Developing and Utilizing a Whole-Cell Mathematical Model of IL-6 in Triple-Negative Breast Cancer: Predicting Cell Behavior and Targeted Anti-Tumorigenic Drug Interactions

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Breast Cancer (BC) is the most commonly diagnosed cancer and is a significant cause of death worldwide, affecting around 2.3 million women annually. Triple-negative breast cancer (TNBC) is a highly aggressive type of breast cancer characterized by the absence of three molecular markers of BC. TNBC represents a therapeutically challenging disease due to the absence of these clinically reliable biomarkers and treatment targets. Hence, there is an urgent need to understand the biological mechanisms of TNBC to identify novel therapeutic targets for addressing TNBC. The Interleukin (IL)-6 cytokine is a key enhancer and regulator of TNBC. Targeting the pathway of IL-6 in the cancer to overcome its critical influence on tumor growth is a new and promising strategy for cancer treatment. However, the exact nature of the effect of IL-6 remains poorly understood, and consequently, new models are needed. Here, a mathematical model of the signal transduction pathway of IL-6 in TNBC was developed; the model is a system of 48 ordinary differential equations. The IL-6 model was used in a sensitivity analysis to identify potential drug targets in the pathway for regulating the expression of IL-6. We further developed a model-based approach for virtual drug-target screening to evaluate the interaction between the potential drugs and targets on the secretion of IL-6. The model developed is the first-ever complete model of IL-6 in breast cancer. Research will be able to utilize this model to predict cell behavior and quantify the expression dynamics of IL-6. As well as to be used to predict the effectiveness of anti-tumorigenic drugs on the regulation of IL-6. This model may help researchers find novel therapeutics for targeting TNBC and provides a direction for drug-target selection.

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Second Award of $2,000