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BRIEF REPORT

Genomic and phenotypic characterization of *Enterococcus faecalis* from broiler sternal bursitis: antimicrobial resistance and one health risks

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Abstract

Enterococcus spp. are opportunistic bacteria capable of acquiring antimicrobial resistance and virulence traits, facilitating their adaptation to multiple ecological niches. Sternal bursitis, a condition affecting poultry welfare and carcass quality, remains poorly characterized from a microbiological perspective. This study provides the first genomic and phenotypic characterization of *Enterococcus* isolates from bursitis lesions in broilers, aiming to assess their antimicrobial resistance profiles, virulence determinants, and genetic diversity within a One Health framework. A total of 44 *Enterococcus* isolates were recovered from 48 sternal bursitis lesions, all identified as *E. faecalis*. Resistance was common for tetracycline (70.5%) and erythromycin (27.3%), while all isolates remained susceptible to critically important antimicrobials, including vancomycin and linezolid. Whole-genome sequencing revealed a genetically diverse population, comprising multiple sequence types, plasmid replicons, and virulence gene profiles, including determinants for adhesion, biofilm formation, capsule synthesis, and extracellular proteases. Ionophore resistance genes (*narA*, *narB*) were also detected in several lineages. The coexistence of antimicrobial resistance and virulence determinants, often linked to mobile genetic elements, highlights the potential of lesion-associated with *E. faecalis* to act as reservoirs of relevant genes with zoonotic implications. Overall, this work highlights the diverse and ecological role of *Enterococcus* in extraintestinal poultry infections, reinforcing the need for continued genomic surveillance to promote animal health, food safety, and antimicrobial stewardship.

Keywords *Enterococcus faecalis* · Broiler chickens · Sternal bursitis · Antimicrobial resistance · Virulence factors

Background

Enterococcus spp. are Gram-positive, facultatively anaerobic cocci that inhabit the intestinal microbiota of humans and animals. Among them, *Enterococcus faecalis* and *Enterococcus faecium* are the most clinically relevant species, showing increasing antimicrobial resistance and diverse virulence determinants (Georges et al. 2022). Their resilience across environments, including hospital and farms, underscores their relevance within the One Health framework (Coelho et al. 2023).

In poultry production, *Enterococcus* spp. are commonly isolated from the intestinal tract, litter, and carcasses, but are also associated with extraintestinal infections, such as septicemia, arthritis, and endocarditis (Bortolaia et al. 2016; Dolka et al. 2017). Sternal bursitis, commonly referred to as "breast blisters", is a condition characterized by inflammation of the sternal bursa, leading to serous or purulent lesions surrounded by inflamed tissue (Silva et al. 2024). Besides its welfare impact, sternal bursitis contributes to carcass downgrading and economic losses in poultry production, and although these lesions have multifactorial causes, they are often associated with prolonged pressure on the keel bone due to poor litter quality, abrasive or wet bedding, or mechanical trauma. Opportunistic bacterial agents, such as *Staphylococcus* spp. and *Escherichia coli*, may also

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been contribute to lesion development and severity (Silva et al. 2024; Ribeiro et al. 2025). Despite the frequent presence of *Enterococcus* spp. in poultry environments, their involvement in bursitis remains underexplored.

Resistance determinants such as *vanA*, *vanB*, *erm(B)*, and tetracycline genes (*tet(M)* and *tet(L)*) have been widely reported in livestock, highlighting the role of poultry as a reservoir of antimicrobial resistance, a situation largely driven by the selective pressure resulting from antimicrobial use in animal production (Ramos et al. 2012). The co-occurrence of resistance and virulence factors, including those mediating adhesion and biofilm formation, can enhance pathogenicity and complicate control measures (Lazar et al. 2023).

Despite increasing evidence of *Enterococcus* spp. involvement in poultry infections, little is known about their genomic diversity and virulence potential in lesions such as bursitis. Thus, focusing on *Enterococcus* spp. from bursitis lesions addresses a clear gap in understanding their pathogenic potential and One Health relevance. This study provides the first phenotypic and genomic characterization of *E. faecalis* from bursitis lesions in broilers, aiming to assess their antimicrobial resistance, virulence profiles, and potential zoonotic relevance within a One Health framework.

Materials and methods

Sample collection

During post-mortem inspection of broilers at a slaughterhouse in Oliveira de Frades (Portugal), 48 samples were collected from distinct animals showing visible sternal bursitis lesions over a two-month period (November-December 2021). The broilers had an average age of 86 days at slaughter. Samples were collected immediately after picking and evisceration. Fibrinous material from the bursa was obtained aseptically by disinfecting the lesion area with 70% ethanol, opening the bursa under sterile conditions, and collecting the material with a sterile swab placed in transport medium for transfer to the laboratory.

Enterococcus spp. isolation

Swabs were placed in 5 ml of Brain Heart Infusion (BHI) broth (LiofilChem, Roseto degli Abruzzi, Italy) and incubated at 37 °C for 24 h. Cultures were then plated on Slanetz-Bartley (SB) agar (Liofilchem, Roseto degli Abruzzi, Italy) with and without 4 mg/L of vancomycin, to isolate both *Enterococcus* spp. and vancomycin-resistant enterococci (VRE), and incubated at 37 °C for 48 h. One presumptive *Enterococcus* colony per sample was subcultured on Kanamycin Aesculin Azide agar (Liofilchem, Roseto degli Abruzzi, Italy) and incubated at 37 °C for 24 h. Isolates able to hydrolyze aesculin were subcultured, in BHI agar, and bacterial stocks obtain from solid media were stored at −80 °C for further characterization.

Antimicrobial susceptibility testing

The antimicrobial susceptibility testing was determined as described by the Clinical and Laboratory Standards Institute (CLSI) guidelines (2024) (CLSI 2024). The tests were performed using the disk diffusion method on Mueller–Hinton (MH) II agar (Oxoid, Basingstoke, UK) and included 10 antibiotics: ampicillin (AMP, 10 µg), vancomycin (VAN, 30 µg), teicoplanin (TEC, 30 µg), erythromycin (ERY, 15 µg), chloramphenicol (C, 30 µg), linezolid (LNZ, 30 µg), quinupristin-dalfopristin (QDA, 15 µg), imipenem (IMI, 10 µg), tetracycline (TET, 30 µg), and ciprofloxacin (CIP, 5 µg).

DNA extraction

The genomic DNA was extracted using the Insta Gene™ Matrix (Bio-Rad, California, USA), according to the manufacturer's instructions. The concentration and purity of the extracted DNA were evaluated using the ND-100 Spectrophotometer, NanoDrop.

Whole-genome sequencing analysis

We selected a subset of 19 isolates for whole-genome sequencing (WGS) based on their phenotypic antimicrobial resistance profiles to maximize genomic diversity. Isolates representing different resistance patterns were prioritized to ensure coverage of potentially distinct genetic backgrounds. WGS was carried out on an Illumina platform. Genome analysis was performed using the INNUca pipeline (v4.2.3–06.3), developed by the Instituto Nacional de Saúde Doutor Ricardo Jorge (Lisbon, Portugal), which includes Multi-Locus Sequence Typing (MLST). Identification of acquired antimicrobial resistance genes was carried out using ResFinder (v4.6.0, Center for Genomic Epidemiology, Technical University of Denmark, Lyngby) and AMRFinderPlus (v4.0.3, National Center for Biotechnology Information, Bethesda, MD, USA). Detection of virulence-related genes was conducted with VirulenceFinder (v2.0, Center for Genomic Epidemiology), and plasmid content was analyzed using PlasmidFinder (v2.1), applying default parameters.

Molecular characterization by PCR

The remaining 25 isolates, showing more homogeneous or less relevant resistance profiles, were characterized by

polymerase chain reaction (PCR) to expand the dataset while maintaining methodological balance according to laboratory resources. DNA amplification reactions were performed using a ProFlex™ PCR System thermal cycler (Applied Biosystems, Waltham, USA) in a final volume of 50 µL, containing: 30.2 µL of ultra-pure water, 5 µL of complete buffer (Bioron, Römerberg, Germany), 1.5 µL of 100 mM $MgCl_2$, 1 µL of dNTPs (10 mM), 1 µL of each primer (50 µM), 0.3 µL of DFS-Taq DNA polymerase (5 U/µL, Bioron®), and 10 µL of template DNA (10 ng). Positive controls were strains from the MicroART collection, and Milli-Q water was used as the negative control. Two *Enterococcus* species were targeted: *E. faecalis* ($ddl_{E.\ faecalis}$), and *E. faecium* ($ddl_{E.\ faecium}$). Thirteen primer sets were employed to screen for resistance genes associated with five antibiotic classes: glycopeptides (*vanA*, *vanB*), macrolides (*erm(A)*, *erm(B)*, and *erm(C)*), phenicols (*catA*), streptogramins (*vatD*, *vatE*), and tetracyclines (*tet(K)*, *tet(L)*, *tet(M)*, *tet(O)*). Additionally, isolates were tested for the virulence-associated gene *gelE* (gelatinase). Primer sequences, PCR conditions, and amplicon sizes are listed in Table 1.

Results

Prevalence of *Enterococcus* spp

A total of 48 bursitis cases were examined from a slaughterhouse in Oliveira de Frades (Portugal), from which 44 (91.7%) yielded *Enterococcus* spp. isolates. Growth was observed exclusively on non-supplemented plates, indicating that none of the isolates exhibited phenotypic resistance to vancomycin. As selective culture methods were used, it cannot be excluded that other bacterial species were present in the lesions.

Antimicrobial susceptibility patterns

The *Enterococcus* spp. strains isolated from sternal bursitis cases in broilers showed resistance to two of the ten antibiotics tested. The highest proportion of resistance was observed for tetracycline (70.5%), followed by erythromycin (27.3%). All isolates were susceptible to ampicillin, vancomycin, teicoplanin, chloramphenicol, linezolid, quinupristin-dalfopristin, imipenem and ciprofloxacin (Fig. 1).

A total of eight distinct antibiotic classes were tested, including β-lactams (AMP, and IMI), glycopeptides (VAN, and TEC), macrolides (ERY), phenicols (C), oxazolidinones (LNZ), streptogramins (QDA), tetracyclines (TET), and fluoroquinolones (CIP). Multidrug resistance, defined as resistance to three or more antibiotic classes, was not identified among the isolates (Ahmed et al. 2023).

Genomic and molecular profiles

WGS was applied to a subset of isolates ($n=19$) to ensure genomic diversity and to perform in-depth genetic characterization, while PCR included the remaining isolates ($n=25$), improving dataset representativeness. All isolates were identified as *E. faecalis*.

MLST analysis of WGS-sequenced isolates, identified seven different sequence types (STs): ST36 (36.8%), ST444 (21.1%), ST82 (10.5%), ST300 (10.5%), ST314 (10.5%), ST245 (5.3%) and ST59 (5.3%). The genetic relationships among these STs are shown in Fig. 2.

Genotypic analysis (Table 2) revealed that *lsa(A)*, which encodes intrinsic resistance to lincosamides and streptogramin A, was the most frequently detected resistance gene (94.7%). Additional resistance determinants included tetracycline resistance genes *tet(O)* (52.6%), *tet(L)* (36.8%), and *tet(M)* (31.6%), and macrolide resistance genes *erm(B)* (31.6%) and *erm(54)* (15.8%). One isolate (5.3%) carried *lnu(G)*, which confers resistance to lincosamides via nucleotidylation. Conversely, no *vanA* or *vanB* glycopeptide resistance genes were detected. Ionophore resistance genes *narA* and *narB* were co-detected in six (31.6%) isolates. The number of virulence-associated genes per isolate ranged from 16 to 32. Adhesion-related genes were highly prevalent, with *efaA* exhibiting 100% detection, followed by *ebpA*, *ebpB*, *ebpC*, *srtC*, *fss1* (each 94.7%) and *bopD* (89.5%). Other adhesion-associated genes were detected at lower frequencies, including *EF0485* (52.6%), *fss2* (15.8%), *fss3*, *prgB/asc10* and *asa1* (each 10.5%), and *ace* (5.3%). The most prevalent capsular polysaccharide genes were *cpsB* (100%), *cpsA* (94.7%), *cpsC*, *cpsD*, *cpsE*, *cpsG*, *cpsH*, *cpsI*, *cpsJ* (63.2% each), and *cpsK* (52.6%). Quorum-sensing regulators were widely distributed, with *fsrB* detected in 100% of isolates, *fsrC* in 94.7%, and *fsrA* in 84.2%. Protease-encoding genes showed a similar distribution, with *gelE* detected in all isolates (100%), and *EF0818* and *sprE* in 94.7% of isolates each. Several of these widely detected genes, including *gelE*, *sprE*, the *fsrA/B/C* quorum-sensing system, and the *ebpA/ebpB/ebpC* pilus cluster are biofilm-associated genes. Cytolysin operon genes (*cylA*, *cylI*, *cylL*, *cylR*, *cylS*) were less frequent, with *cylI* being the most prevalent, detected in five isolates (26.3%). Plasmid replicons r*ep9b* and *repUS43* were each detected in four (21.1%) isolates; however, these replicon types were not co-detected in the same isolates. In addition, *rep6* was found in a single (5.3%) isolate.

The PCR-characterized isolates are summarized in Table 3. The most frequent antimicrobial resistance genes were *tet(O)* (28%) and *tet(M)* (24%). The *erm(B)* gene was also found (16%), indicating resistance to macrolides, particularly erythromycin. Regarding virulence determinants, the *gelE* gene was detected in 96% of isolates.

Table 1 Target genes, primer sequences, conditions, and amplicon sizes used for the identification of *Enterococcus* species, antimicrobial resistance and virulence genes by PCR

Gene	Primer (5' → 3')	Conditions	Size	Reference
$ddl_{E.\ faecalis}$	ATC AAG TAC AGT TAG TCT ACG ATT CAA AGC TAA CTG	94 °C 2 min 94 °C 1 min/46,9 °C 1 min/72 °C 1 min (30 cycles) 72 °C 10 min	941 bp	(Dutka-Malen et al. 1995)[a]
$ddl_{E.\ faecium}$	TAG AGA CAT TGA ATA TGC CTA ACA TCG TGT AAG CT	94 °C 2' 94 °C 1 min/50 °C 1 min/72 °C 1 min (30 cycles) 72 °C 10 min	550 bp	(Dutka-Malen et al. 1995)[a]
vanA	GGG AAA ACG ACA ATT GC GTA CAA TGC GGC CGT TA	94 °C 2 94 °C 1 min/54 °C 1 min/72 °C 1 min (30 cycles) 72 °C 10 min	732 bp	(Dutka-Malen et al. 1995)
vanB	ATG GGA AGC CGA TAG TC GAT TTC GTT CCT CGA CC		635 bp	
erm(A)	TCT AAA AAG CAT GTA AAA GAA CTT CGA TAG TTT ATT AAT ATT AGT	93 °C 3 min 93 °C 1 min/52 °C 1 min/72 °C 1 min (35 cycles) 72 °C 5 min	645 bp	(Sutcliffe et al. 1996)
erm(B)	GAA AAG ATA CTC AAC CAA ATA AGT AAC GGT ACT TAA ATT GTT TAC		639 bp	
erm(C)	TCA AAA CAT AAT ATA GAT AAA GCT AAT ATT GTT TAA ATC GTC AAT		642 bp	
catA	GGA TAT GAA ATT TAT CCC TC CAA TCA TCT ACC CTA TGA AT	94 °C 5 min 94 °C 1 min/50 °C 1 min/72 °C 2 min (30 cycles) 72 °C 7 min	486 bp	(Aarestrup et al. 2000)
vatD	CCG AAT CCT ATG AAA ATG TAT CC GCA GCTACTATTGCACCATCCC	94 °C 2 min 94 °C 1 min/55 °C 1 min/72 °C 3 min (40 cycles) 72 °C 5 min	413 bp	(Robredo et al. 2000)
vatE	ACG TTA CCC ATC ACT ATG GCT CCG ATA ATG GCA CCG AC		282 bp	
tet(K)	TTA GGT GAA GGG TTA GGT CC GCA AAC TCA TTC CAG AAG CA	94 °C 1 min 94 °C 1 min/55 °C 2 min/72 °C 2 min (30 cycles) 72 °C 10 min	697 bp	(Aarestrup et al. 2000)
tet(L)	CAT TTG GTC TTA TTG GAT CG ATT ACA CTT CCG ATT TCG G	94 °C 1' 94 °C 1 min/50 °C 1 min/72 °C 1 min (30 cycles) 72 °C 10 min	456 bp	
tet(M)	GTT AAA TAG TGT TCT TGG AG CTA AGA TAT GGC TCT AAC AA	94 °C 1 min 94 °C 1 min/55 °C 2 min/72 °C 2 min (30 cycles) 72 °C 10 min	576 bp	
tet(O)	ACG GAR AGT TTA TTG TAT ACC TGG CGT ATC TAT AAT GTT GAC	94 °C 5 min 94 °C 30 s/60 °C 30 s/72 °C 30 Sect. (25 cycles) 72 °C 7 min	171 bp	(Aminov et al. 2001)
gelE	AGT TCA TGT CTA TTT TCT TCA C CTT CAT TAT TTA CAC GTT TG	94 °C 3' 94 °C 1 min/55 °C 1 min/72 °C 1 min (30 cycles) 72 °C 5 min	403 bp	(Eaton and Gasson 2001)

[a]annealing temperature adapted from the cited source

Discussion

To our knowledge, this is the first report describing the isolation and genomic characterization of *Enterococcus* spp. from sternal bursitis lesions in broilers. Although the high number of isolates recovered is noteworthy, our findings are from a single slaughterhouse which may not reflect the situation in other production settings in Portugal. *E. faecalis* was the exclusively detected species, highlighting its role as the main extraintestinal enterococcal pathogen in poultry. The absence of *E. faecium*, commonly reported in other poultry studies, may reflect local epidemiological differences or the limited scope of sampling. The frequent detection of *Enterococcus* spp. suggests that these bacteria might contribute to the microbial ecology of sternal bursitis, either as primary opportunistic pathogens or as secondary

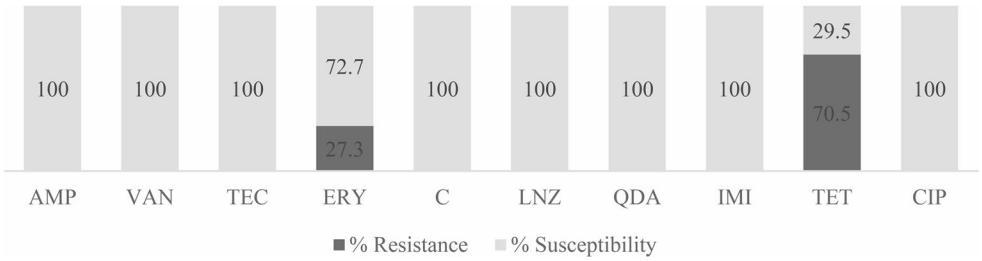

Fig. 1 Phenotypic resistance rates of *Enterococcus* spp. isolated from sternal bursitis (AMP – ampicillin; VAN – vancomycin; TEC – teicoplanin; ERY – erythromycin; C – chloramphenicol; LNZ – linezolid; QDA – quinupristin-dalfopristin; IMI – imipenem; TET – tetracycline; CIP – ciprofloxacin)

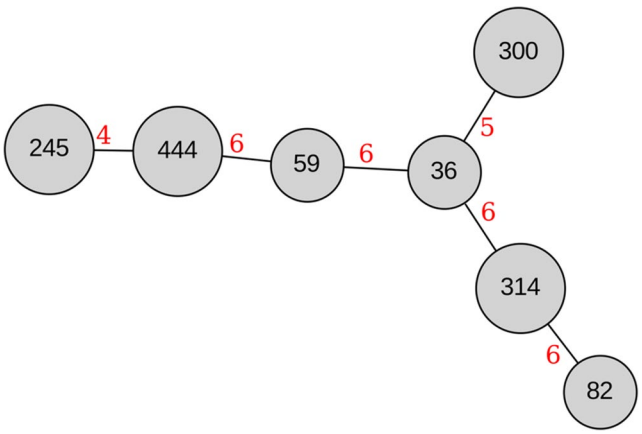

Fig. 2 Minimum spanning tree based on multilocus sequence typing (MLST) of *E. faecalis* isolates. Each node represents a sequence type (ST), and edges connect STs with the smallest allelic distances. Numbers in red indicate the number of differing MLST loci between connected STs

colonizers following tissue damage. Their occurrence likely reflects environmental contamination, and they may colonize damaged tissue due to their ability to form biofilms and withstand harsh environmental conditions (Krawczyk et al. 2021). Further studies across multiple slaughterhouses are needed to confirm these patterns and assess their broader significance.

The presence of seven distinct STs, indicates multiple origins and population dispersal within poultry production systems, rather than clonal expansion alone. The minimum spanning tree corroborates this heterogeneity, with STs separated by four to six allelic differences and the absence of a dominant clonal complex. ST36 has been identified as a central genotype, linking several lineages, suggesting that bursitis cases likely originate from multiple environmental *E. faecalis* lineages rather than a single expanding clone. ST36, previously associated with amyloid arthropathy in chickens, supports the concept of lineages adapted to specific ecological niches. ST444 and ST314, carrying both *narA* and *narB*, indicate potential associations between genetic backgrounds and ionophore resistance (Blanco et al. 2018; Balakuntla et al. 2025). The detection of ST36, ST59, and ST82, previously reported in both animal and human infections, underscores the zoonotic potential of these *E. faecalis* lineages within a One Health framework (Ruiz-Garbajosa et al. 2006). The findings of our study are consistent with a previous study, which has demonstrated that *E. faecalis* from poultry exhibits heterogeneous ST distributions, while sharing lineages that circulate in animals and humans (Blanco et al. 2018).

The high proportion of tetracycline-resistant isolates (70.5%) observed in this study aligns with previous reports from poultry production systems, reflecting historical use of tetracyclines as therapeutic agents and growth promoters in poultry farming (Alzahrani et al. 2022; Garcia-Llorens et al. 2025). In some regions of the world, ongoing use continues to exert strong selective pressure on enterococcal populations (Rahman et al. 2022). Similarly, 27.3% of isolates were resistant to erythromycin, consistent with other geographic settings, including Poland and Zambia, where macrolide resistance is commonly observed among poultry-associated *E. faecalis* (and *E. faecium* in mixed populations) (Stępień-Pyśniak et al. 2016; Mwikuma et al. 2023). The persistence of resistance determinants against older antibiotic classes remains concerning from a One Health perspective. Their continued presence in the bacterial population is likely multifactorial: tetracyclines and macrolides are still largely used in food-production animals in many EU countries, exerting selective pressure that favors the maintenance of *tet* and *erm* genes (EMA 2025). In addition, these genes are frequently plasmid- or transposon-associated, promoting horizontal gene transfer (Chen et al. 2021). In contrast, the full susceptibility observed for several antimicrobials, including ampicillin, vancomycin, teicoplanin, chloramphenicol, linezolid, quinupristin–dalfopristin, imipenem and ciprofloxacin, likely reflects their restricted, limited or absent use in poultry production and the effectiveness of regulatory measures. In particular, the sustained susceptibility to glycopeptides and linezolid is consistent with the long-standing ban on

Table 2 Characterization of *E. faecalis* isolated from sternal bursitis cases in broiler chickens by whole-genome sequencing (WGS): multilocus sequence type (MLST), resistance phenotype and genotype, ionophore resistance genes, virulence factors, and associated plasmids

E. faecalis Isolates	MLST	Antibiotic Resistance		Ionophores	Virulence Factors[a]					Plasmids
		Phenotype	Genotype		Colonization	Immune Evasion	Regulatory	Protein-processing	Cyto-lysin	
JR180	300	ERY-TET	*tet(L)*, *erm(B)*, *lsa(A)*		*EF0485*, *ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *bopD*	*cpsA*, *cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*, *cpsK*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*		*rep9b* (CP002494)
JR181	82	TET	*tet(M)*, *lsa(A)*		*ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *fss2*, *fss3*, *bopD*	*cpsA*, *cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsF*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*, *cpsK*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*	*cylR2*	*repUS43* (CP003584)
JR183	36	TET	*tet(O)*, *lsa(A)*		*EF0485*, *ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *bopD*	*cpsA*, *cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*, *cpsK*	*fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*		
JR184	82	TET	*tet(M)*, *lsa(A)*		*ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *fss2*, *fss3*, *bopD*	*cpsA*, *cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsF*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*, *cpsK*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*	*cylA*, *cylI*, *cylL*, *cylS*, *cylR1*, *cylR2*	*repUS43* (CP003584)
JR185	300	ERY-TET	*tet(L)*, *erm(B)*, *lsa(A)*		*EF0485*, *ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *prgB/asc10*, *bopD*	*cpsA*, *cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*, *cpsK*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*,		*rep9b* (CP002494)

Table 2 (continued)

E. faecalis Isolates	MLST	Antibiotic Resistance		Ionophores	Virulence Factors[a]					Plasmids
		Phenotype	Genotype		Colonization	Immune Evasion	Regulatory	Protein-processing	Cyto-lysin	
JR188	36	TET	*tet(O)*, *lsa(A)*		*EF0485*, *ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*	*cpsA*, *cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*, *cpsK*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*	*cylI*	
JR191	59	ERY	*erm(54)*, *lsa(A)*		*ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *prgB/asc10*, *bopD*	*cpsA*, *cpsB*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*		
JR193	36	TET	*tet(O)*, *lsa(A)*		*EF0485*, *ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *bopD*	*cpsA*, *cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*	*fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*		
JR197	444	ERY-TET	*tet(M)*, *tet(L)*, *erm(B)*, *lsa(A)*	*narA*, *narB*	*ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *bopD*	*cpsA*, *cpsB*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*, *EF3023*		
JR199	444	TET	*tet(O)*, *lsa(A)*	*narA*, *narB*	*ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *bopD*	*cpsA*, *cpsB*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*, *EF3023*		
JR201	314	ERY-TET	*tet(O)*, *lnu(G)*, *erm(54)*, *lsa(A)*	*narA*, *narB*	*ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *asa1 bopD*	*cpsA*, *cpsB*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*, *EF3023*		
JR204	444	ERY-TET	*tet(L)*, *erm(B)*, *lsa(A)*	*narA*, *narB*	*ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *bopD*	*cpsA*, *cpsB*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*, *EF3023*		
JR207	36	TET	*tet(O)*, *tet(M)*, *tet(L)*		*EF0485*, *efaA*	*cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*, *cpsK*	*fsrA*, *fsrB*, *fsrC*	*gelE*	*cylI*	*rep9b* (CP002494), *repUS43* (CP003584)
JR209	36	TET	*tet(O)*, *lsa(A)*		*EF0485*, *ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *bopD*	*cpsA*, *cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*, *cpsK*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*	*cylI*	

Table 2 (continued)

E. faecalis Isolates	MLST	Antibiotic Resistance		Ionophores	Virulence Factors[a]					Plasmids
		Phenotype	Genotype		Colonization	Immune Evasion	Regulatory	Protein-processing	Cyto-lysin	
JR212	245	ERY-TET	*tet(M)*, *tet(L)*, *erm(B)*, *lsa(A)*		*EF0485*, *ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *ace*, *bopD*	*cpsA*, *cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*	*fsrA*, *fsrB*	*EF0818*, *gelE*, *sprE*, *EF3023*		*rep9b* (CP002494), *rep6* (AJ223161), *repUS43* (CP003584)
JR213	314	ERY-TET	*tet(M)*, *tet(L)*, *erm(B)*, *erm(54)*, *lsa(A)*	*narA*, *narB*	*ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *asa1 bopD*	*cpsA*, *cpsB*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*, *EF3023*		
JR216	444	TET	*tet(O)*, *lsa(A)*	*narA*, *narB*	*ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *bopD*	*cpsA*, *cpsB*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*, *EF3023*		
JR220	36	TET	*tet(O)*, *lsa(A)*		*EF0485*, *ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *fss2*, *bopD*	*cpsA*, *cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*, *cpsK*	*fsrA*, *fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*	*cylI*	
JR221	36	TET	*tet(O)*, *lsa(A)*		*EF0485*, *ebpA*, *ebpB*, *ebpC*, *efaA*, *srtC*, *fss1*, *bopD*	*cpsA*, *cpsB*, *cpsC*, *cpsD*, *cpsE*, *cpsG*, *cpsH*, *cpsI*, *cpsJ*, *cpsK*	*fsrB*, *fsrC*	*EF0818*, *gelE*, *sprE*		

TET — Tetracycline; ERY — Erythromycin; [a]Virulence Factors Categories: Colonization – genes involved in adherence, Immune evasion – genes contributing to evasion of host immune responses, Regulatory – quorum-sensing regulators, Protein-processing – genes encoding proteases, Cytolysin – genes of the cytolysin operon

glycopeptide use in livestock and the resulting low selective pressure (European Commision 1997). Overall, these results support an association between antimicrobial usage patterns in animal production and resistance profiles.

The high prevalence of *lsa(A)*, an intrinsic gene conferring resistance to lincosamides and streptogramin A, may provide *E. faecalis* with an adaptive advantage in poultry systems. This gene protects the ribosome from antibiotic inhibition, supporting bacterial survival under relevant selective pressures (Dina et al. 2003). Multiple resistance genes to tetracycline and macrolides were identified, reflecting ongoing selective pressure from antimicrobial use, consistent with previous findings from European and African poultry isolates (Cauwerts et al. 2007; Fatoba et al. 2022). Although the *lnu(G)* gene, which is responsible for encoding clindamycin resistance through ribosomal methylation, suggests horizontal gene transfer, the available data do not allow identification of the donor lineage. However, this gene has been reported in several Gram-positive bacteria common in animal production environments, making environmental commensals a plausible source (Zhu et al. 2017). The co-occurrence of *narA* and *narB* was expected, as these genes are genetically linked and form the *narAB* operon, which confers resistance to polyether ionophores such as narasin, salinomycin and maduramicin. Importantly, recent studies have shown that *narAB* is frequently located on mobile genetic elements, including plasmids, where it often appears near genes mediating resistance to clinically

Table 3 Characterization of the *E. faecalis* isolated from sternal bursitis in broilers

E. faecalis Isolates	Antibiotic Resistance		Virulence Factor
	Phenotype	Genotype	
JR182	TET	*tet(M)*	*gelE*
JR186			*gelE*
JR187	TET	*tet(O)*	*gelE*
JR189	TET	*tet(O)*	*gelE*
JR190	TET	*tet(O)*	*gelE*
JR192	ERY	*erm(B)*	*gelE*
JR194	TET	*tet(M)*	*gelE*
JR195	TET	*tet(O)*	*gelE*
JR196			*gelE*
JR198	TET	*tet(M)*	*gelE*
JR200			*gelE*
JR202	ERY	*erm(B)*	*gelE*
JR203			*gelE*
JR205			*gelE*
JR206			*gelE*
JR208	TET	*tet(M)*	*gelE*
JR210	TET	*tet(M)*	*gelE*
JR211	ERY	*ermB*	*gelE*
JR214	TET	*tet(O)*	*gelE*
JR215	TET	*tet(O)*	*gelE*
JR217	ERY	*erm(B)*	
JR218	TET	*tet(O)*	*gelE*
JR219	TET	*tet(M)*	*gelE*
JR222			*gelE*
JR223			*gelE*

TET—tetracycline; ERY—erythromycin

relevant antibiotics (e.g., vancomycin, erythromycin and tetracycline) (Simjee and Tice 2023). Reports on *nar* genes in *Enterococcus* are scarce, but recent studies have indicated their increasing detection, suggesting these determinants could represent an overlooked component of the antimicrobial resistance in animal production systems (Ibrahim et al. 2025). This trend may be partly driven by the extensive use of ionophore antibiotics in poultry production, which can exert selective pressure favoring the persistence of *nar* genes (Nardulli et al. 2023).

Virulence profiling revealed a broad range of factors associated with adhesion, immune evasion, and tissue degradation, including the *fsr* quorum-sensing system, extracellular proteases, and cytolysin operon genes. Several of the detected virulence genes are known to contribute to biofilm formation (particularly *gelE*, *sprE*, the *fsrA/B/C* quorum-sensing system, and the *ebpA/ebpB/ebpC* pilus cluster), which supports the potential of these isolates to adhere, aggregate, and persist in poultry environments. Similarly, a study from South Africa highlighted the frequent detection of the *fsr* regulatory system and capsular genes among chicken isolates (Fatoba et al. 2022). The widespread presence of *gelE* and other protease-encoding genes supports the view that *E. faecalis* from sternal bursitis lesions is ecologically adapted and genetically diverse, capable of degrading collagen and extracellular matrix components. Cytolysin genes were detected in a subset of isolates, with *cylI* being the most prevalent, present in 26.3% of isolates, underscoring their potential role in enhanced pathogenicity in both avian and human infections (Ahmed et al. 2023). The coexistence of plasmid replicons carrying both resistance and virulence genes highlights the role of plasmid-mediated horizontal gene transfer in pathogenicity and resistome diversification. Our findings are consistent with previous studies in Ghana that reported *rep9* and *repUS43* in poultry-associated *E. faecalis*, often linked to conjugative plasmids carrying *tet* and *erm* resistance determinants (Amuasi et al. 2023).

This study provides the first genomic characterization of *E. faecalis* isolated from sternal bursitis lesions in broilers, highlighting their dual role as opportunistic pathogens and reservoirs of antimicrobial resistance. The coexistence of diverse resistance and virulence determinants, along with ionophore resistance genes and heterogeneous STs, underscores their ecological adaptation and zoonotic relevance. Overall, our findings emphasize the importance of continued genomic surveillance, prudent antimicrobial use, and improved husbandry practices to safeguard poultry health. These measures are crucial to prevent the dissemination of resistant and pathogenic *E. faecalis* across animal, environmental, and human ecosystems, reinforcing the relevance of a One Health approach.

Author contributions Jessica Ribeiro conceived the study, conducted the experimental work, and prepared the original manuscript draft. Vanessa Silva performed data analysis and contributed to visualization. Pedro Pinto, Rita Batista, and Alexandra Nunes assisted in laboratory work and methodological procedures. Lillian Barros validated analytical results. Madalena Vieira-Pinto, João Paulo Gomes, Gilberto Igrejas, and Patrícia Poeta provided essential resources and technical support. Sandrina A. Heleno, Filipa S. Reis, and Patrícia Poeta supervised the research and critically reviewed the manuscript. All authors read and approved the final version of the manuscript.

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Data availability The data and findings generated in this study are available within the article.

Declarations

Ethics approval All data used in this study were anonymized and gathered in full compliance with the decisions of the European Parliament and the Council regarding the epidemiological surveillance and control of communicable diseases within the European Community (Eur-Lex-31998D2119, 1998; Eur-Lex-32000D0096, 2000).

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