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Radiobiological properties of accelerated ion beams along the Bragg curve

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Radiation quality is the determinant of the radiobiological effectiveness of ionising radiation since the molecular damage at the DNA level mainly responsible for observed cytogenetic effects mirrors the pattern of energy deposition by radiation tracks. It is known that high-LET radiations, whose energy loss in the traversed matter is described by the Bragg curve, are more effective than x- or gamma rays at causing acute effects. However, despite the growing use of accelerated ion beams for cancer treatment (e.g. hadrontherapy), substantial uncertainties surround the radiobiological consequences of exposure to charged particles as far as long-term and non-cancer effects are concerned, particularly for the consequences on normal tissue at the beam entrance or in the distal part of the spread-out Bragg peak (SOBP) in therapeutic set up. Moreover, although the relative biological effectiveness (RBE) of particle radiation has been traditionally related to the radiation linear energy transfer (LET), the latter parameter has consistently proven inadequate to interpret particle radiation effectiveness. Hence, a radiobiological characterization of the Bragg curve is needed by which “biological” Bragg curves can be derived. To this purpose, we have measured the induction of lethal and sub-lethal damage in human normal and cancer cells at various positions along pristine and modulated ion beams accelerated at the superconducting cyclotron of the LNS-INFN laboratory (Catania, Italy) and at the 3-MV TTT-3 Tandem accelerator of the Department of Physical Sciences, University of Naples Federico II. In particular, 62 MeV 1H and 62 MeV/n 12 C ions beams, both monoenergetic and with a SOBP have been used at LNS, while effects near ion track end have been studied at Naples facility for 12C, 16O and 19F ion beams of up to 20.3 MeV. The endpoints studied include cell killing assayed by clonogenic survival, chromosomal aberrations as detected by FISH painting and cellular premature senescence by beta-galactosidase activity. Our preliminary results support the idea that DNA damage and cellular response vary significantly along the ion path and that track structure ought to be taken into account to better model charged particle biological effectiveness.

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