

Special Issue

Protective Mechanisms Against DNA Replication Stress

Message from the Guest Editors

Replication stress has emerged as one of the primary challenges our cells face when replicating their genome. Replication forks routinely encounter hindrances that stall the progression of DNA polymerases, higher-order structures that are difficult to replicate, and actively transcribed regions. Replication fork stalling generates single-stranded DNA that triggers protective pathways that either enable the replication fork to restart or protect nascent DNA from degradation. Failure in these mechanisms leads to replisomes' disassembly and collapse into double-strand breaks. During mitosis, unresolved replication stress gives rise to anaphase bridges, fragile sites, and supernumerary centrosomes that lead to multipolar spindles and aneuploidy. Replication stress is therefore a major driving force of genome instability and has been proposed to be involved in early stages of cancer and senescence. Inhibitors of critical replication stress signaling cascades have gained clinical importance in recent years, as has our understanding of how replicative exhaustion accelerates aging and replicative DNA damage induces inflammation.

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Genes is central to our understanding of biology, and modern advances such as genomics and genome editing have maintained genetics as a vibrant, diverse and fast-moving 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. Why not consider *Genes* for your next genetics paper?

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