May 31st, 10:30 AM - 11:00 AM

Serum PSA vs. Tumor Burden: Connecting the Dots with a Model of Angiogenesis

Johnna Barnaby  
*Florida State University, jpb14f@my.fsu.edu*

Harsh Jain  
*Florida State University*

Follow this and additional works at: https://scholarscompass.vcu.edu/bamm

Part of the [Life Sciences Commons](https://scholarscompass.vcu.edu/bamm/), [Medicine and Health Sciences Commons](https://scholarscompass.vcu.edu/bamm/), and the [Physical Sciences and Mathematics Commons](https://scholarscompass.vcu.edu/bamm/)

https://scholarscompass.vcu.edu/bamm/2018/thursday/1

This Event is brought to you for free and open access by the Dept. of Mathematics and Applied Mathematics at VCU Scholars Compass. It has been accepted for inclusion in Biology and Medicine Through Mathematics Conference by an authorized administrator of VCU Scholars Compass. For more information, please contact [libcompass@vcu.edu](mailto:libcompass@vcu.edu).
Prostate-specific antigen (PSA) is used as a diagnostic tool for determining prostate cancer tumor burden. However, clinical studies have shown that it is a poor biomarker that has led to little mortality benefit. To improve its prognostic potential, we need to better understand how PSA leaks out of the tumor and into the serum. In order to do this, we need to consider changes in vascular morphology both in growing tumors as well as in those under treatment. We develop a mathematical model of angiogenesis in relation to tumor growth that incorporates angiogenesis in the context of prostate cancer growth and PSA dynamics. This model is able to replicate a variety of tumor growth dynamics for a given PSA time course profile. Using mouse xenograft data we validate our model, and can use this model to show that for given patient PSA data we may achieve varying tumor growth profiles. Coupled with additional data on vessel morphology, our model can also explain why even as PSA levels decrease, a patient may have poor treatment outcomes.