

Special Issue on  
**Modulation of Host Inflammatory Signaling by Microbial Pathogens**

# CALL FOR PAPERS

The primary role of inflammatory responses is to counteract infections and tissue injuries. Innate immune cells recognize invasion of pathogens or pathogen-driven cell damage by pattern recognition receptors (PRRs). These receptors detect pathogen-associated molecular patterns or damage-associated molecular patterns (PAMPs/DAMPs) from infected or injured cells. Activation of PRRs triggers intracellular signaling cascades and the expression of a variety of proinflammatory molecules, which together form the early host response to infection. This system is tightly regulated and is also a basic requirement for the subsequent activation of adaptive immunity, ideally resulting in control or eradication of bacteria.

In turn, microbial pathogens have evolved intriguing mechanisms of pathogenesis, including effective strategies to subvert host immune defenses. These microbes modulate, circumvent, and undermine innate and adaptive host defense systems for their own benefit and survival. To this end, they employ various molecular mechanisms including strategies to escape or hide from the immune system by altering surface exposed molecules and manipulating host's signaling cascades by a plethora of virulence factors. In addition, some bacteria secrete toxins, which can reverse or inhibit protective host functions and thus promote colonization of the host.

Our understanding of bacterial strategies to manipulate inhibitory signaling pathways can open novel and promising opportunities to counteract microbial escape mechanisms and tip the balance in favor of the host. In addition, novel discoveries of the functional roles of inhibitory pathways may provide useful insights into limiting host responses and into extenuating infection pathologies. Furthermore, unraveling the crosstalk between microbial pathogens and the host immune system may have translational implications for designing more effective regimens to treat immune disorders.

Here, we cordially invite and encourage authors to submit their original research articles as well as review articles that will help in understanding the modulation of inflammatory signaling by microbial pathogens on the molecular basis and cell biology including acute/chronic inflammation and mediators of inflammation, as well as cellular processes and molecular mechanisms involved in the production of inflammatory responses linked to microbial infections. We especially welcome the submission of articles on anti-inflammatory drug development, trials, and therapies based on probiotic microbes or anti-inflammatory microbial molecules.

Potential topics include but are not limited to the following:

- ▶ Microbial infection and inflammation, including acute/chronic inflammation
- ▶ Host cell signaling responses to microbial infections
- ▶ Host cell signaling responses to commensals
- ▶ Cellular processes and molecular mechanisms involved in the production of inflammatory responses
- ▶ Modulation of immune response and cellular signaling by microbial effectors
- ▶ Microbes and microbial molecules as potential therapeutics for autoimmune diseases

Authors can submit their manuscripts through the Manuscript Tracking System at <https://mts.hindawi.com/submit/journals/iji/mhisp/>.

Papers are published upon acceptance, regardless of the Special Issue publication date.

**Lead Guest Editor**

Christian Rüter, University of Münster,  
Münster, Germany  
[rueterc@uni-muenster.de](mailto:rueterc@uni-muenster.de)

**Guest Editors**

Karin Loser, University of Münster,  
Münster, Germany  
[loserk@uni-muenster.de](mailto:loserk@uni-muenster.de)

Stefan Schild, University of Graz, Graz,  
Austria  
[stefan.schild@uni-graz.at](mailto:stefan.schild@uni-graz.at)

Philip R. Hardwidge, Kansas State  
University, Manhattan, USA  
[hardwidg@vet.k-state.edu](mailto:hardwidg@vet.k-state.edu)

**Submission Deadline**

Friday, 9 November 2018

**Publication Date**

March 2019