interfere with three major types of target in the bacterial cell – the cell wall synthesis, proteins synthesis, and DNA access, mainly during replication. Hence, although secondary toxic effects may occur, antibiotics selectively target bacteria, whose cells are destroyed or inhibited to divide, while no harm is anticipated in the host's cells.

Unfortunately, the enthusiasm put on antibiotics as therapeutic agents would not last. Resistance, meaning the ability to survive and proliferate in the presence of antibiotics at concentrations used for therapeutic purposes, is found for all antibiotics, sooner or later after their commercialization (Alanis 2005). When bacteria acquired the capability of recurrently grow in the presence of a clinically used dose of antibiotic, they cause the failure of the antibiotic as therapeutic agent and are, hence, named as antibiotic-resistant bacteria (ARB). The mechanisms used by ARB can be summarized as the ability to (i) alter the antibiotic molecule (degradation, transformation), (ii) control the antibiotic intracellular concentration (efflux, cell impermeabilization), or (iii) modify the cellular antibiotic target (Blair et al. 2015).

10.2 Intrinsic and Acquired Antibiotic Resistance

Some bacteria harbor ancestral traits that confer intrinsic resistance to one or more classes of antibiotics. Intrinsic resistance can result from specific cell properties, such as the absence of cell wall or the presence of an external membrane, or some specific chromosomal genes. In these cases, the genes encoding for such properties are part of the core genome of a given species or genus (EUCAST; Davies and Davies, 2010). Consequently, all members of a given taxonomic group (species, genus, family) share the same resistance phenotype. In contrast, when the genes encoding for antibiotic resistance (ARGs) are part of the accessory genome of a bacterial strain, which includes genetic information that was acquired, antibiotic resistance is only observed in some representatives of a given species (EUCAST). Acquired antibiotic resistance may result from gene mutation or genetic recombination (Martinez and Baquero 2000; Zhang et al. 2009a; Davies and Davies 2010). Gene mutations occur randomly in the genome, often potentiated by mutagenic agents, and when they represent an evolutionary advantage to the cell, they may become dominant through dissemination by vertical transmission (from one generation to the next). Genetic recombination, frequently referred to as horizontal gene transfer (HGT), is believed to be common among bacteria, representing one of the major driving forces for bacterial evolution (Ochman et al. 2000; Davies and Davies 2010; Wiedenbeck and Cohan 2011). The HGT may occur by (i) conjugation among bacteria, which involves the transfer of genetic material from a donor to a recipient cell, requiring that both share the same space, but not necessarily the same species; (ii) transformation, consisting on the uptake of naked DNA released by dead cells; and (iii) transduction, mediated by bacteriophages (Andersson and Hughes 2010).