



Original Contribution

Is Alcohol Consumption Associated With Risk of Early Menopause?

Joshua R. Freeman, Brian W. Whitcomb, Alexandra C. Purdue-Smithe, JoAnn E. Manson, Christine R. Langton, Susan E. Hankinson, Bernard A. Rosner, and Elizabeth R. Bertone-Johnson*

* Correspondence to Dr. Elizabeth R. Bertone-Johnson, Department of Biostatistics and Epidemiology, Department of Health Promotion and Policy, School of Public Health and Health Sciences, University of Massachusetts Amherst, 715 North Pleasant Street, Amherst, MA 01003 (e-mail: ebertone@schoolph.umass.edu)

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Earlier age at menopause is associated with increased long-term health risks. Moderate alcohol intake has been suggested to delay menopause onset, but it is unknown whether alcohol subtypes are associated with early menopause onset at age 45 years. Therefore, we aimed to evaluate risk of early natural menopause among 107,817 members of the Nurses' Health Study II who were followed from 1989 to 2011. Alcohol consumption overall and by subtypes, including beer, red wine, white wine, and liquor, was assessed throughout follow-up. We estimated hazard ratios in multivariable models that were adjusted for age, body mass index, parity, smoking, and other potential confounders. Women who reported moderate current alcohol consumption had lower risks of early menopause than did nondrinkers. Those who reported consuming 10.0–14.9 g/day had a lower risk of early menopause than did nondrinkers (hazard ratio = 0.81, 95% confidence interval: 0.68, 0.97). Among specific beverages, evidence of lower early menopause risk was confined to consumption of white wine and potentially red wine and liquor, but not to beer. Data from this large prospective study suggest a weak association of moderate alcohol intake with lower risk of early menopause, which was most pronounced for consumption of white and red wine and liquor. High consumption was not related to lower risk of early menopause.

alcohol; early menopause; endocrine system; wine

Abbreviations: CI, confidence interval; HT, hormone therapy; NHS2, Nurses' Health Study II.

Early natural menopause, which is cessation of ovarian function prior to age 45 years, affects approximately 10% of Western women (1, 2). Early menopause is associated with adverse outcomes, including cardiovascular disease, osteoporosis, and mortality (3–5). The etiology of early menopause is not fully understood, but behavioral and dietary factors are thought to be involved (2, 6, 7). Alcohol is one such factor. Alcohol consumption may increase circulating estrogen levels and reduce oxidative stress in ovarian tissue, thus delaying menopause (8–12). Research on alcohol intake to date has focused primarily on menopausal timing (13–31). Two cross-sectional studies did not find an association between alcohol intake and risk of early menopause, but they were limited by retrospective recall of alcohol intake (32, 33).

Evidence from a meta-analysis suggested that low-to-moderate alcohol intake is associated with later menopause onset compared with not drinking (8). Although the level of alcohol consumption may contribute to a later age at menopause, it remains unclear whether associations vary by alcohol type (8, 32–34). Specifically, red wine may be related to lower risk, as evidenced by animal studies in which it was suggested that resveratrol in red wine confers ovarian protective effects (11, 12).

To address these questions, we evaluated the associations of prospectively measured total alcohol intake and alcoholic beverage-specific consumption with incident early menopause within the Nurses' Health Study II (NHS2) cohort. We hypothesized that both moderate alcohol intake (defined as ≤ 1 standard drink/day for women) and moderate

red wine consumption would be associated with a lower risk of early menopause (35).

METHODS

The NHS2 is a prospective cohort study of 116,429 female nurses aged 25–42 years who were recruited at baseline in 1989 via a mailed questionnaire. Return of questionnaires was considered implied consent. Study details including recruitment have been published previously (36, 37). Briefly, data on participant demographic characteristics, lifestyle behaviors, medical history, reproductive factors, and prospective outcomes were collected via biennial questionnaires, with a follow-up rate $\geq 89\%$ per questionnaire cycle (7). The study protocol was approved by the institutional review boards of the Brigham and Women's Hospital and the Harvard T.H. Chan School of Public Health.

Assessment of alcohol consumption

At baseline (1989), participants were queried regarding their average overall alcohol consumption over the past year. Intake response options ranged from “none or <1 drink/month” to “40+ drinks/week.” From 1991 onward, alcohol intake was assessed every 4 years using semiquantitative food frequency questionnaires (38). The specific beverages of which intakes were measured included regular beer, light beer, white wine, red wine, and liquor. Response options ranged from “never or <1/month” to “6+/day.” Total alcohol intake (grams) was calculated by multiplying the frequency of each beverage intake by its average alcohol content (beer = 12.8 g/drink; wine = 11.0 g/drink; liquor = 14.0 g/drink) and summing across all beverages (38). This allowed us to account for differences in alcohol content across beverages.

Assessment of early menopause

From baseline onward, each NHS2 questionnaire asked women if their menstrual periods had permanently ceased and if so, the age at cessation and whether periods ceased due to surgery, radiation, or chemotherapy or had stopped naturally (7). Women were also asked about their current and past use of menopausal hormone therapy (HT). Age at natural menopause was defined as age after 12 consecutive months of amenorrhea not due to radiation, chemotherapy, or surgical removal of the ovaries. Few women reported being postmenopausal on one questionnaire and then subsequently reported being premenopausal. For these women, age at menopause was defined as age after which periods were absent for 12 months or more and the status persisted for at least 3 consecutive questionnaires. The present analysis was limited to women who were premenopausal at baseline and provided information on alcohol consumption ($n = 107,817$). We defined early natural menopause as natural menopause before age 45 years (7).

Assessment of covariates

Factors self-reported at baseline included age, height, weight, age at menarche, and race/ethnicity. Data on participant weight, number of full-term pregnancies, breastfeeding duration, age at first birth, oral contraceptive use, HT use, and smoking history were collected at baseline and then biennially (7).

Statistical analyses

Continuous variables were examined for normality. We divided women into categories of grams per day of total alcohol intake. We compared baseline continuous and categorical characteristics of participants by baseline alcohol intake with adjustment for age. We then used Cox proportional hazards models to estimate hazard ratios and 95% confidence intervals for associations of alcohol intake with early natural menopause. For these models, women were followed from baseline until early natural menopause, hysterectomy, oophorectomy, cancer diagnosis (excluding nonmelanoma skin cancer), death, loss-to-follow-up, or age 45 years.

Alcohol was modeled using 3 approaches: 1) baseline consumption (1989) to examine baseline associations between alcohol consumption and risk of early menopause; 2) current consumption (assessed every 4 years) to address changes in alcohol intake over time; and 3) cumulative average consumption to address random error that might occur with use of a single timepoint measure (39). We also evaluated risk of early natural menopause using updated beer, light beer, red wine, white wine, and liquor intakes from the 1991 food frequency questionnaires onward. Beverage-specific models used current consumption with adjustment for confounding (model 1) and mutual adjustment of other beverages (model 2).

In multivariable models, we adjusted for current age, age at menarche, body mass index, parity, smoking duration and status, duration of oral contraceptive use and status, and breastfeeding duration as a priori potential confounders. Additionally, we mutually adjusted for alcohol subtypes to control for other beverages. We also performed 2 sensitivity analyses. First, we restricted to never smokers to address potential residual confounding due to smoking. Second, to evaluate whether HT use influenced associations, we additionally adjusted for HT use and separately censored at first report of HT use. Statistical analyses were conducted using SAS, version 9.3 (SAS Institute, Inc., Cary, North Carolina).

RESULTS

Age-adjusted participant characteristics at baseline are presented in Web Table 1 (available at <https://doi.org/10.1093/aje/kwab182>). Alcohol use was positively associated with smoking and duration of oral contraceptive use and inversely associated with parity and breastfeeding duration. Body mass index did not vary substantially by alcohol intake.

A total of 2,761 early natural menopause cases occurred over approximately 1.5 million person-years of follow-up (Table 1). Baseline alcohol consumption was not related to

Table 1. Hazard Ratios for Early Natural Menopause by Baseline, Current, and Cumulative Average Alcohol Intakes, Nurses' Health Study II, 1989–2011

Time Period and Alcohol Intake, g/day	Age-Adjusted		Model 1 ^a	
	HR	95% CI	HR	95% CI
Baseline (1989)				
Nondrinker	1.00	Referent	1.00	Referent
0.1–4.9	1.02	0.94, 1.11	0.98	0.90, 1.07
5.0–9.9	0.99	0.87, 1.13	0.90	0.79, 1.03
10.0–14.9	1.17	1.00, 1.36	0.99	0.85, 1.16
15.0–29.9	1.09	0.85, 1.41	0.90	0.70, 1.17
≥30.0	1.81	1.29, 2.54	1.31	0.93, 1.84
Current (updated)				
Nondrinker	1.00	Referent	1.00	Referent
0.1–4.9	1.04	0.95, 1.13	1.00	0.92, 1.09
5.0–9.9	1.02	0.89, 1.15	0.93	0.82, 1.06
10.0–14.9	0.94	0.79, 1.11	0.81	0.68, 0.97
15.0–29.9	1.22	0.99, 1.49	1.03	0.84, 1.26
≥30.0	1.20	0.87, 1.65	0.88	0.64, 1.22
Cumulative average				
Nondrinker	1.00	Referent	1.00	Referent
0.1–4.9	1.04	0.96, 1.14	0.99	0.90, 1.08
5.0–9.9	1.07	0.94, 1.21	0.95	0.83, 1.08
10.0–14.9	1.01	0.84, 1.21	0.84	0.70, 1.01
15.0–29.9	1.10	0.87, 1.38	0.87	0.69, 1.10
≥30.0	1.86	1.30, 2.64	1.29	0.90, 1.85

Abbreviations: CI, confidence interval; HR, hazard ratio.

^a Model 1 was adjusted for age (months; continuous), age at menarche (years; continuous), body mass index (weight (kg)/height (m)²; <18.5, 18.5–24.9, 25.0–29.9, or ≥ 30.0), parity (nulliparous, 1–2 children, or ≥3 children), breastfeeding duration (months; ordinal), pack-years of smoking by status (never smoked, past smoker 1–10 years, past smoker 11–20 years, past smoker ≥20 years, current smoker 1–10 years, current smoker 11–20 years, or current smoker ≥20 years), oral contraceptive use (never, past, or current), and duration of oral contraceptive use (months; never, 1–23, 24–47, 48–71, 72–95, 96–119, or ≥120).

risk of early menopause. In fully adjusted models of current alcohol intake, lower risk was observed among women with a moderate intake than among nondrinkers; women who consumed 10.0–14.9 g/day had a hazard ratio of 0.81 (95% confidence interval (CI): 0.68, 0.97). In fully adjusted models of cumulative average intake, similar estimates were observed for women with moderate intakes, though associations were not statistically significant (for women who drank 10.0–14.9 g/day vs. nondrinkers, hazard ratio = 0.84, 95% CI: 0.70, 1.01). Restricting to nonsmokers, adjusting for HT use, and censoring on first HT use in sensitivity analyses did not meaningfully impact estimates (data not shown).

White wine consumption was significantly associated with lower risk of early natural menopause in models that controlled for confounding and other beverage subtypes (Table 2). Red wine and liquor consumption were marginally associated with lower risk of early natural menopause in both models. Beer and light beer were not associated with risk of early natural menopause.

DISCUSSION

In this large, prospective study, moderate alcohol intake was associated with lower risk of early menopause. Magnitudes of association were similar when considering recent and long-term average consumption to account for variation in consumption trends. Our work suggests that white wine intake and potentially red wine and liquor intakes were associated with small reductions in risk of early menopause in models adjusted for confounding, as well as mutually adjusted for other alcoholic beverages. Together with the existing literature, our results suggest that moderate alcohol consumption, specifically wine and liquor, is relevant for lower early menopause risk.

To our knowledge, the present study is among the largest prospective studies to date of alcohol intake and age at menopause and one of the few in which risk of early natural menopause risk was assessed rather than menopausal timing (13–31). In a retrospective study of 2,123 postmenopausal

Table 2. Hazard Ratios for Early Natural Menopause by Current Frequency of Intake of Specific Alcoholic Beverages, Nurses' Health Study II, 1991–2011

Type of Alcohol and Consumption Frequency	Age-Adjusted		Model 1 ^a		Model 2 ^b	
	HR	95% CI	HR	95% CI	HR	95% CI
Regular beer						
<1 per month	1.00	Referent	1.00	Referent	1.00	Referent
1 per month to 1 per week	1.06	0.95, 1.18	1.03	0.92, 1.15	1.04	0.93, 1.16
>1 per week	1.02	0.84, 1.25	0.93	0.77, 1.14	0.94	0.77, 1.15
Light beer						
<1 per month	1.00	Referent	1.00	Referent	1.00	Referent
1 per month to 1 per week	1.02	0.92, 1.13	0.99	0.89, 1.10	1.00	0.90, 1.11
>1 per week	1.18	1.01, 1.38	1.04	0.89, 1.21	1.05	0.90, 1.23
Red wine						
<1 per month	1.00	Referent	1.00	Referent	1.00	Referent
1 per month to 1 per week	0.92	0.83, 1.02	0.91	0.82, 1.01	0.92	0.82, 1.02
>1 per week	0.91	0.75, 1.09	0.85	0.70, 1.03	0.86	0.71, 1.04
White wine						
<1 per month	1.00	Referent	1.00	Referent	1.00	Referent
1 per month to 1 per week	0.99	0.90, 1.07	0.98	0.89, 1.07	0.98	0.89, 1.07
>1 per week	0.91	0.79, 1.05	0.86	0.74, 0.99	0.86	0.73, 1.00
Liquor						
<1 per month	1.00	Referent	1.00	Referent	1.00	Referent
1 per month to 1 per week	1.01	0.91, 1.12	0.95	0.86, 1.06	0.96	0.86, 1.06
>1 per week	1.01	0.82, 1.23	0.85	0.69, 1.04	0.86	0.70, 1.05

Abbreviations: CI, confidence interval; HR, hazard ratio.

^a Model 1 was adjusted for age (months; continuous), age at menarche (years; continuous), parity (nulliparous, 1–2 children, or ≥ 3 children), breastfeeding duration (months; ordinal), body mass index (weight (kg)/height (m)²; <18.5, 18.5–24.9, 25.0–29.9, or ≥ 30.0), pack-years of smoking by status (never smoked, past smoker 1–10 years, past smoker 11–20 years, past smoker ≥ 20 years, current smoker 1–10 years, current smoker 11–20 years, or current smoker ≥ 20 years), oral contraceptive use (never, past, or current), and duration of oral contraceptive use (months; never, 1–23, 24–47, 48–71, 72–95, 96–119, or ≥ 120).

^b Model 2 was adjusted for model 1 covariates and mutually adjusted for other alcoholic beverage types (median g/day; continuous).

members of the Oslo Health Study, odds of natural menopause between ages 40–44 years ($n \approx 200$) were lower in women who reported weekly alcohol intake than in nondrinkers (odds ratio = 0.66, 95% CI: 0.37, 1.18), but the statistical power was limited by the few cases (32). A similar result was observed in a retrospective study of postmenopausal Korean women ($n = 1,599$) that included 261 early natural menopause cases. Ever drinkers had lower likelihood of early menopause (odds ratio = 0.81, 95% CI: 0.57, 1.15) compared with never drinkers; however, results were not adjusted for smoking (33).

Our findings are consistent with literature considering alcohol intake and age at menopause. In a 2016 meta-analysis, researchers observed later age at menopause related to alcohol intake, reporting a summary relative risk for early menopause onset in drinkers versus nondrinkers of 0.86 (95% CI: 0.78, 0.96) in 7 cross-sectional studies and of 0.94 (95% CI: 0.91, 0.97) in 6 prospective studies (8). Among prospective studies, alcohol intake amount was

related to later menopause onset as well; the relative risk for those the highest intake category (> 16 g/day) compared to nondrinkers was 0.89 (95% CI: 0.86, 0.92). More recently, Costanian et al. (40) reported an adjusted hazard ratio of 0.89 for menopause onset in weekly drinkers versus never drinkers (95% CI: 0.81, 0.98) in a cross-sectional analysis from the Canadian Longitudinal Study on Aging.

A strength of our analysis was evaluation of alcoholic beverage-specific associations with early menopause risk while mutually adjusting for other beverage types. Our findings for beer and red wine are consistent with those reported in a prospective study of perimenopause onset among 502 women aged 36–45 years in which the authors reported a null association for beer and an imprecise signal with red wine intakes as low as 1–3 glasses/month (hazard ratio = 0.58, 95% CI: 0.33, 1.03) up to ≥ 2 glasses/week (hazard ratio = 0.67, 95% CI: 0.34, 1.31) (34). In contrast to our findings, this study reported null associations for white wine and liquor intake. However, this study used only baseline

assessments of past year alcoholic beverage intake, which may be more prone to non-differential misclassification in comparison to the cumulatively averaged beverage intakes used in our analyses of longitudinally collected data (39).

The health benefits of wine and liquor may be driven by ethanol and antioxidants. Ethanol has estrogenic effects, and studies of animal models and humans suggest ethanol may modulate aromatase conversion of androgens to estrogen (9). Health benefits of wine and particularly red wine may be attributable to antioxidants in grapes, such as resveratrol (34). Studies in rats have demonstrated resveratrol has many ovarian protective effects, such as reducing follicular atresia and mitigating oxidative stress (11, 12).

Strengths of our study include its prospective design and robust follow-up, which reduced the potential for selection bias. The study has a large sample size with longitudinal measurement of alcohol and alcohol-specific beverage types. The NHS2 is also a homogeneous sample of mostly White women in nursing with a narrow range in socioeconomic position and similar educational ascertainment, which by design enhances the internal validity of the findings. However, some limitations are important to consider when interpreting the findings. Alcohol consumption was self-reported, which is prone to error and could lead to exposure misclassification. Misclassification is likely independent of incident early natural menopause, which could have affected assessment of dose-response patterns unpredictably. To address this possibility, we used cumulative averages, which have been used to reduce the effects of this type of misclassification (39). Despite the large sample size, few participants reported consuming more than 2 alcoholic beverages per day, limiting our statistical power to evaluate associations at higher intakes. Because of multiple comparisons, we may have observed associations due to chance alone, and thus caution is warranted in interpreting our findings. Lastly, 63% of our sample consumed alcohol, and thus our findings may have limited generalizability to populations with different alcohol consumption levels.

Findings of this large prospective study suggest that moderate alcohol consumption, particularly of white wine, is associated with modestly lower risk of early natural menopause. Prior studies have been unable to evaluate alcoholic beverage subtypes, and our analysis suggests this association may be explained by consumption of white and red wine and liquor. Nevertheless, high alcohol intakes, regardless of type, were not associated with lower risk of early natural menopause.

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Author affiliations: Department of Biostatistics and Epidemiology, School of Public Health and Health Sciences, University of Massachusetts Amherst, Amherst, Massachusetts, United States (Joshua R. Freeman, Brian W. Whitcomb, Christine R. Langton, Susan E. Hankinson, Elizabeth R. Bertone-Johnson); Epidemiology Branch, Division of Intramural Population Health Research, Eunice

Kennedy Shriver National Institute of Child Health and Human Development, Bethesda, Maryland, United States (Joshua R. Freeman, Alexandra C. Purdue-Smithe); Channing Division of Network Medicine, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts, United States (JoAnn E. Manson, Susan E. Hankinson, Bernard A. Rosner); Department of Epidemiology, Harvard T.H. Chan School of Public Health, Boston, Massachusetts, United States (JoAnn E. Manson); Division of Preventive Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts, United States (JoAnn E. Manson); Department of Biostatistics, Harvard T.H. Chan School of Public Health, Boston, Massachusetts, United States (Bernard A. Rosner); and Department of Health Promotion and Policy, School of Public Health and Health Sciences, University of Massachusetts Amherst, Amherst, Massachusetts, United States (Elizabeth R. Bertone-Johnson).

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REFERENCES

1. Coulam CB, Adamson SC, Annegers JF. Incidence of premature ovarian failure. *Obstet Gynecol.* 1986;67(4):604–606.
2. Shuster LT, Rhodes DJ, Gostout BS, et al. Premature menopause or early menopause: long-term health consequences. *Maturitas.* 2010;65(2):161–166.
3. Muka T, Oliver-Williams C, Kunutsor S, et al. Association of age at onset of menopause and time since onset of menopause with cardiovascular outcomes, intermediate vascular traits, and all-cause mortality: a systematic review and meta-analysis. *JAMA Cardiol.* 2016;1(7):767–776.
4. Gallagher JC. Effect of early menopause on bone mineral density and fractures. *Menopause.* 2007;14(3 pt 2):567–571.
5. Asllanaj E, Bano A, Glisic M, et al. Age at natural menopause and life expectancy with and without type 2 diabetes. *Menopause.* 2019;26(4):387–394.
6. Boutot ME, Purdue-Smithe A, Whitcomb BW, et al. Dietary protein intake and early menopause in the Nurses' Health Study II. *Am J Epidemiol.* 2018;187(2):270–277.
7. Purdue-Smithe AC, Whitcomb BW, Manson JE, et al. A prospective study of dairy-food intake and early menopause. *Am J Epidemiol.* 2019;188(1):188–196.
8. Taneri PE, Kiefe-de Jong JC, Bramer WM, et al. Association of alcohol consumption with the onset of natural menopause:

- a systematic review and meta-analysis. *Hum Reprod Update*. 2016;22(4):516–528.
9. Purohit V. Can alcohol promote aromatization of androgens to estrogens? A review. *Alcohol*. 2000;22(3):123–127.
 10. Eskew A, Bligard K, Broughton DE, et al. Does alcohol intake impact ovarian reserve? *Fertil Steril*. 2017;108(3):e258.
 11. Özcan P, Fişciyoğlu C, Yildirim OK, et al. Protective effect of resveratrol against oxidative damage to ovarian reserve in female Sprague-Dawley rats. *Reprod Biomed Online*. 2015;31(3):404–410.
 12. Chinwe GS, Azuka OI, Aadaeze NC. Resveratrol supplementation rescues pool of growing follicles and ovarian stroma from cisplatin-induced toxicity on the ovary in Sprague-Dawley rats: an experimental study. *Int J Reprod Biomed (Yazd)*. 2018;16(1):19–30.
 13. Celentano E, Galasso R, Berrino F, et al. Correlates of age at natural menopause in the cohorts of EPIC-Italy. *Tumori*. 2003;89(6):608–614.
 14. Dorjgochoo T, Kallianpur A, Gao YT, et al. Dietary and lifestyle predictors of age at natural menopause and reproductive span in the Shanghai Women's Health Study. *Menopause*. 2008;15(5):924–933.
 15. Gold EB, Crawford SL, Avis NE, et al. Factors related to age at natural menopause: longitudinal analyses from SWAN. *Am J Epidemiol*. 2013;178(1):70–83.
 16. Kinney A, Kline J, Levin B. Alcohol, caffeine and smoking in relation to age at menopause. *Maturitas*. 2006;54(1):27–38.
 17. Morris DH, Jones ME, Schoemaker MJ, et al. Body mass index, exercise, and other lifestyle factors in relation to age at natural menopause: analyses from the Breakthrough Generations Study. *Am J Epidemiol*. 2012;175(10):998–1005.
 18. Nagata C, Wada K, Nakamura K, et al. Associations of physical activity and diet with the onset of menopause in Japanese women. *Menopause*. 2012;19(1):75–81.
 19. Nagel G, Altenburg HP, Nieters A, et al. Reproductive and dietary determinants of the age at menopause in EPIC-Heidelberg. *Maturitas*. 2005;52(3–4):337–347.
 20. Sammel MD, Freeman EW, Liu Z, et al. Factors that influence entry into stages of the menopausal transition. *Menopause*. 2009;16(6):1218–1227.
 21. Santoro N, Brockwell S, Johnston J, et al. Helping midlife women predict the onset of the final menses: SWAN, the Study of Women's Health Across the Nation. *Menopause*. 2007;14(3 pt 1):415–424.
 22. Torgerson DJ, Thomas RE, Campbell MK, et al. Alcohol consumption and age of maternal menopause are associated with menopause onset. *Maturitas*. 1997;26(1):21–25.
 23. Bernis C, Reher DS. Environmental contexts of menopause in Spain: comparative results from recent research. *Menopause*. 2007;14(4):777–787.
 24. Brett KM, Cooper GS. Associations with menopause and menopausal transition in a nationally representative US sample. *Maturitas*. 2003;45(2):89–97.
 25. Cooper GS, Baird DD, Darden FR. Measures of menopausal status in relation to demographic, reproductive, and behavioral characteristics in a population-based study of women aged 35–49 years. *Am J Epidemiol*. 2001;153(12):1159–1165.
 26. Kaczmarek M. The timing of natural menopause in Poland and associated factors. *Maturitas*. 2007;57(2):139–153.
 27. Nagata C, Takatsuka N, Inaba S, et al. Association of diet and other lifestyle with onset of menopause in Japanese women. *Maturitas*. 1998;29(2):105–113.
 28. Neslihan Carda S, Bilge SA, Oztürk TN, et al. The menopausal age, related factors and climacteric symptoms in Turkish women. *Maturitas*. 1998;30(1):37–40.
 29. Pakarinen M, Raitanen J, Kaaja R, et al. Secular trend in the menopausal age in Finland 1997–2007 and correlation with socioeconomic, reproductive and lifestyle factors. *Maturitas*. 2010;66(4):417–422.
 30. Stepaniak U, Szafranec K, Kubinova R, et al. Age at natural menopause in three central and eastern European urban populations: the HAPIEE Study. *Maturitas*. 2013;75(1):87–93.
 31. Torgerson DJ, Avenell A, Russell IT, et al. Factors associated with onset of menopause in women aged 45–49. *Maturitas*. 1994;19(2):83–92.
 32. Mikkelsen TF, Graff-Iversen S, Sundby J, et al. Early menopause, association with tobacco smoking, coffee consumption and other lifestyle factors: a cross-sectional study. *BMC Public Health*. 2007;7:149.
 33. Chang SH, Kim CS, Lee KS, et al. Premenopausal factors influencing premature ovarian failure and early menopause. *Maturitas*. 2007;58(1):19–30.
 34. Phillips GS, Wise LA, Harlow BL. A prospective analysis of alcohol consumption and onset of perimenopause. *Maturitas*. 2007;56(3):263–272.
 35. U.S. Department of Agriculture and U.S. Department of Health and Human Services. *Dietary Guidelines for Americans, 2020–2025*. Washington, DC: US Department of Agriculture; 2020. https://www.dietaryguidelines.gov/sites/default/files/2020-12/Dietary_Guidelines_for_Americans_2020-2025.pdf. Accessed April 5, 2021.
 36. Bao Y, Bertoia ML, Lenart EB, et al. Origin, Methods, and evolution of the three Nurses' health studies. *Am J Public Health*. 2016;106(9):1573–1581.
 37. Nurses' Health Study. History. <https://www.nurseshealthstudy.org/about-nhs/history>. Accessed April 5, 2021.
 38. Kim HJ, Jung S, Eliassen AH, et al. Alcohol consumption and breast cancer risk in younger women according to family history of breast cancer and folate intake. *Am J Epidemiol*. 2017;186(5):524–531.
 39. Willett WC. *Nutritional Epidemiology*. 3rd ed. New York, NY: Oxford University Press; 2013.
 40. Costanian C, McCague H, Tamim H. Age at natural menopause and its associated factors in Canada: cross-sectional analyses from the Canadian Longitudinal Study on Aging. *Menopause*. 2018;25(3):265–272.