Special Issue

Molecular Mechanism of Innate and Acquired Immunity in Tissue Injury

Message from the Guest Editor

Various types of cell death, such as apoptosis, pyroptosis, and necrosis, occur in injured tissues. Dead cells subsequently release danger-associated molecule patterns (DAMPs) including DNA, RNA, ATP, uric acid, high-mobility group box 1 (HMGB1), and S100 proteins. These molecules can collectively regulate the migration and activation of various types of immune cells, including granulocytes, lymphocytes, and dendritic cells. The immune cells can produce various cytokines, chemokines, and lipid mediators, thereby modulating the host response to tissue injury and promoting subsequent tissue repair. Thus, innate and acquired immunity is presumed to contribute crucially to the tissue repair process. However, it remains elusive how DAMPs and other mediators coordinate innate and acquired immune responses to tissue injury. Hence, in this Special Issue on tissue injury, we will discuss various types of mediators including DAMPs, cytokines, chemokines, and lipid mediators, which are deeply involved in the innate and acquired immune responses to tissue injuries—particularly those in clinically relevant situations.

Guest Editor

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The International Journal of Molecular Sciences (*IJMS*, ISSN 1422-0067) is an open access journal, which was established in 2000. The journal aims to provide a forum for scholarly research on a range of topics, including biochemistry, molecular and cell biology, molecular biophysics, molecular medicine, and all aspects of molecular research in chemistry. *IJMS* publishes both original research and review articles, and regularly publishes special issues to highlight advances at the cutting edge of research. We invite you to read recent articles published in *IJMS* and consider publishing your next paper with us.

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