Published in final edited form as:

Am J Obstet Gynecol. 2013 April ; 208(4): 272.e1–272.e13. doi:10.1016/j.ajog.2012.12.034.

Lifetime Abuse Victimization and Risk of Uterine Leiomyomata in Black Women

Lauren A. Wise, Sc.D.¹, Julie R. Palmer, Sc.D.¹, and Lynn Rosenberg, Sc.D.¹

¹Slone Epidemiology Center, Boston University, Boston, MA, 02215

Abstract

Objective—Uterine leiomyomata (UL) are a major contributor to gynecologic morbidity and medical costs, and black women are disproportionately affected by the condition. Previous studies have linked UL to psychosocial stress, including child abuse. We assessed the association between lifetime abuse victimization and UL among 9,910 premenopausal women.

Study Design—Data were derived from the Black Women's Health Study, a prospective cohort study. In 2005, participants reported their experiences of physical and sexual abuse within each life stage (childhood, adolescence, adulthood). Biennial follow-up questionnaires from 2005 through 2011 ascertained new UL diagnoses. Rate ratios (RR) and 95% confidence intervals (CI) were estimated using Cox regression.

Results—There were 1,506 incident UL cases diagnosed by ultrasound or surgery. UL incidence was higher among women who reported child abuse, particularly sexual abuse. Relative to no abuse across the lifespan, RRs were 1.16 (CI, 1.02–1.33) for physical abuse only, 1.34 (CI, 1.09–1.66) for sexual abuse only, and 1.17 (CI, 0.99–1.39) for both physical and sexual abuse in childhood. RRs for 1–3 and 4 incidents of child sexual abuse were 1.29 (CI, 1.04–1.61) and 1.41 (CI, 1.07–1.85), respectively, while RRs for low, intermediate, and high frequencies of child physical abuse were 1.19, 1.04, and 1.23, respectively. The association was strongest for the highest category of child abuse severity (RR=1.57, CI, 1.19–2.07). No associations were found for teen or adult abuse.

Conclusions—In the present study, child sexual abuse was an independent risk factor for UL, supporting the hypothesis that childhood adversity increases UL risk.

Keywords

Uterine neoplasms; Abuse victimization; Prospective Studies; African American

© 2013 Mosby, Inc. All rights reserved.

Please address correspondence to: Lauren A. Wise, Slone Epidemiology Center, 1010 Commonwealth Avenue, Boston, MA, 02215; Telephone (617) 734-6006; Fax (617) 738-5119. lwise@bu.edu.

Disclosure Statement: The authors have no conflicts of interest to disclose.

Paper Presentation Information: These data were presented at the 25th Annual Meeting of the Society for Pediatric and Perinatal Epidemiology in Minneapolis, Minnesota, June 25–26, 2012; the 46th Annual Meeting of the Society for Epidemiologic Research, Minneapolis, Minnesota, June 27–30, 2012; and the OB/GYN Grand Rounds at the Mayo Clinic, Rochester, Minnesota, June 24, 2012.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

INTRODUCTION

Uterine leiomyomata (UL) are clinically recognized in 30% of reproductive-aged women^{1–3} and are the primary indication for hysterectomy in the United States.^{4,5} Black women have 2–3 times the UL incidence of white women,^{4,6–8} have earlier ages at diagnosis,^{6,8} and have more symptomatic disease at clinical presentation.^{6,8,9} Differences in the prevalence of known risk factors do not explain the health disparity.^{6,9–11} Although UL etiology is poorly understood, sex steroid hormones are thought to influence their development and growth.^{12,13}

Emerging epidemiologic research suggests that exposure to psychosocial stress influences UL risk.¹⁴ In a cross-sectional study,¹⁵ a positive association was found between UL prevalence and both the number of major life events and “stress intensity.”¹⁶ We reported a positive association between perceived racial discrimination and UL in the Black Women’s Health Study (BWHS), a prospective cohort study. UL incidence was positively associated with greater levels of “everyday racism” and lifetime occurrences of major discrimination, with weaker associations found among those with higher coping skills.¹⁷ A positive association between early-life abuse victimization and UL was found in the Nurses’ Health Study II.¹⁸ Results were attenuated among those reporting high childhood emotional support,¹⁸ which like our study,¹⁷ supports the hypothesis that increased social/emotional support may buffer the negative impact of stress on health.^{19,20}

Severe stress in early life has been associated with altered regulation of the hypothalamic-pituitary-adrenal (HPA) axis and autonomic stress response, which may persist into adulthood.^{21–25} Specifically, childhood sexual trauma^{26–30} and low social support³¹ have been linked to chronic HPA axis dysregulation. Stress can down-regulate the hypothalamic-pituitary support of ovarian hormones,^{32–34} but in some circumstances up-regulate adrenal progesterone.^{35–37} Studies of granulosa cell tissue culture also suggest that under certain conditions, ovarian steroid secretion increases rather than decreases.³⁸ Various growth factors, cytokines, and matrix metalloproteinases involved in UL growth are up-regulated by stress hormones.^{14,35,36} Stress can also adversely affect health-related behaviors, leading to physical inactivity, heavy alcohol consumption, and poor diet, all of which can increase UL risk.^{39–44}

To assess whether there is an individual or cumulative stress burden of abuse across the lifespan, we prospectively evaluated the association of abuse victimization across three life stages—childhood, adolescence, and adulthood—with UL incidence in the BWHS. We assessed abuse according to timing, type, and frequency, and we also evaluated whether coping skills buffered these associations.

MATERIAL AND METHODS

Study population

The Black Women’s Health Study is an ongoing prospective cohort study of 59,000 African-American women aged 21–69 at entry.⁴⁵ In 1995, *Essence* magazine subscribers were mailed an invitation to enroll in a long-term health study by completing a comprehensive self-administered baseline questionnaire. Biennially, participants complete follow-up questionnaires to update their exposure and medical information; cohort retention has exceeded 80% through 2011. Participants reside in more than 17 states, with the majority residing in New York, California, Illinois, Michigan, Georgia, and New Jersey. The institutional review board of Boston University Medical Center approved the study protocol.

Assessment of abuse victimization

On the 2005 BWHS follow-up questionnaire, participants were asked questions about abuse victimization across the lifespan, including exposure as a “child” (up to age 11), “teenager” (age 12–18), and “adult” (ages 19 and older). The nine-item abuse instrument was adapted from the Conflict Tactics Scale and the Pregnancy Abuse Assessment Screen.^{46,47} Response categories were “never,” “1–3 times,” or “4 times.” We defined physical abuse as any report of a perpetrator having “pushed, grabbed, or shoved me,” “threw something at me that could hurt me,” “kicked, bit, or punched me,” “hit me with something including hand or fist,” or “physically attacked me in some other way” at a frequency of 4 times, or either “choked or burned me” or “seriously harmed someone I loved” at any frequency. We defined sexual abuse as any report of a perpetrator having “exposed genitals against my will” 4 times or “been sexual with me against my will” at any frequency.

We created a physical abuse summary score variable by assigning 1 point for each report of a physical abuse item occurring 4 times, with the exception of “choked or burned” or “seriously harmed someone I loved,” where 1 point was assigned for reports that these occurred 1–3 times and 2 points for reports that these occurred 4 times, because these events were considered more severe. The resulting physical abuse severity score, which ranged from 0 to 9, was further categorized as low (score=1), intermediate (score=2) and high (score=3). We also created a summary variable for sexual abuse that classified 4 incidents separately from 1–3 incidents of sexual assault. Finally, within each life stage, we created a “severity” variable that defined “mild” abuse as one type of physical abuse that occurred 4 times; “moderate” abuse as sexual abuse 1–3 times and/or two forms of physical abuse occurring 4 times or a more severe form of physical abuse (burn or choke or seriously harm someone I love) occurring at any frequency; “severe” abuse as 3 or more types of physical abuse occurring 4 times and/or sexual abuse 4 times; and “very severe” abuse includes 3 or more types of physical abuse occurring 4 times and sexual abuse 4 times. These abuse definitions have been used in previous publications from the BWHS^{48–50} and other studies.^{51,52}

Assessment of uterine leiomyomata

On the 2005 questionnaire, women reported whether they had *ever* been diagnosed with “uterine fibroids,” the year of first diagnosis, and whether their diagnosis was confirmed by “ultrasound” and/or “surgery (e.g., hysterectomy),” listed as two separate questions. On the 2007, 2009, and 2011 questionnaires, women reported whether their diagnosis had occurred in the previous two years. Cases were classified as “surgically-confirmed” if they first reported confirmation by “surgery,” regardless of whether they also reported “ultrasound” confirmation. We included cases confirmed by surgery or ultrasound, because surgical cases represent only 10–30% of diagnosed cases.⁵³ Ultrasound has high sensitivity (99%) and specificity (91%) relative to histologic evidence.^{54,55}

Assessment of covariates

On the 1995 and biennial follow-up questionnaires, we collected data on reproductive and contraceptive history, anthropometric factors, lifestyle factors (smoking, alcohol, physical activity), geographic region of residence, socioeconomic correlates (education, marital status, occupation), medical conditions, and Pap smear frequency. In 2003, women reported their household income. In 2007, participants reported their recency of pelvic exam and pelvic ultrasound under two separate questions (never, <5, 5–9, 10 or more years ago). We collected retrospective data on early life factors, including nativity (1997), maternal age (2005), sibship size and birth order (2007), parental education (2009), home ownership in childhood (2009), and passive smoke exposure (1997). In 2005, participants completed an abbreviated (10-item) scale based on an instrument developed by Carver⁵⁶ designed to

assess: 1) active coping, 2) use of emotional support, 3) use of instrumental support, 4) positive reframing, and 5) acceptance. Higher scores on the coping scale indicated higher coping skills.

Validation studies

Uterine leiomyomata—We mailed supplemental questionnaires to a random sample of 248 cases regarding their initial date of diagnosis, method of confirmation, symptoms, and treatment, and asked for permission to review their medical records. We obtained medical records from 127 of the 128 women who granted permission and corroborated the self-report in 122 (96%). Among the 188 (76%) with supplemental data, 71% reported UL-related symptoms prior to diagnosis and 87% reported that their condition came to clinical attention because they sought treatment for symptoms or a tumor was palpable during a routine pelvic exam. There were no appreciable differences between cases who did and did not release medical records with respect to UL risk factors.⁵⁷

Abuse—We conducted a factor analysis of physical and sexual abuse items using an orthogonal rotation, which revealed two factors that confirmed the predetermined “physical” and “sexual” domains of abuse. Among 690 women who inadvertently returned a duplicate questionnaire in 2005, weighted kappa (κ) values indicated good reproducibility of responses to the abuse questions. In general, there was higher agreement in reporting of sexual abuse (child: $\kappa=0.68-0.78$; teen: $\kappa=0.61-0.66$; adult: $\kappa=0.51-0.59$) than physical abuse (child: $\kappa=0.48-0.60$; teen: $\kappa=0.47-0.59$; adult: $\kappa=0.35$ to 0.67), and child abuse was reported with greater reliability than teen or adult abuse. Cronbach alphas for the physical and sexual abuse items, respectively, were 0.80 and 0.82 in childhood, 0.81 and 0.72 in adolescence, and 0.84 and 0.63 in adulthood, indicating high internal consistency.

Restriction criteria

Of the 40,314 women who responded to both the 1997 and 2005 questionnaires, we excluded women who were postmenopausal ($N=14,824$) or had been diagnosed with UL before 2005 ($N=12,907$); women lost to follow-up after 2005 ($N=535$); cases without a year of diagnosis ($N=116$) or method of confirmation ($N=62$); and women with incomplete data on abuse ($N=1,534$) or other covariates ($N=426$), leaving 9,910 women for analysis. Those excluded had lower education (mean: 15.7 vs. 16.0 years) and were more likely to smoke (14% vs. 10%) than those included, but were similar with respect to age at menarche (mean: 12.4 vs. 12.4 years), parity (67% vs. 66%), age at first birth (mean: 22.7 vs. 23.4 years), and other UL risk factors. The incidence rate for UL among the women who did not complete the abuse instrument was 27.7 cases/1,000 person-years, a rate similar to that among the non-abused women (28.1 cases/1,000 person-years).

Data Analysis

We categorized abuse according to stage at first abuse, type, frequency, and severity (defined above). The reference category for all analyses was “no abuse across the lifespan.” Incident cases were women who reported a first diagnosis of UL confirmed by ultrasound or surgery. Person-years at risk were calculated from the start of follow-up (March 2005) until UL diagnosis, menopause, death, loss to follow-up, or the end of follow-up (March 2011), whichever came first. Cox regression models were used to estimate incidence rate ratios (RRs) and 95% confidence intervals (CI) for the associations of interest.

We constructed two multivariable models. The first model controlled for age (1-year intervals) and time period (2-year intervals) in addition to early life factors, including nativity (foreign-born, U.S.-born), maternal age (<20, 20–24, 25–29, 30–34, 35), birth order (first-born, later-born), sibship size (children), childhood passive smoke exposure (yes,

no), parental education (neither, at least 1 parent completed high school), home ownership in childhood (rented only, owned at some point), and age at menarche (years). The second model further controlled for potential mediators, including parity (births), age at first birth (years), years since last birth (<5, 5–9, 10–14, 15, 20), oral contraceptive (OC) use (never, ever), age at first OC use (years), body mass index (BMI; <20, 20–24, 25–29, 30–34, 35 kg/m²), smoking (current, past, never), current alcohol use (<1, 1–6, 7 drinks/week), education (12, 13–15, 16, 17 years), marital status (married/partnered, divorced/separated/widowed, single), occupation (white collar, non-white collar, unemployed), income (<\$25,000, \$25,001–50,000, \$50,001–100,000, >\$100,000), region (South, Northeast, Midwest, and West), type 2 diabetes, and physical activity (MET-hours/week). Factors that changed over time were modeled as time-varying covariates. Multivariable models mutually adjusted for physical and sexual abuse. Adolescent abuse was further adjusted for child abuse, and adult abuse was further adjusted for child and adolescent abuse. Further control for family history of UL or perceived racism^{17,58} made little difference in the effect estimates and were omitted from multivariable models.

We used stratification methods to examine whether associations were modified by other covariates such as coping, education, and income. P-values from interaction tests were obtained using the likelihood ratio test comparing models with and without cross-product terms between abuse and covariates. Departures from proportional hazards were evaluated in the same manner using cross-product terms between abuse and age (<35, 35 years). Analyses were performed using SAS statistical software version 9.2.⁵⁹

RESULTS

Baseline characteristics of participants according to type of abuse, within each life stage, are shown in Table 1. Fifty-one percent reported abuse in childhood (43% physical and 19% sexual), 38% in adolescence (28% physical and 20% sexual), and 39% in adulthood (29% physical and 19% sexual). Relative to no abuse across the lifespan, child abuse was associated with younger age at baseline, younger maternal age, lower parental education, greater exposure to childhood passive smoking, lower prevalence of home ownership in childhood, earlier ages at first birth and first OC use, higher parity, lower adult household income, higher BMI, and greater tobacco and alcohol consumption. No appreciable differences were found for BMI at age 18 or pelvic exam recency. Patterns were generally similar for abuse reported in adolescence and adulthood.

During 47,382 person-years of follow-up, 1,506 incident UL cases diagnosed by ultrasound (N=1,231) or surgery (N=275) were reported. UL incidence was highest among women reporting child abuse, particularly sexual abuse (Table 2). Relative to no abuse across the lifespan, RRs in the fully-adjusted model were 1.16 (95% CI, 1.02–1.33) for physical abuse only, 1.34 (95% CI, 1.09–1.66) for sexual abuse only, and 1.17 (95% CI, 0.99–1.39) for both physical and sexual abuse in childhood. RRs increased monotonically with increasing frequency of child sexual abuse: RRs for 1–3 and 4 incidents of child sexual abuse were 1.29 (95% CI, 1.04–1.61) and 1.41 (95% CI, 1.07–1.85), respectively. There was little evidence of a dose-response relation between frequency of child physical abuse and UL: relative to no abuse, RRs were 1.19, 1.04, and 1.23 for low, intermediate, and high frequency categories, respectively. The highest category of severity of child physical and sexual abuse was associated with a 57% increased rate of UL relative to no abuse (95% CI, 1.19–2.07). Abuse in adolescence or adulthood was not appreciably associated with UL. Results did not vary significantly by age, education, income, or recency of pelvic exam or ultrasound (data not shown).

Associations between child sexual abuse frequency and UL were weaker among women with higher coping skills (Table 3), defined as scoring above the median on the Carver coping scale. Among women with lower coping skills, RRs for 1–3 and 4 incidents of child sexual abuse were 1.50 and 1.98, respectively; among women with higher coping skills, the respective RRs were 1.19 and 1.05 (P -interaction=0.08). Although stronger results were observed for child abuse overall, RRs were not statistically different across coping strata. Retrospective analyses that started follow-up in 1997 and included all incident cases from 1997–2011 (Supplemental Table 1) or all prevalent and incident cases (Supplemental Table 2) were weaker, but still showed positive associations between child abuse and UL risk.

COMMENT

In this prospective cohort study of black women, child sexual abuse was positively associated with UL incidence. The association remained unchanged after control for early life covariates as well as several health-related behaviors associated with psychosocial stress, including smoking, physical inactivity, and alcohol consumption. The association for child sexual abuse frequency was weaker among those with higher coping skills. Results based on all incident cases, with follow-up starting in 1997 and ending in 2011 (including both retrospective (1997–2005) and prospective (2005–2011) incident periods), still showed positive associations between child sexual abuse and UL, but the associations were weaker. It is unclear whether selection or information biases explain the differences in findings across case groups, or whether they are simply due to chance variation.

Our results agree with those from the sole previous study of abuse victimization and UL, a prospective cohort study that followed 68,505 nurses (97% of whom were white) for self-reported incident UL from 1989 through 2005.¹⁸ Abuse histories were ascertained in 2001.¹⁸ Multivariable RRs were 1.08, 1.17, 1.23, 1.24, and 1.36 for exposure categories ranging from mildest to most severe relative to no abuse. In contrast to our study, however, physical and sexual abuse each had the same magnitude of effect (RR=1.09), and the co-occurrence of abuse types was associated with a 23% increased risk. Increased child emotional support attenuated the associations, consistent with our observation of a weaker effect among women with higher coping skills. The buffering of effects by greater emotional support and coping is supported by current research.⁶⁰

Strengths of our study include the prospective design and validation of UL. While we were unable to validate abuse reports, the instruments from which our abuse questions were adapted have been used in different populations and demonstrate high reproducibility in our cohort and in other studies.^{46,47} Factor analysis confirmed the preconceptualized domains of abuse.⁴⁹ Furthermore, there is biologic plausibility for an adverse effect of child sexual abuse on UL. Child sexual trauma has been linked to dysregulation of the HPA axis and the stress response,^{26–30} which may affect ovarian hormone synthesis³⁸ and UL growth.^{12,13} Sexual abuse is also associated with reproductive tract infections,⁶¹ which are hypothesized to increase UL risk through various mechanisms.^{62–64}

UL rates in our cohort are similar to those from other U.S. studies.^{6,7,57,65,66} Self-report was confirmed in almost all participants from whom we obtained medical records, but not all participants were screened. Thus, our case group likely over-represents women with symptomatic tumors given that most cases reported symptoms and a low percentage were detected incidentally. This concern is partly addressed by our observation of similar associations among the 90% of women with a pelvic exam or ultrasound within the last 5 years. However, even within the screened group, there is still some potential for detection bias resulting from differential symptom experience among abused relative to non-abused women given that most^{67–72} but not all^{73,74} studies have documented a higher prevalence of

chronic pelvic pain and health care utilization in women with abuse histories. Evidence against material distortion from such a bias is that IRRs were elevated just for childhood abuse, and not for all stages and types of abuse.

Although participants reside in all regions of the US, the BWHS is a volunteer sample of women with higher levels of education than the general black population. Estimates of established UL risk factors (e.g., age at menarche⁷⁵) and abuse^{76–82} are consistent with national data on African-Americans. The observed associations were also consistent across levels of education and income. Therefore, we expect our findings to extend to the general population of black women.

In conclusion, we found that child sexual abuse was associated with an increased incidence of UL in black women, after controlling for early life and adult correlates of abuse. Our results support the hypothesis that childhood adversity increases UL risk.

Acknowledgments

Sources of Financial Support: This work was supported by Eunice Kennedy Shriver National Institute of Child Health and Human Development grant HD069602 (PI: Wise) and National Cancer Institute grant CA58420 (PI: Rosenberg). The content is solely the responsibility of the authors and does not necessarily represent the official view of the Eunice Kennedy Shriver National Institute of Child Health and Human Development, the National Cancer Institute, or the National Institutes of Health.

Abbreviations

BMI	body mass index
CI	confidence interval
MET	metabolic equivalent
RR	rate ratio
UL	uterine leiomyomata

References

- Buttram VC, Reiter RC. Uterine leiomyomata: etiology, symptomatology, and management. *Fertil Steril*. 1981; 36:433–45. [PubMed: 7026295]
- Coronado GD, Marshall LM, Schwartz SM. Complications in pregnancy, labor, and delivery with uterine leiomyomas: a population-based study. *Obstet Gynecol*. 2000; 95:764–9. [PubMed: 10775744]
- Stewart EA. Uterine fibroids. *Lancet*. 2001; 357:293–8. [PubMed: 11214143]
- Wilcox LS, Koonin LM, Pokras R, et al. Hysterectomy in the United States, 1988–1990. *Obstet Gynecol*. 1994; 83:549–55. [PubMed: 8134065]
- Farquhar CM, Steiner CA. Hysterectomy rates in the United States 1990–1997. *Obstet Gynecol*. 2002; 99:229–34. [PubMed: 11814502]
- Marshall LM, Spiegelman D, Barbieri RL, et al. Variation in the incidence of uterine leiomyoma among premenopausal women by age and race. *Obstet Gynecol*. 1997; 90:967–73. [PubMed: 9397113]
- Brett KM, Marsh JV, Madans JH. Epidemiology of hysterectomy in the United States: demographic and reproductive factors in a nationally representative sample. *J Womens Health*. 1997; 6:309–16. [PubMed: 9201665]
- Baird DD, Dunson DB, Hill MC, Cousins D, Schectman JM. High cumulative incidence of uterine leiomyoma in black and white women: ultrasound evidence. *Am J Obstet Gynecol*. 2003; 188:100–7. [PubMed: 12548202]

9. Kjerulff KH, Langenberg P, Seidman JD, Stolley PD, Guzinski GM. Uterine leiomyomas: racial differences in severity, symptoms, and age at diagnosis. *J Reprod Med*. 1996; 41:483–90. [PubMed: 8829060]
10. Kjerulff KH, Guzinski GM, Langenberg PW, et al. Hysterectomy and race. *Obstet Gynecol*. 1993; 82:757–64. [PubMed: 8414322]
11. Kjerulff KH, Langenberg P, Guzinski GM. The socioeconomic correlates of hysterectomies in the United States. *Am J Public Health*. 1993; 83:106–8. [PubMed: 8417592]
12. Rein MS, Nowak RA. Biology of uterine myomas and myometrium *in vitro*. *Semin Reprod Endocrinol*. 1992; 10:310–9.
13. Rein MS, Barbieri RL, Friedman AJ. Progesterone: a critical role in the pathogenesis of uterine myomas. *Am J Obstet Gynecol*. 1995; 172:14–8. [PubMed: 7847524]
14. Baird D, Wise LA. Childhood abuse and fibroids. *Epidemiology*. 2011; 22:15–7. [PubMed: 21150350]
15. Vines AI, Ta M, Esserman DA. The Association Between Self-Reported Major Life Events and the Presence of Uterine Fibroids Women's Health Issues. 2010; 20:294–8.
16. Holmes TH, Rahe RH. The social readjustment rating scale. *J Psychosom Res*. 1967; 11:213–8. [PubMed: 6059863]
17. Wise LA, Palmer JR, Cozier YC, et al. Perceived racial discrimination and risk of uterine leiomyomata. *Epidemiology*. 2007; 18:747–57. [PubMed: 17917606]
18. Boynton-Jarrett R, Rich-Edwards JW, Jun H-J, Hibert EN, Wright RJ. Abuse in Childhood and Risk of Uterine Leiomyoma: The Role of Emotional Support in Biologic Resilience. *Epidemiology*. 2011; 22:6–14. [PubMed: 21068667]
19. Boney-McCoy S, Finkelhor D. Psychosocial sequelae of violent victimization in a national youth sample. *J Consult Clin Psychol*. 1995; 63:726–36. [PubMed: 7593865]
20. Margolin G, Gordis EB. The effects of family and community violence on children. *Annual Review of Psychology*. 2000; 51:445–79.
21. Heim C, Newport DJ, Heit S, et al. Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *JAMA*. 2000; 284:592–7. [PubMed: 10918705]
22. McEwen BS. Protective and damaging effects of stress mediators: allostasis and allostatic load. *N Eng J Med*. 1998; 338:171–9.
23. McEwen BS. Sex, stress and the hippocampus: allostasis, allostatic load and the aging process. *Neurobiology of Aging*. 2002; 23:921–39. [PubMed: 12392796]
24. McEwen BS. Interacting mediators of allostasis and allostatic load: towards an understanding of resilience in aging. *Metabolism*. 2003; 52:10–6. [PubMed: 14577057]
25. McEwen BS, Seeman T. Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci*. 1999; 896:30–47. [PubMed: 10681886]
26. Heim C, Newport DJ, Heit S, et al. Pituitary-Adrenal and Autonomic Responses to Stress in Women After Sexual and Physical Abuse in Childhood. *JAMA*. 2000; 284:592–7. [PubMed: 10918705]
27. Penza KM, Heim C, Nemeroff CB. Neurobiological effects of childhood abuse: implications for the pathophysiology of depression and anxiety. *Arch Womens Ment Health*. 2004; 6:15–22. [PubMed: 12715261]
28. Heim C, Nemeroff CB. The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies. *Biol Psychiatry*. 2001; 49:1023–39. [PubMed: 11430844]
29. De Bellis MD, Chrousos GP, Dorn LD, et al. Hypothalamic-pituitary-adrenal axis dysregulation in sexually abused girls. *J Clin Endocrinol Metab*. 1994; 78:249–55. [PubMed: 8106608]
30. Bremner JD, Vythilingam M, Anderson G, et al. Assessment of the hypothalamic-pituitary-adrenal axis over a 24-hour diurnal period and in response to neuroendocrine challenges in women with and without childhood sexual abuse and posttraumatic stress disorder. *Biol Psychiatry*. 2003; 54:710–8. [PubMed: 14512211]

31. Meaney MJ, Aitken DH, van Berkel C, Bhatnagar S, Sapolsky RM. Effect of neonatal handling on age-related impairments associated with the hippocampus. *Science*. 1988; 239:766–8. [PubMed: 3340858]
32. Kam K, Park Y, Cheon M, et al. Effects of immobilization stress on estrogen-induced surges of luteinizing hormone and prolactin in ovariectomized rats. *Endocrine*. 2000; 12:279–87. [PubMed: 10963049]
33. Tilbrook AJ, Canny BJ, Serapiglia MD, Ambrose TJ, Clarke IJ. Suppression of the secretion of luteinizing hormone due to isolation/restraint stress in gonadectomised rams and ewes is influenced by sex steroids. *J Endocrinol*. 1999; 160:469–81. [PubMed: 10076193]
34. Breen KM, Billings HJ, Wagenmaker ER, Wessinger EW, Karsch FJ. Endocrine basis for disruptive effects of cortisol on preovulatory events. *Endocrinology*. 2005; 146:2107–15. [PubMed: 15625239]
35. Ferin M. Clinical review 105: Stress and the reproductive cycle. *J Clin Endocrin Metab*. 1999; 84:1768–74.
36. Puder JJ, Freda PU, Goland RS, Ferin M, Wardlaw SL. Stimulatory effects of stress on gonadotropin secretion in estrogen-treated women. *J Clin Endocrin Metab*. 2000; 85:2184–8.
37. Lahoz MM, Nagle CA, Porta M, Farinati Z, Manzur TD. Cortisol response and ovarian hormones in juvenile and cycling female Cebus monkeys: effect of stress and dexamethasone. *Am J Primatol*. 2007; 69:551–61. [PubMed: 17177312]
38. Kornya L, Bodis J, Koppan M, Tinneberg HR, Torok A. Modulatory effect of acetylcholine on gonadotropin-stimulated human granulosa cell steroid secretion. *Gynecol Obstet Invest*. 2001; 52:104–7. [PubMed: 11586037]
39. Wise LA, Palmer JR, Harlow BL, et al. Risk of uterine leiomyomata in relation to tobacco, alcohol and caffeine consumption in the Black Women's Health Study. *Hum Reprod*. 2004; 19:1746–54. [PubMed: 15218005]
40. D'Aloisio, AA.; Baird, DD.; Dunson, DB. Association of alcohol intake with incidence of uterine fibroids [Abstract PP41]. National Institutes of Health; Bethesda, MD: 2005.
41. Wise LA, Radin RG, Palmer JR, et al. Intake of fruit, vegetables, and carotenoids in relation to risk of uterine leiomyomata. *Am J Clin Nutr*. 2011; 94:1620–31. [PubMed: 22071705]
42. Wise LA, Radin RG, Palmer JR, Kumanyika SK, Rosenberg L. A prospective study of dairy intake and risk of uterine leiomyomata. *Am J Epidemiol*. 2010; 171:221–32. [PubMed: 19955473]
43. Radin RG, Palmer JR, Rosenberg L, Kumanyika SK, Wise LA. Dietary glycemic index and load in relation to risk of uterine leiomyomata in the Black Women's Health Study. *Am J Clin Nutr*. 2010; 91:1281–8. [PubMed: 20200259]
44. Chiaffarino F, Parazzini F, La Vecchia C, et al. Diet and uterine myomas. *Obstet Gynecol*. 1999; 94:395–8. [PubMed: 10472866]
45. Rosenberg L, Adams-Campbell LL, Palmer JR. The Black Women's Health Study: a follow-up study for causes and preventions of illness. *J Am Med Womens Assoc*. 1995; 50:56–8. [PubMed: 7722208]
46. Straus MA. Measuring intrafamily conflict and violence: the Conflict Tactics (CT) scales. *J Marriage Fam*. 1979; 41:75–88.
47. McFarlane J, Parker B, Soeken K, Bullock L. Assessing for abuse during pregnancy. Severity and frequency of injuries and associated entry into prenatal care. *JAMA*. 1992; 267:3176–8. [PubMed: 1593739]
48. Wise LA, Palmer JR, Boggs DA, Adams-Campbell LL, Rosenberg L. Abuse victimization and risk of breast cancer in the Black Women's Health Study [corrected]. *Cancer Causes Control*. 2011; 22:659–69. [PubMed: 21327459]
49. Wise LA, Palmer JR, Rothman EF, Rosenberg L. Childhood abuse and early menarche: findings from the black women's health study. *Am J Public Health*. 2009; 99 (Suppl 2):S460–6. [PubMed: 19443822]
50. Boynton-Jarrett R, Rosenberg L, Palmer JR, Boggs DA, Wise LA. Child and Adolescent Abuse in Relation to Obesity in Adulthood: The Black Women's Health Study. *Pediatrics*. 2012; 130:245–53. [PubMed: 22753562]

51. Wise LA, Zierler S, Krieger N, Harlow BL. Adult onset of major depressive disorder in relation to early life violent victimisation: a case-control study. *Lancet*. 2001; 358:881–7. [PubMed: 11567704]
52. Rayworth BB, Wise LA, Harlow BL. Childhood abuse and risk of eating disorders in women. *Epidemiology*. 2004; 15:271–8. [PubMed: 15097006]
53. Schwartz, SM.; Marshall, LM. Uterine leiomyomata. In: Goldman, MB.; Hatch, MC., editors. *Women Health*. San Diego, CA: Academic Press; 2000. p. 240-52.
54. Loutradis D, Antsaklis A, Creatas G, et al. The validity of gynecological ultrasonography. *Gynecol Obstet Invest*. 1990; 29:47–50. [PubMed: 2190879]
55. Dueholm M, Lundorf E, Hansen ES, Ledertoug S, Olesen F. Accuracy of magnetic resonance imaging and transvaginal ultrasonography in the diagnosis, mapping, and measurement of uterine myomas. *Am J Obstet Gynecol*. 2002; 186:409–15. [PubMed: 11904599]
56. Carver CS. You want to measure coping but your protocol's too long: consider the brief COPE. *Int J Behav Med*. 1997; 4:92–100. [PubMed: 16250744]
57. Wise LA, Palmer JR, Stewart EA, Rosenberg L. Age-specific incidence rates for self-reported uterine leiomyomata in the Black Women's Health Study. *Obstet Gynecol*. 2005; 105:563–8. [PubMed: 15738025]
58. Wise LA, Ruiz-Narváez EA, Palmer JR, et al. African ancestry and genetic risk for uterine leiomyomata. *Am J Epidemiol*. 2012; 176:1159–68. [PubMed: 23161897]
59. SAS Institute Inc. *SAS/STAT® 9.2 User's Guide*. Vol. 2008. Cary, NC. Cary, NC: SAS Institute; 2008.
60. Hager AD, Runtz MG. Physical and psychological maltreatment in childhood and later health problems in women: An exploratory investigation of the roles of perceived stress and coping strategies. *Child Abuse Negl*. 2012; 36:393–403. [PubMed: 22609072]
61. Girardet RG, Lahoti S, Howard LA, et al. Epidemiology of sexually transmitted infections in suspected child victims of sexual assault. *Pediatrics*. 2009; 124:79–86. [PubMed: 19564286]
62. Witherspoon JT, Butler VW. The etiology of uterine fibroids with special reference to the frequency of their occurrence in the Negro: a hypothesis. *Surg Gynecol Obstet*. 1934; 58:57–61.
63. Laughlin SK, Schroeder JC, Baird DD. New directions in the epidemiology of uterine fibroids. *Semin Reprod Med*. 2010; 28:204–17. [PubMed: 20414843]
64. Payson M, Malik M, Siti-Nur Morris S, et al. Activating transcription factor 3 gene expression suggests that tissue stress plays a role in leiomyoma development. *Fertil Steril*. 2009; 92:748–55. [PubMed: 18692824]
65. Whiteman MK, Kuklina E, Jamieson DJ, Hillis SD, Marchbanks PA. Inpatient hospitalization for gynecologic disorders in the United States. *Am J Obstet Gynecol*. 2010; 202:541 e1–6. [PubMed: 20132921]
66. Velebil P, Wingo PA, Xia Z, Wilcox LS, Peterson HB. Rate of hospitalization for gynecologic disorders among reproductive-age women in the United States. *Obstet Gynecol*. 1995; 86:764–9. [PubMed: 7566845]
67. Plichta S. The effects of woman abuse on health care utilization and health status: a literature review. *Womens Health Issues*. 1992; 2:154–63. [PubMed: 1422244]
68. Bonomi AE, Anderson ML, Rivara FP, et al. Health care utilization and costs associated with childhood abuse. *J Gen Intern Med*. 2008; 23:294–9. [PubMed: 18204885]
69. Rivara FP, Anderson ML, Fishman P, et al. Healthcare utilization and costs for women with a history of intimate partner violence. *Am J Prev Med*. 2007; 32:89–96. [PubMed: 17234483]
70. Ulrich YC, Cain KC, Sugg NK, et al. Medical care utilization patterns in women with diagnosed domestic violence. *Am J Prev Med*. 2003; 24:9–15. [PubMed: 12554018]
71. Coker AL, Reeder CE, Fadden MK, Smith PH. Physical partner violence and medicaid utilization and expenditures. *Public Health Rep*. 2004; 119:557–67. [PubMed: 15504447]
72. Wisner CL, Gilmer TP, Saltzman LE, Zink TM. Intimate partner violence against women: Do victims cost health plans more? *J Fam Pract*. 1999; 48:439–43. [PubMed: 10386487]

73. Modesitt SC, Gambrell AC, Cottrill HM, et al. Adverse impact of a history of violence for women with breast, cervical, endometrial, or ovarian cancer. *Obstet Gynecol.* 2006; 107:1330–6. [PubMed: 16738160]
74. Gandhi S, Rovi S, Vega M, et al. Intimate partner violence and cancer screening among urban minority women. *J Am Board Fam Med.* 2010; 23:343–53. [PubMed: 20453180]
75. Abma JC, Chandra A, Mosher WD, Peterson LS, Piccinino LJ. Fertility, family planning, and women's health: New data from the 1995 National Survey of Family Growth. *Vital Health Stat.* 1997; 23:19.
76. Finkelhor D. Current information on the scope and nature of child sexual abuse. *Future Child.* 1994; 4:31–53. [PubMed: 7804768]
77. Felitti VJ, Anda R, Nordenberg D, et al. The Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults: The Adverse Childhood Experiences (ACE) Study. *Amer J Prev Med.* 1998; 14:245–58. [PubMed: 9635069]
78. Finkelhor D, Dziuba-Leatherman J. Children as victims of violence: A national survey. *Pediatrics.* 1994; 94:413–20. [PubMed: 7936846]
79. Kilpatrick, DG.; Saunders, BE. Final Report submitted November 1997 to the US Department of Justice, Office of Justice Programs, National Institute of Justice, Grant No 93-IJ-CX-0023. 1997. Prevalence and consequences of child victimization: Results from the national survey of adolescents.
80. Kilpatrick DG, Acierno R, Saunders B, et al. Risk factors for adolescent substance abuse and dependence: Data from a national sample. *J Consult Clin Psychol.* 2000; 68:19–30. [PubMed: 10710837]
81. Finkelhor D, Ormrod RK, Turner H. Re-victimization patterns in a national longitudinal sample of children and youth. *Child Abuse Negl.* 2007; 31:479–502. [PubMed: 17537508]
82. Barnett, OW.; Miller-Perrin, CL.; Perrin, RD. Physical child abuse. In: Barnett, OW.; Miller-Perrin, CL.; Perrin, RD., editors. *Family violence across the lifespan.* Thousand Oaks: SAGE; 1996.

Table 1
Baseline characteristics of 9,910 women according to lifetime abuse victimization, the Black Women's Health Study, 2005.

Characteristic ^a	No abuse across lifespan	Stage and type of abuse							
		Childhood		Adolescence		Adulthood		Physical & sexual	Physical & sexual
		Physical only	Sexual only	Physical only	Sexual only	Physical only	Sexual only		
Number of women	2,971	3,161	629	1,274	1,802	1,030	949	1,961	953
<i>Early life characteristics:</i>									
Foreign-born (%)	5.7	6.0	6.6	6.5	5.9	6.9	5.5	5.3	5.6
First-born (%)	37.8	37.0	43.8	38.7	37.5	40.8	38.0	37.0	38.9
Sibship size (mean)	3.9	4.1	3.9	4.3	4.2	3.9	4.4	4.2	4.2
Maternal age <20 y (%)	12.7	16.2	18.5	24.3	18.9	17.5	24.8	18.9	20.1
Childhood passive smoke exposure (%)	50.0	57.1	53.0	62.9	57.8	58.2	61.2	57.0	59.6
Neither parent finished high school (%)	10.4	11.2	11.3	11.8	11.9	10.1	13.2	12.4	10.9
Home ownership in childhood (%)	65.7	61.4	58.9	54.0	59.1	61.4	54.8	60.6	58.4
Age at menarche, y (mean)	12.4	12.4	12.2	12.3	12.5	12.2	12.4	12.3	12.4
<i>Adult characteristics:</i>									
Age at baseline, y (mean)	41.6	41.0	40.8	40.6	40.8	40.9	41.4	41.3	42.0
Parous, yes (%)	63	67	65	68	66	70	71	71	75
Age at first birth, y (mean) ^b	26.1	25.5	25.0	23.8	24.6	24.5	23.0	23.8	23.5
Age at first OC use, y (mean) ^c	19.7	19.4	19.4	19.1	19.4	19.1	18.6	19.1	18.7
Education, y (mean)	15.0	15.1	15.0	14.9	14.9	15.0	14.7	14.7	14.7
Income \$25,000 in 2003 (%)	10	9	10	12	11	10	14	12	15
BMI at age 18, kg/m ² (mean)	21.9	22.0	22.4	22.2	22.1	22.0	21.9	21.9	22.1
Current BMI, kg/m ² (mean)	29.7	30.1	30.5	30.9	30.3	30.3	30.9	30.4	30.2

Characteristic ^a	Stage and type of abuse									
	No abuse across lifespan	Childhood		Adolescence		Adulthood				
		Physical only	Sexual only	Physical & sexual	Physical only	Sexual only	Physical & sexual	Physical only	Sexual only	Physical & sexual
Physical activity, MET-h/wk (mean)	20.8	20.8	20.9	21.1	21.8	19.5	21.7	20.7	20.8	21.5
Family history of UL, yes (%)	30	35	31	35	34	32	34	33	32	32
Smoking, current (%)	8	11	9	11	14	13	19	16	13	18
Alcohol, current drinks/wk (%)	2	4	3	4	3	4	6	4	4	6
Coping score in 2005 (mean)	28.4	28.9	29.4	29.2	29.2	28.9	29.4	29.2	29.1	29.2
Pelvic exam <5 y in 2007, yes (%)	87	90	88	90	89	91	91	89	90	90

^a Characteristics are presented as means or percents within abuse categories and are age-standardized to distribution of cohort at baseline.

^b Restricted to parous women only.

^c Restricted to ever users of oral contraceptives (OC).

Table 2

Lifetime abuse victimization and risk of uterine leiomyoma, Black Women's Health Study (2005–2011)

	Cases	Person-years	Age-adjusted RR (95% CI)	Multivariable RR (95% CI) ^d	Multivariable RR (95% CI) ^b
No abuse across the lifespan ^c	403	14,360	1.00 (referent)	1.00 (referent)	1.00 (referent)
Any abuse in childhood	842	24,030	1.22 (1.08–1.37)	1.20 (1.07–1.36)	1.19 (1.05–1.34)
Any abuse in adolescence	608	18,015	1.02 (0.84–1.24)	1.02 (0.83–1.24)	0.99 (0.81–1.21)
Any abuse in adulthood	600	18,146	1.03 (0.86–1.24)	1.03 (0.86–1.24)	1.01 (0.84–1.22)
Cumulative exposure to abuse					
Childhood only	251	7,112	1.22 (1.05–1.43)	1.22 (1.04–1.43)	1.22 (1.04–1.43)
Adolescence only	68	2,329	1.03 (0.79–1.33)	1.04 (0.80–1.35)	1.02 (0.79–1.33)
Adulthood only	131	4,454	1.06 (0.87–1.29)	1.06 (0.87–1.29)	1.05 (0.86–1.28)
Childhood and adolescence	184	5,436	1.17 (0.98–1.39)	1.15 (0.97–1.37)	1.13 (0.95–1.35)
Childhood and adulthood	113	3,441	1.16 (0.94–1.43)	1.15 (0.93–1.42)	1.13 (0.91–1.39)
Adolescence and adulthood	62	2,210	1.01 (0.77–1.32)	1.01 (0.78–1.33)	1.00 (0.77–1.31)
All three life stages	294	8,042	1.27 (1.09–1.47)	1.25 (1.08–1.46)	1.25 (1.07–1.46)
Stage at first abuse					
Childhood	842	24,030	1.22 (1.08–1.37)	1.21 (1.07–1.36)	1.19 (1.06–1.35)
Adolescence	130	4,538	1.02 (0.84–1.24)	1.03 (0.84–1.25)	1.01 (0.83–1.24)
Adulthood	131	4,454	1.06 (0.87–1.29)	1.06 (0.87–1.29)	1.05 (0.86–1.28)
Childhood					
Type of abuse					
Physical abuse only	511	15,111	1.18 (1.03–1.34)	1.17 (1.02–1.33)	1.16 (1.02–1.33)
Sexual abuse only	115	2,892	1.38 (1.12–1.70)	1.38 (1.12–1.70)	1.34 (1.09–1.66)
Both physical and sexual abuse	216	6,028	1.24 (1.05–1.46)	1.21 (1.02–1.43)	1.17 (0.99–1.39)
Physical abuse frequency ^b					
Low	319	9,309	1.19 (1.02–1.38)	1.19 (1.02–1.38)	1.19 (1.02–1.38)
Intermediate	143	4,585	1.06 (0.87–1.29)	1.05 (0.86–1.28)	1.04 (0.85–1.27)
High	265	7,245	1.24 (1.05–1.47)	1.23 (1.04–1.45)	1.23 (1.04–1.46)
Sexual abuse frequency ^e					
1–3 incidents	216	5,984	1.34 (1.08–1.66)	1.32 (1.07–1.65)	1.29 (1.04–1.61)
4 incidents	115	2,936	1.43 (1.09–1.88)	1.41 (1.08–1.85)	1.41 (1.07–1.85)

	Cases	Person-years	Age-adjusted RR (95% CI)	Multivariable RR (95% CI) ^a	Multivariable RR (95% CI) ^b
Severity of abuse					
Mild	248	7,181	1.21 (1.04–1.42)	1.21 (1.03–1.42)	1.21 (1.03–1.42)
Moderate	275	7,938	1.21 (1.03–1.40)	1.19 (1.02–1.39)	1.17 (1.00–1.36)
Severe	258	7,642	1.16 (0.99–1.36)	1.14 (0.97–1.34)	1.13 (0.96–1.32)
Very severe	61	1,270	1.66 (1.27–2.17)	1.60 (1.22–2.11)	1.57 (1.19–2.07)
Adolescence					
Type of abuse					
Physical abuse only	272	8,709	0.95 (0.76–1.19)	0.95 (0.76–1.20)	0.92 (0.73–1.15)
Sexual abuse only	159	4,908	1.03 (0.83–1.28)	1.03 (0.83–1.29)	1.03 (0.82–1.28)
Both physical and sexual abuse	177	4,398	1.20 (0.93–1.54)	1.19 (0.92–1.53)	1.13 (0.87–1.47)
Physical abuse frequency ^d					
Low	244	7,159	0.96 (0.74–1.25)	0.97 (0.74–1.26)	0.95 (0.73–1.24)
Intermediate	88	2,706	0.87 (0.62–1.22)	0.87 (0.62–1.22)	0.86 (0.61–1.21)
High	117	3,242	0.94 (0.67–1.30)	0.94 (0.67–1.31)	0.95 (0.68–1.32)
Sexual abuse frequency ^e					
1–3 incidents	280	7,772	1.05 (0.82–1.34)	1.06 (0.83–1.35)	1.06 (0.82–1.35)
4 incidents	56	1,534	1.00 (0.69–1.44)	1.00 (0.69–1.45)	0.96 (0.66–1.40)
Severity of abuse					
Mild	157	5,192	0.93 (0.73–1.18)	0.93 (0.73–1.18)	0.89 (0.70–1.13)
Moderate	304	8,594	1.08 (0.87–1.33)	1.07 (0.87–1.32)	1.06 (0.86–1.31)
Severe	121	3,683	0.95 (0.72–1.24)	0.94 (0.72–1.23)	0.91 (0.69–1.20)
Very severe	26	547	1.42 (0.91–2.21)	1.40 (0.90–2.18)	1.36 (0.86–2.12)
Adulthood					
Type of abuse					
Physical abuse only	299	9,188	1.01 (0.83–1.24)	1.01 (0.83–1.23)	0.99 (0.81–1.21)
Sexual abuse only	147	4,418	1.04 (0.83–1.30)	1.05 (0.84–1.31)	1.03 (0.82–1.29)
Both physical and sexual abuse	154	4,541	1.06 (0.83–1.35)	1.07 (0.84–1.36)	1.08 (0.84–1.38)
Physical abuse frequency ^d					
Low	276	8,373	1.00 (0.81–1.25)	1.00 (0.80–1.24)	0.99 (0.80–1.24)
Intermediate	74	2,306	0.95 (0.70–1.29)	0.96 (0.70–1.30)	0.97 (0.71–1.32)
High	103	3,051	1.00 (0.74–1.33)	0.99 (0.74–1.33)	1.01 (0.75–1.36)

	Cases	Person-years	Age-adjusted RR (95% CI)	Multivariable RR (95% CI) ^a	Multivariable RR (95% CI) ^b
Sexual abuse frequency ^c					
1–3 incidents	259	8,021	0.97 (0.74–1.26)	0.98 (0.75–1.28)	0.96 (0.74–1.26)
4 incidents	42	938	1.33 (0.87–2.03)	1.39 (0.91–2.12)	1.38 (0.90–2.11)
Severity of abuse					
Mild	211	6,266	1.03 (0.84–1.25)	1.02 (0.84–1.25)	1.01 (0.83–1.22)
Moderate	288	9,044	0.98 (0.81–1.18)	0.98 (0.81–1.19)	0.97 (0.80–1.18)
Severe	61	1,732	1.06 (0.78–1.43)	1.06 (0.79–1.44)	1.07 (0.79–1.46)
Very severe	4	98	1.26 (0.46–3.41)	1.26 (0.46–3.43)	1.17 (0.43–3.21)

Abbreviations: UL=uterine leiomyomata, RR=incidence rate ratio, CI=confidence interval.

^aAdjusted for age, time period, foreign-born, maternal age, birth order, sibship size, childhood passive smoke exposure, parental education, home ownership, and age at menarche. Adjusted for type and stage of abuse, when applicable.

^bFurther adjusted for parity, age at first birth, years since last birth, ever use of OCs, age at first OC use, BMI, current alcohol intake, smoking history, education, marital status, household income, occupation, type 2 diabetes, MET-hours per week of physical activity.

^cReference group for all analyses.

^dExcludes women who reported sexual abuse only.

^eExcludes women who reported physical abuse only.

Table 3
Child abuse victimization and risk of uterine leiomyomata, stratified by coping skills (2005–2011)

	Low coping skills (below median of Carver coping scale)			High coping skills (above median of Carver coping scale)		
	Cases	Person-years	Multivariable RR (95% CI) ^a	Cases	Person-years	Multivariable RR (95% CI) ^a P-interaction
No abuse across the lifespan ^b	171	6,343	1.00 (referent)	208	7,067	1.00 (referent)
Any abuse in childhood	357	10,173	1.23 (1.02–1.49)	458	12,968	1.14 (0.97–1.35) 0.47
Type of abuse						
Physical abuse only	214	6,501	1.18 (0.96–1.45)	280	8,045	1.13 (0.94–1.36) 0.54
Sexual abuse only	52	1,125	1.63 (1.18–2.24)	59	1,631	1.16 (0.86–1.55)
Both physical and sexual	91	2,547	1.20 (0.92–1.56)	119	3,293	1.16 (0.92–1.47)
Physical abuse frequency ^c						
Low	136	3,977	1.21 (0.96–1.54)	172	4,959	1.13 (0.91–1.40) 0.94
Intermediate	58	1,992	1.01 (0.74–1.38)	79	2,420	1.06 (0.81–1.39)
High	111	3,079	1.27 (0.98–1.66)	148	3,959	1.20 (0.95–1.51)
Sexual abuse frequency ^d						
1–3 incidents	90	2,535	1.50 (1.07–2.12)	122	3,242	1.19 (0.88–1.62) 0.08
4 incidents	53	1,137	1.98 (1.32–2.97)	56	1,682	1.05 (0.70–1.57)
Severity of abuse ^e						
Mild	104	3,083	1.23 (0.96–1.58)	136	3,798	1.16 (0.91–1.49) 0.79
Moderate	114	3,355	1.18 (0.93–1.51)	152	4,277	1.15 (0.90–1.46)
Severe	114	3,255	1.22 (0.95–1.56)	136	4,147	1.06 (0.81–1.39)
Very severe	25	481	1.79 (1.16–2.77)	34	747	1.44 (0.96–2.17)

Abbreviations: UL=uterine leiomyomata, RR=incidence rate ratio, CI=confidence interval. Note: Median of 10-item abbreviated Carver coping scale was 29 at start of follow-up.

^a Adjusted for age, time period, foreign-born, maternal age, birth order, sibship size, childhood passive smoke exposure, parental education, home ownership, age at menarche, parity, age at first birth, years since last birth, ever use of OCs, age at first OC use, BMI, current alcohol intake, smoking history, education, marital status, household income, occupation, type 2 diabetes, MET-hours per week of physical activity. Adjusted for type and stage of abuse, when applicable.

^b Reference group for all analyses.

^c Excludes women who reported sexual abuse only.

^d Excludes women who reported physical abuse only.