ORIGINAL RESEARCH

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Ascarid infection and respiratory health in feeder pigs raised on pasture or in confinement

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Summary

Objective: To investigate the effects of respiratory disease, ascarid infection, and anthelmintic treatment in pasture-raised feeder pigs.

Method: In field study 1 (FS 1), 30 indoor and 30 outdoor pigs from three different herds were selected at 3 weeks of age, euthanized, and necropsied at 7–10 weeks of age. Pigs were weighed at necropsy, and liver and respiratory tracts were examined. Average daily gain was calculated based on an assumed 3-lb average starting weight. Blood samples were collected for cell differentials. Bronchoalveolar lavage fluid (BALF) was examined for cell differentials, lactate dehydrogenase (LDH), and procoagulant activity (PCA). These procedures were repeated in field study 2 (FS2) for herd 1 only, except that 12 indoor and 12 outdoor pigs were selected and treated with anthelmintics to control ascarids.

Results: Outdoor pigs had a significantly higher ADG, ascarid liver score, PCA, and LDH compared to indoor pigs. In the outdoor pigs, eosinophilia was present in BALF and blood. No changes due to ascarid infection were found in BALF and blood of indoor pigs. Despite the anthelmintic treatment, results in FS2 were similar to those in FS1.

Implications: Average daily gain was improved in outdoor pigs compared to indoor pigs despite the infections in outdoor pigs with *Ascaris suum*.

Keywords: swine, pasture, *Ascaris suum*, anthelmintics

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Traditionally, pigs in the midwestern United States were raised on pasture, with shelters or barns with open fronts to allow them access to outdoor pens.[1,2] Within the past 20 years, such facilities have been largely replaced with strict confinement systems. However, outdoor production systems have become the focus of renewed interest in the swine industry because they require less initial investment capital, promise better air quality, and may promote animal welfare.[3–5]

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Each type of swine facility has its relative advantages and disadvantages. Confinement buildings are densely populated and several environmental stress factors, including airborne contaminants that might be hazardous to animals and their caretakers, are characteristic of these systems.[1,2] Outdoor production systems, on the other hand, have a high prevalence of *Ascaris suum* with attendant economic losses.[6,7] Fewer parasites are found in intensive production confinement systems, because prevalence of infection is closely related to hygiene and housing systems.[6,8,9] However, Roepstorff reported that it was almost impossible to find a Danish swine herd that was totally free of *A. suum*.[10] *Ascarid* eggs are resistant to most environmental factors, such as temperature, chemicals, and desiccation.[10,11]

Outdoor production has been found advantageous in herds with high risks for respiratory problems.[4] Respiratory diseases are the most important disease problem in pigs raised in confinement buildings. In the 1980s, United States slaughterhouse studies of pigs raised in confinement indicated gross lesions of pneumonia and atrophic rhinitis in 100% of swine herds, and pneumonia in 40% and atrophic rhinitis in 70% of market hogs.[12,13] More recent data indicate a similar prevalence of pneumonia, but a decrease in the prevalence of atrophic rhinitis lesions.[14] Pneumonia and rhinitis are estimated to cause annual losses of several hundred million dollars due to mortality and reduced weight gain.[13,15]

Respiratory diseases in pigs are usually of multifactorial etiology.[14] Traditionally, infectious agents have been considered to be the primary etiological agents, but noninfectious airborne contaminants may also be critically important contributing factors. For example, pigs in confinement buildings are continuously exposed to airborne contaminants, including dust, endotoxin, microorganisms, and gases.[16–18]

The following study was originally designed to compare the effects of confinement versus pasture rearing on the morphology and immunologic status of the respiratory system of feeder pigs. We hypothesized that pigs raised indoors were exposed to more environmental challenges than pigs raised outdoors, and that this might introduce differences in respiratory pathology. A second field study was conducted in which both the indoor- and outdoor-raised pigs were intensively and prophylactically treated with anthelmintics. The objective of this paper is to discuss the effects of ascarid infection and treatment failure in pasture-raised feeder pigs.

Material and methods

Herds and animals

Three farrow-to-finish swine herds located in central Wisconsin were selected based on the presence of year-round confinement (indoor) production with additional pasture (outdoor) production of feeder pigs from May to October. The management of indoor and outdoor husbandry was similar in all three herds. Serological screening (Oxford Laboratories; Worthington, Minnesota) for respiratory pathogens indicated antibodies to *Mycoplasma hyopneumoniae*, swine influenza virus, *Haemophilus parasuis*, and *Streptococcus suis* in feeder pigs from all herds.

Field study 1

Field study 1 (FS1) was conducted in all three herds between June and September 1995. Six pigs, respectively, from the indoor and outdoor environments were randomly selected from a minimum of five gilt litters at 7–10 weeks of age in herd 1 (n=12), and 12 pigs from each environment in herds 2 (n=24) and 3 (n=24). In FS 1, a total of 30 pigs from both environments allowed us to detect a statistical difference (95% confidence and 80% power) if the overall prevalence in the outdoor pigs was 5% and in the indoor pigs was 37.5%.

- Outdoor groups of 10–15 gilts farrowed in huts on pasture. Pigs were weaned at 3 weeks of age and kept outdoors until they reached feeder pig size (20 kg, 10 lb) and age (8–9 weeks). Outdoor pigs had almost unlimited pasture space and 0.23 m^2 (2.5 sq ft) per pig in the huts. They were then moved to confinement finishing facilities where they were raised to slaughter weight.
- Indoor groups of 15–20 indoor sows and gilts were confined to standard 2.1 × 1.5 m (6.7 × 4.9 ft) farrowing crates on 30-cm (12-inch) raised decks with slatted floors. The indoor nursery pigs were penned in groups of 15–20 on slatted floors, with about 0.23 m^2 (2.5 sq ft) per pig.

All pigs were fed similar starter (22% crude protein) and nursery (20% crude protein) complex feeds. Pigs received no vaccinations or other treatments. At the end of the study, pigs were weighed, anesthetized with a combination of xylazine (4.4 mg per kg, Rompun®, Miles Inc, Kansas), and tiletamine and zolazepam (4.4 mg per kg, Telazol®, Fort Dodge, Iowa), exsanguinated, and necropsied. The pre-exsanguination anesthesia was necessary to minimize severe congestion and hemorrhages in the lungs, which are induced by electrical or captive bolt pistol stunning. Samples were collected as described below. Average daily gain (ADG) from birth to necropsy was calculated for each pig using an assumed 1.5-kg (3-lb) average starting weight. Pigs were weighed once at necropsy.

Field study 2

Field study 2 (FS2) was conducted only in herd 1 between June–August 1996. Field study 2 was conducted in exactly the same way as FS1, except that in FS2 an intensive anthelmintic treatment plan was initiated to control the heavy ascarid migration found in FS1. Indoor and outdoor gilts were treated with pyrantel tartrate at the recommended dose of 96 g per ton of feed (Banminth®, Pfizer Animal Health). Indoor and outdoor pigs were treated intramuscularly at 10 days of age with ivermectin (0.3 mg per kg, Ivomec®, Merck AgVet Division, New Jersey). From the age of 2 weeks, they were fed a starter feed with added pyrantel tartrate at 96 g per ton of feed (Banminth®). Twelve pigs were randomly selected at 8 weeks of age from both the indoor and outdoor environments (n=24). Necropsy and sampling procedures were as described for FS1.

Blood sample collection

In field study 1, blood samples were collected by *vena cava* puncture prior to necropsy from six 8-week-old pigs from both indoor and outdoor environments from each herd. A 1-mL sample of EDTA blood was collected for total white blood cell count (Coulter Electronics, Florida) and a smear was prepared for cytology. Differential cell counts were determined on 200 cells on stained smears (Diff-Quick, American Scientific Products, Missouri).

Macroscopic evaluation of liver and respiratory tract

At necropsy, livers were subjectively scored for presence and severity of ascarid larval fibroma ("milk spots") so that:

- 0 = no milkspots;
- 1 = < 10 milkspots;
- 2 = > 10 milkspots, but areas of viable liver; and
- 3 = most of the liver surface covered with milkspots.

Pleuritis was also scored on a range from 0–3 gross on an estimated scale from 0%–100% of the lung volume involved, as previously described.[19]

Bronchoalveolar lavage and fixation of lung tissue

A 12-Fr Foley catheter was inserted and sealed into the bronchus of the right diaphragmatic lobe and bronchoalveolar lavage (BAL) was performed. Four times twenty mL of phosphate buffered saline (PBS, pH 7.4) was slowly infused and aspirated immediately. The volumes of aspirated BAL fluid (BALF) were pooled. The percent recovery ranged from 70%–75%. For histopathological examination, the left lung lobes were fixed by bronchial perfusion with formalin at a constant pressure of 30 cm H_2O from the fluid pressure head to the level of the carina.

A 1-mL aliquot of the pooled BALF was collected for total cell count. Total white blood cell counts were determined with an automated cell counter (Coulter Electronics). Cell differentials were determined on 200 cells on stained cytospin smears (Diff-Quick).

BALF was centrifuged at low speed at 4°C (5 minutes, 1000 × g) and supernatant was collected. Procoagulant activity (PCA) was measured by the ability of BALF supernatant to shorten the recalcification time of pooled normal swine plasma in a two-stage clotting assay. Equal volumes of BALF supernatant and pooled normal plasma were incubated for 2 minutes at 37°C. Subsequently, an equal volume of $CaCl_2$ was added and the clotting time was measured (ACL300+ Coagulation

Analyzer, Coulter Electronics). Lactate dehydrogenase (LDH) activity on BALF supernatant was expressed as the rate of oxidation of reduced nicotinamide dinucleotide at 340 nm (Marshfield Laboratories, Wisconsin).

Statistical analysis

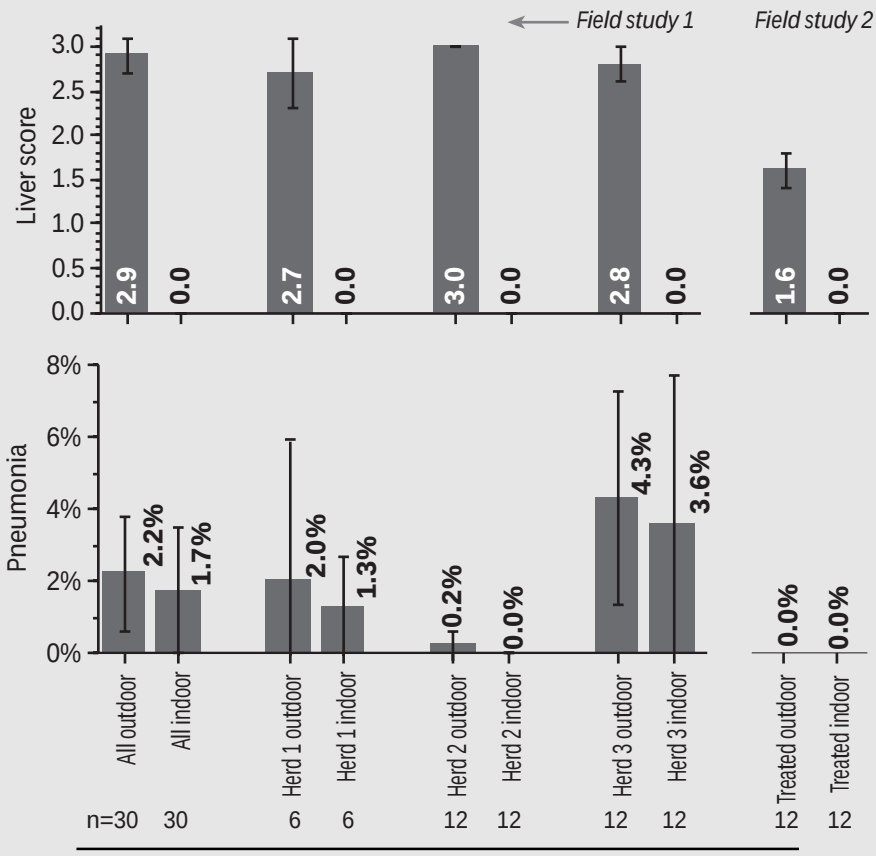
Descriptive statistical analysis of the data was calculated with Systat computer package (Systat Inc., Illinois). Indoor and outdoor herds in FS1 were compared for all variables with multivariate ANOVA using herd as a block (SAS Institute Inc., North Carolina). The overall difference between indoor and outdoor pigs for all variables measured was tested with the Wilks λ procedure. A univariate analysis (F-test) was used to test differences between indoor and outdoor pigs in FS1 and FS2.

Results

Field study 1

The overall analysis of the measured health parameters indicated significant differences between indoor and outdoor pigs ($P < .001$).

Figure 1



Macroscopic lesions at necropsy in field studies 1 and 2

Health

In FS1, outdoor pigs had significantly higher liver scores ($P < .001$) than indoor pigs (Figure 1). The gross pneumonic lesions in both indoor and outdoor pigs were minimal, and indicative of mycoplasma and/or bacterial pneumonia (Figure 1). Small petechial hemorrhages caused by migrating larvae were observed in the lungs of outdoor pigs. On histopathology (Figure 2), the lungs of the outdoor pigs were severely infiltrated with eosinophils. Worm tracks filled with debris and occasional larvae were observed. LDH ($P < .01$) and PCA ($P < .01$) also significantly differed between the indoor and outdoor pigs. Eosinophilia was observed in the cell differentials of blood and BALF of outdoor pigs compared to indoor pigs ($P < .001$). No adult nematodes were observed during necropsy procedures.

Growth

Outdoor pigs had a significantly higher ADG ($P < .001$) than the indoor pigs. Results followed the same trend at the herd level, even though each herd was different and the number of pigs evaluated per herd was small (Figure 3).

Field study 2

Health

Liver lesions in the outdoor pigs were still severe considering the treatment plan, but were slightly less severe compared to those in FS1. Again, no adult nematodes were observed (Figure 1). Procoagulant activity ($P < .05$) and LDH ($P = .08$) activity were significantly higher in the outdoor pigs than in the indoor pigs (Figure 2). Small petechial hemorrhages were found only in the lungs of the outdoor pigs. No other macroscopic lesions of pneumonia were found in either group of pigs (Figure 1). Histopathological lesions in outdoor pigs were less than in those observed in FS1 and consisted of eosinophilic infiltration and occasional ascarid larva. The cell differentials in the BALF indicated a higher number of eosinophils ($P < .05$) in the outdoor pigs and a higher number of lymphocytes ($P < .05$) in the indoor pigs (Figure 4).

Growth

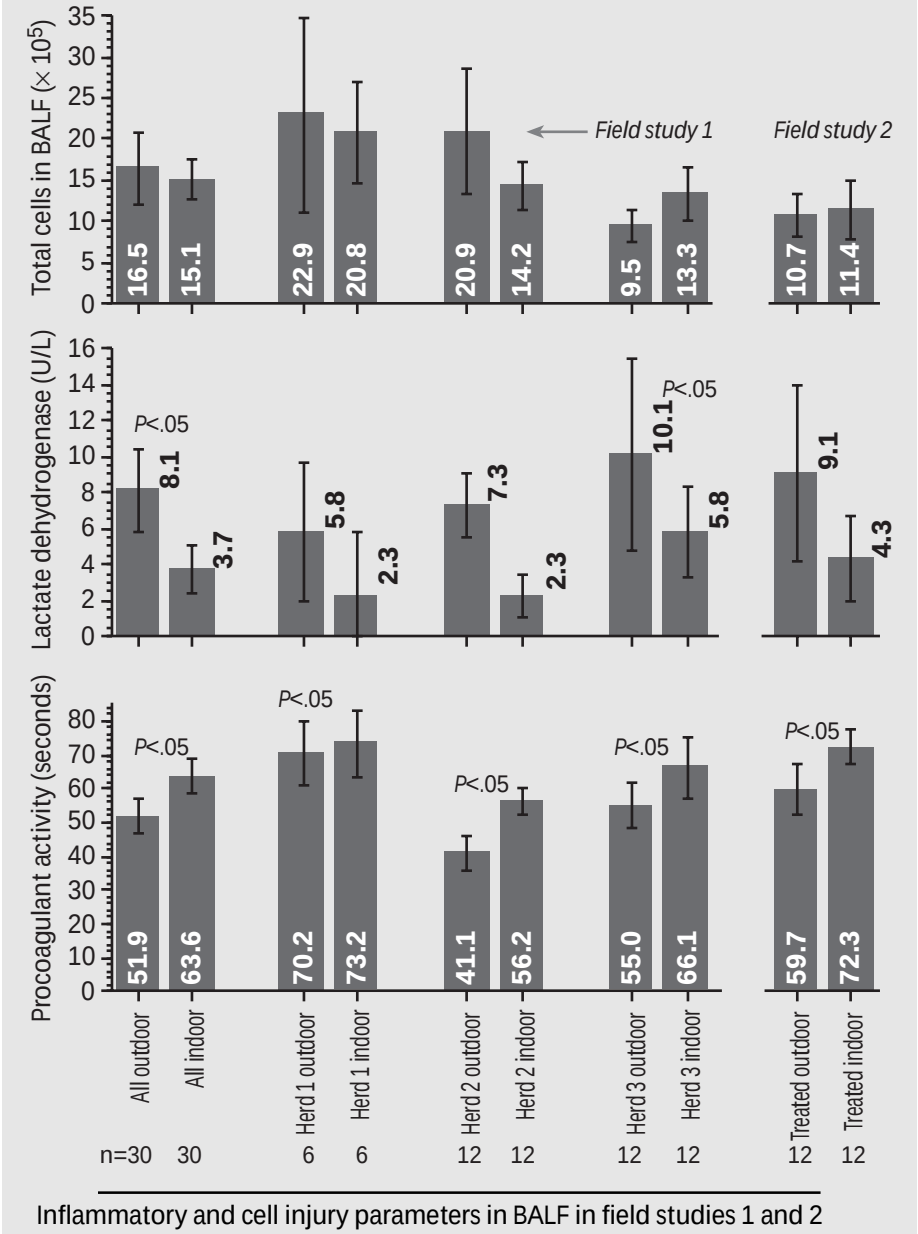
ADG was greater in outdoor pigs, although this difference was not significant ($P = .65$) (Figure 3).

Discussion

This study was originally designed to test the hypothesis that the growth of pigs raised outdoors would be improved over indoor-raised counterparts, given that indoor pigs are more susceptible to respiratory disease because they are continuously exposed to higher concentrations of airborne contaminants. The second field study was conducted after large numbers of ascarid worms were observed in the outdoor pigs in FS1 in an attempt to control for this variable.

Only nursery-aged pigs were included in this study because outdoor pigs in all herds were moved to confinement around 8 weeks of age,

Figure 2



Inflammatory and cell injury parameters in BALF in field studies 1 and 2

i.e., feeder pig age. Therefore, the effect of long-term exposure to airborne contaminants on respiratory health could not be studied and the results apply only to feeder pigs.

At necropsy, livers of outdoor pigs in FS1 had large numbers of small fibromas ("milk spots"), indicating the migration of immature ascarids.[20] Liver lesion scores in FS2 outdoor pigs were lower than those in FS1 and were not resolved by the treatment plan. Liver white spots are indicative of ascarid larval migration and are associated with inflammation. Previously, it has been demonstrated that the number of liver white spots increases until 6–9 weeks of continuous exposure to *A. suum*, followed by a gradual decline even under continuous exposure.[11,21] Based on this finding, the large number of liver white spots observed in the outdoor pigs are the result of an intense transmission of *A. suum* and a severe liver inflammation, which was too intense to resolve prior to the endpoint of the study at 7–10 weeks of age.

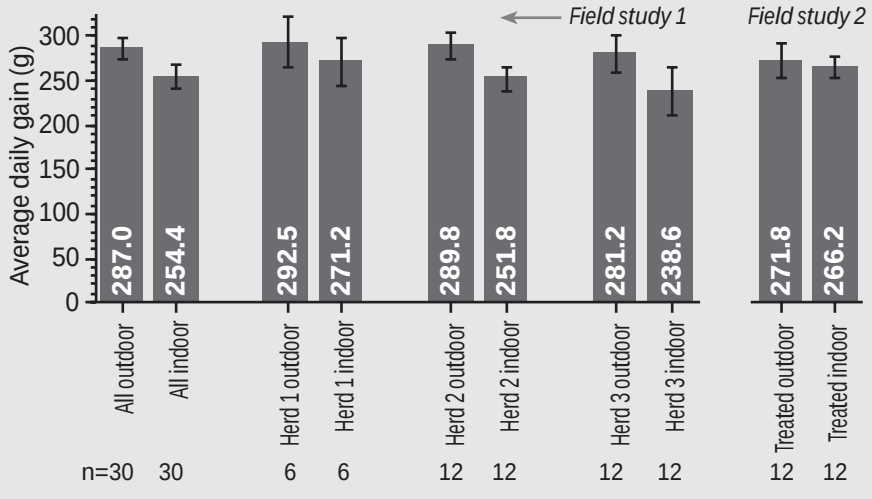
The larval migration also caused small petechial hemorrhages in the lungs of the outdoor pigs. Microscopic evaluation of the lung tissue demonstrated severe eosinophil infiltration and several ascarid larvae. Ascarid larvae have been observed to reach the lungs as third-stage larvae within 4 days after experimental infection.[22] The larval migration might also explain why we found increased numbers of eosinophils in peripheral blood and BALF of outdoor pigs. Fornhem, et al., have reported such eosinophil increases in BAL and lung tissue of pigs experimentally exposed to *A. suum* allergen to study allergen-induced late airway obstruction.[23,24]

Outdoor pigs had increased levels of LDH and PCA in BALF, which are probably also caused by migrating larvae. LDH is a cytosolic enzyme present in nearly all cells and is considered a nonspecific biochemical marker of lung injury.[25] Presence of LDH in BALF is an indicator of a toxic effect on the respiratory cells.[26] Increased PCA in the lung might contribute to inflammatory changes leading to fibrin deposition.[27] PCA is also involved in nonspecific immune reactions and delayed-type hypersensitivity.[27,28]

It would be expected that the severe microscopic lesions we observed in the lungs, due to larval migration, could induce coughing. However, we did not evaluate coughing frequency and/or severity during the course of this study.

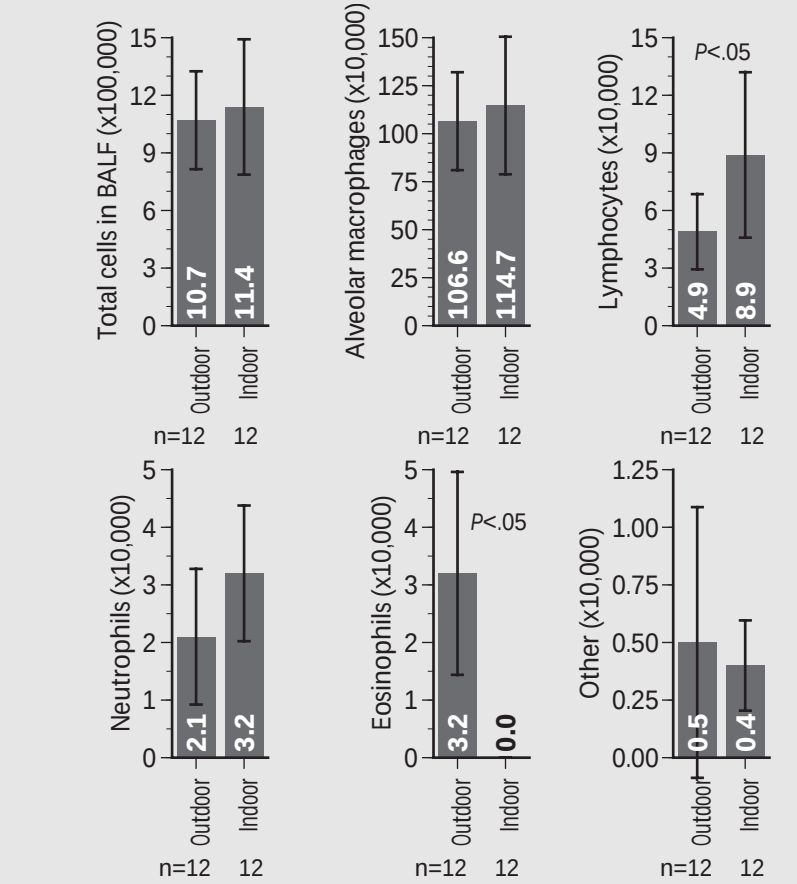
Although ascarid infections are also frequently reported in pigs raised in confinement buildings, none of the lesions described in the outdoor pigs were present in the indoor pigs in this study. Since no fecal samples were collected, it cannot be concluded that the indoor feeder pigs were completely ascarid free. On the other hand, it has been suggested that liver milkspots are a more reliable indicator of larval migration that has occurred within the previous 2–3 weeks than fecal sampling.[11] These results are also consistent with Roepstorff, et al., who reported that transmission of *A. suum* in confinement herds with slatted floors does not occur until the late finishing phase and that the highest intensity of infection is found in sows.[10] Transmission in such herds is low because feces are deposited through the slatted floors, away from the pigs, leading to improved hygiene and physical conditions unsuitable for embryonation of the eggs.[10] In contrast, outdoor-raised pigs have been observed to be already heavily infested with

Figure 3



Comparison of growth in indoor and outdoor pigs in field studies 1 and 2

Figure 4



Cell differentials for BALF in field study 2

A. suum at 10–12 weeks of age, suggesting that transmission occurs soon after birth.[11] This raises the concern that outdoor feeder pigs, which are moved inside for finishing around 8 weeks of age, are a possible infection source for other indoor pigs if they are not intensively treated.

Despite the severe ascarid larval migration in the outdoor pigs of all herds, ADG for outdoor pigs was equal to or better than in the indoor pigs. Because starting weights were not measured and a 3-lb standard was used instead, because feed efficiency was not measured, and because the sample size was only marginally adequate, the interpretation of this data is difficult. It could be that the impact of the larval migration on growth was small and/or that outdoor pigs were less stressed due to a lower population density on pasture and/or less polluted air environment. In addition, outdoor pigs stayed in the same environment until 7–10 weeks of age, while the indoor pigs were moved and commingled in a nursery at weaning. Guy, et al., also measured significantly higher ADG in finishing pigs raised in outdoor paddocks compared to pigs raised in confinement.[5] It is also possible that the entire population of outdoor pigs was heavier because smaller, weaker pigs, which might have survived indoors, were more likely to die in the outdoor environment. It is reasonable to assume that the outdoor pigs in our study would have performed even better, had we been able to control or prevent the parasites. Since our study was terminated when pigs reached 7–10 weeks of age, any long term effects of ascarids could not be measured.

In this study, intensive treatment with approved anthelmintics for *A. suum* at recommended doses had little effect on larval migration in the FS2 outdoor pigs. This could be explained by a combination of three important factors. First and most importantly, our results indicate that anthelmintic treatment of pigs with a heavy parasite infestation due to continuous exposure to worm-infested soil is ineffective. Other investigators have also reported that anthelmintics alone might not control parasite infection on contaminated pastures, where pigs are continuously reinfected.[6,29,30] Because of their transitory effect, anthelmintics need to be used in combination with other control measures, such as pasture rotation and mixed or alternate grazing with other animals.[31]

Secondly, ivermectin injection in 10-day-old pigs does not inhibit larval migration to or within the liver. Ivermectin is indicated for fourth-stage larvae and adult worms and is active for at least 6–12 days.[32] The prepatent period of *A. suum* is 8 weeks and fourth-stage larvae are

present from day 10 on. This implies that the ivermectin injection should have been effective, but the continuous re-infection of pigs might explain the lack of drug efficacy. From the age of 2 weeks on, the pigs continuously received pyrantel tartrate, which is useful against all stages of the *A. suum* life cycle at the recommended dose of 96 g per ton of feed. It is possible that the pigs were not consuming adequate amounts of feed prior to weaning to prevent larval lesions in lung and liver, although this seems rather unlikely given their ADG performance.

Lastly, development of drug resistance by *A. suum* should also be considered.[10] It is not known whether *A. suum* can develop resistance, but this parasite has a high reproductive potential and treatment survivors might produce a more resistant progeny. Widespread use and misuse of some anthelmintics aids in the development of resistance.[10] On the other hand, the highly resistant ascarid eggs may establish a proportion of parasites that escape treatment, which might slow down resistance development.[10]

Overall, our study indicates that current anthelmintic protocols were not effective in pigs with a heavy parasite infestation due to continuous exposure to *A. suum*. In contrast, the indoor pigs had no signs of larval migration, whether they were treated or not (FS1 versus FS2). This suggests that environmental factors, such as slatted floors and sanitary procedures, have a profound impact on the transmission of *A. suum* and raises the question of whether anthelmintics are useful in intensive confinement production systems. Further research is needed to clarify this question due to the renewed interest in outdoor pasture systems.

Implications

- Despite ascarid infection, 8-week-old outdoor pigs had better ADG compared to indoor-raised pigs, probably because of the lower population density on pasture.
- Intensive anthelmintic treatment has a low efficacy in pigs raised on a worm-infested soil.
- Pasture management is required in addition to strategic anthelmintic treatment for parasite control of outdoor raised pigs.
- Outdoor feeder pigs need to be wormed prior to commingling with other indoor pigs.

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