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GENETICS

POP goes the smooth muscle contractile protein

Pelvic organ prolapse (POP) occurs as a result of lost support and suspension that is normally provided by ligaments, endopelvic fascia and the levator ani muscle. Dysfunction of the smooth muscle component of the endopelvic fascia can have a marked effect on vaginal attachment to the pelvic side wall and the support provided by the pelvic floor, leading to POP. The molecular mechanisms underlying these pathologic changes have been shown to include altered expression of *MYH11* (smooth muscle myosin heavy chain) and *CALD1* (caldesmon); however, the role of other genes encoding smooth muscle contractile proteins, such as *ACTG2* (smooth muscle γ-actin) and *TPM1* (tropomyosin), and the influence of ovarian hormone changes, such as menopause, on smooth muscle content in POP, have not been studied. Bortolini *et al.* performed an observational case–control study to examine the differences in mRNA expression of *MYH11*, *CALD1*, *ACTG2* and *TPM1* between women with advanced POP and healthy controls, and the effects of menopausal status.

Biopsy samples of the anterior vaginal wall were taken from 55 women during total hysterectomy for stage ≥3 POP (cases) or benign conditions (controls).

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Of these women, 37 were premenopausal (23 cases and 14 controls) and 18 were postmenopausal (13 cases and 5 controls). Gene expression was assessed using real-time PCR.

In the premenopausal women, only *MHY11* expression was significantly downregulated in patients with POP compared to controls (fivefold decrease, $P = 0.0002$). In the analysis of postmenopausal women, *CALD1* expression was sixfold higher in cases compared to controls ($P = 0.03$), and *ACTG2* expression was increased sixfold (although this difference was not quite statistically significant). When the expression profiles of the premenopausal and postmenopausal controls were compared, all four of the study genes were found to be decreased after the menopause, significantly so in the cases of *CALD1*, *ACTG2* and *TPM1*.

The authors conclude that alterations in the expression of *MHY11* and *CALD1* are associated with POP, and decreases in *CALD1*, *ACTG2* and *TPM1* expression are observed following menopause. These changes are likely to contribute to smooth muscle dysfunction, leading to a loss of vaginal support and, subsequently, POP.

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Original article Bortolini, A. T. *et al.* Expression of genes encoding smooth muscle contractile proteins in vaginal tissue of women with and without pelvic organ prolapse. *Neurourol. Urodynam.* doi:10.1002/nau.21175