Managing sticky situations – anti-biofilm agents

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Editor

Biofilm-related infections are accountable for increasing morbidity and disability, particularly in an era when surgery has evolved to new heights and is able to repair or replace virtually any damaged component of the human body.

Bacteria have recently been redefined as “intelligent” beings, able to communicate, coordinate, and store in their genome information derived from previous experiences, such as the development of antimicrobial resistance, or the acquisition of virulence traits, to name only a few. Bacteria can act both separately and collectively, responding to outside stimuli and making decisions regarding their morphology, metabolism and virulence.

Bacterial intelligence represents more than just the sum of individual behaviors; it is a macromolecular biological network that allows multidirectional interactions between the members of the collectivity. Sociomicrobiology has shown that bacteria display plastic associative learning, as well as decision making skills and the ability to anticipate what comes next (Westerhoff *et al.* *Front Microbiol* 2014;5:379).

By way of distributed information processing (Steinert M. *Front Cell Infect Microbiol* 2014;4:8) and quorum sensing, bacteria can make choices regarding their development as planktonic cells,

biofilms, or as intermediate aggregative communities (Haaber *et al.* *PLoS One* 2012;7:e41075).

When organized in biofilms, bacteria can avoid host immunity, become tolerant to antimicrobials, and down-regulate the metabolism of individual cells while up-regulating their overall virulence.

Biofilms have been associated with protracted, hard-to-treat infections, and there currently is an acute need for the introduction of novel anti-biofilm agents in the therapeutic armamentarium. Such agents would of course target all steps of biofilm formation, namely attachment, aggregation, maturation, and dispersal.

When dealing with sticky situations such as biofilm formation, it becomes important to understand the exact mechanisms whereby biofilm is formed or degraded. It seems only natural that primary attachment is the easiest step to target in the biofilm lifecycle, as any drug with antibacterial activity, or any substance able to inhibit bacterial adherence can prevent the formation of biofilm altogether, and relevant examples include antibacterial agents such as minocycline or rifampin, molecules such as aryl rhodanines, silver ions, bacteriophages, etc. (Chung *et al.* *Pathog Dis.* 2014;70:231-9). Mature biofilm can be disrupted by multiple mechanisms, namely depolymerizing enzymes, dispersin, DNase, proteinase, or bacteriophages.

In conclusion, it is becoming increasingly important to study bacterial communication, aggregation and dispersal, in order to be able to target specific steps in the biofilm lifecycle and to ensure tailored anti-microbial and anti-biofilm therapy.

Conflicts of interest: none to declare.

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