

Mathematical Model of B7-H1-positive Tumor and Cytotoxic T Cell Interaction

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A costimulatory molecule B7-H1, which is ever-present in carcinomas of the lung, ovary and colon and in melanomas but not in most normal tissues, has been experimentally determined to be an anti-apoptotic receptor on cancer cells. B7-H1-positive cancer cells have been shown to be immune resistant, whereas in vitro experiments and mouse models have shown B7-H1-negative tumor cells are significantly more susceptible to being repressed by the immune system. B7-H1 is believed to form a molecular shield which inhibits production and induces apoptosis of cytotoxic T cells. A better understanding of this relationship may allow medical researchers to develop a cancer treatment specifically targeting this molecular shield, allowing the immune system to more effectively repress a tumor. In this work, using experimental data and “first principles” arguments regarding the immune system, tumor growth, and mechanisms of apoptosis, we derive and simulate a mathematical model in order to elucidate the immune system, surface protein B7-H1, and tumor cell interaction dynamics. This is joint work with Doron Levy¹ and Koji Tamada².

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