

EDITORIAL

Menopausal night sweats: more evidence for long-term repercussions of childhood abuse on women's health

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There is now substantial evidence that abuse in childhood carries long-lasting adverse effects on adult health, including increased risk for heart disease, cancer, chronic lung disease, liver disease, diabetes, and arthritis,^{1–4} especially in affected women.^{5–7} Moreover, a higher incidence of “women’s conditions” such as chronic pelvic pain, eating disorders leading to obesity, migraines, irritable bowel, and depression, as well as intimate partner victimization also exist in this female population.⁸ The rates of suicide, alcohol and drug abuse, and psychiatric admissions are also higher compared with women without this history.⁹ To add to the burden, the greater likelihood of exposure to adult sexual violence in child abuse survivors also puts them at higher risk for adverse pregnancy outcomes such as miscarriage and preterm labor.⁸

Despite the important attention to pregnancy outcomes, other reproductive health sequelae have been less studied. In 2008, Thurston et al reported on the first study to evaluate whether vasomotor symptoms (VMS) were more frequent in women exposed to childhood abuse or neglect using data from the Pittsburg cohort of the SWAN Mental Health Study.¹⁰ The Study of Women’s Health Across the Nation (SWAN) is one of the largest and most ethnically diverse longitudinal studies of the menopause experience. Beginning in 1996, SWAN enrolled 3,302 midlife women still having periods across five racial/ethnic groups at five different sites, following them annually over the course of the menopausal transition for related symptoms as well as health behaviors, social and psychological factors, and health outcomes. By 2008, the SWAN data had revealed a wealth of information on associations between VMS and such moderating factors as smoking, obesity, impaired mood, depression, and anxiety¹¹—health problems previously linked to childhood maltreatment—thus setting the stage for interrogating the SWAN data on how VMS and childhood abuse might interact.

In the 2008 report, Thurston et al presented data from SWAN visit 8—a time when many of the carefully screened sample of 332 white and African American women had traversed the menopausal transition, to demonstrate that a history of child abuse or neglect was associated with greater odds of reporting both hot flashes and night sweats in age-adjusted models.¹⁰ Limitations acknowledged by the investigators included the small sample size, retrospective hot flash self-reports, and the lack of data on VMS intensity. A major strength of the study was the use of a multidimensional, validated questionnaire on childhood abuse, the Childhood Trauma Questionnaire (CTQ), that measures five different types of abuse or neglect (emotional, physical, sexual abuse; emotional, physical neglect). In the accompanying editorial, Pinkerton et al speculated that more precise studies using prospective hot flash diaries, monitoring devices to capture hot flash events, and other biomarkers to detect changes in the brain would better define the nature of long-term neurobiologic effects of childhood trauma and its relationship to menopause health and vasomotor symptoms.⁸

Since then, the knowledge base in this area has continued to grow and strengthen the case for long-term repercussions of childhood maltreatment on adult health and women’s health in particular.¹² For example, drawing again on various SWAN data sets (where 38% of the sample reported a history of childhood abuse or neglect using the CTQ), investigators demonstrated associations with midlife adiposity, metabolic syndrome, and subclinical cardiovascular disease.^{13–15} Similar to the findings in the 2008 SWAN report, the link between sexual abuse and more severe sleep problems and VMS was also demonstrated in the Seattle Midlife Women’s Health Study^{16,17} and in the multiethnic cohort of women aged 40 to 80 years enrolled in the Kaiser Permanente Northern California healthcare system.¹⁸ In both cases, however, VMS assessment relied on self-reports which have been shown to be heavily influenced by concurrent emotional mood¹⁹ and highly sensitive to research conditions.⁶

Taking advantage of an existing data set from the MsHEART study (a non-SWAN sample of healthy midlife volunteers) that used the CTQ to classify trauma along with novel ambulatory technologies to characterize subclinical cardiovascular disease and VMS,²⁰ Rebecca Thurston and colleague Mary Carson have now taken a deeper dive into

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understanding the link between the hot flash experience and childhood trauma. In this issue of *Menopause*, Carson and Thurston² present additional data on the rates and type of childhood abuse retrieved from 295 well-screened MsHEART participants aged 40 to 60, dichotomized on the basis of self-reported daily VMS in the last 3 months (yes or no). In the MsHEART study, participants underwent 3 days of prospective assessments for self-reported time-stamped VMS and sleep activity (e-diaries and actigraphy), coupled with 24 hours of sternal skin-conductance monitoring to assess hot flashes and night sweats—those that leave a physiologic fingerprint of both perceived and not perceived electrodermal activity. In this analysis, the investigators report that among women in the “yes” group (daily VMS at baseline), higher rates of physiologically detected night sweats during sleep were demonstrated in those meeting CTQ score cut points for physical or sexual abuse. After controlling for a range of possible confounds, the association with physical abuse was especially prominent among nonwhite participants. At the same time, to the authors’ surprise, no links to wake-time VMS physiologic recordings in the “yes” group were seen across any of the five childhood trauma subtypes. Based on these findings, Carson and Thurston conclude that childhood abuse is associated with more frequent physiologically assessed VMS during sleep, thus adding to the knowledge base about its long-term sequelae on midlife women’s health.

Although in need of replication, these findings are intriguing and, as typical of most novel findings, raise more questions than they answer. To their credit, the authors provide evidence-based rationale to explain much of their data. They argue that physical and sexual abuse are more likely to be recalled versus other forms of abuse (and therefore better captured on the CTQ), and that the higher rates of both child abuse and VMS documented in minority women could have explained the significant associations in the minority subsample. The authors go on to speculate that their failure to demonstrate an association between self-reported VMS diaries and child abuse history might be due to the use of more precise, prospective measures versus retrospective recall used in most prior work. But at the same time, the report is silent on findings from the “no” group—those who reported no daily VMS in the last 3 months—even though they underwent the same detailed VMS and sleep recordings. Indeed, the table of clinical characteristics presents those for the total sample without subdividing by VMS group, leaving the reader to speculate about the extent to which VMS were documented (if any) by physiologic recordings and differed from the symptomatic group. Given that some 43% of the “no” group reported a history of child abuse, it would be interesting to know more about any other differences in clinical or menopause characteristics in these asymptomatic women.

Perhaps more importantly, to help interpret the exclusive link to sleep-related night sweats, the authors point to emerging evidence that emotional trauma carries significant impacts on other aspects of nocturnal physiology beyond sleep, such

as heart rate variability, hinting at the idea that changes in VMS physiology during sleep in trauma sufferers may also be at play. They go on to speculate on how disturbances in the sympathetic nervous system and hypothalamic-pituitary-adrenal axis observed in survivors of childhood trauma might impinge on the pathophysiology of VMS, given the presumed role of these brain centers in VMS mechanisms. To what extent the thermoregulatory controls of VMS, which have yet to be fully elucidated, are magnified by nocturnal disturbances associated with childhood maltreatment in these same systems is unknown but would seem to be a new line of inquiry for the field. Such hypothetical speculation may not be unreasonable given the growing evidence that the kisspeptin-neurokinin B-dynorphin (KNDy) pathway plays a central role in the neurobiology of hot flashes²¹ beyond its role in GnRH regulation, as part of a larger system that receives input from higher centers involved in emotion and cognition. In any case, these findings add fuel to the fire in the search for vasomotor symptom triggers and provide an intriguing argument for more precise analysis of day/night changes in VMS characteristics. We may not yet be ready to use the menopause hot flash as a biomarker for interrogating the neurobiology of trauma, but it would seem that more attention to its assessment is warranted.

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