Influence of DMSO on Carbon K ultrasoft X-rays induced chromosome aberrations in V79 Chinese hamster cells

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ABSTRACT

Ultrasoft X-rays have been shown to be very efficient in inducing chromosomal aberrations in mammalian cells. The present study was aimed to evaluate the modifying effects of DMSO (a potent scavenger of free radicals) on the frequencies of chromosome aberrations induced by soft X-rays. Confluent held G1 Chinese hamster cells (V79) were irradiated with Carbon K ultrasoft X-rays in the presence and absence of 1 M DMSO and frequencies of chromosome aberrations in the first division cells were determined. DMSO reduced the frequencies of exchange types of aberrations (dicentrics and centric rings) by a factor of 2.1–3.5. The results indicate that free radicals induced by ultrasoft X-rays contribute to a great extent to the induction of chromosome aberrations. The possible implications of these results in interpreting the mechanisms involved in the high efficiency of ultrasoft X-rays in the induction of chromosome aberrations are discussed.

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1. Introduction

Ionizing radiations (IR) have been shown to be very efficient in inducing chromosome aberrations (CA) with great efficiency in diverse types of cells from plants to human. High LET radiations (α particles, neutrons) are more effective in inducing CA compared to low LET radiations (X-rays, γ -rays). The yields of exchange type of aberrations (dicentrics and rings) following X-irradiation of G1 cells are known to increase with dose in a linear-quadratic manner and this has been interpreted as due to the interaction of two independent events (DNA double strand breaks – DSB) at high doses. With high LET radiations, these yields are usually linear due to single ionization track can lead to two DSBs due to correlation of events along the track of the particle (for reviews, see Refs. [1,2]).

Ultrasoft X-rays (0.1–5.0 keV) provide a unique tool for probing mechanisms radiation induced biological effects. They interact within the cell almost exclusively by the photoelectric effect producing isolated electron tracks of low, well-defined energy with dimension comparable to DNA and associated structures. Data from a number of laboratories show ultrasoft X-rays to have increased effectiveness for a wide range of biological end-points compared to equal doses of conventional X-rays or γ -rays [3–5], with RBE

(Relative Biological Efficiency) values for a range of biological end-points (cell inactivation, mutation, transformation, DSB induction and chromosome aberration induction) typically increasing with decreasing ultrasoft X-ray energy down to C_K X-rays (0.28 keV). Ultrasoft X-rays show all the characteristic features of low-LET radiation [6] including rejoining kinetics of DSB similar to ^{60}Co γ -rays apart from an indication of a higher residual yield [7]. Carbon K X-rays are of particular interest, because they produce a single photoelectron with a mean range of ~ 4 nm and a practical range of ~ 7 nm [8], which are only slightly larger than the DNA helix width.

Carbon K ultrasoft X-rays have been found to be very efficient in inducing exchange types of CA (dicentrics, translocations) in human and Chinese hamster cells in comparison to 250 kV X-rays [9–12]. A predominantly linear dependency with dose was observed for chromosome exchanges, suggesting a possible recombination misrepair process with undamaged DNA (or non-radiation induced lesions) might be involved, since a track produced by Carbon K X-rays would seldom be expected to traverse more than one DNA helix [12,13]. These Carbon K X-rays also produced a relatively high frequency of complex exchanges with a non-linear dose response and this was proposed to be due to the increased efficiency of DSB induction leading to slow repairing lesions coming together at specific repair sites [12].

It has been demonstrated earlier that dimethyl sulfoxide (DMSO, a potent scavenger of radiation induced free radicals) protects against ionizing radiation (IR) induced biological effects, such as, DNA double strand breaks (DSBs), cell killing, chromosomal aber-

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