Mathematically Modeling Tumorigenesis and Axons Regulation for Pancreatic Cancer

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During tumorigenesis and the development of pancreatic cancer, dynamic changes in the neuronal composition of the pancreas have been experimentally observed. Research has shown that axons play a role in influencing cancer progression. Depending on the type of axon, they can have either a pro-tumoral or anti-tumoral effect. A mathematical model of nerve-tumor interactions has been published, demonstrating that cancer-induced axonal plasticity contributes to transforming an initially protective neural environment into one that favors tumor growth. This previous work showed how important it could be to be able to predict the impact of sequential denervation at different times in pathological progression. However, the micro-environment of pancreatic cancer is highly complex, and many challenging research questions remain regarding the nervous system's impact on tumor progression. Recent studies have observed an interplay between axonogenesis, tumor growth, and changes in tumor tissue stiffness. The aim is to construct a new mathematical model of tumorigenesis and axon regulation at the tissue level, incorporating the effects of stiffness. The objective is to obtain results that will aid in formulating hypotheses to better understand the neurobiology of pancreatic cancer and, in the long term, contribute to strategies for controlling pancreatic cancer by impacting the immune system.

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