Gene structure and virulence factors of vancomycin resistance within enteric and environmental bacteria

1. Introduction

Modern medicine faces an urgent medical challenge because antibiotic-resistant bacteria have become widespread. The discovery of vancomycin-resistant bacteria presents the most alarming resistance mechanism because such bacteria severely diminish available treatment options for serious infections (Dhanda et al., 2018). Healthcare providers use glycopeptide antibiotic vancomycin as their final option to treat multidrug-resistant Gram-positive bacterial infections like *Staphylococcus aureus* and Enterococcus faecium. The widespread occurrence of VanA and VanB resistance genes has stimulated manifold treatment breakdowns and prolonged hospital admissions as well as elevated death rates (Leone et al., 2024, Caglayan et al., 2025).

The enzymes encoded in the VanA and VanB gene clusters transform the terminal D-Ala-D-Ala motif in bacterial cell wall precursors into D-Ala-D-Lac which results in a 1000-fold decrease of vancomycin binding ability. These genes exist on mobile genetic elements such as plasmids and transposons making them easily spread across clinical and environmental bacterial populations by transfer mechanisms. The ability of these genes to move between species creates concerns about their rapid spread between environmental reservoirs and clinical pathogens (Caglayan et al., 2025).

Bacterial organisms possess both resistance genes and virulence factors which are molecular attributes responsible for improving their pathogenic characteristics. Adhesins along with toxins and immune evasion proteins and tissue-degrading enzymes form the basis of bacterial traits required for colonization and tissue destruction. The simultaneous occurrence of resistance and virulence elements increases bacterial pathogenic properties and creates obstacles for treating bacterial infections. Environmental bacteria which were once regarded as non-threatening hosts virulence and resistance genes causing them to function as hidden reservoirs (Darmancier et al., 2022).

Enterococcus faecium serves as the main research subject because it carries VanA/VanB genes prominently in this investigation which analyzes its resistance genes and virulence factors with five other enteric and environmental bacteria species (Wardal et al., 2023). The research explores genomic contexts and sequences along with pathogenic pathways to reveal molecular distinctions between virulent and less pathogenic organisms.

2. Methodology

2.1 Retrieval of Reference Sequences

Researchers retrieved VanA and VanB resistance gene nucleotide reference sequences from the NCBI Nucleotide database (https://www.ncbi.nlm.nih.gov/). The research used Enterococcus faecium sequences in FASTA format as specific reference queries for homology investigations.

2.2 BLAST Search and Identification of Homologs

The reference sequences served as queries for BLASTn searches to discover bacterial species with homologs of both VanA and VanB genes. The bacterial homology research utilized the NCBI BLAST interface (https://blast.ncbi.nlm.nih.gov/ under the “nr/nt”) database with targeted bacterial taxonomic limitations. The identified top BLAST hits contained a minimum of 90% sequence similarity together with substantial alignment coverage. Among five clinic-adjacent and environmental species *Staphylococcus haemolyticus*, *Enterococcus faecalis*, *Enterococcus gallinarum*, *Staphylococcus aureus* and *Clostridioides difficile* met the selection criteria.

2.3 Sequence Alignment and Identity Matrix Generation

The protein sequences of VanA and VanB from selected species were obtained either from GenBank (https://www.ncbi.nlm.nih.gov/genbank/about/). The protein sequence alignment process used Clustal Omega (https://www.ebi.ac.uk/Tools/msa/clustalo/) as the platform. Percent identity matrices emerged from these sequences during the output phase. Assessments of the sequence alignments revealed both preserved areas together with genetic relationship data.

2.4 Phylogenetic Analysis

Using MEGA X version 10.2.6 the NJ method built phylogenetic trees through 1,000 bootstrap replications to validate the reliability of tree branches. The investigators utilized aligned sequences of proteins from both VanA and VanB as their source data. A dedicated tree-building procedure analyzed genetic evolution between selected species using individual trees.

2.5 Genomic Context and Gene Localization

The analysis of VanA and VanB gene positions (plasmid or chromosome) utilized annotated genome assembly files obtained from the NCBI Genome Browser. The locations of genes were viewed by analyzing GenBank files through SnapGene Viewer version 6.1.1. Plasmid origin was verified through the PlasmidFinder web tool accessible at (https://cge.food.dtu.dk/services/PlasmidFinder/).

2.6 Virulence Factor Identification

A search for virulence factors within selected bacterial genomes utilized two databases: VFDB (Virulence Factor Database) (http://www.mgc.ac.cn/VFs/) together with VirulenceFinder (https://cge.food.dtu.dk/services/VirulenceFinder/). The FASTA sequences of genomes received analysis to record annotated virulence genes with their chromosomal/plasmid location and predicted functional data.

Results

1. Presence and Distribution of VanA and VanB Genes

Enterococcus faecium sequence was retrieved from NCBI and blasted with datasets which led us to 5 species that have VanA gene or VanB gene. Among which, 5 species with 90-100% per identity were selected. Selected bacteria are: *Staphylococcus haemolyticus, Enterococcus faecalis, Enterococcus gallinarum, Staphylococcus aureus,* and *Clostridioides difficile*. VanA and VanB genes of *Enterococcus faecium* were blast through genome sequences of *Staphylococcus haemolyticus*, *Staphylococcus aureus*, *Enterococcus faecalis*, *Enterococcus gallinarum*, and *Clostridioides difficile*. The presence and absence along with percent identity of sequences was observed (Table 1).

Table 1: Sequence similarity of *Enterococcus faecium* VanA and VanB genes with other environmental and clinically pathogenic bacteria.

Species	**VanA (Present/Absent; % ID)**	**VanB (Present/Absent; % ID)**
Staphylococcus haemolyticus	Present (99%, 674/677 bp)	Absent
Enterococcus faecalis	Present (99, 680/683 bp)	Present (99%, 599/600 bp)
Enterococcus gallinarum	Present (99, 680/683 bp)	Present (100%, 600/600 bp)
Staphylococcus aureus	Present (99, 681/683 bp)	Absent
Clostridioides difficile	Present (99%, 681/683 bp)	Absent

2. Sequence Similarity and Phylogenetic Analysis of VanA/VanB

Thorough evaluation of VanA and VanB gene ancestries showed distinct evolutionary patterns in their distributions. The VanA sequences presented in Figure 1 maintained high levels of similarity and the Enterococcus species clustered tightly while Staphylococcus species united closely presumably because of horizontal gene transfer events. The phylogenetic analysis in Figure 2 shows that VanB sequences evolved more quickly through extensive divergence because of their limited species dispersal and high evolutionary drift rates compared to VanA.

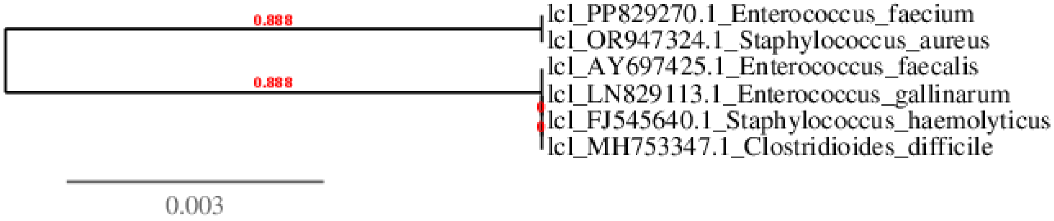


Figure 1: The phylogenetic analysis of VanA protein sequences. Established through Neighbor-Joining method computations in MEGA X at 1,000 bootstrap replicates.

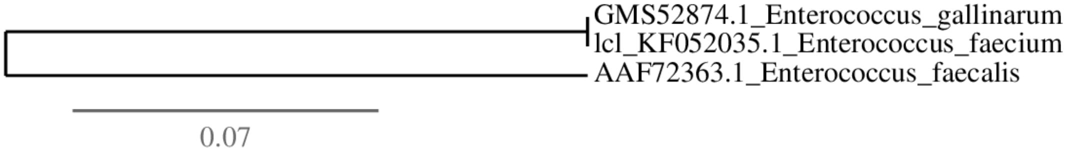


Figure 2: The phylogenetic analysis of VanB protein sequences. Established through Neighbor-Joining method computations in MEGA X at 1,000 bootstrap replicates.

A percent identity analysis matrix determined the sequence similarities among different bacterial isolates with VanA and VanB resistance genes (Table 2). Six sequences from the VanA group showed extensive sequence conservation rates that ranged from 98.51% to 100.00% in their comparisons. The Enterococcus faecium strain (lcl|PP829270.1) showed almost full concordance with Enterococcus and non-Enterococcus strains together with Staphylococcus and *Clostridioides difficile* in sequence similarity tests indicating resistance group cross-taxa similarities. The VanB group appeared more variable than its three member sequences. The two sequences KF052035.1 and GMS52874.1 possessed full identity while the third sequence AAF72363.1 demonstrated around 76.5% similarity with these two. The VanB-type resistance seems to display both highly conserved and divergent genetic characteristics which hint at possible evolutionary or genetic origin differences.

Table 2: Percent identity matrix of VanA and VanB protein sequences among selected bacterial species

#	**ID**	**1**	**2**	**3**	**4**	**5**	**6**
	VanA						
1	lcl|PP829270.1_Enterococcus_faecium	100	99.39	98.96	99.12	98.51	99.11
2	lcl|OR947324.1_Staphylococcus	99.39	100	98.96	99.46	99.35	99.46
3	lcl|AY697425.1_Enterococcus	98.96	98.96	100.0	100.0	100.0	100.0
4	lcl|LN829113.1_Enterococcus_gallinarum	99.12	99.46	100.0	100.0	100.0	100.0
5	lcl|MH753347.1_Clostridioides_difficile	98.51	99.35	100.0	100.0	100.0	100.0
6	lcl|FJ545640.1_Staphylococcus_haemolyticus	99.11	99.46	100.0	100.0	100.0	100.0
	VanB						
1	AAF72363.1[Enterococcus	100.00	76.50	76.61			
2	lcl|KF052035.1_Enterococcus_faecium	76.50	100.00	100.00			
3	GMS52874.1	76.61	100.00	100.00			

Conserved domain analysis

InterProScan analyses showed VanA and VanB proteins maintain their biological functions while conserving protein structures that span multiple species (Figures 3 and 4). Every analysis of the VanA sequences showed the presence of the D-alanine--D-alanine ligase domain which demonstrates their contribution to cell wall synthesis blocking. Some/variations of VanA proteins showed the presence of additional functional domains namely Ddl/VanB, D_ala_D_alaTGR, and Dala_Dala_lig which suggest that these proteins underwent functional modifications. Different from VanB proteins the domain combination of Ddl/VanB and D-alanine--D-alanine ligase appeared in all VanB sequences along with a few proteins which contained one or more tandem domain repeats.

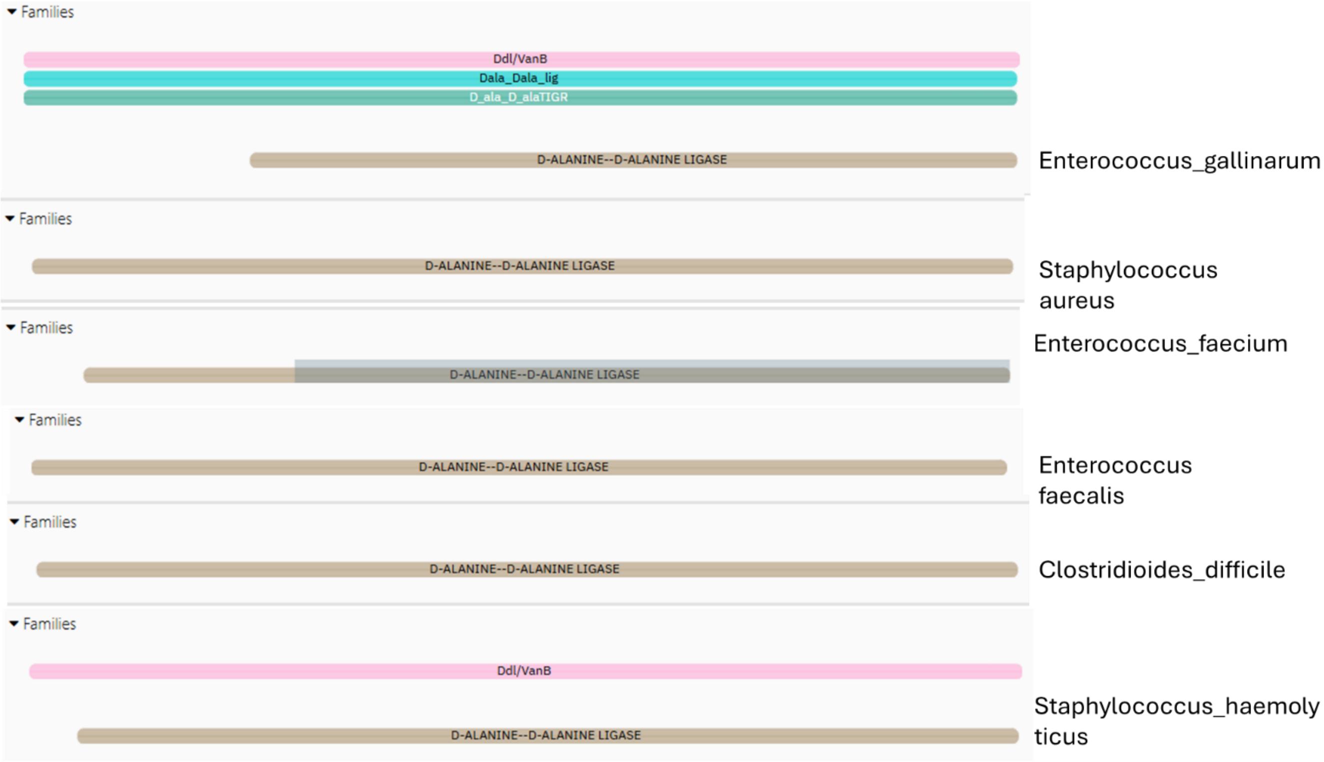


Figure 3: Conserved domain structures of VanA proteins identified through InterProScan

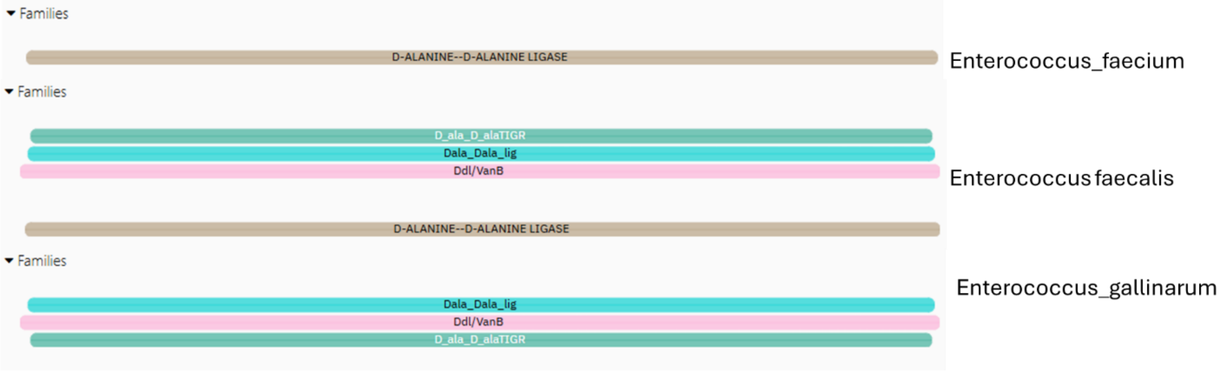


Figure 4: Conserved domain structures of Van**B** proteins identified through InterProScan

Genomic vs Plasmid Location of Van Genes

The VanA element occurs within transposable elements of *S. aureus* along with *E. faecium* while regulatory genes (vanR, vanS, vanX) flank its location (Figure 5). Plasmid-resident VanB has proven contagious because it shares genomic space with the resistance genes fosB in addition to mobile genetic elements thus facilitating viral spread (Figure 6).

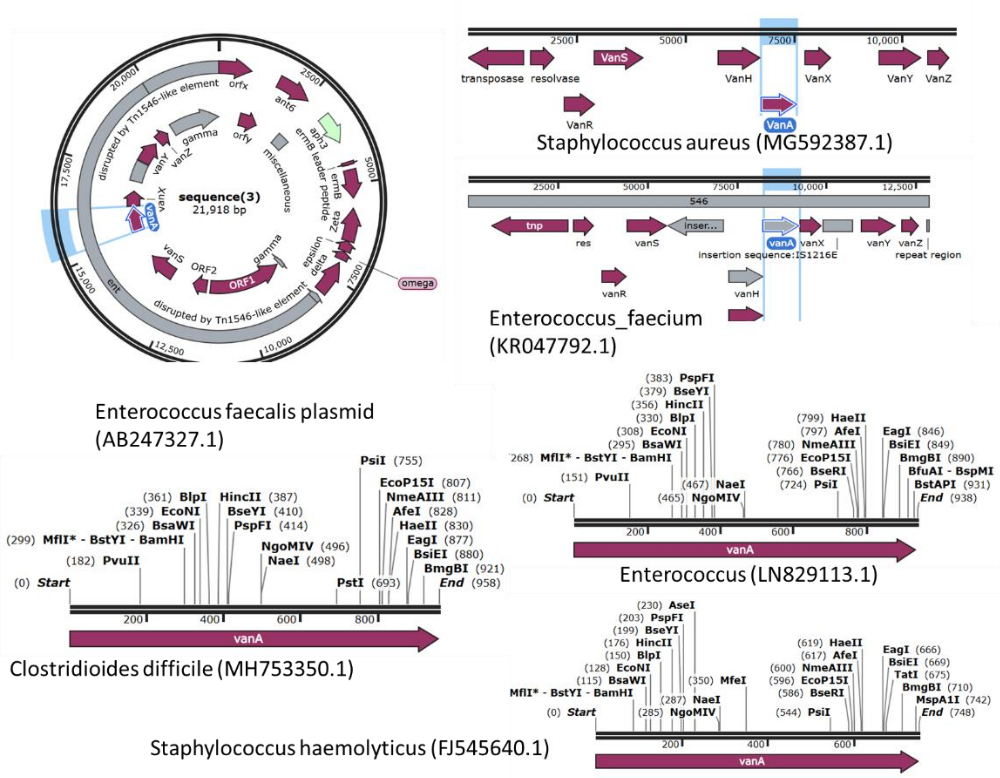
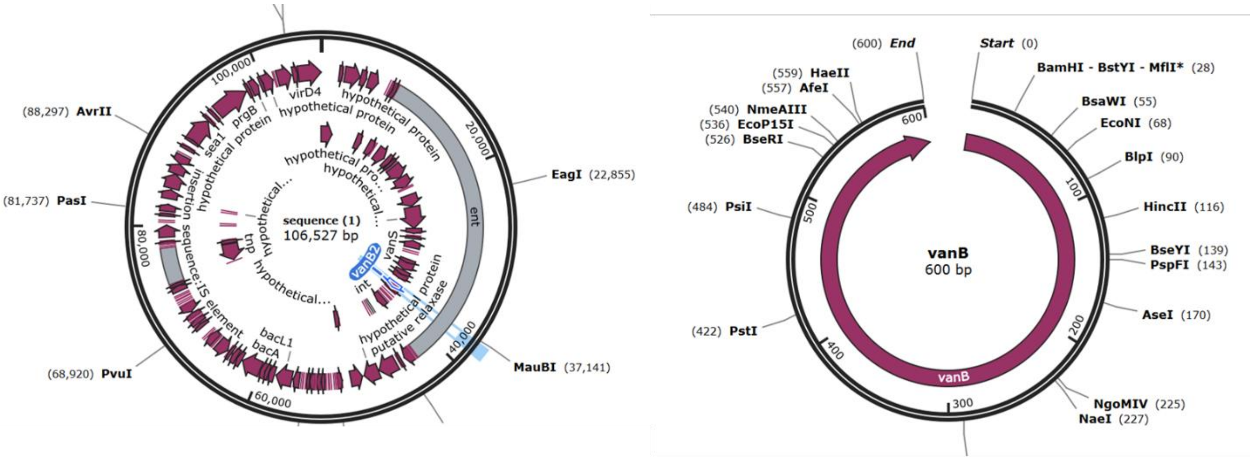
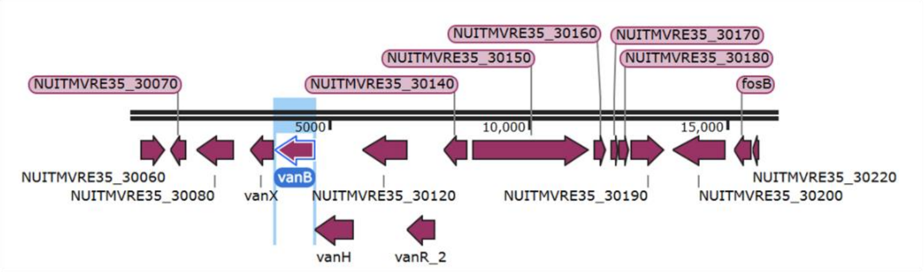


Figure 5: Genomic context and restriction map of the VanA gene in *Staphylococcus aureus* and Enterococcus faecium



Enterococcus faecalis (AF72363.1)

Enterococcus_faecium (KF052035.1)



Enterococcus gallinarum (BTSO01000022.1)

Figure 6: Genomic context and restriction map of the VanB gene in *Staphylococcus aureus* and Enterococcus faecium

Virulence factor analysis

The analysis of virulence genes across different species identified multiple functions along with different positions that also interacted with genes related to vancomycin resistance. *Enterococcus faecalis* contains the esp gene which aids biofilm formation on plasmids that are closely positioned to the VanA resistance locus. *Enterococcus gallinarum* displayed the chromosomal gelE gene which is regularly present in clinical isolates yet shows no connection to resistance loci. The hla toxin gene from *Staphylococcus aureus* exists chromosomally while maintaining a distant location relative to Van resistance genes. The isolated *Clostridioides difficile* possessed tcdA enterotoxin which does not contribute to vancomycin resistance. The plasmid-based icaA gene from *Staphylococcus haemolyticus* supported biofilm formation but did not contain any detected Van genes (Table 3).

Table 3: Virulence genes identified in selected bacterial species, including their function, genomic location, and proximity to VanA/VanB gene

Species	**Virulence Gene**	**Function**	**Location (Chromosome/Plasmid)**	**Proximity to VanA/VanB**	**Notes**
Enterococcus faecalis	esp	Enterococcal surface protein	Plasmid	Nearby VanA	May aid in biofilm formation
Enterococcus gallinarum	gelE	Gelatinase (degrades collagen)	Chromosome	Not nearby	Typical in clinical isolates
Staphylococcus aureus	hla	Alpha-hemolysin (toxin)	Chromosome	Not nearby	Common in MRSA
Clostridioides difficile	tcdA	Enterotoxin A	Chromosome	No Van genes	Part of pathogenicity locus
Staphylococcus haemolyticus	icaA	Biofilm formation (adhesin)	Plasmid	No Van genes	Important for device infections

Discussion

The study establishes an evident link between VanA and VanB gene sequence similarities and bacteria species pathogenicity. A research by Arias and Murray (2012) shows *Enterococcus faecalis* and *Enterococcus gallinarum* have Van gene sequences identical to E. faecium while both host pathogenic factors esp and gelE that allow biofilm development and extracellular matrix destruction (Arias and Murray, 2012). Vancomycin-resistant Enterococcus strains commonly possess multiple virulence factors leading to greater clinical significance which matches these study results (Willems et al., 2023). The multidrug-resistant strains of *Staphylococcus aureus* and S. haemolyticus revealed high VanA identity results which correlated with two virulence factors namely hla and icaA in line with existing research on horizontal gene transfer in staphylococci (Meng et al., 2025).

Together with resistance islands and mobile genetic elements Van genes demonstrate a capacity to enable horizontal gene transfer that strengthens the presence of resistance and virulence features in hospital environments. The occurrence of VanB showed restricted distribution to Enterococcus species.

The practical use and importance of VanA along with VanB genetic elements consists of two main aspects. The genes present consistent patterns across many sequences so they function as dependable indicators in medical tests and antimicrobial resistance monitoring systems. PCR and qPCR along with LAMP detection methods have already been improved as diagnostic tools because their target areas demonstrate stability (Foo et al., 2020). Knowledge of VanA and VanB genes' genetic structure together with their regulatory elements allows researchers to develop therapeutic approaches that use targeted inhibitors or CRISPR-based gene disruption methods to restore sensitivity to vancomycin.

Conclusion

Integrated surveillance of resistance and virulence genes becomes essential because of the established connection between Van gene homology and infection severity. The extensive spread of these elements through mobile components creates persistent infection control challenges yet offers defendable targets for fighting infections that are resistant to multiple drugs.

References

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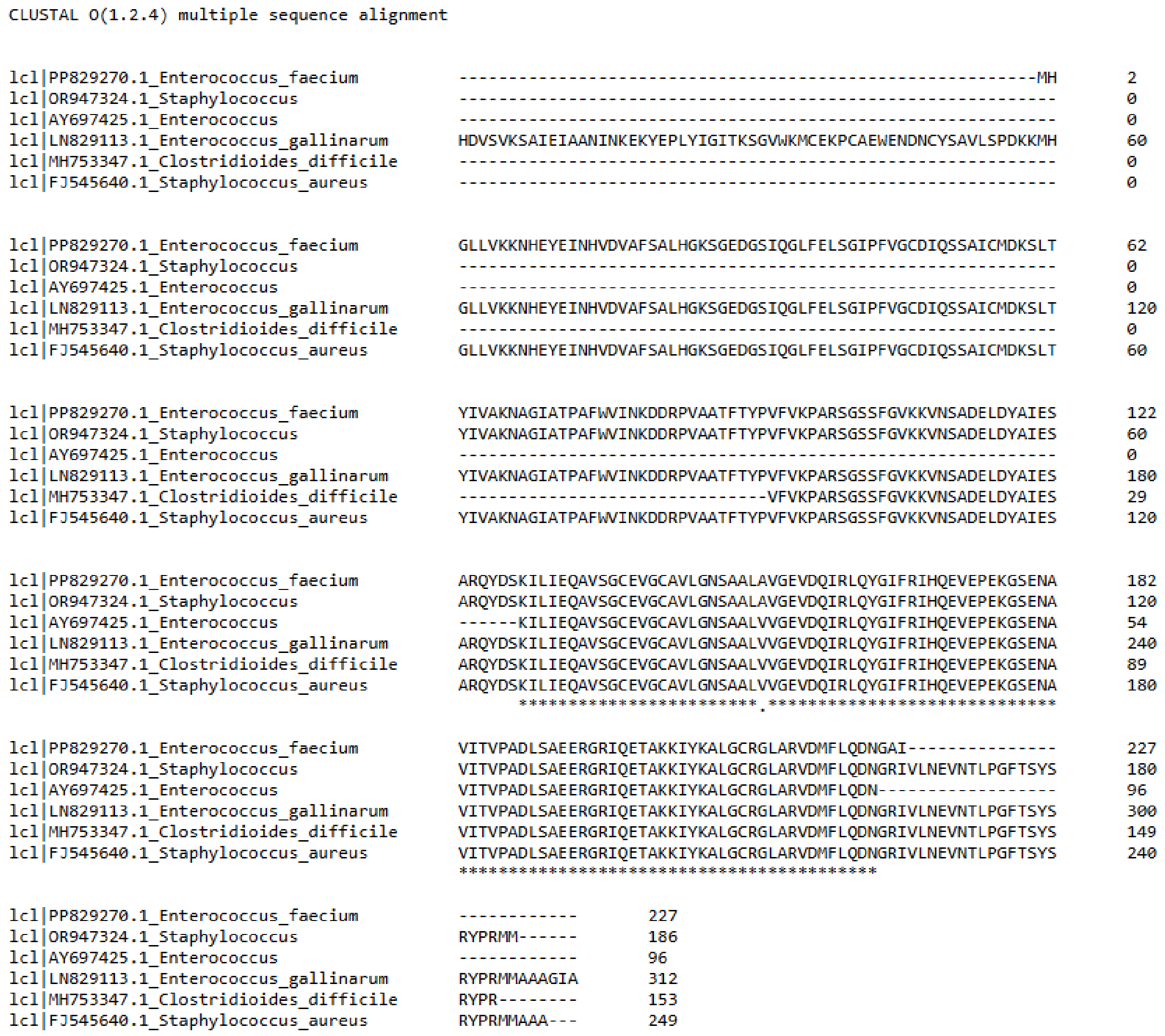
WILLEMS, R. P., VAN DIJK, K., VEHRESCHILD, M. J., BIEHL, L. M., KET, J. C., REMMELZWAAL, S. & VANDENBROUCKE-GRAULS, C. M. 2023. Incidence of infection with multidrug-resistant Gram-negative bacteria and vancomycin-resistant enterococci in carriers: a systematic review and meta-regression analysis. *The Lancet Infectious Diseases,* 23**,** 719-731.

Appendix 1

VanB protein sequeneces MSA



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CLUSTAL O(1.2.4) multiple sequence alignment


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VanB Protein sequences MSA

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CLUSTAL O(1.2.4) multiple sequence alignment


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