Bacterial prints in human infectious diseases

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Ample resources have been dedicated to studying bacterial resistance, biofilm formation, and genetic encoding of resistance, metabolism or fitness mutations. However, less is known about bacterial persister cells that display multidrug tolerance,¹ latency¹ or adaptation.²

Classically considered to be cellular beings that can survive and multiply without the need of host cells, bacteria have also been shown to display phenotypes leading to internalization into human cells, as is the case with *Granulibacter bethesdensis* in monocytes and macrophages,³ *Streptococcus pneumoniae* in erythrocytes,⁴ *Staphylococcus aureus* in epithelial or endothelial cells,^{5,6} or *Escherichia coli* in urothelial cells.⁷ The internalization of bacteria into human cells opens up numerous possibilities for them to evade the immune system and lead to persistence and, potentially, chronic infection, or relapse after an apparently successful antimicrobial therapy.

As the human body is known to be made up of ten times more bacterial cells than human cells (NIH Human Microbiome Project),⁸ it becomes crucial to study the pathogenesis of microbial infection, and to identify and define 'sterile' environments in the human body –if any– as well as potential reservoirs for bacterial latency.

Blood cells play a role in clearing bacterial infection, but can also drive bacterial pathogenesis as they travel throughout the body and are ideal transport vehicles for bacteria, providing both nutrients and, at times, protection from the immune system.

To better describe the phases of bacterial infection, the triggers that lead to expression of particular bacterial phenotypes, and to single out a pathway driving chronicisation of bacterial infection, we aim to perform a study of bacterial prints in human infectious diseases, through means of semi-quantitative PCR, microbiologic identification, or microscopy techniques.

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Conflicts of interest None to declare.

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