Special Issue

New Insights into Psoriasis

Message from the Guest Editors

Psoriasis is a common, chronic, inflammatory, immunemediated skin disease. The main cause of the disease is the overactivation of the skin's innate and acquired immune system cells by external factors and internal factors. These cells secrete cytokines, chemokines and growth factors that stimulate keratinocyte proliferation, neoangiogenesis and inflammation. The IFNN, TNFN and IL-23/IL17 axis, which activates the NF-\□B pathway, plays a dominant role in the pathogenesis of the disease. A complex interaction between epigenetics, genetics and the inflammatory signaling networks of skin and immune cell mediators has been proved in many studies. Psoriasis has been shown to be associated with the mutation of more than 60 genes. Regarding epigenetic mechanisms, histone modifications, promoter methylations, and long noncoding RNA and microRNA hyperexpression are considered to be additional factors contributing to psoriasis pathogenesis. In this Special Issue, we will publish recent data regarding the pathogenesis, comorbidities and therapy of psoriasis. We invite you to submit original and review publications based on the results of molecular research.

Guest Editors

Dr. Bogusław Nedoszytko

Prof. Dr. Agnieszka Owczarczyk-Saczonek

Prof. Dr. Dorota Krasowska

Dr. Aneta Szczerkowska-Dobosz

Deadline for manuscript submissions

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International Journal of Molecular Sciences Editorial Office MDPI, Grosspeteranlage 5 4052 Basel, Switzerland Tel: +41 61 683 77 34 ijms@mdpi.com

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Message from the Editor-in-Chief

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.

Editor-in-Chief

Prof. Dr. Maurizio Battino

Department of Odontostomatologic and Specialized Clinical Sciences, Sez-Biochimica, Faculty of Medicine, Università Politecnica delle Marche, Via Ranieri 65, 60100 Ancona, Italy

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