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Role of ATM and MRE11 in Genomic Stability and Oxidative Stress Responses

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Deadline for manuscript submissions:
closed (15 August 2022)

Message from the Guest Editors

Genetic disorders, which are defective in *ATM* or *MRE11*, are categorized in radiation-hypersensitive disease and show similar cellular phenotypes, such as radioresistant DNA synthesis and chromosome instability, as well as radiation hypersensitivity. Many studies in the last few decades, have identified ATM and MRE11 as critical players in the maintenance of genomic stability against various types of DNA damages, including radiation-induced DNA double-strand break (DSB) and replication stress. The *ATM* gene product shows protein kinase activity, which is activated in response to oxidative stress as well as generation of DSB damages. Such an activity upon oxidative stress is suggested to play a role in repressing neurodegeneration.

In this Special Issue, we welcome reviews and original articles covering many aspects of the role of ATM, MRE11, and related factors (ATR, NBS1, RAD50, etc.) in maintaining genomic stability or resisting oxidative stress. We also welcome reviews or original articles providing the clues to solve the mechanisms of pathogenesis in these genetic disorders. We look forward to your contribution.

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Message from the Editor-in-Chief

Genes are central to our understanding of biology, and modern advances such as genomics and genome editing have maintained genetics as a vibrant, diverse and fastmoving field. There is a need for good quality, open access journals in this area, and the *Genes* team aims to provide expert manuscript handling, serious peer review, and rapid publication across the whole discipline of genetics. Starting in 2010, the journal is now well established and recognised.

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