Special Issue

Complement System Entry Suspense: A Hero or Villain in Rare and Genetic Diseases

Message from the Guest Editor

A complement is a group of about fifty liquid and cell membrane-associated proteins. Complement activation is a complex process largely materializing in three different ways, i.e., through classical, alternative, and lectin pathways. Complement activation using the classical pathway occurs due to the ligation of IgG/IgM immune complexes (ICs) to their receptors and/or C1q, as well as the binding of C1q to certain molecules released from injured cells. The lectin pathway is activated through the binding of the mannan-binding lectin, a serum protein, to mannose-containing carbohydrates or related ficolins to certain carbohydrates or acetylated structures. The alternative pathway can be initiated when a spontaneously activated complement component binds to the surface of a pathogen.

Complement activation and the production of several of its downstream molecules are essential for controlling cellular and metabolic functions. This Special Issue invites original research articles, reviews, and opinions describing the molecular mechanism through which the complement system activates/suppresses and modifies the disease processing of rare and genetic illnesses.

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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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