Data-Driven PDE Models of Tumor–Immune Dynamics in Tumor Micro-Environment

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Abstract

We develop and analyze a family of data-driven partial differential equation (PDE) models that resolve how spatial heterogeneity and tumor–immune interactions shape disease progression across three cancer types: breast cancer [1], colon cancer [2], and osteosarcoma [3]. Building on calibrated kinetic (ODE/QSP) descriptions of key cellular and cytokine networks, we extend the reaction terms to reaction–diffusion–advection PDE systems to capture localization, transport, and proximity-driven regulation inside the tumor microenvironment. In breast cancer, we use spatio-temporal data from the MMTV-PyMT mouse model to study how immune infiltration patterns and boundary influx influence evolving tumor architecture and immune depletion. In colon cancer, we investigate spatial immune organization through a PDE system coupled with tissue mechanics (domain deformation via elasticity), enabling in silico experiments on how microenvironmental structure alters competitive/cooperative signaling. For osteosarcoma, we incorporate biomechanical effects (e.g., porous-media style growth and stress) and immune-context stratification to quantify progression and treatment response across distinct immune phenotypes.

Across these case studies, we emphasize parameter identification from experimental/omics-derived cell abundance estimates, sensitivity/uncertainty analyses, and predictive simulations that support patient- or cluster-specific hypotheses for therapy design and optimization.

References

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