

Radiobiological model for intraoperative radiotherapy with electrons

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In radiotherapy treatments, the Radiobiological Effectiveness of Radiation (RBE) is customary assumed to be proportional to the absorbed dose. We have shown in a recent publication [1], that induced damage at the molecular level in water, in terms of induced molecular dissociations, by a 6MV X-ray beam generated by a clinical LINAC accelerator, is always proportional to the induced ionisation and therefore to the absorbed dose (typically determined with ionization chambers). Identical irradiation of living cells in water showed that biological damage, in terms of early and late apoptosis and DNA damage, resulted to be also proportional to the absorbed dose, within the irradiated area, although residual cellular damage out of this area was also observed and assigned to reactive radical diffusion (see [1] for details). This result may be expected since photon interactions with molecules do not produce significant changes in the beam energy and high energy photoelectrons are generated within the whole irradiated area so their damaging effect is homogeneously distributed within the irradiated volume. However, the situation is completely different when the primary radiation beam is formed by charged particles (protons, heavier ions, electrons or positrons). These are gradually losing their energy by successive collisions with the molecular constituents of the target and therefore the interaction probabilities significantly change along the irradiation volume. In these conditions, proportionality between biological damage and absorbed dose is not expected. In this work we study the correlation between induced molecular and biological damages by a 6MeV electron beam generated by a LIAC clinical accelerator for intraoperative radiotherapy treatments in different irradiation conditions corresponding to the same absorbed dose. The molecular damage is evaluated in terms of induced molecular dissociations, via ionization, electronic and vibrational excitations and electron attachment, by using our Low Energy Particle Track Simulation (LEPTS) code [2]. The biological damage is evaluated via cell survival analysis for a constant 5 Gy dose in different beam spectral conditions.

REFERENCES

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