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Modeling the radiobiological effects of gold nanoparticles in proton therapy of glioblastomas

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Several studies show that the combination of high-Z nanoparticles and external radiotherapy leads to an increased radiation effect in tumoral cells without an increase of the patient dose.

However, it is not yet clear how the sequence of physical, chemical, and biological mechanisms contributes to the observed synergic effect. The objective of this work is to develop simulation tools that allow the analysis and interpretation of radiobiology studies with multifunctional nanoparticles (NPs). To do that, we will develop realistic simulations of the irradiation of monolayer human glioblastomas multiforme (GBM) cell cultures, taking into consideration different concentrations and cellular and subcellular distributions of the gold nanoparticles (AuNPs). The simulations will be implemented based on TOPAS [1] software more specifically the extension TOPASn-Bio [2] that includes models of the physical and chemical processes induced by radiation at the DNA scale.

Several incident beam configurations will be considered (X-rays, Co-60 source and proton beams). So far, simulations of Cobalt-60 irradiation mimicking the one used in the experiments at C2TN were performed.

Based on these simulations, the dose distributions at the subcellular scale were obtained for different AuNPs concentrations. The microdosimetric distributions in cells were used to predict cell survival fractions, using standard mathematical models of the biological effects of radiation as Local Effect Model (LEM) [3] and Microdosimetric Kinetic Model (MKM) [4].

The results obtained in the simulations were compared with the biological in vitro experimental results, and a good agreement between them was verified.

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[4] M. Cunha, et al., "NanOx, a new model to predict cell survival in the context of particle therapy", *Phys. Med. Biol.* 62 1248, 2017

[5] Christian P Karger and Peter Peschke, "RBE and related modeling in carbon-ion therapy", *Phys. Med. Biol.* 63 01TR02, 2018

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