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Acquired Resistance from Gene Transfer

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7.1 Introduction

Antibiotic drugs emerged as unambiguously the most powerful medical tool to combat infectious diseases. They substantially improved human health and significantly increased lifespan. Antibiotic drugs can be divided into several classes due to their structural features and their target site, e.g. β-lactams, aminoglycosides, glycopeptides, tetracyclines, macrolides, lincosamides, streptogramins, sulfonamides, quinolones, and carbapenems (Davies and Davies 2010; Sultan et al. 2018). Antibiotic drugs can render bacteria harmless by interfering with cell wall synthesis, protein synthesis, or the nucleic acid machinery, by affecting metabolic pathways, or by disintegrating bacterial membrane structures (Kohanski et al. 2010) (see Figure 7.1). Several issues, including antibiotic overuse and wrong prescription, fueled the increasing appearance of bacterial strains no longer responding to conventional antibiotic treatment strategies. While antibiotics by themselves do not provoke resistance, frequent exposure and high doses of antibiotic drugs exert selection pressure on bacteria, triggering several resistance strategies. Antibiotic resistances (ABRs) in bacteria have emerged as a global health threat. Several human pathogens associated with epidemics have evolved into multidrug-resistant (MDR) forms. This then leads to a massive reduction of therapeutic options (Davies and Davies 2010; Karam et al. 2016; Aslam et al. 2018; Yelin and Kishony 2018). One alarming development is that bacteria become increasingly resistant to different antibiotic classes via acquisition of resistance genes originating from the same and/or different bacterial species. Dissemination of resistance determinants mostly occurs by horizontal gene transfer (HGT).

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