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Stress urinary incontinence is caused predominantly by urethral support failure

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To the Editors:

In his recent Special Contribution [1], Dr. Bergström states the opinion that “urethral support failure is the predominant factor for SUI” (stress urinary incontinence), consistent with the ‘Urethral Hanging Theory’ (UHT) [1]. Proving or rejecting a theory about the causes of stress incontinence requires comparing data from women with SUI to data from *normal asymptomatic* women. Dr. Bergström specifically attempts to rebut the finding of just such a study in which we showed that impaired urethral closure was the primary causal factor associated with stress incontinence, with urethral support being secondary [2]. Because his article contains several misinterpretations of our work, we wish to correct the record. In so doing, we do not dispute that urethral support plays a role in stress incontinence—only that it is not the primary factor.

First, our finding that urethral closure pressure is the predominant factor causing stress incontinence is based on strong evidence from the large prospective ROSE Study [3]. This case control study compared over 105 women with SUI to 108 asymptomatic controls of similar age, race, and parity. We reported on measurements of 16 potential causal factors of stress incontinence, including several relating to urethral support. The results showed that urethral closure pressure was 42% lower in stress incontinent women, predicting 50% of SUI in logistic regression analysis. At 50%, no one of the many other factors alone could predict more. The most predictive of the urethral support measures explained only 16%. The primary role of reduced urethral closure pressure was confirmed in a separate independent population-based study [3]. These two papers contributed to a major reversal in our previous beliefs that urethral support was the primary factor as suggested in anatomical articles published in 1988 [4] and 1994 as the Hammock Hypothesis [5].

Second, in attempting to counter our findings, the author states: “If Zacharin and the UHT are correct, and urethral support failure is the predominant factor for SUI, how can recent evidence show that urethral function failure is the predominant factor? The answer to this

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question is that the ROSE study was inaccurately designed. The authors measured several parameters but had no valid SUI biomechanical model to test. Without a correct model, it is impossible to know what to measure and how to interpret results." This statement does not give the model in question its due. Our biomechanical model was published in 1994, has been cited 651 times, and is not substantially dissimilar to the UHT. The ROSE study was actually undertaken to prove this hypothesis and determine, mechanistically, what aspect of support was associated with stress incontinence.

When our analysis indicated that it was urethral closure, not support, that was the dominant factor, we were surprised. So we took the additional step of conducting a secondary analysis considering all visible aspects of urethral mobility on ultrasound [6] to see if our measurements had missed something. Experts with published experience evaluating support and stress incontinence evaluated ultrasound recordings of women with and without SUI while blinded to their continence status. Based on the movements they saw during cough, they rendered their opinion of whether they thought that person had stress incontinence.

Third, Dr. Bergström misinterprets this study as a "study by an expert panel, regarding the urethrovesical mobility (point Aa) during coughing." That is not what was done. As the methods section from the paper clearly states: "evaluators were asked to give their opinion on whether or not each subject had SUI. No specific instructions were given to direct the evaluators in their observation of the motion on the ultrasound." The experts were right 57% of the time—only 7% better than chance. Our instructions were specifically stated so the experts had complete freedom to identify any movement patterns that were associated with stress incontinence unencumbered by any metric. Since the experts could use all visible movements, if support was a major factor, it would have been noticed and success with identification would be high.

Fourth, the ultrasound study that Dr. Bergström cites as providing evidence for urethral support being the primary factor over urethral function in stress incontinence had a critical limitation, as its authors acknowledged [7]: it did not have an asymptomatic control group. Instead, it was based on "305 women complaining of symptoms of prolapse or lower urinary tract dysfunction" referred for evaluation. It compared those with stress incontinence to those without. Presumably, most women without stress incontinence referred for evaluation would have had urge incontinence. This is relevant, because data show that Caucasian women with urge incontinence have the same reduced urethral closure pressure as women with stress incontinence—both with mean closure pressures of 45 cm H_2O [3]. Therefore it is not surprising, given that study design, that a comparison between the two groups did not find a difference in urethral function. What that study does not provide is data on asymptomatic continent women. Those data are needed before any conclusions can be drawn about the role of urethral support in stress incontinence.

We have never, and do not now, suggest that urethral support plays no role in SUI; hard data show that it is just not the *primary* factor.

There is value in testing hypotheses with properly chosen controls and being open to unexpected findings. Surprises in science are paradigm-shifting, although they may seem

unwelcome. This fact was vividly expressed by Huxley: "The great tragedy of Science—the slaying of a beautiful hypothesis by an ugly fact" [8].

References

1. Bergstrom BS (2022) Stress urinary incontinence is caused predominantly by urethral support failure. Int Urogynecol J 33(3):523–530. doi:10.1007/s00192-021-05024-1 [PubMed: 35064789]
2. DeLancey JO, Trowbridge ER, Miller JM, Morgan DM, Guire K, Fenner DE, Weadock WJ, Ashton-Miller JA (2008) Stress urinary incontinence: relative importance of urethral support and urethral closure pressure. J Urol 179(6):2286–2290; discussion 2290. doi:10.1016/j.juro.2008.01.098 [PubMed: 18423707]
3. DeLancey JO, Fenner DE, Guire K, Patel DA, Howard D, Miller JM (2010) Differences in continence system between community-dwelling black and white women with and without urinary incontinence in the EPI study. Am J Obstet Gynecol 202(6):584 e581–584 e512. doi:10.1016/j.ajog.2010.04.027 [PubMed: 20510959]
4. DeLancey JO (1988) Structural aspects of the extrinsic continence mechanism. Obstet Gynecol 72(3 Pt 1):296–301 [PubMed: 3405547]
5. DeLancey JO (1994) Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. Am J Obstet Gynecol 170(6):1713–1720; discussion 1720-1713. doi:10.1016/s0002-9378(94)70346-9 [PubMed: 8203431]
6. Lewicky-Gaupp C, Blaivas J, Clark A, McGuire EJ, Schaer G, Tumbarello J, Tunn R, DeLancey JO (2009) "The cough game": are there characteristic urethrovesical movement patterns associated with stress incontinence? Int Urogynecol J Pelvic Floor Dysfunct 20(2):171–175. doi:10.1007/s00192-008-0738-0 [PubMed: 18850057]
7. Pirpiris A, Shek KL, Dietz HP (2010) Urethral mobility and urinary incontinence. Ultrasound Obstet Gynecol 36(4):507–511. doi:10.1002/uog.7658 [PubMed: 20503229]
8. Huxley TH (1870) Biogenisis and Abiogenisis. In: Collected Essays, Vol 8. Cambridge University Press, Cambridge, UK.