Investigação realizada no Serviço de Higiene e Epidemiologia da Faculdade de Medicina da Universidade do Porto, sob orientação do Professor Doutor Nuno Lunet.

Esta dissertação é baseada numa revisão sistemática de artigos científicos publicados sobre a prevalência de úlceras gástricas em suínos e respectivos factores de risco e num trabalho de campo desenvolvido para esta dissertação, intitulado “Prevalence and Risk Factors for Gastric Ulcers in Swine”.

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1. INTRODUCTION

Gastric ulceration is the most common pathology observed in the stomachs of pigs, which contributes to its economic importance (Friendship, 1999). Studies focusing on the frequency and risk factors for gastric ulcers in swine were almost inexistent before the 1960s (Jensen and Frederick, 1939; Kernkamp, 1945). With the impact of technology and industrialization, the importance of the ulcers increased and between 1960 and 1980 several authors have documented the prevalence and risk factors (especially dietary exposures) for gastric ulcers in pigs, in different settings and following heterogeneous methodologies (Muggenburg *et al.*, 1964b; Mahan *et al.*, 1966; Riker *et al.*, 1967b; Berruecos and Robison, 1972; Dobson *et al.*, 1978). Several reports about the presence of Helicobacter species in stomach of the pig were published more recently (Barbosa *et al.*, 1995; Queiroz *et al.*, 1996; Roosendal *et al.*, 2000; Choi *et al.*, 2001; Appino *et al.*, 2006).

In swine, two types of stomach ulcers may be defined and analysed: the ulcers in non-glandular region (*pars oesophagea*) and the ulcers in glandular region (cardiac, fundic and pyloric).

The ulcers in glandular region have been associated with systemic diseases such as salmonellosis, erysipelas or hog cholera infection (Curtin *et al.*, 1963; Muggenburg *et al.*, 1964a), but its prevalence is lower than the observed for ulcers in the *pars oesophagea*. The frequency of *pars oesophagea* ulcers has increased with the introduction of confinement rearing and the use of grain-based processed rations in the diet (Friendship, 1999). In the most recent studies the prevalence of *pars oesophagea* ulcers in pigs ranges between 11.6% (Ramis *et al.*, 2004) and 31.0% (Kopinski and McKenzie, 2007) while the investigations on the frequency of ulcers in the glandular region are much older and yielded prevalence estimates below 1.0% (Berruecos and Robison, 1972; Bivin *et al.,* 1974).

Ulceration of the *pars oesophagea* region can be an important cause of death in certain herds, contributing to economic losses in the pig industry. The major economic concern associated with gastric ulceration is sudden death from bleeding gastric ulcers, the most common cause of mortality during the grower-finisher stage (Melnichouk, 2002; Friendship, 2004*).*

In a survey at the Indiana Swine Evaluation Station Curtin *et al.* (1963) reported 4 deaths due to esophago-gastric ulcers among 443 pigs (4.6%), and in an observation at a slaughterhouse they found 19.6% of esophago-gastric ulcers. The results of these authors showed that pigs were more frequently affected by esophago-gastric ulcers than indicated by clinical signs and deaths.

Davenport (1969) conducted a study at a commercial piggery in South Auckland region (New Zealand), involving detailed necropsy examination of all weaned pigs that died in January 1968, observing 13 deaths due to oesophago-gastric ulceration (large ulcers were present in the *pars oesophagea*) among 48 dead pigs examined. The same author conducted a study for four days, observing 86 stomachs with erosions and ulcers in *pars oesophagea* in 962 (8.9%) pig stomachs examined at an Auckland abattoir. It appears likely that oesophago-gastric ulceration is not uncommon in New Zealand and death may be a consequence on some farms (Davenport, 1969).

In Canada, a study on a large swine farming operation conducted by Melnichouk (2002) reported that post-mortem examination of 146 pigs in one week in April revealed that 39 pigs (27.0%) and during one week of June the necropsy of 137 pigs showed that 37 pigs (27.0%) died in both cases due to severe blood loss caused by gastric ulceration. The annual economic losses attributable to gastric ulceration for this company were estimated to be above 2.3 million US dollars.

Management strategies to prevent ulcer development in swine require the understanding of the effect of factors such as nutritional and management practices. Therefore, we aimed to review systematically the evidence on the prevalence and risk factors for gastric ulcers in swine.

2. SYSTEMATIC REVIEW OF THE PUBLISHED STUDIES ON THE PREVALENCE AND RISK FACTOS FOR GASTRIC ULCERS IN SWINE

2.1. Literature search, selection of studies and data extraction for systematic review

Published articles addressing the prevalence of gastric ulcers in swine or the factors associated with its occurrence were identified in PubMed (http://www.ncbi.nlm.nih.gov/sites/entrez/). The database was searched from inception to June 2008, using the following expression: (pig OR swine OR pigs OR sow) AND ulcer.

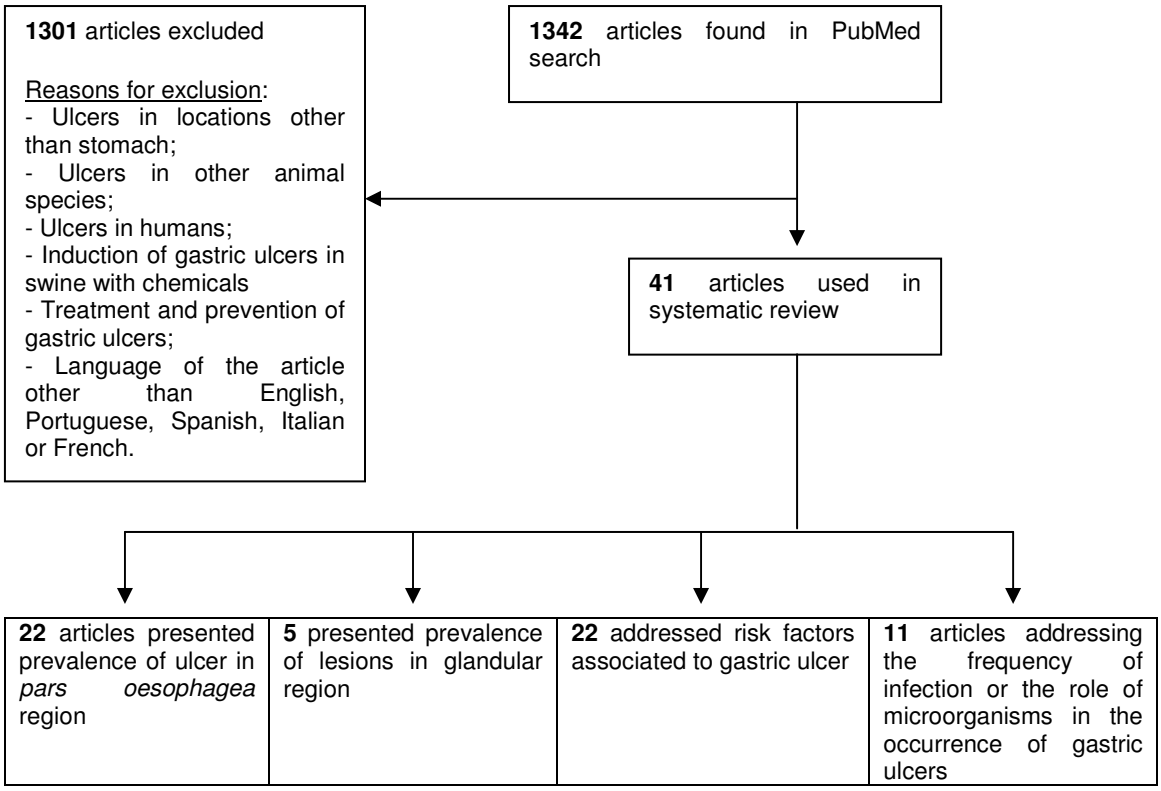
Studies covering the association between infection and stomach ulcers were also considered for review. Only articles in English, Portuguese, French, Italian and Spanish published as a full paper or letter to the editor and referring to original research were eligible.

From each study, information was extracted regarding de following characteristics: year of publication, country, prevalence of lesions in the stomach or specific stomach anatomical location (*pars oesophagea*, cardiac, fundic and pyloric regions), factors associated with the occurrence of gastric lesions (*e.g.*: age, gender, growth rate/weight/back-fat, genetic origin, nutritional factors, frequency of feeding, ambient temperature, source of water, microorganisms).

2.2. Systematic review flow chart and description of the studies

The electronic database search yielded 1342 articles covering a period from 1951 to 2008, from which 41 were considered eligible for the systematic review, as shown in the systematic review flow-chart (Figure 1).

Figure 1. Systematic review flow-chart.



From the 41 papers included in the systematic review, 22 referred to studies conducted in the North America (19 in the United States – Curtin *et al.*, 1963; Muggenburg *et al.*, 1964a; Muggenburg *et al.*, 1964b; Mahan *et al.*, 1966; Gamble *et al.*, 1967; Chamberlain *et al.*, 1967; Riker *et al.*, 1967a; Riker *et al.*, 1967b; Wallin *et al.*, 1969 ; Pickett *et al.*, 1969; Muggenburg *et al.*, 1971; Berruecos and Robison, 1972; Suarez *et al.*, 1997; Krakowka *et al.*, 1998; Lawrence *et al.*, 1998; Eisemann and Argenzio, 1999; Doster, 2000; Choi *et al.*, 2001; Eisemann *et al.*, 2002. Two in Canada

– Robert *et al.*, 1991; Melnichouk, 2002 and one study conducted by USA and Canada – Krakowka and Ellis, 2006), 8 in Europe (Flatlandsmo and Slagsvold, 1971; Elbers *et al.*, 1995; Guise *et al.*, 1997; De Groote *et al.* 1999; Roosendaal *et al.*, 2000; Ramis *et al.* , 2004; Amory *et al.*, 2006; Appino *et al.*, 2006), 3 in Africa (Makinde and Gous, 1998; Banga-Mboko *et al.*, 2003; Mall *et al.*, 2004), 3 in South America (all from Brazil – Bivin *et al.* , 1974; Barbosa *et al.*, 1995; Queiroz *et al.*, 1996), 3 from Australia (Dobson *et al.*, 1978; Robertson *et al.*, 2002; Kopinski and McKenzie, 2007), 2 from New Zealand (Davenport, 1969; Tannock and Smith, 1970).

The most recent studies quantified the frequency of gastric lesions in animals selected at the slaughterhouses (Melnichouk, 2002; Kopinski and McKenzie, 2007) or investigated risk factors for gastric ulcers (Robertson *et al.*, 2002; Amory *et al.*, 2006). There are also recent studies about the association of Helicobacter spp. and the occurrence of gastric ulcers in swine (Roosendaal *et al.*, 2000; Choi *et al.*, 2001; Krakowka and Ellis, 2006). The oldest studies were mostly experimental research or reporting on the relationship between nutritional factors and gastric ulcers (Mahan *et al.*, 1966; Pickett *et al.*, 1969; Flatlandsmo and Slagsvold, 1971; Dobson *et al.*, 1978).

2.3. Prevalence of ulcers in swine

2.3.1. Ulcers in glandular region

Five studies estimated the prevalence of lesions in glandular region of the stomach, mainly in the fundic and pyloric regions of the stomach (Table 1).

The prevalence of ulcers in glandular region ranged from 0.19% (Bivin *et al.*, 1974) to 2.1% (Muggenburg *et al.*, 1964b), although the latter estimate included erosions and ulcers.

Table 1. Prevalence of ulcers in the glandular region of the stomach, in swine.

Author, year	Country	Sample size (% females)	Cardiac ulcer (%)	Fundic ulcer (%)	Pyloric ulcer (%)
Curtin *et al.*, 1963	USA	443 (68.4)	---	0.9	0.22
Muggenburg *et al.*, 1964a	USA	594 (NS)	0.1	1.1	---
Muggenburg *et al.*, 1964b	USA	3753 (a sample of 1019 barrows and 597 gilts was used to compare the prevalence of gastric lesions between males and females; 464 gilts and 1002 sows were used to address age differences)		2.1 [1]	
Berruecos and Robison, 1972	USA	263 (0)	---	0.7	---
Bivin *et al.*, 1974	Brazil	3113 (NS)	0.06	---	0.12

[1] Erosions are included
NS – Not specified in the original article

2.3.2. Ulcers in *pars oesophagea*

Twenty two studies presented results for prevalence of lesions in *pars oesophagea* (table 2).

Prevalence of ulcers in the *pars oesophagea* region of the stomach in pigs ranged from 2.3% to 66%, as described in studies conducted in the USA, Canada, Brazil, South of Africa, Burkina Faso, New Zealand, Australia, Norway, Netherlands, United Kingdom, Italy and Spain, although most studies found in this systematic review are from USA. In the latter country, the prevalence of ulcers in *pars oesophagea* ranged from 5.5% (Muggenburg *et al.*, 1964b), in a sample also including sows, to 66% in an experimental survey with finishing pigs (Wallin *et al.*, 1969).

Melnichouk (2002) found a 15.5% prevalence of ulcers in *pars oesophagea* in a recent Canadian survey.

In Europe the prevalence of *pars oesophagea* ulcers ranged from 11.6% in Spain (Ramis *et al.*, 2004) to 19.1% in United Kingdom (Amory *et al.*, 2006), if studies that consider erosions for calculation of the prevalence of gastric ulcer are excluded.

Two studies were conducted in Africa, both showing a low prevalence of gastro-oesophageal ulceration (South of Africa: 5.1% and Burkina Faso: 10%) and Brazil has similar prevalence of gastric ulcer in oesophageal region.

Studies from New Zealand and Australia used the term "oesophago-gastric ulcers - OGU" as referring to both ulcers and erosions (Davenport *et al.* 1969; Robertson *et al.*, 2002) and the figures obtained are not directly comparable with those from the remaining investigations. Other reports, from countries such as Norway, Netherlands, United Kingdom and USA also took erosions into account when computing the prevalence of *pars oesophagea* ulcers (Flatlandsmo and Slagsvold, 1971; Elbers *et al.*, 1995; Suarez *et al.*, 1997; Amory *et al.*, 2006).

Four of the studies reporting on the prevalence of gastric ulcer were experimental research (Mahan *et al.*, 1966; Riker *et al.*, 1967b; Wallin *et al.* 1969; Flatlandsmo and Slagsvold, 1971), aiming to evaluate the influence of nutritional factors in the development of gastric ulcers. Eisemann *et al.* (2002) assessed the effect of feed withdrawal in the prevalence of gastric ulcers.

Kopinski and McKenzie (2007) developed a visual guide of morphological changes that can occur in the *pars oesophagea* region. Thirty-one percent of the finishing pigs developed ulcers with or without stenosis.

Table 2. Prevalence of ulcers in the *pars oesophagea* of finishing pigs and culled breeding animals.

Author, year	Country	Sample size (% females)	Lesions evaluated in *pars oesophagea* region	*Pars oesophagea* ulcer (%)
Curtin *et al*., 1963	USA	443 (68.4)	Large ulcer or ulcer scar	19.6
Muggenburg *et al*., 1964a	USA	594 (NS)	Sub-acute and chronic ulcers	34.3
Muggenburg *et al*., 1964b	USA	3753 (For comparison of gastric lesions between males and females a sample of 1019 barrows and 597 gilts was used For comparison of gastric lesions across age groups, a sample of 464 gilts and 1002 sows was used)	Sub-acute and chronic ulcers	5.5 [1]
Mahan *et al*., 1966	USA	120 (48.3)	Number of ulcers	10.8 [2]
Riker *et al*., 1967b	USA	102 (32.3)	Number of ulcers	13.7
Davenport, 1969	New Zealand	962 (NS)	Erosions and ulcers	8.9
Wallin *et al*., 1969	USA	47 (NS)	Number of ulcers, including pigs that died with gastric ulcer	66.0
Flatlandsmo and Slagsvold, 1971	Norway	341 (NS)	Erosions and ulcers	2.3
Berruecos and Robison, 1972	USA	263 (0)	Ulcers	21
Bivin *et al.* , 1974	Brazil	3113 (NS)	Acute and chronic ulcers	11 [2]
Elbers *et al*., 1995	Netherlands	458 (50.4)	Hiperkeratosis and more than 10 erosions and/or erosions larger than 5 cm and/or ulcers or stenosis of the oesophagus towards the stomach	10.7
Suarez *et al*., 1997	USA	86 (NS)	Erosions and ulcers	24.4
Guise *et al*., 1997	United Kingdom	1242 (Only 358 pigs in the study were distinguished by gender with 150 females)	Severe ulceration with larger break(s) in the mucous membrane	13.4

Table 2. ***(Cont.)*** Prevalence of ulcers in the *pars oesophagea* of finishing pigs and culled breeding

Author, year	Country	Sample size (% females)	Lesions evaluated in *pars oesophagea* region	*Pars oesophagea* ulcer (%)
Makinde and Gous, 1998	South of Africa	4320 (NS)	Complete epithelial loss with exposure of the underlying muscular layer	5.1
Melnichouk, 2002	Canada	1021 (NS)	Deep or extensive ulcers	15.5
Eisemann *et al*., 2002	USA	754 (NS)	Active ulceration and/or extensive, active ulceration with epithelial loss	16.4
Robertson *et al.* 2002	Australia	15741 (Only animals from one herd were distinguished by gender Sows came from three herds but the number of these animals is not specified by the author)	Erosions and ulcers	17 [1]
Banga-Mboko *et al.* 2003	Burkina Faso	114 (NS)	Ulcers	10
Ramis *et al.* 2004	Spain	20796 (NS)	Deeper, hardened and roughened ulcer, with haemorraghic points and/or chronic ulcer without bleeding	11.6
Amory *et al*., 2006	United Kingdom	800 (NS)	Erosions and/or ulcers (with or without bleeding) or stenosis of the oesophagus towards the stomach	19.1
Appino *et al*., 2006	Italy	595 (NS)	Acute and chronic ulcers	12.6
Kopinski and McKenzie, 2007	Australia	280 (NS)	Developed ulcers, haemorrhage and stenosis present	31

[1] culled breeding animals are included.
[2] Values calculated by the author
NS – Not specified in the original article

2.4. Association between characteristics of the animals and the occurrence of gastric lesions

Eleven studies evaluated the prevalence of gastric lesions according to the characteristics of the animals.

Gender, age and growth rate/weight/ back-fat are factors inherent to the animal (table 3 and table 4). Some studies reported the influence of genetic origin in gastric ulcer.

2.4.1. Gender

The older studies (Curtin *et al.*, 1963; Muggenburg *et al.*, 1964b) described a higher prevalence of oesophago-gastric lesions in males compared to females. However, our calculations using their data showed no statistically significant effect of gender in oesophago-gastric lesions. Elbers *et al.* (1995), Guise *et al.* (1997) and Robertson *et al.* (2002) found no statistically significant gender differences in the prevalence of oesophago-gastric lesions, though the prevalence tended to be higher in males.

Mahan *et al.* (1966) observed no statistically significant differences in the incidence of esophago-gastric ulcers related to gender and Flatlandsmo and Slagsvold (1971) had the same conclusion but their papers do not mention the results related to gender.

2.4.2. Age

Most studies evaluated finishing pigs, and therefore there is almost no variation in their age at the slaughter. Only two studies observed stomachs of the culled sows to compare with finishing pigs, with contradictory results. Muggenburg *et al.* (1964b)

found no difference in the prevalence of gastric lesions between culled sows and gilts, but Robertson *et al.* (2002) detected a significantly higher prevalence of gastric ulcer in sows compared to finishing pigs.

Table 3. Association between gender and age and the occurrence of gastric ulcers in swine.

Author, year	Country	Gender	Age
Curtin *et al*, 1963	USA	Castrate males vs. Females: Ulcers = 23.6% vs. 17.8% OR = 1.42 (95% CI: 0.85 to 2.38)	---
Muggenburg *et al.*, 1964b	USA	Barrows vs. Gilts: Ulcers = 7.0% vs. 5.2% OR = 1.39 (95% CI: 0.88 to 2.19)	Sows vs. Gilts: Ulcers = 6.0% vs. 5.1% OR =1.17 (95% CI: 0.70 to 1.96)
Mahan *et al.*, 1966	USA	There were no differences in the incidence of esophago-gastric ulcers related to gender (NS)	---
Flatlandsmo and Slagsvold, 1971	Norway	There were no differences in the mean gastric ulcer index in relation to gender (NS)	---
Elbers *et al.*, 1995	Netherland	Barrows vs. Gilts: Ulcers = 12.0% vs. 9.5% OR = 1.28 (95% CI: 0.68 to 2.42)	---
Guise *et al.*, 1997	United Kingdom	Only 358 pigs in the study were distinguished by gender Males vs. Females: Ulcers = 57.2% vs. 49.3% OR = 1.37 (95% CI: 0.88 to 2.14)	---
Robertson *et al.*, 2002	Australia	There were no significant differences in the prevalence of oesophago-gastric ulcers in male (35%) and female (32%) pigs sampled from the same Western Australian piggery (p=0.54)	Sows had a significantly higher prevalence of oesophago-gastric ulcers than finishing pigs (p<0.05)

OR – Odds Ratio; 95% CI – 95% Confidence Interval
OR and 95% CI computed by the author
NS – Not specified in the original article

2.4.3. Growth Rate / Weight / Back-fat

The cross-sectional nature of the observations does not allow the clarification of the temporal relation between the occurrence of ulcers and growth rate/weight. Also, the association between chronic ulceration of the *pars oesophagea* and growth rate and feed efficiency is difficult to summarize due to the contradictory nature of the findings (Doster, 2000).

Wallin *et al.* (1969), Dobson *et al.* (1978), Guise *et al.* (1997) and Robertson *et al.* (2002) found no association between the presence of gastric lesions in pigs and growth rate, whereas Elbers *et al.* (1995) and Eisemann *et al.* (2002) found a negative association with growth rate or carcass weight. Confounding factors not taken into account in the analyses, such as health, feeding regime or management could be responsible for the different results of investigations about growth rate/weight and gastric ulcers (Robertson *et al.*, 2002).

Berruecos and Robison (1972) reported a negative association between low back-fat and the prevalence of gastric ulcers but their results indicated that selection for efficiency or fast growing pigs would not increase the incidence of ulcers.

Table 4 shows the summary of the results from studies addressing the relation between the existence of gastric lesions and growth rate/weight/back-fat.

Table 4. Association between the occurrence of gastric ulcers and growth rate/weight/back-fat in swine.

Author, year	Country	Results / Conclusions
Wallin *et al*., 1969	USA	There was no apparent relationship between the presence of gastric ulcers and rate of gain in body weight (NS).
Berruecos and Robison, 1972	USA	There is high negative correlations between live back-fat and ulcer score (selection for low back-fat may cause an increase in the occurrence of ulcers) (NS).
Dobson *et al*. , 1978	Australia	There was no significant difference in growth rate of pigs with ulcers when compared with pigs without ulcers (NS).
Elbers *et al*., 1995	Netherland	This study indicated that finishing pigs with extensive erosions and/or ulceration of the *pars oesophagea* gained 50 to 75g/day less than finishing pigs with no lesions or only slight oesophago-gastric lesions.
Guise *et al*., 1997	United Kingdom	There was no significant difference in the daily liveweight gain of the male (0.079kg) and female (0.062kg) with ulcers when compared with males (0.074kg) and females (0.083kg) without ulcers.
Robertson *et al*., 2002	Australia	There was no detectable difference in the weight gain of pigs with and those without gastric ulcers.
Eisemann *et al*., 2002	USA	When hot carcass weight (kg) was partitioned into quartiles (< 71.2; 71.2-76.1; 76.2-80.3; >80.3), the prevalence of gastric ulcers increased when carcass weight decreased ($p<0.01$).

NS – Not specified in the original article

2.4.4. Genetic Origin

Studies addressing the relationship between genetic background and gastric lesions did not yield consistent results to prove such an association.

In the Curtin's study (1963) ulcers in *pars oesophagea* occurred in 8 of 9 breeds (Yorkshire – 35.6%; Hampshire - 40.2%; Landrace – 11.5%; Berkshire – 2.3%; Spotted Poland China – 4.6%; Poland China – 1.2%; Tamworth – 2.3%; Duroc Jersey – 2.3%) during the survey but the number of pigs in several of the breeds was so small that prevalence of ulceration could not be significantly ascertained from the data. They found no ulcers in four Chester White pigs.

Muggenburg *et al.* (1964b) showed no significant differences in the prevalence of gastric ulcers between Poland China (4.1% of *pars oesophagea* ulcers), Chester White (8.4% of *pars oesophagea* ulcers) and Hampshire (3.8% of *pars oesophagea* ulcers) breeds and no difference between the Yorkshire (12.4% of *pars oesophagea* ulcers) and Duroc (17.6% of *pars oesophagea* ulcers) breeds in their survey.

Berruecos and Robison (1972) are the only authors that showed the breed effects on gastric ulcer and they found significantly higher incidence of ulcers lesions in Duroc (29%) compared with the Yorkshire breed (12%).

Mahan *et al.* (1966) found a prevalence of gastric ulcer approximately eight times as higher in crossbred pigs YorkshireXHampshire (22.2%) than in crossbred spotted pigs (2.8%). Although these data do not prove a genetic relationship to the prevalence of gastric ulcer, the authors suggested that genetic origin may be another factor associated with the occurrence of the disease and may partially explain the differences obtained in various studies.

Elbers *et al.* (1995) mentioned that the differences between litters observed in their study (all the finishing pigs were F_2 crossbred from different breeding company) may also be an indication that some of the differences in the prevalence of *pars oesophagea* lesions can be attributed to a genetic origin but, the relationship between genetic and gastric lesions was not specifically investigated.

2.5. Association between characteristics of the farms and the occurrence of gastric lesions

Seventeen studies presented results for farm-related factors associated with the occurrence of gastric lesions in swine.

After birth, piglets are fed with maternal milk and some ration during twenty five days. At weaning, the pigs pass to the growing phase until 75 days of age. Afterwards, the animals go to fattening phase until five or six months of age. The feed management during these phases is variable between farms. Most of the studies about nutritional

factors are experimental, which allows results less prone to bias and more consistent findings.

2.5.1. Nutritional factors

To achieve optimal pig performance it is necessary to process cereal grains through a hammer mill or roller mill to reduce particle size. Reduction of particle size increases surface area of the grain, increasing the surface area for enzyme action, improving the efficiency of digestion and ultimately the efficiency of body weight gain. Furthermore, particle size reduction allows uniform mixing of grain with protein, vitamin and mineral supplements. The finely ground can be mechanically processed into small structures similar to tubes called pelleted feeds. This type of diet allows an easier handling and improved feeding practices, being generally associated with a better performance. Expanded grain is produced through a heat treatment of the cereal grain and was described as a risk factor for gastric ulcer by Pickett *et al.* (1969).

Mahan *et al.* (1966), Dobson *et al.* (1978) and Lawrence *et al.* (1998) reported that finely ground diets are associated with an increased prevalence of gastric lesions compared to the observed when animals are fed with coarsely ground diets.

Chamberlain *et al.* (1967), Gamble *et al.* (1967), Flatlandsmo and Slagsvold (1971), Eisemann and Argenzio (1999), Robertson *et al.* (2002) and Amory *et al.* (2006) showed that feeding of pelleted food is involved in the development of gastric lesions. Amory *et al.* (2006) showed that pelleted feed had less influence than the slatted floors in the occurrence of gastric ulcers in the herds.

Mahan *et al.* (1966), Riker *et al.* (1967a) and Pickett *et al.* (1969) reported that feeding with expanded corn was associated with a higher frequency of ulcers when compared to raw corn. With exception of the study reported by Robertson *et al.* (2002) and Amory *et al.* (2006), all studies are experimental. The main results and conclusions are presented in table 5.

Table 5. Association between diet and the occurrence of gastric lesions in swine (experimental studies).

Authors, year	Results/Conclusions
Mahan *et al.*,1966	More lesions developed with finely ground corn than with coarsely ground corn diets and expanded corn produced more ulcers than did the various grinds of unprocessed corn. These results indicated that, although feed fineness is a factor in precipitating ulcers, it is not solely responsible.
Riker *et al.*, 1967a	The expansion of the grains increased the incidence of lesions, expressed on the basis of the ulcer index, significantly only in the case of corn or milo but there was no significant difference in severity of the lesions between pigs fed raw or expanded wheat or barley, indicating that expansion per se was not the sole factor influencing ulcer formation.
Chamberlain *et al.*, 1967	Pigs consuming pelleting diet had a significantly higher mean ulcer score than those consuming either of the unpelleted feeds.
Gamble *et al.*, 1967	Pelleted diets produced significantly more ulcers than did the use of meal.
Pickett *et al.*, 1969	This study indicated finely ground diets as a contributing factor to the occurrence of oesophago-gastric lesions and the expansion of the grains in diet increased the incidence of oesophago-gastric lesions.
Flatlandsmo and Slagsvold, 1971	Finely ground and use of pellets in the diet appeared to be the most consistent factors related to gastric lesions.
Dobson *et al.*, 1978	Finely ground rations were more likely to produce ulcers than either the coarsely ground wheat rations or the standard barley ration but the effect of pelleting process was less clear.
Lawrence *et al.*, 1998	Feeding finely ground diet increased the prevalence of *pars oesophagea* abnormalities.
Eisemann and Argenzio, 1999	Pigs fed the finely ground and pelleted diet had greater incidence of lesions in stomachs than pigs fed a coarse diet.

2.5.2. Frequency of feeding

Two studies addressed the influence of feeding frequency in the occurrence of gastric ulcers, with inconsistent results.

Robert *et al.* (1991) observed a higher frequency of gastric ulcers in the restricted-fed pigs and suggested that feed restriction could be a factor of stress for the pigs. Another explanation for these results is that the group of restricted-feed pigs could have lower amounts of crude fibre in their diet when compared with the diet of the group *ad libitum* pigs. Robertson *et al.* (2002) reported that pigs fed *ad libitum* had higher prevalence of oesophago-gastric ulcer.

2.5.3. Farm size

Ramis *et al.* (2004) reported a higher prevalence of oesophago-gastric lesions in largest farms (more than 50000 finishing pigs). One possible explanation is the usual mixing of pigs from several sources in the large finishing units, causing a higher level of respiratory and digestive diseases between animals. Another possible reason is the use of pelleted diet by large producers to improve feed conversion rates (rapid growth and lean deposition) and farms smaller normally use meal to feed their animals.

2.5.4. Ambient temperature

Curtin *et al.* (1963) observed more deaths due to gastric ulcers during late spring and early winter, when there are wider variations in ambient temperature. Riker *et al.* (1967b) showed that pigs living in environments with wide variations of the temperature had more gastric lesions than those remaining in environments with stable temperature, which could be attributed to the continuous stress caused by temperature fluctuations.

Muggenburg *et al.* (1964b) did not reach conclusive findings in a first study on this topic, but latter on (Muggenburg *et al.*, 1971) concluded that there is no association between variations of the ambient temperature and the development of gastric lesions.

Robertson *et al.* (2002) could not show a significant association between factors such as the type of ventilation (mechanical / natural air) or the existence of heating system and the occurrence of gastric ulcers.

Amory *et al.* (2006) reported that finisher pigs with controlled environment (ventilation or heating system) had a higher prevalence of gastric ulcer than those raised in a non-controlled environment.

2.5.5. Type of floor

The effect of the type of floor in gastric ulcers in pigs had been reported by only one study of Amory *et al.* (2006). The authors concluded that pigs kept on slatted floors had significantly more gastric ulceration than pigs kept on a solid floor, and that these had significantly higher ulcer scores than pigs provided with straw bedding. The authors suggested that animals housed in the slatted floors may have a high level of respiratory diseases, which would cause more interruptions on feeding, and a consequent increase of gastric ulcer prevalence. They, also, suggested that the straw provided an additional source of fibre which may prevent the gastric ulcers.

2.5.6. Water

Robertson *et al.* (2002) showed an association between the source of water and oesophago-gastric ulcer. The animals receiving dam water had a higher prevalence of oesophago-gastric ulcer than those from farms using water from a river or bore. The authors suggested that dam water often has bacterial and algal blooms during hot weather and the microbiological quality of the water may have influence in the

occurrence of oesophago-gastric ulcer. In humans, excretion of Helicobacter species in feces and subsequent contamination of water has been suggested (Choi *et al.*, 2001), and it may be important to evaluate this pathway as source of Helicobacter spp. to infect the stomach of the pig. Another possible explanation for the importance of drinking water is that water from different sources may have different pH values and different buffering effect than can influence the development of gastric ulcer. But in this study it was not possible to evaluate the quality of drinking water of the farms.

In table 6 we present a summary of the results from studies about the relation between the characteristics of the farms and with the occurrence of gastric lesions in swine.

Table 6. Association between the characteristics of the farm and the occurrence of gastric lesions in swine.

Authors, year	Frequency of feeding	Farm size	Temperature	Type of floor	Water
Curtin *et al*, 1963	---	---	Seasonal incidence of deaths suggested that variations in ambient temperature are a factor in precipitating the clinical signs associated with esophago-gastric ulcers.	---	---
Muggenburg *et al*., 1964b	---	---	No conclusion on the seasonal variation of gastric ulcers was reached.	---	---
Riker *et al*., 1967b	---	---	Pigs which were rotated between the 29.4ºC and the 18.3ºC every 3 days showed more lesions than those remaining in constant environment (p>0.01)	---	---
Muggenburg *et al*. , 1971	---	---	No differences were observed in number and severity of gastric lesions between pigs exposed to high or low temperature.	---	---
Robert *et al*., 1991	The percentage of animals showing sever ulcers was nevertheless higher in the group of pigs on restricted feed than in pigs given continual access to feed.	---	---	---	---
Robertson *et al*., 2002	Pigs fed *ad libitum* vs. pigs fed a restricted diet: OR=3.7 (95% CI: 8.9 to 21)	---	This study did not consider farms with no acclimatization (mechanical ventilation, heating system and cooling system) as factors associated with high prevalence of gastric ulcers.	---	Piggery water from a dam vs. Water from river or bore: OR=3.8 (95% CI: 2.9 to 4.9)
Ramis *et al*., 2004	---	Farms with < 10000 pigs vs. 10000 – 50000 pigs Ulcers = 8.13% vs. 8.42% OR = 1.04 (95% CI: 0.9 to 1.2) Farms with < 10000 pigs vs. > 50000 pigs: Ulcers = 8.13% vs. 19.45% OR = 2.73 (95% CI: 2.5 to 3.0)	---	---	---
Amory *et al*., 2006	---	---	Finisher pigs with controlled environment (thermostatically or ventilation) had a significantly higher mean ulcer score (p< 0.05) than pigs with no controlled environment.	Finisher pigs housed on slatted floor had a significantly higher mean ulcer score (p< 0.001) than pigs housed on solid concrete floors, which had significantly higher score (p<0.01) than pigs housed on straw bedding.	---

OR – Odds Ratio; 95% CI – 95% Confidence Interval

2.6. Association between stomach infections and the occurrence of gastric lesions

The presence of microorganisms in stomach of the pig was referred by eleven articles of this review.

The stomach of the pig was shown to be colonized by different species of microorganisms, such as *Candida albicans* (Curtin *et al.*, 1963, Tannock and Smith, 1970), *Candida slooffi*, *Escherichia coli*, Peptostreptococci, Veillonellae, Proteus species, Clostridium perfringens (Tannock and Smith, 1970), Lactobacillus (Tannock and Smith, 1970; Krakowka *et al.*, 1998), Bacillus (Krakowka *et al.*, 1998), but none of these investigations quantified the association between infection and the occurrence of gastric ulcers in swine.

Barbosa *et al.* (1995) reported that a tightly coiled spiral bacteria in the stomachs of pigs, named *Gastropirillum suis.* After direct PCR on tissue samples from pig stomachs, it was found that this bacterium belongs to the genus Helicobacter and some authors speculated that *Gastropirillum suis* and *Helicobacter heilmannii* type 1 represent the same species (Queiroz *et al.*, 1996; De Groote *et al.*, 1999). It was proposed the name 'Candidatus Helicobacter suis' for this gastric helicobacter identified in pigs (De Groote *et al.*, 1999).

The prevalence of infection with Helicobacter spp was reported to be above 60% in the stomachs of pigs at the age of slaughter (Barbosa *et al.*, 1995; Choi *et al.*, 2001; Queiroz *et al.*, 1996; Roosendaal *et al.*, 2000).

Helicobacter infection has been associated with gastric ulceration in pigs (Barbosa *et al.*, 1995; Queiroz *et al.* ,1996; Roosendal *et al.*, 2000; Choi *et al.*, 2001; Appino *et al.*, 2006) but conflicting results were reported by Krakowka *et al.* (1998) and Mall *et al.* (2004) that found no consistent association between *Helicobacter* infection and *pars oesophagea* ulceration in pig's stomach. In the study of Suarez *et al.* (1997),

they did not detect Helicobacter spp. in stomach of the pigs but they detected Arcobacter spp.

Although Krakowka *et al.* (1998) did not show an association between *Helicobacter* infection and *pars oesophagea* ulceration in pig's stomach, Krakowka and Ellis (2006) reported that a high carbohydrate diet and gastric colonization by porcine *Helicobacter pylori*-like bacteria (closely related to human *Helicobacter pylori* but distinct from *Helicobacter heilmannii*) facilitates the development of clinically significant gastro-esophageal ulcers in piglets.

More studies are needed to clarify the association between Helicobacter and gastric lesions in swine and the mode of transmission (Choi *et al*., 2001).

2.7. Conclusions

The prevalence of ulcers in *pars oesophagea* is high in many studies and is a worldwide problem that deserves more investigations about risk factors associated to the disease.

Inherent characteristics to the animal as gender and age have contradictory results but recent studies showed that males and females have similar prevalence of gastric ulcer and in sows the prevalence of gastric ulcers is significantly higher than in finishing pigs.

Genetic origin may be another factor associated with the occurrence of the disease and may partially explain the differences obtained in various studies but there are no consistent results in the articles reviewed.

The available evidence does not allow conclusions on whether the variations in growth rate/weight are a consequence or a risk factor of gastric ulcer in pigs.

Finely ground and pelleted diets are the risk factor for gastric lesions for which the evidence is more consistent. Other possible risk factors of gastric ulcers related to

farm management as frequency of feeding, ambient temperature and stomach infections had contradictory results in the articles reviewed.

The quality of water could be an important risk factor for gastric lesions in swine and a possible source of Helicobacter spp. for pigs. It is also important to clarify the influence of Helicobacter spp. on gastric lesions and how these species can infect the pig.

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3. OBJECTIVES

Gastric ulceration is the most common pathology observed in the stomachs of the pigs and can be an important cause of death in certain herds. Economics and welfare concerns justify the monitoring of swine populations to determine the prevalence and severity of stomach lesions (Friendship, 2004). The prevalence of ulcers in *pars oesophagea* is high in many studies and is a worldwide problem that deserves more investigations about risk factors associated to the disease because of the contradictory results of the studies, except for the type of diet.

The objectives of this dissertation were:

- To estimate the prevalence of gastric ulcers in swine raised in Portugal;
- To quantify the association between animal and farm characteristics and the occurrence of *pars oesophagea* ulcers lesions.

4. MANUSCRIPT: PREVALENCE AND RISK FACTORS FOR GASTRIC ULCERS IN SWINE

ABSTRACT

Introduction

Gastric ulceration is the most common pathology observed in the pigs' stomachs and can be an important cause of death in certain herds. Economics and welfare concerns justify the monitoring of swine populations to determine the prevalence and severity of gastric lesions.

The prevalence of gastric ulcer in swine is unknown in Portugal and, apart from nutritional factors, its determinants are poorly understood. Therefore, we aimed to quantify the prevalence of gastric ulcers in swine and the association between animal and farm characteristics and the occurrence of these lesions.

Methods

Finishing pigs (n=760), approximately 6 months old, and culled breeding animals (n=127), aged one year or more, were randomly selected at a slaughterhouse in the North of Portugal, among the animals from 9 previously specified farms. Their stomachs were visually evaluated for the presence of ulcers. Information on gender, carcass weight, lean meat percentage, and farm/managing characteristics were obtained through the slaughterhouse records or inquiry to the veterinarians from each farm.

The prevalence of gastric ulcers was computed for the whole sample, according to age and gender, and separately for animals from each farm.

The association between farm characteristics (dimension of the farm, mixing pigs, feeding system, air ventilation, heating system, systematic sanitary disinfection, source of water) and the occurrence of *pars oesophagea* ulcers was quantified through Odds

Ratios and respective 95% Confidence Intervals (95% CI), computed by unconditional logistic regression with robust standard errors (allowing for clustering by farm)

Data were analyzed using STATA®, version 9.2.

Results

The overall prevalence of ulcers in *pars oesophagea* was 18.7%, significantly higher in breeding animals than in finishing pigs (34.6% vs. 16.0%, $p<0.001$), and a wide variation was observed across farms (range: 7.5%-41.2% for finishing pigs, 8.3%-71.4% for breeding animals). The overall prevalence of ulcers in the glandular region was 0.7%.

Finishing pigs from farms with mine/well water supply had a higher prevalence of *pars oesophagea* ulcers than those from farms with pit water supply (OR=3.49, 95% CI: 1.75-6.94).

There was no significant association between gender, carcass weight/back-fat, or managing conditions of the farms (*e.g.* mixing pigs after weaning or before finishing phase, feeding system, type of air ventilation, presence or not of heating system, systematic sanitary disinfection) and the prevalence of *pars oesophagea* ulcers in finishing pigs.

Conclusion

The prevalence of gastric ulcer in this survey is in the upper range of the observation in other countries. Our results suggest that the source of water may be important risk factor to the development of ulcers in *pars oesophagea.*

INTRODUCTION

Gastric ulceration is the most common pathology observed in the stomachs of the pigs and can be an important cause of death in certain herds (Friendship, 1999; Melnichouk, 2002). The major economic concern associated with gastric ulceration is sudden death from bleeding gastric ulcers, the most common cause of mortality during the grower-finisher stage (Melnichouk, 2002; Friendship, 2004). Regarding a possible influence of the gastric ulcers in the live weight gain of the animals, the available evidence is contradictory (Elbers *et al.*, 1995; Guise *et al.*, 1997; Eisemann *et al.*, 2002; Robertson *et al.*, 2002). Moreover the cross-sectional nature of these studies precludes a proper interpretation of the results in what concerns to the temporal relation between these phenomena. Economics and welfare concerns justify the monitoring of swine populations to determine the prevalence and severity of stomach lesions (Friendship, 2004).

Ulceration occurs principally in the non-glandular region of the stomach (*pars oesophagea*), whereas ulceration of the fundic and pyloric region (glandular region) is rare and normally associated with systemic infections (Friendship, 1999). The prevalence of *pars oesophagea* ulcer in finishing pigs varies widely across studies, ranging from 2.3% in Norway (Flatlandsmo and Slagsvold, 1971) or 5.1% in South of Africa (Makinde and Gous, 1998) to 31% in the Australia (Kopinski and McKenzie, 2007) or 34.3% in the USA (Muggenburg *et al.*, 1964a), although these studies span over many years and are heterogeneous in the methods and criteria used to define ulcer.

The aetiology of gastric ulceration is multifactorial, being determined by the physiologic characteristics of the animals, and feeding and management practices, namely those that contribute to the social stress of the animals (*e.g.* type and the frequency of feeding [Flatlandsmo and Slagsvold, 1971; Eisemann and Argenzio,

1999], source of water (Robertson *et al.*, 2002), microbiological environmental contamination and illnesses (Ramis *et al.*, 2004)).

The available evidence strongly supports the association between type of food and the occurrence of gastric ulcers in swine. The use of pelleted ration increases the risk for development of gastric ulcers (Chamberlain *et al.*, 1967; Flatlandsmo and Slagsvold, 1971; Eisemann and Argenzio, 1999; Amory *et al.*, 2006). For other potential determinants of gastric ulceration the findings from different studies are contradictory (Robert *et al.*, 1991; Robertson *et al.*, 2002).

The prevalence of the gastric ulcer in swine is unknown in Portugal and it would be important to estimate the frequency of this disease, as well as the assessment of risk factors in this specific setting.

Our objectives were to quantify the prevalence of gastric ulcer and the association between animal and farm characteristics and the occurrence of these lesions in animals raised in Portugal.

MATERIALS AND METHODS

Finishing pigs (animals with approximately 6 months of age) and culled breeding animals (aged one year or more) were selected at a slaughterhouse in the region of Porto, North of Portugal, chosen due to the proximity to the researcher's place of work. This slaughterhouse receives finishing pigs from national farms and from Spain and the culled breeding animals came only from national farms. Animals from nine portuguese farms are included in this survey, the only for which we could obtain reliable information about the managing conditions.

Between August and December 2006, one farm was randomly selected by simple random sampling, using a random numbers table (EpiInfo 6.04d, Centers for Disease Control and Prevention, EUA), among the farms eligible for this study

providing animals for slaughtering in the same day (when more than one farm was providing animals).

Twenty hybrid finishing pigs were then consecutively selected after the selection of the order number of the first animal to be included in the study by simple random sampling, using a random numbers table. When the selected farm provided less than 20 animals for slaughtering, all the animals from that farm were included in the study. All the culled breeding pigs admitted in the abattoir in the same days were included in the study, regardless of the eligible farm to which they belonged.

The carcasses are numbered by routine in the slaughterhouse, and the corresponding stomachs were identified by the researcher with the same number for the purpose of this study. Each carcass is weighted and submitted to a fat-o-meat'er (FOM S71 - SFK Technology equipment) system to measure fat and muscle thickness on the carcass. With the identification of the stomachs it was possible to get information on gender, carcass weight and lean meat percentage of the corresponding carcass.

The stomachs of all the selected animals were opened along the greater curvature, emptied and inverted to allow the observation of the mucosa. The presence of ulcers and the local of the lesions in the stomach (*pars oesophagea*, cardiac, fundic and pyloric regions) were recorded.

A questionnaire was mailed to the veterinary responsible to obtain information about farm size, the existence of breeding animals, type of the diet, method of feeding, mixing of pigs, source of drinking water, ventilation, heating system and sanitary disinfection of the farm. The only characteristic that did not differ across farms was the type of diet. All animals were fed commercially prepared pelleted ration and finishing pigs were fed *ad libitum.*

The prevalence of gastric ulcers was computed for the whole sample, according to age and gender, and separately for animals from each farm. Quantitative variables

are presented as median, and 25 and 75 percentiles, and were partitioned into quartiles for comparisons across groups.

The association between characteristics of the farm (dimension of the farm, mixing pigs, feeding system, air ventilation, heating system, systematic sanitary disinfection, source of water) and the occurrence of *pars oesophagea* ulcers was quantified through Odds Ratios and 95% Confidence Intervals (95% CI), computed by unconditional logistic regression with robust standard errors (allowing for clustering by farm).

Sample size estimates were computed assuming the assessment of risk factors for *pars oesophagea* ulcers among finishing pigs as the primary objective. About 750 animals need to be evaluated to estimate an association corresponding to an Odds Ratio of 2, assuming a ratio of 6 animals without ulcers per each finishing pig with a *pars oesophagea* ulcer, a proportion of exposure among controls between 25% and 60%, and a design effect up to 1.1.

Data were analyzed using STATA®, version 9.2.

RESULTS

Characterization of the farms and of the selected animals

Tables 1 and 2 present the characteristics of the nine farms selected for this study and the characteristics of the samples available for analysis.

There are differences in management conditions and number of animals between farms. Farms A, B, C, D, E and F have breeding animals for reproduction and growing and fattening phases. Farms C and D mix their animals, farms A and F mix their animals only in fattening phase and farms B and E do not mix their animals at all.

Only farm A has manual feeding system in growing and finishing pigs, the others (B, C, D and E) have automatic system. Farms A and B feed their sows twice a

day with manual system, Farm C feeds their sows twice a day with automatic system and farms D, E and F feed their sows twice a day in gestation phase, and thrice a day, with automatic system, in lactation phase. Regarding the source of water, animals from farm A drink no chlorinated water from a mine, the animals from farm B drink chlorinated water from a well and animals from C, D, E and F drink pit chlorinated water. All theses farms have heating system in the maternity and growing phase and cooling system in maternity, growing and fattening phases. Sanitary disinfection is always made (all in – all out) in growing and fattening phase in farms B, D and E. In farms A, C and F the sanitary disinfection is always made only in growing phase.

Farms G, H and I only have growing and fattening systems, so there are no breeding pigs. The feeding system is manual, the source of water is a pit (no chlorination is done), there are no heating and cooling systems, and sanitary disinfection is always made when all animals get out to the slaughterhouse (system all in – all out).

Eight hundred and eighty seven animals entered this survey (760 commercial hybrids finishing pigs and 127 breeding animals). Among finishing pigs there are 41.7% of females and most of the breeding animals were females (90.5%).

Table 1. Characterization of the farms.

Farm	Number sows	Number Finishing pigs	Mixing animals	Feeding system (finishing pigs)	Feeding system (sows)	Feeding frequency (sows)	Air ventilation	Heating system	Systematic sanitary disinfection	Source of water
A	150	1400	Yes (finishing phase)	Manual	Manual	Twice a day	Mechanical	Yes *	Yes (only in growing phase)	Mine (not chlorinated)
B	120	1000	No	Automatic	Manual	Twice a day	Mechanical	Yes*	Yes (always)	Well (chlorinated)
C	120	1200	Yes (after weaning)	Automatic	Automatic	Twice a day	Mechanical	Yes*	Yes (only in growing phase)	Pit (chlorinated)
D	280	1800	Yes (after weaning)	Automatic	Automatic	Twice a day in pregnancy Thrice a day in lactation	Mechanical	Yes*	Yes (always)	Pit (chlorinated)
E	550	2500	No	Automatic	Automatic	Twice a day in pregnancy Thrice a day in lactation	Mechanical	Yes*	Yes (always)	Pit (chlorinated)
F	120	1500	Yes (finishing phase)	Automatic	Automatic	Twice a day in pregnancy Thrice a day in lactation	Mechanical	Yes*	Yes (only in growing phase)	Pit (chlorinated)
G	---	400	No	Manual	---	---	Natural	No	Yes (always)	Pit (not chlorinated)
H	---	440	No	Manual	---	**---**	Natural	No	Yes (always)	Pit (not chlorinated)
I	---	200	No	Manual	---	**---**	Natural	No	Yes (always)	Pit (not chlorinated)

* Only in growing phase and maternity

Table 2. Characterization of the samples from each farm.

	Stomachs examined			Carcass weight (Kg)			Females		
	N			Median (P25-P75)			%		
Farm	All animals	Finishing pigs	Breeding animals	All animals	Finishing pigs	Breeding animals	All animals	Finishing pigs	Breeding animals
A	109	80	29	76 (75-112)	68 (63-79)	173 (125-179)	63.3	55.0	86.2
B	96	80	16	77.5 (70-90.5)	75.5 (69-82)	167.5 (163-183)	43.7	35.0	87.5
C	84	60	24	78 (66-121.5)	72 (65-79.5)	138.5 (132-152)	53.6	36.7	95.8
D	177	140	37	78 (70-88)	75 (68-80)	150 (128-168)	52.5	41.4	94.6
E	234	220	14	71.5 (65-80)	70 (64-79)	125.5 (110-158)	44.9	42.7	78.6
F	67	60	7	74 (66-79)	71.5 (65.5-76)	192 (129-201)	46.3	40.0	100.0
G	60	60	0	76 (68-79)	76 (68-79)	---	40.0	40.0	---
H	20	20	0	79 (73.5-86)	79 (73.5-86)	---	60.0	60.0	---
I	40	40	0	74 (68-79.5)	74 (68-79.5)	---	27.5	27.5	---
Total	887	760	127	75 (67-84)	73 (66-80)	155 (129-176)	48.7	41.7	90.5

P25 – Percentile 25; P75 – Percentile 75.

Prevalence of gastric ulcer

The prevalence of *pars oesophagea* ulcer in the 887 animals from the 9 farms was 18.7%. All stomachs with ulcers had lesions in the *pars oesophagea*, but some of them had also ulcers in glandular region. One breeding animal had an ulcer in the cardiac region, two finishing pigs had an ulcer in the fundic region, and three cases of ulcers were observed in the pyloric region (one in finishing pigs and two in breeding animals). The overall prevalence of ulcers in the glandular region was 0.7%.

There was a statistically significant difference in the prevalence of *pars oesophagea* ulcers between finishing pigs and breeding animals (16.0% vs. 34.6%, $p<0.001$).

Table 3 summarizes the prevalence of ulcers found in all animals, by farm, showing statistically significant differences in the prevalence of *pars oesophagea* ulcers in finishing pigs across farms ($p<0.001$).

Table 3. Prevalence of *pars oesophagea* ulcer observed in pigs from 9 farms.

Farm	All animals	Finishing pigs	Breeding animals
	n(%)	n(%)	n(%)
A	29 (26.6)	18 (22.5)	11 (37.9)
B	39 (40.6)	33 (41.2)	6 (37.5)
C	12 (14.3)	10 (16.7)	2 (8.3)
D	35 (19.8)	20 (14.3)	15 (40.5)
E	25 (10.7)	20 (9.1)	5 (35.7)
F	13 (19.4)	8 (13.3)	5 (71.4)
G	6 (10.0)	6 (10.0)	---
H	4 (20.0)	4 (20.0)	---
I	3 (7.5)	3 (7.5)	---
Total	166 (18.7)	122 (16.0)	44 (34.6)

The highest prevalence of *pars oesophagea* ulcers in finishing pigs was observed in Farm B (41.2%) followed by farm A (22.5%).

Among breeding animals, there were also statistically significant differences ($p = 0.031$) in the prevalence of *pars oesophagea* ulcers between farms. The highest percentage of ulcers was observed in farm F (71.4%) followed by farm D (40.5%).

There were no statistically significant differences between male and female animals, neither in the group of the finishing pigs (males vs. females: 17.2% vs. 14.5%, $p=0.140$) or among breeding animals (males vs. females: 16.7% vs. 36.5%, $p=0.069$).

Association between farm characteristics and ulcers in finishing pigs and sows

The prevalence of ulcers according to the farm characteristics is presented in table 4.

Management conditions such as mixing pigs after weaning or before finishing phase, feeding system, type of air ventilation, existence of heating system or systematic sanitary disinfection were not significantly associated to the occurrence of *pars oesophagea* ulcers.

Farms with less than 1800 finishing pigs presented higher prevalence of ulcers than farms with more than 1800 finishing pigs ($OR_{<1800 \text{ finishing pigs vs. } \geq 1800 \text{ finishing pigs}} = 2.06$).

Finishing pigs that use water from mine or well had a significant higher prevalence of *pars oesophagea* ulcers than pigs that use water from pit ($OR_{\text{mine or well vs. pit}} = 3.49$). However, no significant differences were found in the prevalence of *pars oesophagea* ulcers between animals using chlorinated water and those receiving non-treated water. Animals from farms with no chlorinated water of mine had a significant higher prevalence of *pars oesophagea* ulcers than animals from farms with non-treated pit water ($OR_{\text{no chlorinated water mine vs. no chlorinated water pit}} = 2.39$). Farms where animals used chlorinated water from well had a significant higher prevalence of *pars oesophagea* ulcers than those from farms with chlorinated pit water ($OR_{\text{chlorinated well vs. chlorinated pit}} = 5.11$).

Table 4. Association between farm characteristics and the occurrence of *pars oesophagea* ulcers in finishing pigs.

CHARACTERISTICS OF FARM	**Animals having *pars oesophagea* ulcers n (%)**	**OR (95%CI)**
Dimension of the farm		
≥ 1800 finishing pigs	40 (11.1)	1 [reference]
< 1800 finishing pigs	82 (20.5)	2.06 (1.01-4.21)
Mixing pigs		
Never	66 (15.7)	1 [reference]
After weaning	30 (15.0)	0.95 (0.36-2.47)
Only in finishing phase	26 (18.5)	1.22 (0.43-3.49)
Feeding system		
Manual	31 (15.5)	1 [reference]
Automatic	91 (16.2)	1.06 (0.43-2.62)
Air ventilation		
Natural	13 (10.8)	1 [reference]
Mechanical	109 (17.0)	1.69 (0.8-3.55)
Heating system		
Absent	13 (10.8)	1 [reference]
In growing phase	109 (17.0)	1.69 (0.8-3.55)
Systematic sanitary disinfection		
Always	86 (15.4)	1 [reference]
Only in growing phase	36 (18.0)	1.21 (0.55-2.68)
Source of water		
Pit	71 (11.8)	1 [reference]
Mine or well	51 (31.9)	3.49 (1.75-6.94)
Chlorinated water		
No	31 (15.5)	1 [reference]
Yes	91 (16.2)	1.05 (0.42-2.62)
No chlorinated water		
Pit	13 (10.8)	1 [reference]
Mine	18 (22.5)	2.39 (1.52-3.76)
Chlorinated water		
Pit	58 (12.1)	1 [reference]
Well	33 (41.2)	5.11 (3.65-7.14)

OR – Odds Ratio; 95% CI – 95% Confidence Interval

Similarly to the observed for finishing pigs, no significant association was observed between the conditions in which breeding animals were raised and the occurrence of ulcers in the *pars oesophagea* (table 5). Sows fed thrice a day during lactation had twice the risk of *pars oesophagea* ulcers compared to those always fed twice a day, though this association was not statistically significant.

Table 5. Association between farm characteristics and the occurrence of *pars oesophagea* ulcers in sows.

CHARACTERISTICS OF FARM	***pars oesophagea* ulcers n (%)**	**OR (95%CI)**
Dimension of the farm		
≥ 1800 finishing pigs	19 (41.3)	1 [reference]
< 1800 finishing pigs	23 (33.3)	0.71 (0.28-1.82)
Feeding system		
Manual	16 (41.0)	1 [reference]
Automatic	26 (34.2)	0.74 (0.3-1.83)
Feeding frequency:		
Twice a day	18 (29.0)	1 [reference]
Twice a day except during lactation (thrice daily)	24 (45.3)	2.0 (0.72-5.7)
Source of water:		
Pit	26 (34.2)	1 [reference]
Mine or well	16 (41.0)	1.34 (0.55-3.27)
Chlorinated water		
No	10 (40.0)	1 [reference]
Yes	32 (35.6)	0.83 (0.39-1.75)
Chlorinated water:		
Pit	26 (34.2)	1 [reference]
Well	6 (42.9)	1.44 (0.58-3.58)

OR – Odds Ratio; 95% CI – 95% Confidence Interval

Association between the occurrence of ulcers and carcass weight and lean meat percentage, in finishing pigs and in breeding animals

There were no statistically significant differences in the prevalence of *pars oesophagea* ulcers for different categories of carcass weight, neither in finishing pigs (tables 6) or breeding animals (tables 7), and similar results were observed for lean meat percentage in finishing animals (table 6).

Table 6. Association between *pars oesophagea* ulcers and carcass weight and lean meat percentage in finishing pigs.

	***pars oesophagea* ulcers n (%)**	**OR (95%CI)**
Carcass weight (Kg)*		
0-66	34 (27.9)	1 [reference]
67-73	30 (24.6)	1.12 (0.63 -1.98)
74-80	28 (22.9)	0.82 (0.29 - 2.29)
81-120	30 (24.6)	1.14 (0.55 - 2.36)
Lean meat (%) †		
0-56.3	21 (13.4)	1 [reference]
56.4-58.0	23 (14.9)	1.14 (0.45 - 2.86)
58.1-59.4	21 (13.5)	1.01 (0.37 - 2.75)
59.5-100	33 (22.0)	1.82 (0.54 - 6.12)

OR – Odds Ratio; 95% CI – 95% Confidence Interval

* Cut points to define categories of carcass weight were quartiles of the distribution among finishing pigs

† Cut points to define categories of lean meat percentage were quartiles of the distribution among finishing pigs

Table 7. Association between *pars oesophagea* ulcers and carcass weight in breeding pigs.

	***pars oesophagea* ulcers n (%)**	**OR (95%CI)**
Carcass weight (Kg)*		
0-129	10 (31.2)	1 [reference]
130-155	10 (31.2)	1.00 (0.20 - 4.95)
156-176	11 (32.3)	1.05 (0.29 - 3.74)
177-220	13 (44.8)	1.79 (0.70 - 4.53)

OR – Odds Ratio; 95% CI – 95% Confidence Interval

* Cut points to define categories of carcass weight were quartiles of the distribution among breeding pigs.

DISCUSSION

The overall prevalence of ulcers in *pars oesophagea* was 18.7%, being nearly two-fold higher in breeding animals than in finishing pigs. The overall prevalence of ulcers in the glandular region was 0.7%. Finishing pigs from farms with mine/well water supply had thrice the risk for *pars oesophagea* ulcers than those from farms with pit water supply, but there were no significant differences according to gender, carcass weight/back-fat, or other managing conditions of the farms than water origin.

The limitations of this study should be recognized. This study was based solely on animals of one slaughterhouse selected by convenience of the researcher. This could contribute to a lower external validity of the prevalence estimates, but it should be taken into account that even in this convenience sample we observed a wide range of prevalence estimates. On the other hand, the estimates for the association between animal and farm characteristics at the occurrence of gastric ulcers in swine is not expected to be biased, although a larger variability of exposures across farms could contribute to disclose other associations.

In this study, 16.0% of finishing pigs presented *pars oesophageal* ulcers, a higher prevalence than the observed in Spain, 11.6%, (Ramis *et al.*, 2004), Italy, 12.6%, (Appino *et al.*, 2006), United Kingdom, 13.4%, (Guise *et al.*, 1997) or Burkina Faso, 10% (Banga-Mboko *et al.*, 2003). A recent study in Australia, 31%, (Kopinski and McKenzie, 2007) found a higher prevalence than in our study, and similar estimates had been found in Canada, 15.5%, (Melnichouk, 2002) and USA, 16.4%, (Eisemann *et al.*, 2002).

The proportion of animals with ulcers in the glandular region was higher than the observed by Bivin *et al.* in 1974, 0.19%, and lower than in the report by Muggenburg *et al.* in 1964(b), 2.1%, although the lack of precision of the estimates, the time span between these investigations, and the heterogeneity of the sampling procedures precludes direct comparisons between these studies.

The *pars oesophagea* ulcers were significantly more frequent in breeding animals than in finishing pigs, despite the limited number of sows recruited in our study, in accordance to the observed by Robertson *et al.* (2002) in Australia, although Muggenburg *et al.* (1964b) found no difference in the prevalence of gastric lesions between culled sows and gilts. These two studies are the only comparing finishing pigs and breeding animals directly.

In the present study, the absence of difference in the frequency of *pars oesophagea* ulcers between males and females of similar age is in accordance with previous reports (Curtin *et al.*, 1963 and Muggenburg *et al.*, 1964b Mahan *et al.*, 1966; Flatlandsmo and Slagsvold, 1971; Elbers *et al.*, 1995; and Robertson *et al.*, 2002).

There was a wide variation in the prevalence of *pars oesophagea* ulcer across farms, both for finishing pigs and breeding animals, suggesting that differences in the management of swine could contribute to the development of *pars oesophagea* ulcers to different extents. A similar finding was reported by Muggenburg *et al.* (1964b), showing significant differences in the percentage of gastric ulcers between two farms from the University of Wisconsin. Notwithstanding some management differences between these two farms no factors were identified to explain the difference in prevalence of the gastric ulcers. In Australia, Robertson *et al.* (2002) observed differences in the prevalence of oesophago-gastric ulcer between herds and in distinct Australian states. The prevalence of oesophago-gastric ulcer in state of Victoria (53%) was significantly higher than in Western Australia (30%) or Queensland (7%). These results also suggest that the management and nutritional practices may explain the differences observed across settings, but their data did not allow the quantification of the association between risk factors inside the herds of different states and the occurrence of gastric ulcers in pigs.

In the present study, the smaller farms had higher prevalence of *pars oesophagea* ulcers than the larger ones, which is in contradiction with the study by Ramis *et al.* (2004). A possible explanation for our results is the fact that the two farms

that had higher prevalence of *pars oesophagea* ulcer in finishing pigs (A and B) are included in the group of the smaller farms due to the cut-off arbitrarily selected for this analysis.

The effect of mixing pigs after weaning or before finishing phase, feeding system, type of air ventilation, heating system, systematic sanitary disinfection are not significantly associated with the occurrence of ulcers in the *pars oesophagea* region. Regarding the feeding system, Robert *et al.* (1991) showed higher frequency of gastric ulcers in restricted-fed pigs, otherwise Robertson *et al.* (2002) reported that pigs fed *ad libitum* had higher prevalence of oesophago-gastric ulcer. Curtin *et al.* (1963), Riker *et al.* (1967) reported that animals living in ambient with temperature fluctuations had higher prevalence of gastric lesions than animals living in constant temperature, but Amory *et al.* (2006) found a higher prevalence of gastric ulcers in pigs with controlled environment than in natural environment. In the study by Robertson *et al.* (2002) the type of air ventilation or the existence/absence of heating system were not associated with a high herd prevalence of gastric ulcers, as in the present investigation.

The influence of the source of water on development of oesophago-gastric ulcers was reported by Robertson *et al.* (2002). They mentioned that water from different sources might have different pH values, buffering abilities and different microbiological quality but in their study the quality of water was not assessed. In our study, three source of water were identified: mine, well and pit. The water from mine was not treated with filtration and using chlorates, all the farms with well had chlorinated water and not all farms with pit had chlorinated water. In the group of finishing pigs, animals receiving water from mine or well had thrice the risk of ulcers in *pars oesophagea* than pigs that drank water from a pit. We may speculate that is due to the contamination of the water with microorganisms in mine and well because the water is less deep than water from a pit. The water from a pit may be less contaminated because water is deeper. However, in the farm with the highest prevalence of ulcers in finishing pigs (farm B) the animals received treated well water.

This could be compatible with our previous hypothesis if the treatment of the water could not decrease microbiological charge in the water to levels low enough. Farm A, in which the second highest prevalence of *pars oesophagea* ulcers was observed, the source of water was a mine, and no chlorination was done. As in humans, the colonization of the stomach for organisms of the Helicobacter-type is currently more accepted and connected with the development of gastric injuries in pigs (Barbosa *et al.*, 1995; Queiroz *et al.*, 1996; Roosendal *et al.*, 2000; Choi *et al.*, 2001; Appino *et al.*, 2006). Our results could be explained if water is a possible source of Helicobacter species that can infect pigs.

We did not find any association between the characteristics of the breeding animals or their conditions in the farm and the development of the ulcers in *pars oesophagea*. Although there are significant differences in prevalence of *pars oesophagea* ulcer of the breeding animals between farms, in this study it was not possible to disclose which factors are responsible for these differences. An interesting result was found in sows fed thrice a day in lactation phase, which had a higher prevalence of *pars oesophagea* ulcers than sows fed only twice a day in the same phase, but this difference is not statistically significant. These results may be seen as suggestive of an association between the frequency of feeding and the occurrence of ulcers.

Diet is the factor most consistently associated with the occurrence of gastric ulcers, but in this study all animals were fed pelleted ration. This fact suggests two interpretations for our results: the diet is not the only major risk factor or pelleted diet of each farm had variations on its composition (differences in type of grain) that can prevent the appearance of this disease.

In Europe, it is considered that the efficient production of pork is better obtained by a combination of quick, thin growth and low deposition of fat. The production of good quality meat can be obtained reducing the total quantity of fat by genetic selection of the pigs, whereas deposits of fat in balanced levels is important for the quality of meat.

Berruecos and Robison (1972) found a negative association between the presence of ulcer in *pars oesophagea* and selection against back-fat. These researchers reported a relationship between low back-fat and a high prevalence of gastric ulcer. In our study, there is no association between carcass weight/lean meat percentage and the development of ulcers.

In the present investigation it was not possible to known the genetic origin of the commercial hybrids finishing pigs and breeding animals, however, the genetic origin could be an important risk factor to the gastric ulcer in pigs. Studies addressing the relationship between genetic background and gastric lesions did not yield consistent results to prove such an association (Curtin *et al*., 1963; Mahan *et al*., 1966; Elbers *et al*., 1995). The possible reason that no studies demonstrated any association between genetic origin and the occurrence of gastric ulcer in swine is the early slaughter of the pigs that does not allow the expression of the genetic susceptibility to ulcers.

CONCLUSION

The prevalence of gastric ulcer in this survey is in the upper range of the observation in other countries. Our results suggest that the source of water may be important risk factor to the development of ulcers in *pars oesophagea* but the quality of the water was not evaluated. Further investigation about risk factors in the development of *pars oesophagea* ulcers must evaluate the quality of drinking water carefully.

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5. ABSTRACT AND CONCLUSIONS

Introduction

Gastric ulceration is the most common pathology observed in the pigs' stomachs and can be an important cause of death in certain herds. Economics and welfare concerns justify the monitoring of swine populations to determine the prevalence and severity of gastric lesions.

The prevalence of gastric ulcer in swine is unknown in Portugal and, apart from nutritional factors, its determinants are poorly understood. Therefore, we aimed to quantify the prevalence of gastric ulcers in swine and the association between animal and farm characteristics and the occurrence of these lesions.

Methods

Finishing pigs (n=760), approximately 6 months old, and culled breeding animals (n=127), aged one year or more, were randomly selected at a slaughterhouse in the North of Portugal, among the animals from 9 previously specified farms. Their stomachs were visually evaluated for the presence of ulcers. Information on gender, carcass weight, lean meat percentage, and farm/managing characteristics were obtained through the slaughterhouse records or inquiry to the veterinarians from each farm.

The prevalence of gastric ulcers was computed for the whole sample, according to age and gender, and separately for animals from each farm.

The association between farm characteristics (dimension of the farm, mixing pigs, feeding system, air ventilation, heating system, systematic sanitary disinfection, source of water) and the occurrence of *pars oesophagea* ulcers was quantified through Odds Ratios and respective 95% Confidence Intervals (95% CI), computed by unconditional logistic regression with robust standard errors (allowing for clustering by farm)

Data were analyzed using STATA®, version 9.2.

Results

The overall prevalence of ulcers in *pars oesophagea* was 18.7%, significantly higher in breeding animals than in finishing pigs (34.6% vs. 16.0%, $p<0.001$), and a wide variation was observed across farms (range: 7.5%-41.2% for finishing pigs, 8.3%-71.4% for breeding animals). The overall prevalence of ulcers in the glandular region was 0.7%.

Finishing pigs from farms with mine/well water supply had a higher prevalence of *pars oesophagea* ulcers than those from farms with pit water supply (OR=3.49, 95% CI: 1.75-6.94).

There was no significant association between gender, carcass weight/back-fat, or managing conditions of the farms (*e.g.* mixing pigs after weaning or before finishing phase, feeding system, type of air ventilation, presence or not of heating system, systematic sanitary disinfection) and the prevalence of *pars oesophagea* ulcers in finishing pigs.

Conclusion

The prevalence of gastric ulcer in this survey is in the upper range of the observation in other countries. Our results suggest that the source of water may be important risk factor to the development of ulcers in *pars oesophagea.*

6. RESUMO E CONCLUSÕES

Introdução

A ulceração gástrica é uma patologia frequentemente observada nos estômagos dos suínos e pode ser uma importante causa de morte em algumas explorações. Os prejuízos económicos e bem-estar animal justificam o estudo da frequência e gravidade das lesões gástricas em suiniculturas, assim como os seus determinantes.

A prevalência da úlcera gástrica em suínos é desconhecida em Portugal e, à excepção de factores nutricionais, os outros determinantes da doença são pouco claros. O objectivo deste estudo é quantificar a prevalência de úlceras gástricas na espécie suína e determinar a associação entre características dos animais e das explorações e a ocorrência de tais lesões.

Métodos

Foram incluídos neste estudo suínos de engorda híbridos (n=760), com aproximadamente 6 meses de idade, e suínos reprodutores (n=127), com idade igual ou superior a um ano. Os suínos foram seleccionados aleatoriamente num matadouro no Norte de Portugal, entre os animais provenientes de 9 explorações previamente especificadas. Os estômagos dos suínos foram avaliados visualmente com o objectivo de identificar úlceras. As informações sobre sexo, peso de carcaça, percentagem de carne magra e as características de maneio nas explorações foram obtidas por consulta dos registos do matadouro ou por inquérito aos médicos veterinários de cada exploração.

A prevalência de úlceras gástricas foi calculada para toda a amostra, de acordo com a idade e sexo, e separadamente para os animais de cada exploração.

A associação entre as características das explorações (dimensão das suiniculturas, mistura de lotes de animais, sistema de alimentação, existência de aquecimento/arrefecimento dos pavilhões, desinfecção sanitária sistemática, fonte de água) e da ocorrência de úlceras na região *pars oesophagea* foi quantificada através

de Odds Ratio e respectivos intervalos de confiança a 95% (IC a 95%), calculados por regressão logística não condicional com erros padrão robustos (considerando cada exploração como um conglomerado).

Os dados foram analisados usando o STATA ®, versão 9.2.

Resultados

A prevalência total de úlceras na *pars oesophagea* foi de 18,7%, significativamente maior nos animais reprodutores (34,6% vs. 16,0%, p <0,001), e observou-se uma grande variação das prevalências entre explorações (entre 7,5% e 41,2% para suínos de engorda e entre 8,3% e 71,4% para os animais reprodutores). A prevalência de úlceras na região glandular foi de 0,7%.

Os animais de engorda provenientes de explorações em que o abatecimento de água é efectuado através de mina ou poço apresentaram uma maior prevalência de úlceras na *pars oesophagea* do que os suínos de engorda provenientes de explorações com abastecimento de água através de furo de captação própria (OR = 3,49, IC 95% : 1.75-6.94).

Não se observou associação significativa entre sexo, peso de carcaça / percentagem de carne magra, condições de maneio das explorações (*e.g.* mistura de lotes de suínos após o desmame ou antes da fase de engorda, sistema de alimentação, existência do sistema de aquecimento/arrefecimento, desinfecção sanitária sistemática) e a prevalência de úlceras na região *pars oesophagea* em suínos de engorda.

Conclusão

Concluímos que a prevalência da úlcera gástrica é alta nos suínos nacionais. Os nossos resultados sugerem que a fonte de água pode ser um importante fator de risco para o desenvolvimento de úlceras na *pars oesophagea* em suínos.