Mathematical Modeling of the role of platelets and immune system in Cancer Metastasis.

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Cancer metastasis is responsible for a large proportion of cancer-associated death. The metastatic process ensues when cancer cells detach from the primary tumor and infiltrate the bloodstream and lymphatic vessels, evolving into circulating tumor cells (CTCs).
Emerging evidence suggests that CD8+ T cells possess the capability to identify and eradicate CTCs. However, this anti-tumoral mechanism remains largely unexplored. Recent investigations have underscored the role of activated platelets in promoting CTC extravasation, thereby augmenting metastatic advancement. In this presentation, we introduce a novel mathematical model describing the interplay between the primary tumor, CTCs, activated platelets, and CD8+ T cells in metastasis.
Stability analysis demonstrates that during the early dissemination of CTCs, effector CD8+ T cells can maintain or constrain the burden of secondary metastatic tumors at a low equilibrium state. Conversely, in instances of late CTC dissemination, CD8+ T cells are less likely to impede the expansion of secondary tumors. Additionally, global sensitivity analysis underscores the substantial impact of primary tumor growth rate, intravascular CTC proliferation, and CD8+ T cell proliferation on the quantity of secondary tumor cells. Furthermore, model simulations indicate that escalated CTC proliferation significantly fosters tumor metastasis and further demonstrate that heightened levels of activated platelets on CTCs correspond to an increased likelihood of secondary tumor establishment.