Profiling immune cell trafficking within the prostate tumor microenvironment

Keith Rennier[1], Dengfeng Cao[2], William Shin[1], Ekaterina Esaulova[2], Max Artyomov[2], Robert Schreiber[2], Brian Zabel[3], **Russell Pachynski[1]**

[1]Division of Oncology, Department of Medicine; [2]Department of Pathology and Immunology, Washington University School of Medicine, St Louis MO; [3]Palo Alto Veterans Institute for Research, Palo Alto, USA

The goal of our proposed studies is to characterize in detail the tumor and immune microenvironments in both normal and malignant prostate tissues; and, further, to see if we can identify particular chemokine/chemoattractant profiles that may correlate with an "inflamed" phenotype. Our third aim focuses on utilization of mouse tumor models to test modulation of key chemokines found to be potentially important in an *in vivo* setting. Our initial studies proposed utilizing MRI-guided fusion biopsies of both normal and lesional (ie likely malignant) tissue. These tissue samples would be used to create single cell suspensions then used to interrogate infiltrating immune subsets as well as characteristics of the tumor epithelial/stromal components. To date, we have worked on tissue acquisition and processing protocols. We have evaluated various methods of cell isolation, and are investigating optimal tumor cell markers to identify malignant samples *ex vivo*. We have identified EpCAM as a reasonable surface tumor marker and are refining our dead cell removal protocols as well. Given the relatively low viable cell yield of our MRI-guided biopsies, we have been focused on collecting larger amounts of tissue from fresh prostatectomy samples, and continue to evaluate these. Ongoing efforts are focused on CyTOF, scRNAseq, and multiplexed IF modalities to evaluate collected prostate tissues. Our mouse models have now produced very promising preliminary data showing the impact of the innate leukocyte chemoattractant chemerin expression in the TRAMP tumor model – supporting our data in melanoma and breast cancer, and supporting our hypothesis that chemerin acts as a tumor suppressor gene in prostate cancer. Additional ongoing efforts are focused on therapeutic translation.

Conflict of Interest: The authors declare no relevant conflicts of interest.
Funding Sources: Prostate Cancer Foundation YIA