States of America (10%), Brazil (8%), India (4%), and Mexico (2%) as the five major consumers.

10.6 Antibiotic Resistance Evolution

It is assumed that the presence of antimicrobials in a given ecological niche will lead to the successive elimination of susceptible bacteria with the simultaneous Darwinian selection of ARB (Founou et al. 2016). Hence, any host, human or animal, consuming antibiotics or environmental compartment contaminated with antibiotics, are potential reservoirs of ARB and ARG. In parallel with selection, the stress imposed by the presence of antibiotic residues may favor the acquisition of ARG by susceptible bacteria, making antibiotic residues important evolution drivers (Andersson and Hughes 2011). Although antibiotic resistance acquisition is common in nature, these processes are facilitated by the massive and intensive use of antibiotics (Sørensen et al. 2005). A high number of reports and studies produced over the last decades show an increase not only of resistance prevalence but also of the diversity and distribution of ARG (EARS-Net; ESAC; NARMS; Knapp et al. 2010). The exposure of the bacterial communities to antibiotics has been suggested as a major driver for the emergence and spread of resistance. It is believed that mechanisms such as the disturbance of the microbial communities, interference with gene expression, or biofilm formation can trigger or act in combination with the horizontal transfer of ARG (Martinez 2008; Andersson and Hughes 2012, 2014; You and Silbergeld 2014). In addition, it has been argued that the levels of resistance observed at any time and place are the result not only of the recent conditions but also of the global history of antibiotic resistance acquisition (O'Brien 2002). This can be explained based on the fact that when the carriage of resistance determinants does not impose a fitness cost, they will not be lost by their host, even in the absence of selective pressures (Andersson and Hughes 2010, 2011).

10.7 Stressors for Antibiotic Resistance

Besides the antibiotic residues or metabolites thereof, some other chemical compounds have been described to contribute to ARB and ARG enrichment due to processes of co- or cross-resistance. Co-resistance to antibiotics and other chemicals occurs when the genes specifying the resistance phenotypes are located together in the same genetic element. In turn, cross-resistance arises from a given mechanism that confers resistance to two or more antimicrobials from different classes. The best example of co-resistance is the genetic linkage of ARG and metal translocation genes (e.g. Hg, Cu, Cd, Zn) (Seiler and Berendonk 2012; Pal et al. 2015). Examples of cross-resistance mechanisms are