Final Report Summary - MUSIC (Modeling and Simulation of Cancer Growth)

Although significant progress has been made in the last 50 years in the understanding, prevention, and treatment of cancer, 3.2 million people are diagnosed with cancer every year in Europe, leading to approximately 1.1 million deaths. Nowadays, the treatment of cancer is based on the so-called diagnostic paradigm. The shift from the traditional diagnostic paradigm to a predictive patient-specific one may lead to more effective therapies, and ultimately to lower morbidity and mortality. The MuSIC project proposed predictive models for cancer growth and new numerical algorithms that permit to obtain fast and accurate simulations based on patient-specific data.

The proposed models are based on partial differential equations emanating from the phase-field method. They account for the tumor and the vasculature that develops around it, which is essential for the tumor to grow beyond a harmless limited size. The effect of vital nutrients on tumor growth, as well as the impact of mechanical stresses on the cell cycle are considered. We developed different models, that operate at different scales (from almost cellular scale to tissue scale). These models will give important insight at different scales, at different stages of tumor growth and for several types of cancer.

We proposed new algorithms based on isogeometric analysis to perform patient-specific simulations that consider the anatomy and physiological information of the patient as extracted from medical images. As an important contribution we brought to the field of cancer modeling the expertise of high-performance computing and the possibility to do computations on patient-specific geometries. Our models also give a concrete example of how simulation and imaging can be combined to produce predictive models for tumor growth.

Our models of tumor growth and angiogenesis have provided insight into several scientific questions that remain open about the early stages of tumor growth. We also proposed models specifically dealing with brain and prostate tumors. The combined use of our brain and prostate models and numerical algorithms on a patient-specific basis may have significant impact in the clinic. In particular, the brain model suggests an explanation of why hypoxia in brain tumors leads to poor prognosis even if the proliferation of cancer cells is slower when the oxygen concentration is low. The prostate cancer model offers an explanation of why prostate cancer patients who also present benign prostatic hyperplasia can expect in general a better outcome.

Reported by

UNIVERSIDADE DA CORUNA
Spain

Subjects

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