Abstract

The distribution of camptothecin (CPT)-induced break points in late S or G2 phase of the cell cycle observed in Chinese hamster chromosomes was analysed in 400 metaphases. Contrary to expectation, they were not localized in the heterochromatic regions, suggesting that these chromatid-type aberrations arise by a mechanism which does not involve collision of the CPT-trapped 'cleavable complex' with the replication fork. Since many break points mapped more frequently to light bands (DAPI negative) than dark bands (DAPI positive) with a frequency of 73 and 15% respectively, it could be argued that the presence of the CPT-trapped 'cleavable complex' probably interferes with chromatin condensation. In fact, the euchromatic regions, which are expected to be more actively condensed in G2 phase, were more involved in chromosomal damage. These results do not completely confirm the idea that some residual DNA synthesis occurring in G2 is responsible for the G2 clastogenic effects of CPT as the heterochromatic regions should, in this case, be more involved.