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Gastric Ulcers in Finishing Pigs at Slaughter in Western Santa Catarina, Brazil: Frequency and Severity

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ABSTRACT

***Background*:** Gastric ulceration of the *pars oesophagea* is a multifactorial disorder of intensively raised pigs that may impair welfare and performance and cause gastrointestinal bleeding or sudden death. Reported prevalence varies among production systems, and lesions may remain subclinical until slaughter. Slaughterhouse inspection therefore offers an opportunity to estimate lesion burden and identify factors that merit on-farm investigation. This study evaluated the frequency and severity of gastric lesions in finishing pigs at slaughter in western Santa Catarina, Brazil.

***Materials, Methods & Results*:** A cross-sectional observational study was conducted between February and May 2020 at a federally inspected abattoir. Pigs originated from 8 commercial farms in the same integrated production system. The farms housed 7,293 finishing pigs; 3,798 were followed at slaughter, and 380 stomachs, approximately 10% of each slaughter batch, were selected systematically. Each stomach was opened along the greater curvature, emptied, washed and examined macroscopically. Lesions in the *pars oesophagea* were classified from grade 0 to grade 4: normal mucosa (grade 0), parakeratosis (grade 1), parakeratosis with ulceration affecting < 33% (grade 2), ulceration affecting 34-66% (grade 3), or ulceration affecting 67-100% of the mucosal surface (grade 4). For analysis, grades 1 and 2 were considered mild and grades 3 and 4 severe. Production records included flooring type, mortality, disease occurrence and treatment, and environmental enrichment. Absolute and relative frequencies were calculated, and univariate logistic regression was used to test associations between lesion severity and flooring or enrichment at a significance level of 5%. Gastric lesions were detected in 376/380 stomachs (98.9%); 4/380 (1.1%) had no lesions. Grade 1 was most frequent (178/380; 46.8%), followed by grade 2 (155/380; 40.8%), grade 3 (30/380; 7.9%) and grade 4 (13/380; 3.4%). Mild lesions represented 87.6% and severe lesions 11.3% of examined stomachs. Lesion distributions were similar across farms. Grade 0 ranged from 0 to 7.7% by farm, grade 1 from 35.9 to 53.1%, grade 2 from 33.3 to 57.1%, grade 3 from 4.0 to 12.0%, and grade 4 from 0 to 9.5%. Neither flooring type nor environmental enrichment was significantly associated with lesion severity ($P > 0.05$). During finishing, 163 pigs died, corresponding to a mean mortality of 2.23% across farms; respiratory disease accounted for 66 deaths and gastric ulcers for 27. Six farms had no environmental enrichment, 1 had partial enrichment, and 1 had enrichment in all pens. Six farms used solid flooring with a water layer, whereas 2 used partially slatted flooring.

***Discussion*:** The predominance of mild lesions and the absence of associations with the evaluated housing variables suggested that unmeasured factors, including feeding practices and pre-slaughter management, may have contributed to lesion occurrence. Because the farms shared 1 integrated production system, limited variation in management may also have reduced the ability to detect associations. The high prevalence confirmed that clinically silent gastric lesions were common in finishing pigs. Routine slaughterhouse monitoring, particularly of severe lesions, was therefore considered a useful tool for herd-level surveillance of gastrointestinal health and animal welfare and for guiding targeted investigation of nutritional, health and management risk factors.

Keywords: gastric ulceration, *pars oesophagea*, slaughterhouse monitoring, animal welfare, prevalence, swine.

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INTRODUCTION

Gastric ulceration of the non-glandular *pars oesophagea* is common in intensively raised pigs, with reported prevalence ranging from 20.7% [16] to 79% [32]. Unlike the glandular mucosa, this region lacks mucus-secreting cells and is therefore vulnerable to exposure to acidic gastric contents [17]. Lesions may progress from parakeratosis to ulceration and cause gastrointestinal bleeding, anaemia, impaired growth, oesophageal stenosis or sudden death, although extensively affected animals can remain asymptomatic [2,15,18,33].

Predisposing factors include genetics, feed particle size and physical form, fasting, housing conditions, stress and concurrent disease [7,8,11,18]. Factors that increase the fluidity of gastric contents can disrupt the gastric pH gradient and expose the *pars oesophagea* to acid [11,22]. Because lesions can be clinically silent and may regress over time, inspection of stomachs at slaughter provides a practical means of estimating their frequency and severity [20,31].

The present study aimed to evaluate the frequency and severity of gastric ulcers in finishing pigs slaughtered in western Santa Catarina, Brazil.

MATERIALS AND METHODS

Study design and origin of the animals

A cross-sectional observational study was conducted to evaluate the frequency and severity of gastric lesions in finishing pigs at slaughter. The evaluations were carried out in an abattoir operating under Federal Inspection Service (SIF), located in the western region of the state of Santa Catarina, Brazil, between February and May 2020. This study used material obtained from routine slaughter procedures, with no experimental manipulation of the animals.

The animals evaluated originated from 8 commercial pig farms integrated within the same production system. During the study period, these farms totalled 7,293 pigs housed during the finishing phase. The slaughtered animals consisted of immunocastrated males and non-castrated females with age and body weight compatible with the final stage of production.

Sampling

A total of 3,798 pigs from the 8 evaluated farms were followed at slaughter. For the analysis of gastric lesions, systematic sampling corresponding to approximately 10% of the animals slaughtered per batch was performed, resulting in a total of 380 stomachs evaluated. Stomachs were selected randomly and systematically along the slaughter line before the organs were opened.

Macroscopic evaluation of the stomachs

The stomachs were evaluated in the upper offal processing area of the abattoir after separation from the intestinal tract. Each stomach was opened along the greater curvature, emptied and washed with running water to remove the gastric contents.

A macroscopic inspection of the *pars oesophagea*, also referred to as the oesophageal quadrangle, was then performed in order to identify lesions compatible with gastric ulceration.

Lesions were classified using a previously described scoring system [31], which considers 5 degrees of lesion severity: Grade 0 (normal stomach, oesophagogastric mucosa with smooth, shiny epithelium and no visible alterations); Grade 1 (parakeratosis, oesophagogastric mucosa with proliferative, rough and dull epithelium, possibly presenting small erosions); Grade 2 (parakeratosis with ulceration affecting less than 33% of the *pars oesophagea*); Grade 3 (parakeratosis with ulceration affecting between 34% and 66% of the mucosal surface); Grade 4 (parakeratosis with ulceration affecting between 67% and 100% of the mucosal surface).

Collection of management information

In addition to the evaluation of gastric lesions, information related to the production history and housing conditions of the evaluated farms was collected. For each batch, the following variables were recorded: type of flooring in the facilities, mortality history, occurrence of diseases and treatments performed in the batch, and the presence of environmental enrichment in the pens.

Data analysis

The collected data were organised and initially analysed using descriptive statistics, considering absolute and relative frequencies of the different lesion grades. To explore possible associations between management variables and lesion severity, the data were grouped into 2 outcomes: presence of mild lesions (Grades 1 & 2) and presence of severe lesions (Grades 3 & 4).

Univariate logistic regression was performed to evaluate the association between lesion severity and the variables flooring type and environmental enrichment. Prevalence ratios (PR) were calculated using the online statistical tool VassarStats[1]. The level of statistical significance adopted was 5% ($P < 0.05$).

RESULTS

Eight commercial pig farms were evaluated, totalling 7,293 pigs housed during the study period. Of these, 3,798 animals were monitored at slaughter, corresponding to approximately 52% of the total pigs housed. From these farms, 380 stomachs were collected and evaluated, representing approximately 10% of the animals slaughtered (Table 1).

The macroscopic evaluation of the *pars oesophagea* revealed a high frequency of gastric lesions. Of the total stomachs evaluated, 98.9% (376/380) presented some degree of alteration compatible with gastric ulceration, whereas only 1.1% (4/380) showed no lesions.

Among the observed lesion grades, mild lesions were predominant, representing 87.6% of the cases. Grade 1 lesions were the most frequent, accounting for 46.8% (178/380) of the evaluated stomachs, followed by Grade 2 lesions with 40.8% (155/380). Lesions classified as severe were less frequent, being observed in 11.3% of the stomachs, with 7.9% (30/380) classified as Grade 3 and 3.4% (13/380) as Grade 4 (Figures 1 & 2).

When analysed by farm (Figure 3), the frequency of lesions was similar among the 8 evaluated farms. Only farms 2 and 3 presented stomachs without lesions in the oesophageal region (*pars oesophagea*). Furthermore, only farms 2, 4 and 7 showed no animals presenting Grade 4 lesions.

The frequency of stomachs without lesions (Grade 0) ranged from 0% to 7.7% among the evaluated farms. Grade 1 lesions ranged from 35.9% to 53.1%, whereas Grade 2 lesions varied between 33.3% and 57.1%. The occurrence of Grade 3 lesions ranged from 4.0% to 12.0% across the farms. The most severe lesions (Grade 4) varied from 0% to 9.5% among the evaluated farms.

The highest frequency of stomachs without lesions (Grade 0) was observed in farm 2, representing 7.7% of the stomachs evaluated in that farm. Grades 1 and 2 lesions were most frequently observed in farms 4 (53.1%) and 5 (57.1%), respectively. Moreover, farm 1 showed the highest proportion of Grade 3 lesions (12.0%), whereas farm 6 presented the highest frequency of Grade 4 lesions (9.5%).

Statistical analysis did not demonstrate a significant association between gastric lesion grades and the evaluated environmental variables, including flooring type in the facilities and the presence of environmental enrichment.

Regarding the sanitary history of the farms, complete information was not available for farms 1, 2 and 5. In farms 3, 4 and 6, animals showing respiratory clinical signs during the finishing phase were reported. In farm 3, an episode of diarrhoea with the presence of blood was also reported. In farms 7 and 8, no episodes of clinical disease were reported during the evaluated period. In farms where diseases occurred, treatments were administered collectively through the drinking water according to the protocol established by the integrating company.

During the finishing period, a total mortality of 163 animals was recorded, corresponding to an average mortality rate of 2.23% among the evaluated farms. Mortality ranged from 0.63% to 5.34% among the farms. Farm 1 presented the highest mortality rate (5.34%), whereas farm 8 presented the lowest mortality rate (0.63%).

The main recorded causes of mortality were respiratory diseases, responsible for 66 deaths, followed by gastric ulcers (27 cases), runting (16 cases) and sudden death (10 cases).

Regarding housing characteristics, 75% of the farms had solid flooring with a water layer system, whereas 25% had a combination of solid flooring in the front part of the pen and slatted flooring in the rear section. Concerning environmental enrichment, only 1 farm had enrichment in all pens, 1 farm had partial enrichment, and 6 farms had no environmental enrichment in their facilities.

DISCUSSION

Gastric ulceration is a condition that affects pigs worldwide, and several studies have investigated its prevalence [16,18,32]. In the present study, a high frequency of gastric lesions was observed in pigs evaluated at slaughter, with 98.9% of the stomachs presenting some degree of alteration, predominantly mild lesions (Grades 1 & 2). These findings reinforce the high occurrence of alterations in the oesophagogastric mucosa in intensive pig production systems.

Table 1. Number of pigs housed, slaughtered and stomachs evaluated, distribution of gastric lesion grades in the *pars oesophagea*, and mortality rate in pigs originating from eight commercial farms.

Farms	Total housed	No. animals slaughtered	No. stomachs evaluated	No lesion	Grade 1	Grade 2	Grade 3	Grade 4	Mortality (%)
1	1042	496	50	0	22	20	6	2	5.34
2	400	391	39	3	14	18	4	0	2.24
3	850	415	41	1	17	17	3	3	2.47
4	497	486	49	0	26	21	2	0	1.2
5	380	211	21	0	6	12	2	1	1.36
6	2000	628	63	0	31	21	5	6	1.5
7	1024	722	72	0	39	28	5	0	2.44
8	1100	449	45	0	23	18	3	1	0.63
Total	7293	3798	380	4	178	155	30	13	
%	100%	52%	10%	1%	47%	41%	8%	3%	2.23%

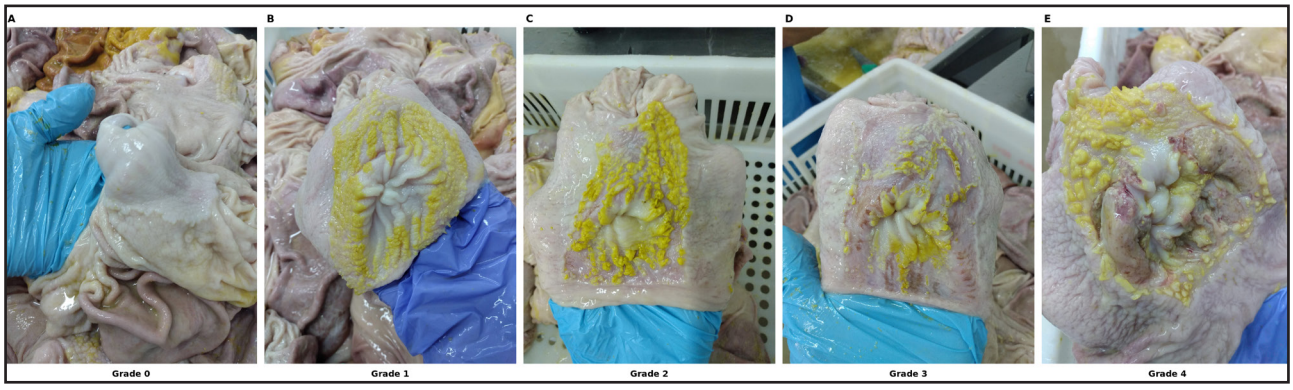

Figure 1. Macroscopic appearance of gastric lesions in the *pars oesophagea* of pigs at slaughter, illustrating the different lesion grades according to the classification system used in this study: A- Grade 0: normal mucosa without lesions; B- Grade 1: parakeratosis with thickened and rough mucosa; C- Grade 2: parakeratosis with ulceration affecting less than 33% of the mucosal surface; D- Grade 3: ulcerative lesion affecting between 34% and 66% of the mucosal surface; and E- Grade 4: extensive ulceration affecting more than 67% of the mucosal surface.

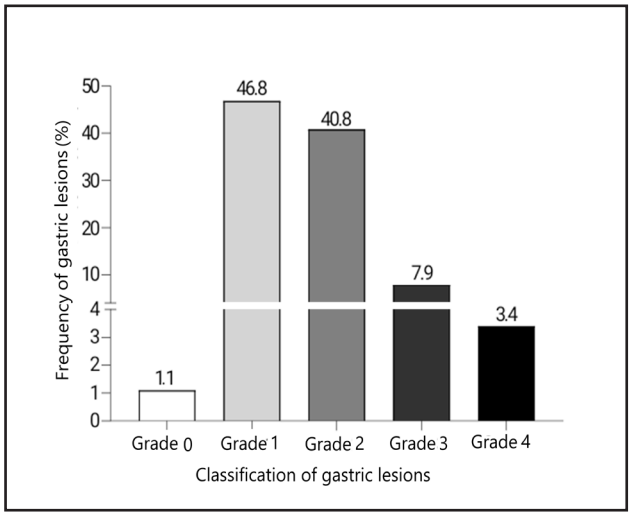

Figure 2. Occurrence of gastric ulcers and their classification according to severity in 380 evaluated stomachs.

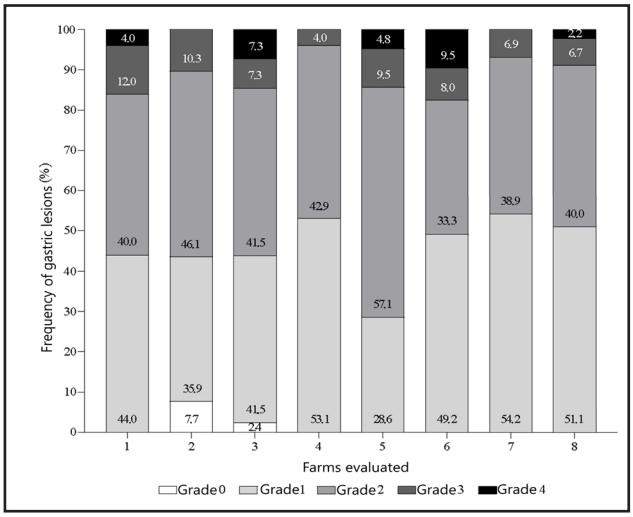

Figure 3. Percentage distribution of gastric ulcer grades observed in each evaluated farm.

Several studies report variable prevalences of gastric lesions in pigs at slaughter. An evaluation of 15,741 stomachs in Australian abattoirs found that approximately 17% of the animals presented severe ulcerative lesions, whereas around 20% showed no alterations [29]. A study of slaughter pigs in Nigeria reported a prevalence of 32.9% of lesions in the *pars oesophagea* [26]. In the United Kingdom, mild lesions were detected in 73% of the pigs, while the prevalence of severe lesions ranged from 0.8% to 19% [32]. Similarly, studies conducted in Brazil have also demonstrated a high frequency of alterations in the gastric mucosa. An evaluation of more than 20,000 stomachs in the state of Rio Grande do Sul found that 89.7% presented some degree of lesion, with Grade 1 lesions being the most frequent [25].

A study of 400 stomachs from pigs slaughtered in the state of São Paulo, at approximately 120 days of age and an average weight of 105 kg, found a high frequency of gastric lesions [21]. In that study, 98.75% of the stomachs presented some degree of lesion in the *pars oesophagea*, with Grade 3 lesions being the most frequent (43.25%), whereas parakeratosis (Grade 1) was observed in only 16.5% of the animals. Similar results were obtained in an evaluation of 250 stomachs from pigs slaughtered in the state of Paraná, aged between 135 and 150 days and weighing between 90 and 110 kg [3]. In that study, the overall prevalence of gastric lesions was 64.4%, with Grade 3 lesions again being the most frequent (27.2%). These findings reinforce that alterations in the mucosa of the *pars oesophagea* are common in finishing pigs, although the distribution of lesion grades may vary among different production systems.

Among the factors described in the literature as predisposing to the development of gastric ulcers in pigs, nutritional aspects such as fine feed particle size and physical form of the diet are frequently highlighted, in addition to pre-slaughter management factors including prolonged fasting periods [9,19,24]. Fasting periods between 14 and 23 h may increase the occurrence of parakeratosis in the gastric mucosa [10]. A higher frequency of lesions in the *pars oesophagea* has also been reported in pigs fed diets containing finer particles, highlighting the role of feed particle size in the pathogenesis of gastric lesions [19]. Parakeratotic lesions were observed in approximately 20% of stomachs evaluated during the nursery phase [25]. These lesions occurring during the nursery phase may later result in chronic ulcerative lesions or even oesophageal stenosis during the finishing phase.

In the present study, nutritional variables were not evaluated, which limits the identification of possible dietary factors that could explain the high frequency of lesions observed. However, considering that the evaluated farms belonged to the same integrated production system, it is likely that feeding practices, diet formulation and nutritional management were relatively similar among the evaluated farms.

In this study, none of the analysed variables were statistically associated with gastric ulceration. This may be related to the high similarity among the farms belonging to the same integrating company, which results in very similar management practices. Nevertheless, several authors highlight that environmental, nutritional and management factors may favour the development of gastric ulcer lesions [7,8].

An evaluation of 50 animals from 16 farms investigated the association between environmental factors in the housing facilities and the occurrence of gastric ulcers [1]. Among the 21 variables evaluated, flooring type was considered the most important factor. In slatted floor systems, the frequency of severe gastric ulcers reached up to 60%, compared with 35% in pigs housed on solid floors. In contrast, in straw-based systems the frequency varied between 0% and 4%. Further studies of flooring type reported consistent results, with solid floors being less associated with gastric ulcer development than slatted flooring [8,16]. A proposed explanation is that some housing systems may increase the occurrence of respiratory disease, resulting in reduced feed intake and consequently increasing the risk of gastric ulceration. In the present study, the majority of the farms used solid concrete flooring with a water layer system, and only 25% of the farms had partially slatted floors.

Behavioural patterns indicative of stress, such as exploratory behaviours directed towards pen mates (for example belly-nosing, ear biting and tail manipulation), as well as spending more time sitting or inactive, have been positively associated with keratinisation in the *pars oesophagea* region [14,16]. Environmental enrichment using chains or tyres may reduce some of these behavioural patterns while allowing pigs to express exploratory behaviour [27].

Several studies have compared enriched environments that promote reduced stress with stan-

dard production environments. One study evaluated stomachs of pigs housed in 2 different environments: an enriched environment with straw bedding and a conventional environment with slatted flooring and no enrichment [28]. Lesions were classified using a different method from that used in the present study, consisting of 7 grades. In slaughter monitoring, 17.5% of animals from the conventional environment presented severe ulcerative lesions, whereas none of the animals housed in enriched environments showed severe lesions. Environmental enrichment using wood or suspended chains has also shown a protective effect against gastric lesions [16].

When analysing the mortality history of the evaluated farms in the present study, the main causes of death were pneumonia and gastric ulcers, accounting for 66 and 27 deaths (41% and 16.8%), respectively. These findings are consistent with evidence that gastric ulcers contribute significantly to mortality in finishing pigs [23].

Respiratory diseases occurring throughout the productive life of the farms may also act as predisposing factors for the development of gastric ulcers in pigs. This effect may be related mainly to reduced feed intake commonly observed during respiratory disease episodes, which increases the fluidity of gastric contents and favours the exposure of the *pars oesophagea* mucosa to gastric acid [11]. Additionally, infectious processes may increase histamine release, stimulating hydrochloric acid secretion and contributing to the development of lesions in the gastric mucosa [33].

A possible association between porcine circovirus type 2 (PCV2) infection and gastric ulceration was investigated by evaluating 24 stomachs from pigs aged 3 to 5 months, originating from different farms with a history of PCV2 infection and sudden death associated with gastric ulcers [6]. Histological evaluation identified areas of necrosis in gastric glands in the cardia and pyloric antrum regions, as well as large amounts of viral particles within cells and in cellular debris associated with necrotic areas. Based on these findings, it was hypothesised that PCV2 may affect mucus-producing cells, compromise the gastric buffering system and reduce gastric pH, thereby contributing to the development of ulcerative lesions.

An association between infection with *Actinobacillus pleuropneumoniae* serotype 6 and an increased risk of severe gastric ulcers has been observed [5]. In addition, anorexia resulting from infection with Influenza A virus has been associated with an increased predisposition to gastric ulceration in pigs [12].

Pigs affected by gastric ulcers may exhibit behavioural and postural alterations compared with healthy animals [30]. Affected animals tend to spend more time standing, show increased restlessness and, when lying down, spend less time resting on the left side. These behavioural patterns may represent attempts to prevent the accumulation of gastric contents in the cranial portion of the stomach, as well as attempts to alleviate discomfort and pain caused by ulcerative lesions.

In addition to negatively affecting animal welfare, gastric ulcers may also impair productive performance in pigs [13]. An evaluation of 80 pigs during the growing phase found reduced average daily gain in animals affected by moderate and severe gastric ulcers [4]. No significant difference in mean body weight was detected between animals with moderate and severe lesions. However, pigs that died during the experiment as a result of gastric ulcers and oesophageal stenosis showed poorer growth performance compared with animals presenting only mild lesions.

These findings reinforce that gastric ulcers remain an important subclinical problem in pig production, frequently silent, but capable of negatively affecting both animal welfare and productive performance even before the appearance of evident clinical signs.

CONCLUSION

The present study demonstrated a high frequency of pigs affected by gastric ulcers in the western region of Santa Catarina, Brazil, with mild lesions being the most commonly observed. The environmental and management factors evaluated in this study were not associated with the presence of lesions. Gastric ulcer lesions, particularly those of lower severity, may be related to other factors not assessed in the present study, possibly including management practices during the final stages of the finishing phase and the pre-slaughter period.

Slaughterhouse monitoring proved to be an effective tool for the detection of gastric ulcers. For the practical application of these data in the field, such monitoring may serve as an indicator of animal welfare in pig farms and as a tool for identifying predisposing factors. In this context, evaluating the frequency of severe lesions may be sufficient, as these are more closely associated with chronic stress.

MANUFACTURER

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***Declaration of interest*.** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

***Artificial intelligence statement*.** The authors declare that no artificial intelligence (AI) tools were used to generate, analyze, or interpret the data or to produce the scientific content of this manuscript. The authors are solely responsible for all aspects of the work.

REFERENCES

1 **Amory J.R., Mackenzie A.M. & Pearce G.P. 2006.** Factors in the housing environment of finisher pigs associated with the development of gastric ulcers. *Veterinary Record*. 158(8): 260-264. DOI: 10.1136/vr.158.8.260.

2 **Anderson L.C., Mitchell J., Ringler D.H. & Bohr D.F. 1981.** Cimetidine prophylaxis for gastric ulcers in laboratory swine. *Laboratory Animal Science*. 31(5 Pt 1): 498-501.

3 **Araújo Jr. G.V., Begotti I.L., Moraes F.F., Merlini N.B. & Merlini L.S. 2012.** Lesões gástricas em suínos e sua relação com a presença de *Helicobacter* spp. *Arquivos de Ciências Veterinárias e Zoologia da UNIPAR*. 15(2): 99-102.

4 **Ayles H.L., Friendship R.M., Ball R.O. & Barber S.M. 1996.** Effect of dietary particle size on gastric ulcers, assessed by endoscopic examination, and relationship between ulcer severity and growth performance of individually fed pigs. *Swine Health and Production*. 4(5): 211-216.

5 **Busch M.E. & Nielsen E.O. 2016.** Herd level risk factors for stomach ulcers in finishing pigs. In: *Proceedings of the 24th International Pig Veterinary Society Congress* (Dublin, Ireland). p.143.

6 **Corrêa A.M.R., Zlotowski P., Barcellos D.E.S.N., Cruz C.E.F. & Driemeier D. 2008.** Gastric ulcers in pigs affected with postweaning multisystemic wasting syndrome. *Pesquisa Veterinária Brasileira*. 28(12): 601-605. DOI: 10.1590/S0100-736X2008001200007.

7 **Cybulski P., Larska M., Woźniak A., Jabłoński A. & Stadejek T. 2021.** The dietary risk factors of gastric ulcers in finishing pigs from 16 Polish farms. *Agriculture*. 11(8): 719. DOI: 10.3390/agriculture11080719.

8 **Cybulski P., Woźniak A., Larska M., Jabłoński A. & Stadejek T. 2024.** Gastric ulcers in finishing pigs: the evaluation of selected non-dietary risk factors and impact on production performance. *Porcine Health Management*. 10(1): 11. DOI: 10.1186/s40813-024-00362-0.

9 **Dalla Costa O.A., Coldebella A., Costa M.J.R.P., Faucitano L., Peloso J.V., Ludke J.V. & Scheuermann G.N. 2006.** Período de descanso dos suínos no frigorífico e seu impacto na perda de peso corporal e em características do estômago. *Ciência Rural*. 36(5): 1582-1588. DOI: 10.1590/S0103-84782006000500038.

10 **Dalla Costa O.A., Costa M.J.R.P., Ludke J.V., Coldebella A., Kich J.D., Peloso J.V., Faucitano L. & Dalla Roza D. 2008.** Tempo de jejum dos suínos no manejo pré-abate sobre a perda de peso corporal, o peso do conteúdo estomacal e a incidência de úlcera esofágica-gástrica. *Ciência Rural*. 38(1): 199-205. DOI: 10.1590/S0103-84782008000100032.

11 **De Witte C., Ducatelle R. & Haesebrouck F. 2018.** The role of infectious agents in the development of porcine gastric ulceration. *The Veterinary Journal*. 236: 56-61. DOI: 10.1016/j.tvjl.2018.04.015.

12 **Detmer S.E. 2016.** The clinical features, pathobiology, and epidemiology of influenza infections in pigs. In: Swayne D.E. (Ed). *Animal Influenza*. 2nd edn. Hoboken: Wiley-Blackwell, pp.435-451. DOI: 10.1002/9781118924341.ch16.

13 **Dunlop H., Timmer J. & McOrist S. 2021.** Impact of gastric ulceration on weight gain in finisher pigs. *Livestock Science*. 249: 104527. DOI: 10.1016/j.livsci.2021.104527.

14 **Dybkjær L., Vraa-Andersen L., Paisley L.G., Møller K., Christensen G. & Agger J.F. 1994.** Associations between behaviour and stomach lesions in slaughter pigs. *Preventive Veterinary Medicine*. 19(2): 101-112. DOI: 10.1016/0167-5877(94)90042-6.

15 **Friendship R.M. 2004.** Gastric ulceration in swine. *Journal of Swine Health and Production*. 12(1): 34-35. DOI: 10.54846/jshap/381.

16 **Gottardo F., Scollo A., Contiero B., Bottacini M., Mazzoni C. & Edwards S.A. 2017.** Prevalence and risk factors for gastric ulceration in pigs slaughtered at 170 kg. *Animal*. 11(11): 2010-2018. DOI: 10.1017/S1751731117000799.

17 **König H.E. & Liebich H.G. 2016.** Sistema digestivo: parte cranial do canal alimentar (esôfago e estômago). In: König H.E. & Liebich H.G. (Eds). *Anatomia dos Animais Domésticos: Texto e Atlas Colorido*. 6th edn. Porto Alegre: Artmed, pp.307-376.

18 **Krepelková Z., Novotný J., Bárdová K., Link R. & Csörgö A. 2024.** Gastric ulcers in pigs - a review. *Folia Veterinaria*. 68(2): 33-42. DOI: 10.2478/fv-2024-0015.

19 **Lawrence B.V., Anderson D.B., Adeola O. & Cline T.R. 1998.** Changes in pars esophageal tissue appearance of the porcine stomach in response to transportation, feed deprivation, and diet composition. *Journal of Animal Science*. 76(3): 788-795. DOI: 10.2527/1998.763788x.

20 **Lippke R.T., Kummer R., Marques B.M.F.P.P., Mores T.J., Gonçalves M.A.D. & Barcellos D.E.S.N. 2009.** Monitoria sanitária em suinocultura. *Acta Scientiae Veterinariae*. 37(Suppl 1): s133-s146.

21 **Marchini C.F.P., Martins P.M. & Rabelo R.N. 2017.** Prevalence of gastric lesion in pigs. *Investigação*. 16(8): 50-55. DOI: 10.26843/investigacao.v16i8.1737.

22 **Marques J.L.L., Mores N., Sobestiansky J. & Zanella J.R.C. 1989.** Causas de morte súbita em suínos: III úlcera gástrica. *Concórdia: EMBRAPA-CNPSA. Comunicado Técnico 142*. 4p.

23 **Melnichouk S.I. 2002.** Mortality associated with gastric ulceration in swine. *The Canadian Veterinary Journal*. 43(3): 223-225.

24 **Mößeler A., Köttendorf S., Große Liesner V. & Kamphues J. 2010.** Impact of diets' physical form (particle size; meal/pelleted) on the stomach content (dry matter content, pH and chloride concentration) of pigs. *Livestock Science*. 134(1-3): 146-148. DOI: 10.1016/j.livsci.2010.06.121.

25 **Oliveira S.J., Bernardi R.T., Vogt F.I., Scartezzini M., Hepp D. & Lunge V.R. 2010.** Úlceras gástricas em suínos de abate: cultivo de *Arcobacter* spp. a partir de estômagos com diferentes graus de lesões. *Acta Scientiae Veterinariae*. 38(4): 351-356.

26 **Omotosho O.O., Emikpe B.O., Lasisi O.T. & Jarikre T.A. 2016.** Prevalence, distribution and pattern of gastric lesions in slaughtered pigs in south-western Nigeria. *Onderstepoort Journal of Veterinary Research*. 83(1): a1063. DOI: 10.4102/ojvr.v83i1.1063.

27 **Pearce G.P., Paterson A.M. & Pearce A.N. 1989.** The influence of pleasant and unpleasant handling and the provision of toys on the growth and behaviour of male pigs. *Applied Animal Behaviour Science*. 23(1-2): 27-37. DOI: 10.1016/0168-1591(89)90004-X.

28 **Ramis G., Gómez S., Pallarés F.J. & Muñoz A. 2005.** Comparison of the severity of esophagogastric, lung and limb lesions at slaughter in pigs reared under standard and enriched conditions. *Animal Welfare*. 14(1): 27-34. DOI: 10.1017/S096272860002889X.

29 **Robertson I.D., Accioly J.M., Moore K.M., Driesen S.J., Pethick D.W. & Hampson D.J. 2002.** Risk factors for gastric ulcers in Australian pigs at slaughter. *Preventive Veterinary Medicine*. 53(4): 293-303. DOI: 10.1016/S0167-5877(01)00286-0.

30 **Rutherford K.M.D., Thompson C.S., Thomson J.R., Lawrence A.B., Nielsen E.O., Busch M.E., Haugegaard S. & Sandøe P. 2018.** A study of associations between gastric ulcers and the behaviour of finisher pigs. *Livestock Science*. 212: 45-51. DOI: 10.1016/j.livsci.2018.03.013.

31 **Sobestiansky J., Barcellos D.E.S.N., Driemeier D. & Matos M.P.C. 2012.** Monitoramento de abate. In: Sobestiansky J. & Barcellos D.E.S.N. (Eds). *Doenças dos Suínos*. 2nd edn. Goiânia: Cânone, pp.910-931.

32 **Swaby H. & Gregory N.G. 2012.** A note on the frequency of gastric ulcers detected during *post-mortem* examination at a pig abattoir. *Meat Science*. 90(1): 269-271. DOI: 10.1016/j.meatsci.2011.06.015.

33 **Thomson J.R. & Friendship R.M. 2019.** Digestive system. In: Zimmerman J.J., Karriker L.A., Ramirez A., Schwartz K.J., Stevenson G.W. & Zhang J. (Eds). *Diseases of Swine*. 11th edn. Hoboken: Wiley-Blackwell, pp.234-263. DOI: 10.1002/9781119350927.ch15.