A. suum* 2 weeks prior to slaughter as well as 33% of livers with white spots at slaughter.

Farm B produced only fatteners, which were obtained from an *A. suum*-free supplier herd at a weight of approx. 30 kg. The farm consisted of one medium and one large fattening unit, of which only the large unit was included in the study. The large unit consisted of 22 pens containing 48 pigs each, all of which were fed restrictively with liquid feed. Pre-study examination of fattening pigs revealed 46% of fecal samples positive for *A. suum* 2 weeks prior to slaughter as well as 10% of pigs with liver white spots at slaughter.

2.2. Study design

The study was designed in such a way that if a minimum of 27 pens per treatment per farm were included (statistical power = 80%), a possible difference in average daily gain of 30 g between treatment groups could be detected (if present) with 95% confidence.

A total of 1624 (Farm A) and 2374 (Farm B) fattening pigs, produced in five and three batches, respectively,

were included in the study. Upon arrival in the fattening unit, pigs were weighed pen-wise. Subsequently, pens were randomly allocated to either anthelmintic treatment with flubendazole (Flubenol®, Janssen Animal Health) or treatment with placebo (lactose), but care was taken to balance total body weight per pen between both treatment groups. The study was conducted as a blind trial, which meant that both formulations were coded using different colours, and neither farm staff nor veterinarian had access to the code.

Pigs were treated by administering anthelmintic (5 mg/kg body weight) or placebo mixed in the feed for five consecutive days (Monday to Friday). The pigs were treated twice during the fattening period: during the 1st week (day 2–6) and the 6th week (day 36–40) of the fattening period. Treatments were carried out pen-wise, with each pen initially housing 28 (Farm A) or 48 (Farm B) pigs.

The fattening period in both herds was 12 weeks, i.e. pigs were sent for slaughter on Thursday in week 10, 11 and 12 of the fattening period at a live weight of approx. 100 kg. This meant that, in each batch, the last pigs were slaughtered 41 days after the second treatment, which is beyond the pretant period of *A. suum*. However, the withdrawal time for Flubenol® in Denmark was 30 days at the time of the study, which made it impossible to include a third treatment closer to the time of slaughter.

2.3. Parasitological examinations

Pooled pen floor fecal samples were collected in each pen 9 weeks after introduction to the fattening units, to establish if adult worm infections were present. The samples were examined using a modified McMaster method (Roepstorff and Nansen, 1998) with a detection level of five eggs per gram feces (EPG).

To compare the use of pooled fecal samples with the more frequently used individual fecal samples taken per rectum, individual rectal samples were collected from all pigs treated with placebo in batch 2 and 3 in Farm A and batch 3 in Farm B. The individual fecal samples were analysed using the McMaster method mentioned above.

At the abattoir, the liver of each pig was examined for the presence of white spots (WS) according to a semi-quantitative scoring system: score 0: no WS; score 1: 1–5 WS; score 2: 6–15 WS; score 3: >15 WS (Christensen and Enøe, 1999).

2.4. Performance parameters

Pigs were weighed pen-wise upon arrival at the fattening unit. At slaughter, individual carcass weight was recorded. Based on these data and adjusted for pigs that had been removed during the course of the study, average daily gain (ADG) during the fattening period (roughly from 30 to 100 kg live weight) was calculated at pen level. In addition, feed intake was registered for each pen once weekly. When a pig died or was removed from the pen, the amount of feed administered to the remaining pigs was adjusted accordingly. Finally, feed conversion rates were calculated at pen level for the entire fattening period. Lean meat percentage was recorded at slaughter on an individual basis.

2.5. Clinical examinations

The pigs were inspected daily by the farm staff and once a month by the practising veterinarian. The number and probable cause of individual treatments for other disease (e.g. diarrhea, lameness, tail biting), pigs moved to hospital pens, as well as mortality were recorded at pen level. Autopsy was performed by the veterinary practitioner on the majority of pigs that died on the farm during the study. Pigs removed from the study pens due to other disease were not returned to these pens during the study period.

2.6. Statistical analysis

The results were analysed using the SAS system for mixed models (Littell et al., 1996). For each herd, a mixed model (PROC MIXED) was fitted to the data using feed conversion as response variable—and a similar model was built for average daily gain and liver white spots. A P-value of <0.05 was used as criterion for including a variable into a model. The effect of interactions between explanatory variables was checked. As a model control, residuals arising from each model were inspected.

3. Results

3.1. Parasitological findings

In Farm A, both *A. suum* eggs and strongyle eggs were found whereas in Farm B only *A. suum* eggs were detected. None of the pigs from 57 pens treated with anthelmintic excreted helminth eggs in the feces during the entire study. In contrast, pigs in 18.2% of 57 pens treated with placebo showed positive EPG values prior to slaughter (Table 1). However, Table 1 also shows that marked differences in prevalence based on egg excretion in the placebo groups were observed between and within the two farms. Using pen floor samples, egg excretion was detected in 6.7% of placebo pens in Farm A, whereas in Farm B 29.6% of placebo pens were positive for *A. suum* eggs (Table 1). All egg-positive pen floor samples were very low, ranging from 5 to 65 EPG, except one sample containing 5000 EPG.

Based on rectal fecal samples taken in the placebo group, 16 of 299 (5.4%) fatteners in Farm A and 3 of 402 (0.75%) fatteners in Farm B were positive for *A. suum*. Comparison of pen floor samples and rectal samples revealed considerable discrepancy in number of placebo pens diagnosed positive for *A. suum* (Table 2a). Of nine pens that were egg-positive judged by rectal samples out of a total of 21 pens, only two were diagnosed positive based on pen floor samples. Average EPG in *A. suum*-positive individual rectal samples was 750 EPG (range: 5–5000 EPG), and samples with high EPG values were collected in several pens with negative pen floor samples (Table 2b).

Strongyle eggs were found in Farm B in 14 of 299 (4.7%) pigs, corresponding to 6 out of 12 (50%) pens. Identification of strongyle eggs using fecal cultures was not attempted, but based on available reports from Denmark (Roepstorff et al., 1998) they were presumed to be *Oesophagostomum* spp. eggs.

Table 1
Fecal egg excretion and liver white spots due to *Ascaris suum* infection in fattening pigs treated with anthelmintic (flubendazole) or placebo during a clinical trial in two conventional Danish swine herds, 2004–2005.

Farm and batch	Fattening period	Egg excretion (week 9 of fattening)				White spots (week 10, 11 and 12 of fattening)			
		Flubendazole		Placebo		Flubendazole		Placebo	
		Pos. pens/ total pens	EPG range	Pos. pens/ total pens	EPG range	Pos. pigs/ total pigs	%	Pos. pigs/ total pigs	%
Farm A									
Batch 1	17 May–5 August 2004	0/6	0	0/6	0	51/163	31.3	45/158	28.5
Batch 2	7 June–26 August 2004	0/6	0	1/6	0–65	69/156	44.2	80/157	50.9
Batch 3	30 June–16 September 2004	0/6	0	1/6	0–10	67/147	45.6	60/138	43.5
Batch 4	19 July–7 October 2004	0/6	0	0/6	0	72/158	45.6	65/161	40.4
Batch 5[a]	9 August–28 October 2004	0/6	0	0/6	0	62/164	37.8	55/159	34.6
All A batches		0/30		2/30 (6.7%)		321/788	40.7	305/773	39.5
Farm B									
Batch 1	16 August–1 November 2004	0/9	0	4/9	0–5000[c]	69/293	23.6	55/291	18.9
Batch 2[b]	19 November 2004–16 February 2005	0/9	0	2/9	0–45[d]	52/276	18.8	18/294	6.1
Batch 3[b]	4 March–27 May 2005	0/9	0	0/9	0	10/326	3.1	18/313	5.8
All B batches		0/27		6/27 (29.6%)		131/895	14.6	91/898	10.1
Total (A + B)		0/57		8/57 (18.2%)		452/1683	26.7	396/1671	23.7

[a] In Farm A, batch 5 was housed in the same unit as batch 1, and pen treatment allocation was not changed between batches.
[b] In Farm B, batch 2 and 3 were housed in the same pens as batch 1, but pen treatment allocation was changed randomly between batches.
[c] Batch 1—positive EPG values: 5, 10 and 5000.
[d] Batch 2—positive EPG values: 10 and 45.

The occurrence of white spots in pigs from both farms at slaughter is shown in Table 1. On average, the prevalence of WS was slightly higher in pigs treated with anthelmintic compared to untreated pigs. In Farm A, a high prevalence of WS was recorded but the difference between the flubendazole group (40.7%) and the placebo group (39.5%) was not significant. In Farm B, WS prevalence was generally lower and overall a statistically significant difference ($P < 0.01$) was found between the flubendazole group (14.6%) and the placebo group (10.1%).

The distribution of liver lesion scores was not influenced by de-worming (Fig. 1). Within batches of pigs delivered to the abattoir, the distribution of liver lesion scores (i.e. the ratio between lightly, moderately and severely affected livers) was remarkably constant between pigs treated with flubendazole and pigs treated with placebo.

In contrast to Farm A, the prevalence of WS declined over time in Farm B (Fig. 2). This decline occurred earlier in the placebo group compared to the treatment group. A statistical analysis of batch 2 that was slaughtered in February 2005 revealed that WS prevalence was significantly lower ($P < 0.05$) in pigs treated with placebo compared to the treatment group.

Table 2a
Comparison[a] of detection of fecal egg excretion using pen floor samples or individual rectal samples in 21 untreated pens housing fattener pigs infected with *Ascaris suum*, 2004–2005.

Pen diagnosis for *Ascaris suum* egg excretion		Individual rectal fecal samples	
		Positive	Negative
Pen floor fecal sample	Positive	2	0
	Negative	7	12

[a] Fisher's exact test $P = 0.17$.

Table 2b
Ascaris suum egg excretion in untreated Danish fattener pigs. Comparison of pens with an *Ascaris*-positive pen floor fecal sample and/or at least one *Ascaris*-positive rectal fecal sample.

Pen No.	EPG value of pen floor sample	No. of positive EPG values in individual pigs			
		1	2	3	4
1	0	455	5	5	–
2	0	85	–	–	–
3	0	1225	–	–	–
4	10	5000	1610	1205	10
5	0	880	285	5	5
6	0	85	–	–	–
7	65	690	–	–	–
8	0	520	–	–	–
9	0	950	680	595	–

3.2. Performance

The average performance results for pigs treated with flubendazole and pigs treated with a placebo are shown in Table 3. There were no significant differences in slaughter weight and lean meat percentage between the flubendazole and placebo groups.

ADG was significantly influenced by weight at the start of the fattening period ($P < 0.0001$) and batch ($P < 0.0001$) in both farms. In contrast, treatment (flubendazole or placebo) and degree of worm infection (number of WS)

Table 3
Performance of 3932 Danish fattening pigs infected with *Ascaris suum* and treated with anthelmintic (flubendazole) or placebo, 2004–2005.

	Farm A		Farm B	
	Flubendazole	Placebo	Flubendazole	Placebo
Number of pigs	787	771	1160	1214
Carcass weight (kg)	74.2	73.9	76.9	77.1
Lean meat percentage (%)	60.9	60.9	60.9	60.9
Overall ADG[a] (g)	830	822	843	850
ADG batch 1	767	727	855	859
ADG batch 2	828	817	877	887
ADG batch 3	807	827	798	806
ADG batch 4	836	840	–	–
ADG batch 5	907	902	–	–
Overall FC[b] (kg/kg)	2.91	2.92	2.74	2.72
FC batch 1	2.87	2.92	2.70	2.67
FC batch 2	2.87	2.87	2.72	2.70
FC batch 3	2.94	2.90	2.81	2.79
FC batch 4	2.96	2.96	–	–
FC batch 5	2.94	2.94	–	–

[a] ADG: average daily gain.
[b] FC: feed conversion.

did not have a significant effect on ADG. Following correction for start weight and batch, the mean difference in ADG between flubendazole and placebo groups was +5.2 g in Farm A ($P = 0.46$) and −5.9 g in Farm B ($P = 0.33$).

Feed conversion was also significantly influenced by batch in Farm A ($P = 0.02$) and Farm B ($P < 0.0001$) but not by start weight. Again, treatment and degree of worm infection did not have a significant effect on feed conversion. After correction for batch, the mean difference in feed conversion between flubendazole and placebo groups was −0.003 kg feed/kg gain in Farm A ($P = 0.87$) and +0.015 kg feed/kg gain in Farm B ($P = 0.35$).

In Farm A, a marked improvement in ADG and feed conversion was observed during the course of the study. At the beginning of the study (batch 1 and 2) there was an apparent effect of treatment on ADG and feed conversion. However, the effect of treatment disappeared gradually as ADG and feed conversion improved in both treatment groups (Table 3). This improvement in pig performance coincided with the onset of mass treatment with antibiotics (batch 3, 4 and 5) following a period with diarrhea problems due to *Lawsonia intracellularis*. The difference in effect of treatment between batch on ADG and feed conversion was analysed statistically by checking for interactions between ADG and feed conversion on the one hand and batch on the other hand. No significant interaction was found ($P = 0.17$). This means that the difference in ADG and feed conversion observed between treatment groups could not be attributed to anthelmintic treatment.

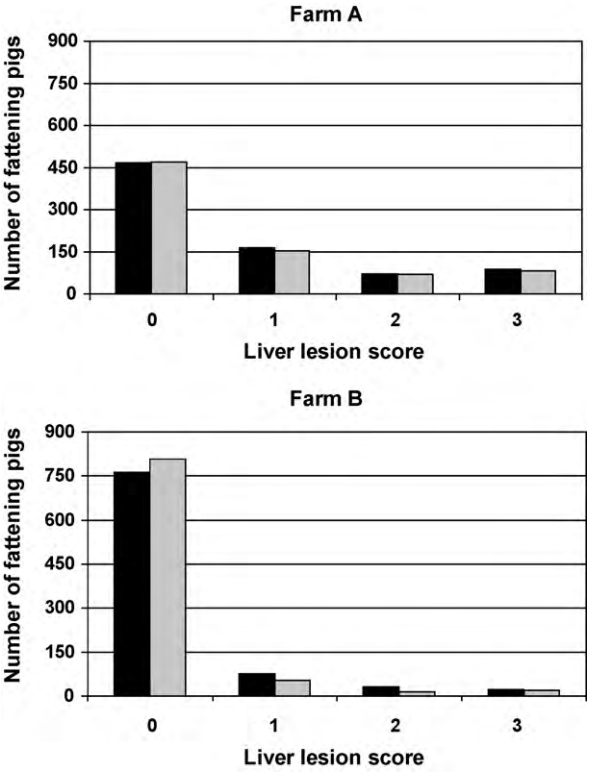

Fig. 1. Frequency of liver lesion scores (white spots, WS) at slaughter in 3354 Danish fattening pigs infected with *Ascaris suum* and treated with flubendazole (black bars) or placebo (grey bars) in two Danish pig herds (Farm A and B), 2004–2005. Score 0: no WS; score 1: 1–5 WS; score 2: 6–15 WS; score 3: >15 WS.

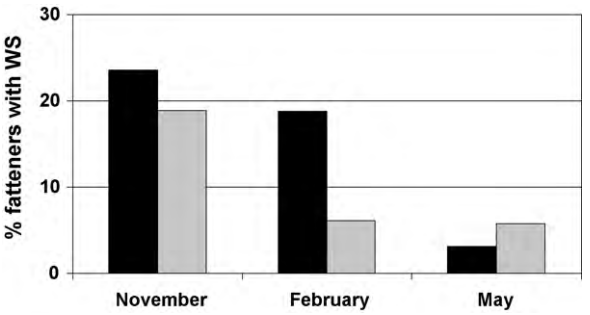

Fig. 2. Prevalence of liver white spots (WS) in 1793 Danish fattening pigs infected with *A. suum* and treated with anthelmintic (black bars) or placebo (grey bars) according to month of slaughter, 2004–2005.

In Farm B, a significant reduction in ADG ($P < 0.0001$) and a significant increase in feed conversion ($P < 0.0001$) were observed in batch 3, which was introduced to the fattening unit in February 2005, compared to batch 1 and 2 (Table 3). However, the decrease in ADG occurred in both treatment groups. Likewise, feed conversion increased at a similar rate in the flubendazole and placebo group, respectively. Also Farm B experienced diarrhea problems, especially in batch 3.

3.3. Clinical observations

Aside from transient coughing, the pigs under study did not show any clinical signs of *A. suum* infection. Mortality during the fattening period was low in both herds and not significantly different in pigs treated with anthelmintic (1.7%) compared to pigs treated with placebo (1.9%). Treatment due to other disease consisted largely of treatment against *Lawsonia* diarrhea using antibiotics (batch 3, 4 and 5 in Farm A; batch 2 and 3 in Farm B). The number of treatment days per pig against diarrhea was not significantly different for pigs treated with anthelmintic (0.06 days/pig) compared to pigs treated with placebo (0.05 days/pig).

In Farm A, post-mortem examination was performed on 29 pigs. White spots on the liver were only observed in one of these pigs. The most common clinical findings were stomach ulcers, haemorrhagic bowel syndrome and pale soft exudative musculature. In Farm B, nine pigs were examined post-mortem and white spots were found on the liver of one pig. The most common clinical findings were stomach ulcers and haemorrhagic bowel syndrome. In both farms, post-mortem findings were indicative of *L. intracellularis* infection.

4. Discussion

A common statement about *A. suum* infections in pigs is that they might cause reduced performance, i.e. decreased daily gain and increased feed conversion (Stewart and Hale, 1988; Urquhart et al., 1996). Much of the evidence for a negative impact of *A. suum* infections on pig productivity has been derived from experimental studies, typically showing significant differences in weight gain and feed conversion between pigs with and without *A. suum* (Stephenson et al., 1980; Forsum et al., 1981; Hale et al., 1985; Stewart and Hale, 1988). On the other hand, very little is known about the effect of *A. suum* infection on pig performance under commercial farming conditions, and opinions on the importance of worm infections in pigs differ remarkably among veterinary practitioners. Bernardo et al. (1990) calculated a life-time *A. suum* worm burden for pigs at slaughter and found a significant negative association with daily weight gain. In another abattoir study on finisher pigs, *A. suum* liver lesion scores were associated with a significant decrease in ADG on one farm, and an increase in ADG on another farm (Regula et al., 2000). Not only is evidence scarce and inconclusive for a possible impact of *A. suum* on pig performance under farming conditions; it is also difficult to obtain evidence if appropriate control groups are not included. Therefore, we chose to investigate the effect of *A. suum* infection on performance under commercial farming conditions using treatment of pigs with an anthelmintic or placebo.

Overall, we found no significant difference in performance of pigs treated against *A. suum* with anthelmintics and pigs treated with a placebo, indicating that *A. suum* did not have a significant impact on the performance of finisher pigs raised in the two commercial farms included in our study. This is in contrast to other reports on *A. suum* and performance of pigs in naturally contaminated farm environments (Urban et al., 1989; Bernardo et al., 1990; Regula et al., 2000). It may be argued that our study design of anthelmintic treatment twice during the fattening period only interrupted the life cycle of *A. suum* without eliminating exposure to infection. However, several studies have shown that the effect of *A. suum* on productivity is most pronounced in patent infections, i.e. when large worms are present in the small intestine (Stephenson et al., 1980; Hale et al., 1985). Therefore, our design aimed at eliminating adult worm infections, with the additional effect of reducing egg excretion and hence infection pressure in the long-term. Our results indicate that the presence of patent *A. suum* infections *per se* did not influence pig productivity. In theory, the pigs slaughtered last in each batch (41 days after the second anthelmintic treatment) might have harboured patent infections. However, no significant difference in WS prevalence or ADG was found between these pigs and pigs slaughtered 1 or 2 weeks earlier (results not shown). To improve pig performance significantly, repeated anthelmintic treatments over several consecutive fattening rounds might be necessary.

Another possible confounding factor could be that infection pressure was either insufficient or too variable between pens and batches or over time. For example, use of slatted floors in both farms might have resulted in reduced exposure to infective parasite eggs (e.g. Roepstorff and Jorsal, 1990). In our study, assessment of infection pressure or intensity was based on pen floor fecal samples and liver white spots at slaughter. This was done not just for convenience but, as opposed to counting worm burdens, because these are the tools available to pig farmers and veterinarians for deciding whether anthelmintic control strategies are justified. It may be argued that, based on fecal egg counts, infection intensity was rather low (Table 1). However, the frequency of WS was moderate to high in both herds, and despite a rather poor correlation between egg counts and white spots (Bøgh et al., 1994), WS are considered an inequivocal sign of infection (Goodall et al., 1991). It cannot be excluded that the study farms, both with a history of anthelmintic treatment against worms, through their management already had attained a reduced environmental contamination (i.e. low intensity of infection) at the start of the study. However, this underpins the dilemma these farms are facing—should they continue anthelmintic treatment and if so, will treatment be cost-effective, i.e. will pig performance improve sufficiently to bear the costs?

We observed differences in *A. suum* prevalence based on both egg excretion and liver white spots between batches within farms and between farms. In Farm A, low egg excretion levels combined with a high WS prevalence indicated high transmission among large fatteners, i.e. at the end of

the finishing period. In contrast, egg excretion was substantial in Farm B while WS prevalence was moderate, indicating that transmission primarily occurred early in the fattening period. However, even though infection pressure was different between the two farms, the results showed that transmission was substantial. In addition, possible variation of infection pressure between pens was taken into account by randomization during allocation of treatment.

A common and easy method to monitor *A. suum* infection pressure in a pig house is by fecal egg count examination. While in the majority of experimental and cross-sectional studies egg excretion is studied using individual rectal fecal samples (e.g. Roepstorff and Jorsal, 1989; Boes et al., 1997; Roepstorff et al., 1998), veterinary practitioners often rely on (pooled) pen floor samples. Since there are no studies that have compared the sensitivity of rectal sampling and pen floor sampling with regard to the detection of helminth infections, we decided to include a small scale comparison in our study. According to our results, pen floor sampling seriously underestimates the prevalence of *A. suum* in a fattening unit or farm. Only 22% of pens where *A. suum* was present were diagnosed correctly by both sampling methods. However, due to the small scale nature of our experiment the data should be interpreted with caution. It should also be noted that almost all EPG values from pen floor samples were low, i.e. below 100 EPG. Rectal fecal samples with an *A. suum* EPG below 200 are generally regarded as false-positive (Boes et al., 1997); however, whether this applies to pooled pen floor samples has yet to be determined. Thus, our results suggest that sampling strategies for helminth infections in commercial pig farms may have to be reconsidered, which will require further study.

An interesting observation was the impact of concurrent *A. suum* infection and other disease, in this case diarrhea caused by *L. intracellularis*, on pig performance. In Farm A, performance was initially poor due to diarrhea problems, and especially pigs not receiving anthelmintic treatment showed reduced productivity, suggesting a negative effect of concurring *A. suum* and *Lawsonia* infection. A negative interaction between migrating *A. suum* larvae and occurrence of pneumonia in pigs has been reported previously (Flesjå and Ulvesæter, 1980; Martinsson et al., 1991; Bouwkamp et al., 2006), but to our knowledge interactions between *A. suum* and enteric pathogens in clinical disease (diarrhea) in pigs have not been studied in detail. Interestingly, performance improved markedly in pigs treated with flubendazole as well as the control pigs following treatment with antibiotics (see Table 3), resulting in almost similar ADG and feed conversion in both groups. This suggests that enteric disease due to other intestinal pathogens such as *Lawsonia* might be more deleterious for the performance of fattening pigs than infection with *A. suum*, which deserves further study.

Treatment with flubendazole twice during the fattening period prevented *A. suum* egg excretion but did not lead to a reduction in the occurrence of liver white spots at slaughter. In fact, WS prevalence was higher in pigs treated with anthelmintic, especially in Farm B. This paradox of anthelmintic treatment leading to an increase in WS prevalence has been reported previously (Goodall et al., 1991; Huiskes et al., 2001). As discussed by Roepstorff (2003), a late onset of exposure to infective *A. suum* eggs might result in higher WS numbers when pigs are slaughtered. Anthelmintic treatment 5 or 6 weeks into the fattening period mimics late exposure and increased *A. suum* transmission, as it removes the existing worm infection and creates entry for new migrating larvae. Since *Ascaris*-induced WS heal in the course of 3–6 weeks post-infection (Eriksen et al., 1992), the interval between last anthelmintic treatment and slaughter will determine the number of WS in pigs at time of slaughter. As with performance, it cannot be excluded that repeated anthelmintic treatments over several consecutive fattening rounds might be necessary to reduce infection pressure significantly, thereby reducing WS prevalence (Kanora et al., 2004).

The prevalence of WS in slaughter pigs shows a seasonal pattern, with the highest level of liver condemnations in late summer/early autumn (Goodall et al., 1991; Christensen and Enøe, 1999). In the current study, seasonal effects on WS prevalence could only be assessed in Farm B. As can be seen in Fig. 2, between autumn 2004 and spring 2005 WS prevalence dropped both in pigs treated with anthelmintic and in pigs treated with placebo. This suggests that the reduction in WS prevalence was due to seasonal fluctuations rather than reduced transmission as a result of anthelmintic treatment. In fact, the difference in WS prevalence between pigs treated with anthelmintic and pigs treated with placebo in batch 2 (slaughtered in February 2005) suggests that anthelmintic treatment delayed the seasonal effect.

Our study design allowed for two anthelmintic treatments during the fattening period. The treatments were carried out at an interval of 30 days in order to interrupt the *A. suum* life cycle. Due to the Danish withdrawal time for Flubenol®, which was 30 days at the time of the study, it was impossible to include a third treatment closer to the time of slaughter, as recommended by the manufacturers. A third treatment e.g. 1 week prior to shipment of the first fattening pigs might have further reduced the prevalence of WS in the flubendazole group, as suggested by Kanora et al. (2004). However, treatment of market weight pigs is costly due to the large amounts of anthelmintic required, and the additional effect on productivity during the last part of the fattening period will be limited. Furthermore, the possible economical benefits of such a treatment protocol will largely depend on whether the pig producer is deducted a slaughter penalty for livers with white spots, which is the case at present e.g. in The Netherlands and Belgium, but not in Denmark.

5. Conclusions

- No significant differences in average daily gain, feed conversion or lean meat percentage between pigs treated with anthelmintic or placebo could be demonstrated in this study.
- Treatment with flubendazole on day 2–6 and day 36–40 of the fattening period prevented *A. suum* egg excretion but did not reduce the occurrence of liver white spots.

- A possible negative influence of the combination of *A. suum* infection and *L. intracellularis* diarrhea on pig performance could not be excluded.
- Pen floor fecal sampling underestimated the prevalence of *A. suum* in the fattening units compared to individual rectal sampling.
- To improve performance significantly, repeated treatments over several consecutive rounds might be necessary.

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