Lessons from an integrated model of prostate to bone metastasis

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008 Kemeny, 4:00PM
3:30 pm, 300 Kemeny

Abstract

Bone metastasis is common in prostate cancer progression. In bone, prostate cancer cells derive factors necessary for progression by manipulating bone forming osteoblasts and bone resorbing osteoclasts, resulting in areas of excessive osteogenesis and osteolysis, respectively. We developed an agent-based mathematical model where the interactions between key cell types and their role on the evolutionary dynamics of the tumor can be studied. The dialogue between the mathematical model and biological experiments have expanded our understanding of the process of prostate to bone metastasis. We have generated a series of new insights on posible future treatments such as the inhibition of transforming growth factor beta (TGFBeta); a key factor in the progression of bone metastases. Therapeutic inhibition of TGFBeta however, presents a dilemma since it can have differential effects on various cell types in the tumor-bone microenvironment. We will discuss the many challenges of using mathematical and in vivo models to test the impact of TGFBeta inhibition on prostate to bone metastases.

This talk should be accessible to graduate students.