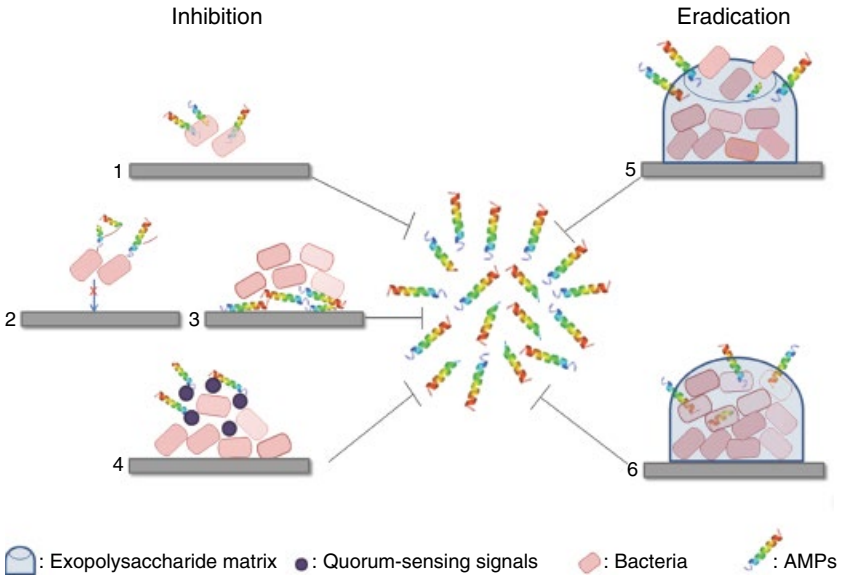

Figure 20.1 Mechanisms of anti-biofilm action of AMPs. (1) Inhibition of biofilm formation by targeting cells in the planktonic state. (2) Inhibition of adhesion on biotic surface reaching the regulatory pathways responsible for the production of adhesion defects, such as pili and flagellum. (3) Inhibition of adhesion by abiotic surface coating. (4) Inhibition of quorum sensing. (5) Eradication of mature biofilm by disruption of the extracellular matrix. (6) Eradication of the mature biofilm through the death of the adhered cells and incorporated into the extracellular matrix. *(See insert for color representation of the figure.)*

initial adhesion through the abiotic surface coating; (iii) inhibiting the adhesion on a biotic surface, reaching the regulatory pathways responsible for the production of adhesion structures; or (iv) binding to the molecules involved in *quorum sensing*, inhibiting bacterial communication (Jorge et al. 2012; Di Luca et al. 2014; Fuente-Núñez et al. 2016).

At the molecular level, studies also demonstrate that peptides can prevent the formation of pathogenic biofilms by disrupting nucleotide signaling, important signaling molecules that allow microorganisms to control the formation and maintenance of biofilm (Pletzer et al. 2016). For example, it has been described that AMPs can bind and degrade intracellular nucleotides (p)ppGpp (guanosine tetra- and pentaphosphate), thus preventing the intracellular accumulation of this molecule and consequently inhibiting the formation of pathogenic biofilms (Fuente-Núñez et al. 2015). The AMPs can also bind to the nucleotide c-di-GMP (bis-(3′,5′)-cyclic dimeric guanosine monophosphate) and prevent the expression of extracellular matrix components, also inhibiting biofilm formation (Foletti et al. 2018). In addition, they can also inhibit quorum-sensing sensors such as trp RNA-binding attenuation protein (TRAP)