Proceedings of ICMMGH 2026 Symposium: Cell and Molecular Biology: Understanding Immune Cell Functions
DOI: 10.54254/2753-8818/2026.PJ31041

Horizontal Gene Transfer: An Underestimated Driving Force for the Dissemination of Antibiotic Resistance Genes

Haoyang Du

Clongowes Wood College, Dublin, Ireland
sunnydu1225@outlook.com

Abstract. Antibiotic resistance (AMR) threatens the foundation of modern medicine, accelerated by horizontal gene transfer (HGT) of antibiotic resistance genes (ARGs) among bacteria. This review examines HGT as the principal mechanism connecting immense ecological reservoirs of ARGs to clinical pathogens. The global “resistome” encompasses ancient ARGs in natural environments such as soil and water, which have intensified in human-impacted systems including wastewater treatment plants and gut microbiomes. The main molecular processes driving HGT—conjugation, transformation, and transduction—serve as key routes for ARG dissemination. Human activities, including antibiotic overuse, co-selection by heavy metals and biocides, and the generation of anthropogenic terrestrial hotspots, further promote resistance selection and increase gene transfer rates. Tackling AMR requires shifting from a 'clinical-pathogen-focused’ view to an ecological and evolutionary approach based on surveillance and management of gene flow at the human-animal-environment interface. Prospective approaches could concentrate on the direct disruption of HGT mechanisms and MGEs, as these could be considered as novel and more sustainable ways to mitigate the spreading of resistances.

Keywords: Horizontal Gene Transfer, Antibiotic Resistance, Conjugation, Transformation, Transduction

1. Introduction

The escalating problem of antimicrobial resistance (AMR) poses one of the 21st century's most significant threats to global health, with bacteria disseminating resistance far faster than new antimicrobials are developed. Therefore, it is critical to understand the ecological and molecular mechanisms driving the rapid spread of resistance.

Antibiotic resistance genes (ARGs)—genetic elements encoding resistance—are not a recent phenomenon. Ancient metagenomic data suggest ARGs are ancient components of the microbial pangenome, co-evolving over millennia [1]. The collection of these genes, found in pathogenic and non-pathogenic bacteria across all environments, is known as the 'resistome.’ This vast genetic repository is associated with natural (e.g., soil) and human-related (e.g., wastewater, microbiomes) environments. The clinical AMR problem stems not from the resistome's existence but the unprecedented availability of these ARGs from environmental reservoirs to human pathogens. This transmission is primarily driven by horizontal gene transfer (HGT). The powerful effect of HGT is

DOI: 10.54254/2753-8818/2026.PJ31041

highlighted by cases like blaCTX-M extended-spectrum \beta-lactamase genes, which mobilized from environmental Kluyvera species into pathogenic Enterobacteriaceae [2]. This mobility transforms a quiescent environmental gene pool into a direct clinical hazard.

HGT is not a single mechanism but a set of molecular processes by which bacteria share genetic material. These include conjugation (cell-to-cell contact), transformation (uptake of environmental DNA), and transduction (viral-mediated transfer), among others. These mechanisms differ in their genetic cargo, transfer rates, and host ranges, collectively forming a powerful system for genetic exchange.

This review will examine the dissemination of ARGs by first outlining the key ecological reservoirs of the resistome (Chapter 2). We will then detail the molecular mechanisms of HGT—conjugation, transformation, and transduction—driving ARG mobility (Chapter 3). Finally, we will address the anthropogenic factors and selective pressures, such as antibiotic use and pollution, that accelerate these transfers.

2. The resistome–ecological reservoirs of antibiotic resistance genes

The resistome refers to the collection of all ARGs and their precursors present in both pathogenic and non-pathogenic bacteria that are accessible for transfer via HGT. This vast genetic resource is not the result of modern antibiotic use but a long-standing and evolving feature of the microbial world [1]. However, human activities have dramatically accelerated the mobilization and spread of these genes, transforming them from local ecosystems to global public-health disasters. The key issue is not the presence of ARGs per se, but their increasing association with MGEs that allow movement across phylogenetic and ecological barriers. Understanding the structure of these reservoirs—where these genes aggregate and recombine—holds the key to the scope of the antibiotic resistance problem. Broadly, these reservoirs can be categorized as environmental or host-associated, which are increasingly linked as a consequence of anthropogenic impacts.

2.1. Environmental reservoirs: soil and water

Soil and water environments have long served as the primary natural reservoirs of ARGs. These settings harbored resistance elements for millions of years. But human activity has produced artificial environments that magnify vessels and portals for transfer to clinically relevant bacteria.

2.1.1. Ancient origins and natural environments

ARGs have been detected in ancient and remote environments, implicating them as active participants in natural microbial ecology and potentially serving as defense molecules in the chemical warfare among competing microbes [1]. Active ARGs, such as those encoding resistance to β-lactams, tetracyclines, and aminoglycosides, have also been progressively identified in ancient permafrost cores, unspoiled cave microbiomes, and terrestrial sediments. This is from the perspective that the resistome is a natural portion of the microbial pangenome. ARGs are widespread in clean environments where they exist in ecological balances and are stabilized by non-pathogenic environmental bacteria. The primary concern today is that anthropogenic pollution is mobilizing these ancient genes from their wild hosts into pathogens, disrupting long-established ecological balances.

DOI: 10.54254/2753-8818/2026.PJ31041

2.1.2. Anthropogenic hotspots: wastewater treatment plants and effluents

While ARGs originate from natural habitats, certain human-made systems act as hotspots for their amplification and exchange. Wastewater treatment plants (WWTPs) are a key example [3]. They receive domestic, hospital, and industrial discharges, creating a one-of-a-kind selective environment. It is a mix of commensal and pathogenic bacteria and sub-inhibitory levels of a variety of antibiotics, disinfectants, and heavy metals that exert a strong selective pressure [4]. The overarching definition that encompasses these hotspots is a high-density bacterial population combined with a strong antibiotic selection pressure, maximizing the chance for HGT.

Metagenomic analysis revealed that while WWTPs may reduce bacteria, their effluents can occasionally be enriched with ARGs and MGEs at higher relative abundances than those in the influents [3]. Such installations are genetic exchange hubs, where ARGs from environmental and clinical bacteria can be exchanged, commonly with the assistance of plasmids, transposons, and integrons, and introduced into downstream aquatic ecosystems [5].

2.2. Host-associated reservoirs

The human and animal microbiomes form another important component of the global resistome. Dense microbial populations within hosts serve as reservoirs where ARGs are easily exchanged with invading pathogens.

2.2.1. The animal gut: agriculture and aquaculture

Intensive animal farming practices have significantly expanded the host-associated resistome. Antibiotics are widely used in livestock and aquaculture to promote growth, prevent disease, and treat infections, subjecting the gut microbiota of farm animals to tremendous selective pressure [6]. This has resulted in a high-density reservoir of resistant bacteria, often carrying mobile plasmids capable of interspecies gene transfer. These ARGs may spread into the broader environment via manure as fertilizer or directly into the aquatic environment from fish farms. Moreover, the evidence is mounting towards an animal production environment resistome-human pathogen resistome direct connection, implicating the food chain as a major conduit for resistance transmission [6].

2.2.2. The human microbiome: a commensal arsenal

The human microbiome, especially in the gut, oral cavity, and skin, is a dynamic reservoir of ARGs. Non-pathogenic gut flora can contain numerous resistance genes, forming an "on-demand gene supply" for pathogens [7]. Antibiotic treatment disrupts this balance by suppressing susceptible populations, allowing resistant strains to proliferate. This enhances both the abundance of ARGs and their potential for exchange with transient or invasive pathogens.

Globalization further accelerates resistome intermixing: international travel has been recognized as a major factor in the acquisition and dissemination of multi-drug resistant organisms [8]. Thus, the human resistome is a fluid and adaptable system, influenced by diet, health status, and antibiotic use.

2.3. Connectivity and flow among reservoirs

While environmental and host-associated resistomes are treated as distinct, in reality their boundaries are porous. Gene exchange occurs continuously across ecological, spatial, and temporal

DOI: 10.54254/2753-8818/2026.PJ31041

scales.

2.3.1. Clinical genes from environmental origins

Many clinically important ARGs can be traced to environmental origins. Such “opportunistic” resistance genes thrived innocuously in environmental microorganisms for ages before being seized by human pathogens. A classic example is the family of blaCTX-M extended-spectrum β-lactamase (ESBL) genes, which are increasingly being recognized as the major contributors to resistance in Enterobacteriaceae. Genomic analyses have identified their ancestral source as chromosomes of environmental Kluyvera spp., which were captured by plasmids and then dispersed worldwide in clinical isolates [2]. In the same manner, the plasmid-mediated quinolone resistance (qnr) genes, e.g., qnrA, appear to have a prevailing origin from the aquatic bacterium, Shewanella algae [9]. These instances highlight an important paradigm: the environment resistome is the ultimate source of new resistance determinants for the pathogens of the clinic.

2.3.2. Evidence from genomic tracing studies

Advances in metagenomic and comparative genomics have enabled detailed tracking of ARG transfer across ecosystems. By analyzing the genetic contexts of ARGs and associated MGEs, researchers have traced gene flow from agricultural systems to human pathogens. Recent studies demonstrate convergence of certain ARGs and MGEs between aquaculture environments and the human gut, evidencing cross-reservoir transmission [6]. Similarly, the plasmid-mediated colistin resistance gene mcr-1, first identified in agricultural settings in China, rapidly spread to clinical isolates and environmental samples worldwide, highlighting the speed and scale of global ARG propagation.

The resistome represents a highly connected ecological matrix, linking ancient environmental gene pools with anthropogenic hotspots and host-associated microbiomes. Having these genes is a natural phenomenon, but their accelerated mobilization into pathogens is a human-driven crisis. Understanding these reservoirs and their interconnections sets the stage for the next discussion: how these genes move—the mechanisms of HGT explored in Chapter 3.

3. Mechanisms of horizontal gene transfer–molecular conduits

The large, interconnected reservoirs of ARGs described previously would have limited clinical impact without the potent molecular mechanisms that mediate their dissemination among bacteria. HGT renders the resistome dynamic, enabling resistance determinants to migrate across species and even phylum boundaries [1]. This process transforms environmental ARGs into clinical risk. The three principal mechanisms—conjugation, transformation, and transduction—each have particular features, donor-recipient requirements, and genetic playloads. A fourth, less well-characterized pathway, mediated by GTAs, also contributes to gene flow. Understanding these processes is fundamental to explaining how antibiotic resistance spreads so quickly.

3.1. Conjugation: plasmid-mediated transfer

Conjugation is one of the most important mechanisms driving the spread of multidrug resistance in clinical settings, as it efficiently transfers large and complex genetic elements across wide phylogenetic distances.

DOI: 10.54254/2753-8818/2026.PJ31041

3.1.1. Machinery and process

Conjugation is a contact-dependent process that facilitates the transfer of plasmid DNA from a donor to a recipient cell. It is regulated by transfer (tra) genes encoded within the conjugative element. These genes produce a type IV secretion (T4SS), a complex molecular apparatus central to DNA transfer. A key element of this system is the pilus, an extracellular appendage that initiates contact with a potential recipient and brings cells together. Once connected, a single strand of plasmid DNA is cleaved and transferred unidirectionally from donor to recipient. Each cell then synthesizes a complementary strand, resulting in two plasmid-bearing donor-capable cells. This process enables rapid, exponential dissemination of plasmids within bacterial populations.

3.1.2. Broad-host-range plasmids and multidrug resistance

Conjugation is particularly significant because it can mobilize broad-host-range plasmids that carry multiple ARGs, thereby conferring an instant MDR phenotype upon recipients. Many such plasmids are "promiscuous," capable of persistence across multiple bacterial species, facilitating interspecies or even interphylum gene flow. For example, plasmids carrying genes like blaNDM-1 (bla New Delhi metallo-β-lactamase) have spread among different Gram-negative pathogens worldwide primarily through conjugation. This mechanism is effective even within biofilms, where dense cell proximity enhances plasmid transfer [10]. The development of conjugation inhibitors as a therapeutic target further emphasizes the relevance of this pathway in ARG dissemination [11].

3.2. Transformation: free DNA uptake

In contrast to the structured machinery of conjugation, transformation involves the uptake of exposed, foreign DNA (eDNA) from the environment and its potential incorporation into the host genome.

3.2.1. Natural competence and environmental triggers

To undergo transformation, bacterial cells must enter a transiently competent state in which they express proteins necessary for DNA binding, uptake, and recombination. This state is typically induced by environmental cues such as nutrient limitation, high cell density (quorum sensing), or stress. Interestingly, a major inducer of competence in some bacteria is the presence of sublethal levels of antibiotics. For instance, some β-lactam antibiotics promote competence in S. pneumoniae by inhibiting cell wall synthesis and thus leading to the induction of genes necessary for DNA uptake [12]. This creates a self-reinforcing feedback loop: antibiotic exposure can directly promote the gathering of resistance genes for it. The eDNA available for uptake may be derived from locally lysed bacteria, creating a communal pool of genetic material, including ARGs.

3.2.2. The role of mosaic genes in resistance

Transformation contributes to antibiotic resistance primarily through the modification of existing chromosomal genes. If the internalized fragment has homology to a stretch of the recipient chromosome, it can be ligated via homologous recombination. And indeed, if such an event occurs more than once with DNA from multiple, albeit related, sources, it may give rise to "mosaic" genes —chimeric sequences derived from multiple parents. This process underlies the evolution of penicillin resistance by pathogens like Streptococcus pneumoniae and Neisseria gonorrhoeae. They

DOI: 10.54254/2753-8818/2026.PJ31041

have maintained their penicillin-binding protein (PBP) genes as mosaics with DNA from closely related commensals, which results in reduced β-lactam antibiotic binding. Transformation thus provides a mechanism for gradual, stepwise evolution of resistance via allelic replacement [1].

3.3. Transduction: bacteriophage as a carrier

Transduction is a mechanism of HGT mediated by bacteriophages, in which bacterial DNA is packaged into viral particles and transmitted to new host cells during phage infection cycles.

3.3.1. Generalized vs. specialized transduction

Transduction occurs in two main forms. Generalized transduction is a mistake-driven process during the phage lytic cycle: occasionally, fragments of bacterial DNA, chromosome, or plasmids, are mistakenly packaged into phage heads. These defective phages cannot propagate infection but can inject this DNA into new hosts, thereby acting as vehicles for gene transfer.

In contrast, specialized transduction occurs during the lysogenic cycle when a temperate phage excises imprecisely from the host genome, carrying neighboring host genes with it. These genes become part of the phage genome and are propagated to subsequent bacterial hosts during infection.

3.3.2. Metagenomic evidence for phage-mediated ARG spread

Although once thought to be minor in ARG dissemination, accumulating evidence highlights the virome as a significant contributor. Phage fractions isolated from environments such as freshwater, wastewater, and the human gut have been shown to contain functional ARGs [13]. Phages thus act as a vast, dynamic gene reservoir, shuttling resistance genes among bacteria, particularly within dense microbial systems such as the gut microbiome [14]. While ARGs are relatively rare in phage genomes compared with plasmids, the enormous abundance and rapid turnover of phages mean even infrequent transfer events can have substantial epidemiological impacts [15].

3.4. Gene transfer agents: a phage-like mechanism

GTAs are defective bacteriophage-like particles that package random segments of the host DNA and that are generated by certain bacteria and archaea. They are a distinct, hybrid type of HGT.

3.4.1. Characteristics and production

Unlike infectious bacteriophages, GTAs do not package their own DNA and thus are non-infectious in the conventional sense. Their production is controlled by the host's cellular machinery and is often associated with quorum sensing, and a fraction of cells lyse to release GTA particles. Each GTA particle carries a small, random piece of the host genome, which can subsequently be delivered into recipient cells, enabling gene exchange between otherwise unrelated bacteria.

3.4.2. Potential for high-frequency interphylum transfer

Some GTAs exhibit remarkably broad host ranges, with documented transfers spanning distinct bacterial phyla [1]. This promiscuity suggests that GTAs could play a meaningful ecological role in the spread of ARGs in complex, polymicrobial systems such as soil and marine sediments. Despite their potential importance, GTAs remain the least studied HGT mechanism in the context of

DOI: 10.54254/2753-8818/2026.PJ31041

antibiotic resistance. Their unique features imply that they may constitute a hidden but significant pathway for ARG mobilization among distantly related environmental bacteria.

While these HGT mechanisms are intrinsic to microbial evolution, their activities are amplified by human influence, in particular by antibiotic exposure, pollution, and intensive agriculture. These human-driven magnifiers will be investigated in Chapter 4.

4. The human amplifier: anthropogenic activities accelerating HGT

Although HGT is a natural aspect of microbial evolution, human activities have significantly increased its frequency and magnitude. These anthropogenic factors not only select for resistant bacteria already present in microbial communities but also generate environmental factors that promote the efficiency and speed of HGT mechanisms themselves. This section describes how the use of antibiotics, industrial contamination, and the formation of new ecological niches have converged to transform an evolutionary mechanism into a global amplifier for spreading ARGs.

4.1. Antibiotic selection: a direct accelerator of gene flow

The most immediate and direct human influence is the widespread use of antibiotics, which promotes HGT by two synergistic mechanisms. The major mechanism is direct selection. When antibiotics enter an environment, susceptible bacteria are eliminated, enriching the microbial community with resistant strains. This enrichment increases the density of potential ARG donors, thus enhancing the likelihood of HGT events. Therefore, resistant populations gain a greater capacity to disseminate their genetic determinants to other bacteria.

A more subtle but equally powerful mechanism arises from sub-inhibitory antibiotic concentrations, which indirectly induce HGT-related processes. At doses insufficient to kill bacteria, certain antibiotics serve as signaling molecules that induce SOS and other stress responses that enhance the activity of MGEs and prophage induction. For instance, fluoroquinolones are strong inducers of the SOS response, facilitating the mobilization of transposons and integrons harboring ARGs. Similarly, tetracyclines can upregulate conjugation-related (tra) genes on plasmids, thereby increasing transfer rates among bacterial cells. This creates a pernicious feedback loop where the very drugs intended to eliminate pathogens instead stimulate the mechanisms that proliferate resistance [12].

4.2. Co-selection by heavy metals and biocides: silent partners

Selective pressure on ARGs does not stem solely from antibiotics. Heavy metals (e.g., copper, zinc, and mercury) and biocides (e.g., quaternary ammonium compounds, QACs), widely used in agriculture, industry, and medicine, act as “silent co-selectors.” Resistance genes for antibiotics, metals, and biocides are often co-localized on the same MGE, such as plasmids or transposons. When microbial communities are exposed to heavy-metal contamination from mining effluents or agricultural feed additives, these selective pressures enrich not only metal resistance genes but also linked ARGs, even in the absence of antibiotics.

Metagenomic studies consistently confirm this phenomenon: soils exposed to mining activities or long-term manure application harbor more numerous and diverse ARGs than unpolluted soils. Similarly, QAC-based disinfectants may co-select for bacteria carrying plasmids with both QAC resistance and clinically relevant ARGs, such as extended-spectrum β-lactamases (ESBLs). This co-selection underscores a critical paradox, efforts to inhibit bacterial propagation using non-antibiotic

DOI: 10.54254/2753-8818/2026.PJ31041

agents may inadvertently sustain and augment ARG reservoirs, complicating AMR remediation efforts [1].

4.3. Man-made hotspots: ecological mixers of genes

Unique landscapes have been human-fabricated to act as specialized reactors for HGT. At the convergence of a number of factors is a perfect storm for genetic transfer: incredible densities of a wide variety of bacteria, a complex cocktail of sublethal antibiotics and co-selecting pollutants, and the opportunity for intimate association in the form of biofilms.

WWTPs are the quintessential example. They are exposed to a mixed influent from hospitals, households, and industry comprising human pathogens, commensal bacteria, and environmental bacteria together with a mixture of antibiotics and other chemicals [5,3]. Metagenomic studies have confirmed that WWTPs are indeed reservoirs of a high number of ARGs and MGEs, and there is evidence that, although bacterial densities might decrease after the treatment step, the relative abundance of mobile ARGs in the final effluents may increase [4].

Similarly, animal manure lagoons associated with concentrated animal feeding operations (CAFOs) represent intense HGT hotspots. Continuous antibiotic use in livestock produces manure abundant in ARGs, MGEs, and resistant bacteria. The nutrient-rich, microbially active conditions within these lagoons promote extensive gene exchange between indigenous bacteria and those introduced through animal waste. When such manure is applied to farmland, it perpetuates a cycle of ARG dissemination from farm to environment. Collectively, these human-made hotspots act as pivotal hubs in the global ARG flow network, linking anthropogenic waste streams with natural microbial reservoirs.

4.4. Biofilms: structured hubs for horizontal gene transfer

Biofilms, organized microbial communities embedded within a self-generated extracellular matrix, constitute another important HGT-favoring milieu. Found on natural and artificial surfaces such as river sediments, water pipes, catheters, and other medical implants, biofilms substantially enhance HGT through spatial proximity and stability.

The dense structure of biofilms enables close cell-to-cell contact, promoting HGT, especially conjugation [10]. Also, cells in biofilms are resistant to external stress and therefore interact for longer periods. In addition, the biofilm matrix is at least partially composed of eDNA released from lysed cells. The elevated level of local eDNA results in more opportunities for natural transformation to occur, and competent cells within the biofilm may gain new genes, such as ARGs. Experimental studies have demonstrated that the rate of plasmid conjugation is much (up to several orders of magnitude) higher in the biofilm than that in planktonic (free-swimming) cells. Thus, biofilms are simultaneously a refuge for resistant bacteria and a highly reactive foundry for new combinations of ARG on mobile elements.

In a sense, these anthropogenic pressures are reshaping the microbial world on a global level—and that is a problem that we need to address strategically, which will be addressed in the final chapter.

5. Conclusion

This paper has consolidated extensive evidence to reinforce a core thesis: ARGs are an ancient and common feature of the microbial pangenome, but their transformation into a global health crisis is

DOI: 10.54254/2753-8818/2026.PJ31041

driven by the extraordinary mobility provided by HGT. Human activities have magnified these natural processes to such an extent that the proliferation of ARGs can now be expected to increase exponentially, especially in environments that act as transmission bridges between environmental reservoirs and clinical pathogens. Therefore, the AMR challenge should not be viewed merely as a clinical problem arising from therapeutic failure but as a planetary-scale issue rooted in the ecological and evolutionary connectivity of genes. Recognizing this reality requires a paradigm shift. By curbing the propagation of MGEs, the global expansion of ARGs could be prevented, and a tactical and sustainable long-term strategy for waste management could be implemented.

Future strategies should be holistic, anticipatory, and cross-sectoral. Three priorities emerge as particularly crucial. First, a one-health surveillance system must be set up and maintained at first. These organizations will need to expand even further past tracking resistant pathogens within hospitals and begin to collect genomic and metagenomic data on animal, agricultural, and environmental (e.g., wastewater) sources to monitor the development and transmission of high-risk MGEs across these interconnected fields. Second, there is also a compelling need for mechanistic investigation of HGT processes in natural/complex environs. Lab models are helpful, but it is necessary to understand how factors within a wastewater biofilm, or an animal's gut, might influence rates of gene transfer to build models that can tell us what might happen in the future and what we can do to try to mitigate the risk. Third, we must accelerate the development of novel approaches that specifically block HGT pathways. These are additions to anti-conjugal compounds (that inhibit plasmid transfer), CRISPR-based methods to remove plasmids from bacteria, and next-generation wastewater treatment technologies designed to eliminate or inactivate MGEs instead of just the host cells.

Ultimately, combating AMR requires a strategic transition from a pathogen-centric mode of defense to one that operates at the microbial gene flow ecology level. The global resistome is an interconnected network, and interventions confined to the clinic sphere will remain insufficient. Targeting the molecular mechanisms that enable HGT offers the most powerful leverage in mitigating the relentless spread of antibiotic resistance. Only by addressing the evolutionary machinery that underpins ARG dissemination can humanity hope to regain control over the microbial world.

References

[1] Arnold, B. J., Huang, I. T., & Hanage, W. P. (2022) Horizontal gene transfer and adaptive evolution in bacteria. Nature Reviews Microbiology, 20: 206–218.

[2] Rodriguez, M. M., Power, P., Sader, H., Galleni, M., & Gutkind, G. (2021) Redefining the origin and evolution of chromosomally encoded blaCTX-M/KLU in the context of a revised taxonomy of genus Kluyvera. Antimicrobial Agents and Chemotherapy, 65(7): e02424-20.

[3] Lira, F., Vaz-Moreira, I., Tamames, J., Manaia, C. M., & Martínez, J. L. (2020) Metagenomic analysis of an urban resistome before and after wastewater treatment. Scientific Reports, 10: 8174.

[4] Yin, X., Deng, Y., Ma, L., Wang, Y., Chan, L. Y. L., & Zhang, T. (2019) Exploration of the antibiotic resistome in a wastewater treatment plant by a nine-year longitudinal metagenomic study. Environment International, 133(Pt B): 105270.

[5] Abdulkadir, N., Saraiva, J. P., Zhang, J., Stolte, S., Gillor, O., Harms, H., & Rocha, U. (2024) Genome-centric analyses of 165 metagenomes show that mobile genetic elements are crucial for the transmission of antimicrobial resistance genes to pathogens in activated sludge and wastewater. Microbiology Spectrum, 12(3): e02918-23.

[6] Tian, L., Fang, G., Li, G., Li, L., Zhang, T., & Mao, Y. (2024) Metagenomic approach revealed the mobility and co-occurrence of antibiotic resistomes between non-intensive aquaculture environment and human. Microbiome, 12: 107.

[7] Fredriksen, S., de Warle, S., van Baarlen, P., Boekhorst, J., & Wells, J. M. (2023) Resistome expansion in disease-associated human gut microbiomes. Microbiome, 11: 166.

DOI: 10.54254/2753-8818/2026.PJ31041

[8] Wuerz, T. C., Kassim, S. S., & Atkins, K. E. (2020) Acquisition of extended-spectrum beta-lactamase-producing Enterobacteriaceae carriage after exposure to systemic antimicrobials during travel: Systematic review and meta-analysis. Travel Medicine and Infectious Disease, 37: 101823.
[9] Melvold, J. A., & Wyrsch, E. R. (2017) Identification of a novel qnrA allele, qnrA8, in environmental Shewanella algae. Journal of Antimicrobial Chemotherapy, 72(10): 2949–2952.
[10] Element, S. J., Moran, R. A., Beattie, E., Hall, R. J., van Schaik, W., & Buckner, M. M. C. (2023) Growth in a biofilm promotes conjugation of a blaNDM-1-bearing plasmid between Klebsiella pneumoniae strains. mSphere, 8(4): e00170-23.
[11] Palencia-Gándara, C., Getino, M., Moyano, G., Redondo, S., Fernández-López, R., González-Zorn, B., & de la Cruz, F. (2021) Conjugation inhibitors effectively prevent plasmid transmission in natural environments. mBio, 12(4): e01277-21.
[12] Domenech, A., Slager, J., & Veening, J.-W. (2018) Antibiotic-induced cell chaining triggers pneumococcal competence by reshaping quorum sensing to autocrine-like signaling. Cell Reports, 25(9): 2390–2400.e3.
[13] Moon, K., Jeon, J. H., Kang, I., Park, K. S., Lee, K., Cha, C.-J., & Cho, J.-C. (2020) Freshwater viral metagenome reveals novel and functional phage-borne antibiotic resistance genes. Microbiome, 8(1): 75.
[14] Borodovich, T., Shkoporov, A. N., Hill, C., & Clooney, A. G. (2022) Phage-mediated horizontal gene transfer and its implications for the human gut microbiome. Gastroenterology Report, 10: goac012.
[15] Enault, F., Briet, A., & Petit, M.-A. (2017) Phages rarely encode antibiotic resistance genes: A cautionary tale for virome analyses. The ISME Journal, 11(1): 237–247.