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Horizontal gene transfer and its role in spreading resistance genes among bacterial populations

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Abstract

Background: Horizontal gene transfer (HGT) is a critical process that enables bacteria to acquire and disseminate genetic material, particularly antibiotic resistance genes. Despite extensive research, there is still uncertainty about the role of HGT in the rapid spread of resistance genes across bacterial populations and its implications for public health. **Main Body:** This review addresses how HGT mechanisms contribute to the spread of antibiotic resistance, with a specific focus on the roles of mobile genetic elements (MGEs) like plasmids, transposons, and integrons. This study employed a comprehensive survey of peer-reviewed literature, focusing on studies from the past decade, which examine HGT mechanisms, MGEs, and the spread of antibiotic resistance. Results showed that environmental and anthropogenic factors, such as antibiotic use in agriculture, wastewater discharge, and hospital settings, significantly amplify the occurrence of HGT, facilitating the spread of resistance genes. The integration of biofilm dynamics with HGT is particularly concerning, as it promotes the persistence of multidrug-resistant strains. These findings underscore the need for real-time surveillance systems and environmental monitoring to track resistance gene flow across different ecosystems. By improving our understanding of HGT in both clinical and environmental settings, we can design more effective intervention strategies. **Conclusion:** The findings of this review have broad implications for public health, antimicrobial stewardship, and the development of novel therapies to combat antibiotic-resistant infections.

Keywords: Antibiotic resistance genes, Antimicrobial resistance, Bacterial infections, Biofilms, Horizontal gene transfer

1. Background

Horizontal gene transfer (HGT) refers to the non-reproductive transfer of genetic material between organisms, allowing genes to move laterally across species or strains. This process contrasts with vertical gene transfer, which involves the inheritance of genetic information across successive generations. The concept of HGT emerged prominently in the mid-20th century, following observations that certain bacteria could rapidly acquire new traits, such as antibiotic resistance.[1]

Although the molecular mechanisms of HGT – including transformation, transduction, and conjugation – have been extensively investigated, important gaps remain in understanding how environmental reservoirs, anthropogenic pressures, and microbial community dynamics collectively influence resistance gene dissemination. Increasing evidence suggests that resistance genes circulate between environmental, agricultural, and clinical settings, creating interconnected reservoirs that sustain and amplify antimicrobial resistance (AMR).

This review explores the mechanisms of HGT, focusing on its role in the spread of antibiotic resistance genes (ARGs). It also examines the types of resistance genes that are most commonly transferred, including those conferring resistance to beta-lactams, aminoglycosides, and macrolides. In addition, the review discusses the ecological and environmental factors that facilitate HGT, including the influence of anthropogenic activities like antibiotic use in medicine, agriculture, and aquaculture. The present review highlights the clinical and public health implications of HGT, emphasizing the spread of multidrug-resistant (MDR) pathogens in healthcare settings and the challenges posed by HGT in both pathogenic and commensal bacteria.

The literature for this review was gathered through an extensive search of peer-reviewed journals, including PubMed, Scopus, and Web of Science. The search terms included “horizontal gene transfer,” “antibiotic resistance,” “mobile genetic elements,” “plasmid transfer,” and “conjugation in bacteria.” Studies published between 2005 and 2026 were prioritized, focusing on the most recent advancements in HGT mechanisms, the ecological and clinical implications of resistance gene spread, and emerging

genomic tools for monitoring HGT. Key references were also drawn from books and reviews that provided foundational knowledge of HGT, as well as case studies that illustrated its role in public health outbreaks.

2. Main body

2.1. Mechanisms of HGT

Bacteria utilize several distinct mechanisms for HGT, each involving different vehicles for gene exchange. The classical modes are transformation, transduction, and conjugation, which together liberate genes from the confines of vertical inheritance.

Transformation involves the uptake of free DNA fragments from the environment into a bacterial cell. This process requires the recipient bacterium to be in a physiologically competent state, which can be naturally induced or artificially triggered. Conditions facilitating transformation include environmental stress, nutrient limitation, and specific growth phases that promote competence development.[2] Examples of naturally competent bacteria include *Streptococcus pneumoniae*, *Bacillus subtilis*, and *Neisseria gonorrhoeae*, which can internalize exogenous DNA and incorporate it into their genomes through homologous recombination.[3]

Transduction is mediated by bacteriophages, which transfer genetic material between bacterial cells.[4] Bacteriophages are viruses that infect bacterial cells. There are two primary types of transduction: Generalized and specialized. Generalized transduction occurs when a phage accidentally packages random fragments of the host bacterial DNA during assembly, transferring these genes to a new host on infection. Specialized transduction involves the transfer of specific bacterial genes located near prophage integration sites when the prophage excises incorrectly. Phages play a critical role in mobilizing these genes by facilitating their horizontal movement across bacterial populations, contributing significantly to the spread of antibiotic resistance.[5]

Conjugation is the direct transfer of genetic material between bacterial cells via cell-to-cell contact, typically mediated by conjugative plasmids or conjugative transposons. Conjugative elements possess a complex structure that includes the origin of transfer, genes encoding the mating pair formation apparatus, and relaxase enzymes that initiate DNA processing for transfer. Broad-host-range plasmids, capable of transferring across diverse bacterial species, have substantial epidemiological significance as vectors for disseminating antibiotic resistance and virulence factors across ecological and clinical settings.[6,7]

2.2. Types of resistance genes spread by HGT

Horizontal transfer plays a central role in disseminating various types of genes that help bacteria survive antibiotics and other stressors.[8] Key categories of resistance genes that spread via HGT include:

2.2.1. ARGs

Each major class of antibiotic is associated with resistance genes that have propagated through bacterial populations by HGT. These include genes that encode drug-inactivating enzymes, efflux pumps, and target modification. Many ARGs did not originate in pathogens. Instead, they often come from environmental bacteria and were later acquired by disease-causing bacteria through HGT.[9] Among the most clinically important ARGs moved by HGT are β-lactamases.[10] β-lactamases are enzymes that hydrolyze β-lactam antibiotics such as penicillins, cephalosporins, and carbapenems, thereby inactivating the drug.[11] These genes are frequently plasmid-borne and can rapidly spread between strains/species, creating abrupt shifts in resistance profiles within hospitals and communities.[12] Genes encoding efflux pump proteins contribute to multidrug resistance by actively expelling antibiotics from bacterial cells; these genes can be horizontally transferred, increasing bacterial survival under antibiotic pressure.[13] Target modification genes alter antibiotic binding sites, which reduces drug efficacy. These commonly include ribosomal modification, ribosomal protection proteins, and cell-wall target alteration modules.[14]

2.2.2. Heavy metal resistance genes (HMRGs) and co-selection

Heavy metals such as copper, zinc, mercury, and arsenic can co-select for antibiotic resistance even in the absence of antibiotic exposure through interconnected mechanisms. One major mechanism is co-resistance in which HMRGs and ARGs are physically linked on the same mobile genetic elements (MGEs) such as plasmids, transposons, or integrons, so that selection pressure for metal resistance simultaneously preserves antibiotic resistance. In addition, cross-resistance and co-regulation mechanisms contribute to this phenomenon, as shared systems, particularly efflux pumps and general stress response pathways, can enhance tolerance to both heavy metals and antimicrobial agents, thereby promoting the persistence and dissemination of multidrug resistance in diverse environments.[15]

2.2.3. Virulence factors linked to resistance

MGEs sometimes carry not only resistance genes but also virulence determinants.[16] The co-localization of virulence and resistance in one transmissible element is particularly concerning, as it can create highly pathogenic and drug-resistant strains in one step.[17] *Staphylococcus aureus*' staphylococcal cassette chromosome mec element carries the *mecA* gene conferring methicillin resistance, and may also associate with other factors influencing immune evasion and pathogenicity.[18] In enteric pathogens such as *Escherichia coli* and *Klebsiella pneumoniae*, plasmids and integrative elements have been shown to harbor combinations of virulence genes alongside antibiotic resistance determinants, illustrating how MGEs can assemble multi-trait packages and drive the spread of both virulence and resistance. Genome analyses have found that while virulence and resistance genes are not universally linked, specific combinations do co-occur more frequently than expected by chance. In some pathogens, there is a preferential association of particular virulence systems with certain ARGs on the same element or in the same strains.[19] This suggests that bacteria under intense selective pressure may favor acquisition of gene sets that enhance both survival in the host (virulence) and survival against antibiotics. The net effect is the emergence of "high-risk" clones that are both hard to treat and highly aggressive.

One documented outbreak in Germany in 2011, reported by Navarro-Garcia (2014),[20] described the emergence of *E. coli* O104:H4. A normally benign enteroaggregative *E. coli* strain had acquired a Shiga toxin phage (virulence) and an extended-spectrum beta-lactamase plasmid (resistance), producing a pathogen that caused severe disease and resisted third-generation cephalosporins.

2.2.4. MGEs carrying resistance determinants

Plasmids, transposons, and integrons constitute MGEs that are responsible for the dissemination of ARGs. Plasmids are widely recognized as major vectors of resistance spread. They facilitate rapid horizontal transfer of ARGs between strains and species in both community and healthcare settings. Transposons and related insertion sequences further enhance their mobility by enabling resistance genes to move within and between DNA molecules. This includes the transfer between chromosomal and plasmid locations, and they frequently serve as scaffolds for multi-gene resistance clusters.[21] Integrons mainly function as gene capture and expression systems capable of accumulating arrays of resistance gene cassettes, particularly Class 1 integrons, which are strongly associated with multidrug resistance. Integrons are commonly embedded within transposons and plasmids, which form highly mobile resistance platforms, and in some cases may also be linked with virulence-associated determinants, thereby contributing to the co-localization and co-dissemination of resistance and pathogenic traits.[22]

2.2.5. Ecological and environmental perspectives

The environment plays a pivotal role in the emergence and spread of resistance genes through horizontal transfer. Bacterial communities in soils, waters, and associated with plants and animals act as vast reservoirs of ARGs. HGT in these settings can mobilize resistance genes even before they are exposed to clinical antibiotics, and anthropogenic activities can accelerate the transfer of these genes.

2.2.6. Natural environments as reservoirs and hotspots

Natural ecosystems constituting soil, freshwater, and marine waters, sediments, and host-associated microbiomes act as long-term reservoirs of diverse resistance genes and MGEs. These ecosystems create repeated opportunities for HGT.[23] Soil is particularly important because it contains an enormous microbial biomass and a naturally rich "resistome." Large-scale metagenomic work supports soil as a meaningful reservoir with potential connectivity to human-associated resistomes.[24] Aquatic systems act as mixing zones where bacteria from humans, animals, and the environment co-occur, which increases contact rates among donors, recipients, and gene vectors.[25] In parallel, host-associated microbiomes such as gut, skin, and respiratory niches are dense communities where mobile elements are common, making HGT a pervasive evolutionary force and a direct route for resistance determinants to move across commensals and opportunistic pathogens.[26]

2.2.7. Anthropogenic drivers - medicine, agriculture, aquaculture

Human activity increases both the selection pressure and the connectivity needed for resistance spread. Intensive antibiotic exposure selects strongly for resistant strains and maintains plasmids and other mobile elements that carry ARGs.[27] Antibiotic use in food animals is repeatedly implicated in the selection and amplification of resistant bacteria and resistance genes that can move across ecological boundaries (animals ↔ environment ↔ humans) through multiple transmission routes.[28] Aquaculture adds an additional, often underestimated pathway: Antimicrobials used in fish farming can enter surrounding waters and sediments, where they can continue selecting for resistant bacteria and enriching resistance genes in environmental

microbiota.[29] These pressures do not act in isolation. Co-stressors, including pollutants and metals, and environmental transport processes such as runoff and wastewater discharge, can sustain and redistribute resistance determinants far beyond the original site of antibiotic use.[30]

2.2.7. Biofilms

Biofilms are not just "sticky communities"; they are HGT-friendly architectures. Their high cell density, prolonged cell-to-cell contact, and extracellular DNA-rich matrix create ideal conditions for conjugation, transformation, and transduction, and multiple reviews conclude that HGT can occur more frequently in biofilms than in planktonic cultures.[31] Plasmids, in particular, are deeply intertwined with biofilm ecology: biofilms can help stabilize plasmids in populations, while plasmids can encode traits that enhance biofilm formation—forming a feedback loop that increases both persistence and transfer opportunities for resistance genes.[32] This matters ecologically because biofilms dominate many real-world habitats such as pipes, medical devices, river stones, sediments, and microplastics, transforming these surfaces into microbial interaction hotspots that facilitate resistance gene exchange.[33]

2.3. Clinical and public health implications

The spread of antibiotic resistance through HGT is a major challenge in clinical and public healthcare settings. HGT not only accelerates the emergence of MDR pathogens but also blurs the line between harmless commensals and dangerous pathogens by enabling gene exchange among them.[34] In healthcare environments, HGT accelerates the emergence and spread of MDR organisms by allowing high-risk resistance determinants, especially plasmid-borne carbapenemases to move across strains and even species while patients, staff, and equipment provide dense contact networks.[35] This is why hospitals can see resistance expand faster than clonal spread alone would predict: the key epidemiological entity in healthcare environments is frequently the MGEs persisting across patients and wards, rather than an individual bacterial strain.[36]

2.4. Outbreak case studies linked to HGT events

There are numerous documented cases in which HGT was directly responsible for sparking outbreaks. The study by (Baker *et al.*, 2018),[37] reports that the horizontal transfer of a single azithromycin resistance plasmid (pKSR100) significantly contributed to multiple *Shigella* epidemics. The *pKSR100* plasmid facilitated epidemic outbreaks of different *Shigella* species, which contrasts with the slower spread of chromosomally inherited AMR traits. Genomic epidemiology revealed that a single conjugative plasmid carrying an azithromycin resistance gene (an *mphA* macrolide phosphotransferase) moved repeatedly between different *Shigella* species and strains in the men who have sex with men community. This plasmid transfer created multiple azithromycin-resistant *Shigella* lineages (in *Shigella flexneri* 2a, *S. flexneri* 3a, *Shigella sonnei*, etc.) that drove concurrent outbreaks, whereas resistance that had to evolve via chromosomal mutation showed a slower, incremental spread. The analysis demonstrated in real time that HGT of one plasmid facilitated epidemic emergence of Shigellosis in that network.

Another case reported by (Périchon and Courvalin, 2009)[38] is the vancomycin-resistant *S. aureus* (VRSA) incidents. *S. aureus* does not easily develop vancomycin resistance on its own, but in several patients co-infected with Vancomycin-Resistant *Enterococcus* (*Enterococcus faecalis* carrying the vanA plasmid) and methicillin-resistant *S. aureus*, the vanA resistance transposon was transferred by conjugation from Enterococcus to *S. aureus*, yielding VRSA. This was first reported in 2002 in the United States of America and has since occurred sporadically.

3. Conclusion

HGT is a central driver of antibiotic resistance dissemination across clinical, environmental, and agricultural settings. This review highlights how MGEs, including plasmids, transposons, integrons, and bacteriophages, facilitate rapid interspecies gene exchange, enabling the emergence and persistence of MDR pathogens. Environmental pressures and anthropogenic activities further amplify these transfer events, creating interconnected resistance reservoirs that transcend traditional ecological boundaries. The evidence underscores that resistance spread is not solely dependent on clonal expansion of individual bacterial strains but is frequently mediated by the movement of genetic elements across diverse microbial populations. Consequently, effective infection control strategies must incorporate genomic surveillance approaches capable of tracking resistance genes and mobile elements alongside bacterial lineages. These conclusions reinforce the importance of integrating molecular epidemiology, environmental monitoring, and antimicrobial stewardship programs to mitigate resistance dissemination. A deeper understanding of HGT dynamics will be critical for developing targeted interventions, informing public health policy, and guiding future therapeutic innovations aimed at combating the global AMR crisis.

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5. Authors' contributions

The author conceptualized the review, conducted the literature search, analyzed and synthesized the literature, and drafted and approved the final manuscript.

6. Ethics approval and consent to participate

Not applicable. This study is a review of previously published literature and does not involve human participants, human data, human tissue, or animal experimentation.

7. Consent for publication

Not applicable. This manuscript does not contain any individual person's data in any form.

8. Availability of data and materials

All data generated or analysed during this study are included in this published article. No new datasets were generated or analysed as part of this review.

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References

1. Tiwari P, Bae H. Horizontal gene transfer and endophytes: An implication for the acquisition of novel traits. Plants (Basel) 2020;9:305.
2. Winter M, Buckling A, Harms K, Johnsen PJ, Vos M. Antimicrobial resistance acquisition via natural transformation: Context is everything. Curr Opin Microbiol 2021;64:133-8.
3. Sharma DK, Misra HS, Bihani SC, Rajpurohit YS. Biochemical properties and roles of DprA protein in bacterial natural transformation, virulence, and pilin variation. J Bacteriol 2023;205:e0046522.
4. Colavecchio A, Cadieux B, Lo A, Goodridge LD. Bacteriophages contribute to the spread of antibiotic resistance genes among foodborne pathogens of the Enterobacteriaceae family-a review. Front Microbiol 2017;8:1108.
5. Borodovich T, Shkoporov AN, Ross RP, Hill C. Phage-mediated horizontal gene transfer and its implications for the human gut microbiome. Gastroenterol Rep (Oxf) 2022;10:goac012.
6. Domingues CP, Rebelo JS, Dionisio F, Nogueira T. Clinical and environmental plasmids: Antibiotic resistance, virulence, mobility, and ESKAPEE pathogens. Antibiotics (Basel) 2025;15:29.
7. Garcillán-Barcia MP, De La Cruz F, Rocha EP. The extended mobility of plasmids. Nucleic Acids Res 2025;53:gkaf652.
8. Murray LM, Hayes A, Snape J, Kasprzyk-Hordern B, Gaze WH, Murray AK. Co-selection for antibiotic resistance by environmental contaminants. NPJ Antimicrob Resist 2024;2:9.
9. Tao S, Chen H, Li N, Wang T, Liang W. The spread of antibiotic resistance genes *in vivo* model. Can J Infect Dis Med Microbiol 2022;2022:3348695.
10. Ajayi AO, Odeyemi AT, Akinjogunla OJ, Adeyeye AB, Ayo-Ajayi IB. Review of antibiotic-resistant bacteria and antibiotic resistance genes within the one health framework. Infect Ecol Epidemiol 2024;14:2312953.
11. Oelschlaeger P. β-Lactamases: Sequence, structure, function, and inhibition. Biomolecules 2021;11:986.

12. Haavisto V. Plasmids and the Spread of Antibiotic Resistance Genes. Washington, DC: American Society for Microbiology; 2023. Available from: https://asm.org/articles/2023/january/plasmids-and-the-spread-of-antibiotic-resistance-g [Last accessed on 2026 Feb 20].
13. Yu XH, Hao ZH, Liu PL, Liu MM, Zhao LL, Zhao X. Increased expression of efflux pump NorA drives the rapid evolutionary trajectory from tolerance to resistance against ciprofloxacin in *Staphylococcus aureus*. Antimicrob Agents Chemother 2022;66:e0059422.
14. Partridge SR, Kwong SM, Firth N, Jensen SO. Mobile genetic elements associated with antimicrobial resistance. Clin Microbiol Rev 2018;31:e00088-17.
15. Gillieatt BF, Coleman NV. Unravelling the mechanisms of antibiotic and heavy metal resistance co-selection in environmental bacteria. FEMS Microbiol Rev 2024;48:fuae017.
16. Morales G, Abelson B, Reasoner S, Miller J, Earl AM, Hadjifrangiskou M, *et al*. The role of mobile genetic elements in virulence factor carriage from symptomatic and asymptomatic cases of *Escherichia coli* bacteriuria. Microbiol Spectr 2023;11:e0471022.
17. Yu M, Yu T, Yu S, Li L, Chen S, Wang Y, *et al*. Drug-resistant genes, virulence characteristics, and molecular typing of clindamycin-resistant *Streptococcus agalactiae* in late pregnancy. Front Cell Infect Microbiol 2025;15:1721174.
18. Darmancier H, Domingues CP, Rebelo JS, Amaro A, Dionísio F, Pothier J, *et al*. Are virulence and antibiotic resistance genes linked? A comprehensive analysis of bacterial chromosomes and plasmids. Antibiotics (Basel) 2022;11:706.
19. Johansson MH, Petersen TN, Nag S, Lagermann TM, Birkedahl LE, Tafaj S, *et al*. Investigation of mobile genetic elements and their association with antibiotic resistance genes in clinical pathogens worldwide. PLoS One 2025;20:e0330304.
20. Navarro-Garcia F. *Escherichia coli* O104:H4 pathogenesis: An enteroaggregative *Escherichia coli*/Shiga toxin-producing *Escherichia coli* explosive cocktail of high virulence. In: Torres AG, editor. Enterohemorrhagic *Escherichia coli* and other Shiga Toxin-Producing *Escherichia coli*. United States: ASM Press; 2015. p. 503-29.
21. Guo X, Long X, Li J, Wu J, Zhu X, Zhu Y, *et al*. Effects of antibiotics and heavy metals on antibiotic resistance genes and mobile gene elements in agricultural activity. Environ Sci Eur 2025;37:87.
22. Ali N, Ali I, Din AU, Akhtar K, He B, Wen R. Integrons in the age of antibiotic resistance: Evolution, mechanisms, and environmental implications: A review. Microorganisms 2024;12:2579.
23. Delgado-Baquerizo M, Hu HW, Maestre FT, Guerra CA, Eisenhauer N, Eldridge DJ, *et al*. The global distribution and environmental drivers of the soil antibiotic resistome. Microbiome 2022;10:219.
24. Zhao Y, Li L, Huang Y, Xu X, Liu Z, Li S, *et al*. Global soil antibiotic resistance genes are associated with increasing risk and connectivity to human resistome. Nat Commun 2025;16:7141.
25. Delgado-Blas JF, Ovejero CM, David S, Montero N, Calero-Caceres W, Garcillan-Barcia MP, *et al*. Population genomics and antimicrobial resistance dynamics of *Escherichia coli* in wastewater and river environments. Commun Biol 2021;4:457.
26. Diebold PJ, Rhee MW, Shi Q, Trung NV, Umrani F, Ahmed S, *et al*. Clinically relevant antibiotic resistance genes are linked to a limited set of taxa within gut microbiome worldwide. Nat Commun 2023;14:7366.
27. Zhu YG, Gillings M, Simonet P, Stekel D, Banwart S, Penuelas J. Human dissemination of genes and microorganisms in Earth's Critical Zone. Glob Chang Biol 2018;24:1488-99.
28. Pandey S, Doo H, Keum GB, Kim ES, Kwak J, Ryu S, *et al*. Antibiotic resistance in livestock, environment and humans: One Health perspective. J Anim Sci Technol 2024;66:266-78.
29. Lastauskienė E, Valskys V, Stankevičiūtė J, Kalcienė V, Gėgžna V, Kavoliūnas J, *et al*. The impact of intensive fish farming on pond sediment microbiome and antibiotic resistance gene composition. Front Vet Sci 2021;8:673756.
30. Seethalakshmi PS, Prabhakaran A, Kiran GS, Selvin J. Genomic insights into plasmid-mediated antimicrobial resistance in the bacterium *Bhargavaea beijingensis* strain PS04. Arch Microbiol 2024;206:33.
31. Abe K, Nomura N, Suzuki S. Biofilms: Hot spots of horizontal gene transfer (HGT) in aquatic environments, with a focus on a new HGT mechanism. FEMS Microbiol Ecol 2020;96:fiaa031.
32. Metzger GA, Ridenhour BJ, France M, Gliniewicz K, Millstein J, Settles ML, *et al*. Biofilms preserve the transmissibility of a multi-drug resistance plasmid. NPJ Biofilms Microbiomes 2022;8:95.
33. Zhang X, Dong Z, Zhang S, Ma J, Liu S. Microplastic biofilm as hotspots of antibiotic resistance genes and potential pathogens. NPJ Biofilms Microbiomes 2025;12:24.

34. Von Wintersdorff CJ, Penders J, Van Niekerk JM, Mills ND, Majumder S, Van Alphen LB, *et al*. Dissemination of antimicrobial resistance in microbial ecosystems through horizontal gene transfer. Front Microbiol 2016;7:173.
35. Koh V, Cabrera R, Sridatta PS, Thevasagayam NM, Lim ZQ, Marimuthu K, *et al*. Plasmid dynamics driving carbapenemase gene dissemination in healthcare environments: A nationwide analysis of closed Enterobacterales genomes. Nat Commun 2025;16:9522.
36. Sobkowiak A, Schwierzeck V, Van Almsick V, Scherff N, Schuler F, Bessonov K, *et al*. The dark matter of bacterial genomic surveillance-antimicrobial resistance plasmid transmissions in the hospital setting. J Clin Microbiol 2025;63:e0012125.
37. Baker KS, Dallman TJ, Field N, Childs T, Mitchell H, Day M, *et al*. Horizontal antimicrobial resistance transfer drives epidemics of multiple *Shigella* species. Nat Commun 2018;9:1462.
38. Périchon B, Courvalin P. VanA-type vancomycin-resistant *Staphylococcus aureus*. Antimicrob Agents Chemother 2009;53:4580-7.

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