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Early-life factors and breast cancer risk in Hispanic women: The role of adolescent body size

Meera Sangaramoorthy¹, Amanda I. Phipps², Pamela L. Horn-Ross^{1,3}, Jocelyn Koo¹, and Esther M. John^{1,3}

¹Cancer Prevention Institute of California, Fremont, CA 94538

²Public Health Sciences Division, Fred Hutchinson Cancer Research Center, Seattle, WA 98109

³Division of Epidemiology, Department of Health Research and Policy, Stanford University School of Medicine, and Stanford Cancer Institute, Stanford, CA 94305

Abstract

Background—Adult body size has long been known to influence breast cancer risk, and there is now increasing evidence that childhood and adolescent body size may also play a role.

Methods—We assessed the association with body size at ages 10, 15, and 20 years in 475 premenopausal and 775 postmenopausal Hispanic women who participated in a population-based case-control study of breast cancer conducted from 1995 to 2004 in the San Francisco Bay Area. We used unconditional logistic regression to estimate odds ratios (OR) and 95% confidence intervals (CI) for the associations with self-reported relative weight compared to peers and body build at ages 10, 15, and 20 years.

Results—In premenopausal women, we found inverse associations with relative weight compared to peers, with ORs of 0.63 ($P_{\text{trend}} = 0.05$), 0.31 ($P_{\text{trend}} < 0.01$), and 0.44 ($P_{\text{trend}} = 0.02$) for heavier vs. lighter weight at ages 10, 15, and 20 years, respectively. These inverse associations were stronger in currently overweight women and US-born women and did not differ significantly for case groups defined by estrogen receptor status. Inverse associations were stronger in US-born than foreign-born Hispanics. In postmenopausal women not currently using hormone therapy, inverse associations with relative weight were limited to US-born Hispanics.

Conclusions—Large body size at a young age may have a long-lasting influence on breast cancer risk in premenopausal, and possibly postmenopausal, Hispanic women that is independent of current BMI.

Impact—These findings need to be weighed against adverse health effects associated with early-life obesity.

Keywords

Adolescence; BMI; body size; breast cancer; estrogen receptor

INTRODUCTION

Studying migration patterns and breast cancer risk in Hispanic women, we have previously reported a trend of increasing risk with earlier age at migration to the US (1), suggesting that migration-related changes in early-life exposures contribute to the increasing breast cancer

incidence rates in Hispanic immigrants. It has been shown that body size is one factor that can change substantially with migration, particularly in children and adolescents (2).

Adult body size has long been shown to be an important modifiable risk factor for breast cancer, with dual effects depending on menopausal status. Large body size in adulthood is inversely associated with breast cancer risk in premenopausal women (3), whereas in postmenopausal women not using hormone therapy (HT), body size is positively associated with risk (4). More recent epidemiological studies also suggest a possible etiologic role for body size during adolescence, when developing mammary glands may be particularly susceptible to breast carcinogenesis (5-7). Tall height and rapid change in height in childhood and adolescence have been associated with increased breast cancer risk later in life (8, 9). In contrast, high adolescent BMI (9-11), higher relative weight compared to peers (12, 13), and heavy body build in childhood or adolescence (8, 11, 14-16) have been associated with decreased risk, although results are not consistent across studies (17, 18). It remains uncertain whether the relation between breast cancer and adolescent body size are independent of adult adiposity (11, 14, 19), and whether these associations differ by hormone receptor status (10, 13, 16, 20, 21).

Most prior reports on adolescent body size and breast cancer risk included non-Hispanic white women, with only limited data available for Hispanic women (20). Analyzing data from the San Francisco Bay Area Breast Cancer Study, a population-based case-control study conducted in a multiethnic population, we assessed the association between self-assessed relative and absolute body size at ages 10, 15, and 20 years and breast cancer risk in premenopausal and postmenopausal Hispanic women.

MATERIALS AND METHODS

Study population

This population-based case-control study of breast cancer has been described elsewhere (1, 22). We report here on a subset of cases diagnosed from May 1, 1998 to April 30, 2002 and frequency-matched controls ascertained between 1998 and 2003 for whom information on adolescent body size measures was collected. Briefly, 9,978 newly diagnosed breast cancer cases aged 35-79 years were identified through the Greater Bay Area Cancer Registry. Of 8,756 cases who could be contacted (alive, with a valid address and without physician-reported contraindications), 7,704 (88%) completed a brief telephone screening interview that assessed study eligibility and self-reported race/ethnicity. A total of 1,031 cases were selected to participate in the study (all Hispanics diagnosed from 1998 to 2002, all African-Americans diagnosed from 1998 to 1999 and a 10% random sample of eligible non-Hispanic whites diagnosed from 1998 to 1999). Of these cases, 931 (90%) completed an in-person interview, including 650 Hispanics, 134 African Americans, and 147 non-Hispanic whites.

Population controls aged 35-79 years were identified through random-digit dialing (22). Of 87,035 randomly generated telephone numbers, a household enumeration was obtained for 32,802 (86%) of 38,015 residential phone numbers. From the pool of potentially eligible females, 1,478 controls were randomly selected by frequency matching on race/ethnicity and the expected 5-year age distribution of cases. Of the 1,386 selected controls who could be contacted (alive and with valid address), 1,269 (92%) completed the telephone screening interview that assessed self-identified race/ethnicity and history of breast cancer. Of 1,198 eligible controls without a history of breast cancer, 1,050 (88%) completed the in-person interview, including 766 Hispanics, 137 African Americans and 147 non-Hispanic whites. Given the relatively small number of African American and non-Hispanic white women with early-life body size information, we restricted the present analysis to Hispanic women.

Data collection

Trained bilingual, bicultural professional interviewers administered a structured questionnaire in English or Spanish at the participants' home and collected information on known and suspected breast cancer risk factors. In addition to information on body size (described below), lifetime histories of physical activity from multiple sources (22), usual dietary intake including alcohol consumption (23), and menstrual, reproductive, medical and breast cancer family histories were assessed. Information on estrogen receptor (ER) and progesterone receptor (PR) status was available from cancer registry records for 86% of cases. Study participants provided written informed consent and the study was approved by the Institutional Review Board of the Cancer Prevention Institute of California (formerly the Northern California Cancer Center).

Body size variables

Body build at ages 10, 15 and 20 years was assessed using 9 line drawings of body figures ranging from slim to heavy (24) and categorized as slim (figures 1-2), average (figures 3-4), or heavy (figures 5-9). Weight comparisons to peers at ages 10, 15, and 20 years were assessed using a 5-point scale (1=much lighter, 2=lighter, 3=same, 4=heavier, and 5=much heavier) and categorized as lighter (1-2), same (3), or heavier (4-5). Current body mass index (BMI) was calculated as weight (in kilograms [kg]) divided by height squared (in meters [m]), based on measured height at interview (or self-reported adult height for 3% of cases and 2% of controls who declined the measurements) and self-reported weight in the reference year (or measured weight for 1% of cases and 4% of controls without self-report). Among controls with both self-reported weight in the reference year and measured weight at interview, the two weights were highly correlated ($r=0.91$), justifying the substitution of measured weight for self-reported weight when the latter was missing. Adult BMI was classified as normal weight (18.5-24.9 kg/m²), overweight (25.0-29.9 kg/m²), or obese (≥ 30.0 kg/m²) (25). Women who were underweight (BMI <18.5 kg/m²; 2 cases and 3 controls) were grouped with normal weight women.

Statistical analysis

We performed separate analyses for premenopausal and postmenopausal Hispanic women. Premenopausal women included those who were pregnant, breast-feeding, or reported that they were still having menstrual periods in the reference year. The reference year was defined as the calendar year prior to diagnosis (cases) or selection into the study (controls). Women were considered postmenopausal if they reported that their periods had stopped more than one year prior to diagnosis (cases) or selection into the study (controls), if they had had a bilateral oophorectomy, or if they were aged 55 years or older at the time of diagnosis or selection and had either started HT use prior to cessation of menses or had had a simple hysterectomy (without oophorectomy). Women for whom menopausal status could not be determined based on these criteria (59 cases, 62 controls) and women with missing information on HT use (8 cases, 2 controls) were excluded from all analyses.

Unconditional logistic regression was used to estimate breast cancer risk associated with adolescent body size measures comparing cases to controls. Polytomous logistic regression was used to compare cases with ER+ or ER- tumors to controls. Analyses by joint ER and PR status (data not shown) were unstable due to small numbers and did not alter our interpretation of the data.

Multivariate adjusted odds ratios (OR) and 95% confidence intervals (CI) were estimated, adjusting for age (years, continuous) and other established risk factors for breast cancer, including country of birth (U.S.-born, foreign-born), education (some high school or less, high school graduate or technical school, some college, college graduate), family history of

breast cancer in first-degree relatives (no, yes), prior biopsy-confirmed history of benign breast disease (no, yes), number of full-term pregnancies (nulliparous, 1, 2, 3, 4), lifetime breastfeeding (nulliparous, 0, 6, 7-12, 13-24, 25 months), age at first full-term pregnancy (nulliparous, 19, 20-24, 25-29, 30 years), use of oral contraceptives (0, <5, 5 years), adult height (cm, quartiles among controls), alcohol consumption during the reference year (0, 0.1-4.9, 5-9.9, 10-19.9, 20 g/day), and caloric intake during the reference year (kcal/day, quartiles among controls). Because age at menarche could be an intermediate variable that is affected by early-life body size, we performed secondary analyses with and without adjustment for age at menarche. Separate analyses were also performed adjusting for and then stratifying by current BMI (<25.0, 25.0-29.9, 30.0 kg/m²). In postmenopausal women, we stratified the analyses by HT use (current use vs. past or never use) given evidence of potential heterogeneity in associations with adult BMI according to current HT use (4). Linear trends were assessed across ordinal values of categorical variables. Differences in ORs between the two case groups defined by hormone receptor status were tested using the Wald statistic *p*-value, which was calculated from the polytomous regression model. All tests of significance were two-sided and *p*-values <0.05 were considered statistically significant. Analyses were performed using SAS Version 9.1 (SAS Institute, Cary, NC).

For the multivariate analyses we excluded 20 women (7 cases, 13 controls) with missing information on covariates, and 15 women (12 cases, 3 controls) with a daily caloric intake that was considered unreliable (<600 Kcal or >5,000 Kcal), leaving 1,250 women in the analysis (564 cases, 686 controls). Of these, 475 were premenopausal (210 cases, 265 controls), 580 were postmenopausal with past or never use of HT (205 cases, 375 controls), and 195 were postmenopausal with current HT use (149 cases, 46 controls).

RESULTS

Characteristics of cases and controls by menopausal status are shown in Table 1. Compared to controls, cases were more likely to have been born in the US, had a higher level of education, were more likely to report a family history of breast cancer in first-degree relatives or a personal history of benign breast disease, had a younger age at menarche, were more likely to be nulliparous, had fewer full-term pregnancies, a later age at first full-term pregnancy, a shorter duration of breastfeeding, and higher use of oral contraceptives. Cases were taller, less likely to be currently obese, and had higher alcohol consumption and caloric intake. More postmenopausal cases than controls were current HT users. A higher percentage of US-born than foreign-born control women reported heavier relative weight compared to peers at ages 10 years (24% vs. 16%) and 15 years (27% vs. 22%) and heavy body build at age 20 years (33% vs. 24%).

Among premenopausal women, breast cancer risk was inversely associated with relative weight compared to peers, with ORs of 0.63 ($P_{\text{trend}} = 0.05$), 0.31 ($P_{\text{trend}} < 0.01$), and 0.44 ($P_{\text{trend}} = 0.02$) for heavier vs. lighter relative weight at ages 10, 15, and 20 years, respectively (Table 2). These associations were independent of current BMI. Additional adjustment for age at menarche did not alter the results (data not shown). Inverse associations with relative weight were somewhat stronger for US-born than foreign-born premenopausal women, even after adjustment for current BMI, with ORs ranging from 0.22 to 0.26 for heavier vs. lighter relative weight at ages 10, 15, and 20 years (Table 3). Among foreign-born premenopausal women, relative weight at ages 15 and 20 years were also inversely associated with risk.

Considering the joint effects of current BMI and adolescent body size (Table 4), we found that among overweight women (BMI ≥ 25 kg/m²), breast cancer risk decreased with heavier relative weight at ages 15 ($P_{\text{trend}} < 0.01$) and 20 years ($P_{\text{trend}} = 0.02$) and heavy body build

at age 20 years ($P_{\text{trend}} = 0.02$). There were very few currently normal-weight women (BMI $<25 \text{ kg/m}^2$) who reported having a heavy body build or heavier relative weight compared to their peers at ages 10, 15, or 20 years; comparisons in this currently normal-weight group revealed no significant associations with adolescent body size. Compared to currently normal-weight (BMI $<25 \text{ kg/m}^2$) women who were lighter than their peers at age 10 years, risk was lowest in currently overweight women (BMI $\geq 25 \text{ kg/m}^2$) who were heavier than their peers at age 10 years (OR=0.31, 95% CI: 0.14-0.68), with similar corresponding ORs for relative weight at ages 15 and 20 years (OR=0.23, 95% CI: 0.10-0.51, and OR=0.25, 95% CI: 0.12-0.52, respectively).

When stratifying the analyses by ER status (Table 4), premenopausal cases with ER+ disease were more likely to report a lighter relative weight and slim body build at ages 10, 15 and 20 years, with significant inverse associations found for relative weight at ages 10 ($P_{\text{trend}} = 0.04$) and 15 years ($P_{\text{trend}} < 0.01$). Similarly, although numbers were limited for premenopausal cases with ER- disease, we found that these cases were more likely to report a lighter relative weight at ages 10, 15, and 20 years or a slim body build at age 20 years. Overall, inverse associations did not differ significantly for ER+ and ER- disease ($P_{\text{heterogeneity}} > 0.05$ for all comparisons).

In postmenopausal women, there were no significant inverse associations with childhood or adolescent body size measures in women not currently using HT (Table 2), both among never HT users and past HT users (data not shown), or in women currently using HT (data not shown). Among women not currently using HT, inverse associations with large body size measures were limited to US-born Hispanic women only (Supplemental Table 1). We found inverse trends for relative weight at ages 10 ($P_{\text{trend}} = 0.01$), 15 ($P_{\text{trend}} = 0.08$), and 20 years ($P_{\text{trend}} = 0.04$) and body build at ages 10 ($P_{\text{trend}} = 0.08$) and 15 years ($P_{\text{trend}} = 0.05$), that were slightly attenuated after adjustment for current BMI. No statistically significant associations were found for ER+ or ER- tumors in postmenopausal women (Supplemental Table 1).

DISCUSSION

Similar to earlier reports in non-Hispanic white women, we found that early-life body size influences risk of premenopausal breast cancer in Hispanic women. There was a more consistent pattern of inverse associations for relative weight than for body build. US-born controls were more likely than foreign-born controls to report large childhood and adolescent body size, and particularly strong inverse associations with relative weight were found in US-born women and women with a current BMI $\geq 25 \text{ kg/m}^2$. We found no associations with childhood or adolescent body size in postmenopausal women, except for some suggestive inverse associations in US-born women not currently using HT.

Consistent with our findings, the only other study that reported on adolescent body size and breast cancer risk in Hispanic women also found a suggestive inverse association with high BMI (≥ 22.5 vs. < 18.2) at age 15 years (OR=0.65, 95% CI: 0.39-1.08, $P_{\text{trend}} = 0.09$) (20). Our findings for premenopausal breast cancer are also consistent with previous reports in mostly non-Hispanic white women. Inverse associations have been found for high relative weight (12, 19), heavy body build (15, 16), or high BMI (10) at various ages in childhood or adolescence, although some studies did not find associations with adolescent body size (11, 17, 18). We found inverse trends for relative weight at ages 10, 15, and 20 years and, consistent with other studies (15, 16), we found that the inverse association of premenopausal breast cancer risk with early-life body size was independent of current BMI, a strong predictor of premenopausal breast cancer risk in this study (26). Similar to other cohort (19) and case-control (27) studies, we found the greatest reduction in premenopausal

breast cancer risk in women who were heavy both in childhood or adolescence and adulthood.

Unlike some other studies that reported reduced risks of postmenopausal breast cancer risk associated with large adolescent body size (11, 13, 16, 28), the present study found little evidence of inverse associations in postmenopausal women, regardless of HT use, except for suggestive inverse associations among US-born women not currently using HT. In the Nurses' Health Study (16), the largest study on adolescent body size and breast cancer risk to date, inverse associations did not differ between pre- and postmenopausal women.

Given the small number of African American and non-Hispanic white subjects for whom we collected information on early-life body size, we restricted the analysis to Hispanic women. When we included women of all three race/ethnicities, the results for premenopausal women (276 cases, 345 controls) and postmenopausal women not currently using HT (312 cases, 511 controls) were essentially the same (data not shown).

Few studies have assessed whether the association with adolescent body size is modified by hormone receptor status (10, 13, 16, 20, 21) and findings are inconsistent. In the Nurses' Health Study (16), the inverse association with adolescent body build among pre- and postmenopausal women combined was stronger for ER- tumors than ER+ tumors, and PR status was not found to be an important modifying factor. Other studies have also reported stronger inverse associations for ER- tumors with BMI at age 15 years in Hispanics (pre- and postmenopausal women combined) (20), and with body build at age 7 years in a population of Swedish postmenopausal women (21). In contrast, some studies found inverse associations with high BMI at age 18 years limited to ER+ premenopausal breast cancer (10), and with high relative weight at age 12 years limited to PR-postmenopausal breast cancer (13). In the present study, inverse associations of premenopausal breast cancer risk with large childhood and adolescent body size were more consistent for ER+ tumors, but associations were not significantly different by ER status.

Some potential limitations need to be considered when interpreting our results. The analyses for premenopausal women, particularly those for breast cancer defined by ER status, were limited by small sample sizes. Our findings suggest potentially different effects of early-life body size on breast cancer risk in US-born vs. foreign-born Hispanic women, but our sample size was too small to examine these complex interactions. Exposure ascertainment required recall of body size many years in the past and, thus, could be subject to errors in recall. To minimize recall error, we asked about comparative weight, which may be easier to recall than absolute weight at young ages (29), and we used visual aids, such as line drawings of body figures, that have been validated as a reliable self-reported measure of adult (29) and adolescent (28, 30) body size. Other studies have shown that self-reported body build at 18 years is strongly correlated with recorded BMI at age 18 years (28), and that BMI measured at menarche is strongly correlated with body figures recalled 30 years later (30). It is reassuring that, in our study, relative weight at age 10 years (among control women who had not started menstruation by age 10 years) was inversely associated with age at menarche, with mean ages of 13.4, 13.0, and 12.7 years for lighter, same, and heavier relative weight, respectively. This inverse trend is consistent with other reports (16, 21). When we adjusted the multivariate analyses for age at menarche, the inverse associations were not altered. Our findings on adolescent body size were stronger for relative weight compared to peers than for body build assessed by figure drawings, possibly due to better recall of the former adolescent body size measure. There is some evidence that birth size and infant growth may be related to breast cancer risk later in life and that the effect of adolescent body size may be mediated through these very early life factors (31, 32). We did not collect information on birth weight or infant growth, and therefore could not consider these factors in our analyses.

The biologic explanations for the inverse association between large adolescent body size and premenopausal breast cancer risk remain uncertain. Childhood obesity is associated with earlier menarche, a well established risk factor for breast cancer. This association seems incongruent with the observed inverse association with large adolescent body size. Several underlying mechanisms have been postulated (33). It has been suggested that overweight adolescents experience slower pubertal growth and sexual maturation (8), despite having, on average, an earlier onset of puberty (34). Overweight adolescents may also experience a longer interval between menarche and the onset of regular menstrual cycles (14), which may be indicative of a greater frequency of anovulatory cycles (6) and, thus, lower cumulative exposure to ovarian sex hormones. Obesity in young girls has been associated with lower sex hormone binding globulin levels (35) and higher androgen levels (6, 35); estrogen and progesterone concentrations, on the other hand, do not appear to vary with BMI in adolescence (35, 36). Obesity in young girls has been associated with higher levels of insulin (37) and insulin-like growth factor-1 (38), and it has been hypothesized that obesity at a young age could influence the metabolic set point for life through an insulin-related pathway (20). Findings on hormone levels in young girls, however, are not consistent and not necessarily compatible with decreased breast cancer risk in adult life, suggesting complex biologic pathways. Furthermore, it is not known whether hormonal changes associated with adolescent obesity persist in adult life. It has also been suggested that higher levels of estrogen and other hormones in young overweight girls may induce earlier differentiation of breast tissue, with terminally differentiated cells being less susceptible to malignant transformation (16), and that body fat at a young age influences the histologic constituents of breast tissue (11).

Risk factors for breast cancer in Hispanic women have not been extensively studied. Interestingly, in the present analysis the inverse associations with early-life body size in premenopausal women were stronger for US-born than foreign-born Hispanics, and in postmenopausal women not currently using HT, they were limited to US-born Hispanics. The basis for this difference by country of birth is unclear; it is possible that correlates of and contributors to childhood body size differ across these populations. Large body size in childhood or adolescence was more frequently reported by US-born than foreign-born Hispanic women. Similarly, in a study of children from Mexican descent, the prevalence of obesity was considerably higher in those living in California than in Mexico (2). It is possible that the earlier onset of childhood obesity in US-born Hispanics exerts a greater protective effect on breast cancer risk than in foreign-born Hispanics. US-born and foreign-born Hispanics also differ in other factors; foreign-born controls had higher levels of physical activity before age 20 years, later age at menarche, and shorter height (unpublished data), suggesting complex interactions between several early-life factors.

Given increasing trends of obesity in Hispanic children and adolescents (39), it remains to be seen whether or not childhood and adolescent obesity today has the same underlying pathology or biologic consequences as it did in the past and, thus, whether it will lead to lower breast cancer incidence rates in the future. Furthermore, the potential benefit of adolescent obesity in reducing breast cancer risk need to be weighed against obesity-related risks of other chronic diseases, such as diabetes, which is increasingly prevalent in obese children and adolescents (40).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Abbreviations

BMI	body mass index
CI	confidence interval
ER	estrogen receptor
HT	hormone therapy
OR	odds ratio
PR	progesterone receptor

REFERENCES

1. John EM, Phipps AI, Davis A, Koo J. Migration history, acculturation, and breast cancer risk in Hispanic women. *Cancer Epidemiol Biomarkers Prev.* 2005; 14:2905–13. [PubMed: 16365008]
2. Rosas LG, Guendelman S, Harley K, Fernald LC, Neufeld L, Mejia F, et al. Factors associated with overweight and obesity among children of Mexican descent: results of a binational study. *J Immigr Minor Health.* 2011; 13:169–80. [PubMed: 20217234]
3. Friedenreich CM. Review of anthropometric factors and breast cancer risk. *Eur J Cancer Prev.* 2001; 10:15–32. [PubMed: 11263588]
4. Morimoto LM, White E, Chen Z, Chlebowski RT, Hays J, Kuller L, et al. Obesity, body size, and risk of postmenopausal breast cancer: the Women's Health Initiative (United States). *Cancer Causes Control.* 2002; 13:741–51. [PubMed: 12420953]
5. Russo J, Tay LK, Russo IH. Differentiation of the mammary gland and susceptibility to carcinogenesis. *Breast Cancer Res Treat.* 1982; 2:5–73. [PubMed: 6216933]
6. Stoll BA. Teenage obesity in relation to breast cancer risk. *Int J Obes Relat Metab Disord.* 1998; 22:1035–40. [PubMed: 9822939]
7. Michels KB, Willett WC. Breast cancer--early life matters. *N Engl J Med.* 2004; 351:1679–81. [PubMed: 15483288]
8. Berkey CS, Frazier AL, Gardner JD, Colditz GA. Adolescence and breast carcinoma risk. *Cancer.* 1999; 85:2400–9. [PubMed: 10357411]
9. Ahlgren M, Melbye M, Wohlfahrt J, Sorensen TI. Growth patterns and the risk of breast cancer in women. *N Engl J Med.* 2004; 351:1619–26. [PubMed: 15483280]
10. Michels KB, Terry KL, Willett WC. Longitudinal study on the role of body size in premenopausal breast cancer. *Arch Intern Med.* 2006; 166:2395–402. [PubMed: 17130395]
11. Berstad P, Coates RJ, Bernstein L, Folger SG, Malone KE, Marchbanks PA, et al. A case-control study of body mass index and breast cancer risk in white and African-American women. *Cancer Epidemiol Biomarkers Prev.* 2010; 19:1532–44. [PubMed: 20501755]
12. Coates RJ, Uhler RJ, Hall HI, Potischman N, Brinton LA, Ballard-Barbash R, et al. Risk of breast cancer in young women in relation to body size and weight gain in adolescence and early adulthood. *Br J Cancer.* 1999; 81:167–74. [PubMed: 10487629]
13. Bardia A, Vachon CM, Olson JE, Vierkant RA, Wang AH, Hartmann LC, et al. Relative weight at age 12 and risk of postmenopausal breast cancer. *Cancer Epidemiol Biomarkers Prev.* 2008; 17:374–8. [PubMed: 18250344]
14. Tehard B, Kaaks R, Clavel-Chapelon F. Body silhouette, menstrual function at adolescence and breast cancer risk in the E3N cohort study. *Br J Cancer.* 2005; 92:2042–8. [PubMed: 15928661]

15. Baer HJ, Colditz GA, Rosner B, Michels KB, Rich-Edwards JW, Hunter DJ, et al. Body fatness during childhood and adolescence and incidence of breast cancer in premenopausal women: a prospective study. *Breast Cancer Research*. 2005; 7:R314–R325. [PubMed: 15987426]
16. Baer HJ, Tworoger SS, Hankinson SE, Willett WC. Body fatness at young ages and risk of breast cancer throughout life. *Am J Epidemiol*. 2010; 171:1183–94. [PubMed: 20460303]
17. Franceschi S, Favero A, La Vecchia C, Baron AE, Negri E, Dal Maso L, et al. Body size indices and breast cancer risk before and after menopause. *Int J Cancer*. 1996; 67:181–6. [PubMed: 8760584]
18. Sanderson M, Shu XO, Jin F, Dai Q, Ruan Z, Gao YT, et al. Weight at birth and adolescence and premenopausal breast cancer risk in a low-risk population. *Br J Cancer*. 2002; 86:84–8. [PubMed: 11857016]
19. Weiderpass E, Braaten T, Magnusson C, Kumle M, Vainio H, Lund E, et al. A prospective study of body size in different periods of life and risk of premenopausal breast cancer. *Cancer Epidemiol Biomarkers Prev*. 2004; 13:1121–7. [PubMed: 15247122]
20. Slattery ML, Sweeney C, Edwards S, Herrick J, Baumgartner K, Wolff R, et al. Body size, weight change, fat distribution and breast cancer risk in Hispanic and non-Hispanic white women. *Breast Cancer Res Treat*. 2007; 102:85–101. [PubMed: 17080310]
21. Li J, Humphreys K, Eriksson L, Czene K, Liu J, Hall P. Effects of childhood body size on breast cancer tumour characteristics. *Breast Cancer Res*. 2010; 12:R23. [PubMed: 20398298]
22. John EM, Horn-Ross PL, Koo J. Lifetime physical activity and breast cancer risk in a multiethnic population: the San Francisco Bay area breast cancer study. *Cancer Epidemiol Biomarkers Prev*. 2003; 12:1143–52. [PubMed: 14652273]
23. Horn-Ross PL, John EM, Lee M, Stewart SL, Koo J, Sakoda LC, et al. Phytoestrogen consumption and breast cancer risk in a multiethnic population: the Bay Area Breast Cancer Study. *Am J Epidemiol*. 2001; 154:434–41. [PubMed: 11532785]
24. Stunkard AJ, Sorensen T, Schulsinger F. Use of the Danish Adoption Register for the study of obesity and thinness. *Res Publ Assoc Res Nerv Ment Dis*. 1983; 60:115–20. [PubMed: 6823524]
25. WHO. Obesity: preventing and managing the global epidemic: report of a WHO Consultation. World Health Organization; Geneva: 2000. p. 0512-3054.
26. John EM, Sangaramoorthy M, Phipps AI, Koo J, Horn-Ross PL. Adult body size, hormone receptor status, and premenopausal breast cancer risk in a multiethnic population: the San Francisco Bay Area breast cancer study. *Am J Epidemiol*. 2010; 173:201–16. [PubMed: 21084558]
27. Magnusson CM, Roddam AW, Pike MC, Chilvers C, Crossley B, Hermon C, et al. Body fatness and physical activity at young ages and the risk of breast cancer in premenopausal women. *Br J Cancer*. 2005; 93:817–24. [PubMed: 16160699]
28. Magnusson C, Baron J, Persson I, Wolk A, Bergstrom R, Trichopoulos D, et al. Body size in different periods of life and breast cancer risk in post-menopausal women. *Int J Cancer*. 1998; 76:29–34. [PubMed: 9533758]
29. Bulik CM, Wade TD, Heath AC, Martin NG, Stunkard AJ, Eaves LJ. Relating body mass index to figural stimuli: population-based normative data for Caucasians. *Int J Obes Relat Metab Disord*. 2001; 25:1517–24. [PubMed: 11673775]
30. Must A, Phillips SM, Naumova EN, Blum M, Harris S, Dawson-Hughes B, et al. Recall of early menstrual history and menarcheal body size: after 30 years, how well do women remember? *Am J Epidemiol*. 2002; 155:672–9. [PubMed: 11914195]
31. Michels KB, Trichopoulos D, Robins JM, Rosner BA, Manson JE, Hunter DJ, et al. Birthweight as a risk factor for breast cancer. *Lancet*. 1996; 348:1542–6. [PubMed: 8950880]
32. dos Santos Silva I, De Stavola BL, Hardy RJ, Kuh DJ, McCormack VA, Wadsworth ME. Is the association of birth weight with premenopausal breast cancer risk mediated through childhood growth? *Br J Cancer*. 2004; 91:519–24. [PubMed: 15266328]
33. Magnusson CM, Roddam AW. Breast cancer and childhood anthropometry: emerging hypotheses? *Breast Cancer Res*. 2005; 7:83. [PubMed: 15987435]

34. Kaplowitz PB, Slora EJ, Wasserman RC, Pedlow SE, Herman-Giddens ME. Earlier onset of puberty in girls: relation to increased body mass index and race. *Pediatrics*. 2001; 108:347–53. [PubMed: 11483799]
35. Baer HJ, Colditz GA, Willett WC, Dorgan JF. Adiposity and sex hormones in girls. *Cancer Epidemiol Biomarkers Prev*. 2007; 16:1880–8. [PubMed: 17855709]
36. Larmore KA, O'Connor D, Sherman TI, Funanage VL, Hassink SG, Klein KO. Leptin and estradiol as related to change in pubertal status and body weight. *Med Sci Monit*. 2002; 8:CR206–10. [PubMed: 11889458]
37. Caprio S, Hyman LD, Limb C, McCarthy S, Lange R, Sherwin RS, et al. Central adiposity and its metabolic correlates in obese adolescent girls. *Am J Physiol*. 1995; 269:E118–26. [PubMed: 7631766]
38. Fall CH, Pandit AN, Law CM, Yajnik CS, Clark PM, Breier B, et al. Size at birth and plasma insulin-like growth factor-1 concentrations. *Arch Dis Child*. 1995; 73:287–93. [PubMed: 7492190]
39. Flegal KM, Ogden CL, Carroll MD. Prevalence and trends in overweight in Mexican-american adults and children. *Nutr Rev*. 2004; 62:S144–8. [PubMed: 15387481]
40. Dabelea D, Bell RA, D'Agostino RB Jr, Imperatore G, Johansen JM, Linder B, et al. Incidence of diabetes in youth in the United States. *JAMA*. 2007; 297:2716–24. [PubMed: 17595272]

Table 1

Characteristics of Study Population, by Menopausal Status^a

	Premenopausal				Postmenopausal			
	Cases N = 210		Controls N = 265		Cases N = 354		Controls N = 421	
	N	%	N	%	N	%	N	%
Age (years)								
35-44	112	53	157	59	10	3	9	2
45-54	96	46	106	40	62	18	98	23
55-64	2	1	2	1	147	42	166	39
65-74					99	28	126	30
75					36	10	22	5
Estrogen receptor (ER) status								
ER+	124	59			241	68		
ER-	52	25			69	19		
Unknown ER	34	16			44	12		
Country of birth								
US-born ^c	94	45	75	28	185	52	155	37
Foreign-born	116	55	190	72	169	48	266	63
Education								
Some high school or less	74	35	138	52	169	48	242	58
High school graduate or technical school	63	30	66	25	99	28	102	24
Some college	45	21	29	11	46	13	39	9
College graduate	28	13	32	12	40	11	28	9
Family history of breast cancer in first-degree relatives								
No	186	89	246	93	292	83	374	89
Yes	24	11	19	7	62	18	47	11
Prior biopsy-confirmed history of benign breast disease								

	Premenopausal						Postmenopausal					
	Cases N = 210			Controls N = 265			Cases N = 354			Controls N = 421		
	N	%		N	%	p-value ^b	N	%		N	%	p-value ^b
No	183	87		247	93	0.03	285	81		358	85	0.10
Yes	27	13		18	7		69	20		63	15	
Age at menarche (years)												
11	56	27		67	25	<0.01	96	27		100	24	<0.15
12	59	28		42	16		85	24		81	19	
13	50	24		67	25		70	20		91	22	
14	44	21		88	33		101	29		145	35	
Number of full-term pregnancies												
Nulliparous	34	16		18	7	<0.01	24	7		21	5	0.20
1	30	14		28	11		38	11		38	9	
2	62	30		64	24		75	21		69	16	
3	52	25		79	30		74	21		98	23	
4	32	15		76	29		143	40		195	46	
Lifetime breastfeeding (months) ^d												
0	50	28		52	21	<0.01	138	42		127	32	0.04
6	44	25		37	15		75	23		89	22	
7-12	24	14		36	15		27	8		40	10	
13-24	29	16		49	20		41	12		59	15	
25	29	16		73	30		49	15		85	21	
Age at first full-term pregnancy (years) ^d												
19	33	19		80	32	<0.01	85	26		110	28	0.03
20-24	75	43		81	33		149	45		161	40	
25-29	37	21		53	21		53	16		94	24	
30	31	18		33	13		43	13		35	9	
Oral contraceptive use (years)												
0	50	24		76	29	0.04	152	43		190	45	0.22
<5	92	44		131	49		124	35		159	38	

	Premenopausal						Postmenopausal					
	Cases N = 210			Controls N = 265			Cases N = 354			Controls N = 421		
	N	%	N	%	N	%	N	%	N	%	N	%
5	68	32	58	22			78	22	72	17		
Menopausal hormone therapy use												
Never							132	37	194	46		<0.01
Past							73	21	181	43		
Current							149	42	46	11		
Current body mass index (kg/m ²)												
<25.0	74	35	60	23			79	22	69	16		0.11
25.0-29.9	75	36	101	38			119	34	149	35		
30.0	61	29	104	39			156	44	203	48		
Adult height (cm) ^e												
152.8	36	17	68	26			61	17	104	25		0.02
152.9-157.6	50	24	66	25			93	26	104	25		
157.7-162.6	44	21	67	25			84	24	108	26		
162.7	80	38	64	24			116	33	105	25		
Average alcohol consumption (g/day) ^f												
0	121	58	182	67			238	67	298	71		0.11
0.1-4.9	36	17	54	20			62	18	71	17		
5.0-9.9	14	7	12	5			17	5	27	6		
10.0-19.9	22	11	14	5			21	6	18	4		
20	17	8	3	1			16	5	7	2		
Average caloric intake (kcal/day) ^{e, f}												
1477.5	44	21	66	25			67	19	107	25		0.06
1477.6-1961.5	38	18	67	25			88	25	103	25		
1961.6-2541.5	35	31	66	25			86	24	107	25		
2541.6	63	60	66	25			113	32	104	25		

^a Percentages may not add up to 100 due to rounding.

- ^b Chi square p-value for difference between cases and controls.
- ^c Includes 2 cases and 3 controls born in western countries such as Canada, Europe, Australia, and New Zealand.
- ^d Among parous women.
- ^e Quartiles among controls.
- ^f In reference year.

Table 2
Adolescent Body Size and Breast Cancer Risk in Hispanic Women, by Menopausal Status

	Premenopausal				Postmenopausal, not currently using hormone therapy							
	Cases N = 210		Controls N = 265		Cases N = 205		Controls N = 375					
	N	%	N	%	OR ^a , 95% CI	OR ^b , 95% CI	N	%	OR ^a , 95% CI	OR ^b , 95% CI		
Relative weight compared to peers at age 10 years												
Lighter	109	52	100	40	1.00	1.00	114	58	188	52	1.00	
Same	68	33	106	42	0.55 0.35-0.88	0.58 0.37-0.94	61	31	123	34	0.79 0.52-1.20	0.84 0.55-1.29
Heavier	31	15	46	18	0.52 0.28-0.97	0.63 0.33-1.20	23	12	51	14	0.61 0.34-1.11	0.68 0.37-1.25
P for trend					p=0.01	p=0.05			p=0.08	p=0.19		
Relative weight compared to peers at age 15 years												
Lighter	91	44	69	27	1.00	1.00	85	43	156	43	1.00	
Same	89	43	129	50	0.49 0.30-0.79	0.53 0.32-0.86	81	41	161	44	0.95 0.63-1.42	1.04 0.69-1.57
Heavier	29	14	61	24	0.28 0.15-0.52	0.31 0.16-0.61	31	16	47	13	1.14 0.64-2.03	1.32 0.73-2.38
P for trend					p<0.01	p<0.01			p=0.80	p=0.43		
Relative weight compared to peers at age 20 years												
Lighter	80	38	67	26	1.00	1.00	67	34	123	34	1.00	
Same	95	46	120	46	0.76 0.47-1.23	0.82 0.50-1.35	97	49	176	79	0.99 0.65-1.50	1.10 0.72-1.69
Heavier	34	16	73	28	0.38 0.21-0.68	0.44 0.24-0.84	34	17	64	18	1.04 0.59-1.82	1.19 0.69-2.12
P for trend					p<0.01	p=0.02			p=0.92	p=0.54		
Body build at age 10 years												
Slim	147	71	169	65	1.00	1.00	155	76	262	72	1.00	
Average	42	20	69	27	0.60 0.36-1.01	0.68 0.40-1.15	38	19	73	20	0.83 0.52-1.33	0.90 0.55-1.45
Heavy	18	9	22	9	0.93 0.44-1.99	1.11 0.51-2.41	11	5	28	8	0.55 0.25-1.19	0.57 0.26-1.24
P for trend					p=0.26	p=0.60			p=0.11	p=0.18		
Body build at age 15 years												
Slim	88	42	90	34	1.00	1.00	99	49	156	43	1.00	
Average	90	43	132	50	0.70 0.44-1.10	0.76 0.48-1.20	81	40	178	48	0.71 0.48-1.06	0.79 0.51-1.13

	Premenopausal						Postmenopausal, not currently using hormone therapy					
	Cases N = 210			Controls N = 265			Cases N = 205			Controls N = 375		
	N	%	N	%	OR ^a , 95% CI	OR ^b , 95% CI	N	%	N	%	OR ^a , 95% CI	OR ^b , 95% CI
Heavy	30	14	42	16	0.72 0.39-1.35	0.86 0.45-1.64	23	11	34	9	0.90 0.48-1.68	0.96 0.51-1.82
<i>P</i> for trend					<i>p</i> =0.18	<i>p</i> =0.45					<i>p</i> =0.28	<i>p</i> =0.46
Body build at age 20 years												
Slim	52	25	46	17	1.00	1.00	50	24	87	24	1.00	1.00
Average	117	56	148	56	0.66 0.39-1.12	0.71 0.41-1.20	120	59	224	61	0.96 0.61-1.49	1.03 0.666-1.62
Heavy	39	19	70	27	0.47 0.25-0.88	0.57 0.29-1.12	35	17	58	16	1.04 0.58-1.87	1.16 0.64-2.10
<i>P</i> for trend					<i>p</i> =0.02	<i>p</i> =0.10					<i>p</i> =0.93	<i>p</i> =0.65

^aOdds ratios (OR) and 95% confidence intervals (CI), adjusted for age (years, continuous), country of birth (US-born, foreign-born), education (some high school or less, high school graduate or technical school, some college, college graduate), first-degree family history of breast cancer (no, yes), prior biopsy-confirmed history of benign breast disease (no, yes), number of full-term pregnancies (nulliparous, 1, 2, 3, 4), lifetime breastfeeding (nulliparous, 0, 6, 7-12, 13-24, 25 months), age at first fullterm pregnancy (nulliparous, 19, 20-24, 25-29, 30 years), oral contraceptive use (never <5, 5 years), adult height (cm, quartiles among controls), average alcohol consumption (0, 0.1-4.9, 5-9.9, 10-19.9, 20 g/day), and average caloric intake (kcal/day, quartiles among controls).

^bAdjusted additionally for current BMI (<25.0, 25.0-29.9, 30.0 kg/m²).

Table 3
Adolescent Body Size and Breast Cancer Risk in Hispanic Women, by Place of Birth

	US-born						Foreign-born					
	Cases N = 94			Controls N = 75			Cases N = 116			Controls N = 190		
	N	%	N	%	OR ^a , 95% CI	OR ^b , 95% CI	N	%	N	%	OR ^a , 95% CI	OR ^b , 95% CI
Relative weight compared to peers at age 10 years												
Lighter	54	58	23	31	1.00	1.00	55	48	77	43	1.00	1.00
Same	27	29	33	45	0.29 0.12-0.73	0.33 0.12-0.91	41	36	73	41	0.80 0.44-1.44	0.77 0.42-1.41
Heavier	13	14	18	24	0.23 0.07-0.72	0.26 0.08-0.85	18	16	28	16	0.71 0.32-1.60	0.92 0.39-2.14
<i>P</i> for trend					<i>p</i> =0.01	<i>p</i> =0.02					<i>p</i> =0.35	<i>p</i> =0.61
Relative weight compared to peers at age 15 years												
Lighter	44	47	18	24	1.00	1.00	47	41	51	28	1.00	1.00
Same	37	39	37	49	0.45 0.17-1.14	0.52 0.19-1.47	52	45	92	50	0.53 0.29-0.98	0.55 0.30-1.03
Heavier	13	14	20	27	0.18 0.06-0.59	0.22 0.06-0.81	16	14	41	22	0.30 0.14-0.68	0.34 0.15-0.79
<i>P</i> for trend					<i>p</i> <0.01	<i>p</i> =0.02					<i>p</i> <0.01	<i>p</i> =0.01
Relative weight compared to peers at age 20 years												
Lighter	45	48	20	27	1.00	1.00	35	30	47	25	1.00	1.00
Same	30	32	33	44	0.53 0.21-1.35	0.57 0.21-1.49	65	57	87	47	0.87 0.47-1.61	0.91 0.48-1.72
Heavier	19	20	22	29	0.30 0.11-0.83	0.25 0.07-0.87	15	13	51	28	0.38 0.17-0.86	0.45 0.19-1.06
<i>P</i> for trend					<i>p</i> <0.02	<i>p</i> =0.03					<i>p</i> =0.03	<i>p</i> =0.08
Body build at age 10 years												
Slim	62	67	39	52	1.00	1.00	85	74	130	70	1.00	1.00
Average	24	26	29	39	0.51 0.21-1.25	0.64 0.24-1.70	18	16	40	22	0.66 0.33-1.33	0.72 0.35-1.47
Heavy	6	7	7	9	0.57 0.13-2.41	0.76 0.17-3.35	12	10	15	8	1.13 0.43-2.95	1.43 0.52-3.88
<i>P</i> for trend					<i>p</i> =0.18	<i>p</i> =0.48					<i>p</i> =0.70	<i>p</i> =0.93
Body build at age 15 years												
Slim	36	39	23	31	1.00	1.00	52	45	67	36	1.00	1.00
Average	46	50	40	53	1.09 0.45-2.65	1.49 0.58-3.84	44	38	92	49	0.57 0.32-1.04	0.60 0.33-1.09

	US-born						Foreign-born					
	Cases N = 94			Controls N = 75			Cases N = 116			Controls N = 190		
	N	%	N	%	OR ^a , 95% CI	OR ^b , 95% CI	N	%	N	%	OR ^a , 95% CI	OR ^b , 95% CI
Heavy	10	11	12	16	0.57 0.16-1.99	1.02 0.26-4.06	20	17	30	16	0.80 0.38-1.71	0.91 0.42-1.98
<i>P</i> for trend					<i>p</i> =0.50	<i>p</i> =0.80					<i>p</i> =0.28	<i>p</i> =0.46
Body build at age 20 years												
Slim	22	24	12	16	1.00	1.00	30	26	34	18	1.00	1.00
Average	52	57	38	51	1.35 0.46-3.97	1.96 0.62-6.25	65	56	110	58	0.49 0.25-0.96	0.50 0.25-0.99
Heavy	18	20	25	33	0.51 0.16-1.62	0.77 0.20-2.92	21	18	45	24	0.50 0.23-1.12	0.62 0.27-1.43
<i>P</i> for trend					<i>p</i> =0.17	<i>p</i> =0.53					<i>p</i> =0.09	<i>p</i> =0.22

Odds ratios (OR) and 95% confidence intervals (CI), adjusted for age (years, continuous), education (some high school or less, high school graduate or technical school, some college, college graduate), first-degree family history of breast cancer (no, yes), prior biopsy-confirmed history of benign breast disease (no, yes), number of fullterm pregnancies (nulliparous, 1, 2, 3, 4), lifetime breastfeeding (nulliparous, 0, 6, 7-12, 13-24, 25 months), age at first full-term pregnancy (nulliparous, 19, 20-24, 25-29, 30 years), oral contraceptive use (never, <5, 5 years), adult height (cm, quartiles among controls), average alcohol consumption (0, 0.1-4.9, 5-9.9, 10-19.9, 20 g/day), and average caloric intake (kcal/day, quartiles among controls).

^b Adjusted additionally for current BMI (<25.0, 25.0-29.9, 30.0 kg/m²).

Table 4
Adolescent Body Size and Breast Cancer Risk in Premenopausal Hispanic Women, by Current BMI and Tumor Estrogen Receptor Status

	Current BMI (kg/m ²) <25.0				Current BMI (kg/m ²) 25.0				Estrogen receptor positive		Estrogen receptor negative	
	Cs N = 74	Cn N = 60	OR ^a , 95% CI	Cs N = 136	Cn N = 205	OR ^a , 95% CI	Cn N = 265	ER+ Cs N = 124	OR ^b , 95% CI	ER- Cs N = 52	OR ^b , 95% CI	
Relative weight compared to peers at age 10 years												
Lighter	48	30	1.00	61	70	1.00	100	68	1.00	23	1.00	
Same	26 ^c	30 ^c	0.51 0.16-1.62 ^c	49	79	0.73 0.41-1.29	106	38	0.50 0.29-0.88	19	0.87 0.41-1.83	
Heavier				34	43	0.61 0.30-1.24	46	18	0.58 0.26-1.26	8	0.81 0.29-2.33	
P for trend			p=0.26			p=0.14			p=0.04		p=0.66	
Relative weight compared to peers at age 15 years												
Lighter	41	27	1.00	50	42	1.00	69	57	1.00	20	1.00	
Same	33 ^c	32 ^c	0.89 0.30-2.62 ^c	63	104	0.47 0.26-0.86	129	49	0.41 0.23-0.73	26	0.71 0.33-1.51	
Heavier				22	54	0.26 0.12-0.56	61	18	0.29 0.13-0.63	5	0.22 0.07-0.76	
P for trend			p=0.83			p<0.01			p<0.01		p=0.02	
Relative weight compared to peers at age 20 years												
Lighter	42	28	1.00	38	39	1.00	37	45	1.00	19	1.00	
Same	32 ^c	32 ^c	0.99 0.34-2.86 ^c	70	92	0.92 0.49-1.73	120	57	0.87 0.48-1.57	24	1.01 0.46-2.22	
Heavier				27	69	0.42 0.21-0.87	73	22	0.58 0.27-1.24	8	0.37 0.13-1.06	
P for trend			p=0.980.02			p=0.02			p=0.17		p=0.09	
Body build at age 10 years												
Slim	60	44	1.00	87	125	1.00	169	89	1.00	35	1.00	
Average	13 ^d	15 ^d	0.30 0.08-1.10 ^d	32	54	0.89 0.48-1.64	69	27	0.68 0.36-1.26	7	0.52 0.20-1.37	
Heavy				15	22	1.01 0.45-2.26	22	7	0.67 0.24-1.87	9	2.00 0.72-5.52	
P for trend			p=0.07			p=0.89			p=0.24		p=0.55	
Body build at age 15 years												
Slim	38	26	1.00	50	64	1.00	90	54	1.00	21	1.00	
Average	35 ^d	34 ^d	1.47 0.54-3.98 ^d	61	102	0.70 0.40-1.25	132	54	0.65 0.37-1.12	21	0.79 0.37-1.67	

	Current BMI (kg/m ²) <25.0				Current BMI (kg/m ²) 25.0				Estrogen receptor positive		Estrogen receptor negative	
	Cs N = 74	Cn N = 60	OR ^a , 95% CI	Cs N = 136	Cn N = 205	OR ^a , 95% CI	Cn N = 265	ER+ Cs N = 124	OR ^b , 95% CI	ER- Cs N = 52	OR ^b , 95% CI	
Heavy				24	38	0.77 0.38-1.59	42	15	0.59 0.26-1.33	9	0.95 0.35-2.54	
<i>P</i> for trend			<i>p</i> =0.45			<i>p</i> =0.39			<i>p</i> =0.12		<i>p</i> =0.78	
Body build at age 20 years												
Slim	23	20	1.00	29	26	1.00	46	30	1.00	15	1.00	
Average	50 ^d	40 ^d	1.95 0.58-6.57 ^d	73	111	0.48 0.24-0.95	148	69	0.72 0.39-1.36	36	0.51 0.23-1.13	
Heavy				33	67	0.37 0.17-0.79	70	24	0.64 0.29-1.43	10	0.38 0.13-1.06	
<i>P</i> for trend			<i>p</i> =0.028			<i>p</i> =0.02			<i>p</i> =0.28		<i>p</i> =0.06	

^aOdds ratios (OR) and 95% confidence intervals (CI), adjusted for age (years, continuous), country of birth (US-born, foreign-born), education (some high school or less, high school graduate or technical school, some college, college graduate), first-degree family history of breast cancer (no, yes), prior biopsy-confirmed history of benign breast disease (no, yes), number of full-term pregnancies (nulliparous, 1, 2, 3, 4), lifetime breastfeeding (nulliparous, 0, 6, 7-12, 13-24, 25 months), age at first full-term pregnancy (nulliparous, 19, 20-24, 25-29, 30 years), oral contraceptive use (never, <5, 5 years), adult height (cm, quartiles among controls), average alcohol consumption (0, 0.1-4.9, 5-9.9, 10-19.9, 20 g/day), and average caloric intake (kcal/day, quartiles among controls).

^bAdjusted additionally for current BMI (<25.0, 25.0-29.9, 30.0 kg/m²).

^cCombines same and heavier relative weight categories.

^dCombines average and heavy body build categories.