Astronauts' lymphocytes taken from post-flight blood samples often show increased levels of chromosome aberrations [1]. CA can be used for risk estimation [2], since specific aberrations are strongly correlated with some cancer types [3]. Furthermore, particular aberration categories can be regarded as biomarkers of the radiation quality [4,5]. Modeling approaches based on track-structure simulations can shed light on the mechanisms underlying CA induction, as well as provide estimates where experimental data are not available, typically at low doses.

In this work, a mechanistic model and a Monte Carlo code simulating radiation-induced chromosome aberrations in human lymphocytes will be presented. The current version of the model [6] can predict dose-response curves for the main aberration types (dicentrics, translocations, rings, complex exchanges and deletions) following irradiation with low- and high-LET radiation, as well as with mixed fields. The model is based on the assumption that clustered DNA breaks play a fundamental role in the process of aberration formation, and that only break free-ends in neighboring chromosome territories can interact and form exchanges. Such lesions are distributed within a 3-μm radius sphere representing a lymphocyte nucleus according to the radiation track structure. Interphase chromosome territories are explicitly simulated, that allows us to obtain a large number of different configurations in which each chromosome occupies a compact domain with volume proportional to its DNA content. In view of performing predictions down to low doses at the cGy level, background aberrations are included in the model. The very good agreement between model predictions and experimental data available in the literature provides a validation of the model both in terms of the adopted assumptions and in terms of the simulation techniques.

As an application in the field of radiation protection at low doses, estimates of Chronic Myeloid Leukemia risk will be shown, based on the calculated yields of translocations involving the BCR and ABL genes, which are considered as a major cause for CML [7]. Furthermore, the ratio of dicentrics to centric rings and that of complex to simple exchanges will be discussed as possible biomarkers of the radiation quality.

REFERENCES