JOURNAL OF WOMEN'S HEALTH
Volume 21, Number 1, 2012

DOI: 10.1089/jwh.2011.2745

Type and Timing of Menopause and Later Life Mortality Among Women in the Iowa Established Populations for the Epidemiological Study of the Elderly Cohort

Sarah E. Tom, Ph.D.,[1] Rachel Cooper, Ph.D.,[2] Robert B. Wallace, M.D.,[3] and Jack M. Guralnik, M.D., Ph.D.[4]

Abstract

Background: The relationship between menopausal characteristics and later life mortality is unclear. We tested the hypotheses that women with surgical menopause would have increased all-cause and cardiovascular mortality compared with women with natural menopause, and that women with earlier ages at natural or surgical menopause would have greater all-cause and cardiovascular mortality than women with later ages at menopause.
Methods: Women who participated in the Iowa cohort of the Established Populations for the Epidemiologic Study of the Elderly ($n = 1684$) reported menopausal characteristics and potential confounding variables at baseline and were followed up for up to 24 years. Participants were aged 65 years or older at baseline and lived in rural areas. We used survival analysis to examine the relationships between menopausal characteristics and all-cause and cardiovascular mortality.
Results: A total of 1477 women (87.7% of respondents) died during the study interval. Women with an age at natural menopause ≥ 55 years had increased all-cause and cardiovascular disease mortality compared with women who had natural menopause at younger ages. Type of menopause and age at surgical menopause were not related to mortality. These patterns persisted after adjustment for potential confounding variables.
Conclusions: Among an older group of women from a rural area of the United States, later age at natural menopause was related to increased all-cause and cardiovascular mortality. Monitoring the cardiovascular health of this group of older women may contribute to improved survival times.

Introduction

If age at menopause is a marker of the overall intrinsic rate of biological aging, then women with later menopause should have improved survival in later life. Older women are no longer acutely exposed to fluctuations in endogenous reproductive hormones in the same way that they were earlier in their lives and during the menopausal transition. However, characteristics of menopause may indicate later life health risks, related to hormonal exposures or other processes that may influence the risk for cardiovascular mortality in particular.[1,2] Most previous analyses have found relationships of moderate strength between early age at natural menopause and increased risk of all-cause[1,3–11] and cardiovascular mortality.[1,5,7,9,10,12–14] Surgical menopause may also be related to the overall intrinsic rate of biological aging. Women who experience surgical menopause, and in particular early surgical menopause, may have less favorable underlying health profiles and poorer health later in life[15–18] compared with women who experience natural menopause. These differences may relate to long-term health and survival differentials. However, studies have not detected differences in all-cause or cardiovascular mortality between women with natural versus surgical menopause.[10,19] Some studies have shown no relationship between age at surgical menopause and all-cause mortality between women with natural or surgical menopause,[3,4] but the majority have demonstrated a relationship between early surgical menopause and increased cardiovascular and all-cause mortality risks.[10,14,20,21]

The relationships between age at menopause and cardiovascular and all-cause mortality may differ by attained age at death. The association between younger age at natural

[1]Department of Preventive Medicine and Community Health, University of Texas Medical Branch, Galveston, Texas.
[2]MRC Unit for Lifelong Health and Ageing, Division of Population Health, University College London, United Kingdom.
[3]Department of Epidemiology, University of Iowa, Iowa City, Iowa.
[4]Department of Epidemiology and Public Health, University of Maryland School of Medicine, Baltimore, Maryland.

menopause and mortality may be stronger in women with younger rather than older attained age.[6,8,14] However, some studies have found that ischemic heart disease mortality risk for both women with early and older age at natural menopause is stronger in women with younger rather than older attained age.[13,22] Yet other studies have demonstrated that early age at menopause is associated with mortality only in women with older attained age.[1,7]

The objectives of this study were to investigate associations of type and timing of menopause with all-cause and cardiovascular mortality over 24 years of follow-up among a group of women who were aged 65 years and over at baseline. The older age distribution of participants will further clarify the associations between type and age at menopause with mortality at oldest attained ages. We hypothesized that women with surgical menopause would have increased all-cause and cardiovascular mortality compared with women with natural menopause, and that women with earlier ages at natural or surgical menopause would have increased all-cause and cardiovascular mortality compared with other women.

Materials and Methods

Data

The Established Populations for the Epidemiologic Study of the Elderly (EPESE) . is a longitudinal study of community-dwelling adults aged 65 years and over in the United States that began in the early 1980s in New Haven, Connecticut; north central North Carolina; East Boston, Massachusetts; and Iowa and Washington counties, Iowa.[23] The study continued to track individuals who moved into nursing homes during follow-up. This analysis utilizes the Iowa cohort, comprised of all individuals aged ≥ 65 years living in two counties. The Iowa cohort was the only EPESE cohort to ascertain information on menopause. A total of 2253 female study participants enrolled at baseline in 1981–1983.

Main explanatory factors

Women reported the type and timing of their menopause at the baseline data collection during in-person interviews. We categorized type of menopause as surgical or natural based on the reason the participant gave for her menstrual periods stopping. We considered women who reported their periods stopping because of natural reasons as having undergone a natural menopause, whereas we categorized women who reported surgery as the reason for cessation as having a surgical menopause. Because the survey did not include further details of surgery, this second group included women who had undergone hysterectomy with or without bilateral oophorectomy and also those who had undergone bilateral oophorectomy without hysterectomy. Women who reported other reasons for their periods stopping, who did not know the reason, or who did not respond to this question were excluded from analyses ($n=290$). Women also reported age at last menstrual period, which we used for age at menopause in analyses.

Outcome measures

We ascertained mortality by linkage to the National Death Index with follow-up until 31 December 2005. We grouped cause of death by underlying cause of death according to the International Classification of Diseases, Ninth Revision (ICD-9).[24] We defined deaths due to cardiovascular diseases by ICD-9 codes 390–434 and 436–448.

Covariates

At baseline women reported age, educational attainment, number of pregnancies, age at menarche, smoking, height, weight, and use of estrogen therapy, all of which we included as covariates in analyses. We created a binary variable to indicate whether the respondent had completed at least high school. We categorized number of pregnancies as none, 1, 2, 3, or 4 or more. Categories for age at menarche in years were ≤ 12, 13, 14, or ≥ 15 to form approximate quarters. We calculated body mass index as weight in kilograms/height in meters squared and categorized this variable as ≤ 25, 25.1–29.9, and ≥ 30. Due to the small percentage of women who had used estrogen therapy for more than 2 years, we defined estrogen therapy use as never versus ever user. We excluded women who were missing data on any covariate from analyses ($n=107$). Of the 2253 women who participated in the baseline Iowa EPESE, 1707 provided information on menopause. Of these 1707 women we were able to include 1684 women who had complete data on all covariates in the analysis.

Statistical analysis

In descriptive analyses of covariates comparing women who had undergone natural menopause with women who had undergone surgical menopause, we used a Wilcoxon–Mann–Whitney test for age and age at surgical and natural menopause, chi-squared tests for all other categorical variables, and a *t*-test for age at menarche. We used Cox proportional hazards models to examine the associations of type and timing of menopause with all-cause mortality. Attained age was the time variable, with those participants with no known date of death or attrition censored at their attained age by 31 December 2005. We ran one set of models to test the association between type of menopause and mortality and a second set of models for age at menopause. Because women with surgical menopause experienced this event on average at earlier ages than women with natural menopause, we ran the analysis for age at menopause by type of menopause in order to isolate relationships with age at menopause from type of menopause. First we ran an unadjusted model, and then we ran a model adjusted for all covariates.

We used a competing risks framework for the outcome of cardiovascular mortality,[25,26] with cardiovascular deaths as the outcome and deaths from noncardiovascular causes censored. We present results for cardiovascular mortality excluding women with unknown cause of death ($n=31$). Including these women in models yielded similar results. We tested for time dependence for type of menopause and age at menopause according to log-log plots, Kaplan–Meier curves, and likelihood ratio tests for the inclusion of a time interaction for type of menopause and age at menopause. Additionally, we stratified the sample on the attained age of 90 years, the approximate median attained age of the cohort. None of these tests showed strong evidence of time dependence. We performed analyses using Stata SE version 10.

Results

Over 24 years of follow-up, 1477 women (87.7% of respondents) had documented deaths. Of deaths with a known

cause, 41% were related to cardiovascular causes. Women with surgical menopause had a younger median age at baseline (74 years, interquartile range [IQR] = 68, 80) than women with natural menopause (73 years, IQR = 67, 78) (Table 1). Women with natural menopause had an older median age at menopause than women with surgical menopause. Women with surgical menopause had a slightly younger age at menarche than women with natural menopause. A larger percentage of women with surgical menopause had ever used estrogen therapy.

Type of menopause was not related to all-cause, cardiovascular, or noncardiovascular mortality (Table 2). The risk of all-cause mortality moderately increased with older age at natural menopause ($p = 0.03$ for linear trend; Table 3). Women with age at natural menopause ≥55 years had a 29% increased rate of cardiovascular mortality (HR 1.29, 95% CI 1.02, 1.63), compared with women with natural menopause at age 50–54 years. Further adjustment of confounding variables did not attenuate this relationship. Age at surgical menopause was not related to mortality risk (Table 4).

Table 1. Sample Characteristics by Type of Menopause

Characteristic	*% Natural menopause (n = 1247)*	*% Surgical menopause (n = 437)*	p *value*[a]
Age at baseline (years)[b]			
65–69	26.5	30.9	< 0.01
70–74	25.3	30	
75–79	22.8	20.8	
≥80	25.3	18.3	
Median (IQR[c])	74 (69, 80)	73 (69, 78)	
Age at natural menopause (years)			< 0.01
<45	13.1		
45–49	25.6		
50–54	47.0		
≥55	14.3		
Median (IQR)	50 (42, 47)		
Age at surgical menopause (years)			
<40		22.0	
40–44		24.5	
45–49		22.0	
≥50		31.5	
Median (IQR)		45 (40, 50)	
No. of pregnancies			
0	16.6	16.9	0.67
1	32.2	33	
2	16.1	15.6	
3	13	15.4	
4 or more	22.1	19.2	
Age at menarche (years)			< 0.01
<12	33.9	41.4	
13	27.4	28.2	
14	21.9	16.9	
≥15	16.8	13.5	
Education			
Less than high school	44.9	43.8	0.77
High school or more	55.1	56.2	
Smoking			
Ever smoked	13.8	14.2	0.75
Never smoked	86.2	85.8	
Body mass index (kg/m^2)			
≤25	55.7	55.1	0.97
25.1–29.9	30.2	30.5	
≥30	14.1	14.5	
Ever used estrogen therapy			
Yes	17.2	33.6	< 0.01
No	82.8	66.4	

[a]Wilcoxon-Mann-Whitney test for age, age at surgical menopause versus age at natural menopause; *t* test for age at menarche, chi-squared test for categorical variables.
[b]Categories displayed here for descriptive purposes; age included as a linear term in models.
[c]IQR, interquartile range.

Discussion

In a cohort of women aged 65 years and older at baseline with up to 24 years of follow-up, age at natural menopause was related to mortality. Women with older ages at natural menopause had higher rates of all-cause mortality than women with younger ages at natural menopause, while the oldest ages at natural menopause only were related to increased cardiovascular mortality. However, type of menopause and age at surgical menopause were not related to all-cause or cardiovascular mortality.

Comparisons with other studies

Our results for age at natural menopause contrast those of previous studies. Other studies with the most comparable age ranges (approximately ≥80 years of attained age) have found no relationship between age at natural menopause and mortality[8,13,14,22] or increased mortality risk among women with early menopause only.[1,7,8] However, the younger subsamples of 70–79 years[13] and a sample with a broader age range[12] showed a U-shaped relationship between age at natural menopause and risk for ischemic heart disease mortality.

Differences in the pattern of association between natural age at menopause and mortality between the Iowa EPESE and the results from previous cohorts may relate to variation in the distribution of attained age at death. The Iowa EPESE cohort had an older distribution of age at death than other cohorts. The median age at death in this cohort was 90 years. By contrast, only 24% of deaths occurred at attained age ≥85 years in a subset of women with attained age ≥80 years in another cohort.[13] Therefore, our sample may capture mortality of an older age group. Age differences may also explain why women with a later but not younger age at natural menopause had an elevated risk for mortality in the Iowa EPESE. Since previous studies have shown a relationship between early age at menopause and increased mortality among women with younger attained age,[6,8,14] this group of women with younger age at menopause in the Iowa EPESE may be more robust than the women with a similar age at menopause in younger age groups.[12] This survival difference may explain why we see an increase in mortality only for older age at menopause as opposed to a U-shaped relationship.

Our results for type of menopause and age at surgical menopause support some previous findings but conflict with others. Our finding of no relationship between type of menopause and mortality agree with two previous studies.[10,19] We did not find a relationship between age at surgical menopause and mortality, similar to some studies of women of a broader age range.[3,4] However, some other studies found a

Table 2. Mortality by Type of Menopause

	n	*Mortality rate*[a]	*Age-adjusted hazard ratio (95% CI)*	*Fully adjusted hazard ratio*[b] *(95% CI)*
All-cause mortality				
Surgical menopause	1247	66.54	1.01 (0.90, 1.13)	0.98 (0.87, 1.10)
Natural menopause	437	58.2	1.00	1.00
Cardiovascular mortality				
Surgical menopause	1231	35.51	0.92 (0.78, 1.09)	0.90 (0.76, 1.07)
Natural menopause	422	27.47	1.00	1.00

[a]Per 1000 person-years.
[b]Adjusts for age, educational attainment, number of pregnancies, age at menarche, smoking, height, weight, and use of estrogen therapy.

relationship between early age at oophorectomy and increased mortality,[10,14,20,21] One difference between the Iowa EPESE and the other cohorts is that we were unable to separate women who had oophorectomy from those who had hysterectomy only. The combination of these groups of women may mask an effect of early menopause on increased mortality only for women with oophorectomy. Another possibility is that although the age range in these studies was broad, if a majority of deaths related to early age at surgical menopause occurred early, these results may largely reflect those early deaths. Such early deaths may capture women who had high mortality before they would have been eligible to enter the Iowa EPESE at a minimum age of 65 years.

Mechanisms

One of the leading hypotheses proposed to explain the potential relationship between age at menopause and mortality concerns exposure to endogenous estrogen, which supports increased mortality among women with earlier ages at menopause. However, we found no association between age at surgical menopause and mortality and unexpectedly detected increased mortality among women with later ages at natural menopause. If estrogen offers protective effects against health conditions, it is possible that the strength of these effects diminishes as time since menopause elapses.[8,14] By age 65 years, the minimum age of entry to the EPESE, it is possible that any variability in endogenous estrogen exposure related to type and timing of surgical menopause was no longer affecting health. Another explanation is that women who survive to at least age 65 years may have a bias towards positive health characteristics, as women with early menopause have a more adverse profile in many aspects of health.[15–18] These characteristics may be unrelated to estrogen or may counter the effects of loss of estrogen. The biological mechanisms underlying the relationship between later age at natural menopause and increased mortality are unclear. One speculation is that a hormonal imbalance may delay menopause or encourage bleeding and increase risk of myocardial infarction.[12]

Methodological considerations

The strengths of this cohort include a long follow-up period, inclusion of women with surgical menopause, and assessment of relevant covariates. Although the sample is small, the high number of deaths during the follow-up provides the statistical power to examine the relationship between characteristics of menopause and mortality. Some previous studies have excluded women with hysterectomy or oophorectomy.[1,5,8,11,12] However, as women with surgical menopause account for 26% of women in the Iowa EPESE, we felt that it was important to include this substantial proportion of women in the analysis.

However, the study was limited in aspects of the scope of variables and design. Because women were required to recall age at menopause and reason for menopause, there may be some reporting inaccuracies because these events took place over 20 years in the past. Previous work has shown that there is a high level of accuracy when recalling age at surgical

Table 3. Mortality by Age at Natural Menopause

	n	*Mortality rate*[a]	*Age-adjusted hazard ratio (95% CI)*	*Fully adjusted hazard ratio*[b] *(95% CI)*	p *value*[c]
All-cause mortality (years)					
<45	163	72.69	0.89 (0.74, 1.07)	0.88 (0.73, 1.06)	0.03
45–49	319	64.45	0.95 (0.82, 1.10)	0.96 (0.83, 1.12)	
50–54	586	65.92	1.00	1.00	
≥55	179	66.98	1.12 (0.94, 1.34)	1.13 (0.95, 1.35)	
Cardiovascular mortality (years)					
<45	160	40.92	0.97 (0.76, 1.24)	0.96 (0.75, 1.23)	0.17
45–49	316	32.92	0.93 (0.76, 1.14)	0.95 (0.77, 1.16)	
50–54	576	33.92	1.00	1.00	
≥55	179	40.52	1.29 (1.02, 1.63)	1.30 (1.03, 1.65)	

[a]Per 1000 person-years.
[b]Adjusts for age, educational attainment, number of pregnancies, age at menarche, smoking, height, weight, and use of estrogen therapy.
[c]From test of linear trend for age at natural menopause.

Table 4. Mortality by Age at Surgical Menopause

	n	*Mortality rate*[a]	*Age-adjusted hazard ratio (95% CI)*	*Fully adjusted hazard ratio*[b] *(95% CI)*	p *value*[c]
All-cause mortality (years)					
<40	96	144.37	1.25 (0.92, 1.69)	1.22 (0.89, 1.67)	0.80
40–44	107	63.23	1.21 (0.90, 1.63)	1.24 (0.91, 1.68)	
45–49	96	19.38	1.00	1.00	
≥50	138	11.18	1.18 (0.88, 1.56)	1.19 (0.89, 1.59)	
Cardiovascular mortality (years)					
<40	93	73.05	1.25 (0.82, 1.92)	1.24 (0.80, 1.93)	0.28
40–44	102	28.62	1.16 (0.76, 1.78)	1.18 (0.76, 1.84)	
45–49	94	10.04	1.00	1.00	
≥50	113	4.8	0.98 (0.65, 1.48)	0.99 (0.65, 1.52)	

[a]Per 1,000 person-years.
[b]Adjusts for age, educational attainment, number of pregnancies, age at menarche, smoking, height, weight, and use of estrogen therapy.
[c]From test of linear trend for age at surgical menopause.

menopause and type of procedure.[27–32] However, recalled age at natural menopause has been shown to be less accurate.[27,30,33] Expected trends, such as a younger age at menopause for surgical menopause and greater use of hormone therapy in surgical than natural menopause, suggest that inaccurate reporting concerning type of menopause and age at menopause is not likely to strongly bias our results.

No details about type of surgery were provided, which would have allowed us to further examine whether early age at oophorectomy but not hysterectomy alone was related to increased mortality, as previous studies have shown.[14,20,21] Based on national estimates, we expect that between 25% and 41% of women had oophorectomy with hysterectomy, which were the estimates in 1965 and 1984. Most women in the Iowa EPESE would have experienced surgical menopause during this time interval.[34] While bilateral oophorectomy results in menopause, hysterectomy alone may accelerate the onset of menopause.[35,36] Therefore, age at surgical menopause may represent an approximate age for up to 60% of women with surgical menopause who we expect had hysterectomy only. This study cannot test associations of type and timing of menopause with mortality closer to menopause because women had to have survived to at least age 65 years to be included in the EPESE. For example, some previous studies have demonstrated particularly high mortality within 20 years of surgery for women who experienced surgical menopause prior to age 45 involving bilateral oophorectomy.[10,14,20,21] If this trend occurred in the population of women from which the Iowa EPESE was sampled, women who experienced increased mortality risk before 65 years due to early oophorectomy will have been excluded from the Iowa EPESE. While some women were excluded for lack of information on type and age at menopause due to the retrospective nature of the study, the hazard ratio and percentage dead by the end of follow-up showed that all-cause mortality for women who did and did not have information and type or age at menopause was similar, suggesting that sample selection should not substantially bias results.[4]

These results may not be generalizable to younger cohorts or more geographically diverse groups of women. At the time that women of the Iowa EPESE underwent surgical menopause, hysterectomy was used for some gynecological conditions that would now be treated with less invasive techniques, including hysteroscopic surgery and uterine artery fibroid embolization. Screening programs have also become more frequent. These advances have contributed to falling rates of hysterectomy in the United States in the latter part of the 20th century.[37–41] Study members with surgical menopause in our study were hence likely to be a more heterogeneous group than contemporary women with surgical menopause in terms of reasons for hysterectomy, whereby it is possible that the relationship of type of menopause with mortality may be different.[38,40–43] Rates of oophorectomy have also fluctuated over time.[39,40] Another limitation to the generalizability of results is that the members of the Iowa EPESE lived in rural counties. Older women living in urban areas may have different risk profiles than women in rural areas due to environmental and cultural differences.[44] We were unable to include women from the other thee EPESE sites in the analysis because the questionnaires from the other EPESE sites did not include information on menopause. The ability to utilize all four sites would have resulted in a sample that was more diverse in terms of race and socioeconomic position. However, the Iowa EPESE provides a unique opportunity to examine mortality among the oldest women. Our results suggest that later age at menopause is a risk factor for increased mortality in this rapidly growing segment of the population.

Conclusion

Later age at natural menopause was related to increased rates of all-cause and cardiovascular mortality in later life. Monitoring the cardiovascular health of this group of older women who experience natural menopause at later ages may contribute to improved survival.

Acknowledgments

The authors thank Joanne Calabro and Caroline Phillips for assistance with data preparation and Binbing Yu for statistical consulting. This study was supported in part by the Intramural Research Program, National Institute on Aging, National Institutes of Health. Dr. Cooper is funded by the New Dynamics of Ageing (RES-353-25-0001). Dr. Tom, a University of Texas Medical Branch Building Interdisciplinary Research Careers in Women's Health (BIRCWH) Scholar, is supported by a research career development award (K12HD052023, PI: Berenson), that is co-funded by the Eunice

Kennedy Shriver National Institute of Child Health & Human Development, the Office of Research on Women's Health, and the National Institute of Allergy & Infectious Diseases. The content is solely the responsibility of the authors and does not necessarily represent the official views of the Eunice Kennedy Shriver National Institute of Child Health & Human Development or the National Institutes of Health.

Disclosure Statement

No competing financial interests exist.

References

1. Mondul AM, Rodriguez C, Jacobs EJ, Calle EE. Age at natural menopause and cause-specific mortality. Am J Epidemiol 2005;162:1089–1097.
2. Gruber CJ, Tschugguel W, Schneeberger C, Huber JC. Production and actions of estrogens. N Engl J Med 2002;346: 340–352.
3. Amagai Y, Ishikawa S, Gotoh T, Kayaba K, Nakamura Y, Kajii E. Age at menopause and mortality in Japan: the Jichi Medical School Cohort Study. J Epidemiol 2006;16:161–166.
4. Cooper GS, Baird DD, Weinberg CR, Ephross SA, Sandler DP. Age at menopause and childbearing patterns in relation to mortality. Am J Epidemiol 2000;151:620–623.
5. Cooper GS, Sandler DP. Age at natural menopause and mortality. Ann Epidemiol 1998;8:229–235.
6. Cui R, Iso H, Toyoshima H, et al. Relationships of age at menarche and menopause, and reproductive year with mortality from cardiovascular disease in Japanese postmenopausal women: the JACC study. J Epidemiol 2006;16:177–184.
7. Hong JS, Yi SW, Kang HC, et al. Age at menopause and cause-specific mortality in South Korean women: Kangwha Cohort Study. Maturitas 2007;56:411–419.
8. Jacobsen BK, Heuch I, Kvåle G. Age at natural menopause and all-cause mortality: a 37-year follow-up of 19,731 Norwegian women. Am J Epidemiol 2003;157:923–929.
9. Jansen SC, Temme EH, Schouten EG. Lifetime estrogen exposure versus age at menopause as mortality predictor. Maturitas 2002;43:105–112.
10. Ossewaarde ME, Bots ML, Verbeek AL, et al. Age at menopause, cause-specific mortality and total life expectancy. Epidemiology 2005;16:556–562.
11. Snowdon DA, Kane RL, Beeson WL, et al. Is early natural menopause a biologic marker of health and aging? Am J Public Health 1989;79:709–714.
12. Jacobsen BK, Knutsen SF, Fraser GE. Age at natural menopause and total mortality and mortality from ischemic heart disease: the Adventist Health Study. J Clin Epidemiol 1999;52:303–307.
13. Jacobsen BK, Nilssen S, Heuch I, Kvåle G. Does age at natural menopause affect mortality from ischemic heart disease? J Clin Epidemiol 1997;50:475–479.
14. van der Schouw YT, van der Graaf Y, Steyerberg EW, Eijkemans JC, Banga JD. Age at menopause as a risk factor for cardiovascular mortality. Lancet 1996;347:714–718.
15. Brett KM. Can hysterectomy be considered a risk factor for cardiovascular disease? Circulation 2005;111:1456–1458.
16. Cooper R, Kuh D, Hardy R, Power C. Is there an association between hysterectomy and subsequent adiposity? Maturitas 2007;58:296–307.
17. Cooper R, Lawlor DA, Hardy R, et al. Socio-economic position across the life course and hysterectomy in three British cohorts: a cross-cohort comparative study. BJOG 2005;112: 1126–1133.
18. Cooper R, Mishra G, Hardy R, Kuh D. Hysterectomy and subsequent psychological health: findings from a British birth cohort study. J Affect Disord 2009;115:122–130.
19. Iversen L, Hannaford PC, Elliott AM, Lee AJ. Long term effects of hysterectomy on mortality: nested cohort study. BMJ 2005;330:1482.
20. Rivera CM, Grossardt BR, Rhodes DJ, et al. Increased cardiovascular mortality after early bilateral oophorectomy. Menopause 2009;16:15–23.
21. Rocca WA, Grossardt BR, de Andrade M, Malkasian GD, Melton LJ. Survival patterns after oophorectomy in premenopausal women: a population-based cohort study. Lancet Oncol 2006;7:821–828.
22. Jacobsen BK, Heuch I, Kvåle G. On mortality from ischemic heart disease in women with very late menopause. J Clin Epidemiol 2000;53:435–436.
23. Cornoni-Huntley J, Ostfeld AM, Taylor JO, et al. Established populations for epidemiologic studies of the elderly: study design and methodology. Aging (Milano) 1993;5:27–37.
24. World Health Organization. International Classification of Disease, Ninth Revision. Geneva: World Health Organization, 1980.
25. Farewell V. An application of Cox's proportional hazards model to multiple infection data. J R Stat Soc Ser C: Appl Stat 1979;28:136–143.
26. Prentice RL, Kalbfleisch JD, Peterson AV, Flournoy N, Farewell VT, Breslow NE. The analysis of failure times in the presence of competing risks. Biometrics 1978;34:541–554.
27. Bean JA, Leeper JD, Wallace RB, Sherman BM, Jagger H. Variations in the reporting of menstrual histories. Am J Epidemiol 1979;109:181–185.
28. Brett KM, Madans JH. Hysterectomy use: the correspondence between self-reports and hospital records. Am J Public Health 1994;84:1653–1655.
29. Coulter A, McPherson K, Elliott S, Whiting B. Accuracy of recall of surgical histories: a comparison of postal survey data and general practice records. Community Med 1985;7: 186–189.
30. Hahn RA, Eaker E, Rolka H. Reliability of reported age at menopause. Am J Epidemiol 1997;146:771–775.
31. Harlow SD, Linet MS. Agreement between questionnaire data and medical records. The evidence for accuracy of recall. Am J Epidemiol 1989;129:233–248.
32. Treloar SA, Do KA, O'Connor VM, O'Connor DT, Yeo MA, Martin NG. Predictors of hysterectomy: an Australian study. Am J Obstet Gynecol 1999;180:945–954.
33. Colditz GA, Stampfer MJ, Willett WC, et al. Reproducibility and validity of self-reported menopausal status in a prospective cohort study. Am J Epidemiol 1987;126:319–325.
34. Pokras R, Hufnagel VG. Hysterectomy in the United States, 1965–84. Am J Public Health 1988;78:852–853.
35. Farquhar CM, Sadler L, Harvey SA, Stewart AW. The association of hysterectomy and menopause: a prospective cohort study. BJOG 2005;112:956–962.
36. Cooper GS, Thorp JM. FSH levels in relation to hysterectomy and to unilateral oophorectomy. Obstet Gynecol 1999;94: 969–972.
37. Babalola EO, Bharucha AE, Schleck CD, Gebhart JB, Zinsmeister AR, Melton LJ. Decreasing utilization of hysterectomy: a population-based study in Olmsted County, Minnesota, 1965–2002. Am J Obstet Gynecol 2007;196: 214.e211–217.

38. Baskett TF. Hysterectomy: evolution and trends. Best Pract Res Clin Obstet Gynaecol 2005;19:295–305.
39. Keshavarz H, Hillis SD, Kieke BA, Marchbanks PA. Hysterectomy surveillance—United States, 1994–1999. MMWR Surveill Summ 2002;51:1–8.
40. Lepine LA, Hillis SD, Marchbanks PA, et al. Hysterectomy surveillance—United States, 1980–1993. MMWR CDC Surveill Summ 1997;46:1–15.
41. Sattin RW, Rubin GL, Hughes JM. Hysterectomy among women of reproductive age, United States, update for 1979–1980. MMWR CDC Surveill Summ 1983;32:1SS–7SS.
42. Brieger GH , Jones HW Jr. Operative gynecology before the era of laparoscopy: a brief history. In: Rock JA, Jones HW, eds. Te Linde's Operative Gynecology 2008. Philadelphia: Wolters Kluwer Health/Lippincott Williams and Wilkins, 2008:1–14.
43. Jones HW III. Abdominal hysterectomy. In: Rock JA, Jones HW, III, eds. Te Linde's Operative Gynecology 2008. Philadelphia: Wolters Kluwer Health/Lippincott Williams and Wikins, 2008:727–743.
44. Hartley D. Rural health disparities, population health, and rural culture. Am J Public Health 2004;94:1675–1678.

Address correspondence to:
Sarah E. Tom, Ph.D.
Department of Preventive Medicine and Community Health
University of Texas Medical Branch
301 University Boulevard
Galveston, TX 77555

E-mail: setom@utmb.edu