Lungworms (*Metastrongylus Spp.*.) Demonstrated in Domestic Pigs with Respiratory Disease - What was the True Relevance of that?

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Case Report

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Abstract

Background

An outdoor pig herd was affected by severe respiratory disease in one out of three pastures. At necropsy, *Mycoplasma hyopneumoniae* and *Pasteurella multocida* were detected in the lungs, as well as the lung worm *Metastrongylus apri*. The life cycle of *Metastrongylus* spp. includes earth worms as an intermediate host, and since domesticated pigs mainly are reared indoors lungworms has not been diagnosed in domestic pigs in Sweden for decades, not even in pigs reared outdoors. Therefore, this disease outbreak was scrutinised from the view of validating the impact of *Metastrongylus* spp..

Results

At the time of the disease outbreak, neither eggs of *Metastrongylus* spp. nor *Ascaris suum* were detected in faeces of pigs aged ten weeks. In contrast, five-months-old pigs at the pasture with respiratory disease shed large amounts of eggs from *Ascaris suum*, whereas *Ascaris suum* not was demonstrated in healthy pigs aged six months at another pasture. Low numbers of eggs from *Metastrongylus* spp. were seen in faecal samples from both these age categories.

At slaughter, seven weeks later, ten normal weighted pigs in the preceding healthy batch were compared with ten normal weighted and five small pigs from the affected batch. Healing *Mycoplasma*-like pneumonic lesions were seen in all groups. Small pigs had more white spot liver lesions, and all small pigs shed eggs of *Ascaris suum* in faeces, compared to around 50% of the pigs in the normally sized groups. *Metastrongylus* spp. were demonstrated in 13 of the 25 pigs (52%), representing all groups included.

Conclusion

As *Metastrongylus* spp. were demonstrated regardless of health status, and in another healthy outdoor herd, the impact of *Metastrongylus* spp. on the outbreak of respiratory disease was depreciated. Instead, *Metastrongylus* spp. was suggested to be common in outdoor production, although rarely diagnosed. The reason for this is because they will escape detection at routine inspection at slaughterhouses, and that they appeared to generally not induce clinical signs of respiratory disease. Instead, a possible association with a high burden of *Ascaris suum* was suggested to have preceded the severe outbreak with respiratory disease.

Background

Porcine lungworms (*Metastrongylus* spp.) are nematodes with earthworms as intermediate host (1). Pigs may become infected when they ingest earth worms with third stage lungworm larvae. Further development of the lungworm occurs in the pig and adult lungworms can be found in the bronchus and bronchioles of the lungs. Adult lungworms are thin, but can reach a length of approximately 50 mm. They
may cause respiratory illness (1), especially in young (small) individuals where bronchioles may be obstructed by adult lungworms (2). Such problems may be enhanced by other concurrent infections (3) and/or by nutritional deficiencies (4). Lungworms are however often neglected as a cause of respiratory diseases in domestic pigs. The reason for this is that pigs are mainly reared indoors where there is no access to the intermediate host (5), and eggs of *Metastrongylus* spp. were not demonstrated in any faecal sample in a recently published Swedish parasite-point-prevalence study (6). Thus, lungworms are rarely diagnosed in domestic pigs reared indoors, and therefore also rarely discussed in pigs reared outdoors.

During the summer of 2021 a pasture housing a batch with 131 growing pigs outdoors from an approximate mean live weigh of 30 kg to around 130 kg was affected by severe respiratory disease. In total, 15 of the pigs died, whereof seven had been unsuccessfully treated with penicillin. However, another 23 affected pigs that had been medicated with penicillin early during the disease survived. Two pigs that had died were necropsied and diagnosed with pneumonic lesions resembling those caused by *Mycoplasma hyopneumoniae*. Nevertheless, pigs that were medically treated early during the diseases responded well to treatment with penicillin. Since *Mycoplasma* spp. lack a cell wall and therefore are naturally resistant to penicillin (7) it was assumed that an initial infection with *M. hyopneumoniae* had been secondarily infected with *Pasteurella multocida*. That assumption explained both the severity of the disease, and the positive effect of the treatment with penicillin early during the infection. That assumption was also supported by the fact that *P. multocida* was demonstrated in the lungs of both pigs, as well as *M. hyopneumoniae* and *M. hyorhinis*.

However, the necropsy also identified the porcine lungworm *Metastrongylus apri* in the lungs of the affected pigs. Since lungworms had not been demonstrated at all in domesticated pigs in Sweden since decades, their impact on the disease outbreak caused by *M. hyopneumoniae* and subsequently infected with *P. multocida* at the pasture was discussed.

The affected farm had two other pastures, but none of them had been affected by respiratory disease. The pasture that had been affected by respiratory disease was geographically the most remote pasture (Figure 1), and wild boars were common in the neighbourhood. Lung worms are globally common in wild boars (8, 9), including Sweden (10), and the prevalence of lungworms did not differ between geographically isolated wild boar populations north and south of the Alpes, although species within the *Metastrongylus* family differed between the two regions (11).

Due to the rare finding of lungworms, an intensified investigation with the aim to scrutinise the true impact of lungworms on the clinical course of the respiratory disease at the affected pasture was initiated.

**Results**

**Clinical signs**
Among the 131 fatteners that were allocated to pasture B, one pig was found dead four weeks after arrival, and occasionally dead pigs were found during the following week. Thereafter, the incidence of pigs affected by respiratory diseases increased dramatically. In total, 30 of the 131 pigs (23%) were parenterally medicated with penicillin during the subsequent two weeks. The treatment efficacy was considered good if treatment was initiated during the early course of disease, but seven of the 30 treated pigs (23%) died. In total, another eight pigs either died or were euthanised without initiating treatment, whereof two were necropsied (see below). To conclude, 15 out of 131 pigs (11%) died during a period of three weeks. Pigs affected by the disease searched shelter in the huts. As the weather was warm and dry, the huts were removed with the aim to prevent spread of disease, and thereafter the transmission of respiratory diseases decreased.

No similar signs of disease were recorded in other parts of the farm. Neither on the other two pastures (Figure 1; A and C) nor on the two concrete slabs (Figure 1; D and E).

**Necropsies**

Pneumonic lesions resembling those caused by *M. hyopneumoniae* were seen in the two pigs from pasture B that had been sent for necropsy. *M. hyopneumoniae*, *M. hyorhinis* and *P. multocida* were demonstrated in the lungs. Lungworms were found in the airways of both pigs and these were identified as *Metastrongylus apri*.

**Parasitological investigations from faecal samples collected in the herd**

No parasite eggs were detected in the faecal samples from growers aged ten weeks (n=8) and not yet transferred to the pastures, *i.e.*, when still at the indoor facilities (Table 1). Nor were any parasite eggs detected in non-dewormed pregnant sows that also were housed indoors (n=8).

During the outbreak of severe respiratory disease on pasture B, faecal samples from outdoor fatteners were collected from all pastures and concrete slabs that housed pigs (Table 2). In pigs aged five months at pasture B with respiratory disease, the faecal egg count (FEC) was high for *A. suum* (mean >1,500 egg per gram; Epg) and occasionally also for *Trichuris suis* (Max = 1,100 Epg). In contrast, no eggs of these parasites were detected in faecal samples collected from pigs aged six months grazing on pasture A, nor from pigs aged four months and grazing on pasture C. Still, high FEC of *A. suum* were detected in pigs aged seven months on the concrete slab D that had been populated with pigs from pasture A. *Metastrongylus* spp. were demonstrated in faeces from all pigs aged 5 months or more (Table 2), but the faecal egg count was low (mean <40 egg per gram).

**Results obtained at slaughter**
The live weight corresponded to 129.2 ± 5.5 kg for the ten pigs that had reached market weight from pasture B, and 126.1 ± 9.7 kg for the ten market weight pigs from pasture A. The live weight for the five small pigs from pasture B was 70.5 ± 6.1 kg, which differed significantly (p<0.001) from the pigs that had reached market weight (Figure 2). The live weight of the seven pigs from herd V that was situated 150 km from the affected herd was 126.7 ± 18.8 kg (Figure 2).

Most pigs had pneumonic lesions resembling mycoplasmosis, but these lesions were without exception in healing phases (12), i.e., none of them were actively in progress at the time of slaughter. Thereby they were not recorded by the official meat inspection at slaughter, which only register active processes (12). Pleuritis was registered in one of the small pigs from pasture B, but not in any of the other pigs. The number of white spot liver lesions were somewhat higher among the small pigs from pasture B, but white spots were present in all categories of pigs. Also, lungworms were found in all categories of pigs (Figure 2).

**Haematology at slaughter**

The small pigs from pasture B had numerically but not significantly (p>0.05) higher leukocytes concentrations than the heavier pigs from pasture B and the healthy pigs from pasture A. (Figure 3). They also had a significantly (p<0.05) lower percentage of lymphocytes and somewhat (p=0.07) higher percentage of granulocytes than the larger pigs from the same pasture, which altogether indicated a higher activation due to infections compared with the haematology of the larger pigs. All three categories had normal levels of hemoglobin (Hb), but the Hb was significantly (p<0.05) higher in the large pigs from pasture B than in the small pigs (Figure 3).

**Serological reactions at slaughter**

All three categories of pigs were clearly seropositive to *M. hyopneumoniae*, and the absorbance levels were numerically but not significantly (p>0.05) higher in the small pigs from pasture B than in large pigs (Figure 4). There were seroreactors to *P. multocida* in all categories of pigs, but the mean absorbance levels were moderate and did not differ (p>0.05) between pig categories. All pigs were seronegative to *Actinobacillus pleuropneumoniae* serotypes 2 and 3.

**Parasitological findings in faeces collected at slaughter**

The 25 pigs were also examined for the presence of nematode eggs in faecal samples at an individual level. With the exception of *A. suum* where eggs were detected in all small pigs from pasture B (n = 5), the parasitological findings were similar in all three categories of pigs (Figure 5).

Low levels of eggs from *T. suis* and *Metastrongylus* spp. were found in all categories of pigs. In contrast, the level of *A. suum* eggs in individual pigs could be high (Figure 5).
Discussion

Lungworms are rarely detected and rarely discussed in the pig production of today (6). A recent finding of lungworm in an outdoor pig herd that also suffered a severe outbreak of respiratory disease therefore initiated this extended disease investigation. What was the true impact of the lungworms?

Growers that were transferred to a pasture when they were aged 10-11 weeks neither shed eggs of *A. suum* nor *Metastrongylus* spp. at that time. However, during the acute outbreak of respiratory disease in five-month-old pigs at pasture B, faecal analysis showed a low faecal egg count of *Metastrongylus* spp. and a high faecal egg count of *A. suum*. Both these parasites can contribute to respiratory disorders, *Metastrongylus* spp. by being present in the airways and *A. suum* because the parasite life cycle includes migration of larvae through the lungs (1).

It should however be noted that low faecal egg counts of *Metastrongylus* spp. also was demonstrated in faecal samples from older outdoor pigs that did not show any clinical signs of respiratory disease, *i.e.* from six-month-old pigs at pasture A, as well as from seven-month-old pigs at the concrete slab D. In contrast, no eggs of *A. suum* were demonstrated in the pigs at pasture A, but a high FEC was demonstrated in pigs from that pasture that had been transferred to the concrete slab D prior to slaughter. This implied that pigs had been negative to *A. suum* at pasture A but became infected with a residual infection when transferred to the concrete slab prior to slaughter.

The results from the extended survey of lungs at slaughter to a large extent showed the same picture. Adult lungworms were demonstrated in the airways of all groups of pigs that had been reared outdoors, *i.e.* not only from pigs reared at pasture B were severe respiratory diseases had been diagnosed. Adult lung worms were also demonstrated in the airways of the pigs from pasture A that apparently had been healthy during the rearing period – as well as in lungs of apparently healthy pigs from herd V that reared pigs outdoors 150 km away from the affected herd. The incidence of mature lungworms in the airways of pigs from pasture B did not differ between small and large pigs.

The severity of the disease outbreak at pasture B was underlined by the high mortality and the weight difference of 50 kg between small and market weight pigs, which suggested a prolonged rearing time with around two months for the small pigs to reach desired market weight. In comparison with the market weight pigs from both pastures B and A, the small pigs from pasture B had a higher incidence of pigs with more than 20 white spots in the liver that presumably were caused by *A. suum* (1). The small pigs also had numerically higher amounts of antibodies to *M. hyopneumoniae* than the heavier pigs. In addition, the small pigs from pasture B also had higher levels of leukocytes with lower percentages of lymphocytes but higher percentages of granulocytes than the heavier pigs, signs that indicated an ongoing response to infections. By merging these observations, the poor growth of the small pigs could be suggested to have been caused by infections that to some extent lasted throughout the whole rearing period (13).
Altogether, the recordings at slaughter and the necropsies strengthened the conclusion that the respiratory outbreak to a large extent was caused by *M. hyopneumoniae*. However, as affected pigs responded well to penicillin that does not combat *Mycoplasma* spp. (7) *P. multocida* was probably an important secondary pathogen in pigs that developed severe clinical signs during the disease outbreak. However, as the levels of antibodies to *P. multocida* were low in these pigs that had survived the acute disease outbreak, the long-term effect of *P. multocida* on surviving pigs appeared to have been marginal. This could probably be explained by the combination of initiating treatments early during the course of the infection and the removal of the huts where affected pigs searched shelter. As the huts had limited air spaces they were regarded to condense the pathogen load and, as the weather conditions allowed it, they were removed.

Considering the high FEC of *A. suum* in the five-month-old pigs at pasture B during the acute disease outbreak, it can not be ruled out that *A. suum* contributed to the disease. The larvae of *A. suum* migrate through liver and lungs before maturation into adults (1) and thereby induce lesions in the lungs that may facilitates infections with *e.g.* *M. hyopneumoniae* and/or *P. multocida*. Indeed, it was notable that there were no signs at all of infections with *A. suum* in the older pigs at pasture A that had remained healthy during the time of the disease outbreak in pasture B. So why did these pigs not turn unhealthy when moved to the concrete slabs where they apparently became infected with *A. suum*? The explanation for this certainly is to find in the fact that the pigs at the time for transfer to the concrete slab since long had been exposed to *M. hyopneumoniae*. Thereby they had developed both antibodies and immunity towards (re)infections with *M. hyopneumoniae* at the time point when the larvae of *A. suum* migrated through their lungs (14, 15).

Due to these findings the impact of *Metastrongylus* spp. at the severe outbreak of respiratory disease in July 2021 was downgraded. The amount of *Metastrongylus* spp. eggs in the faecal samples collected at slaughter from pigs that had grazed at pasture B was low and comparable with the levels of eggs fond in the healthy pigs from pasture A. In addition, adult lungworms were found in the lungs of 20-30% of the pigs from both pastures, and lungworms were also detected in another farm rearing pigs outdoors that was located 150 km away and not had reported any problems with respiratory diseases. The common observation of adult lungworms was somewhat surprising, but it must be emphasised that the routine inspections made at slaughter does not include inspection of the airways by opening them (16), and therefore the presence of adult lungworms in the airways of pigs will escape detection at routine meat inspections.

By opening the airways of the lungs in the extended inspection we discovered adult lungworms, but it is notable that the incidence of lungworms probably was underdiagnosed despite the extended measures undertaken. We did not find adult worms in the lungs in all pigs that shed eggs of *Metastrongylus* spp. in the faeces. Eggs were demonstrated in totally nine of the 25 pigs from the affected herd, but adult worms were only demonstrated in the lungs in three of these pigs. These three pigs shed 250, 300 and 1950 eggs per gram faeces, respectively.
Looking at this phenomenon the other way around; we found adult lungworms in totally seven pigs, but we were only able to detect *Metastrongylus* eggs in faeces from three of them. This could theoretically have been because the lung worms either not yet had started to produce eggs or due to an unbalanced gender distribution of the lungworms where up to at least 75% of the worms may belong to one sex (11). However, the low amounts of eggs shed by the faeces also made underdiagnosing plausible.

So, despite extended efforts there is obviously a risk that *Metastrongylus* spp. remain undetected in individual pigs, as well macroscopically in the lungs as microscopically in faeces. Possible explanations for this could be that there is no correlation between the number of adults in the lungs and the number of eggs shed in faeces (11) and that the number of eggs shed per gram faeces was low; 400±602 - or 206±169 if the only pig that shed more than 1,000 eggs per gram faeces was excluded. Regardless of these diagnostic shortcomings we managed to demonstrate adult lungworms or their eggs in 13 out of 25 (52%) examined pigs from the affected herd at slaughter of which at least 10 pigs had not been associated with respiratory diseases at all.

Seen from this perspective, and by also including the demonstration of adult lungworms in the other outdoor herd located 150 km away, it appeared reasonable to assume that *Metastrongylus* spp. also is present in other herds rearing pigs in outdoor systems. Maybe especially in areas with high densities of wild boars since the density of wild boars has been shown to be proportional to the presence of *Metastrongulys* spp. in earthworms (17), as to the parasitic burden in the wild boars themselves (18). For this reason, parasites that can affect both wild boars and domesticated pigs will be accumulated around feeding spots for wild boars (18), and it can be concluded that it must be inappropriate to establish feeding spots for wild boars in the neighbourhood of pig herds. Especially pig herds with access to outdoor grazing. It cannot be excluded that wild boars actively visit pastures for aimed for domesticated pigs in search for food, which of course may lead to exchange of microorganisms between the two species. If the wild boars carry *Metastrongylus* spp., which appears likely (19, 8, 9, 10), the soil of the pastures will be contaminated with *Metastrongylus* eggs that will turn infective for pigs by earthworms (1).

The wild boar population in Sweden has been evolving since decades and during hunting lungworms are frequently seen in animals younger than one year, but less frequently in older animals (10). This is in accordance with observations from Corsica (19), Spain (8) and Italy (9), and it has been assumed that wild boars older than one year have developed an immunity to the parasite. However, the observations made in his study indicated that the load of *Metastrongylus* spp. was low to moderate in affected domesticated pigs in the herds examined. The clinical influence of lungworms therefore was suggested to be limited, possibly with exception for a risk to facilitate the entrance of secondary invaders (3).

The results obtained at the farrowing site located indoors revealed that the parasitic burden there was low to absent. Consequently, the presence of both *A. suum* and *Metastrongylus* spp. was related to the pastures and not to the growers that populated the pastures. Due to the high load of *A. suum* demonstrated in pasture B during the disease outbreak, it was decided to plough up that pasture and with
the aim to reduce the parasitic load grow cereals the next season instead of grazing pigs. The long-term efficacy of that measure could of course be discussed due to the high survival capacity of the eggs to *A. suum*, infective eggs have been demonstrated in a pig stable that had been empty for 13 years (20), but by rotating pastures the parasitic burden could hopefully remain at a moderate level.

The high numbers of eggs to *A. suum* determined at the concrete slabs where pigs spent their last weeks prior to slaughter was explained by the fact that pigs if not already infected with *A. suum* on arrival turned infected due to residual eggs to *A. suum* in the slabs. This residual infection ought to be reduced by improved hygienic measures between batches (6). As stated above that would not kill the eggs, but a thorough cleaning may hopefully remove a large number of them from the slabs.

The high load of *A. suum* in combination with the potential correlation between that parasite and the severe outbreak of respiratory disease at pasture B highlighted that rotation of pastures ought to be considered when rearing pigs outdoors. The best option would of course be to only use a pasture for one year in combination with growing cereals for as many years as possible before again grazing pigs, but in reality the access to pastures and the extent of the production will decide the turn-over time for rotation of pastures. Most likely, also labour needed and expenses for establishment of new pastures must be taken into account, making rotation of pastures into a question that also concern economy. On the other hand, the economic losses for disease outbreaks like the one described in pasture B will be significant, and costs for improved measures to prevent such outbreaks ought to be regarded as profitable in relation to disease outbreaks (13).

**Conclusion**

As *Metastrongylus* spp. were demonstrated regardless of health status, and also in another healthy outdoor herd, the impact of *Metastrongylus* spp. on the outbreak of respiratory disease was depreciated. Instead, *Metastrongulys* spp. was suggested to be common in outdoor production, although rarely diagnosed. The reason for this is because they will escape detection at routine inspections at slaughterhouses, and that they appeared to generally not induce clinical signs of respiratory disease. Instead, a possible association with a high burden of *Ascaris suum* was suggested to have preceded the severe respiratory disease in pasture B.

**Materials And Methods**

**Herd**

The affected herd was an integrated herd with 88 sows were eight sows farrowed indoors every second week in an age segregated system. The offspring were reared indoors until the age of around eleven weeks when they were transferred to one out of three pastures outdoors (Figure 1; A, B or C). The pastures had huts where pigs could seek shelter from harsh weather. Around one month before reaching market weight of approximately 130 kg live weight, the pigs were transferred to concrete slabs located in
absolute vicinity to the abattoir (Figure 1, D or E). These concrete slabs were also located outdoors. Pigs in the slabs had access to ponds and could seek shelter from harsh weather in the surrounding building.

**Clinical signs initiating the study**

In May 2021, 131 pigs with a mean weight of approximately 30 kg were transferred to pasture B. A severe outbreak of respiratory disease took place in this group 4-7 weeks after the allocation. Two pigs that had died were sent for necropsies at the National Veterinary Institute SVA, and the disease outbreak was monitored by looking into the records and treatment journals of the herd.

There had been no signs of respiratory disease at the other pastures (Figure 1; A and C) or at the concrete slabs (Figure 1; D and E).

**Parasitological examinations of the herd**

Coinciding in time with the disease outbreak in pasture B, faecal samplings were collected from growers at all pastures (Figure 1; A, B and C) as well as from one of the concrete slabs (D) that housed pigs close to market weight that previously had been grazing at pasture A.

To get information regarding the parasitological status of sows and of the offspring when transferred from the indoor facilities to the pastures, individual faecal samples were collected from one group of non-dewormed pregnant sows (n=8) and pen samples were collected from 8 different pens housing non-dewormed growers aged 10 weeks.

All faecal samples were analysed for presence of parasites with a centrifugal flotation technique. Nematode eggs were identified and quantified by a modified McMaster technique with a lower detection limit of 50 Epg faeces (21).

**Examinations made at slaughter**

When the first pigs in the batch that had been affected with the severe respiratory disease outbreak at pasture B reached market weight, ten market weight pigs and five of the smallest pigs from that batch were slaughtered and examined with focus on presence of parasites in lungs and in faeces. As a control, ten other pigs that had been grazing on pasture A and not attended with respiratory disease were also slaughtered and examined in the same way.

During the same day, outdoor pigs from another herd (Herd V), located 150 km away from the affected herd, were also slaughtered at the abattoir. The lungs from seven pigs from Herd V were examined for presence or parasites in the lungs.
The individual slaughter weights were recorded for all these animals, and the live weight was calculated as the slaughter weight * 1.34. The internal organs were inspected in detail; the number of assumed A. suum-induced white spots in the livers were counted, and the lungs were carefully examined with respect to lesions and presence of parasites.

In addition, individual faecal samples were collected from the 25 pigs of the effected herd that were examined (n= 10 + 5 that had been grazing at the affected pasture B and the 10 healthy pigs that had been grazing at pasture A). The faecal samples were analysed for presence of parasite eggs using centrifugal flotation technique and quantified by a modified McMaster technique (21).

Individual blood samples without additives were also collected from the 25 pigs from pasture B (n=10 + 5) and pasture A (n=10). The samples were centrifuged and serum stored at -18°C until analysed for presence of antibodies to respiratory pathogens with different ELISA systems; M. hyopneumoniae (IDEXX M. hyo. Ab test, IDEXX, Westbrook, USA) A. pleuropneumoniae serotype 2 and 3 (22), and P. multocida (23).

Individual blood samples were also collected with EDTA as additive. These samples were analysed with respect to concentrations of hemoglobin and leukocytes, and the differential counts of the subpopulations of leukocytes (Exigo, Boule Medical AB, Spånga, Sweden).

Statistics

Measurements presented are, unless specified otherwise, presented as mean values with standard deviations. Statistical analyses regarding body weights, levels of antibodies, leukocytes and hemoglobin were carried out using student t tests.

Abbreviations

°C = Degree Celsius

A. Pleuropneumoniae = Actinobacillus pleuropneumoniae

A. suum = Ascaris suum

Epg = Egg per gram

FEC = Faecal Egg Count

Hb = Hemoglobulin

M. hyopneumoniae = Mycoplasma hyopneumoniae

M. hyorhinis = Mycoplasma hyorhinis
L = litre

*P. multocida* = *Pasteurella multocida*

Spp. = Species

**Declarations**

**Availability of data and materials**

Not applicable.

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**Contributions**

PW was contacted by a practitioner about the disease outbreak and thereafter contacted the herd owner. PW designed the study in collaboration with EP. EP made all copromicroscopical analyses and PW responded for the other analysis. PW and EP wrote the manuscript.

**Ethics approval and consent to participate**
This study was approved by an ethical permission from the Swedish Board of Agriculture, processed by the committee för ethics of Uppsala in Sweden (Dnr 5.8.18-06256/2019) entitled Scientific investigations effectuated following ordinary disease investigations in animals.

**Consent for publication**

The authors declare that the owners of the pigs, which were equal to the owners of the abattoir, gave a consent for publication of the data.

**Competing interests**

The authors declare no conflict of interest.

**Additional information**

None

**Supplementary information**

Not added

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**Tables**

**Table 1**

*Presence of parasite eggs in the indoor facilities. Faecal samples were collected from individual sows ante partus and from pens with growers aged 10 weeks. None of the pigs had been treated with anthelmintic drugs.*

| Age          | n  | Roundworms (*Ascaris suum*) | Whipworms (*Trichuris suis*) | Lungworms (*Metastrongylus* spp.) |
|--------------|----|-----------------------------|------------------------------|----------------------------------|
| Sows         | 8  | 0                           | 0                            | 0                                |
| Growers      | 8  | 0                           | 0                            | 0                                |

**Table 2**

*Presence of parasite eggs at the pastures and a concrete slab during the disease outbreak at pasture B*
| Pasture | Roundworms (*Ascaris suum*) | Whipworms (*Trichuris suis*) | Lungworms (*Metastrongylus spp.*) |
|---------|-----------------------------|-------------------------------|----------------------------------|
| **Health status** |               |                               |                                  |
| Age     |               |                               |                                  |
| **Pasture C** | Positive 0 / 3 | 0 / 3 | 0 / 3 | 0 % | 0 % | 0 % |
| **Healthy pigs** | prevalence 0 % | 0 % | 0 % |                                  |
| 4 months | Epg            |                               |                                  |
|         | Mean 0         | 0                              | 0                                |
|         | Max 0          | 0                              | 0                                |
|         | Min 0          | 0                              | 0                                |
| **Pasture B** | Positive 4 / 5 | 4 / 5 | 1 / 5 | 80 % | 80 % | 20 % |
| **Affected pigs** | prevalence 80 % | 80 % | 20 % |                                  |
| 5 months | Epg            |                               |                                  |
|         | Mean 2090 ± 1545 | 350 ± 453 | 10 ± 22 |                                  |
|         | Max 3800       | 1100                           | 50                               |
|         | Min 0          | 0                              | 0                                |
| **Pasture A** | Positive 1 / 5 | 0 / 5 | 1 / 5 | 1 / 5 | 20 % | 20 % |
| **Healthy pigs** | prevalence 0 % | 0 % | 20 % |                                  |
| 6 months | Epg            |                               |                                  |
|         | Mean 0         | 0                              | 10 ± 23                          |
| Slab D (From A) | Positive prevalence | 3 / 4 | 0 / 4 | 1 / 4 |
|----------------|---------------------|-------|-------|-------|
|                | prevalence          | 75 %  | 0 %   | 25 %  |

**Healthy pigs**

7 months **Epg**

|               | Mean    | Max   | Min   |
|---------------|---------|-------|-------|
| **Mean**      | 1525 ± 2205 | 4800  | 0     |
| **Max**       |          | 4800  | 150   |
| **Min**       |          | 0     | 0     |

**Figures**
Figure 1

Geographic overview of the pastures and the flow of animals. Greene areas represent forests, white areas represent buildings or cultivated land, blue areas represent water. The black lines represent roads (a).

At the age of around 11 weeks, growers were transferred from the indoor facilities to pasture A, B or C. Approximately one month before reaching market weight they were transferred to either outdoor concrete
slab D or E located close to the abattoir.

Figure 2

Mean values and standard deviations for live weights (kg), and incidences (%) of macroscopical lesions in the lungs at slaughter (healing mycoplasma-like pneumonias, pleuritis and presence of lungworms). Regarding white spots in livers, the mean number of white spots per pig is shown.
Figure 3

Mean values and standard deviations for live weights (kg), hemoglobin (gram per L) and total number of leukocytes ($10^8$ per L). The figure also shows the subpopulations of the leukocytes as percentages of the total number of leukocytes.
Figure 4

Absorbance levels (mean + standard deviations) of serum antibodies to Mycoplasma hyopneumoniae, Pasteurella multocida and Actinobacillus pleuropneumoniae serotypes 2 and 3. The cut off-value for positive reactions was 0.5 for all tests.

Figure 5

Incidence of parasite eggs in faeces collected at an individual level at slaughter (left), and the maximal amount of eggs demonstrated per gram faeces in an individual pig (right).