Special Issue

Fragile X Syndrome: Molecular Mechanisms, Cellular and Animal Models, and Targeted Therapeutics

Message from the Guest Editors

The discovery of the *FMR1* gene as the cause of the fragile X syndrome, a frequent form of intellectual disability and autism, can be considered one of the major breakthroughs in medical genetics. A dynamic expansion of a trinucleotide CGG repeat from parent to child explained the "anticipation" in the families, e.g., the increase in the number of affected patients with generations. The neurodevelopmental disorder is caused by a full expansion of the CGG repeat accompanied by epigenetic alterations in the youngest generations. In the three decades that passed since the gene discovery, understanding the molecular mechanisms underlying fragile X syndrome, generation of cell and animal models, and new therapeutic strategies has been the focus in fragile X syndrome research. This Special Issue aims to provides a snapshot of our continuing search for causes and treatment of the fragile X syndrome, which has evolved as a prime example for translational research in neurodevelopmental disorders.

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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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