The Role of Plasmid in Antibiotic Resistance

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ABSTRACT

Plasmids are central engines of antibiotic resistance because they efficiently collect resistance genes, move them within and between species, and persist across clinical, agricultural, and environmental settings. Certain plasmid types and mobile elements (integrons, insertion sequences, and phage-plasmids) are especially important in generating multidrug-resistant "superbugs." Environmental conditions, biofilms, and cooperative resistance further amplify spread, while plasmid stability and adaptation make control difficult, motivating strategies that explicitly target plasmids and horizontal gene transfer. This article aimed to highlight the plasmid and its role in antibiotic resistance.

Keywords: Plasmid, Caring genes, Antibiotic resistance, DNA.

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Introduction

Plasmids are small, usually circular DNA molecules that move between bacteria and are central to the modern antibiotic resistance crisis. They collect, assemble, and spread antibiotic resistance genes (ARGs) within and across species, environments, and even continents, creating multidrug-resistant "superbugs" (1, 2).

Antibiotics have been used clinically for only a few decades, yet resistance is now a major global problem, reflecting the speed of bacterial adaptation (1). Bacteria acquire resistance either by mutation or, far more rapidly, by horizontal gene transfer (HGT) via plasmids, phages, and integrative elements. MDR "superbugs" frequently harbor multiple resistance genes on plasmids, allowing them to resist various drug classes. Examples of these resistance mechanisms include beta-lactamase, efflux pumps, and enzymes that modify the antibiotic structure, rendering it ineffective. The purpose of this article is to emphasize the role of plasmids in contributing to antibiotic resistance genes (1, 3).

Functions of Plasmids

Plasmids serve various roles, depending on the genes they carry:

- Numerous plasmids contain proteins that either render antibiotics inactive or drive them out of the cell (antibiotic resistance).
- Plasmids encoding poisons or adhesion factors are frequently carried by pathogenic bacteria.
- Certain plasmids enable bacteria to break down uncommon or harmful substances.
- Bacteria can transfer genetic material via direct contact thanks to some plasmids (conjugation).
- Foreign genes are introduced and expressed in target cells using engineered plasmids (1,3).

Mechanisms of Plasmid-Mediated Resistance

- **Gene capture and assembly**: Plasmids act as genetic platforms where multiple ARGs accumulate via transposons, insertion sequences, and integrons, creating multidrug resistance cassettes (1, 4).
- **Horizontal gene transfer (HGT):** Conjugative plasmids transfer by cell-to-cell contact; mobilizable plasmids hitchhike with them (5,6). This allows rapid spread of ARGs compared with slow mutation-based evolution.
- **Inter-plasmid exchange:** ARGs frequently move between plasmids within the same cell; in one large analysis, 87% of plasmid-borne ARGs showed potential inter-plasmid transfer, often driven by IS26 and integrons (2, 4).
- **Phage-plasmids:** It is believed that phages and plasmids are two different kinds of genetic material that is mobile that use horizontal gene transfer to propel bacterial evolution. e.g., colistin, carbapenem resistance) (7).

How Plasmids Build Multidrug Resistance

- Plasmids function as frameworks that gather various resistance genes through integrons, transposons, and insertion sequences, creating potent platforms for multidrug resistance (2,6).
- ARGs commonly transfer between plasmids within the same cell, according to extensive genomic surveys; these transfers happen between compatible plasmids, quickly constructing complex resistance combinations (1).
- Not all mechanisms of resistance are equally transferable, as evidenced by the far higher likelihood of accessory (non-core) chromosomal genes for resistance being on plasmids than core genes (2).

Environments and Conditions That Boost Transfer

- There are strong interconnections among clinical, agricultural, and environmental settings, as plasmids facilitate the transfer of antimicrobial resistance genes (ARGs) between humans, animals, plants, food chains, soil, and water (8, 9).
- Although the in-situ dynamics of plasmid transfer under actual pollution conditions and within various communities remain poorly understood, studies indicate that antibiotics and pollutants—such as disinfectants, metals, microplastics, and pharmaceuticals—can enhance the rate of plasmid transfer in laboratory models (10, 11).
- Conjugative plasmids and other mobile components are more effective at disseminating antimicrobial resistance genes (ARGs) in biofilms than in free-living cells, both in clinical and environmental contexts (11, 12).

Why Plasmid-Mediated Resistance Is Hard to Control

- Bacteria can eventually compensate for the fitness costs associated with resistance plasmids, allowing them to stabilize even without the presence of antibiotics (6, 13). Certain enzymes encoded by plasmids, such as KPC-2 β-lactamase, provide cooperative protection to neighboring cells, making them more likely to be transformed into new carriers of the plasmid (2).
- Although these control methods remain largely experimental, they propose strategies such as chemical or biological "plasmid curing" and the inhibition of plasmid transfer, which specifically target plasmids and other mobile genetic elements (14, 15).

Conclusion

Plasmids primarily drive the development, assembly, and global spread of antibiotic resistance. These genetic elements contribute to multidrug resistance in hospitals, agriculture, and ecological systems by acquiring various resistance genes, rapidly transferring between bacteria and environments, and merging with other mobile genetic elements. To design effective therapies that target plasmids and curb the spread of untreatable diseases, it is essential to understand these mechanisms.

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