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Notable and Newsworthy



Dr. David Jarrard served as Faculty Presenter at the 2021 Prostate Cancer Patient Conference. This year's conference was held virtually. You can view his presentation, "Treating Metastatic Prostate Cancer" here: <https://www.youtube.com/watch?v=cC5yP3bQFjg>

Dr. Vinaya Bhatia has accepted the position of Director of Medical Student Programs in the Department. She will be directing all aspects of medical student teaching in the Department of Urology.

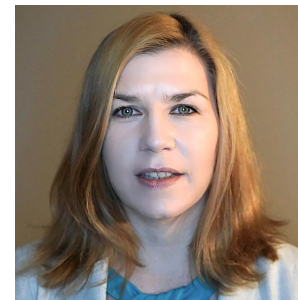


Dr. Teresa Liu, PhD, Scientist I and K01 Scholar in Dr. William Ricke's lab, has been awarded a UAB Nathan Schock Pilot/Feasibility Grant Award for her "*Mitochondrial Dysfunction Leads to Dysregulation of the Krebs Cycle in BPH/LUTS*".

UW Scientist Explores Novel Target for Treatment of LUTS

More than half of men over the age of 50 experience lower urinary tract symptoms (LUTS) secondary to benign prostatic hyperplasia (BPH). While LUTS is a common, benign condition, it reduces the quality of life for millions of men as they age.

UW Department of Urology Scientist, Petra Popovics, PhD, hopes to one day provide a new approach for treating the condition by exploring the role of a pro-inflammatory protein called osteopontin in the development of LUTS. When she began her research, "no one had looked at the connection between osteopontin and benign prostatic diseases before." This fact alone makes her research potentially groundbreaking.



Dr. Popovics designed her project after she observed that osteopontin is highly elevated when inflammation and fibrosis occur in the prostate. This observation, coupled with the fact that an increase in inflammatory cells is almost always found in the prostate in patients with LUTS, led her to several key "what if" questions:

What happens if we take osteopontin out of the system? Do we get less inflammation in the prostate and less fibrosis? Would it be worth targeting osteopontin further down the line in the treatment of LUTS?

To begin to tackle these questions, Dr. Popovics hypothesized that osteopontin levels induced by inflammation stimulate both prostatic fibrosis and LUTS. She is using the NIH K01 Research Scientist Development Award she received earlier this year to research this hypothesis.

An early experiment has already yielded striking results. Using an osteopontin “knockout mouse” model (mice that were missing osteopontin throughout the body), Dr. Popovics initiated prostatic inflammation via bacterial infection in both knockout mice and in control mice with osteopontin present. Within seven days, both the control and knockout mice showed a substantial increase in inflammation and fibrosis. Two months later, the inflammation was sustained and there was a “huge fibrosis” in most of the mice with osteopontin. Conversely, in most of the mice that were missing osteopontin, the inflammation was healed, and the fibrosis disappeared.

“I didn’t expect the fibrosis to be cleared out from the prostate – it was really fascinating to see. This result gives hope that we can not only stop the process, but we can potentially reverse it.”

Increased inflammation in the prostate caused by hormonal imbalance is believed to play a major role in the development of BPH and LUTS. Thus, Dr. Popovics is next looking at what happens if we remove osteopontin from the system when inflammation is initiated by hormonal imbalance.

Ultimately, she hopes to “develop better tools and therapies, and improve the quality of life for the millions of patients who suffer from BPH and LUTS.”

In addition to her research on osteopontin, Dr. Popovics, a member of UW’s NIH U54 O’Brien Center for Benign Urologic Research, is leading efforts to establish a tissue repository for samples from patients who have undergone prostatic surgeries to relieve symptoms. This resource will “help discover urine fibrotic markers to identify patients with significant prostatic fibrosis,” and help establish “personalized treatments for male lower urinary tract symptoms.”



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