AbstractID: 4978 Title: A Monte Carlo Based Tumor Model

Purpose: To develop a stochastic, radiobiological tumor model to conduct *in silico* simulations of cell proliferation and to quantitatively assess biological effects of delivered dose and different irradiation regimes.
Method and Materials: We developed a Monte Carlo model to perform computer simulations on initial tumor growth, progression of predetermined cell distributions and cellular response to irradiation following the linear-quadratic model. The single-cell-based configuration is composed of a volumetric grid lattice, with each grid site representing a capillary, clonogenic or normal tissue cell. Mean values for biological parameters such as cell cycle time and cell cycle phase dependent radiosensitivity were adopted from literature and sampled restrictively from Gaussian distributions for each cell. Additionally, angiogenesis, apoptosis and necrosis were implemented and random processes like cell displacement after proliferation and radiation-induced DNA damages were modeled using probability density functions.
Results: Simulations of initial tumor formation and ongoing proliferation illustrate a decelerated growth rate with increasing cell cycle times and hypoxia occurrence, whereas low thresholds for capillary stimulation through tumor angiogenesis factors lead to accelerated proliferation. Analyses of tumor response to different fractionation patterns show faster and stronger expression of necrosis after accelerated time-dose-patterns. A benchmark against published experimental data with human HNSCC-6 tumor cell lines demonstrates good quantitative agreement.
Conclusions: Our model is able to qualitatively predict basic radiobiological behavior and implicitly includes the ‘four Rs of radiotherapy’ as a result of the cellular approach. In order to apply the model to a specific tumor, it has to be tuned by including *in vivo* data and benchmarked against experiments. Given the adequate biological input parameters, good quantitative agreement can be achieved. This model could be enhanced to help predicting treatment response and bring us one step closer to biological optimization models.