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The role of oxidative stress as a mutational mechanism on telomeric deletion events

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Telomeres are nucleoprotein structures that contain non-coding (TTAGGG) tandem repeats and associated telomere binding proteins at the end of chromosomes. As a consequence of end-replication losses, telomeres undergo gradual erosion with ongoing cell division. It is hypothesized that in addition to the end-replication problem, mutational mechanisms may contribute to telomere erosion and generates large-scale telomeric deletions. As short dysfunctional telomeres are capable of fusion to other chromosome ends, large-scale telomeric deletion can lead to genomic instability that may drive tumor progression. The aim of this study is to observe if oxidative stress contributes to telomere erosion and large-scale telomeric deletion. By undertaking a comprehensive analysis of telomere dynamics following the induction of oxidative stress, the data presented here showed that oxidative damage does not appear to affect the rate of telomere erosion or the frequency of large-scale telomeric deletion. Instead prolonged exposure to oxidative stress results in the preferential loss from the culture of sub-populations of cells that exhibit short telomeres. We conclude that loss of these cells from the culture may be due to a preferential sensitivity to damage that may be related to these cells being closer to their replicative limit. These data are more consistent with the view that premature senescence does not arise as a consequence of accelerated telomere erosion but instead more likely results from stochastic DNA damage across the rest of the genome.

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