PRESPECTIVE

# Two-way communication between microbe and host

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The importance of back-and-forth communication in host–pathogen relationships has long been recognized in commensalism and microbial disease. We've been studying these bacteria in our environment for generations, yet there are still many unanswered concerns about the evolutionary cross-talk between host and germ. Basic immunological problems, such as how enormous numbers of microbes may dwell within animals and humans while maintaining a healthy functional immune system, have resurfaced as a result of the recent spike in research interest in the commensal microbiome. How do some microorganisms manage to evade and subvert the immune system while others don't? The communication network we must tap into as researchers to address these issues is the sophisticated and important-to-remember two-way interaction and coevolution of host and microorganism.

The Immune System of the Host the innate and adaptive immune systems are the two primary branches of the vertebrate immune system. Because the innate immune system is the first line of defence against foreign invaders, innate immunity cells are equipped with Pattern Recognition Receptors, (PRRs) which are "generalist" receptors that recognize molecular patterns seen on bacteria Immunological cells' PRRs attach to germs or microbial components and induce immune responses. This recognition is not microbe specific, and instead of a discriminating antigen, it depends on recognition motifs found in a range of bacteria. The adaptive immune system is more evolved and capable of distinguishing different microorganisms.

In the example of bacteria, when elimination of bacteria is not carried out by the innate PRR induced activities, both bacteria lysis and phagocytosis can be induced by opsonization facilitated by the adaptive immune system as well other adaptive responses Complement mediated lysis of invasive bacteria outside the cell requires the binding of specific antibodies to the target bacteria to initiate a complex capable of invading and disrupting the bacterial cell. These antibodies are produced by B-cells of the adaptive immune system. Phagocytosis can also be initiated by the binding of antibodies and acute phase proteins such as C-reactive protein and serum amyloid A and P to allow presentation on Major Histocompatibility Complex (MHC)-II of macrophages. TCR binding to MHC-II induces the generation of cytokines, which in turn activates intracellular bacteria killing mechanisms such lysozyme activity. A number of studies have described modifications in host immunological signaling caused by bacteria]. As previously stated, activation of PRRs is one of the host's initial lines of defence against germs. Toll-Like Receptors (TLRs), nucleotide-binding and oligomerization (NOD) like receptors (NLRs), and C-type lectin receptors (CLRs) are the three main PRRs involved in bacterial elimination by the innate immune system.

Following the activation of these receptors, signaling cascades involving Mitogen-Activated Protein Kinase (MAPK), interferon nuclear Factor kappa-light-chain enhancer of activated B cells, as well as other signaling proteins that induce inflammatory responses and clear bacteria, are activated.

Protein kinases are essential for the activation and control of these pathways. Later, we'll look at how bacteria can use these kinases to impact the immunological response of the host. Bacterial Inflammation of the Host a significant percentage of immunological research on the host–pathogen interaction focuses on the negative consequences of its (dys) function or how it can be controlled in a sick state. This is understandable given that researchers are attempting to investigate important disease-causing pathogens. Understanding the immune system's optimal functioning in the setting of a quickly removed infectious threat (rather than a serious or chronic disease) may help us understand the immune system's core processes and optimal response to microorganisms. What dynamic are we examining, an efficient immune mobilization to treat an infectious agent or a microbial beneficial response tailored to dodge the immunological response, for researchers studying host–pathogen interactions that result in disease states? Salmonella, for example, causes inflammatory reactions in the gut epithelial layer and

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then uses the resulting permeability to penetrate the gut barrier]. While the inflammatory reaction kills many bacteria, it also allows Salmonella to colonies the host. Salmonella's survival and spread within the host are dependent on this invasion across the endothelium. Although Salmonella has been found to thrive in the lumen it is an extremely unfriendly environment for long-term survival.

Salmonella is carried by some species, which accept it and allow it to become part of the commensal microbiota, allowing it to shed continuously. In other species, such as humans, Salmonella-induced inflammatory immune responses cause diarrhoea, which clears the lumen's contents, including nutrients, debris, and bacteria, necessitating invasion. Salmonella's capacity to take advantage of the inflammatory immune response is both advantageous and necessary for its survival. Infection of the epithelial or immune cells that line the lumen can cause the epithelial barrier to be disrupted, allowing bacteria to spread to the lamina propria through inflammation produced by their presence. Salmonella must also pass the endothelium barrier and move through the bloodstream to successfully infect organs outside of the Gastrointestinal (GI) tract. This is advantageous to the bacteria because colonization various organs are the best way for them to thrive and survive. Crossing the endothelium into the blood causes septicemia and other life-threatening health issues, especially in immunocompromised individuals. In this situation, who is replying to whom? Perhaps the answer to such concerns eludes us because studying the immune system when it is functioning routinely and appropriately, or in homeostatic balance with a microbial population is challenging. Bacteria Kinases as an Example the various distinct alterations in signal transduction that can occur during exposure to pathogenic and commensal bacteria lead host immune responses to bacteria to vary. Bacteria have the ability to influence not only the host gene expression but also the proteome by modifying proteins directly or indirectly. We'll look at one example, phosphorylation of the host proteome by bacterial kinases, as a Post-Translational Modification (PTM). The addition of a gamma phosphate from an Adenosine Triphosphate (ATP) molecule to a specific serine, threonine, or tyrosine amino acid residue in a protein is referred to as phosphorylation. Kinases are enzymes that catalyze this covalent activity. Kinases can be found in both eukaryotes and prokaryotes. Kinase comes from the Greek word kinein, which meaning "to move." Kinases are phosphotransferases, which means they transfer phosphate groups from one chemical molecule to another. They aren't just found in proteins. Canonical kinases and pseudo kinases are two types of kinases. Canonical kinases are catalytically active, while pseudo kinases are those that have lost activity due to evolution. Pseudo kinases, on the other hand, are inactive kinases that developed alongside catalytically active kinases but lack crucial kinase needs. According to research, pseudo kinases may play a key function in the regulation of other kinases. Pseudo kinases are found in eukaryotes and prokaryotes alike. . A wide superfamily of canonical protein kinases contains biologically important kinases that are known to be involved in regulatory cellular functions such as metabolism, immunological control, cell maintenance, and so on. The protein-Serine Threonine Kinases (STKs) and the Protein Tyrosine Kinases (PTKs) are the two primary subfamilies of canonical protein kinases in eukaryotes. Many cellular activities rely on these kinases to turn on and off. Protein serine threonine kinases phosphorylate the oxygen of a Hydroxyl (OH) group on serine or threonine amino acids and are either membrane-bound or intracellular signaling proteins. MAPK kinases, protein kinase (A, B, C), Casein kinase, calcium calmodulin kinases, and other protein-serine threonine kinases are examples of protein-serine threonine kinases. Protein-Tyrosine Kinases (PTKs) are protein kinases that phosphorylate the tyrosine residue of a protein. They can be transmembrane or cytoplasmic. PTKs include receptor tyrosine kinases like Epithelial Growth Factor Receptor (EGFR), Platelet Derived Growth Factor Receptor (PDGFR), and Vascular Endothelial Growth Factor Receptor (VEGFR), as well as non-receptor tyrosine kinases Including Janus kinase, SRC, and SYK family. One of the four main groups of bacterial kinases contains homologous and orthologous STK subfamily kinases. Eukaryotic-like serine threonine kinases are a class of bacterial kinases that are comparable to STKs (eSTKs). ESTKs have a variety of roles in the survival of bacteria in their hosts, including modification of the host proteome, bacterial cell wall construction, and bacterial metabolism, among others (Ozone 2005).

Salmonella and Yersinia eSTKs have been demonstrated to directly regulate host actin and cytoskeletal activity via MAP, myosin, and Rho/Rac kinase activities, altering processes like phagocytosis, cell proliferation, and vesicle formation. M. tuberculosis kinases have the ability to change metabolism in host cells by blocking the control of tricarboxylic Acid (TCA) cycle enzymes. This bacterium's kinases have also been demonstrated to reduce the host's inflammatory response while increasing bacteria burdens]. Bacterial Tyrosine (BY) kinase is another notable class of bacteria kinases. BY kinases are phosphorylases that phosphorylate tyrosine residues in bacteria. They are not similar to eukaryote Hanks-type tyrosine kinases, but more research is needed to establish orthology. For decades, human medicine has recognized the value of modulating kinase function in health and disease, and a multibillion-dollar research and development effort has concentrated on inhibiting kinase activity in the treatment of malignancies. Microbes have taken advantage of the hSost's usage of kinase-mediated signal transduction to aid invasion and persistence, which is a great example of microbe–host coevolution. To underline the importance of kinases even more, a curation of human protein kinases involved in immune response using Uniprot KB yielded 234 reviewed entries. Over 200 of the 234 kinases are also involved in metabolism, demonstrating the critical role of immune metabolism in properly comprehending microbe–host interactions.

The results of this curation reveal that bacteria have a plethora of targets of opportunity in the context of kinases that can influence the immune system.

Crosstalk between microbes and hosts, as well as commensals the interactions between pathogenic bacteria with the host have been investigated in greater depth than commensal microorganisms. Despite commensals' evident benefits and fascinating immune system impacts, more is known about their kinase action on hosts than commensal bacteria. Balance and homeostasis are essential for the microbiota and host to interact in a mutually beneficial way. The microbiota finds a safe haven in the host, where temperature, oxygen levels, and nutrition availability are all beneficial. The microbiota helps the host's metabolism through its actions. For example, by generating vitamins and boosting the digestion of complex carbs, and therefore increasing nutrient availability. Pathogens are competitively excluded by a well-established microbiota, which outcompetes pathogens for resources and an ecological niche within the host. For the immune system's development and the

host. For the immune system's development and maturation, the commensal microbiota delivers vital signals. The number of microbes in the digestive tract, predominantly bacteria, demonstrates the intricate interaction between the microbiota and the human immune system. If bacteria cross the epithelial barrier or non-commensal microbes enter the intestinal lumen unchecked, the microbiota can be hazardous to the host. Life events can disturb the microbial niche, resulting in symbiosis, which can favor infections.

Apart from the metabolic benefits mentioned above, such disturbance of the microbiome may reduce the tolerogenic signals produced by the microbiome, resulting in pro inflammatory responses that impair immunological homeostasis. Such unfavourable effects have been connected to disorders in many other systems of the host's body, including diabetes, atopic dermatitis, multiple sclerosis, asthma, and others]. Secretory immunoglobulin A is an example of an immune response with subtle cross-talk between the host and the pathogen (sIgA). SIgAs are key antibodies in the mucosal lining for starting immune responses and competitive inhibition of bacteria attaching to epithelial cells in the mucosa. The outcomes of the studies on the specificity of sIgA in eliminating commensal bacteria versus harmful germs are mixed.

The host immune system does not respond to commensal bacteria during homeostasis, at least not in the same way that it responds to pathogenic or foreign bacteria. Perhaps this is the outcome of a symbiotic coevolution between the microbiota and its hosts, which has resulted in a permanent alteration in physiology to allow microbial tolerance]. Researchers believe that bacteria and vertebrates have coexisted for around 0.5 billion years; this dynamic interaction has altered the microbial community and immune system, allowing the immune system to tolerate the microbiota]. The evolution of PRR in innate immune cells can be traced back to the need for immunological specificity in Cnidarians (invertebrates) to identify symbionts from infections. The evolution of vertebrates parallels the evolution of the adaptive immune system. Similar to their invertebrate forerunners, the oldest vertebrates needed antigen recognition receptors on T-cells and B-cells, as well as long-lasting immune memory, to allow precise identification of and appropriate response to a vast majority of antigens, not only as an upgrade to the host defence but also to distinguish between friends and foes.