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Short communication

Gastric pH and *Fusobacterium gastrosuis* detection in relation to gastric lesions in fattening pigs

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Abstract

The development of gastric ulcers in fattening pigs is a multifactorial process influenced by gastric acidity and bacterial colonization. The aim of this study was to investigate the relationship between gastric pH and the presence of *Fusobacterium gastrosuis* in different regions of the stomach in relation to ulcer formation. Gastric pH in the *pars oesophagea* was measured in 112 fattening pigs and showed a negative correlation with lesion severity. Qualitative real-time PCR detected *F. gastrosuis* in 70% of samples from the glandular region and in 50% of *pars oesophagea* samples, with detection based on the 16S rRNA gene being more sensitive than gyrB. Although ulcers occurred predominantly in the *pars oesophagea*, colonization by *F. gastrosuis* was more pronounced in the glandular region of the stomach, suggesting that the bacterium may act as an opportunistic pathogen and, in combination with low gastric pH, indirectly contribute to mucosal damage. These findings support the hypothesis of a multifactorial pathogenesis of gastric ulcers in pigs and highlight the importance of combined monitoring of gastric pH and bacterial colonization in ulcer prevention.

Keywords: *Fusobacterium gastrosuis*, gastric ulcers, pigs, gastric microbiota

Introduction

Gastric ulcers are a common condition in pigs, predominantly affecting the *pars oesophagea*, causing significant economic losses and showing a high prevalence in fattening pigs (Doster 2000, Friendship 2004). Their occurrence may vary depending on husbandry conditions (Lin et al. 2024). The development of gastric ulcers is influenced by multiple factors, including diet composition, feeding practices, and physiological conditions (Szabó et al. 2023, Kamphues et al. 2025). In addition, recent field studies have also shown that gastric lesions in finishing pigs are influenced by multiple non-dietary risk factors and may negatively affect production performance, further supporting the multifactorial nature of this condition (Cybulski et al. 2024).

Low gastric pH plays a significant role, as it can disrupt the protective barriers of the non-keratinized epithelium of the *pars oesophagea*, thereby increasing the risk of erosions and ulcerations. The physiological pH of the *pars oesophagea* typically ranges between 5 and 7, while the glandular regions of the stomach have a lower pH (approximately 2-3; Doster et al. 2000, Taillieu et al. 2024). A pH below 4 in the *pars oesophagea* has been associated with an increased risk of mucosal erosion and ulceration in pigs (Doster 2000, Mösseler et al. 2012).

Fusobacterium gastrosuis is a Gram-negative bacterium that has been detected in gastric samples from pigs, and its presence together with other microorganisms suggests a potential role in gastric diseases (De Witte et al. 2019, Cortez Nunes et al., 2022). The aim of this study was to investigate the presence of *F. gastrosuis* in the stomachs of fattening pigs and its potential association with the development of gastric ulcers.

Materials and Methods

Gastric samples were collected post-mortem from fattening pigs following standard procedures and in accordance with ethical principles (ethical approval was not required according to national legislation). Gastric pH was measured in the *pars oesophagea* in 112 stomachs immediately after sampling using a calibrated pH meter. Macroscopic lesions in the *pars oesophagea* were scored on a four-point scale (0-3), where 0 = no lesions and 3 = severe ulceration.

For molecular analysis, 20 stomachs were selected to represent the full range of lesion scores. From each stomach, tissue samples were collected from the *pars oesophagea* (non-glandular region) and the fundic (glandular) region. Total RNA and reverse transcription of isolated mRNA to cDNA from the samples was carried out using RNeasy Mini Kit (Qiagen, Hilden, Germany) and iScript cDNA Synthesis Kit (Bio-Rad, Hercules, California, USA) according to the manufacturers' protocols. Detection of *Fusobacterium gastrosuis*-specific genes was performed by qualitative real-time PCR as previously described by Karaffová et al. (2017). Primer sequences used in the study are listed in Table 1.

Statistical analysis: Differences in gastric pH among lesion scores were assessed using the Kruskal-Wallis test. The association between pH and lesion severity was evaluated using Spearman's rank correlation coefficient. Statistical significance was set at $p<0.05$.

Results and Discussion

Measurements of pH in the *pars oesophagea* of pigs showed a negative correlation with lesion severity, confirming that low pH represents a risk factor for mucosal damage (Mösseler et al. 2012). pH values ranged from 3.2 to 6.1, with the lowest pH associated with the most severe ulcerations. Mean pH values gradually decreased with increasing lesion scores: 0 (pH 5.2-6.1), 1 (pH 4.5-5.0), 2 (pH 3.8-4.6), and 3 (pH 3.2-3.8; Table 2). Mean pH decreased significantly with increasing lesion scores (Kruskal-Wallis test, $p<0.001$), and Spearman's correlation confirmed a strong negative association between pH and lesion severity ($r = -0.68$, $p<0.001$). These observations are in line with our previous post-mortem investigation, which demonstrated that ulcerative and pre-ulcerative lesions predominantly affect the *pars oesophagea*, whereas bacterial colonization by gastric pathogens occurs mainly in the glandular regions of the stomach. This supports the concept that acid-related mucosal injury represents a primary driver of lesion development, while microbial factors may act as secondary or modifying contributors rather than direct causative agents (Krepelková et al. 2025).

Recent studies suggest that alterations in the microbiota, together with low pH, may affect epithelial integrity in the *pars oesophagea* (Taillieu et al. 2024). Experimental studies have demonstrated that infections can damage the gastric epithelium, indicating that interactions between microorganisms influence ulcer development (Hellemans et al. 2007).

Detection of *Fusobacterium gastrosuis* DNA revealed differential colonization across gastric regions. The bacterium was more frequently identified using the 16S rRNA gene marker than gyrB. In the fundic region, 14 samples (70%) were positive for 16S rRNA and 6 samples (30%) for gyrB, whereas in the *pars oesophagea*, 10 samples (50%) were positive for 16S rRNA

Table 1. Primers used for detection of selected genes of Fusobacterium gastrosuis by qualitative real-time PCR.

Primer	Sequences 5'-3'	References
F. gastrosuis 16S rRNA Fw	CAGTGGGGAATATTGGACAA	Taillieu et al. 2024
F. gastrosuis 16S rRNA Rev	GGTACCGTCACTTTCTTCTT	
F. gastrosuis *gyrB* Fw	GAAGACAACCCAGCTGTAACA	
F. gastrosuis *gyrB* Rev	CAGCTAATTTCCCAGGAAGTGA	

Table 2. Measured pH values according to lesion score.

Ulcer score	Gastric pH (range)
0	5.2 - 6.1
1	4.5 - 5.0
2	3.8 - 4.6
3	3.2 - 3.8

Note: Values represent the ranges of measured gastric pH in the *pars oesophagea* region.

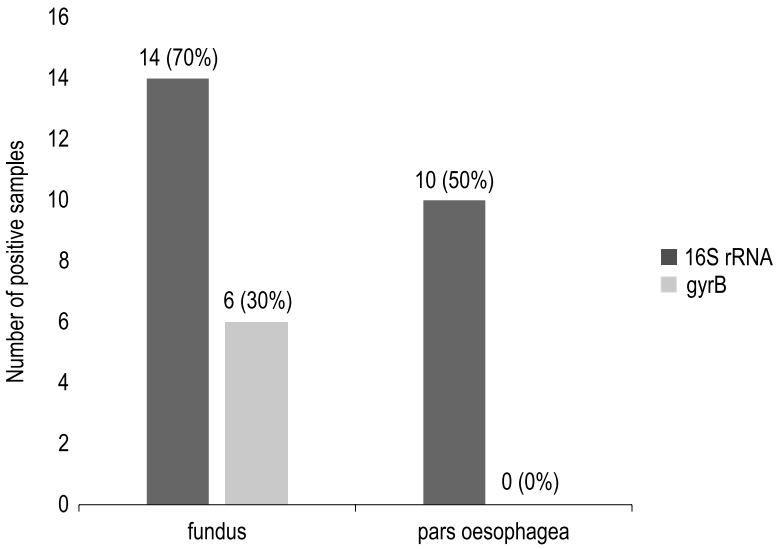

Fig. 1. Qualitative real-time PCR results for selected Fusobacterium gastrosuis genes in different stomach regions.

and gyrB was not detected in this region (Fig. 1). This discrepancy between markers reflects the higher sensitivity of 16S rRNA and the lower specificity of gyrB, consistent with previous findings on *Fusobacterium* detection within complex microbiota (Cortez Nunes et al. 2022).

Although erosive and ulcerative lesions occurred almost exclusively in the *pars oesophagea*, colonization by *F. gastrosuis* was more pronounced in the glandular regions of the stomach, suggesting that the mere presence of the bacterium does not explain ulcer formation. In this context, *Fusobacterium* species have been increasingly associated with mucosal inflammation and epithelial barrier disruption in gastric disease contexts, supporting the plausibility of indirect mechanisms mediated by inflammatory signaling rather than direct lesion-site pathogenesis. *Fusobacterium* spp., particularly in human gastric disease contexts, have been associated with the activation of inflammatory pathways, including increased production of pro-inflammatory cytokines and disruption of epithelial barrier function (Sorino et al. 2025). Such inflammatory signaling originating in the glandular mucosa may contribute to alterations in gastric physiology, including changes in acid secretion or mucosal defense mechanisms. In this context, dysbiosis in the glandular region could indirectly compromise the integrity of the *pars oesophagea*, rendering it more susceptible to acid-induced injury, even in the absence of direct bacterial colonization at the lesion site. *Fusobacterium gastrosuis* was frequently detected in the gastric samples, often together with one or more *Helicobacter* species, including

H. suis, suggesting potential co-occurrence in the gastric microbiota of pigs (De Witte et al. 2019, Cortez Nunes et al. 2022). These bacteria, including their lysates, have been shown to damage epithelial cells in experimental models, indicating their potential role in mucosal injury. A similar regional distribution pattern has been consistently observed in our earlier work focusing on *Helicobacter* spp., where the highest bacterial detection rates were recorded in the fundic region, despite the predominance of lesions in the *pars oesophagea*. Together, these findings challenge a simplistic model of direct bacterial pathogenesis at the lesion site and instead suggest an indirect mechanism involving regional dysbiosis, inflammatory signaling, or alterations in gastric physiology originating from the glandular mucosa (Krepelková et al. 2025).

The use of qualitative real-time PCR enabled high sensitivity in detecting selected bacterial genes, even in areas with low colonization density, supporting the relevance of this method for comprehensive monitoring of gastric pathogens. Overall, these results emphasize that gastric ulcer formation in pigs is a multifactorial process influenced by a combination of physicochemical conditions and microbial interactions.

Several limitations of the present study should be acknowledged. Gastric pH was assessed post-mortem and therefore represents a static measurement that does not capture diurnal fluctuations occurring in vivo. Nevertheless, when applied consistently across animals, post-mortem pH assessment has been shown to provide biologically meaningful associations with gastric pathology, as demonstrated in our previous investigations. Furthermore, the qualitative nature of the real-time PCR approach does not allow estimation of bacterial load; thus, the presence of *Fusobacterium gastrosuis* should be interpreted as indicative of colonization rather than direct pathogenicity. The cross-sectional design additionally precludes causal inference, and future longitudinal studies will be required to clarify temporal relationships between gastric acidity, microbial colonization, and ulcer development.

Conclusion

The present study indicates that the development of ulcers in the *pars oesophagea* of fattening pigs is primarily associated with low gastric pH, while the presence of *Fusobacterium gastrosuis* may represent a secondary, opportunistic factor indirectly involved in mucosal damage. Although ulcers occurred predominantly in the *pars oesophagea*, colonization by *F. gastrosuis* was higher in the glandular region of the stomach, supporting its potential role as an opportunistic pathogen, particularly in combination with adverse physicochemical conditions. These findings support the concept of a multifactorial pathogenesis of gastric ulcers and highlight the importance of simultaneous monitoring of gastric pH and bacterial colonization in preventing gastric ulcers in pigs.

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Autor Declarations

Ethics approval

Ethical approval was not required because all samples were collected post-mortem from routinely slaughtered animals in accordance with national legislation.

Use of generative artificial intelligence

Generative artificial intelligence tools were used solely for language editing and improvement of English grammar. The authors take full responsibility for the content of the manuscript.

Conflict of interest

The authors declare that they have no conflict of interest.

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