Special Issue

Autophagy in the Nervous System

Message from the Guest Editor

Autophagy is considered to be one of the main A□-eliminating pathways under normal conditions. The ubiquitin proteasomal system may be the primary mechanism to degrade endogenous tau, while aggregated tau does not undergo degradation by the proteasome. Autophagy is involved in the clearance of soluble and aggregated tau and NFT in the cell. Moreover, autophagy activation can reduce the secretion of tau and tau propagation. These facts imply that autophagy activation can be beneficial for AD via clearance of A□ or tau. In fact, several therapeutic studies have been performed by modulating autophagy, including mTORC1-dependent and -independent autophagy inducers, other autophagy-inducing drugs, and gene therapy, including microRNA. However, it has been reported that A□ can be produced in autophagy, and A□ secretion can also be done by autophagy. This Special Issue will offer us precious information regarding autophagy and A□ or tau, and autophagy and alpha synuclein, TDP43, and huntingtin. Additionally, it may shed light on whether autophagy activation is beneficial for AD and other neurodegenerative disorders causing dementia.

Guest Editor

Dr. Tadanori Hamano

Second Department of Internal Medicine, Division of Neurology, University of Fukui, Fukui, Japan

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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

mdpi.com/journal/ijms

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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.

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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