

Enterococcus Infection and Susceptibility to Antibiotics in Animals in Oregon, USA, from 2021-2025

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Abstract

Introduction: *Enterococcus* sp. are commensal and pathogenic bacteria that colonize humans and a wide range of animals. Those bacteria are encountered in many habitats including the intestinal tract microbiome of humans and animals. The widespread application of antibiotics in hospitals and agriculture has contributed to the emergence of antibiotic resistant Enterococci, including vancomycin resistant *Enterococcus faecium*. Because animals and humans live in close contact, we analyzed the infections caused by Enterococci in animal species in Oregon from 2021 to 2025. **Methods:** Specimens delivered to the diagnostic laboratory were cultured on blood agar and additional media as required. Bacterial colonies were identified using Mass spectrometry MALDI-TOF (*Matrix-Assisted Laser Desorption/Ionization—Time-of-Flight*) technology. Those identified colonies were then used to determine antibiotic susceptibility using either disc diffusion method or broth microdilution according to the CLSI (*Clinical Laboratory Standards Institute*). **Results:** *Enterococcus* sp. isolated from 2021 to 2025 were included in the analysis. In large animals, eleven infections were caused by *E. faecalis*, five by *E. faecium*, and four by *Enterococcus* sp. In addition, ten infections belong to other species such as *E. avium*, *E. casseliflavus*, *E. mundtii*, and *E. gallinarum*. Those Enterococci isolated were from caprine (5 cases), feline (1 case), porcine (1 case), alpaca (1 case), and equine (2 cases). *E. faecium* was isolated from surgical incisions, urine and wound. Most of the strains were resistant to the majority of the antibiotics tested except for vancomycin. *E. faecalis* was susceptible to amoxicillin/clavulanate and vancomycin, with only one isolate from a horse wound showing resistance to the antibiotics. In dogs and cats, *E. faecalis* was isolated from 71 infections, while *E. faecium* was identified as the pathogen in 37 in-

fections. The most common sites of infection by both *Enterococcus* species in dogs was bile, ear, urine and surgical incision. *E. faecium* was isolated from two pleural fluids. All *E. faecalis* were susceptible to ampicillin and vancomycin. In the case of *E. faecium*, approximately 40% were resistant to aminopenicillins and one isolate from a dog's urine was vancomycin resistant. Among 12 canine and one cat case(s), of Enterococci, one *E. casseliflavus* isolated from a foot infection, was vancomycin resistant. **Conclusion:** The number of infections caused by *Enterococcus* sp. were commonly seen in diverse infection sites of domestic animals, being more frequently isolated from incision or surgical infections in horses and GI tract and surgical sites in dogs and cats. Over the period of 5 years very few isolates showed to be resistant to vancomycin, although resistance to other antibiotics were common.

Keywords

Enterococcus spp, Infection, Antibiotic Susceptibility, Animal Infection, Sites of Infection

1. Introduction

Enterococcus species are Gram-positive bacteria, that occur in soils, water, and plants. Enterococcus spp. are also common inhabitants of both humans and a wide range of animals' intestinal tract. The Enterococcus genus comprise more than 50 species, with broad distribution in nature associated with many factors, such as host species, age, diet, environmental stress [1]. Among the enterococci, two species exhibiting a dual lifestyle as commensal and pathogens, *Enterococcus faecalis* and *Enterococcus faecium*, are predominant [2]. Both species are common colonizers of the gastrointestinal and genitourinary tracts and have become important causes of hospital associated infections [3]. In recent years, a significant increase in resistance to antibiotics has been reported [4] [5].

E. faecalis, *E. faecium*, *Enterococcus hirae*, and *Enterococcus durans* are the species frequently isolated from the intestines of mammals [6]. Enterococcus is associated with a range of infections such urinary tract, wounds, intra-abdominal, bacteremia and endocarditis [7] [8].

Enterococcus spp. has plastic genome, which facilitates the acquisition of many antibiotic resistant genes [9]. In fact, more than 300 different plasmids have been sequenced from genomes of *E. faecium*, which provides a pool of genes that have role on antibiotic resistance. The latest (2024) European Center of Disease Prevention and Control Report specified Enterococcus spp. as a major cause of hospital-related infections [10]. It is now clear that Enterococcus sp. have the ability to easily adapt to different hostile environments, and colonized individuals and animals. Those individuals become potential reservoirs for antibiotic-resistant Enterococcus, representing a source of pathogen transmission [11] [12].

Recent study attempted to determine the clinical relevance of Enterococcus spp.

isolated from different sources, such as fish, vegetables, and human intestinal content [6]. *Enterococcus* spp. are known for their ability for biofilm formation and a large percentage of the diseases caused by *Enterococcus* spp. can be traced to biofilms in the environment, suggesting that antibiotic resistance and virulence are common features in the isolates.

Infections caused by *Enterococcus* spp. in animals are a common observation [13]. In Oregon, however, no study has been conducted looking at the epidemiology of *Enterococcus* infections in different animals' species, many of them living in close contact to humans. Furthermore, antibiotic resistance in *Enterococcus* isolated from animal infections has not been investigated. Therefore, we reviewed the infection cases from 2021-2025 to determine antibiotic susceptibility of enterococci and whether resistance to vancomycin was commonly observed during the period.

2. Methods and Materials

Subjects

Large animal (equine, caprine, porcine, alpaca, and wild felid) and small animal (canine and domestic feline) patients presented to the Veterinary Teaching Hospital (VTH) at the Carlson College of Veterinary Medicine (CCVM) between 2021 and 2025 were retrospectively evaluated for *Enterococcus* spp. infections and associated antimicrobial susceptibility data. Cases represented a variety of infection sites and clinical presentations. Clinical specimens collected from these patients were submitted to the Oregon Veterinary Diagnostic Laboratory (OVDL) for bacterial culture, organism identification, and antimicrobial susceptibility testing. Susceptibility data were retrieved from the laboratory information system for cases originating from the VTH in which *Enterococcus* spp. were identified. The recorded cases represent unique isolates in animals. Episodes or sites were counted once.

Surveillance and Reporting

Only cases in which *Enterococcus* spp. were identified between 2021 and 2025 were included in the analysis. *Enterococcus* isolates were included when the organism was identified as a predominant isolate in culture and was assumed to represent a primary pathogen. Antimicrobial agents tested against these isolates were compiled and analyzed to evaluate the antimicrobial susceptibility and resistance.

Microbiological Identification

Clinical specimens submitted to the diagnostic laboratory were cultured on 5% sheep blood agar (Thermo Scientific, Remel) and additional selective or differential media when appropriate. Specimens were processed under sterile conditions within a biological safety cabinet and streaked for isolation using a four-quadrant technique. Culture plates were incubated for 16 - 24 hours at 33 - 37°C in an atmosphere containing 6% CO₂.

Bacterial identification was performed using matrix-assisted laser desorp-

tion/ionization time-of-flight mass spectrometry (MALDI-TOF MS) with the VITEK MS system (bioMérieux).

Antimicrobial Susceptibility Testing

Antimicrobial susceptibility testing was performed on isolates considered to be the predominant or clinically relevant pathogen. Pure isolates were used to prepare a 0.5 McFarland standard prior to testing. Susceptibility testing was performed using either disk diffusion (Kirby–Bauer) or broth microdilution (Sensititre, Thermo Fisher Scientific) methods in accordance with Clinical and Laboratory Standards Institute (CLSI) guidelines.

For disk diffusion testing, zones of inhibition were measured in millimeters using an automated BIOMIC V3 reader (Giles Scientific) and subsequently verified by a microbiologist. The BIOMIC V3 system converts measured zone diameters into estimated minimum inhibitory concentration (MIC) values comparable to those generated through broth microdilution testing.

Small animal isolates collected from 2021–2024 were tested using Kirby–Bauer disk diffusion (Hardy Diagnostics HardyDisk; Thermo Scientific Oxoid; BD BBL Sensi-Disc). Antimicrobials tested included amoxicillin/clavulanic acid, amikacin, ampicillin, streptomycin, doxycycline, enrofloxacin, penicillin G, rifampin, trimethoprim/sulfamethoxazole, and tetracycline.

Beginning in 2024, small animal isolates were tested using broth microdilution (Thermo Fisher Scientific Sensititre; Companion Animal Gram-Positive COMPGP1F Vet AST plate). Antimicrobials tested included amoxicillin/clavulanic acid, ampicillin, cefazolin, cefovecin, cefpodoxime, cephalothin, clindamycin, doxycycline, enrofloxacin, erythromycin, marbofloxacin, minocycline, nitrofurantoin, penicillin G, pradofloxacin, rifampin, tetracycline, trimethoprim/sulfamethoxazole, and vancomycin. Vancomycin results were not reported in accordance with CLSI guidelines.

Large animal isolates collected from 2021–2024 were tested using Kirby–Bauer disk diffusion (Hardy Diagnostics HardyDisk; Thermo Scientific Oxoid; BD BBL Sensi-Disc). Antimicrobials tested included amoxicillin/clavulanic acid, amikacin, chloramphenicol, ampicillin, streptomycin, doxycycline, enrofloxacin, penicillin G, rifampin, trimethoprim/sulfamethoxazole, tetracycline, cephalothin, and ciprofloxacin.

Beginning in 2024, equine isolates were tested using broth microdilution to determine MIC values (Thermo Fisher Scientific Sensititre; EQUIN2F plate). Antimicrobials tested included amikacin, ampicillin, cefazolin, ceftazidime, ceftiofur, chloramphenicol, clarithromycin, doxycycline, enrofloxacin, erythromycin, gentamicin, imipenem, minocycline, oxacillin, penicillin, rifampin, tetracycline, and trimethoprim/sulfamethoxazole.

Data Analysis

Antimicrobial susceptibility results were compiled and organized to evaluate resistance and susceptibility trends across host species and study years. Isolates were categorized as susceptible, intermediate, or resistant according to Clinical

and Laboratory Standards Institute (CLSI) interpretive criteria applicable at the time of testing. For analyses evaluating resistance trends, isolates categorized as intermediate were grouped with resistant isolates.

Descriptive statistics were used to summarize the frequency of *Enterococcus* spp. isolation and the proportion of isolates demonstrating resistance to each antimicrobial agent. Resistance proportions were calculated for each antimicrobial and stratified by host species group (small animal vs. large animal) and by testing period when relevant. Temporal trends in antimicrobial resistance were evaluated across the study period (2021–2025).

Data were organized and analyzed using statistical software, and results are presented as counts, percentages, and resistance proportions for each antimicrobial agent tested.

3. Results

Most Common Sites Infected by Enterococci in Small Animals and Large Animals

The most common infection sites in small animals, by order of frequency, were urine, bile, surgical incision, and intraabdominal. In contrast, in large animals the most frequent sites were surgical incision, wounds, and intraabdominal infection (Table 1).

Table 1. Incidence per site of infection in small animals caused by *E. faecalis*, *E. faecium*, and other *Enterococcus* species.

Year	Animal sp	Abscess Aspirate	Foot	Incision Wound	Thorax Abdom Bladder	Urine	Tracheal	Perianal	Bile	Ear
<i>E. faecalis</i>										
2021	Canine	X	X	X	X	X				
	Feline						X			
2022	Canine					X	X	X	X	X
	Feline					X	X			
2023	Canine			X		X			X	X
	Feline					X				
2024	Canine	X		X		X				
	Feline									
2025	Canine					X				
	Feline	X				X				
<i>E. faecium</i>										
2021	Canine		X	X	X	X		X	X	
	Feline									
2022	Canine		X		X	X		X	X	
	Feline				X	X				

Continued

2023	Canine			X	X					
	Feline								X	
2024	Canine			X		X				
	Feline	X								
2025	Canine	X		X						
	Feline									
Other species of Enterococcus										
2021	Canine		X		X	X			X	
	Feline									
2022	Canine								X	
	Feline									
2023	Canine									X
	Feline			X						

Infections in Small Animals Caused by *E. faecalis*

Seventy-one infections were diagnosed in small animals caused by *E. faecalis* from 2021 to 2025. *E. faecalis* was isolated from four different sites in dogs in 2021 (lung aspirate, foot, wound, urine) and one urinary infection was diagnosed from a cat. Infections from anal gland, bile, bladder, ear and urine sites were seen in dogs and two infection sites (urine and transtracheal aspirate) were seen in cats during 2022. Five sites were associated with infection (bile, ear, surgical incision, urine, and wound).

In 2024, infections from four different sites (abscess, surgical infection, joint and urine) were seen in dogs and no infection by *E. faecalis* was observed in cats. In 2025, only urine was diagnosed as infection site in dogs, while urine and abscess were seen in cats. **Table 2** shows the susceptibility of the isolates to antibiotics. As can be observed, all the isolates tested showed resistance to amikacin and susceptibility to ampicillin. Out of the 63 strains tested for enrofloxacin activity, 60 showed resistance and only 3 were susceptible. A large percentage was susceptible to penicillin and all of the tested strains were shown to be susceptible to vancomycin.

Table 2. Susceptibility of *Enterococcus faecalis* isolated from small animals from 2021-2025, as absolute numbers.

Phenotype	AK	Amox/Cla	AMP	Cefaz	Cefovecin	Cefpodox	Doxy	Enro	Gen	Pen	Van
R	42	39	0	8	8	8	20	60	8	0	0
S	0	3	61	0	0	0	41	3	0	61	61

Antibiotics: AK: Amikacin, AMP: Ampicillin, Cefaz: Cefazoline, Cefpodox: Cefpodoxime, Doxy: Doxycycline, Enro: Enrofloxacin, Gen: Gentamicin, Pen: Penicillin, Van: Vancomycin. R: Resistant, S: Susceptible.

The number may not agree with the total number of infections due to the difference in panels for different species.

Infection in Small Animals Caused by *E. faecium*

Thirty-seven cases of *E. faecium* were identified in small animals during the period investigated. In 2021 seven sites were infected (abdominal cavity, bile, colon, cyst, surgical infusion, urine and wound), while in 2022 infections were isolated from 8 sites, abdominal cavity, anal gland, abdominal aspirate, bile, liver biopsy, lung, pleural fluid, and urine. Two sites were identified with infection, urine, and liver biopsy. In 2023 cases were isolated from dogs (abdomen cavity, bile gallbladder and urine) and one case in a cat, isolated from the bile. In both 2024 and 2025 two sites in each year have been associated with *E. faecium* infection in dogs, urine, and surgical incision in 2024 and pleural fluid and surgical incision in 2025. Only one case was identified in cats in 2024, (abscess) and none in 2025.

An important finding was observed that a urine from a dog grew *E. faecium* resistant to vancomycin, as shown in **Table 3**.

Table 3. Susceptibility of *Enterococcus faecium* isolated from small animals from 2021-2025.

Phenotype	AK	Amox/Cla	AMP	Cefaz	Cefovecin	Cefpodox	Doxy	Enro	Gen	Pen	Van
R	37	19	25	6	6	6	18	34	7	29	1
S	0	16	13	0	0	0	19	3	1	6	36

Antibiotics: AK: Amikacin, AMP: Ampicillin, Cefaz: Cefazoline, Cefpodox: Cefpodoxime, Doxy: Doxycycline, Enro: Enrofloxacin, Gen: Gentamicin, Pen: Penicillin, Van: Vancomycin. R: Resistant, S: Susceptible.

The number may not agree with the total number of infections due to the difference in panels for different species.

Other Species of *Enterococcus* Diagnosed causing infections in small animals from 2021-2025

In 2021, five sites were identified in dogs, abdominal cavity, bile, foot, liver biopsy and urine. In 2022 and 2023 one infection was diagnosed in dogs each year, both in bile, and one wound infection was seen in a cat (2024). The species identified were *Enterococcus raffinosus* in four infections, *Enterococcus gallinarum* (one infection), *Enterococcus casseliflavos* (5 infections), *Enterococcus hirae* (one infection), and *Enterococcus avium* (2 infections).

One *E. casseliflavos* isolated from the foot of a dog (infection site) was resistant to vancomycin (**Table 4**).

Table 4. Susceptibility of *Enterococcus* sp. other than *E. faecalis* and *E. faecium* to antibiotics.

Phenotype	AK	Amox/Cla	AMP	Doxy	Enro	Pen	Van
R	13	1	1	2	11	2	1
S	0	12	12	11	2	8	10

Antibiotics: AK: Amikacin, AMP: Ampicillin, Doxy: Doxycycline, Enro: Enrofloxacin, Pen: Penicillin, Van: Vancomycin. R: Resistant, S: Susceptible.

The number may not agree with the total number of infections due to the difference in panels for different species.

Infections caused by *E. faecalis* in large animals from 2021-2025

Eleven infections by *E. faecalis* were diagnosed during the period of 5 years in large animals. In 2021, three infection sites were associated with infections in horses, one intraabdominal, one in a wound and one in the eye. In 2022, one infection site was a skin abscess in a goat, and three sites in horses (intraabdominal, joint and umbilicus). In 2023 one skin abscess was cultured positively in a goat and two sites were identified in horses (hoof, and surgical incision).

While in 2024 no infection had *E. faecalis* as a cause, in 2025 one wound in a horse grew *E. faecalis* (Table 5).

The susceptibility of the *E. faecalis* isolates are shown in Table 5. One of the intraabdominal infections grew a vancomycin resistant bacterium.

Table 5. Susceptibility of *E. faecalis* isolated from infections in large animals from 2021-2025.

Phenotype	AK	Amox/Cla	AMP	Cefaz	Cefovecin	Cephalotin	Doxy	Enro	Gen	Pen	Van
R	11	0	-	2	1	2	5	11	1	1	1
S	0	10	-	-	-	-	4	-	1	9	7

Antibiotics: AK: Amikacin, AMP: Ampicillin, Cefaz: Cefazoline, Doxy: Doxycycline, Enro: Enrofloxacin, Gen: Gentamicin, Pen: Penicillin, Van: Vancomycin. R: Resistant, S: Susceptible.

The number may not agree with the total number of infections due to the difference in panels for different species.

***E. faecium* isolates from infection sites in large animals from 2021 to 2025**

Only 5 infections caused by *E. faecium* were diagnosed in large animals in the period of five years. Two infections in horses in 2021 (both intraabdominal), one infection in a goat urine in 2023 and 2 infections in horses in 2025 (surgical site and wound). The susceptibility to antibiotics is shown in Table 6.

Table 6. Susceptibility of *E. faecium* strains isolated from large animals' infection sites from 2021 to 2025.

Phenotype	AK	Amox/Cla	AMP	Cefaz	Cefovecin	Ceftiofur	Doxy	Enro	Gen	Pen	Van
R	5	2	2	2	0	3	2	5	2	3	0
S	0	1	3	0	0	0	3	0	0	0	3

Antibiotics: AK: Amikacin, AMP: Ampicillin, Cefaz: Cefazoline, Doxy: Doxycycline, Enro: Enrofloxacin, Gen: Gentamicin, Pen: Penicillin, Van: Vancomycin. R: Resistant, S: Susceptible.

The number may not agree with the total number of infections due to the difference in panels for different species.

Infectious Caused the Other Enterococcus sp that are not *E. faecalis* of *E. faecium* in Large Animals

Other Enterococcus sp caused 10 infections in large animals during the last five years. One infection in goat (vaginal) in 2021 and two infections in horses (surgi-

cal incisions). In 2022, one infection was diagnosed in an alpaca (wound) and two infections in young horses (blood and umbilicus). In 2023, two horses were diagnosed with infection in a surgical incision and in a tendon sheath, while in 2024 two horse infections were seen in bone and wound.

The bacteria associated with those infections outline above were *E. avium* (one infection), *E. casseliflavus* (4 infections), *Enterococcus mundtii* (two infections) and *E. gallinarium* (3 infections). Susceptibility to antibiotics is outlined in **Table 7**. One of the strains of *E. casseliflavus* showed resistance to vancomycin.

Table 7. Susceptibility of *Enterococcus* sp other than- *E. faecalis* or *E. faecium* to antibiotics.

Species	Amoc/Cla	AMP	Cefaz	Ceftiofur	Doxy	Enro	Gen	Pen	Van
<i>E. avium</i> R/S	1/0	1/0	-	-	1/0	1/0	1/0	1/0	0/1
<i>E. casseliflavus</i> R/S	0/2	1/3	2/0	3/0	2/0	4/0	1/1	1/2	1/3
<i>E. mundtii</i> R/S	-	0/2	1/0	1/0	0/1	0/2	1/0	0/1	0/1
<i>E. gallinarium</i> R/S	-	1/2	-	-	1/2	2/1	-	1/0	0/1

Antibiotics: Amox/Cla: Amoxillin/Clavulanic acid, AMP: Ampicillin, Cefaz: Cefazoline, Doxy: Doxycycline, Enro: Enrofloxacin, Gen: Gentamicin, Pen: Penicillin, Van: Vancomycin. R: Resistant, S: Susceptible.

The number may not agree with the total number of infections due to the difference in antibiotic susceptibility panels for different species.

4. Discussion

Enterococci spp are human and animal commensals as well as pathogens. The *E. faecalis* and *E. faecium* colonize human and many animal species, adapting to the diverse environmental hash conditions [14]. Despite being regarded in general as harmless commensals, *Enterococcus* plays a significant role in many patients with underlying diseases [15]. Enterococci attracted more attention due to their resistance to antibiotics, which is caused by the noticeable plasticity of their genomes. In particular, vancomycin resistant strains are of increase importance in hospital-related infections [16] [17].

Vancomycin resistant enterococci has been classified by WHO as “high priority” [18]. The resistance is associated with the acquisition of the *van* operon, allowing the pathogen to produce altered cell wall peptidoglycan precursors, not recognized by glycopeptides antibiotics [19]. The finding of vancomycin-resistant enterococci in animals is a major concern because of the proximity to humans and maybe other animals. Colonized individuals or animals are potential reservoirs of multi-drug resistant *Enterococcus* spp, representing a significant source of pathogen transmission [20]-[22].

One of the most important pathogenic characteristics of enterococci is the ability to form biofilms [23] [24]. Most of the enterococci-related infections involve biofilm formation, such a urinary tract infection, endocarditis, gallbladder infection and wounds. The bacteria have a high capacity of persisting in environmental

surfaces, and to survive as commensal in the gastrointestinal tract of humans and animals until optimal conditions to develop infection is achieved [24]. A study in Portugal aimed in the discovering of enterococci reservoirs outside the hospitals, identified only *E. faecalis* and *E. faecium* as the predominant enterococci in human related environment, and *E. faecalis* and other species in animal environment [25]. In that study, the investigators cultured enterococci from the nasal cavity of pigs, which is not surprising considering animal habits.

In our study covering 5 years, only 4 isolates of *Enterococcus* (one *E. faecium*, two *E. faecalis* and one *E. casseliflavus*) from animal infections were shown to be vancomycin resistance. Similar findings, of low level of vancomycin resistance, has been observed by Lopes and colleagues in Portugal [14]. In fact, it is curious since some of the vancomycin resistant genes like *vanC* are considered to be encountered in *E. gallinarum*, *E. casseliflavus* and *E. flavescens* [20].

One of the interesting observations is that in despite that both *E. faecium* and *E. faecalis* showed almost complete resistance to first and third generation cephalosporins, the majority of the isolates were susceptible to the aminopenicillins. Although resistance to aminoglycosides is common among the *Enterococcus*, the combination of an aminopenicillins with an aminoglycoside would be still a viable combination. *E. faecium*, however, is expected to be intrinsic resistance to aminoglycosides by producing a methyltransferase *EfmM* which target the 16S RNA 1404 nucleotide [26]. *Enterococcus* is also known to express a phosphotransferase, and a chromosomal acetyl transferase enzyme which results in resistance to kanamycin and amikacin [27]. The resistance to aminoglycosides is also mediated by poor uptake of the antibiotic class.

Enterococcus spp, particularly *E. faecalis* and *E. faecium* are well known to be intrinsically resistant to cephalosporins, due to the low affinity binding of cephalosporins to the bacterial penicillin-binding proteins, in particular PBP5, a class B enzyme, which relies on a glycosyltransferase for an effective peptidoglycan synthesis [28] [29]. PBP divided into two groups, class A of bifunctional enzymes and class B which has only a transpeptidase domain. All *Enterococci* produce at least five PBPs. In *E. faecium*, PBP5 exists in an operon [30] [31]. Sequence variation of PBP5 can differentiate two groups of *E. faecium*, one with high level of ampicillin resistance associated with hospital environment, and a community associated variant that results in lower MIC to ampicillin [32] [33]. PBP5 in *E. faecalis* is not in the same operon as in *E. faecium*, and it is associated with low MIC for ampicillin [32]-[34]. More recently, a Ser/Thr kinase has also been implicated as necessary for *E. faecalis* resistance to cephalosporins [28], which suggest that other mechanisms may influence the expression of the resistant phenotype.

Enterococci have low level of intrinsic resistance to the quinolones. Our results indicate that while non-*E. faecalis* and non-*E. faecium* enterococci had shown susceptibility to quinolones, the great majority of the *E. faecium* and *E. faecalis* were resistant to the second generation quinolones. The quinolones target two enzymes, GyrA and Gyr B (DNA gyrase) and ParC and ParE (topoisomerase IV).

Efflux pumps is also a well describe mechanism of antibiotic resistance to quinolones, whereas NorA and qnr are proteins associated with the protection of the Gyr target, which have been described in *Enterococcus* [35].

Limitations of this work are that although the data represents results of tests of animals all over the State of Oregon, it likely missies not submitted to the College diagnostic laboratory. In addition, due to individual financial constrains not all infections have the etiologic agent identified and susceptibility performed.

5. Conclusion

In summary, treatment of *Enterococcus* infection in animals is not associated with vancomycin resistance, however, since the antibiotic is not used as routine in veterinary medicine, a limited number of compounds is available for the treatment of those infections.

Participation

SP: Performed assays, reviewed the cases and helped assemble the data. Wrote portion of the manuscript, edited the manuscript. BH: Performed the assays, participate in the analysis, SB: Performed the assays, participated in the analysis of the data; AM: collected the information, assemble the data; LEB: Idealized the study, analyzed the data, wrote portions of the manuscript, edited the manuscript, secured funds for the study.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

- [1] Murray, B.E. (1990) The Life and Times of the *Enterococcus*. *Clinical Microbiology Reviews*, **3**, 46-65. <https://doi.org/10.1128/cmr.3.1.46>
- [2] Arias, C.A. and Murray, B.E. (2012) The Rise of the *Enterococcus*: Beyond Vancomycin Resistance. *Nature Reviews Microbiology*, **10**, 266-278. <https://doi.org/10.1038/nrmicro2761>
- [3] García-Solache, M. and Rice, L.B. (2019) The *Enterococcus*: A Model of Adaptability to Its Environment. *Clinical Microbiology Reviews*, **32**, e00058-18. <https://doi.org/10.1128/cmr.00058-18>
- [4] Cetinkaya, Y., Falk, P. and Mayhall, C.G. (2000) Vancomycin-Resistant *Enterococci*. *Clinical Microbiology Reviews*, **13**, 686-707. <https://doi.org/10.1128/cmr.13.4.686>
- [5] Weiner-Lastinger, L.M., Abner, S., Edwards, J.R., Kallen, A.J., Karlsson, M., Magill, S.S., *et al.* (2020) Antimicrobial-Resistant Pathogens Associated with Adult Healthcare-Associated Infections: Summary of Data Reported to the National Healthcare Safety Network, 2015-2017. *Infection Control & Hospital Epidemiology*,

- 41, 1-18. <https://doi.org/10.1017/ice.2019.296>
- [6] Mubarak, A.G., El-Zamkan, M.A., Younis, W., Saleh, S.O., Abd-Elhafeez, H.H. and Yoseef, A.G. (2024) Phenotypic and Genotypic Characterization of *Enterococcus Faecalis* and *Enterococcus Faecium* Isolated from Fish, Vegetables, and Humans. *Scientific Reports*, **14**, Article No. 21741. <https://doi.org/10.1038/s41598-024-71610-0>
 - [7] Flores-Mireles, A.L., Walker, J.N., Caparon, M. and Hultgren, S.J. (2015) Urinary Tract Infections: Epidemiology, Mechanisms of Infection and Treatment Options. *Nature Reviews Microbiology*, **13**, 269-284. <https://doi.org/10.1038/nrmicro3432>
 - [8] Fiore, E., Van Tyne, D. and Gilmore, M.S. (2019) Pathogenicity of *Enterococci*. *Microbiology Spectrum*, **7**, 1-23. <https://doi.org/10.1128/microbiolspec.gpp3-0053-2018>
 - [9] Hollenbeck, B.L. and Rice, L.B. (2012) Intrinsic and Acquired Resistance Mechanisms in *Enterococcus*. *Virulence*, **3**, 421-569. <https://doi.org/10.4161/viru.21282>
 - [10] European Center for Disease Prevention and Control (2024) Point Prevalence Survey of Healthcare-Associated Infections and Antimicrobial Use in European Acute Care Hospitals. European Center for Disease Prevention and Control.
 - [11] O'Toole, R.F., Leong, K.W.C., Cumming, V. and Van Hal, S.J. (2023) Vancomycin-resistant *Enterococcus Faecium* and the Emergence of New Sequence Types Associated with Hospital Infection. *Research in Microbiology*, **174**, Article 104046. <https://doi.org/10.1016/j.resmic.2023.104046>
 - [12] Joshi, S., Shallal, A. and Zervos, M. (2021) Vancomycin-Resistant *Enterococci*: Epidemiology, Infection Prevention, and Control. *Infectious Disease Clinics of North America*, **35**, 953-968.
 - [13] Hammerum, A.M. (2012) *Enterococci* of Animal Origin and Their Significance for Public Health. *Clinical Microbiology and Infection*, **18**, 619-625. <https://doi.org/10.1111/j.1469-0691.2012.03829.x>
 - [14] Lopes, J., de Lencastre, H. and Conceição, T. (2024) Genomic Analysis of *Enterococcus Faecium* from Non-Clinical Settings: Antimicrobial Resistance, Virulence, and Clonal Population in Livestock and the Urban Environment. *Frontiers in Microbiology*, **15**, Article ID: 1466990. <https://doi.org/10.3389/fmicb.2024.1466990>
 - [15] Braga, T.M., Pomba, C. and Lopes, M.F.S. (2013) High-Level Vancomycin Resistant *Enterococcus Faecium* Related to Humans and Pigs Found in Dust from Pig Breeding Facilities. *Veterinary Microbiology*, **161**, 344-349. <https://doi.org/10.1016/j.vetmic.2012.07.034>
 - [16] Gao, W., Howden, B.P. and Stinear, T.P. (2017) Evolution of Virulence in *Enterococcus Faecium*, a Hospital-Adapted Opportunistic Pathogen. *Current Opinion in Microbiology*, **41**, 76-82. <https://doi.org/10.1016/j.mib.2017.11.030>
 - [17] Mlaga, K.D., Garcia, V., Colson, P., Ruimy, R., Rolain, J. and Diene, S.M. (2021) Extensive Comparative Genomic Analysis of *Enterococcus Faecalis* and *Enterococcus Faecium* Reveals a Direct Association between the Absence of Crispr-Cas Systems, the Presence of Anti-Endonuclease (*ardA*) and the Acquisition of Vancomycin Resistance in *E. Faecium*. *Microorganisms*, **9**, Article 1118. <https://doi.org/10.3390/microorganisms9061118>
 - [18] WHO (2017) Global Priority List of Antibiotic-Resistant Bacteria to Guide Research, Discovery and Development of New Antibiotics. WHO.
 - [19] Lombardi, A., Ripabelli, G., Sammarco, M.L. and Tamburro, M. (2025) *Enterococcus Faecium* as an Emerging Pathogen: Molecular Epidemiology and Antimicrobial Resistance in Clinical Strains. *Pathogens*, **14**, Article 483.

- <https://doi.org/10.3390/pathogens14050483>
- [20] Torres, C., Alonso, C.A., Ruiz-Ripa, L., León-Sampedro, R., del Campo, R. and Coque, T.M. (2018) Antimicrobial Resistance in *Enterococcus* spp. of Animal Origin. *Microbiol Spectr*, **6**, 1-41. <https://doi.org/10.1128/9781555819804.ch9>
 - [21] Wada, Y., Irekeola, A.A., E.A.R., E.N.S., Yusof, W., Lih Huey, L., Ladan Muhammad, S., *et al.* (2021) Prevalence of Vancomycin-Resistant *Enterococcus* (VRE) in Companion Animals: The First Meta-Analysis and Systematic Review. *Antibiotics*, **10**, Article 138. <https://doi.org/10.3390/antibiotics10020138>
 - [22] Ahmed, M.O. and Baptiste, K.E. (2018) Vancomycin-Resistant Enterococci: A Review of Antimicrobial Resistance Mechanisms and Perspectives of Human and Animal Health. *Microbial Drug Resistance*, **24**, 590-606. <https://doi.org/10.1089/mdr.2017.0147>
 - [23] Jovanović, M., Velebit, B., Tošić, T., Maki, G., Pavić, S., Jovanović, S., *et al.* (2023) Comparative Study of Virulence Factor Genes, β -Hemolysis and Biofilm Production in Invasive and Colonizing Enterococci. *European Journal of Inflammation*, **21**, Article No. 1721. <https://doi.org/10.1177/1721727x231156333>
 - [24] Hayden, M.K., Blom, D.W., Lyle, E.A., Moore, C.G. and Weinstein, R.A. (2008) Risk of Hand or Glove Contamination after Contact with Patients Colonized with Vancomycin-Resistant *Enterococcus* or the Colonized Patients' Environment. *Infection Control & Hospital Epidemiology*, **29**, 149-154. <https://doi.org/10.1086/524331>
 - [25] Gouliouris, T., Raven, K.E., Ludden, C., Blane, B., Corander, J., Horner, C.S., *et al.* (2018) Genomic Surveillance of *Enterococcus Faecium* Reveals Limited Sharing of Strains and Resistance Genes between Livestock and Humans in the United Kingdom. *mBio*, **9**, 1-15. <https://doi.org/10.1128/mbio.01780-18>
 - [26] Galimand, M., Schmitt, E., Panvert, M., Desmolaize, B., Douthwaite, S., Mechulam, Y., *et al.* (2011) Intrinsic Resistance to Aminoglycosides in *Enterococcus faecium* Is Conferred by the 16S rRNA M⁵c1404-Specific Methyltransferase EfmM. *RNA*, **17**, 251-262. <https://doi.org/10.1261/rna.2233511>
 - [27] Wei, Y., Palacios Araya, D. and Palmer, K.L. (2024) *Enterococcus Faecium*: Evolution, Adaptation, Pathogenesis and Emerging Therapeutics. *Nature Reviews Microbiology*, **22**, 705-721. <https://doi.org/10.1038/s41579-024-01058-6>
 - [28] Kristich, C.J., Little, J.L., Hall, C.L. and Hoff, J.S. (2011) Reciprocal Regulation of Cephalosporin Resistance in *Enterococcus Faecalis*. *mBio*, **2**, e00199-11. <https://doi.org/10.1128/mbio.00199-11>
 - [29] Williamson, R., Gutmann, L., Horaud, T., Delbos, F. and Acar, J.F. (1986) Use of Penicillin-Binding Proteins for the Identification of Enterococci. *Microbiology*, **132**, 1929-1937. <https://doi.org/10.1099/00221287-132-7-1929>
 - [30] Rice, L.B., Carias, L.L., Rudin, S., Hutton, R., Marshall, S., Hassan, M., *et al.* (2009) Role of Class a Penicillin-Binding Proteins in the Expression of β -Lactam Resistance in *enterococcus Faecium*. *Journal of Bacteriology*, **191**, 3649-3656. <https://doi.org/10.1128/jb.01834-08>
 - [31] Sifaoui, F., Arthur, M., Rice, L. and Gutmann, L. (2001) Role of Penicillin-Binding Protein 5 in Expression of Ampicillin Resistance and Peptidoglycan Structure in *Enterococcus faecium*. *Antimicrobial Agents and Chemotherapy*, **45**, 2594-2597. <https://doi.org/10.1128/aac.45.9.2594-2597.2001>
 - [32] Miller, W.R., Munita, J.M. and Arias, C.A. (2014) Mechanisms of Antibiotic Resistance in Enterococci. *Expert Review of Anti-Infective Therapy*, **12**, 1221-1236. <https://doi.org/10.1586/14787210.2014.956092>

- [33] Galloway-Peña, J.R., Rice, L.B. and Murray, B.E. (2011) Analysis of PBP5 of Early U.S. Isolates of *Enterococcus Faecium*: Sequence Variation Alone Does Not Explain Increasing Ampicillin Resistance over Time. *Antimicrobial Agents and Chemotherapy*, **55**, 3272-3277. <https://doi.org/10.1128/aac.00099-11>
- [34] Darehkordi, H., Saffari, F., Mollaei, H.R. and Ahmadrajabi, R. (2019) Amino Acid Substitution Mutations and mRNA Expression Levels of the *pbp5* Gene in Clinical *Enterococcus faecium* Isolates Conferring High Level Ampicillin Resistance. *APMIS*, **127**, 115-122. <https://doi.org/10.1111/apm.12922>
- [35] Hooper, D.C. (2000) Mechanisms of Action and Resistance of Older and Newer Fluoroquinolones. *Clinical Infectious Diseases*, **31**, S24-S28. <https://doi.org/10.1086/314056>