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

Guest Editors:

Dr. Rob Willemsen
Department of Clinical Genetics, Erasmus Medical Center DR Rotterdam, Rotterdam, The Netherlands

Dr. Frank Kooy
Department of Medical Genetics, Universiteit Antwerpen, Antwerpen, Belgium

Deadline for manuscript submissions:
closed (1 March 2022)

Message from the Guest Editors

Dear Colleagues,

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.

Dr. Rob Willemsen

Dr. Frank Kooy
Guest Editors

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Editors-in-Chief

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Prof. Dr. Cord Brakebusch
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Message from the Editorial Board

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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Contact Us

Cells Editorial Office
MDPI, St. Alban-Anlage 66
4052 Basel, Switzerland

Tel: +41 61 683 77 34
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cells@mdpi.com
𝕏@Cells_MDPI