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EVALUATING THE EFFECT OF TISSUE ANISOTROPY IN BRAIN TUMOR GROWTH USING A MECHANICALLY-COUPLED REACTION-DIFFUSION MODEL

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Summary: Glioblastoma (GBM), the most frequent malignant brain tumor in adults, is characterized by rapid growth and healthy tissue invasion. Long-term prognosis for GBM remains poor with median overall survival between 1 y to 2 y [1]. GBM presents with different growth phenotypes, ranging from invasive lesions without notable mass-effect to strongly displacing lesions that induce mechanical stresses and result in healthy-tissue deformation, midline shift or herniation. Biomechanical forces, such as those resulting from displacive tumour growth, shape the tumor environment and contribute to tumor progression [2]. We therefore expect that mechanical forces exerted by lesions on the brain parenchyma have implications on the biophysical level, and that they may affect treatment response and outcome.

Previously, we presented a mechanically–coupled reaction-diffusion model of brain tissue that computes tumor-induced strains based on local tumor cell concentration [3]. The framework simulates tumor evolution over time and across different brain regions using literature-based parameter estimates for tumor cell proliferation, as well as isotropic motility, and mechanical tissue properties. This model yielded realistic estimates of the mechanical impact of a growing tumor on intra-cranial pressure. However, comparison to imaging data showed that asymmetric shapes could not be reproduced by isotropic growth assumptions.

Here we present an extended version of the model that accounts for tissue anisotropy, based on MRI diffusion tensor imaging (MR-DTI), which is known to affect the mechanical behavior of brain tissue and the directionality of tumor cell migration. Tumors were seeded at multiple locations in a human MR-DTI brain atlas and the spatio-temporal evolution of tumor cell concentration and mechanical impact was simulated using the Finite-Element Method. We evaluate the impact of tissue anisotropy on the model's ability to reproduce features of real pathologies by comparing predicted lesions to publicly available GBM imaging datasets. We plan to use the model to characterize GBM growth phenotypes and study implications for treatment.

References

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