

## Abstract

Frequencies of symmetrical and asymmetrical exchange aberrations induced by two inhibitors of topoisomerase II, namely, 4'-(9-acridinylamino) methanesulfon-m-anisidide (m-AMSA) and etoposide (VP16), were estimated in human peripheral blood lymphocytes. The aberrations were scored using conventional Giemsa staining and fluorescence in situ hybridization (FISH) techniques, using chromosome-specific DNA libraries. Stable aberrations (translocations) were detected using two cocktails of DNA libraries specific for three chromosomes, namely 1, 3 and X and 2, 4 and 8, representing approximately 40% of the whole human genome. The frequencies of dicentrics and translocations increased in a dose-dependent manner, however, m-AMSA was found to be a more potent inducer of chromosomal aberrations in comparison with VP16 (at concentrations at which comparable frequencies of aberrations were induced) by 20- to 30-fold. When corrected for DNA content of chromosomes in each cocktail, a higher frequency of translocations with the cocktail consisting of chromosomes 2, 4 and 8 in comparison with 1, 3 and X was evident. The genomic translocation frequency calculated from chromosome painting analysis for m-AMSA exceeded that estimated for dicentrics by approximately 2-fold. However, for VP16 almost equal frequencies of both types of chromosome exchange were found