Special Issue

Myasthenia Gravis and Innate Immunity—Dedicated to the Memory of Dr. Pia Bernasconi

Message from the Guest Editors

This Special Issue is dedicated to the memory of our colleague and friend Pia Bernasconi, an enthusiastic scientist with a great passion for research, whose studies significantly contributed to the understanding of the pathogenetic link between innate immunity and autoimmunity in myasthenia gravis.

Myasthenia gravis (MG) is a prototypic B cell-mediated autoimmune disease, mainly caused by autoantibodies to the acetylcholine receptor (AChR) located at the neuromuscular junction. Its pathogenesis is closely linked to morphological and functional alterations (follicular hyperplasia, thymoma) of the thymus, which is widely considered the main site of autoimmunity onset in MG patients. Over the past decade, an increasing body of evidence has been accumulated showing that pathogen infections (e.g. Epstein–Barr virus), dysregulated Toll-like receptor-mediated signaling pathways, interferon overexpression, and chronic inflammation are critically involved in the intrathymic MG development. This Special Issue aims to summarize new insights on the pathogenic cross-talk between innate immunity and autoimmunity in MG.

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Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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