Quantification of γ-H2AX foci after exposure to I-123-iododeoxyuridine in comparison to γ- and α-irradiation

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Introduction: Phosphorylation of histone H2AX occurs at sites flanking DNA double-strand breaks (DSBs) and can provide an indirect measure for the number of DSBs within a cell. Recent publications suggest LET-dependent differences in the intensity and size of γ-H2AX foci. To determine whether γ-H2AX foci caused by DNA-associated Auger electron emitters (AEE) induce high-LET type γ-H2AX foci we investigated the mean intensity as well as the mean number of γ-H2AX foci after exposure to I-123, high- and low-LET radiation.

Methods: Human T-lymphoma Jurkat cells were either exposed to I-123-iododeoxyuridine (I-123-UdR; 2-200 kBq per 10E6 cells) for 20 h or irradiated with different doses of low-LET Cs-137 γ-rays respectively high-LET Am-241 α-particles. The γ-H2AX foci were quantified by measuring the mean signal intensity using flow cytometry and by counting the number of γ-H2AX foci microscopically by eye. Co-localization experiments were performed with the DNA-repair associated protein 53BP1 employing confocal microscopy.

Results: The mean numbers of γ-H2AX foci per cell showed a much more pronounced increase after exposure to I-123 when compared to γ- and α-irradiation. However, the mean intensity of γ-H2AX signals per cell nucleus, was very similar after I-123 and α-particle exposure. The individual γ-H2AX foci induced by I-123 resemble γ-H2AX foci induced by γ-rays and appear to be smaller, more distinct and/or less intense stained than those after α-irradiation. 53BP1 foci do not always co-localize with γ-H2AX foci.

Conclusions: The presumed complexity of the DNA-lesion caused by DNA-associated AEE is not reflected in the size and the intensity of γ-H2AX foci.

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