



Innate and induced resistance mechanisms of single and mixed bacterial biofilms – role of environmental stress agents

Olívia Pereira, Maria João Vieira

*IBB-Institute for Biotechnology and Bioengineering, Centre of Biological Engineering,
Universidade do Minho, Campus de Gualtar, 4710-057 Braga, Portugal.*

Bacteria attach readily to surfaces in both natural and man-made ecosystems, and if they are left undisturbed biofilms may form. The attached cells normally display an altered phenotype that is believed to be responsible for the less susceptibility to antimicrobials. Moreover, bacteria may also acquire a resistant phenotype in response to those antimicrobials pressure. These innate and induced reduced antimicrobial susceptibilities contribute to the recalcitrance of biofilms and persistence of infections such as those associated with medical equipment and implanted devices.

In order to comprehend the protective mechanisms in biofilms that are responsible for conventional antimicrobial resistance, we have been characterizing and modeling biofilm formation in different apparatus using different surfaces, different liquid phases, operational conditions, and stress agents, mimicking several environments: industrial, clinical and drinking water. In this topic, we are developing high-throughput platforms for the rapid and systematic characterization of biofilm formation and control (1). The phenotype and proteome of bacteria embedded in single and mixed biofilms, formed by type, adapted and isolated strains, is also being characterized. In order to design suitable disinfection and sanitation protocols of medical surfaces and equipment, several mitigation measures are being tested through the use of traditional and innovative chemical or natural antimicrobials, such as surfactants, antimicrobial peptides, bacteriocins, antibiotics, etc. (2). The main resistance mechanisms of the planktonic and sessile bacteria to those antimicrobials are being studied with the aim of establishing their contribution to the occurrence of persister cells and the persistence of nosocomial biofilm-related infections and to gather information that can lead us to the study of methods to block the arise of those mechanisms.

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