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Horizontal Gene Transfer and the Collapse of Bacterial Diversity under Antibiotic Stress

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ABSTRACT

Horizontal gene transfer (HGT) is a biological process that enables bacteria to exchange genetic material independently of cell division and plays a key role in the spread of antibiotic resistance (ABR). This review provides an overview of how antibiotic stress, particularly misuse and low-dose exposure, can enhance HGT and alter bacterial community diversity. Based on evidence from environmental, clinical, and agriculture studies, we highlight that antibiotic pressure can facilitate HGT through several mechanisms, including increased cell-to-cell contact, activation of stress response pathways, and the formation of biofilms that act as hotspots for genetic exchange. As HGT promotes the spread of resistance genes and improves bacterial survival under antibiotic pressure, gradually, resistant strains may become dominant within microbial communities, leading to a decline in bacterial diversity. Such loss of diversity may result in significant ecological consequences, including reduced efficiency of pollutant degradation and disruption of nitrogen cycling processes. Furthermore, emerging strategies aimed at limiting HGT under antibiotic pressure are discussed; particularly those that may help preserve microbial diversity, support ecosystem stability, and contribute to improved public health outcomes.

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1. INTRODUCTION

Bacterial diversity refers to the variety of bacterial species present within an ecosystem. Microorganisms inhabit every environment, from soil and water to the human body, and are also considered the most abundant form of life on Earth [1]. Microorganisms play a crucial ecological role in the decomposition of organic matter, nutrient recycling, and biogeochemical cycles [2]. They also form symbiotic relationships with plants, animals, and other microorganisms. High bacterial diversity contributes to ecosystem stability, resilience, and productivity [3]. HGT in bacteria, also known as lateral gene transfer, is a process in which a bacterium transfers genetic material to another bacterium that is not its next generation without the need for cell division [4]. HGT can occur within bacterial species and genera, including pathogenic, commensal, and probiotic bacteria. In pathogenic or commensal bacterial strains, their resistance genes pose a potential threat to public health because they contribute to the expansion of the resistance gene pool where the pathogenic bacteria can acquire new resistance traits [5].

Early studies in the late 1920s provided the first evidence of HGT. The recognition of DNA as the primary genetic material enabled the expansion of methodologies for studying gene transfer. Normally, HGT occurs within the same bacterial species; it crosses phylogenetic barriers, thus, bacteria are not limited to acquiring resistance from closely related species and can obtain resistance genes from evolutionarily distant bacteria in various environments [6]-[7]. The initial steps in genetic diversification begin with a point mutation or recombination that alters the bacterial genome. Genetic material of bacteria is transmitted both vertically, from parents to offspring, and horizontally via HGT. Through HGT, harmless bacteria may acquire virulence traits and become pathogenic.

HGT occurs through one of three mechanisms: direct cell-to-cell contact (conjugation), naked DNA uptake by competent cells (transformation), or bacteriophage mediation (transduction). See Figure 1, which illustrates HGT mechanisms. Plasmids, ICEs [Integrative and Conjugative Elements], integrons, or transposons are mobile genetic elements (MGEs) that are considered the driving factors of HGT [8]-[11]. Under natural conditions, most bacteria existed as biofilms, providing a favorable environment that enhanced HGT compared to planktonic cells [12]-[13]. Also, inside bacterial chromosomes, PRCIs (phage related chromosomal islands) are MGEs but rely on helper phages for mobilization. It attaches and injects their DNA into recipient bacteria and facilitating HGT [14].

GTAs (gene transfer agents) are virus-like elements retained by bacteria that also contribute to HGT, particularly under starvation or stress conditions. They release random chunks of the host's DNA fragments into the environment and later mediate HGT between bacterial cells [15]. The antibiotics can exert selective pressure on the microbial community, rendering susceptible microorganisms resistant [16]-[18]. From clinical settings where the antibiotics used in treatment and prevention (promoting the rise of ABR in hospitals and patients' bodies) [18] to agriculture, where antibiotics are used frequently in growth promotion and disease prevention in livestock and aquaculture, contributing in bacterial resistance that can spread to humans by the food chain [19]-[20].

Moreover, the hospital effluents, manure-contaminated runoff, and pharmaceutical waste will introduce antibiotics to the soil and aquatic ecosystem creating a reservoir of resistant genes and facilitating HGT, threatening both microbial biodiversity and public health [21]- [23].

Although horizontal gene transfer and antibiotic resistance have been widely discussed in the literature, relatively few review articles have specifically examined how antibiotic-induced HGT contributes to the collapse of bacterial diversity across different ecosystems. This review aims to integrate findings from environmental, clinical, and agricultural studies in order to provide a broader perspective on how antibiotic pressure reshapes microbial communities. By linking gene transfer mechanisms to ecological consequences, this review highlights the role of HGT in promoting the emergence of resistant populations and reducing microbial diversity. Based on the literature reviewed in this article, the relationship between antibiotic pressure, HGT mechanisms, and the resulting decline in bacterial diversity is summarized in Table 1.

Table 1: Summary of Key Studies and Mechanisms Linking Antibiotic Stress, HGT, and Bacterial Diversity

Key Theme	Mechanisms/ Factors Involved	Impact on Bacterial Community	Main Findings/ Consequences	References
Mechanisms of HGT	Conjugation, Transformation, Transduction, and Gene Transfer Agents (GTAs	Rapid dissemination of ARGs	Facilitates survival under changing environmental conditions.	[1], [10], [11]
Biofilms as Hotspots	High cell density and stable physical contact.	Facilitates inter-species HGT.	Biofilms act as reservoirs for Antibiotic Resistance Genes (ARGs).	[12], [13], [14]
Antibiotic Stress (SICs)	Sub-inhibitory Concentrations (SICs) of antibiotics	Activation of SOS response and RpoS stress pathway	Enhances genetic recombination and increases HGT frequency.	[41],[44],[45],[46]
Diversity Collapse	Selective pressure favoring resistant strains.	Out-competition of sensitive specialized strains.	Loss of microbial diversity leads to functional degradation of ecosystems.	[59], [60], [61], [62]
Environmental Impacts	Spread of ARGs in soil and water (e.g., via Manure).	Disruption of nitrogen cycling and nutrient turnover.	Compromised soil health and increased risk to food security.	[65], [71], [75]

2. LITERATURE SEARCH STRATEGY

A comprehensive literature search was conducted to identify relevant studies discussing horizontal gene transfer and its role in the spread of antibiotic resistance and changes in bacterial diversity. Several scientific databases were used, including PubMed, Scopus, Web of Science, and Google Scholar.

Publications from 2000 to 2025 were considered in order to capture both foundational and recent research in this field. The search was performed using combinations of the following keywords: "horizontal gene transfer", "antibiotic resistance", "bacterial diversity", "antibiotic stress", "mobile genetic elements", and "biofilms". Only peer-reviewed articles written in English and directly related to microbial gene transfer and antibiotic-driven ecological changes were included.

3. MECHANISMS OF HGT

HGT occurs primarily through three main mechanisms: transformation, conjugation, and transduction. These mechanisms differ in the way genetic material is transferred between bacterial cells, as illustrated in Figure 1.

3.1. Transformation

The process of natural transformation was first discovered by Griffith in Streptococcus pneumoniae in 1928. The Gram-negative bacteria most frequently studied include *Vibrio cholerae*, *Neisseria gonorrhoeae*, and *Acinetobacter baumannii*. Among Gram-positive bacteria, *Bacillus subtilis* and *Streptococcus pneumoniae* have been the subject of the most in-depth studies [24]. It occurs when a competent bacterial cell (naturally or artificially) uptakes a naked DNA fragment from the environment (from lysed cells). This process requires the recipient cell to be in a physiological state capable of binding and importing DNA. The DNA fragment can re-combine within the host genome or can exist as a plasmid [25]-[27].

3.2. Conjugation

It is direct DNA transfer from one cell to another through physical contact (pilus) [28]-[29]. Among bacterial communities, conjugation is a widespread process in different environments, including soil, plant surfaces, aquatic systems, sewage, and host-associated biofilms [30]. In these environments, conjugation promotes bacterial adaptation through the spread of advantageous genetic traits, such as those related to symbiotic, virulence, or resistance to antibiotics and heavy metals. Conjugation is driving the rapid evolution of bacterial genomes [30]-[32].

3.3. Transduction

It is bacteriophage-mediated DNA transfer (transduction) [31]. In this process, genes are transferred to another cell by infection and the recipient cell acquires the corresponding genetic traits. [33]-[34]. The role of transduction contribution to HGT in natural environments, deserves greater attention [35]. In addition, phages that harbor antibiotics resistance genes (ARGs) have been found in sewage, animals, surface water, and even human samples. This emphasizes the ecological significance of phages as reservoirs of resistance [36]. Show Figure 1.

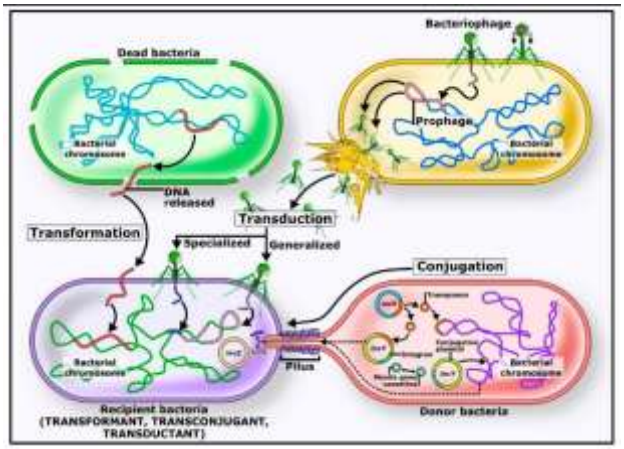

Figure 1. Comparison between major mechanisms of HGT.

4. ANTIBIOTIC STRESS (FACILITATE HGT)

Bacteria acquire antibiotic resistance through multiple pathways and as antibiotic pressure activates bacterial stress-response pathways [37, 38], like HGT, selective drug pressure, and mutations in hypermutator bacterial strains [39]. For example, DNA-damaging antibiotics (Quinolones), can induce resistance via SOS-independent recombination [40]. Moreover, studies have proven that low doses of antibiotic treatment can promote mutations that induce the resistance [40]-[43]. For instance, the SOS response could be activated by antibiotic concentrations lower than those used in clinical treatment, known as subinhibitory concentrations (SICs) [43]. Also, antibiotic-inducing lysis, like β-lactams (penicillin) or glycopeptides (vancomycin) can increase the availability of extracellular DNA in the environment and enhance transformation [44].

The lysis and release of DNA can have significant ecological consequences, influencing the distribution and diversity of bacterial populations [45]. In addition, the misuse/overuse of antibiotics (in human medication) creates a hotspot for HGT and increases pressure on microbial populations. These conditions will accelerate HGT of ARGs, promote their mobilization and spread to pathogenic species. Together, these processes are contributed in the global spread of ABR in different environments included water systems, farms, and hospitals [46]-[48]. Figure 2 summarizes these responses and their effects on gene exchange.

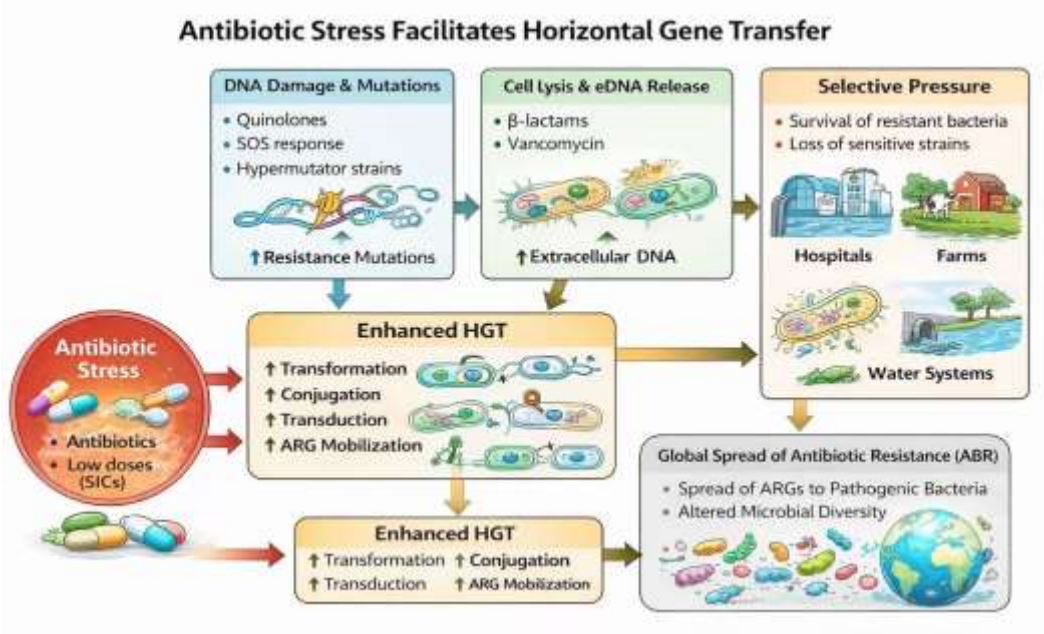

Figure 2. Antibiotic and sub-inhibitory concentrations facilitate HGT.

5. HOW TO LIMIT HGT UNDER ANTIBIOTIC STRESS

New good strategies are needed that will reduce selective pressure and genetic mobility to limit the impact of antibiotics stress. By avoiding the prolonged, sub inhibitory, or broad-spectrum treatments concentrations of antibiotics, this will significantly decrease the induced genetic transfer by antibiotics. Also, targeting MGEs, this will disrupt the machinery that allows genes (resistance gene) to transfer from one cell to other, like; inhibition of conjugative transfer by blocking relaxase activity [49], targeting enzymes like integrase and recombinase or their specific sites to block gene spread and maintain antibiotic efficacy [50].

Moreover, as antibiotics causing SOS activation [DNA damage response network], this will activate the RecA protein [DNA repair/recombination enzymes], causing prophages to excise and potentially spreading antibiotic resistance genes through HGT. To reduce this activation, Blocking RecA activity will directly stop the SOS induction, also the synergy relation between phages and antibiotics PAS (Phage-Antibiotic Synergies) are useful, as antibiotic can activate the latent viruses inside the bacterial cells this lead to eradication of persistent cells which are difficult to treat [51]-[52]. In the same context and to treat biofilm-associated ABR and HGT, Anti-Biofilm strategies have been developed [53]-[54]. These strategies vary from the use of physical methods (ultrasound and light), disrupting EPS (extracellular polymeric substances), prevention of biofilm development by Anti-QS Agents (quorum sensing inhibition) and targeting persister cells with nano-particles [54]-[55]. This will enhance antibiotic susceptibility and prevent new resistance to develop [55].

Many recent studies about the promising technology (CRISPR-Cas tools) in the field of ABR that target and cut specific DNA sequences that responsible of resistance genes or cells function, but its effectiveness varies between bacterial species due to the difference in CRISPR loci, and till now, in the field of biofilms, it remains as ongoing research area [56]-[57]. These strategies are still largely experimental, and are not yet widely used in clinical practice. Focusing on reducing selective pressure through the appropriate use of antibiotics is what has been adopted as the primary preventive ways.

6. HGT AND BACTERIAL DIVERSITY COLLAPSE

As mentioned earlier, HGT among bacteria has been shown to play a key role in the development and spread of multidrug resistance [3],[58]. When an environment (human body, soil, or water) is exposed to an antibiotic, it imposes selective pressure on the microorganisms in it. Under these conditions, antibiotic-resistant strains become more viable and multiply, while non-resistant or specialized strains (which lack resistance genes or cannot adapt quickly) are weakened or extinct [59],[60].

This leads to a decrease in microbial diversity and promotes the spread of resistance, and the dominance of dangerous strains that may be more harmful or resistant to treatment. It contributes to the spread of antibiotic-resistant diseases.

Moreover, essential ecosystem services like nutrient cycling, symbiotic interactions and detoxification are compromised due to a reduction in functional diversity inside microbial communities. This reduction came from Antibiotic pressure that selects for bacteria (which acquired resistance or survival-enhancing genes via HGT), which often outcompete a wider variety of other functionally specialized microbes and strains that are less competitive but functionally diverse, thereby diminishing or eliminating them [60].

HGT lead to genetic homogenization within bacterial community, through sharing beneficial genes between diverse bacterial species (more common trait within community). When some traits became dominant, a reduction in community diversity occurs, this lead to reduce the abundance of ecologically important bacteria. Like those who treated polluted soil and wastewater, or those responsible for degrading organic pollutants, nitrogen, and other essential ecosystem services. So, HGT decreases functional diversity at community levels [61]-[62]. Also, when a gene is shared repeatedly throughout HGT (plasmids, transposons, or phages), some of the unrelated bacteria will acquire it, reducing genetic differences and the microbial community will shift to a more uniform genetic profile, which we can call the "genetic homogenization". Genetic homogenization is a process where genetic differences are reduced, leading to increased similarity within a gene pool [63]. Also leads to reduction in functional diversity.

Recently, bioinformatics studies have proven that environmental conditions strongly affect bacterial fitness consequences of HGT [36]. Certain genes, like; ABR genes may offer a selective advantage to bacteria under antibiotics pressure [64]. Likewise, producing metabolic enzymes in the absence of an energy source will waste resources, leading to the evolution of complex regulatory systems among microbes [65]. Importantly, this varies across ecosystems [66]. In soil environments, spatial structure and availability of heterogeneous nutrients, will affect the retention of ABR genes and transferred metabolic genes. While, in aquatic environments, fluctuating conditions (pH, UV radiation, temperature, salinity ... etc.) and dilution will limit their long-term persistence. In plant associated microbes like (Rhizosphere), HGT are affected by the interplay between host-driven selection and root exudates chemical environment. So, within bacterial species, HGT affects population structure and diversity depending on ecosystem type, consequently contributing to genomic variation [66]-[67]. Despite its crucial role, systematic experimental studies on environmental impacts and the persistence of HGT remain lacking.

7. BIOFILMS FACILITATING HGT

Since biofilms provide a dense and stable bacterial population, by this close proximity, the conjugation is increased (direct transfer of plasmid and mobile genetic elements between cells). Sub-lethal concentrations of antibiotics found within biofilms select for resistance variants. Lead to the dissemination of resistance genes via plasmids and other MGEs [68]. Since, Biofilms provide a favorable environment for horizontal gene transfer the role of biofilms as hotspots for genetic exchange is illustrated in Figure 3.

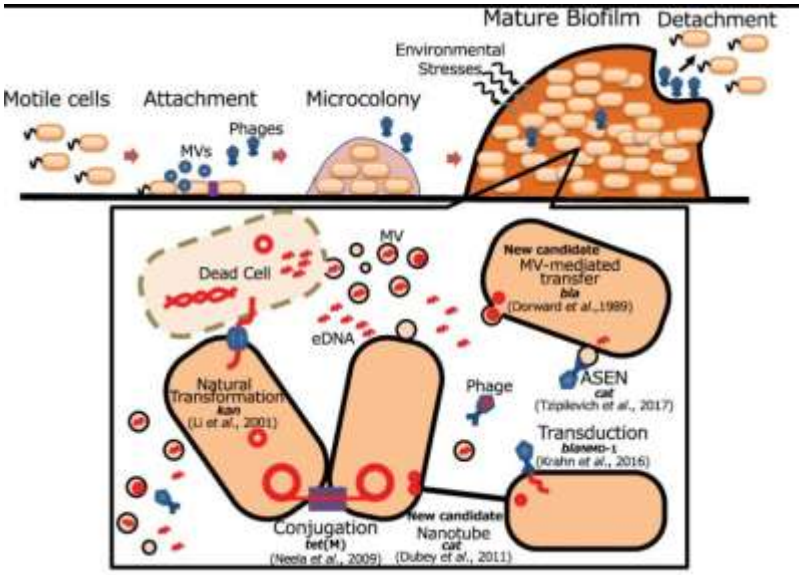

Figure 3. Biofilms as hotspots for HGT and genetic homogenization under antibiotic pressure.

The biofilms contain various bacterial species, facilitating inter-species HGT, including the transfer of ARGs across genera or phyla. This contributes to the homogenization of the genetic structure between bacteria, that is, many different species begin to have the same genes (especially resistance genes) due to their transmission through horizontal transport. As a result, these diverse species begin to resemble genetically rather than being distinct. This leads to a disturbance in environmental specialization, that is, each type of bacteria loses its unique role in the environment, whether in breaking down certain compounds, living with specific species or contributing to a certain dietary cycle. Over time, all this leads to a decrease in general microbial diversity, because the bacterial community becomes composed of genetically similar and less specialized species, which impairs the functioning and stability of the ecosystem [69], accelerating the collapse of bacterial diversity under antibiotic stress.

eDNA (extracellular DNA), derived from cell lysis or actively secreted by bacteria, significantly contributes to biofilm development, structural stability, and resilience against environmental stressors. It is important to understand the role of eDNA in biofilms, due to its implications on both human health and the ecological system [70].

8. ECOLOGICAL CONSEQUENCES

The ecological consequences of increased horizontal gene transfer under antibiotic pressure include the expansion of resistant bacterial populations and the reduction of microbial diversity. These potential impacts are summarized in Figure 4.

Figure 4. The misuse of antibiotic in hospitals and agriculture will release ARGs to the environment, enhances HGT in biofilms, and accelerates genetic homogenization.

Reducing functional diversity of bacteria in soil and aquatic environments, e.g.: pollutant-degrading communities shrink when resistance traits dominate, making ecosystems more vulnerable to disturbances [71], and disruption in plant-microbial interactions such as in rhizobia (nitrogen-fixing bacteria) [72]. Rhizobia, Gram-negative bacteria, live in soils and inter an intracellular symbiotic relationship with plant hosts (nodule formation on roots) [73], and reduce N_2 into ammonia (most bioavailable forms for plant). Symbiotic nitrogen fixation (SNF) is the most efficient biological nitrogen fixation system in nature. About 40 million tons of N are fixed by the rhizobium–legume SNF per year, accounting for about 65% of the total input of biological nitrogen fixation in agricultural systems [74]. By HGT, and the spread of symbiosis genes to inefficient strains, this made the bacterial community genetically rich but, functionally poor. Reducing nitrogen fixation and lowering soil fertility and crop yield [75]. As microbes play an important role in nutrient cycling, pollination and soil fertility. As these processes are important for food production, the reduction in biodiversity of these microbes will affect these processes and lead to the loss in crop yields and low quality of food. Also, this loss of biodiversity will provide an opportunity for harmful pathogens to thrive, causing disease outbreaks in crops which will subsequently affect both the quantity and safety of food [75]-[76].

9. CLINICAL CONSEQUENCES

Human pathogens can acquire ARGs from non-pathogenic environmental bacteria through HGT by MGEs like plasmids, intergons and bacteriophage [3]. Wastewater from hospitals, act as mixing areas where residual antibiotics, high bacterial density, and diverse resistance genes will promote genetic exchange between species. Hence, environments facilitate resistant infections (disease derived from environmental gene pool) [65]. Some studies revealed that migrating across multiple bacterial species, leading to nosocomial infections to spread. 11 out of 12 top WHO priority pathogens are naturally transformable; which highlights the part that HGT plays in hospital outbreaks [76],[6].

10. MANAGEMENT STRATIGIES

To archive one Health–based and multi-layered approach that combines environmental protection, agricultural practices, and human health, an effective management of ABR and its associated consequences on microbial diversity is needed. Stewardship Programs (ASPs) are important to minimize antibiotic exposure by ensuring the right dose, duration, time, and drug. Also, the primary goal of the ASP is to optimize clinical outcomes while minimizing unintended consequences related to antimicrobial usage, such as toxicities. Its success came from the effective collaborative effort between medicine, pharmacy, and infection control (IC), microbiology, and information technology [77].
So, such programs are important in: -

- drug optimization
- restrictions on broad spectrum antibiotics
- reducing costs
- improving patients safety

From the environmental overview above, hospital effluent and wastewater treatment plants represent reservoirs for ARGs and can spread ABR through HGT to environmental microbial communities, which can later re-enter human and animal populations. So, we need to strengthen the technology of wastewater treatment like: (membrane filtration, UV light technology as disinfectant, Ozonation, nanotechnology, Phytoremediation) to improve water quality and reduce the resistant gene flow in the environment. It is worth noting the environmental-derived outbreaks of multidrug-resistant infections like: beta-lactam resistance, colistin resistance, and vancomycin resistance that leads to serious public health issues [78]. Another steps at the agricultural level and to reduce resistance in animal-associated microbiomes, are to restricting the non-therapeutic use of antibiotics for growth promotion and assess veterinary stewardship. Also, controlling agricultural waste and manure management will further limit the spread of ABR. New drugs and technologies show promising potentials to limit HGT:

- Bacteriophage genetic engineering [79].
- Biofilm inhibition: Quorum sensing inhibitors [80].
- CRISPR Antimicrobial strategy [57].
- Conjugation Inhibitors – COINs [81].

Surveillance systems depend on data from environment, agriculture, and clinics should be used in early detection of microbial diversity collapse. Such surveillance in the field of resistance and microbial diversity enables timely intervention before irreversible ecosystem dysfunction occurs. Restore microbial balance after antibiotic exposure with targeted probiotics to restore lost microbial function and enhance natural gut diversity. Additionally, encourage dietary fiber intake to support commensal microbes [82].

11. KNOWLEDGE GAPS

Despite what we know about HGT and antibiotic resistance, there are still important questions that need answers:

- Real-world Studies: Most research happens in labs. We need more studies on how HGT works in real environments like soil and water over long periods.
- The Cost of Resistance: We don't fully understand if bacteria lose their "fitness" or health when they carry new resistance genes. Future research should check if these bacteria can survive long-term without antibiotic pressure.
- Other Transfer Methods: Most studies focus on plasmids. We need to look more at other ways genes move, such as through viruses (phages) or small vesicles.
- Impact on Ecosystems: It is still unclear how much bacterial diversity we can lose before nature stops performing its basic jobs, like cleaning water or helping plants grow.
- New Solutions: We need to find new medicines that stop the "transfer" of genes itself, not just medicines that act like biocides.

12. CONCLUSION

This review highlights the critical role of HGT as a primary driver of bacterial evolution and the dissemination of antibiotic resistance genes (ARGs) under environmental and clinical stress. We have demonstrated that antibiotic pressure, particularly at sub-inhibitory concentrations, acts as a potent catalyst for genetic exchange by activating stress-response pathways (e.g., SOS response) and promoting biofilm formation. The most alarming implication of this synergy is the "diversity collapse" within microbial communities, where the dominance of resistant strains leads to a loss of specialized bacterial species and essential ecosystem functions. Beyond clinical settings, this genetic homogenization poses a severe threat to soil health, nutrient cycling, and global food security. Future strategies must shift from merely developing new antibiotics to inhibiting HGT mechanisms and preserving microbial diversity. Protecting these "invisible" ecological networks is vital to mitigating the long-term impacts of the antibiotic resistance crisis.

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