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Mathematical Models for Tumor-Immune Interactions and Their Applications

In this talk we will describe our recent mathematical models of the interaction between the immune system and cancer focusing on two specific components: TGF-β and B7-H1. TGF-β is an immunoregulatory protein that contributes to inadequate antitumor immune responses in cancer patients. Recent experimental data suggests that TGF-β inhibition alone, provides few clinical benefits, yet it can significantly amplify the anti-tumor immune response when combined with a tumor vaccine. We develop a mathematical model in order to gain insight into the cooperative interaction between anti- TGF-β and vaccine treatments. We show that our model is capable of capturing the observed experimental results, and hence can be potentially used in designing future experiments involving this approach to immunotherapy.

The second part of the talk will be devoted to the surface protein B7-H1. B7-H1 is found on carcinomas of the lung, ovary, colon, and melanomas but not on most normal tissues. B7-H1 has been experimentally determined to be an antiapoptotic receptor on cancer cells, where B7-H1-positive cancer cells have been shown to be immune resistant, and *in vitro* experiments and mouse models have shown that B7-H1-negative tumor cells are significantly more susceptible to being repressed by the immune system. We derive a new mathematical model for studying the interaction between cytotoxic T cells and tumor cells as affected by B7-H1. By integrating experimental data into the model, we isolate the parameters that control the dynamics and obtain insights on the mechanisms that control apoptosis. This is a joint work with Amanda Galante, Shelby Wilson, and Koji Tamada.