other molecules extracted from essential oils have been tested for their anti-biofilm properties (Miladi et al. 2017; Oh et al. 2017; Artini et al. 2018) and been reported to inhibit bacteria by damaging the cell membrane, altering the lipid profile, and inhibiting ATPases, cell division, membrane porins, motility, and biofilm formation, but the molecular mechanisms involved remain unknown.

Lytic bacteriophages are viruses that are able to infect specific bacterial species and can be used in combination with antibiotics to potentiate their action for the treatment of biofilm infections (Chaudhry et al. 2017). Alves et al. (2016) tested two biofilm models (static and dynamic) with a phage cocktail to assess the ability to reduce and disperse *P. aeruginosa* biofilm biomass. In the static model, after four hours of contact with the phage suspension, more than 95% of the biofilm biomass was eliminated. In the flow biofilm model, the biofilm was dispersed after 48 hours. Kumaran et al. (2018) investigated the ability of phage treatment to enhance the activity of antibiotics cefazolin, vancomycin, dicloxacillin, tetracycline, and linezolid against biofilm-forming *S. aureus*. They showed a significant reduction of up to 3 log colony forming unit (CFU) per milliliter when the phage treatment preceded antibiotics and a more important effect with vancomycin and cefazolin, particularly at lower antibiotic concentrations.

9.3.2 Efflux Pump Inhibitors

Efflux pump inhibitors are compounds that meet the following criteria (Soto 2013): enhanced activity of the pump substrates, no activity in efflux pump mutants, increased accumulation and decreased extrusion of efflux pump substrates, no action on the proton gradient across the cytoplasmic membrane, and inhibition of this efflux activity.

9.3.3 Anti-Persisters: Quorum-Sensing Inhibitors

Many anti-persister strategies have been attempted, as prolonged treatment with aminoglycosides that cause mistranslation, leading to misfolded peptides, can sterilize a stationary culture of *P. aeruginosa*, a pathogen responsible for chronic, highly tolerant infection of cystic fibrosis patients. One of the best bactericidal agents is rifampin, an inhibitor of RNA polymerase, and Keren et al. (2012) suggested that it "kills" by preventing persister resuscitation. The anticancer drug cisplatin [*cis*-diamminodichloroplatinum(II)], which mainly forms intra-strand DNA cross-links, can eradicate *E. coli* K-12 persister cells through a growth-independent mechanism (Chowdhury et al. 2016). A combination of tobramycin and fumarate has been tested successfully (Koeva et al. 2017) as an antibacterial potentiator for eliminating recurrent *P. aeruginosa* infections in cystic fibrosis patients through the eradication of bacterial