Special Issue

miRNA-Mediated Posttranscriptional Regulation in the Nervous System

Message from the Guest Editors

MicroRNAs (miRNAs) are small non-coding RNAs (sncRNAs) that, by binding to the 3'UTR of target mRNAs, suppress their translation and/or initiate their degradation. Two decades of research on the functional and molecular characterization of neuronal miRNAs has clearly demonstrated that they are key regulators of neuronal differentiation, brain development and plasticity mechanisms in post-natal neurons. In the nervous system, miRNAs have very diverse and highly specific spatiotemporal patterns of expression and function. Interestingly, external stimuli and neuronal activity modulate their expression and function, through regulatory pathways that involve RNA binding proteins (RPBs) and other RNA interactors. The essential contribution of miRNAs to brain homeostasis is proven by the fact that miRNA loss of function or deregulation is highly associated with neural disease.

This Special Issue aims to discuss recent findings on the molecular mechanism of miRNA-dependent gene expression regulation in the brain. We welcome original research, reviews, and other article types presenting newly discovered miRNA molecular mechanisms and their co-actors in the nervous system.

Guest Editors

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Deadline for manuscript submissions

closed (30 December 2023)



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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

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