A Computational Model for Studying Cancer: Detecting Immediate Drivers for Malignant Tumor Progression

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Unlike homogeneous cellular bodies, cancer is best understood as a diverse community of heterogeneous cells that interacts through complex ecological interactions. Intratumor interactions, such as competition and cooperation, occur constantly within malignant tumors and are analogous to interactions between organisms in an ecosystem. Accounting for these interactions in conjunction with genetic mutations currently poses the greatest obstacle to effective cancer treatment. However, due to ongoing advances in single cell technology, genetic sequences and phenotypic parameters of individual cancer cells can now be captured precisely, providing researchers with access to unprecedented data. Utilizing the breakthroughs of single cell sequencing, a two-phase approach was created to effectively interpret single cell data collected while targeting both tumor heterogeneity and complex ecological interactions. Previously, to combat tumor heterogeneity, a computational model was constructed that employs statistical analysis through t-tests and ANOVA to effectively detect driver mutations for cancer development. In addition to refining this model, a mathematical approach was developed to analyze ecological interactions present within tumors. The approach employs ordinary differential equations to analyze the allometric relationship between measurable phenotypic factors of intratumor cells, enabling the quantification of ecological interactions among cell subpopulations. Ultimately, this two-phase approach can help dually identify intratumor cell populations most conducive to tumor growth and genetic mutations through which cooperative ecological relationships promote overall tumor progression, laying the foundation for personalized cancer intervention, such as gene editing.

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