Tail and ear necrosis in piglets of sows with increased weight loss over the suckling period

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Schwanz- und Ohrnekrosen bei Ferkeln von Sauen mit erhöhtem Gewichtsverlust während der Säugezeit

Ein Betrieb aus einem Schweizer Abferkelring meldete vermehrt Fälle von Nekrosen an Schwanzansatz oder Ohren der Saugferkel. Während einer Bestandsuntersuchung im Februar 2021 wurde festgestellt, dass etwa die Hälfte aller untersuchten Würfe Ferkel enthielt, die Nekrosen an verschiedenen Stellen aufwiesen und dass die Sauen dieser Ferkel eher dünn waren. Der Landwirt dokumentierte daraufhin vor und nach dem Absetzen den Body Condition Score (*BCS*) und das Gewicht, sowie die Anzahl der lebendgeborenen Ferkel mit Schwanz- oder Ohrennekrose der nächsten vier Abferkelpartien. Insgesamt wurden Daten von 97 Sauen mit 1214 lebendgeborenen Ferkeln ausgewertet. Nachträglich wurden die Sauen in zwei Gruppen eingeteilt: Muttersauen mit Ferkeln die Ohr- und/oder Schwanznekrose (NE) aufwiesen und solche ohne (WN). Von den 97 Würfen enthielten 40 Würfe Ferkel mit Nekrosen. Von diesen betroffenen Würfen enthielten 28 nur Ferkel mit Schwanznekrosen, 8 nur mit Ohrnekrosen und 4 Würfen hatten Ferkel mit beiden Arten von Nekrosen. Die Muttersauen der Gruppe NE verlor signifikant mehr Gewicht und BCS-Punkte während der Säugezeit als die Gruppe WN, mit einer Tendenz zu einem niedrigeren BCS nach dem Absetzen (2,0 vs. 2,25/5,0). Blutproben von fünf Sauen wurden analysiert und positiv auf das *Fusarium*-Mykotoxin Deoxynivalenol (DON) getestet. Möglicherweise haben die Sauen zuvor DON-kontaminiertes Futter aufgenommen, das dann in ihrem Fettgewebe gespeichert und bei zunehmendem Gewichtsverlust wieder in die Blutbahn abgegeben wurde. Da DON während der Trächtigkeit oder Laktation von der Muttersau auf ihre Ferkel übertragen werden kann, könnte dies bei den Ferkeln zu Schwanz- oder Ohrennekrose geführt haben. Um diese Hypothese zu überprüfen braucht es jedoch noch diverse Studien.

Schlüsselwörter: Mykotoxin, Nekrose, Ferkel, Gewichtsverlust

Summary

A farm belonging to a Swiss sow pool system reported increased cases of necrosis on the base of the tail or ears in their piglets. Therefore, herd examination was performed in February 2021, and it was found that about half of all examined litters included piglets with necrosis of different locations, and that the sows of these piglets were rather thin. Upon instruction, the farmer then documented the body condition score (*BCS*) and weight before farrowing and after weaning, and the number of liveborn piglets affected by necrosis of the tail or ear of the next four farrowing batches. In total, data of 97 sows with 1214 liveborn piglets were evaluated. Sows were retrospectively allocated into two groups: Those with piglets with ear and/or tail necrosis (NE), and those without (WN). Of the 97 litters, 40 included piglets with necrosis, with 28 of them having piglets only with tail necrosis, 8 only with ear necrosis, and 4 litters included piglets with both types of necrosis. The group NE lost significantly more weight and *BCS* points over the suckling period than the group WN, with a tendency of having a lower *BCS* after weaning (2,0 vs. 2,25/5,0). Blood samples of five sows were analyzed and tested positive for the *Fusarium* mycotoxin deoxynivalenol (DON). It could be possible that the sows previously consumed DON contaminated feed, which was then stored in their fat tissue, and released again into the blood stream during increased weight loss. Since DON can be transferred from the sow to her piglets during gestation or lactation, this release might have affected the piglets, leading to tail or ear necrosis. However, causative studies are needed to confirm this hypothesis.

Keywords: mycotoxin, necrosis, piglets, weight loss

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Introduction

Mycotoxins are secondary metabolites of toxigenic fungi that can accumulate in feed before and after harvest, with *Fusarium* toxins being the most frequently detected ones.[6,11] Pigs are highly sensitive to feed contamination with deoxynivalenol (DON), the major *Fusarium* mycotoxin, formerly known as «vomitoxin» because of its induction of vomitus.[21] However, what clinical symptoms the pigs show depends on the concentration and duration of DON exposition.[18,22] The EU maximal guidance value[4] for feed for pigs is 0,9 mg/kg. Decreased feed intake and decreased weight gain in pigs is seen above approximately 1 mg/kg, total feed refusal at 12 mg/kg, and vomiting at 20 mg/kg.[12, 22] The growth depression increases with increasing concentration of DON in feed.[3] Additionally, it seems that even short DON exposition can have long-term effects on the gut integrity.[16] Apart from symptoms in the digestive tract, other organs can be affected like the immune system, the liver, the uterus, the placenta, or the fetus in uteri.[17,24]

DON can be found in cereal crops such as wheat, and its contamination of animal feed is a relevant problem.[14] An increase of DON contamination of wheat due to climate changes is expected in the future.[19] Higher mean DON concentrations were found in feed and sow plasma of herds affected with tail necrosis in piglets than in unaffected herds.[20] However, the pathogenesis or connection between DON and necrosis in piglets has not been determined yet. DON can be transferred via the placental barrier from the sow to her piglets during gestation, or via milk during lactation.[1,2] It can be detected in blood, milk, fat, liver tissue (also of the fetus), and bile, with the highest concentrations found in bile.[15] A value of approximately 30 µg/L DON concentration in blood should not be exceeded since it can lead to fertility problems and performance depression, while in bile, the limit is set around 80 µg/L.[15]

Table 1: Differences for measured parameters between sows with piglets with tail or ear necrosis (NE) and those without necrosis (WN), including weight *BCS*[1] before farrowing («in») and after weaning («out»), leading to the weight and *BCS* loss over the suckling period. Data are presented as median or mean value ± standard deviation. Significance was assumed when $P < 0,05$ and is marked with**. A tendency towards significance was assumed when $0,05 < P < 0,10$ and is marked with*.

Parameter	P-Value	Group NE	Group WN
Parity	0,51	3	2
Weight in (kg)	0,10	250,2 ± 33,7	237,3 ± 39,4
Weight out (kg)	0,66	205,7 ± 30,3	202,5 ± 36,9
Weightloss (kg)**	0,01	-44,6 ± 15,8	-34,8 ± 17,8
Weightloss (%)**	0,02	-17,7 ± 6,0	-14,6 ± 6,9
BCS in	0,32	2,50	2,50
BCS out*	0,05	2,00	2,25
BCS loss**	0,03	-0,50	-0,50
Liveborn piglets per farrow	0,17	13,00	12,00

[1]*BCS* = Body Condition Score

Maintaining an optimal body condition throughout sows' production cycle is of great importance, because especially poor/low body condition is associated with various diseases and can also affect the new litter.[13] Furthermore, assuming that DON is released from fat stores during catabolic situations and transferred into milk, feed contamination with DON does not only affect the sow that primarily ingested DON from feed, but also her suckling piglets.

This case report describes the correlation of increased weight loss during the suckling period in 97 sows with the occurrence of tail or ear necrosis in their piglets.

Case presentation

A farm belonging to a Swiss sow pool system in the canton of Berne, Switzerland, reported increased cases of tail necrosis in piglets since a couple of months. The sows were of mixed breed (Swiss Large White x Landrace) and inseminated with the semen of Piétrain. In February 2021, the Clinic for Swine, Vetsuisse Faculty, University of Bern was contacted by the herd attending veterinarian. Obviously, beginning one day *post-partum*, most of the litters had piglets with necrosis on the tail, ear, or teats, affecting approximately 20 % of all piglets, with higher percentages estimated in the past. Some lesions healed during the suckling period, some tails fell off around 1 cm behind the base of the tail. Piglets of sows of this sow pool system in other farrowing farms did allegedly not show any signs of tail necrosis. Sows in the mating units were fed with corn cobs before they were transported to the gestation unit approximately one week before the expected farrowing. There they were fed with 2,6 kg mixed feed per day (14 MJ VES/kg), that amount was reduced to 2 kg before farrowing. Piglet losses were estimated at about 10 %. Food and straw were tested negative for DON.

Herd examination

During the herd examination in the farrowing unit, it was found that about half of all litters included piglets with necrosis on the base of the tail or ear, the vulva or teats, or a combination of them (Figure 1 a-d). Additionally, reddish discoloration was sometimes observed on the sole of the claw. The piglets were of good general health and body condition. Sows of affected litters had a *BCS* of 2,0–3,0 /5,0 at 4 d *post-partum*. The feces of the sows were rather dry. In the piglet rearing barn, some of the newly moved piglets showed a 1 cm long healed tail, while in the group of the three weeks older piglets, around 10 % showed lesions on the base of the tail of different severity.

To determine if mostly piglets of thin sows were affected, a documentation of the next farrowing batches was recommended. Additionally, the private veterinarian took blood samples from five sows with affected litters that were ana-

lyzed in a laboratory for DON, using high performance liquid chromatography (BioCheck, Leipzig).

Data analyses

In February 2022, the documentation of four farrowing batches with 23 to 25 sows per batch that farrowed between the end of February 2021 and the beginning of June 2021 was provided. The farmer documented the parity, the weight and *BCS* before moving into the farrowing unit (around four to six days before farrowing; =«in») and again when leaving it (after weaning, =«out»), as well as the number of liveborn piglets per litter and the number of piglets affected by necrosis of the tail or ear. From this data, we calculated the weight and *BCS* loss over the suckling period, and the percentage of piglets of affected litters that were affected by necrosis. For statistical analyses, the NCSS statistical software was used. All parameters were analyzed descriptively and tested for normality using the Shapiro-Wilk normality test. Afterwards, two groups were formed with the sows: Sows that had piglets with ear and/or tail necrosis (NE) and those without necrosis (WN). Normally distributed data were analyzed using the t-test, non-normally distributed data were analyzed using the Wilcoxon Rank-Sum test. Statistical significance was assumed when $P < 0,05$.

Results

In total, data of 97 sows with 1214 liveborn piglets were included, with a median of 12 (range 4 to 26) liveborn piglets per sow. Sows were of a median parity of 3 (range: 1 to 10), a median *BCS* of 2,5/5,0 (range 1 to 4) and an average weight of 242,6 ± 37,5 kg (mean value ± standard deviation; with a median of 247 kg, range 160 kg to 345 kg) before farrowing. Sows (n=97) left the farrowing pen with a median *BCS* of 2,0/5,0 (range 1 to 3,5) and an average weight of 203,8 ±34,2. They therefore lost -0,5 *BCS* points (range -1 to +0,25) and 38,8 ±17,6 kg (15,9 ± 6,7 % of the starting weight) during the suckling period. Of the 97 litters, 40 included piglets with necrosis: 28 only with tail necrosis, 8 only with ear necrosis, and 4 litters included piglets with both ear and tail necrosis. Within an affected litter, on average 28,1 % of the liveborn piglets were affected by tail necrosis, while 38,2 % were affected by ear necrosis.

The group NE lost significantly more weight in total (-44,6 ± 15,8 kg vs. -34,8 ± 17,8; $P = 0,01$) and proportionally (-17,7± 6,0 % vs. -14,6 ± 6,9 %; $P = 0,02$) over the suckling period than the group WN (*Table 1*). Additionally, their *BCS* loss was significantly greater than that of the WN group ($P = 0,03$), with a tendency of having a lower *BCS* after the suckling period (2,0 vs. 2,25; $P = 0,05$). No differences were found for parity, the total weight and *BCS* shortly before farrowing (= weight in) and the weight shortly after weaning (= weight out; *Table 1*).

The blood analysis of the five sows with affected piglets from one farrowing group detected total values between 148,3–199,7 µg/L. The applied method measures DON and its toxic metabolites 3- and 15 acetyl-DON, and it includes the degradation product deepoxy-deoxynivalenol DOM, which accounted for 44–60 µg/L of our total values.

Discussion

Data analyses showed that sows having piglets with tail or ear necrosis lost more weight during the suckling period than sows without. Even though the pathogenesis of DON exposition of the sow and tail necrosis of their piglets has not been fully determined yet, another study found a correlation between tail necrosis in new born piglets and elevated concentration of DON in feed and sow plasma, and it has already been demonstrated that DON can be transferred from the sow to her piglets.[1,20] However, they also

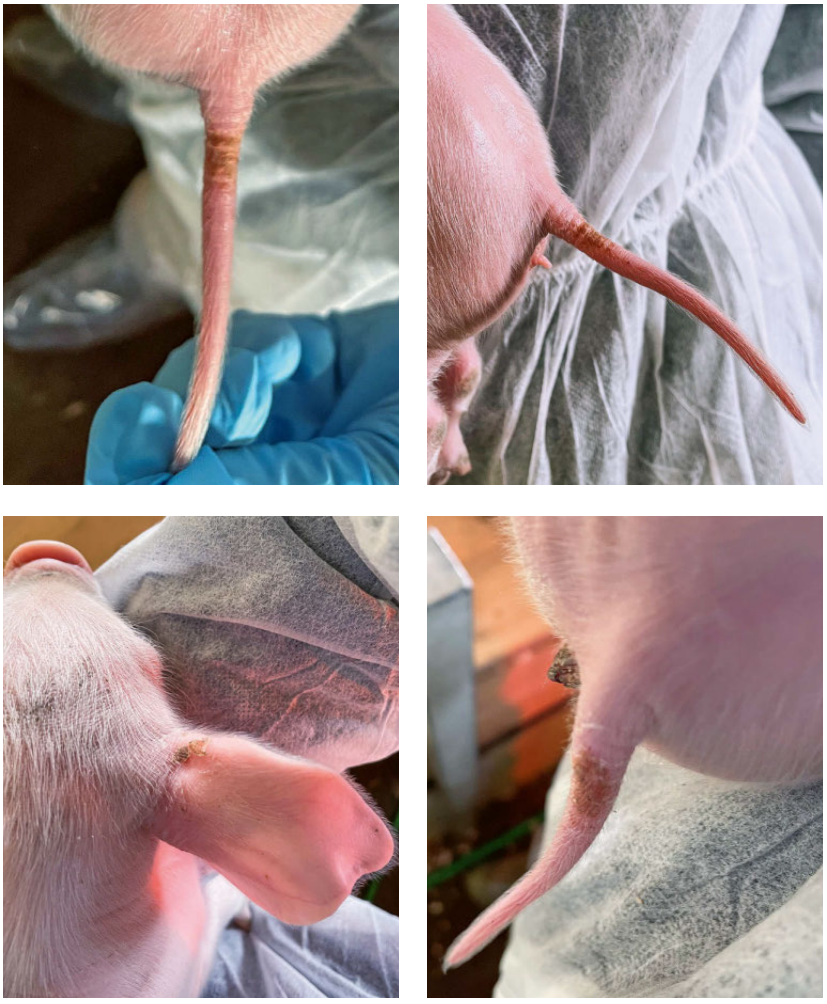

Figure 1 a–d: Piglets born within the last 3 days showing necrosis on the base of the tail or ear, the vulva or teats, or a combination of them.

discussed that another study experimentally exposing piglets to DON via the placenta did not describe neonatal tail necrosis, and that also other factors might play a part in the occurrence of this phenomen.[2,20]

Even though the feed samples that were tested for DON by the farmer were negative, a contamination cannot be excluded, especially since the samples were only taken once, and not repeatedly during the period of data collection, and also because the sows could have consumed the contaminated feed at another farm of the sow pool system. Additionally, van Limbergen et al. (2017) discussed that even DON-concentrations in feed lower than the EU maximum guidance value might affect the piglets. However, feed contamination with DON can be rather high, as one study found up to 89 % of tested feed samples to be contaminated.[6] But also a co-contamination has to be kept in mind since often more than one mycotoxin is found, like additionally zearalenone or fumonisins.[6] Additionally, the rainy weather, as it was seen last year (2021) in Switzerland, may have increased the risk of contaminated cereals.[7,10] Even though the source of contamination in this case was not detected, analyzed blood samples of five sows revealed increased DON values,[15] proofing that the sows had contact to this mycotoxin at some point. A good indicator to differentiate between prenatal DON exposition and DON uptake via colostrum would have been to test the blood of the newborn piglets after birth, before and after colostrum uptake.

Though only found in very low concentrations, DON was detected in the back fat of fattening pigs, and others found DON in fat even in a similar concentration as in blood.[5,15] It could therefore be possible that the uptake of DON by the sow was not at the same time as the clinical signs in the piglets were visible. The sows could have stored DON in fat, which was then released during fat loss. This would also explain why there were only signs in the piglets, but not the sows. However, a moderately reduced feed intake in the sows could have gone unnoticed, but the sows of the two groups did not significantly differ in weight or *BCS* before entering the farrowing barn. Clinical signs of DON exposition are reduced growth performance in pigs due to vomiting, reduced feed intake, decreased glucose uptake, disruption of the intestinal barrier, and more.[6] Whether the piglets also showed other symptoms apart from the necroses like reduced growth performance was not examined in our case, but the farmer did not specifically notice anything out of the order. Additionally, a possible adaptation of the sows to DON during prolonged exposure has to be kept in mind.[3] Regardless of the group, the *BCS* of all the sows were rather low when entering the farrowing unit. This might have affected the piglets, since low backfat thickness is associated with various reproductive performances like smaller litter sizes or weaning weight of the piglets.[13] Indeed, the median of liveborn piglets in our case report was 12 for the group without necrosis and 13 for the group with necrosis, while another recent study in Switzerland found a median of 15 liveborn piglets per sow.[9] However, *BCS* scoring is not the most reliable tool to examine changes in the body condition, because it is very subjective and only poorly correlated to backfat thickness.[23]

The pathogenesis and cause of necrosis of different localisations in piglets has not yet been determined. It was suggested that, at least in newborn piglets, it is more of a syndrome of inflammation and necrosis (SINS) with a mainly endogenous origin, in which mycotoxins could act as a possible co-cause.[8] Our case report did not investigate different possible causes or the interactions between them and DON, but further studies analyzing this would be of interest. The breed seems to play a role in the occurrence of this syndrome, since offspring of Piétrain boars had higher SINS scores than those of Duroc boars, and the sows reported in this case were inseminated with the breed Piétrain.[8] However, different endogenous and exogenous factors might play a part and need further examinations.

The pathogenesis of how feed contamination with DON, increased weight loss of the sow, and necrosis in their piglets are connected is not fully known yet. We do not know at what point in time the sows ingested DON in this case. However, we hypothesize that they ingested DON, stored it in fat, and then released it during a katabolic situation. It is known that the DON ingestion itself can lead to the decreased feed intake what may logically lead to fat loss,[12] but since the farmer did not notice any reduction in feed intake, the other way around seems to be more plausible. Interestingly, even though it was not significant, the sows that had piglets with necrosis seemed to be heavier when entering the farrowing pen than the sows without. Therefore, they could have had more fat stores to mobilize thus releasing more DON. Causative studies are needed to further investigate this phenomenon.

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Rapport de cas : Nécrose de la queue et des oreilles chez les porcelets de truies présentant une perte de poids accrue pendant la période d'allaitement

Une exploitation appartenant à un système suisse de pool de truies a signalé une augmentation des cas de nécrose à la base de la queue ou des oreilles chez ses porcelets. Par conséquent, un examen de l'effectif a été effectué en février 2021 et il a été constaté qu'environ la moitié de toutes les portées examinées comprenaient des porcelets présentant des nécroses à différents endroits et que les mères de ces porcelets étaient plutôt maigres. Sur instruction, l'éleveur a ensuite documenté la note d'état corporel (BCS) et le poids avant la mise bas et après le sevrage, ainsi que le nombre de porcelets nés vivants affectés par une nécrose de la queue ou de l'oreille des quatre lots de mise bas suivants. Au total, les données de 97 truies avec 1214 porcelets nés vivants ont été évaluées. Les truies ont été réparties rétrospectivement en deux groupes : Celles dont les porcelets présentaient une nécrose de l'oreille et/ou de la queue (NE), et celles qui n'en présentaient pas (WN). Sur les 97 portées, 40 comprenaient des porcelets atteints de nécrose, dont 28 uniquement avec une nécrose de la queue, 8 uniquement avec une nécrose de l'oreille et 4 avec les deux types de nécrose. Le groupe NE a perdu beaucoup plus de poids et de points BCS pendant la période d'allaitement que le groupe WN, avec une tendance à avoir un BCS plus faible après le sevrage (2,0 vs. 2,25/5,0). Les échantillons de sang de cinq truies ont été analysés et se sont révélés positifs pour la mycotoxine de Fusarium, le déoxynivalénol (DON). Il est possible que les truies aient consommé des aliments contaminés par le DON qui a ensuite été stocké dans leur tissu adipeux puis libéré dans le sang lors de la perte de poids. Comme le DON peut être transféré de la truie à ses porcelets pendant la gestation ou la lactation, cette libération pourrait avoir affecté les porcelets, entraînant une nécrose de la queue ou des oreilles. Cependant, des études causales sont nécessaires pour confirmer cette hypothèse.

Mots clés: mycotoxine, nécrose, porcelets, perte de poids

Caso: coda e orecchie necrosate in suinetti di scrofe con un'aumentata perdita di peso durante il periodo dell'allattamento

Un allevamento appartenente a un sistema svizzero di pool di scrofe ha segnalato un aumento dei casi di necrosi alla base della coda o delle orecchie nei suinetti. Per questo motivo, nel febbraio 2021, è stato effettuato un esame della mandria ed è stato riscontrato che circa la metà di tutte le figliate esaminate comprendeva suinetti con necrosi in diverse sedi e che le scrofe di questi suinetti erano piuttosto magre. Su istruzione, l'allevatore ha quindi documentato il punteggio di condizione corporea (BCS) e il peso prima del parto e dopo lo svezzamento, nonché il numero di suinetti nati vivi affetti da necrosi della coda o dell'orecchio dei quattro lotti successivi. In totale, sono stati valutati i dati di 97 scrofe con 1214 suinetti nati vivi. Le scrofe sono state assegnate retrospettivamente in due gruppi: quelle con suinetti con necrosi dell'orecchio e/o della coda (NE) e quelle senza (WN). Delle 97 figliate, 40 includevano suinetti con necrosi, di cui 28 solo con necrosi della coda, 8 solo con necrosi dell'orecchio e 4 cucciolate includevano suinetti con entrambi i tipi di necrosi. Il gruppo NE ha perso significativamente più peso e punti BCS durante il periodo di allattamento rispetto al gruppo WN, con una tendenza ad avere un BCS più basso dopo lo svezzamento (2,0 vs. 2,25/5,0). I campioni di sangue di cinque scrofe sono stati analizzati e sono risultati positivi alla micotossina *Fusarium* deossinivalenolo (DON). Si pensa che le scrofe abbiano consumato in precedenza mangime contaminato da DON, quest'ultimo poi è stato immagazzinato nel tessuto adiposo e rilasciato nuovamente nel flusso sanguigno durante l›aumento della perdita di peso. Poiché il DON può essere trasferito dalla scrofa ai suoi suinetti durante la gestazione o l›allattamento, questo rilascio potrebbe aver colpito i suinetti, portando alla necrosi della coda o delle orecchie. Tuttavia, sono necessari studi causali per confermare questa ipotesi.

Parole chiave: micotossina, necrosi, suinetti, perdita di peso

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