Abstract Submitted
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Defects and DNA replication[1] MICHEL GAUTHIER, JOHN HERRICK, JOHN BECHHOEFER, Simon Fraser University — In higher organisms, DNA replication is initiated at distinct sites called replication origins, where pairs of replication forks begin to duplicate DNA bi-directionally outward from the origin site until they eventually coalesce with another fork. Unfortunately, defects along the DNA (such as single-strand DNA lesions or double-strand breaks) can slow, or even stall, replication forks. We introduce a master-equation formalism to study DNA replication kinetics in the presence of defects resulting from DNA damage and find a crossover between two regimes: a normal regime, where the influence of defects is local, and an initiation-limited regime. In the latter, defects have a global impact on replication, whose progress is set by the rate at which origins of replication are activated, or initiated. Normal, healthy cells have defect densities in the normal regime. Our model can explain an observed correlation between interorigin separation and rate of DNA replication.

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