



REVIEW ARTICLE

GASTRIC LESIONS IN DOMESTIC PIGS AND WILD BOARS: ETIOLOGY, PATHOLOGY AND PREDISPOSING FACTORS

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Ethical considerations: When reporting experiments on animals Observation of the ARRIVE guidelines 2.0: Updated guidelines for reporting animal research, published on July 14, 2020 (DOI: 10.1371/journal.pbio.3000410), is applied. The authors ensure that all procedures were performed in compliance with the guidelines for animal care of their institutions or with national/international guidelines.

ABSTRACT

Gastric lesions represent a significant health concern in domestic pigs and, to a lesser extent, in wild boars. Their development reflects a multifactorial interaction of dietary characteristics, stress exposure, microbial balance, and infection with *Helicobacter* spp. Domestic pigs show a high prevalence and severity of lesions, primarily in the *pars oesophagea*, driven by finely ground low-fibre feed, intensive housing, and dysbiosis. In contrast, wild boars typically display mild, often asymptomatic lesions associated with their fibre-rich diet, natural social structure, and stable microbiota. This review compares etiological factors and pathological features across both populations and highlights implications for prevention. Special attention is given to the ecology and zoonotic relevance of *Helicobacter suis*. Understanding these mechanisms is essential for optimizing nutritional strategies, minimizing stress, and mitigating risks of interspecies transmission.

Key words: domestic pigs; gastric ulcers; *Helicobacter suis*; microbiota; pathology; prevention; stress; wild boars

INTRODUCTION

Gastric lesions in pigs constitute an important welfare and productivity issue, with consequences ranging from reduced growth rates to sudden death [1, 2]. Domestic pigs are particularly vulnerable due to the combined effects of intensive management, low-structural diets, and repeated exposure to stress [3, 4]. Anatomically, the non-glandular *pars oesophagea*, lined by stratified squamous epithelium

and lacking mucous protection, represents the primary site affected by parakeratosis, erosions, and ulceration [5, 6].

Recent research has highlighted the value of comparing domestic pigs with wild boars, which share the same species lineage but differ markedly in diet, environment, and microbiota [7, 8]. Wild boars consume diverse, fibre-rich natural diets and experience lower chronic stress, factors that influence gastric physiology and markedly reduce ulcer severity [9, 10]. They also serve as reservoirs for sev-

eral *Helicobacter* species, including *H. suis*, *H. apri*, and *H. pylori*, with implications for animal and human health [11, 12].

This review synthesizes etiological, pathological, microbial, and zoonotic aspects of gastric lesions in domestic pigs and wild boars to identify key mechanisms and inform prevention strategies.

LITERATURE SEARCH STRATEGY

Relevant literature was identified through PubMed, Scopus, Web of Science, and Google Scholar using combinations of the terms: gastric ulcers, *pars oesophagea*, *Helicobacter suis*, wild boar, domestic pig, gastric microbiota, and porcine stomach pathology. Publications from 1995 to 2025 were considered, with emphasis on peer-reviewed articles. Additional sources were obtained from the bibliographies of selected papers. Studies describing etiology, risk factors, microbiology, diet, pathology, or comparative aspects between domestic pigs and wild boars were included.

ETIOLOGICAL FACTORS

Feed structure and dietary composition

Feed particle size is one of the strongest determinants of gastric lesion development. Finely ground or pelleted diets reduce gastric content stratification, allowing acidic digesta to reach the *pars oesophagea* and initiate epithelial damage [13, 14]. Low-fibre diets further enhance acidity and reduce mucosal resilience, whereas coarse or fibre-rich feeds promote buffering and protective layering [15, 16, 17]. High-fibre feeding enhances digesta layering, reduces acid back-flow, and supports beneficial microbiota [7, 15].

Although the importance of feed structure in gastric ulcer development is well established, the role of dietary composition remains less clearly defined. Several studies indicate that cereal type, fibre level, feeding regime, and the inclusion of organic acids or selected micronutrients may modulate ulcer risk; however, the reported effects remain inconsistent and highly context-dependent [3, 14, 16, 18].

Wild boars consume structurally rich natural feeds: roots, fruits, vegetation, and insects, supporting digesta separation and reducing acid contact [7, 19]. Seasonal di-

etary changes alter gastric pH and microbiota but rarely lead to severe lesions [8]. These mechanisms clarify why domestic pigs, exposed to intense production pressures, consistently develop more severe lesions compared to wild boars. Environmental and nutritional constraints, rather than inherent anatomical susceptibility, account for the contrasting pathological outcomes [3, 20].

Stress and environmental conditions

Chronic stress in domestic pigs results from high stocking density, regrouping, transport, and competition for feed and space. These factors activate endocrine pathways that increase gastric acidity, reduce mucosal perfusion, and impair tissue repair [21, 22]. Stress also disrupts microbial balance and enhances susceptibility to ulceration. Stress-induced hyperacidity decreases epithelial repair capacity, further compromising gastric mucosal integrity [13, 14, 18, 23].

Wild boars, in contrast, live in stable social groups, display natural behaviour, and maintain continuous movement, all of which support gastric motility and mucosal integrity [9, 10].

Gastric microbiota and microbial dysbiosis

A stable microbiota plays a crucial role in maintaining gastric homeostasis. Domestic pigs frequently exhibit reduced *Lactobacillus* spp. and increased pro-inflammatory genera such as *Fusobacterium* [23, 24]. These shifts facilitate inflammation, compromise the mucus barrier, and predispose mucosa to erosion. Dysbiosis weakens mucosal protection and interacts synergistically with dietary factors and stress to increase ulcer risk [13, 18].

Wild boars generally maintain a stable microbiota, which supports gastric resilience and contributes to the typically mild or asymptomatic lesions observed in this population [7, 10].

Helicobacter suis is widely recognized as a significant pathogen in pigs, inducing chronic gastritis, epithelial alteration, and enhanced sensitivity to concurrent stressors [25, 26]. Experimental infection confirms its capacity to elicit mucosal inflammation and impair growth [27]. Wild boars often harbour *H. suis*, *H. apri*, and occasionally *H. pylori* with minimal pathology, acting as natural reservoirs [11, 28].

These observations highlight the significance of *Helicobacter* spp. in both domestic pigs and wild boars and

set the stage for considering their potential zoonotic relevance, as discussed below.

***Helicobacter* spp. and zoonotic aspects**

Helicobacter suis is the primary *Helicobacter* species in pigs but also infects humans, where it is linked to chronic gastritis and more severe gastric disorders [29, 30]. Genetic analyses reveal similarities between strains recovered from pigs, wild boars, and human patients, supporting zoonotic transmission potential [11, 31].

Wild boars play a key ecological role by maintaining a reservoir of diverse *Helicobacter* species [28, 32], increasing the relevance of wildlife-livestock interfaces for disease control. Biosecurity and monitoring are therefore critical in mixed-use landscapes.

Key differences in etiological factors, pathogenic mechanisms, and resulting gastric pathology between domestic pigs and wild boars are summarized in Tables 1 and 2.

COMPARATIVE PATHOLOGY OF DOMESTIC PIGS AND WILD BOARS

Domestic pigs

Lesions in domestic pigs predominantly involve the *pars oesophagea*, with pathology ranging from subclinical hyperkeratosis to deep, hemorrhagic ulceration [15, 33, 34]. Subacute lesions present as thickened, roughened

epithelium due to parakeratosis [5]. Erosion progresses to crateriform ulcers penetrating submucosa, often accompanied by necrotic debris and clotted blood [35]. Sudden death may occur when ulcers erode sub-mucosal arteries [36] (Fig. 1).

Microscopically, lesions reveal epithelial necrosis, inflammatory infiltrates of lymphocytes and neutrophils, vascular dilation, and mucin depletion [28, 33]. *Helicobacter suis* infection induces lymphoplasmacytic gastritis, epithelial atrophy, and mucus barrier disruption, often in combination with *Fusobacterium gastrosuis* [37].

Clinical signs include pale mucosae, reduced feed intake, lethargy, decreased weight gain, and, in chronic cases, iron-deficiency anaemia.

Wild boars

Wild boars generally exhibit mild gastric lesions characterized by superficial hyperkeratosis or limited erosions [12, 38] (Fig. 2, Fig. 3). Deep ulceration and hemorrhage are rare, and histological inflammation is minimal [39]. Their high-fibre diet and stable microbiota promote mucosal resilience [10], while natural activity patterns support gastric motility and perfusion.

Despite their carrier status for multiple *Helicobacter* spp., wild boars seldom show extensive glandular pathology, underscoring the importance of environmental moderation of disease expression [11].

Table 1. Comparison of the etiology and pathogenesis of gastric lesions in domestic pigs and wild boars (authors' own table)

Factor	Domestic pigs	Wild boars
Primary type of feed	Finely ground/pelleted diet, low fibre content	Diverse natural diet, high fibre content
Effect of feed on <i>pars oesophagea</i>	Increased digesta mixing → acidic content contacts epithelium → parakeratosis, erosions	Thicker digesta layer → reduced acid backflow
Aggressive factors	Acid, pepsin, mechanical abrasion, stress-induced hyperacidity	Mainly seasonal changes in diet and energy intake
Protective factors	Weaker → low fibre, stress, rapid growth, intensive production	Stronger → fibre, stable microbiota, lower stress
<i>Helicobacter</i> spp.	<i>H. suis</i> → major pathogen; synergizes with <i>Fusobacterium gastrosuis</i>	<i>H. suis</i> , <i>H. apri</i> , <i>H. pylori</i> → mostly asymptomatic
Microbiota	Affected by dysbiosis with finely ground feed → reduced <i>Lactobacillus</i>	Stable, diverse, adapted to high fibre
Key trigger of ulceration	Fine particle size + stress + <i>H. suis</i>	Seasonal dietary changes, rarely pathogenic bacteria
Ulcer prevalence	High, mainly in finishing pigs	Low, mostly mild lesions
Clinical course	Often peracute, silent bleeding, sudden deaths	Mostly latent, clinically silent
Severity of pathology	Moderate to high	Low

Table 2. Comparison of gastric pathology, risk factors, and predisposing conditions in domestic pigs and wild boars (authors' own table)

Parameter	Domestic pigs (<i>Sus scrofa domesticus</i>)	Wild boars (<i>Sus scrofa</i>)
Lesion prevalence	High: 30–70%, mainly in <i>pars oesophagea</i>	Low: 5–10%, mostly mild erosions or hyperkeratosis
Most common location	<i>Pars oesophagea</i> – deep erosions and ulcers	<i>Pars oesophagea</i> – mainly superficial changes
Typical pathologies	Parakeratosis, deep ulcerations, bleeding, scarring	Mild parakeratosis, superficial erosions, rarely ulcerations
Diet	Finely ground or pelleted feed, low fibre content, monodiet	Diverse natural diet: roots, fruits, fibre, insects, vegetation
Nutritional effects	High risk: low structural fibre → higher acidity in the cardia	Protective: fibre → more stable digestion → less acid–epithelium contact
Microbiota	Lower diversity, frequent dysbiosis; reduced <i>Lactobacillus</i> , increased <i>Fusobacterium</i>	Higher diversity, more stable microbiota; lower dysbiosis risk
<i>Helicobacter</i> spp.	<i>H. suis</i> commonly pathogenic → chronic gastritis, ulcerations, dysbiosis	Frequent carriers of <i>H. suis</i> , <i>H. apri</i> , <i>H. pylori</i> – mostly asymptomatic
Zoonotic potential	Infected populations may transmit <i>H. suis</i> to humans	Reservoir of <i>Helicobacter</i> spp., high zoonotic relevance
Stress factors	High: stocking density, transport, handling, hierarchy, low enrichment	Low to moderate: natural environment, free movement
Effect of stress on stomach	Increased cortisol → hyperacidity → deep ulcers → higher risk	Lower stress → more stable mucosal barrier
Role of genetics	Selected for high growth → may be more susceptible	Less selected, more robust digestion
Clinical signs	Often hidden → sudden death possible due to bleeding; anemia, melena, weakness	Mostly asymptomatic, rare clinical signs
Economic impact	Significant: reduced growth, poor feed conversion, mortality	Minimal (mainly in wild populations)
Environmental factors	Intensive housing → limited movement → higher stress	Forest environment → natural behaviour, seasonal dietary shifts
Protective factors	Coarse fibre, probiotics, good stress management	Natural diet, low stress, microbial diversity

IMPLICATIONS FOR PREVENTION AND MANAGEMENT

Preventive strategies must consider the interplay of diet, stress, microbiota, and infection pressure. Increasing structural fibre, reducing excessive feed grinding, maintaining consistent feeding routines, and ensuring gradual dietary transitions significantly lower ulcer risk [40, 41, 42]. Stress reduction through stable group management, minimized transport, adequate space, bedding, and enrichment improves mucosal protection [22, 43].

Microbiota-supporting interventions, including probiotics, fermented feeds, and organic acids, can reinforce gastric stability [44]. Monitoring and controlling *H. suis* infections remain essential, particularly in herds with recurrent ulcer problems [24].

Economically, gastric lesions contribute to decreased growth, poorer feed conversion, and sudden mortality in domestic pigs, supporting the integration of nutritional and

welfare-based prevention within herd health planning [3, 45].

Preventive approaches targeting nutrition, stress reduction, microbiota stabilization, and infection control in domestic pigs and wild boars are summarized in Table 3.

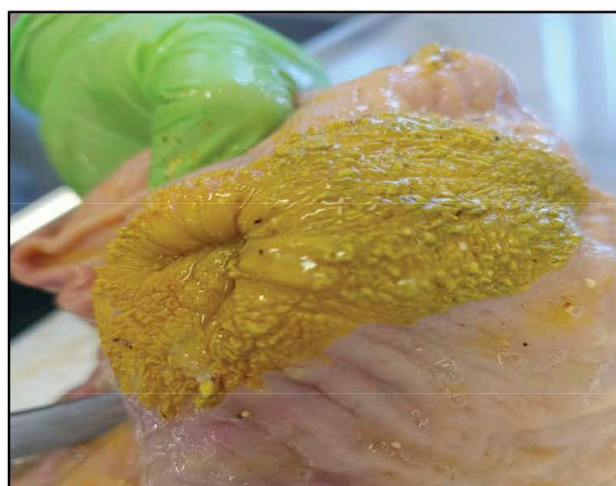
CONCLUSIONS

Gastric lesions in domestic pigs and wild boars arise from the interaction of nutritional, microbial, environmental, and host-related factors. Domestic pigs are predisposed to severe *pars oesophagea* lesions due to finely ground low-fibre diets, high stress exposure, and microbiota instability, often compounded by *H. suis* infection. Wild boars, by contrast, typically develop mild lesions associated with their natural diet, low stress levels, and stable gastric microbiota.

Comparative evaluation highlights that management and dietary structure, more than species-related anatomy,



Score 0 – healthy *pars oesophagea*



Score 1 - parakeratosis



Score 2 - erosions



Score 3 – ulceration

Fig. 1. Lesion score of the gastric mucosa in a finishing pig from 0 to 3
Source: (authors' own photographs)



Fig. 2. Stomach of a wild sow and yearling with healthy mucosa – score 0
Source: (authors' own photographs)



Fig. 3. Stomach of a wild boar (yearling) – glandular (fundic) region with erosions present

Source: (authors' own photographs)

Table 3. Preventive strategies – comparison of domestic pigs and wild boars (authors' own table)

Prevention area	Domestic pigs	Wild boars
Nutrition and feeding	- Increase fibre content (wheat bran, beet pulp) - Use coarser particle size - Avoid extremely fine grinding - Consistent feeding and gradual feed transitions	No intervention – natural diet provides sufficient structure and fibre
Management	- Minimize stress (transport, regrouping) - Optimize stocking density - Improve bedding and environmental enrichment	Not applicable (natural conditions)
Microbiota	- Probiotics (<i>Lactobacillus</i> spp.) - Fermented feed - Support SCFA production - Monitoring	Stable microbiota → minimal need for intervention
Control of <i>H. suis</i> infection	- Farm hygiene - Age group separation	Natural reservoir; interventions only feasible in farmed wild boars
Seasonal risks	Low	High → fluctuations in diet may alter pH and microbiota

determine gastric outcomes. Future research should focus on:

- (1) characterizing gastric microbiota composition and seasonal variability in wild boars;
- (2) clarifying host–pathogen interactions involving *Helicobacter* spp.;
- (3) developing targeted feeding and welfare practices to reduce ulcer prevalence; and

- (4) assessing zoonotic transmission risks at wildlife–livestock interfaces.

Comprehensive integration of nutritional strategies, stress mitigation, microbial monitoring, and biosecurity offers the most effective pathway to reducing ulcer incidence while improving welfare and production efficiency in domestic pigs.

Ethical Statement

No Ethical Approval was necessary for this study.

Conflict of Interest

The authors declare no conflicts of interest.

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Generative AI statement

The authors declare that no generative AI or AI-assisted technologies were used in the writing of this manuscript.

Authors' Contributions

ZK – Conceptualization, Methodology, Literature review, Writing – original draft, Writing – review and editing, Supervision.

JN – Conceptualization, Methodology, Investigation, Writing – original draft, Writing – review and editing, Funding acquisition, Supervision.

KB – Literature review, Writing – original draft, Visualization.

JČ – Contribution to wildlife ecology section, Writing – review and editing.

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