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RECEIVED 03 October 2024 ACCEPTED 07 October 2024 PUBLISHED 18 October 2024

CITATION

Planz O and Kircheis R (2024) Editorial: Community series in the role of toll-like receptors and their related signaling pathways in viral infection and inflammation, volume II. Front. Immunol. 15:1505715. doi: 10.3389/fimmu.2024.1505715

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Editorial: Community series in the role of toll-like receptors and their related signaling pathways in viral infection and inflammation, volume II

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KEYWORDS

toll-like receptor, signaling pathway, viral infection, inflammation, tolerance

Editorial on the Research Topic

Community series in the role of toll-like receptors and their related signaling pathways in viral infection and inflammation, volume II

The second volume of our Community Series on "The Role of Toll-like Receptors and their Related Signaling Pathways in Viral Infection and Inflammation" delves into the intricate mechanisms through which TLRs influence the immune response against viral pathogens. This Research Topic, led by diverse researchers, expands our understanding of TLRs and their role in inflammation.

Included studies comprehensively examine how TLR activation regulates different stages of inflammation. For example, research by Butcher et al. highlights macrophage tolerance in TLR signaling, crucial for adapting to chronic endotoxin exposure and mitigating prolonged pro-inflammatory cytokine production. This adaptation aims to protect host tissues from damage, observed across various TLR ligands, particularly TLR4's ligand lipopolysaccharide (LPS), which induces global shifts towards anti-inflammatory responses.

Additionally, Ricci-Azevedo et al. discuss lectins as TLR agonists with immunomodulatory properties. Plant lectin ArtinM and microbial lectins interact with TLR2 and TLR4, triggering NF-κB activation and IL-12 production *in vitro*. *In vivo* studies show these lectins confer resistance to intracellular pathogens. Lectins from pathogens like Toxoplasma gondii and Paracoccidioides brasiliensis also activate TLRs, suggesting potential for new pharmaceutical tools against infections and tumors.

Moreover, Lu et al.'s review underscores TLRs' significant role in inflammatory bowel disease (IBD), a chronic global condition. Dysfunctions in TLR-mediated pathways

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contribute to IBD pathogenesis and influence treatment efficacy, prompting exploration of novel therapeutic strategies.

Lastly, Popotas et al. explore sex-specific immune responses in acute inflammatory diseases, attributing these differences to genes on the X chromosome encoding TLRs. Their review highlights distinct inflammatory patterns between sexes, crucial for tailoring clinical approaches.

This volume aims to advance our understanding of TLRs in inflammation, driving targeted therapy development and improving patient outcomes. We extend gratitude to all contributors for their invaluable insights and dedication to this critical field.

Author contributions

OP: Conceptualization, Writing – original draft. RK: Writing – review & editing.

Conflict of interest

Author RK was employed by company Syntacoll GmbH.

The remaining author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

The author(s) declared that they were an editorial board member of Frontiers, at the time of submission. This had no impact on the peer review process and the final decision.

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