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The Dietary Risk Factors of Gastric Ulcers in Finishing Pigs from 16 Polish Farms

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Abstract: Sudden death caused by the acute form of gastric ulceration has developed into a widely recognised health and welfare problem. The importance of different triggering factors is poorly understood. The study was carried out on finishers slaughtered in Poland. The collected animal-level data were transformed to a herd-level dataset, which included 27 predictor variables. From a total of 32,264 pig stomachs examined, 23,188 (71.9%) had gastric lesions. Total of 17,703 organs (54.9%) had ulcers. Scores 1 (hyperkeratotic) and 2 (erosions) were observed in 2958 (9.2%) and 2527 (7.8%), respectively. A significant ($p < 0.05$) association of the ulcer prevalence was found for a number of variables. Raising the level of protein significantly ($p = 0.04$) increased ($\rho = 0.22$) the occurrence of gastric ulcers. The addition of wheat bran ($p = 0.02$) and its growing share in feed composition had the contrary effect ($\rho = -0.27$). Feeding pelleted feed was significantly associated ($p = 0.001$) with the higher prevalence of the disease, opposite to liquid feeding ($p = 0.0002$) and supplementation of a mycotoxin deactivator ($p = 0.0008$). Although, the immediate transition from one feeding system to another in most of the farms is impossible, the addition of materials such as wheat bran, and routine supplementation with mycotoxin deactivators may be a practical and relatively low-cost solution.

Keywords: pigs; finishers; gastric ulcers; abattoir survey; risk factors; feeding

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1. Introduction

The anatomy of porcine stomach translates into the species-specific phenomenon of gastric ulceration. Contrary to other animal species, (except the squamous region of the horse's stomach), gastric lesions in pigs almost exclusively occur in the non-glandular part of the organ. Parakeratosis, as a primary defence mechanism, protects the deeper layers of the non-glandular stomach in pigs (pars oesophagea) from injury. However, if the influence of the noxious stimuli persists, parakeratosis becomes a precursor of secondary lesions. Gradual breakage of intercellular junctions results in the formation of erosions. Subsequently, destruction of the lamina propria opens access to deeper tissues, which results in ulcer formation [1].

Sudden deaths caused by the acute ulceration of the pars oesophagea has recently developed into a widely recognised health and welfare problem in most swine-producing countries [2–7]. However, due to the legal regulations which allow to exclude examination of the organ from a routine post slaughter inspection, the epidemiological data from many countries are missing. Even though the evaluation of organ integrity has a clearly satisfactory cost-benefit ratio, almost the entire swine industry ignores the value of such data and its possible influence on improving animal welfare.

In experimental conditions, pathological lesions of the pars oesophagea were induced in pigs using different nutritional, surgical, pharmacological and other factors [8–13].

Under field conditions, dietary determinants have already been demonstrated to affect continuity of the pars oesophagea and are commonly thought to be of primary importance for the disease development. However, some of the previous experiments were biased by the short-term observations of relatively smaller groups of animals fed with feed, composition of which had been drastically and selectively changed for the purpose of the experiment [14–17], and not reflecting the real conditions of a modern farming. Furthermore, some research assessing the risk factors was based on the questionnaires filled by farm managers or owners, which may significantly impair the data quality and bias the results [18,19].

The importance of different disease triggering factors is poorly understood. In spite of the fact that the disease has become an inherent element of modern swine farming, practical and economically rational solutions have not been established so far. Irrespective of the conclusions drawn in previous publications, various feed components and additives are still generally assumed to have a gastroprotective effect, which usually instigates serious setbacks and financial losses.

The objective of the present study was to determine the influence of different dietary determinants, including analytical and botanical feed composition, as well as several feed additives and feed contaminants, on the development of gastric ulcers in fatteners from 16 modern, finishing farms in Poland.

2. Materials and Methods

The present study was carried out on finishers which were slaughtered between January 2013 and February 2017 in a single slaughterhouse in Poland. The animals were born in one of 6 commercial high-performing sow herds (1500–5000 sows in each) belonging to a single production company. All animals were reared in three-phase production using weekly batches and all-in all-out system. Piglets were weaned after 4 weeks of lactation and transported to the weaner farm assigned to the particular sow farm. After another 7 to 8 weeks weaners were transported to one of 16 finishing farms (T1–T16), with up to 16,000 fattening places in each. All the locations met the legal requirements of animal welfare on pig farms in the EU (Council Directive 2008/120/EC of 18 December 2008). The pig flow remained unchanged during the study.

All diets were formulated from grains using two feed mills and a wet feeding system. The pelleted feed for finishing farms T1–T7, T9–T14, T16 was produced in feed mill 1. The mash feed for farm T8 was manufactured in feed mill 2. In farm T15, the feed (containing 66% of water) was prepared on-site for the wet feeding system installed there. All purchased raw materials were subjected to strict quality control. Feed recipes were adjusted due to the seasonal availability and the price of particular components. The portioning scales were calibrated every 6 months to assure consistency of feed batches.

In feed mill 1, supplying farms T1–T7, T9–T14, T16, feed grains were disintegrated using hammer mill, then in order to produce a 4 × 25 mm cylindrical pellet, feed was dry steam conditioned and pelleted at 80 °C. In feed mill 2, supplying farm T8 with mash feed, all feed grains (except barley) were ground by hammer mill. Barley grains were milled using disc mill. In farm T15, where wet feeding was used, maize seeds were disintegrated (lack of whole kernels) using mobile hammer mill before ensiling in bunker silos.

Finishers fed with pelleted or mash feed had uninterrupted access to feeders. The farm equipped with a wet feeding system fed the animals three times a day (at 6 a.m., 11:30 a.m. and 6 p.m.) using throughs whose length allowed all the pigs to consume feed simultaneously.

In order to assess the level of protein, fat, fibre and water, the samples collected from every batch of feed (e.g., 20 tons) were analysed with InfraXact device (FOSS A/S, Denmark) using NIRS technology (near infrared spectroscopy) in a wavelength range between 570 and 1850 nm. For the determination of grinding intensity, the diets were sampled before pelleting (500 g) and examined using a LAB-11-200 laboratory shaker (Zakład Naukowo-Techniczny Eko-Lab, Brzesko, Poland) equipped with the sieve stack, 3, 2, 1 mm

in screen diameter (HAVER&BOECHER OHG Drahtweberei und Maschinenfabrik, Oelde, Germany) and 10 min of working time. The presence of mycotoxins, deoxynivalenol (DON), and ochratoxin A (OTA), was assessed with Veratox ELISA test (Neogen Corp., Lansing, MI, USA). The detection limit for deoxynivalenol and ochratoxin A was 0.1 and 1 ppb, respectively.

The finishers (23 weeks old, from 96.3 to 123.96 kg) were transported to the slaughterhouse on trucks meeting the requirements of Council Reregulation No 1/2005 of 22 December 2004 on the protection of animals during transport and related operations and amending Directives 64/432/EEC and 93/119/EC and Regulation No 1255/97. The feed was withdrawn for 12 h prior to the transportation. Depending on the farm location, transportation time varied from 15 min to 4 h. During the study, 32,264 stomachs were examined (Table 1). At the abattoir, the animals were stunned using carbon dioxide, suspended vertically and bled out through the neck tissue. The stomachs were opened along the greater curvature and examined at 20 min post slaughter after expelling the digesta and rinsing its wall with running water. Pathological alterations were graded by the same veterinarian using four-point scale outlined by Kopinski and McKenzie as follows [20]: grade 0—intact epithelium; 1—parakeratosis, 2—erosions, 3—developed ulcers.

Table 1. Herd-level prevalence of different stomach scores (*n* of observations = 360).

Score	Mean (%)	SD	Range in Batches (%)
0 (normal)	27.9	19.3	0–82.5
1 (hyperkeratotic)	9.2	6.9	0–44.4
2 (erosion present)	7.8	3.4	1.2–15.3
3 (gastric ulcers present)	55.1	18.4	14–89.8

All the statistical analyses were performed using STATA v.13.0 software (StataCorp LP, TX, USA). The p value ≤ 0.05 was considered significant in all the analysis. Spearman rank correlation and Kruskal–Wallis equality-of-populations rank test were used to identify relationships between the prevalence of gastric ulcers and continuous (content of particles <1 mm, protein, fat, feed humidity, fibre, triticale, barley, rye, soybean meal, wheat bran, deoxynivalenol) and nominal variables (dry feed, liquid feed, wheat, sunflower meal, alfalfa, maize, oat, rapeseed meal, lupine, faba bean, olive oil, mycotoxin deactivator, prebiotic, probiotic, mould inhibitor, detection of ochratoxin A), respectively. The normality of the data distribution was evaluated using quantile plots and Shapiro–Wilk test.

3. Results

From a total of 32,264 pig stomachs examined in the study, 23,188 (71.9%) had gastric lesions. About 17,703 stomachs (54.9%) had ulcers (score 3, i.e., defects extending into the submucosa, taking the form of a crater, with a usually smooth bottom macroscopically resembling intact tissue). In the rest of the animals, scores 1 (hyperkeratotic) and 2 (erosions) were observed in 2958 (9.2%) and 2527 (7.8%), respectively. Overall, only 9076 (28.1%) of evaluated pigs were classified as score 0 (intact epithelium).

The herd-level prevalence values of the different score lesions differed slightly from the animal-level one (Table 1). The variation between the batches was evident, and from 0 to 82.5% animals had intact stomach, 0 to 44.4% had hyperkeratosis, 1.2 to 15.3% erosions and 14 to 89.9% had gastric ulcers. The differences were statistically significant ($\chi^2 = 222.1$; $p = 0.0001$).

The collected animal-level data were transformed to a herd-level dataset, which included 27 predictor variables divided into five subcategories (diet form, analytical components, botanical components, feed additives, feed contaminants) and the percentage of pigs with developed ulcers of the pars oesophagea (score 3) originating from one herd as response variable. The strength of the associations was tested separately for all continuous (Table 2) and nominal (Table 3) explanatory variables.

Table 2. Descriptive statistics of the continuous predictor variables and their association with the gastric ulcers prevalence in pig herds.

Variable	Number of Observations	Median	Range	Spearman's ρ	*p*
Diet form					
Content of particles <1 mm (%)	30	61.4	53.6–76.6	−0.08	0.67
Analytical components					
Protein (%)	83	15.0	14.7–15.9	0.22	**0.04**
Fat (%)	83	3.69	3.13–4.48	−0.17	0.13
Feed humidity (%)	83	11.85	11.03–13.7	0.12	0.29
Fibre (%)	83	4.81	3.57–6.07	−0.18	0.11
Botanical components					
Triticale (kg/T)	76	208.5	100–400	−0.10	0.41
Barley (kg/T)	76	303.4	200–400	−0.05	0.65
Rye (kg/T)	76	100	0–205.5	0.08	0.51
Soybean meal (kg/T)	76	80	27.1–100	0.07	0.56
Wheat bran (kg/T)	76	50	20–75	−0.27	**0.02**
Feed contaminants					
Deoxynilvalenol (mg/kg)	53	0.089	0–0.74	0.15	0.25

Table 3. Descriptive statistics of the nominal predictor variables and their association to the gastric ulcers prevalence in pig herds.

Variable	n_1	n_n	n_1/n_n [a]	χ^2	*p*
Diet form					
Dry diet feeding	Mash	Pelleted	13/77	24.00	**0.001**
Liquid feeding	No	Yes	68/6	14.0	**0.0002**
Botanical components					
Wheat [b]	No	Yes	38/38	7.46	0.19
Sunflower meal [c]	No	Yes	43/33	4.95	0.42
Alfalfa [d]	No	Yes	15/61	0.47	0.49
Maize [e]	No	Yes	67/9	0.07	0.79
Oat [f]	No	Yes	67/9	0.05	0.83
Rapeseed meal [g]	No	Yes	33/43	0.15	0.70
Lupine [h]	No	Yes	68/8	0.31	0.58
Faba bean [i]	No	Yes	69/7	2.36	0.12
Feed additives					
Olive oil [j]	No	Yes	89/1	0.82	0.37
Mycotoxin deactivator [k] (no/0.5/1)	No	0.5/1	28/50/6	14.3	**0.0008**
Prebiotic [l]	No	Yes	76/8	1.10	0.30
Probiotic [m]	No	Yes	80/4	0.24	0.62
Mould inhibitor [n]	No	Yes	76/8	1.88	0.17
Feed contaminants					
Ochratoxin A detected	No	Yes	75/15	0.62	0.43

[a] number of observations first category/number of observation following categories; in bold—the variables significantly ($p < 0.05$) associated with the prevalence of gastric ulcers in pig herds [b]—90, 100 or 200 kg/T. [c]—80 or 110 kg/T. [d]—20 kg/T. [e]—100 kg/T. [f]—30 kg/T. [g]—70, 80 or 100 kg/T. [h]—30 kg/T. [i]—10 or 30 kg/T. [j]—40 kg/T. [k]—Toxi-Tect A (LIKRA Tierernährung GmbH, Austria). [l]—BioAktiv T (Prostafeed B.V., Bodegraven, the Netherlands), 0.2 kg/T. [m]—BioPlus YC (Biochem Zusatzstoffe Handels- und Produktionsgesellschaft mbH, Germany), 0.4 kg/T. [n]—Maxi Mill (Anitox Corp., Northamptonshire, UK), 0.5 kg/T.

A significant ($p < 0.05$) association of the ulcer prevalence was found for a number of variables. Among 11 continuous factors analysed in the study, raising the level of protein

was identified as the only analytical component which significantly ($p = 0.04$) increased ($\rho = 0.22$) the occurrence of gastric ulcers in the herds. Among the botanical components, the addition of wheat bran ($p = 0.02$) and its growing share in feed composition had the contrary effect ($\rho = -0.27$). There was no statistically significant correlation between the other dietary factors divided into four subcategories, i.e., diet form (content of particle size <1 mm), analytical components (fat, feed humidity, fibre), botanical components (triticale, barley, rye, soybean) and feed contaminants (detection of DON).

Among 16 nominal values, both variables representing different diet forms were proven to have a crucial role in gastric ulceration in finishing pigs. Feeding pelleted feed was found to be significantly associated ($p = 0.001$) with the higher prevalence of the disease. The opposite effect had liquid feeding ($p = 0.0002$) and supplementation of a mycotoxin deactivator ($p = 0.0008$). For the other 13 nominal determinants, categorised into three groups, i.e., botanical components (wheat, sunflower, alfalfa, maize, oat, rapeseed, lupine, faba), feed additives (olive oil, probiotics, mould inhibitor) and feed contaminant (detection of OTA), statistically significant correlation was not found.

4. Discussion

All species belonging to the family *Suidae* are highly versatile omnivores and feeding habits of those living in the wild are clearly influenced by a local climate and its seasonality. However, the process of domestication has irreversible eliminated many of the natural components from their diets and in modern times replaced them with feed grains. Domestic pigs are not capable to masticate cereals into elements whose size would guarantee proper contact with digestive enzymes, and thus feed conversion rate and growth performance are desired by modern swine production. The appropriate surface-to-volume ratio of feed particles can only be ensured by a mechanical disintegration of raw materials during the production process. It has been shown that the reduction of the average maize-based feed particle size by every 100, between 1200 and 400 µm, directly translates into 1.3% of the average daily gain (ADG) improvement [21]. On the other hand, excessive fragmentation of feedstuffs is at the top of the list of nutritional factors significantly increasing the risk of the disease [22,23].

From the technological point of view, the pelleting process significantly decreases bulk density of feed, which allows to reduce costs of repeated handling and storage. In this way, pelleted feed is currently the most commonly used in fattening farms. However, multiple reports indicate that feed pelleting is associated with the occurrence of gastric ulcers [19,24–29]. The present study further supported this evidence, and indicated that feeding pigs pelleted feed is significantly correlated with the development of gastric ulcers ($p = 0.001$). There are two factors that could explain it. Before pelleting, the feed is steam-conditioned in order to compress it through the die holes. The process breaks the intermolecular starch bonds, leading to its gelatinization. Additionally, steam-conditioning changes the tertiary structure of proteins. While digestibility of the final product is significantly improved, starch gelatinization increases viscosity of digesta which promotes its prolonged contact with the pars oesophagea and may have detrimental effect on the organ integrity [30,31]. During pelleting process, a secondary milling of feed particles occurs, while the pellets are formed under the combination of temperature and high pressure. Thus, even though the primary grinding may be considered optimal and not posing any risk of ulcer formation, the final production step significantly increases the risk. Unfortunately, the importance of this process for the generation of the excessive amounts of the finest particles is very difficult, to impossible, to quantify. Additionally, it has already been shown that dry feeding ad libitum, as in most of the herds evaluated in our study, may result in a higher ulcer prevalence [19].

The present study showed that the use of liquid feeding based on maize silage (containing 66% water) was associated with significantly lower incidence of gastric ulcers in the herd ($p = 0.0002$). One of the possible explanation for lower aggressiveness of liquid feed is dilution of stomach acidic content. However, the latest scientific data, indicate that

the water content itself does not have any impact on the emergence of the problem [32]. Regardless of the technological details and ingredients used, in most large-scale farms liquid feeding is one of the forms of the most restrictive feeding, that could suggest the opposite impact on ulcer formation than described here. However, using long troughs as in farm T15 synchronizes feed intake among those inhabiting one pen and subsequently eliminates the social stress factors influencing the extension of the time between feeding events in the dominated subpopulation of animals. Thus, it can be speculated that the potentially negative impact of liquid feeding may be limited or eliminated if the feed provision system allows unrestricted access to all animals, limiting the stress.

In the present study, apart from the impact of wheat bran content ($p = 0.02$), none of the analysed botanical ingredients was significantly associated with gastric ulceration in finishers. Before, several experimental studies addressed the gastroprotective effects of some of botanical components in finishers feed. For example, advantage of barley over wheat, as a main cereal component of dry feed, and protective against ulcers, was suggested [15]. Similar conclusions were also drawn for oats in relation to maize [33]. However, the major problem with the aforementioned studies is that the experimental and control fodders were not normalized for the analytical feed composition. Actually, most of the experiments involved gross changes of botanical composition leading to major shifts in the chemical content of the final product. For example, the suggested advantage of barley over wheat [15], may be a result of the differences in chemical composition of the experimental feed, and not of the unexplained gastroprotective properties of barley grain. The fact that the crude fibre level in wheat is only approximately 60% of it in barley could explain the observed differences [34].

It is commonly assumed that fibre provides the layering of the stomach content, which protects against direct and prolonged contact of digesta with the pars oesophagea. In the presented study, the crude fibre content alone did not significantly correlate with disease prevalence ($p = 0.11$). However, a low strength negative correlation ($\rho = -0.27$) between the content of wheat bran ($p = 0.02$) and the prevalence of the disease indicates that parallel to the increase in the level of the discussed material, there is a significant increase in another fraction of total dietary fibre, which might be accountable for its gastroprotective function. Previous research clearly showed the beneficial impact of feed components with a higher (than other tested materials) fibre level [15,33]. It is difficult to compare the results of different studies on the impact of the fibre content on gastric pathology, as different definitions of dietary fibre, depending on the analysis method adopted, were used. In the studies conducted on pigs fed with feed rich in non-starch polysaccharides (NSP) (31.4% of NSP, obtained by replacing a portion of wheat with beet pulp), a significantly lower intensity of ulceration was observed compared to the animals fed with a standard diet (19% of NSP) [35]. Similarly, in the case of crude fibre, it has been shown that increasing its level (even up to 9%) reduces the problem [36,37]. In another study, an attempt to increase the level of crude fibre by adding 2.5% lignocellulose did not have a positive effect [38]. The cited research clearly shows the diversity of biological functions of individual dietary components, collectively referred to as fibre, is not fully understood and requires further studies, also on the impact they may have on ulcer formation.

In our study, the only chemical component significantly associated with the occurrence of ulcers is the level of protein ($p = 0.04$), positively correlated with the prevalence of ulcers (low strength, $\rho = 0.22$). The preceding analyses of the effect of protein on gastric pathology were carried out in the 1960s, only using low-protein diets [39]. Considering the p value obtained in our study, and the fact that different levels of protein of feeds used today have not been tested for the ulcerogenic activity so far, the need of further research clarifying the role of protein in the disease development is justified.

Another feed factor that is thought to contribute to gastric ulceration is mycotoxin contamination, which can result either in significant reduction of feed intake or exacerbation of the negative effects of feed structure on gastric epithelium. The example of DON shows that the feed consumption decreases by 5% for every 1 mg increase in concentration

above the critical level of 0.9 mg/kg of feed [40]. In another experiment, carried out on weaned piglets fed with heavily contaminated feed (3.4–3.6 mg DON/kg) over a period of 5 weeks, no macroscopic lesions in the non-glandular stomach were found. The DON contamination increased the detrimental effect of finely ground feed, expressed by a significantly more pronounced lymphoplasmacytic infiltration both of the non-glandular and glandular stomach mucosa [41]. The same conclusions regarding gastric histopathology (also without accompanying gross lesions) were drawn from the studies on fatteners fed with fodder with an unchanged milling structure and a high DON concentration (5 mg/kg) for 120 consecutive days [42]. None of the DON concentrations indicated as critical were found in the feed fed to fatteners in the present study and the concentrations up to 0.75 mg/kg had no impact on ulcer formation. Moreover, OTA levels were very low on the detection limit. Surprisingly, the supplementation of the mycotoxin deactivator Toxi-Tect A (LIKRA Tierernährung GmbH, Linz, Austria), containing clinoptilolite, bentonite, beta-glucans and algae, significantly correlated (p = 0.0008) with the protection against gastric ulceration. This can indirectly indicate the negative subclinical impact of feed contamination with mycotoxins other than OTA and DON. Regardless of the fact that hundreds of fungal metabolites have been described, the impact on pig health was studied in case of a few of them. Thus, the consequences of feeding finishers with feed contaminated by the overwhelming majority of mycotoxins, particularly in concentrations below feed refusal, are not fully understood, but may have adverse effects on local immune response in stomach and epithelial lesions.

The addition of a mould inhibitor (Maxi Mill, Anitox Corp., Wellingborough, UK), investigated in our study was insignificant for the problem (p = 0.17). Such supplementation could have, in theory, a gastroprotective effect under feed storage conditions leading to fungal growth; however, contamination by metabolites of field fungi seems to be a considerably greater threat. However, supplementation of a mould inhibitor might prove beneficial in farms with poor feedstuffs warehousing, exposed to high humidity.

The direct impact of the supplementation with probiotic bacteria and yeast on the disease has not been the subject of previous studies, however, from the perspective of the latest reports on the role of gastric microbiota [43,44], the focus of future research on the gastroprotective effect of probiotics could prove beneficial. However, the supplementation of probiotic in the feed (BioPlus YC, Biochem Zusatzstoffe Handels- und Produktionsgesellschaft mbH, Lohne, Germany), that contained spore-forming microorganisms, i.e., *Bacillus licheniformis*, *B. subtilis*, did not have a statistically significant effect on the prevalence of gastric ulcers in the present study. The same conclusions apply to prebiotic BioAktiv T (Prostafeed B.V., Bodegraven, The Netherlands).

5. Conclusions

Despite the fact that our study on 27 dietary risk factors of gastric ulcers was conducted on the largest to date group of finishers, only liquid feeding, addition of wheat bran and supplementation of the mycotoxin deactivator, were found to be significantly associated with the lower occurrence of the lesions. Only the feed protein level and pelleting were found to increase the risk. Although, the immediate transition from one feeding system to another in most of the farms is impossible, the addition of materials such as wheat bran and routine supplementation with mycotoxin deactivators may be a practical and relatively low-cost solution limiting stomach-ulcer-related losses in modern finishing farms.

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References

1. Roels, S.; Ducatelle, R.; Broekaert, D. Keratin pattern in hyperkeratotic and ulcerated gastric pars esophagea in pigs. *Res. Vet. Sci.* **1997**, *62*, 165–169. [CrossRef]
2. Melnichouk, S. Mortality associated with gastric ulceration in swine. *Can. Vet. J.* **2002**, *43*, 223–225.
3. Makinde, M.; Gous, T. Prevalence of gastro-oesophageal ulcers in grower-finisher pigs in the Northern Province of South Africa: Research communication. *J. S. Afr. Vet.* **1998**, *69*, 59–60. [CrossRef] [PubMed]
4. Marchini, C.F.P.; Martins, P.M.; Rabelo, R.N. Prevalence of gastric lesions in pigs. *Investigacao* **2017**, *16*, 50–55. [CrossRef]
5. Proietti, P.C.; Bietta, A.; Brachelente, C.; Lepri, E.; Davidson, I.; Franciosini, P.F. Detection of Helicobacter spp. In gastric, fecal and saliva samples from swine affected by gastric ulceration. *J. Vet. Sci.* **2010**, *11*, 221–225. [CrossRef]
6. Rutheford, K.M.D.; Thompson, C.S.; Thomson, J.R.; Lawrence, A.B.; Nielsen, O.N.; Busch, E.M.; Haugegaard, S.; Sandøe, P. A study of associations between gastric ulcers and the behaviour of finisher pigs. *Livest. Sci.* **2018**, *212*, 45–51. [CrossRef]
7. Swaby, H.; Gregory, N.G. A note on the frequency of gastric ulcers detected during post-mortem examination at a pig abattoir. *Meat Sci.* **2012**, *90*, 269–271. [CrossRef] [PubMed]
8. Gaafar, S.M.; Keittevuti, B. Experimental induction of esophagogastric ulcers without ioculations of Ascaris suum eggs in swine. *Gastroenterology* **1972**, *63*, 423–426. [CrossRef]
9. Kokue, E.; Nakamura, T.; Hayama, T. Experimental production of porcine gastroesophageal ulcers by betazole and reserpine. *J. Vet. Pharmacol. Therap.* **1978**, *1*, 217–224. [CrossRef]
10. Mall, A.; Fourie, J.; McLeod, H.; Muschol, A.; Campbell, J.A.H.; Hickman, R. Administration of sucralfate prolongs survival of animals with experimental peptic ulceration. *Am. J. Med.* **1991**, *91*, 37–42. [CrossRef]
11. Nafstad, I.; Tollersrud, S.; Baustad, B. Gastric ulcers in swine: 3. Effects of different proteins and fats on their development. *Path Vet.* **1967**, *4*, 23–30. [CrossRef]
12. Riker, J.T.; Perry, T.W.; Pickett, R.A.; Heidenreich, C.J.; Curtin, T.M. Influence of controlled ambient temperatures and diets on the incidence of esophagogastric ulcers in swine. *J. Anim. Sci.* **1967**, *26*, 736–740. [CrossRef] [PubMed]
13. Wondra, K.J.; Hancock, J.D.; Behnke, K.C.; Hines, R.H.; Stark, C.R. Effects of particle size and pelleting on growth performance, nutrient digestability, and stomach morphology in finishing pigs. *J. Anim. Sci.* **1995**, *73*, 757–763. [CrossRef] [PubMed]
14. Jakubowski, K.; Flis, M.; Sobotka, W. Stan zdrowotny żołądka świń żywionych mieszankami z udziałem rożnie rozdrobnionego ziarna jęczmienia i pszenżyta. *Med. Wet.* **2002**, *58*, 201–204.
15. Nielsen, E.K.; Ingvartsen, K.L. Effect of cereal type, disintegration method and pelleting on stomach content, weight and ulcers and performance in growing pigs. *Livest. Prod. Sci.* **2000**, *66*, 271–282. [CrossRef]
16. Paulk, C.; Hancock, J.D.; Fahrenholz, A.; Wilson, J.; McKinney, L.J.; Benhke, K.C.; Nietfeld, J.C. Effects of feeding cracked corn to nursery and finishing pigs. *J. Anim. Sci.* **2015**, *93*, 1710–1720. [CrossRef]
17. Reese, N.A.; Muggenburg, B.A.; Kowalczyk, T.; Hoekstea, W.G.; Grummer, R.H. Effects of Corn, Wheat, Oats and Alfalfa Leaf Meal on the Development of Gastric Ulcers in Swine. *J. Anim. Sci.* **1966**, *25*, 21–24. [CrossRef]
18. Gottardo, F.; Scollo, A.; Contiero, B.; Bottacini, M.; Mazzoni, C.; Edwards, S.A. Prevalence and risk factors for gastric ulceration in pigs slaughtered at 170kg. *Animal* **2017**, *11*, 2010–2018. [CrossRef]
19. Robertson, I.D.; Accioly, J.M.; Moore, K.M.; Driesen, S.J.; Pethick, D.W.; Hampson, D.J. Risk factors for gastric ulcers in Australian pigs at slaughter. *Prev. Vet. Med.* **2002**, *53*, 293–303. [CrossRef]
20. Kopinski, J.S.; McKenzie, R.A. Oesophagogastric ulceration in pigs: A visual morphological scoring guide. *Aust. Vet. J.* **2007**, *85*, 356–361. [CrossRef] [PubMed]
21. Hancock, J.D. The role of feed and feed processing in development of gastric ulcers. *Leman Swine Conf.* **2000**, *1*, 98–99.
22. Deen, J. Epidemiology of gastroesophageal ulcers. *Leman Swine Conf.* **2000**, *1*, 96–97.
23. Mahan, D.C.; Pickett, R.A.; Perry, T.W.; Curtin, T.M.; Featherson, W.R.; Beeson, W.M. Influence of various nutritional factors and physical form of feed on esophagogastric ulcers in swine. *J. Anim. Sci.* **1966**, *25*, 1019–1023. [CrossRef] [PubMed]
24. Amornthewaphat, N.; Hancock, J.D.; Behnke, K.C.; Hines, R.H.; Kennedy, G.A.; Cao, H.; Park, J.S.; Maloney, C.S.; Dean, D.W.; Derouchey, J.M.; et al. Effects of feeder design and pellet quality on growth performance, nutrient digestibility, carcass characteristics, and water usage in finishing pigs. *J. Anim. Sci.* **1999**, *77*, 55.
25. Flatlandsmo, K.; Slagsvold, P. Effect of grain particle size and pellets on development of gastric ulcers in swine. *J. Anim. Sci.* **1971**, *33*, 1263–1265. [CrossRef]
26. Gamble, C.T.; Chamberlain, C.C.; Merriman, G.M.; Lidvall, E.R. Effects of pelleting, pasture and selected diet ingredients on the incidence of esophagogastric ulcers in swine. *J. Anim. Sci.* **1967**, *26*, 1054–1058. [CrossRef]

27. Goodband, B.; Tokach, M.; DeRouchey, J.; Patience, J.; Dritz, S.; Woodworth, J. Latest field research on feed efficiency. *Proc. AASV* **2016**, *1*, 11–13.
28. Pocock, E.F.; Bayley, H.S.; Roe, C.K.; Slinger, S.J. Dietary factors affecting the development of esophagogastric ulcers in swine. *J. Anim. Sci.* **1969**, *29*, 591–597. [CrossRef]
29. Potkins, Z.V.; Lawrence, T.L.; Thomlinson, J.R. Oesophagogastric parakeratosis in the growing pig: Effects of the physical form of barley-based diets and added fibre. *Res. Vet. Sci.* **1989**, *47*, 60–67. [CrossRef]
30. Maxson, D.W.; Stanley, G.R.; Perry, T.W.; Pickett, R.A.; Curtin, T.M. Influence of various rations of raw and gelatinized corn, oats, oat components and sand on the incidence of esophagogastric lesions in swine. *J. Anim. Sci.* **1968**, *2*, 1006–1010. [CrossRef]
31. Nuwer, A.J.; Perry, W.; Pickett, A.; Curtin, T.M. Expanded of heat-processed fractions of corn and their relative ability to elicit esophagogastric ucers in swine. *J. Anim. Sci.* **1967**, *26*, 518–525. [CrossRef]
32. Mösseler, A.; Köttendorf, S.; Grosse Liesner, V.; Kamphues, J. Impact of diets' physical form (particle size; meal/pelleted) on the stomach content (dry matter content, pH, chloride concentration) of pigs. *Livest. Sci.* **2010**, *134*, 146–148. [CrossRef]
33. Maxwell, C.V.; Reese, N.A.; Muggenburg, B.A.; Reimann, E.M.; Kowalczyk, T.; Grummer, R.H.; Hoekstra, W.G. Effect of oat hulls and other oat fractions on the development of gastric ulcers in swine. *J. Anim. Sci.* **1967**, *26*, 1312–1318. [CrossRef] [PubMed]
34. Knudsen, K.E.B. Carbohydrate and lignin contents of plant materials used in animal feeding. *Anim. Feed Sci.* **1997**, *67*, 319–338. [CrossRef]
35. Laitat, M.; Antoine, N.; Cabaraux, J.F.; Cassart, D.; Mainil, J.; Moula, N.; Nicks, B.; Wavreille, J.; Philippe, F.X. Influence of sugar beet pulp on feeding behavior, growth performance, carcass quality and gut health of fattening pigs. *Biotechnol. Agron. Soc. Environ.* **2015**, *19*, 20–31.
36. Millet, S.; Kumar, S.; De Boever, J.; Meyns, T.; Aluwe, M.; De Brabander, D.; Ducatelle, R. Effect of particle size distribution and dietary crude fibre content on growth performance and gastric mucosa integrity of growing-finishing pigs. *Vet. J.* **2012**, *192*, 316–321. [CrossRef] [PubMed]
37. Millet, S.; Meyns, T.; Aluwe, M.; De Brabander, D.; Ducatelle, R. Effect of grinding intensity and crude fibre content of the feed on growth performance and gastric mucosa integrity of growing-finishing pigs. *Livest. Sci.* **2010**, *134*, 152–154. [CrossRef]
38. Grosse Liesner, V.; Taube, V.; Leonhard-Marek, S.; Bieneke, A.; Kamphues, J. Integrity of gastric mucosa in reared piglets—Effects of physical form of diets (meal/pellets), pre-processing grinding (coarse/fine) and addition of lignocellulose (0/2,5%). *J. Anim. Physol. Anim. Nutr.* **2009**, *93*, 373–380. [CrossRef]
39. Nafstad, I. Gastric ulcers in swine: 1. Effect of dietary protein, dietary fat and vitamin E on ulcer development. *Path Vet.* **1967**, *4*, 1–14. [CrossRef]
40. Döll, S.; Dänicke, S. The Fusarium toxins deoxynivalenol (DON) and zearalenone (ZON) in animal feeding. *Prev. Vet. Med.* **2011**, *102*, 132–145. [CrossRef]
41. Dänicke, S.; Beineke, A.; Berk, A.; Kersten, S. Deoxynilvalenol (DON) contamination of feed and grinding finess: Are there interactive implications of stomach integrity and health of piglets? *Toxins* **2017**, *9*, 16. [CrossRef] [PubMed]
42. Madson, D.M.; Ensley, S.M.; Patience, J.F.; Gauger, P.C.; Main, R.M. Diagnostic assessment and lesion evaluation of chronic deoxynilvalenol ingestion in growing swine. *J. Swine Health Prod.* **2014**, *22*, 78–83.
43. Almeida, L.R.; Costa, P.S.; Nascimento, A.M.A.; Reis, A.C.P.; Barros, K.O.; Alvim, L.B.; Nunes, A.C.; Queiroz, D.M.M.; Rocha, G.A.; Nicoli, J.R.; et al. Porcine stomachs with and without castric ulcer differ in Lactobacillus load and strain characteristics. *Can. J. Microbiol.* **2018**, *64*, 493–499. [CrossRef] [PubMed]
44. De Witte, C.; Demeyere, K.; De Bruyckere, S.; Taminiau, B.; Daube, G.; Ducatelle, R.; Meyer, E.; Haesebrouck, F. Charecterization of the non-glandular gastric region microbiota in Helicobacter suis-infected versus non-infected pigs identifies a potential role for Fusobacterium gastrosuis in gastric ulceration. *Vet. Res.* **2019**, *50*, 1–18. [CrossRef]