Bacterial infection and symbiosis

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The "Bacterial Infection and Symbiosis" Minisymposium covered topics ranging from a description of bacterial weapons and their toxic ammunition, to composition and functions of bacterial communities, to consequences of bacterial infection for the host cells, to novel models and approaches to study host–bacteria interactions.

The human body is colonized by vast amounts of various bacteria, which influence well-being. Therefore, it is important to understand the composition and structure as well as metabolic activity of such communities. **Steven A. Wilbert** (Borisy lab, The Forsyth Institute, Cambridge, MA) showed how composition and structure of the microbiome can be studied using multiplexed fluorescence in situ hybridization (Mark Welch *et al.*, 2016) and identified the members of the tongue microbiome that could play a role in nitric oxide homeostasis. Another way that the microbiome might influence human life is by changing host metabolism. **Meng C. Wang** (Baylor College of Medicine, Houston, TX) showed that metabolites secreted by bacteria present in the microbiome influence balance between fusion and fission of mitochondria and thus change host lipid metabolism and affect longevity (Han *et al.*, 2017).

Bacteria also influence their environment directly by secreting a diverse set of proteins by using various nanomachines. As discussed by **Marek Basler** (University of Basel, Basel, Switzerland), cryo–electron microscopy and live-cell fluorescence microscopy have been used recently to gain insights into the mode of action of the Type VI secretion system (Nazarov *et al.*, 2017). Importantly, secreted effectors are often critical for pathogenesis. For example, for intracellular bacteria it is essential to be able to manipulate the infected host cell to allow bacterial replication. **Joanne N. Engel** (University of California, San Francisco) explained that *Chlamydia trachomatis* secretes effector proteins localizing to the bacterial inclusion membrane. One of those effectors, IncE, disrupts interaction of the SNX5 protein of the retromer with the mannose-6-phosphate receptor, which blocks retromer-mediated trafficking in human cells and promotes *Chlamydia* infection (Elwell *et al.*, 2017). **Massimiliano Baldassarre** (University of Aberdeen, United Kingdom) showed that another intracellular pathogen, *Salmonella*, inhibits a Rab32 GTPase-dependent host-defense pathway in order to survive intracellularly (Spanò *et al.*, 2016).

Bacterial infections stimulate various host cell pathways, which in many cases evolved to restrict the growth of the pathogens. **Feng Shao** (National Institute of Biological Sciences, Beijing, China) talked about the roles of caspase-activated proteins, Gasdermins, which form pores in the membrane of cells and thus execute cell death during pyroptosis (Shi *et al.*, 2017). It is becoming clear that Gasdermins play different roles in cell death, and understanding their mode of action and mechanisms of activation could not only help to control infections but also treat cancer. Interestingly, **Lilliana Radoshevich** (University of Iowa, Iowa City) showed that protein modification by an interferon-stimulated, ubiquitin-like protein, ISG15, is yet another mechanism by which cells react to bacterial infections. Mapping the ISGylome following *Listeria monocytogenes* infection identified modified ER and Golgi proteins and initiated a search for understanding the role of this ubiquitin-like protein in restricting infection (Radoshevich *et al.*, 2015).

Many complex interactions occurring during bacterial infection cannot be easily replicated using simple in vitro assays, and thus whole tissues or animals have to be used to test various hypotheses. **Qian Yu** (Song lab, University of Maryland, College Park) showed that *Neisseria gonorrhoeae* infection of human cervix tissue explants provides a useful tool for addressing the role of bacterial adhesins in tissue colonization and penetration (Wang *et al.*, 2017). The ability to visualize progression of an infection ex vivo and in vivo may provide important insights. Using a transparent zebrafish as a model of inflammatory bowel disease, **Ling-shiang Chuang** (Cho lab, Icahn School of Medicine at Mount Sinai, New York) monitored the spread of fluorescently labeled bacteria through the animal and tested the effects of treatments such as application of prostaglandin E2.

Finally, precise quantification and modeling of bacterial behavior is often required to unravel strategies that bacteria use to infect and colonize hosts. **Fabian E. Ortega** (Theriot lab, Stanford School of Medicine, Stanford, CA) showed that *L. monocytogenes* dispersal over long distances during cell-to-cell spread is critical for establishing successful infection (Ortega *et al.*, 2017).

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