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EDITORIAL

WILEY

Pathogen evasion strategies

Pathogens are faced with a number of physical barriers upon entry in their animal host, whether it be the mucocilliary escalator of the respiratory tract or the acidic environment of the stomach. Following this, they must also overcome innate immune cells expressing pattern recognition receptors that are specialised in identifying and eliminating invading pathogens. During the course of co-evolution, all forms of life have developed sophisticated means by which they can respond to, and fight infection and in turn, pathogens have evolved even more complex mechanisms to subvert these efforts. These mechanisms, such as effector molecules or lifestyle advantages, have always drawn significant attention for their possible exploitation as therapeutic targets. With the rise of antibiotic resistance already at catastrophic levels in some countries, the search for exploitable, novel strategies to target virulence mechanisms is at an all-time high.

Mechanisms employed by pathogens to evade the host innate and adaptive immune responses, as well as therapeutic interventions, come in many flavours, and while it is tempting to highlight them all, we can distil them down to a few fundamental concepts; *concealment*, *disruption* and *destruction*. Each of these concepts underpins an important evolutionary advantage that bacteria, fungi, viruses and parasites use to ensure their survival.

1 | CONCEALMENT

The previous virtual issue dealt with one of the most well-studied concealment strategies of pathogens; intracellular survival within host cells. Therefore, for this issue, we will highlight another important tactic employed by pathogens, biofilm formation. It has been hypothesised that the preferred lifestyle of almost all microorganisms on earth is not in a planktonic state, but in fact within these adherent structures composed of an impenetrable matrix consisting of proteins, lipids, DNA and sugars. Due to the presence of this matrix, as well as persister cells, biofilms of bacterial and fungal origin are highly tolerant to both antibiotics and the host immune response. A primary example of the detrimental effects of biofilms is seen with *Pseudomonas aeruginosa*, a common nosocomial pathogen that is perhaps most well known for its debilitating effects in cystic fibrosis patients. Alternatively, as highlighted in this issue, biofilms are also synonymous with dental plaques and thus, play an important role in oral health (Volume 21, Issue 10 and Volume 22, Issue 8). Currently, the search for essential genes required for biofilm formation is an ever-growing field with the goal to help develop combinational therapies that have better efficacy against these highly resistant microbial communities.

2 | DISRUPTION


In case concealment itself is not a viable strategy, other more subtle mechanisms are utilised. Oftentimes, these involve the interference of innate immune effector functions, such as the complement cascade, phagocytosis machinery or cell death pathways. In a previous Editor's Choice article (Volume 21, Issue 6) the human respiratory pathogen *Legionella pneumophila* was shown to disrupt actin polymerisation in host cells, leading to a potential enhancement of intracellular replication. Another Editor's Choice article (Volume 20, Issue 6) highlighted in this issue, focused on the ability of the protozoan parasite *Cryptosporidium parvum* to alter the expression of integral junction proteins and disrupt the integrity of intestinal epithelial barrier function. As a consequence, this would enhance the invasive capabilities of the parasite, favouring colonisation. Collectively, these mechanisms aim to bypass host defences without causing extensive damage, and allow pathogens to disseminate and grow within their host unabated.

3 | DESTRUCTION

For many pathogens, the host intracellular environment poses an incredible challenge for survival, whether it be by the classical phagolysosomal pathway, autophagy, generation of reactive oxygen/nitrogen species, inflammasome activation, or intracellular sensing pathways such as retinoic acid-inducible gene I (RIG-I)-like receptor, nucleotide-binding oligomerisation domain (NOD)-like receptors and toll-like receptors (TLRs). Pathogens, in particular bacteria, such as *Mycobacterium tuberculosis* (Volume 22, Issue 1), *Leptospira interrogans* (Volume 21, Issue 1) and *Vibrio cholerae* (Volume 21, Issue 8) to name a few, respond by secreting effector molecules and toxins that induce host cell death to render these above-mentioned anti-microbial mechanisms ineffective, and promote their replication. However, these mechanisms pose a significant threat to the host, with cell death and the subsequent release of cytosolic components potentially inducing a potent pro-inflammatory response that leads to leukocyte infiltration and tissue damage. In a more recent example, various effector proteins from SARS-CoV-2, the causative agent of the COVID-19 pandemic, have been implicated in the disruption of the antiviral interferon response, promoting subsequent immune evasion and viral replication. Ultimately, this can lead to an overproduction of proinflammatory cytokines, known as a cytokine storm, which can result in death.

In this virtual issue, we revisit a number of influential publications in Cellular Microbiology over the years that have examined the multitude of strategies employed by pathogens to subvert host defences.

With this, we hope to shine a light on not only cutting-edge research articles, but also in-depth reviews that focus on expanding our knowledge of pathogen evasion strategies. Through these virtual issues, we aspire to highlight the efforts of the scientific community in aiding in the future development of therapeutic approaches.

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