

STONES

[Pathophysiology and Treatment of Enteric Hyperoxaluria](#)

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Enteric hyperoxaluria is a distinct entity that can occur as a result of a diverse set of gastrointestinal disorders that promote fat malabsorption. This, in turn, leads to excess absorption of dietary oxalate and increased urinary oxalate excretion. Hyperoxaluria increases the risk of kidney stones and, in more severe cases, CKD and even kidney failure. The prevalence of enteric hyperoxaluria has increased over recent decades, largely because of the increased use of malabsorptive bariatric surgical procedures for medically complicated obesity. This systematic review of enteric hyperoxaluria was completed as part of a Kidney Health Initiative-sponsored project to describe enteric hyperoxaluria pathophysiology, causes, outcomes, and therapies. Current therapeutic options are limited to correcting the underlying gastrointestinal disorder, intensive dietary modifications, and use of calcium salts to bind oxalate in the gut. Evidence for the effect of these treatments on clinically significant outcomes, including kidney stone events or CKD, is currently lacking.

[Tri-modality cavitation mapping in shock wave lithotripsy](#)

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Shock wave lithotripsy (SWL) has been widely used for non-invasive treatment of kidney stones. Cavitation plays an important role in stone fragmentation, yet it may also contribute to renal injury during SWL. It is therefore crucial to determine the spatiotemporal distributions of cavitation activities to maximize stone fragmentation while minimizing tissue injury. Traditional cavitation detection methods include high-speed optical imaging, active cavitation mapping (ACM), and passive cavitation mapping (PCM). While each of the three methods provides unique

information about the dynamics of the bubbles, PCM has most practical applications in biological tissues. To image the dynamics of cavitation bubble collapse, we previously developed a sliding-window PCM (SW-PCM) method to identify each bubble collapse with high temporal and spatial resolution. In this work, to further validate and optimize the SW-PCM method, we have developed tri-modality cavitation imaging that includes three-dimensional high-speed optical imaging, ACM, and PCM seamlessly integrated in a single system. Using the tri-modality system, we imaged and analyzed laser-induced single cavitation bubbles in both free field and constricted space and shock wave-induced cavitation clusters. Collectively, our results have demonstrated the high reliability and spatial-temporal accuracy of the SW-PCM approach, which paves the way for the future in vivo applications on large animals and humans in SWL.

PROSTATE

[E-cadherin expression is inversely correlated with aging and inflammation in the prostate](#)

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Benign prostatic hyperplasia (BPH) is a prostatic disease that is significantly associated with aging. However, it is not well understood how aging contributes to BPH pathogenesis. Several factors associated with an increased risk of BPH are also associated with increasing age, including chronic inflammation and declining epithelial barrier function. Thus, this study explored the potential associations between aging, loss of adherens junction protein E-cadherin and the presence of inflammatory mediators in prostate tissue specimens from healthy young donor and BPH patients. E-cadherin was decreased in aged NAP tissues and in BPH compared to young donor tissue. E-cadherin was inversely correlated with age and infiltration of inflammatory cells in NAP compared to young healthy donor prostate. Stromal COX-2 was positively

correlated with age and inflammation. E-cadherin was further down-regulated in BPH, while COX-2 H-Scores were not significantly altered in BPH compared to NAP. These findings suggest that aging is associated with down-regulation of E-cadherin and up-regulation of stromal COX-2 immunostaining in the prostate. E-cadherin immunostaining was inversely associated with age and inflammation, while stromal COX-2 immunostaining was positively associated with age and inflammation in the prostate. These findings suggest that the prostate epithelial barrier is altered and inflammation is increased with age in the prostate. These changes are further exacerbated in BPH, and may be involved in BPH pathogenesis.

[Claudin-1 down-regulation in the prostate is associated with aging and increased infiltration of inflammatory cells in BPH](#)

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Benign prostatic hyperplasia (BPH) is an age-related disease that is frequently associated with chronic prostatic inflammation. In previous studies, we detected the presence of PSA protein in the stroma of BPH nodules and down-regulation of junction proteins E-cadherin and claudin-1. Transmission electron microscopy (TEM) imaging showed a decrease in tight junctions suggesting the luminal epithelial barrier in BPH tissues may be compromised. Recent in vitro studies showed that stimulation of benign prostate epithelial cell lines with TGF- β 1 induced a decrease in claudin-1 expression suggesting that inflammation might be associated with alterations in the prostate epithelial barrier. This study explored the potential associations between aging and loss of junction proteins and the presence of inflammatory cells in prostate tissue specimens from young healthy donors and aged BPH patients. Claudin-1 immunostaining was inversely associated with increasing age, and inflammation in prostate specimens. B-

cell infiltration increased with age and BPH was associated with an increased infiltration of T-cells and macrophages compared to NAP. These findings suggest that aging is associated with down-regulation of claudin-1 and claudin-1 is further decreased in BPH. Claudin-1

down-regulation was associated with increased infiltration of inflammatory cells in both NAP and BPH tissues. Claudin-1 down-regulation in the aging prostate could contribute to increased prostatic inflammation, subsequently contributing to BPH pathogenesis.

- Jennifer Allmaras, MPH, 4/22/2021

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