Calculation of dose deposition in 3D voxels by heavy ions and simulation of γ-H2AX experiments


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ABSTRACT
The biological response to high-LET radiation is different from low-LET radiation due to several factors, notably difference in energy deposition and formation of radiolytic species. Of particular importance in radiobiology is the formation of double-strand breaks (DSB), which can be detected by γ-H2AX foci experiments. These experiments have revealed important differences in the spatial distribution of DSB induced by low- and high-LET radiations [1,2]. To simulate γ-H2AX experiments, models based on amorphous track with radial dose are often combined with random walk chromosome models [3,4]. In this work, a new approach using the Monte-Carlo track structure code RITRACKS [5] and chromosome models have been used to simulate DSB formation. At first, RITRACKS have been used to simulate the irradiation of a cubic volume of 5 µm by 1) 450 H+ ions of 300 MeV (LET ∼0.3 keV/µm) and 2) by 1 56Fe26+ ion of 1 GeV/amu (LET ∼150 keV/µm). All energy deposition events are recorded to calculate dose in voxels of 20 µm. The dose voxels are distributed randomly and scattered uniformly within the volume irradiated by low-LET radiation. Many differences are found in the spatial distribution of dose voxels for the 56Fe26+ ion. The track structure can be distinguished, and voxels with very high dose are found in the region corresponding to the track "core". These high-dose voxels are not found in the low-LET irradiation simulation and indicate clustered energy deposition, which may be responsible for complex DSB. In the second step, assuming that DSB will be found only in voxels where energy is deposited by the radiation, the intersection points between voxels with dose > 0 and simulated chromosomes were obtained. The spatial distribution of the intersection points is similar to γ-H2AX foci experiments. These preliminary results suggest that combining stochastic track structure and chromosome models could be a good approach to understand radiation-induced DSB and chromosome aberrations.

REFERENCES