## **Special Issue**

# Pertussis Toxin and Research on Pertussis Vaccine

## Message from the Guest Editors

Pertussis toxin (PT) is a key virulence factor of the whooping cough agent Bordetella pertussis. It induces leukocytosis and immune suppression resulting in potentially critical illness in infants. Chemically or genetically toxoided forms of PT are key protective antigens of acellular pertussis vaccines. However, many aspects of PT biogenesis, structure-function relationships, intracellular trafficking within host cells, as well as the many targets and physiological and immunological consequences of PT's action in host cells and tissues remain insufficiently characterized. The aim of this SI is to re-invigorate the interest in basic studies on PT and on its use in improved pertussis vaccines to help containing the ongoing resurgence of the disease, even in the highly vaccinated populations. Submissions are welcome on following topics:

- Biogenesis and secretion of PT
- Structure-function relationships, cellular targets, and mechanism of PT action
- Receptors, intracellular trafficking, translocation of PT
- Physiological and immunological effects of PT
- Production, purification, detoxification and vaccine use of PT
- Immune responses to PT-based vaccines

## **Guest Editors**

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## Deadline for manuscript submissions

closed (15 September 2021)



## **Toxins**

an Open Access Journal by MDPI

Impact Factor 4.0
CiteScore 8.2
Indexed in PubMed



mdpi.com/si/64940

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Toxinology is an incredibly diverse area of study, ranging from field surveys of environmental toxins to the study of toxin action at the molecular level. The editorial board and staff of *Toxins* are dedicated to providing a timely, peer-reviewed outlet for exciting, innovative primary research articles and concise, informative reviews from investigators in the myriad of disciplines contributing to our knowledge on toxins. We are committed to meeting the needs of the toxin research community by offering useful and timely reviews of all manuscripts submitted. Please consider *Toxins* when submitting your work for publication.

#### **Editor-in-Chief**

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