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Commentary

Interactions between the Host and the Pathogen

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COMMENTARY

This issue contains a collection of articles on how bacteria interact with their hosts' microenvironments, divided into four sections: how bacteria sense their hosts, how bacteria respond appropriately, how bacteria remodel and alter their microenvironments, and what factors characterise these microenvironments. This issue will focus on some of the specific environments that bacteria encounter, such as intracellular vacuoles and tissue micro domains, at the host-pathogen interface. Bacterial pathogens face a unique signalling scenario when they come into contact with a host. We look at how the host affects bacterial signalling and how bacterial infections affect host signalling in this paper. I include information about new methodologies and technologies that provide intriguing new ways of looking at host-pathogen interactions, as well as more defined information regarding host niches.

The EnvZ/OmpR two component regulatory system, which represses the cadC/BA system and prevents bacterial cytoplasm neutralisation, is a crucial sensing system. The application of single cell techniques revealed a surprise finding: the bacterial cytoplasm does acidify, and acidification is employed as a signal to induce pathogenicity. The pathogenicity of the cytosolic pathogens *Burkholderia pseudomallei* and *Listeria monocytogenes* is regulated by host glutathione. Joanne Ku and Yunn-Hwen Gan discuss how pathogens use host glutathione as a spatiotemporal cue to control virulence through bacterial fitness change in their article "Modulation of bacterial virulence and fitness by host glutathione."

In their ode to Jane Austen's little-known microbiology interest, "Sense and sensor ability: redox-responsive regulators in *Listeria monocytogenes*," Brittany R. Ruhland and Michelle L. Reniere describe how *Listeria monocytogenes* senses reactive oxygen species. L. monocytogenes has a remarkable five redox-responsive regulators that detect a wide range of redox stresses, including organic hydroperoxides, peroxides), NAD^+/NADH homeostasis, disulfide stress), and infection redox stress. Sulfur is one of the most important nutrients for bacterial pathogens. Joshua Lensmire and Neal Hammer explore the astonishing array of techniques infections use to scavenge both organic and inorganic sulfur-containing metabolites in their paper "Nutrient sulphur acquisition strategies employed by bacterial pathogens." They believe that blocking this wide spectrum of sulfur-gathering capacities as an antibiotic target will necessitate complex tactics.

The authors of this paper, Leou Ismael Banla, Nita H Salzman, and Christopher J. Kristich, are interested in how Enterococci react to the host environment. These bacteria occupy the mammalian gastrointestinal tract as harmless residents, but they can cause disease under certain circumstances, such as antibiotic-induced dysbiosis. The authors describe our understanding of how Enterococci colonise the gastrointestinal tract, including their use of transcriptional reprogramming, the contributions of specific genes, genome plasticity, and roles for intra-species and inter-species interactions, in their review "Colonization of the mammalian intestinal tract by enterococci."

In response to environmental changes, Bordetella's BvgAS system directly activates a large number of virulence genes. Qing Chen and Scott Stibitz present further complexity in the BvgASR regulon of *Bordetella pertussis* in their work.

The BvgASR regulon of *Bordetella pertussis*

The first is by the action of BvgR, a phosphodiesterase that lowers cyclic-diGMP levels. The vrg genes are a group of important virulence genes that are repressed when cyclic-diGMP levels are low. RisAP, a non-canonical response regulator that is strangely not phosphorylated by its corresponding kinase RisS, controls the vrg genes as well. In B. pertussis, deletion of risS increased phosphorylation of RisA during evolution, but not in *B. bronchiseptica's* progenitor. The capacity to activate the vrg genes could help B. pertussis spread via the air.

Christina Yang and Karen Ottemann conclude the issue by talking about significant microniches in the gastrointestinal tract, such as the gastric glands and intestinal crypts. They describe host processes that limit bacterial colonisation in glands and crypts, such as the immune system, acid, mucin, oxygen, and reactive oxygen species, as well as bacterial adaptations that allow growth in these niches, such as

bacterial immunomodulatory molecules, chemotaxis, and the use of specific metabolites, in their article "Control of bacterial colonisation in the glands and crypts." Secretion of effector proteins via secretion systems such as the type three secretion systems is one technique bacteria can change their environments (T3SS).

In their paper "Vibrio variants on a type three theme," Kelly A. Miller, Katharine F. Tomberlin, and Michelle Dziejman discuss similar work. They report that *Vibrio* spp., including some strains of *V. cholerae*, employ T3SSs to regulate a range of host pathways, including innate immunological signalling pathways and actin dynamics, so there is still much more to learn about this process. In their essay "Emerging insights into bacterial deubiquitinases," Tomoko Kubori, Tomoe Kitao, and Hiroki Nagai demonstrate how various bacteria can change host ubiquitination. They discuss the recent discovery that bacterial effector proteins, which serve as ubiquitin ligases and deubiquitinases, target multiple components of the host ubiquitin system. *Salmonella, E. coli, Legionella, Chlamydia, Burkholderia,* and *Xanthomonas* are among the bacteria that do so, causing changes in NF-kB signalling and host autophagy.

Once within the host, germs require a variety of qualities in order to proliferate and cause disease. In her work "The function of cytokine production during *Campylobacter jejuni* colonisation and pathogenesis," Victoria Korolik highlights one of them, the capacity to perform directed mobility. Loss of chemotaxis signalling, and occasionally even a single chemosensory receptor, can drastically limit *C. jejuni's* ability to colonise and cause disease across many animal hosts, that according studies. The most cutting-edge methods for identifying chemoreceptor ligands are also addressed.