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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Deadline for manuscript submissions

closed (31 January 2020)

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Impact Factor 2.8
CiteScore 5.2
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