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Author Reply

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As our understanding of the biological systems that affect bladder function evolves, our model that lower urinary tract symptoms (LUTS) are simply a result of biomechanical obstructive forces is being challenged. Our study identifies the possibility that poor sleep may predispose men for worse LUTS. The commenter has provided a very interesting discussion on recent literature regarding the circadian hormone melatonin and its relationship with nocturia. The hypothesis is that complex interactions between melatonin levels and other hormones may result in more nighttime urine production, and as a result may exacerbate LUTS.

Another potential mechanism may include the dampening effect of melatonin on bladder contractility. Recent animal model studies have identified that melatonin receptors in the bladder can reduce the contractile response elicited by muscarinic agonists or electrical field stimulation.[1] It is tempting to speculate that as melatonin levels decrease with increasing age, loss of melatonin's protective function on bladder contractility exacerbates the mechanical obstruction that results from benign prostatic hyperplasia and the increase in urine output resulting from altered hormone signaling.

In 1995, Garfinkel and colleagues published a landmark study in *Lancet* demonstrating the efficacy of melatonin to improve sleep quality in elderly subjects.[2] We support this idea that melatonin should be investigated as a potential therapeutic for LUTS. Melatonin is a

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promising link that may explain the complex relationship between impaired sleep quality as a driving force of LUTS.

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