HHS Public Access

Author manuscript

Cancer Causes Control. Author manuscript; available in PMC 2014 December 01.

Published in final edited form as:

Cancer Causes Control. 2013 December ; 24(12): 2231–2243. doi:10.1007/s10552-013-0302-1.

Body size in early life and breast cancer risk in African American and European American women

Elisa V. Bandera[1,2], **Urmila Chandran**[1,2], **Gary Zirpoli**[3], **Gregory Ciupak**[3], **Dana H. Bovbjerg**[4], **Lina Jandorf**[5], **Karen Pawlish**[6], **Jo L. Freudenheim**[7], and **Christine B. Ambrosone**[3]

[1]Rutgers Cancer Institute of New Jersey, 195 Little Albany St, New Brunswick, NJ 08903

[2]Rutgers School of Public Health, 683 Hoes Lane West, Piscataway, NJ 08854

[3]Department of Cancer Prevention & Control, Roswell Park Cancer Institute, Elm & Carlton Sts, Buffalo, NY 14263

[4]University of Pittsburgh Cancer Institute, University of Pittsburgh, 5115 Centre Ave, Pittsburgh, PA 15232

[5]Icahn School of Medicine at Mount Sinai, 1428 Madison Ave, New York, NY 10029

[6]New Jersey State Cancer Registry, New Jersey Department of Health, Trenton, NJ 08625

[7]Department of Social and Preventive Medicine, University at Buffalo, 270 Farber Hall, Buffalo, NY 14260-1660

Abstract

Purpose—There is growing evidence that body size in early life influences lifetime breast cancer risk, but little is known for African American (AA) women.

Methods—We evaluated body size during childhood and young adulthood and breast cancer risk among 1,751 cases [979 AA and 772 European American (EA)] and 1,673 controls (958 AA and 715 EA) in the Women's Circle of Health Study. Odds ratio (OR) and 95% confidence intervals (CI) were computed using logistic regression models while adjusting for potential covariates.

Results—Among AA women, being shorter at 7–8 y compared to peers was associated with increased postmenopausal breast cancer risk (OR: 1.68, 95% CI: 1.02–2.74), and being heavier at menarche with decreased postmenopausal breast cancer risk, although of borderline significance (OR: 0.45, 95% CI: 0.20–1.02). For EA women, being shorter from childhood through adolescence, particularly at menarche, was associated with reduced premenopausal breast cancer risk (OR: 0.55, 95% CI: 0.31–0.98). After excluding hormone replacement therapy users, an inverse association with postmenopausal breast cancer was found among EA women reporting to be heavier than their peers at menarche (OR: 0.18, 95% CI: 0.04–0.79). The inverse relationship

Corresponding author: Dr. Elisa V. Bandera, Rutgers Cancer Institute of New Jersey, 195 Little Albany St., New Brunswick, NJ 08903, elisa.bandera@rutgers.edu.

Conflict of interest: The authors declare that they have no conflict of interest.

between BMI at age 20 and breast cancer risk was stronger and only statistically significant in EA women. No clear association with weight gain since age 20 was found.

Conclusions—Findings suggest that the impact of childhood height on breast cancer risk may differ for EA and AA women and confirm the inverse association previously reported in EA populations with adolescent body fatness, in AA women.

Keywords

Breast cancer; African American; childhood; adolescence; young adult; BMI

Introduction

There is growing interest in the role of early life factors on breast cancer etiology, with strong experimental and epidemiologic evidence indicating that the time from peri-puberty to first childbirth is a critical period of susceptibility for breast cancer (1). During this period, the mammary gland is undergoing rapid division, which increases the opportunity for DNA damage and mutations (2). Furthermore, the mammary gland is not fully differentiated until the first full term pregnancy, which makes it more susceptible to carcinogens (3). Epidemiologic studies have generally found that greater adolescent height, growth rate, and final height are associated with increased lifetime breast cancer risk, while a higher body mass during adolescence and young adulthood is associated with lower lifetime risk (4–6). Weight gain since young adulthood has also been generally shown to increase postmenopausal breast cancer risk (7). However, the overall body of evidence for the role of body mass over the life-course is largely based on studies in European American (EA) women, with little known for African American (AA) women.

Obesity is currently a major public health concern in the United States, but particularly for AA women because of the much higher prevalence of overweight and obesity both in AA children and adults compared to white women. According to NHANES 2009–2010 data, 82.1% of Black women (vs. 59.5% of white women) (8) and 41.3% of Black girls (vs. 25.6% of white girls) were overweight or obese (9). At the same time, breast cancer in AA women tends to occur at an earlier age and to have more aggressive features associated with poor prognosis (10, 11), and some differences in the epidemiology of the disease have been noted between AA and EA women (12). The few studies that evaluated the impact of adult obesity on breast cancer risk in AA women have suggested that a higher body mass index may play a different role than in EAs, with most studies showing no association for adult BMI (13, 14). Only four studies have previously evaluated the role of BMI as a young adult on breast cancer risk in AA women, with inconclusive results (15–20).

The aims of this study were to evaluate the impact of body size at age 7–8 years, at menarche, at ages 15–16 years and young adulthood, as well as weight changes during adulthood on breast cancer risk among AA and EA women participating in the Women's Circle of Health Study. To our knowledge, this is the first study evaluating the impact of perceived body size during childhood and adolescence on breast cancer risk in AA women.

Methods

Study population

The Women's Circle of Health Study (WCHS) has been described elsewhere (21). In brief, the WCHS is a case-control study in New York City (NYC) and New Jersey (NJ). Cases were self-identified AA and Caucasian women, 20–75 years, able to complete an interview in English, with no previous history of cancer other than non-melanoma skin cancer, and with newly diagnosed, histologically confirmed ductal carcinoma in situ (DCIS) or invasive breast cancer, stage I-IV. Controls had no history of cancer other than non-melanoma skin cancer and met the same age and language eligibility criteria as cases. In NYC, recruitment took place between January 2002 and December 2008, with cases identified through the hospitals with the largest referral patterns for AA women. Controls in NYC were recruited using random digit dialing (RDD), with sampling based on the same telephone exchanges as cases receiving medical care at the participating hospitals. Controls were frequency matched to cases by age and race. In NJ, recruitment started in March 2006 and is ongoing. Phase I of the study (WCHS) covered seven counties in NJ and ended in April 2012, with Phase II extending recruitment of AA women to two additional counties for a total of nine counties (WCHS-2). Cases in NJ were identified by rapid case ascertainment conducted by the NJ State Cancer Registry. Controls were initially identified by RDD, frequency matched to cases by age group and county of residence. We complemented RDD to recruit AA controls in NJ using community-based recruitment. This was facilitated by working closely with AA breast cancer advocates, AA churches, senior citizen centers, and cancer support organizations such as the American Cancer Society. The community recruitment process and representativeness of the control group have been described in detail previously (22). For this analysis, we included women recruited up to December 2012.

Data collection

After confirming eligibility, an in-person interview was scheduled using the same training manual, methods and study materials at the two recruiting sites. The mean time between diagnosis and interview was 8.5 months. At the interview, informed consent, including a release to obtain medical records, pathology records, and tissue blocks from treating hospitals, was obtained. Several questionnaires were completed then, and body measurements and a saliva sample collected. The questionnaires included questions on established and suspected risk factors for breast cancer, including family and reproductive history, hormone use, alcohol intake, smoking, and occupational history. The questionnaire also assessed perceived weight (thinnest, much thinner, somewhat thinner, about the same, somewhat heavier, much heavier, heaviest) and height (shortest, much shorter, somewhat shorter, about the same, somewhat taller, much taller, and tallest) at age 7–8 years, at menarche, and 15–16 years, compared to other girls of the same age, based on questions from the Women's Interview Study of Health (23). Height and weight at age 20 years and one year before the reference date (date of diagnosis for cases and comparable date for controls) were also obtained. A food frequency questionnaire was used to assess dietary intake one year before the reference date.

Anthropometric measurements were taken at the interview. Height was measured once to the nearest 0.1 cm, and two measurements for waist and hip circumferences were obtained to the nearest 0.1 cm. Waist measurement was taken using a measuring tape around the waist covering the umbilicus, while hip measurement involved placing the measuring tape at the maximum extension of the buttocks in a horizontal plane. Body composition measures (lean and fat mass, percent body fat) were obtained by bioelectrical impedance analysis using a Tanita® TBF-300A scale. Weight was also obtained with the Tanita scale. Information on tumor hormone receptor status was abstracted from pathology reports received from the hospitals where surgeries were performed.

The study was approved by the Institutional Review Boards at the Cancer Institute of New Jersey (now Rutgers Cancer Institute of New Jersey), Mount Sinai School of Medicine (now the Icahn School of Medicine at Mount Sinai), the referring hospitals in NYC, and Roswell Park Cancer Institute and all participants provided written informed consent before participating in the study. A total of 1,751 cases (979 AA and 772 EA) and 1,673 controls (958 AA and 715 EA) completed the interview.

Statistical analyses

BMI was computed as weight in kilograms (kg) divided by the square of height in meters (m) and categorized according to the World Health Organization (WHO) International Classification. For analysis regarding weight gain we used race specific quantiles, with cutpoints based on the distribution of controls, as the distribution was considerably different in the two groups. We used the same cutpoints in pre- and postmenopausal women to be able to compare risk estimates across categories in the AA and EA groups.

Mean values for weight at age 20 and weight gain since age 20 were compared for AA and EA women using t-tests. Distributions for body size at different ages were compared using Chi-square tests. Multivariable unconditional logistic regression was used to estimate odds ratios (OR) and 95% confidence intervals (CI), controlling for relevant confounders. Tests for trend were derived by assigning the median value to each category. Covariates considered included age at reference date, ethnicity (Hispanic vs. not Hispanic), country of origin (United States, Caribbean countries, other), education, family history of breast cancer, history of benign breast disease, age at menarche, age at menopause, parity, breastfeeding (never/ever), age at first birth, hormone replacement therapy use (never/ever), oral contraceptive use (never/ever), and body size at other ages. For example, when evaluating relative weight at childhood and adolescence, we considered BMI at age 20 and current BMI as possible confounders.

We conducted stratified analyses by menopausal status and by major subtypes according to hormone receptor status [estrogen receptor (ER)+/progesterone receptor (PR) + and ER−/PR −]. We also repeated analyses excluding HRT users and cases with non-invasive tumors. Over 95% of the study population was non-Hispanic and therefore, stratified analyses by ethnicity could not be conducted.

SAS version 9.2 (SAS Institute, Cary NC) was used for analysis.

Results

Demographic, reproductive and lifestyle characteristics have been reported elsewhere (24). In summary, EA women tended to have higher levels of education, fewer children, and were less likely to be obese than AA women. Among AA women, cases were more likely to have been older at menopause and to have been HRT users. Among EA women, controls were more likely to have used oral contraceptives and to have breastfed their children. There were no major differences in age at menarche between cases and control in EA or AA women.

The distributions for relative height and weight, BMI at age 20, current BMI, and weight changes by case-control status and AA and EA women are shown in Table 1. There were no major differences in relative height between cases and controls or between AA and EA women. However, cases were more likely to report being thinner than peers compared to controls during the three age periods under evaluation, with the differences being larger and only statistically significant among AA women. On the other hand, both EA and AA cases were less likely to report being overweight and obese at age 20 than controls ($p<0.01$). For current BMI, while the percentage of AA overweight and obese was considerably higher than that for EA women, distributions were similar in cases and controls in the two groups. Most women reported having gained weight from age 20 to the time of interview, with AA women gaining more weight than EA women. However, there were no major differences in the average weight gained during this time period between cases and controls in either group.

Risk estimates and 95% confidence intervals for relative height and weight at various time points, BMI at age 20, and weight gain since age 20 for AA and EA women by menopausal status are presented in Tables 2 and 3. For AA women (Table 2), when we evaluated childhood and adolescent height and breast cancer risk, we found that, among postmenopausal women, childhood height was associated with increased risk (OR: 1.68; 95% CI: 1.02–2.74) for those reporting to be shortest or much shorter than girls their age when they were 7–8 y, after controlling for BMI at age 20 and other relevant covariates. When we adjusted for weight at age 7–8 y instead of BMI at age 20, risk estimates essentially did not change (data not shown). There was also some suggestion that being heavier during adolescence was associated with reduced postmenopausal breast cancer risk. Although there was a suggestion of an inverse association with BMI at age 20 for both pre- and postmenopausal women, risk estimates were attenuated when we included in the model relative weight and height at menarche and confidence intervals included the null. There was also a suggestion of increased risk with higher weight gain since age 20 for both pre- and postmenopausal women after adjusting for current BMI, but confidence intervals included the null.

For EA women (Table 3), we found a suggestion that being shorter from childhood through adolescence was associated with reduced premenopausal breast cancer risk, with a stronger and significant association for relative height at menarche. Being heavier than other girls at menarche also appeared to reduce postmenopausal breast cancer risk, but after further adjusting for BMI at age 20 y, risk estimates were no longer statistically significant. There was also a suggestion of decreased risk for postmenopausal women reporting to be thinner

than their peers. However, BMI at age 20 was strongly inversely associated with breast cancer risk in both pre- and postmenopausal women. There was also a trend of decreased breast cancer risk with higher weight gain among premenopausal women but confidence intervals included the null. Adjusting for current BMI did not have an impact in these analyses in either AA or EA women.

Because other studies have suggested that the association between body fatness and breast cancer may be restricted to non-HRT users (25, 26), we also repeated analyses among postmenopausal women excluding HRT users (Table 4). While results tended to be in the same direction, some of the associations noted above became stronger. For example, the OR was 1.95 (95% CI: 1.12–3.42) for AA women reporting to be shorter than their peers at age 7–8 y. Being heavier at menarche was strongly inversely associated with breast cancer risk for both AA and EA women with ORs of 0.23 (95% CI: 0.08–0.63) and 0.18 (95%: 0.04–0.79), respectively. However, no association was found with weight gain after excluding HRT users. Nevertheless, these analyses should be viewed with caution because they were based on small numbers and require replication.

We also conducted stratified analyses by the most common tumor subtypes according to hormone receptor status: ER+/PR+ and ER-/PR- (data not shown), but no major patterns emerged from these analyses. We also repeated analyses excluding DCIS cases (153 AA and 138 EA) and results were essentially the same.

Discussion

In this study we found that among AA women, among postmenopausal women, being shorter at age 7–8 y compared to peers was associated with increased breast cancer risk, while being heavier during adolescence and young adulthood was inversely associated with risk. For EA women, being shorter from childhood through adolescence, particularly at menarche, was associated with reduced premenopausal breast cancer risk, and, after excluding HRT users, a strong inverse association was found with postmenopausal breast cancer for those reporting to be heavier than their peers at menarche. Although there was a suggestion that BMI at age 20 was inversely related to breast cancer in AA and EA women, the association was stronger and statistically significant only in EA women. We did not find a clear association for weight gain since age 20 in either group. In our study, no clear pattern emerged when we assessed effect modification of pre-adolescent and adolescent body size and weight changes on breast cancer risk by ER/PR status, which has only been previously evaluated in a few studies (27–31) with inconsistent results.

There is growing evidence that the timing of exposure is critical in breast cancer etiology, with certain factors, such as estrogens, having different or even opposing effects on breast cancer risk depending on the period when exposure occurs (32). We evaluated risk for several critical periods: age 7–8 y, corresponding in most girls with the period right before thelarche, which marks the onset of puberty; age at menarche, which follows thelarche after a few years; age 15–16 y, at which most girls have attained their final height; and young adulthood. The hormonal processes triggering and regulating these developmental changes are not totally understood, but it is clear that estrogens (33) and Insulin-like Growth Factor

(IGF)-1 (34) play critical roles. Associations between childhood and adolescent growth, adiposity, sexual maturation milestones (such as thelarche and menarche), and breast cancer risk are complex. For example, rapid weight gain during childhood typically leads to accelerated growth, and taller girls tend to have an earlier onset of menses, which results, in turn, in accelerated skeletal maturation, and shorter height (34). However, this seems contradictory, as studies have also shown that both an earlier menarche and adult tallness are associated with increased breast cancer risk (3). A key to understanding these complex relationships might be the important influence of growth rate in breast cancer risk. Age at peak growth has been inversely associated with breast cancer risk in Danish women (35), while higher peak height velocity increased breast cancer risk in the Nurses' Health Study (36). Growth is a dynamic process and one single height measure may be just reflecting early hormonal markers and early life growth rates. Furthermore, adult height reflects *in-utero* and childhood development events (34), and therefore, the onset of menarche and final height might be serving as markers of early life events affecting breast cancer risk with different endocrine consequences.

One of the most striking findings in our study is the suggestion of opposite effects for childhood height on breast cancer risk in AA and EA women. Our results in EA women were in general agreement with the majority of previous studies in this population, which found decreased risk for shorter relative adolescent stature (37) or increased risk with greater measured height during childhood (35, 38)/adolescence (35, 38–40), or with higher peak height velocity (36, 41) or childhood growth rate (38). In contrast, some studies found no association with childhood stature (39, 42). However, our findings of an increased risk for postmenopausal breast cancer associated with shorter stature at age 7–8 y in AA women was unexpected. To our knowledge, this is the first study evaluating childhood and adolescent body size and breast cancer risk in AA women and, therefore, we cannot compare our results with other studies. Nevertheless, previous studies have reported important differences between AA and EA girls in growth and sexual development. For example, AA girls experience earlier onset of thelarche and menarche (43) and advance through the Tanner stages of development at younger ages (31). By age 8 y, 38% of AA girls and only 10% of white girls have reached the onset of puberty, according to data from the Pediatric Research in Office Settings (PROCS) Study (44). Therefore, AA and EA girls may be at different developmental stages at age 7–8 y. Also, height at that age may be reflecting postnatal weight gain, as it has been suggested that height trajectory may be established during the first 2 years of life (45). Furthermore, factors affecting development or serving as markers of the various development stages may differ in AA and EA women. When evaluating predictors of earlier menarche in AA, BMI explained less than 20% of the overall variation in age at menarche, while childhood poverty reduced the age at menarche only in whites but not in AA girls (46). On the other hand, shorter height has been associated with early life experiences of psychological stress (e.g., family tension, divorce, separation or desertion), socioeconomic factors, and chronic illness and infections (47), which may in turn increase breast cancer risk by affecting hormonal and immune factors.

Our study generally supported earlier findings that being overweight in early-life decreases breast cancer risk in EA women (reviewed in (6)), independent of age at menarche and

current BMI. To our knowledge, this is the first study evaluating relative weight and height during childhood and at menarche and breast cancer risk in AA women, and we found associations similar to those previously reported in EA (6) and Hispanic women (30, 48).

The inverse association between childhood obesity and breast cancer risk seems to be independent of the age at menarche, as shown in the study by Ahlgren et al. (35) as well as in our study. Overweight adolescent girls, despite having earlier onset of puberty, have been shown to experience slower pubertal growth and sexual maturation (36) and longer time to regular menstrual cycles (49), which may results in more frequent anovulatory cycles and lower exposure to ovarian hormones. While the biological mechanisms are unknown, some investigators have proposed that the inverse association with childhood and adolescent BMI may be mediated by a reduction in IGF-1 levels (50). IGF-1 has been shown to be strongly associated with breast cancer risk (4) and a recent study found that birth weight, body fatness in childhood and BMI at age 18 y were inversely associated with adult IGF-1 levels (50). It has also been suggested that exposure to estrogens at this early age may induce differentiation of the breast epithelium (40).

The association of breast cancer risk with BMI in young adulthood (18–21 yrs) has been evaluated in a few studies, mostly conducted in EA women, with inconsistent results. An inverse association in EA was reported in the Nurses' Health Study for premenopausal breast cancer (27) and in the NIH-AARP Diet and Health Study for postmenopausal women (51), but no association with breast cancer was found in other studies (19, 30, 52, 53). For AA women, after excluding HRT users and adjusting for current BMI, there was no evidence of an association. Results from the few previous studies that evaluated BMI at ages 18–21 yrs and breast cancer risk in AA women are inconsistent, with some finding an inverse association for pre- and postmenopausal women (17), for pre-menopausal women (18) or no association (15, 16, 19, 20).

An association between weight gain and postmenopausal breast cancer has been fairly consistently reported in previous studies, largely conducted in EA women (7). In contrast, we found no evidence that weight gain since young adulthood increased risk in EA or AA women after we adjusted for current BMI and excluded HRT users. No association with weight gain in AA women was also reported in the Black Women's Health Study (17) or in the Women's CARE Study (18). In contrast, increased risk for postmenopausal AA and EA women was found in the Multiethnic Cohort (20).

A limitation of the current study is that data on body size in childhood, adolescence and young adulthood as well as weight changes were based on self-report and recall, employing face valid questions from the Women's Interview Study of Health (23). As a result, there may be both inaccuracies in recall of the body measures as well as possible differential error for cases and controls. Furthermore, we collected data on relative body size at childhood, menarche, and adolescence compared to other girls of the same age, rather than actual body measures. This method has been shown to be more accurate than ascertaining actual weight and height in the distant past, which varies much from year to year during pubertal growth (54). Moreover, a strong correlation has been reported between long-term recall of perceived body size at menarche and weight and height at menarche collected in monthly

questionnaires from mothers approximately 30 years earlier (Spearman's r=0.6, p<0.01) (54). Another study also found a strong correlation between long-term recall of body size and actual weight during adolescence (Pearson's r=0.87) (55). However, the validity of recall in AA women or whether recall differs in cases and controls has not been determined, therefore, the possibility of recall bias remains. Nevertheless, several studies that measured height and weight during childhood or adolescence (rather than basing body size on recall) among EA also found that higher body mass was associated with lower breast cancer risk (35, 38, 40, 42).

Conclusions

Our study suggested that childhood height may play a different role in breast cancer risk in EA and AA women. These novel findings warrant further research, particularly with a more detailed and longitudinal evaluation of growth rates through the early childhood and pubertal periods. The current evidence suggests that higher adiposity during early years and young adulthood may decrease breast cancer risk, which is of interest in providing insight into the etiology of breast cancer. Nonetheless, because childhood obesity tends to persist in adulthood (56) and adult obesity has been associated with increased risk of type II diabetes, cardiovascular disease (57), and several types of cancer (58), maintaining a healthy weight throughout the life-course should be a goal for overall health.

Acknowledgments

This work was supported by grants from the US Army Medical Research and Material Command (DAMD-17-01-1-0334), the National Cancer Institute (R01 CA100598,P01 CA151135, K22 CA138563, P30CA072720, P30 CA016056), the Breast Cancer Research Foundation and a gift from the Philip L Hubbell family. The New Jersey State Cancer Registry (NJSCR) is a participant in the Centers for Disease Control and Prevention's National Program of Cancer Registries and is a National Cancer Institute Surveillance, Epidemiology, and End Results (SEER) Expansion Registry. The NJSCR is supported by the Centers for Disease Control and Prevention under cooperative agreement 1US58DP003931-01 awarded to the New Jersey Department of Health. The collection of New Jersey cancer incidence data is also supported by the National Cancer Institute's SEER Program under contract N01-PC-2010-00027 and the State of New Jersey. The funding agents played no role in design, in the collection, analysis, and interpretation of data, in the writing of the manuscript, or in the decision to submit the manuscript for publication.

We thank the colleagues, physicians and clinical staff in New York and New Jersey who facilitated identification and enrollment of cases into the study: Kandace Amend (i3 Drug Safety), Helena Furberg (Memorial Sloan-Kettering Cancer Center), Thomas Rohan and Joseph Sparano (Albert Einstein College of Medicine), Paul Tartter and Alison Estabrook (St. Luke's Roosevelt Hospital), James Reilly (Kings County Hospital Center), Benjamin Pace, George Raptis, and Christina Weltz (Mount Sinai School of Medicine), Maria Castaldi (Jacob Medical Center), Sheldon Feldman (New York-Presbyterian), and Margaret Kemeny (Queens Hospital Center). We also thank our research personnel at the Rutgers Cancer Institute of New Jersey, Roswell Park Cancer Institute, Mount Sinai School of Medicine, Rutgers School of Public Health, and the New Jersey State Cancer Registry, as well as our African American breast cancer advocates and community partners, and all the women who generously donated their time to participate in the study.

List of abbreviations

AA	African ancestry
EA	European ancestry
WCHS	Women's Circle of Health Study

BMI	body mass index
OR	odds ratio
CI	confidence interval
HRT	hormone replacement therapy
NYC	New York City
NJ	New Jersey
DCIS	ductal carcinoma in situ
RDD	random digit dialing
WHO	World Health Organization
ER	estrogen receptor
PR	progesterone receptor
IGF	insulin growth factor

References

1. Colditz GA, Frazier AL. Models of breast cancer show that risk is set by events of early life: prevention efforts must shift focus. Cancer Epidemiol Biomarkers Prev. 1995; 4:567–71. [PubMed: 7549816]
2. Michels KB, Willett WC. Breast cancer--early life matters. N Engl J Med. 2004; 351:1679–81. [PubMed: 15483288]
3. Ruder EH, Dorgan JF, Kranz S, Kris-Etherton PM, Hartman TJ. Examining breast cancer growth and lifestyle risk factors: early life, childhood, and adolescence. Clin Breast Cancer. 2008; 8:334–42. [PubMed: 18757260]
4. Uauy R, Solomons N. Diet, nutrition, and the life-course approach to cancer prevention. J Nutr. 2005; 135:2934S–45S. [PubMed: 16382507]
5. Forman MR, Cantwell MM, Ronckers C, Zhang Y. Through the looking glass at early-life exposures and breast cancer risk. Cancer Invest. 2005; 23:609–24. [PubMed: 16305989]
6. Fuemmeler BF, Pendzich MK, Tercyak KP. Weight, dietary behavior, and physical activity in childhood and adolescence: implications for adult cancer risk. Obes Facts. 2009; 2:179–86. [PubMed: 20054223]
7. World Cancer Research Fund/American Institute for Cancer Research. Continuous Update: Project Report Summary. Food, Nutrition, Physical Activity, and the Prevention of Breast Cancer. London, UK: World Cancer Research Fund International; 2010.
8. Flegal KM, Carroll MD, Kit BK, Ogden CL. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999–2010. JAMA. 2012; 307:491–7. [PubMed: 22253363]
9. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity and trends in body mass index among US children and adolescents, 1999–2010. JAMA. 2012; 307:483–90. [PubMed: 22253364]
10. Amend K, Hicks D, Ambrosone CB. Breast cancer in African-American women: differences in tumor biology from European-American women. Cancer Res. 2006; 66:8327–30. [PubMed: 16951137]
11. Desantis C, Naishadham D, Jemal A. Cancer statistics for African Americans, 2013. CA Cancer J Clin. 2013 Epub ahead of print.
12. Millikan RC, Newman B, Tse CK, et al. Epidemiology of basal-like breast cancer. Breast Cancer Res Treat. 2008; 109:123–39. [PubMed: 17578664]

13. Chandran U, Hirshfield KM, Bandera EV. The role of anthropometric and nutritional factors on breast cancer risk in African-American women. Public Health Nutr. 2012; 15:738–48. [PubMed: 22122844]
14. Sexton KR, Franzini L, Day RS, Brewster A, Vernon SW, Bondy ML. A review of body size and breast cancer risk in Hispanic and African American women. Cancer. 2011; 117:5271–81. [PubMed: 21598244]
15. Mayberry RM. Age-specific patterns of association between breast cancer and risk factors in black women, ages 20 to 39 and 40 to 54. Annals of epidemiology. 1994; 4:205–13. [PubMed: 8055121]
16. Zhu K, Caulfield J, Hunter S, Roland CL, Payne-Wilks K, Texter L. Body mass index and breast cancer risk in African American women. Annals of epidemiology. 2005; 15:123–8. [PubMed: 15652717]
17. Palmer JR, Adams-Campbell LL, Boggs DA, Wise LA, Rosenberg L. A prospective study of body size and breast cancer in black women. Cancer Epidemiol Biomarkers Prev. 2007; 16:1795–802. [PubMed: 17855697]
18. Berstad P, Coates RJ, Bernstein L, 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]
19. 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. 2011; 173:201–16. [PubMed: 21084558]
20. White KK, Park SY, Kolonel LN, Henderson BE, Wilkens LR. Body size and breast cancer risk: the Multiethnic Cohort. Int J Cancer. 2012; 131:E705–16. [PubMed: 22120517]
21. Ambrosone CB, Ciupak GL, Bandera EV, et al. Conducting Molecular Epidemiological Research in the Age of HIPAA: A Multi-Institutional Case-Control Study of Breast Cancer in African-American and European-American Women. J Oncol. 2009; 2009:871250. [PubMed: 19865486]
22. Bandera EV, Chandran U, Zirpoli G, McCann SE, Ciupak G, Ambrosone CB. Rethinking sources of representative controls for the conduct of case-control studies in minority populations. BMC Med Res Methodol. 2013; 13:71. [PubMed: 23721229]
23. Brinton LA, Swanson CA. Height and weight at various ages and risk of breast cancer. Annals of epidemiology. 1992; 2:597–609. [PubMed: 1342311]
24. Gong Z, Ambrosone CB, McCann SE, et al. Associations of dietary folate, vitamin B6, B12 and methionine intake with risk of breast cancer among African American (AA) and European American (EA) women. Int J Cancer. 2013
25. van den Brandt PA, Spiegelman D, Yaun SS, et al. Pooled analysis of prospective cohort studies on height, weight, and breast cancer risk. Am J Epidemiol. 2000; 152:514–27. [PubMed: 10997541]
26. Morimoto LM, White E, Chen Z, 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]
27. 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]
28. Bardia A, Vachon CM, Olson JE, et al. Relative weight at age 12 and risk of postmenopausal breast cancer. Cancer Epidemiol Biomarkers Prev. 2008; 17:374–8. [PubMed: 18250344]
29. 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]
30. Slattery ML, Sweeney C, Edwards S, 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]
31. Hui SL, Perkins AJ, Harezlak J, Peacock M, McClintock CL, Johnston CC Jr. Velocities of bone mineral accrual in black and white American children. J Bone Miner Res. 2010; 25:1527–35. [PubMed: 20200959]
32. Hilakivi-Clarke L, Cho E, de Assis S, et al. Maternal and prepubertal diet, mammary development and breast cancer risk. J Nutr. 2001; 131:154S–7S. [PubMed: 11208953]

33. Carel JC, Lahlou N, Roger M, Chaussain JL. Precocious puberty and statural growth. Hum Reprod Update. 2004; 10:135–47. [PubMed: 15073143]
34. Okasha M, Gunnell D, Holly J, Davey Smith G. Childhood growth and adult cancer. Best Pract Res Clin Endocrinol Metab. 2002; 16:225–41. [PubMed: 12064890]
35. Ahlgren M, Melbye M, Wohlfahrt J, Sorensen TI. Growth patterns and the risk of breast cancer in women. Int J Gynecol Cancer. 2006; 16(Suppl 2):569–75. [PubMed: 17010075]
36. Berkey CS, Frazier AL, Gardner JD, Colditz GA. Adolescence and breast carcinoma risk. Cancer. 1999; 85:2400–9. [PubMed: 10357411]
37. Coates RJ, Uhler RJ, Hall HI, 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]
38. De Stavola BL, dos Santos Silva I, McCormack V, Hardy RJ, Kuh DJ, Wadsworth ME. Childhood growth and breast cancer. Am J Epidemiol. 2004; 159:671–82. [PubMed: 15033645]
39. Herrinton LJ, Husson G. Relation of childhood height and later risk of breast cancer. Am J Epidemiol. 2001; 154:618–23. [PubMed: 11581095]
40. Hilakivi-Clarke L, Forsen T, Eriksson JG, et al. Tallness and overweight during childhood have opposing effects on breast cancer risk. Br J Cancer. 2001; 85:1680–4. [PubMed: 11742488]
41. Li CI, Malone KE, White E, Daling JR. Age when maximum height is reached as a risk factor for breast cancer among young U.S. women. Epidemiology. 1997; 8:559–65. [PubMed: 9270959]
42. Le Marchand L, Kolonel LN, Earle ME, Mi MP. Body size at different periods of life and breast cancer risk. Am J Epidemiol. 1988; 128:137–52. [PubMed: 3381822]
43. Biro FM, Huang B, Crawford PB, et al. Pubertal correlates in black and white girls. J Pediatr. 2006; 148:234–40. [PubMed: 16492435]
44. Herman-Giddens ME, Kaplowitz PB, Wasserman R. Navigating the recent articles on girls' puberty in Pediatrics: what do we know and where do we go from here? Pediatrics. 2004; 113:911–7. [PubMed: 15060243]
45. Dunger DB, Ahmed ML, Ong KK. Effects of obesity on growth and puberty. Best Pract Res Clin Endocrinol Metab. 2005; 19:375–90. [PubMed: 16150381]
46. Reagan PB, Salsberry PJ, Fang MZ, Gardner WP, Pajer K. African-American/white differences in the age of menarche: accounting for the difference. Soc Sci Med. 2012; 75:1263–70. [PubMed: 22726619]
47. Batty GD, Shipley MJ, Gunnell D, et al. Height, wealth, and health: an overview with new data from three longitudinal studies. Econ Hum Biol. 2009; 7:137–52. [PubMed: 19628438]
48. Sangaramoorthy M, Phipps AI, Horn-Ross PL, Koo J, John EM. Early-life factors and breast cancer risk in Hispanic women: the role of adolescent body size. Cancer Epidemiol Biomarkers Prev. 2011; 20:2572–82. [PubMed: 22056503]
49. 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]
50. Poole EM, Tworoger SS, Hankinson SE, Schernhammer ES, Pollak MN, Baer HJ. Body size in early life and adult levels of insulin-like growth factor 1 and insulin-like growth factor binding protein 3. Am J Epidemiol. 2011; 174:642–51. [PubMed: 21828371]
51. Ahn J, Schatzkin A, Lacey JV Jr, et al. Adiposity, adult weight change, and postmenopausal breast cancer risk. Arch Intern Med. 2007; 167:2091–102. [PubMed: 17954804]
52. Weiderpass E, Braaten T, Magnusson C, 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]
53. Trentham-Dietz A, Newcomb PA, Storer BE, et al. Body size and risk of breast cancer. Am J Epidemiol. 1997; 145:1011–9. [PubMed: 9169910]
54. Must A, Phillips SM, Naumova EN, 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]

55. Casey VA, Dwyer JT, Berkey CS, Coleman KA, Gardner J, Valadian I. Long-term memory of body weight and past weight satisfaction: a longitudinal follow-up study. Am J Clin Nutr. 1991; 53:1493–8. [PubMed: 2035478]

56. Dixon B, Pena MM, Taveras EM. Lifecourse approach to racial/ethnic disparities in childhood obesity. Adv Nutr. 2012; 3:73–82. [PubMed: 22332105]

57. Ogden CL, Yanovski SZ, Carroll MD, Flegal KM. The epidemiology of obesity. Gastroenterology. 2007; 132:2087–102. [PubMed: 17498505]

58. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity and the Prevention of Cancer: A Global Perpective. Washington, DC: AICR; 2007.

Table 1

Body size in early life among participants in the Women's Circle of Health Study.

	AA women (n=1937)			EA women (n=1487)		
	Cases	Controls	p value	Cases	Controls	p value
	n (%)	n (%)		n (%)	n (%)	
Relative height at age 7 or 8*			0.20			0.27
Shortest	61 (6.3)	46 (4.8)		41 (5.3)	59 (8.3)	
Much shorter	26 (2.7)	22 (2.3)		35 (4.6)	33 (4.6)	
Somewhat shorter	98 (10)	123 (12.8)		93 (12.1)	95 (13.3)	
About the same	525 (53.8)	540 (56.4)		350 (45.5)	296 (41.5)	
Somewhat taller	128 (13.1)	113 (11.8)		141 (18.3)	119 (16.7)	
Much taller	45 (4.6)	37 (3.9)		53 (6.9)	53 (7.4)	
Tallest	93 (9.5)	77 (8)		57 (7.4)	59 (8.3)	
Relative height at menarche*			0.20			0.12
Shortest	46 (4.7)	39 (4.1)		26 (3.4)	40 (5.6)	
Much shorter	27 (2.8)	21 (2.2)		27 (3.5)	26 (3.6)	
Somewhat shorter	102 (10.5)	118 (12.3)		100 (13)	111 (15.6)	
About the same	528 (54.1)	535 (56)		360 (46.8)	312 (43.7)	
Somewhat taller	134 (13.7)	135 (14.1)		141 (18.3)	116 (16.3)	
Much taller	51 (5.2)	50 (5.2)		66 (8.6)	51 (7.1)	
Tallest	88 (9)	58 (6.1)		49 (6.4)	58 (8.1)	
Relative height at age 15 or 16*			0.19			0.08
Shortest	43 (4.4)	37 (3.9)		24 (3.1)	35 (4.9)	
Much shorter	25 (2.6)	19 (2.00)		32 (4.2)	28 (3.9)	
Somewhat shorter	112 (11.5)	137 (14.3)		130 (16.9)	152 (21.3)	
About the same	575 (58.9)	566 (59.1)		381 (49.4)	315 (44.1)	
Somewhat taller	113 (11.6)	120 (12.5)		118 (15.3)	115 (16.1)	
Much taller	43 (4.4)	31 (3.2)		51 (6.6)	35 (4.9)	
Tallest	66 (6.8)	48 (5)		35 (4.5)	35 (4.9)	
Relative weight at age 7 or 8*			0.002			0.50
Thinnest	85 (8.7)	92 (9.6)		65 (8.4)	55 (7.7)	
Much thinner	80 (8.2)	53 (5.5)		74 (9.6)	65 (9.1)	
Somewhat thinner	244 (25.1)	189 (19.7)		161 (20.9)	128 (17.9)	
About the same	426 (43.7)	441 (46)		330 (42.8)	312 (43.6)	
Somewhat heavier	107 (11)	143 (14.9)		116 (15.1)	120 (16.8)	
Much heavier	22 (2.3)	20 (2.1)		20 (2.6)	28 (3.9)	
Heaviest	10 (1)	20 (2.1)		5 (0.7)	7 (1)	
Relative weight at menarche*			<0.001			0.08
Thinnest	72 (7.4)	80 (8.4)		46 (6)	42 (5.9)	
Much thinner	79 (8.1)	49 (5.1)		59 (7.7)	60 (8.4)	

	AA women (n=1937)			EA women (n=1487)		
	Cases	**Controls**	**p value**	**Cases**	**Controls**	**p value**
	n (%)	**n (%)**		**n (%)**	**n (%)**	
Somewhat thinner	217 (22.3)	174 (18.2)		159 (20.6)	126 (17.7)	
About the same	461 (47.3)	441 (46)		345 (44.8)	286 (40.1)	
Somewhat heavier	121 (12.4)	171 (17.9)		138 (17.9)	168 (23.5)	
Much heavier	17 (1.7)	28 (2.9)		18 (2.3)	24 (3.4)	
Heaviest	8 (0.8)	15 (1.6)		6 (0.8)	8 (1.1)	
Relative weight at 15 or 16*			<0.001			0.11
Thinnest	62 (6.4)	71 (7.4)		39 (5.1)	38 (5.3)	
Much thinner	70 (7.2)	40 (4.2)		48 (6.2)	54 (7.6)	
Somewhat thinner	198 (20.3)	155 (16.2)		167 (21.6)	126 (17.6)	
About the same	499 (51.1)	489 (51)		375 (48.6)	325 (45.5)	
Somewhat heavier	123 (12.6)	157 (16.4)		121 (15.7)	142 (19.9)	
Much heavier	18 (1.8)	32 (3.3)		17 (2.2)	23 (3.2)	
Heaviest	6 (0.6)	14 (1.5)		5 (0.7)	7 (1)	
BMI at age 20 yrs.			0.002			0.001
Underweight/Normal (<25)	739 (82.6)	705 (76.9)		699 (93)	625 (88.7)	
Overweight (25–29.99)	116 (13)	139 (15.2)		44 (5.9)	49 (7)	
Obese (≥30)	40 (4.5)	73 (8)		9 (1.2)	31 (4.4)	
Current BMI			0.99			0.99
Underweight/Normal (<25)	176 (18)	171 (17.9)		353 (45.7)	328 (45.9)	
Overweight (25–29.99)	281 (28.7)	274 (28.6)		209 (27.1)	191 (26.8)	
Obese (≥30)	521 (53.3)	513 (53.6)		210 (27.2)	195 (27.3)	
Weight change since age 20			0.75			0.31
Lost weight	46 (5.2)	50 (5.5)		87 (11.8)	94 (13.6)	
Stable weight	0	0		0	0	
Gained weight	846 (94.8)	861 (94.5)		649 (88.2)	596 (86.4)	
	mean±SD	**mean±SD**		**mean±SD**	**mean±SD**	
Weight at age 20 yrs (kg)	58.65±11.71	61.38±14.30	<0.001	56.14±7.83	57.74±10.34	0.001
Weight gain since age 20 (kg)	26.62±16.11	25.96±16.63	0.40	17.75±13.86	17.94±14.35	0.81

* compared to peers

Table 2

Early life body size and breast cancer risk by menopausal status in AA women, Women's Circle of Health Study.

	Pre-menopausal AA women (n=953)					Post-menopausal AA women (n=984)				
	Cases/Controls	**OR1**	**95% CI**	**OR2**	**95% CI**	**Cases/Controls**	**OR1**	**95% CI**	**OR2**	**95% CI**
Relative height at 7 or 8[*a]										
Shortest/much shorter	34/34	1.29	0.76–2.20	1.20	0.70–2.07	53/34	1.56	0.96–2.53	1.68	1.02–2.74
Somewhat shorter	46/62	0.92	0.59–1.44	0.85	0.53–1.37	52/61	0.83	0.54–1.28	0.92	0.59–1.43
About the same	248/276	REF		REF		277/264	REF		REF	
Somewhat taller	65/51	1.52	0.99–2.32	1.43	0.93–2.22	63/62	1.02	0.68–1.54	1.07	0.70–1.63
Tallest/much taller	77/59	1.44	0.96–2.15	1.39	0.92–2.10	61/55	1.17	0.77–1.80	1.16	0.75–1.79
Relative height at menarche[*a]										
Shortest/much shorter	31/30	1.15	0.66–2.00	1.14	0.65–2.00	42/30	1.42	0.84–2.40	1.51	0.89–2.57
Somewhat shorter	43/62	0.83	0.53–1.31	0.84	0.52–1.34	59/56	1.08	0.70–1.65	1.18	0.76–1.82
About the same	249/268	REF		REF		279/267	REF		REF	
Somewhat taller	71/61	1.34	0.89–2.00	1.36	0.90–2.06	63/74	0.89	0.60–1.32	0.91	0.61–1.37
Tallest/much taller	75/60	1.33	0.89–1.99	1.26	0.83–1.91	64/48	1.38	0.89–2.12	1.36	0.87–2.11
Relative height at 15 or 16[*a]										
Shortest/much shorter	31/28	1.27	0.72–2.24	1.24	0.70–2.21	37/28	1.31	0.76–2.25	1.44	0.83–2.51
Somewhat shorter	51/72	0.84	0.56–1.28	0.87	0.57–1.34	61/65	1.05	0.70–1.57	1.16	0.77–1.76
About the same	271/284	REF		REF		304/282	REF		REF	
Somewhat taller	61/57	1.26	0.83–1.92	1.21	0.79–1.87	52/63	0.85	0.56–1.31	0.87	0.56–1.36
Tallest/much taller	56/41	1.37	0.86–2.17	1.25	0.78–2.02	53/38	1.43	0.89–2.29	1.40	0.86–2.26
Relative weight at 7 or 8[*a]										
Thinnest/much thinner	82/71	1.24	0.84–1.85	1.08	0.71–1.65	83/74	1.13	0.77–1.66	1.04	0.69–1.55
Somewhat thinner	122/104	1.22	0.87–1.72	1.15	0.81–1.65	122/85	1.50	1.06–2.13	1.44	1.01–2.07
About the same	195/207	REF		REF		231/234	REF		REF	
Somewhat heavier	51/82	0.74	0.48–1.12	0.75	0.48–1.17	56/61	0.87	0.56–1.34	0.90	0.57–1.44
Heaviest/much heavier	20/18	1.25	0.62–2.52	1.40	0.67–2.92	12/22	0.52	0.24–1.11	0.54	0.23–1.29
Relative weight at menarche[*a]										
Thinnest/much thinner	74/59	1.27	0.84–1.92	1.22	0.79–1.88	77/70	1.06	0.71–1.57	0.97	0.64–1.46

	Pre-menopausal AA women (n=953)					Post-menopausal AA women (n=984)				
	Cases/Controls	**OR1**	**95% CI**	**OR2**	**95% CI**	**Cases/Controls**	**OR1**	**95% CI**	**OR2**	**95% CI**
Somewhat thinner	107/91	1.12	0.78–1.59	1.02	0.70–1.48	110/83	1.29	0.91–1.85	1.17	0.81–1.69
About the same	212/214	REF		REF		249/227	REF		REF	
Somewhat heavier	63/100	0.70	0.47–1.03	0.71	0.47–1.08	58/71	0.85	0.56–1.29	0.85	0.54–1.32
Heaviest/much heavier	14/18	0.86	0.40–1.83	0.89	0.39–2.00	11/25	0.40	0.18–0.86	0.45	0.20–1.02
Relative weight at 15 or 16[*a]										
Thinnest/much thinner	64/54	1.19	0.78–1.83	1.18	0.75–1.86	68/57	1.20	0.79–1.82	1.12	0.72–1.73
Somewhat thinner	96/79	1.19	0.82–1.73	1.14	0.77–1.68	102/76	1.39	0.97–2.00	1.31	0.90–1.91
About the same	231/236	REF		REF		268/253	REF		REF	
Somewhat heavier	65/91	0.77	0.52–1.13	0.79	0.52–1.20	58/66	0.94	0.62–1.42	0.93	0.59–1.48
Heaviest/much heavier	14/22	0.63	0.30–1.30	0.62	0.28–1.39	10/24	0.42	0.19–0.92	0.49	0.20–1.20
BMI at age 20 yrs.[b]										
<25	347/340	REF		REF		392/365	REF		REF	
25–29.9	64/75	0.89	0.61–1.31	1.02	0.67–1.56	52/64	0.87	0.57–1.31	1.01	0.65–1.58
≥30	23/47	0.65	0.37–1.12	0.77	0.42–1.40	17/26	0.65	0.34–1.25	0.88	0.43–1.81
p for trend			0.12		0.52			0.16		0.82
Weight gain since age 20, kg[c]										
Q 1 (≤13.82)	114/130	REF		REF		75/84	REF		REF	
Q 2 (13.83–23.72)	118/111	1.15	0.78–1.68	1.27	0.86–1.89	115/106	1.30	0.84–2.00	1.35	0.87–2.10
Q 3 (23.73–34.56)	93/102	0.93	0.62–1.38	1.15	0.73–1.82	110/112	1.20	0.77–1.85	1.29	0.80–2.09
Q 4 (>34.56)	82/91	0.96	0.63–1.47	1.49	0.81–2.73	139/125	1.22	0.80–1.86	1.42	0.80–2.53
p for trend			0.63		0.27			0.60		0.34

[*] compared to peers

OR1: Adjusted for age, ethnicity (Hispanic/non-Hispanic), country of origin, education, family history of breast cancer, history of benign breast disease, age at menarche, age at menopause (for postmenopausal women), parity, breastfeeding status, age at first birth, HRT use, oral contraceptive use.

OR2:

[a] further adjusted for BMI at age 20;

[b] further adjusted for height and weight at menarche;

[c] further adjusted for current BMI

Table 3

Early life body size and breast cancer risk by menopausal status in EA women, Women's Circle of Health Study.

	Pre-menopausal EA women (n=794)					Post-menopausal EA women (n=693)				
	Cases/Controls	**OR**	**95% CI**	**OR2**	**95% CI**	**Cases/Controls**	**OR**	**95% CI**	**OR**	**95% CI**
Relative height at 7 or 8[*a]										
Shortest/much shorter	41/56	0.65	0.40–1.06	0.63	0.38–1.04	35/36	1.01	0.57–1.78	1.00	0.56–1.77
Somewhat shorter	51/52	0.93	0.58–1.49	0.90	0.56–1.46	42/43	0.92	0.54–1.57	0.91	0.53–1.56
About the same	170/151	REF		REF		180/145	REF		REF	
Somewhat taller	76/70	0.98	0.64–1.48	1.00	0.65–1.52	65/49	1.34	0.83–2.17	1.32	0.81–2.15
Tallest/much taller	62/64	0.85	0.55–1.32	0.82	0.52–1.30	48/48	1.00	0.61–1.67	1.02	0.61–1.71
Relative height at menarche[*a]										
Shortest/much shorter	26/41	0.57	0.32–1.01	0.55	0.31–0.98	27/25	1.15	0.61–2.18	1.24	0.65–2.38
Somewhat shorter	46/62	0.70	0.44–1.10	0.66	0.41–1.06	54/49	1.34	0.81–2.20	1.37	0.83–2.25
About the same	184/160	REF		REF		176/152	REF		REF	
Somewhat taller	77/71	0.96	0.64–1.45	0.96	0.63–1.45	64/45	1.41	0.86–2.29	1.38	0.84–2.27
Tallest/much taller	65/59	0.96	0.62–1.50	0.93	0.59–1.47	50/50	1.11	0.67–1.82	1.07	0.64–1.77
Relative height at 15 or 16[*a]										
Shortest/much shorter	29/40	0.63	0.36–1.10	0.61	0.35–1.07	27/23	1.11	0.58–2.12	1.16	0.60–2.23
Somewhat shorter	62/84	0.67	0.45–1.02	0.65	0.43–0.99	68/68	1.00	0.64–1.57	1.07	0.68–1.70
About the same	193/168	REF		REF		188/147	REF		REF	
Somewhat taller	66/62	0.82	0.53–1.26	0.78	0.50–1.22	52/53	0.88	0.54–1.44	0.88	0.54–1.45
Tallest/much taller	50/39	1.07	0.65–1.75	1.05	0.63–1.76	36/31	1.17	0.66–2.09	1.12	0.62–2.01
Relative weight at 7 or 8[*a]										
Thinnest/much thinner	81/72	1.08	0.71–1.64	1.06	0.69–1.62	58/48	0.85	0.51–1.42	0.70	0.41–1.18
Somewhat thinner	79/67	1.22	0.81–1.84	1.21	0.80–1.84	82/61	0.86	0.55–1.35	0.72	0.45–1.14
About the same	169/185	REF		REF		161/127	REF		REF	
Somewhat heavier	58/50	1.30	0.82–2.06	1.49	0.92–2.40	58/70	0.60	0.38–0.96	0.67	0.41–1.10
Heaviest/much heavier	14/19	0.86	0.41–1.84	0.96	0.43–2.12	11/16	0.51	0.21–1.22	0.72	0.29–1.84
Relative weight at menarche[*a]										
Thinnest/much thinner	64/65	0.73	0.47–1.14	0.68	0.43–1.08	41/37	0.66	0.37–1.16	0.56	0.31–1.02

	Pre-menopausal EA women (n=794)					Post-menopausal EA women (n=693)				
	Cases/Controls	**OR**	**95% CI**	**OR2**	**95% CI**	**Cases/Controls**	**OR**	**95% CI**	**OR**	**95% CI**
Somewhat thinner	71/71	0.83	0.55–1.26	0.80	0.52–1.22	88/55	1.14	0.73–1.80	1.02	0.64–1.63
About the same	181/160	REF		REF		164/126	REF		REF	
Somewhat heavier	69/81	0.78	0.52–1.18	0.85	0.55–1.31	69/87	0.52	0.33–0.80	0.60	0.38–0.95
Heaviest/much heavier	15/16	0.93	0.43–2.00	1.21	0.53–2.74	9/16	0.36	0.14–0.92	0.46	0.16–1.30
Relative weight at 15 or 16[*a]										
Thinnest/much thinner	52/61	0.64	0.40–1.02	0.59	0.36–0.96	35/31	0.77	0.42–1.40	0.66	0.36–1.23
Somewhat thinner	77/73	0.93	0.62–1.39	0.90	0.59–1.35	90/53	1.35	0.87–2.11	1.23	0.78–1.95
About the same	196/174	REF		REF		179/151	REF		REF	
Somewhat heavier	63/69	0.85	0.56–1.30	1.02	0.65–1.61	58/73	0.63	0.40–1.00	0.77	0.47–1.26
Heaviest/much heavier	13/16	0.78	0.36–1.71	1.14	0.46–2.85	9/14	0.47	0.18–1.22	0.65	0.22–1.89
BMI at age 20 yrs.[b]										
<25	357/343	REF		REF		342/282	REF		REF	
25–29.9	27/28	1.06	0.59–1.89	0.98	0.53–1.82	17/21	0.69	0.33–1.44	0.82	0.38–1.77
≥30	5/15	0.32	0.11–0.92	0.29	0.09–0.86	4/16	0.10	0.03–0.39	0.15	0.04–0.60
p for trend			0.11		0.07			0.001		0.01
Weight gain since age 20, kg[c]										
Q 1 (≤7.57)	93/86	REF		REF		75/63	REF		REF	
Q 2 (7.58–14.57)	85/87	0.91	0.58–1.43	0.88	0.55–1.40	77/62	0.90	0.53–1.52	0.97	0.56–1.66
Q 3 (14.58–24.52)	75/76	0.70	0.43–1.13	0.64	0.36–1.13	91/73	0.78	0.47–1.31	0.90	0.52–1.57
Q 4 (>24.52)	63/71	0.63	0.37–1.04	0.51	0.23–1.16	90/78	0.68	0.41–1.14	0.95	0.46–1.95
p for trend			0.05		0.10			0.13		0.88

[*] compared to peers

OR1: Adjusted for age, ethnicity, country of origin, education, family history of breast cancer, history of benign breast disease, age at menarche, age at menopause (for postmenopausal women), parity, breastfeeding status, age at first birth, HRT use, oral contraceptive use.

OR2:

[a] further adjusted for BMI at age 20;

[b] further adjusted for height and weight at menarche;

[c] further adjusted for current BMI

Table 4

Early body size and breast cancer risk among AA and EA postmenopausal women (excluding HRT users).

	AA (n=752)					EA (n=375)				
	Cases/Controls	**OR**	**95% CI**	**OR2**	**95% CI**	**Cases/Controls**	**OR**	**95% CI**	**OR**	**95% CI**
Relative height at 7 or 8[*a]										
Shortest/much shorter	43/26	1.84	1.06–3.17	1.95	1.12–3.42	20/22	0.82	0.38–1.80	0.76	0.34–1.69
Somewhat shorter	38/51	0.76	0.47–1.24	0.86	0.52–1.42	22/22	0.91	0.42–1.99	0.87	0.39–1.92
About the same	201/211	REF		REF		105/78	REF		REF	
Somewhat taller	43/49	0.90	0.56–1.46	0.96	0.59–1.57	31/26	1.17	0.59–2.34	1.17	0.58–2.37
Tallest/much taller	47/41	1.38	0.85–2.26	1.34	0.81–2.21	24/24	1.00	0.48–2.08	1.03	0.48–2.19
Relative height at menarche[*a]										
Shortest/much shorter	33/23	1.68	0.93–3.03	1.76	0.96–3.20	16/16	0.77	0.32–1.86	0.80	0.32–1.99
Somewhat shorter	45/45	1.05	0.65–1.70	1.12	0.68–1.84	27/29	1.19	0.59–2.40	1.12	0.55–2.28
About the same	202/214	REF		REF		105/78	REF		REF	
Somewhat taller	46/58	0.87	0.55–1.38	0.89	0.55–1.42	28/25	0.75	0.37–1.53	0.70	0.34–1.44
Tallest/much taller	47/37	1.48	0.90–2.45	1.40	0.84–2.33	27/23	1.19	0.58–2.46	1.05	0.50–2.21
Relative height at 15 or 16[*a]										
Shortest/much shorter	29/23	1.45	0.79–2.65	1.58	0.85–2.94	17/14	1.02	0.42–2.46	0.99	0.40–2.42
Somewhat shorter	48/53	1.05	0.66–1.66	1.12	0.70–1.79	37/41	0.90	0.47–1.72	0.94	0.49–1.81
About the same	219/228	REF		REF		105/76	REF		REF	
Somewhat taller	38/43	1.01	0.61–1.67	1.00	0.60–1.69	25/29	0.61	0.30–1.24	0.61	0.29–1.26
Tallest/much taller	39/31	1.48	0.87–2.53	1.37	0.80–2.37	19/12	1.82	0.75–4.44	1.69	0.68–4.21
Relative weight at 7 or 8[*a]										
Thinnest/much thinner	62/56	1.20	0.77–1.86	1.11	0.70–1.76	29/20	1.19	0.56–2.53	0.95	0.43–2.09
Somewhat thinner	84/70	1.27	0.85–1.89	1.23	0.81–1.86	46/30	1.07	0.56–2.05	0.86	0.44–1.69
About the same	174/183	REF		REF		90/80	REF		REF	
Somewhat heavier	43/48	0.87	0.53–1.41	0.83	0.49–1.40	32/33	0.65	0.34–1.26	0.71	0.35–1.44
Heaviest/much heavier	7/21	0.34	0.13–0.85	0.31	0.11–0.88	5/9	0.36	0.10–1.29	0.62	0.15–2.49
Relative weight at menarche[*a]										
Thinnest/much thinner	58/54	1.14	0.73–1.79	1.06	0.66–1.71	22/15	0.91	0.39–2.13	0.78	0.33–1.89

	AA (n=752)					EA (n=375)				
	Cases/Controls	**OR**	**95% CI**	**OR2**	**95% CI**	**Cases/Controls**	**OR**	**95% CI**	**OR**	**95% CI**
Somewhat thinner	76/67	1.19	0.79–1.80	1.13	0.74–1.72	48/30	1.00	0.52–1.93	0.85	0.43–1.67
About the same	186/181	REF		REF		93/70	REF		REF	
Somewhat heavier	45/52	0.97	0.60–1.56	0.89	0.53–1.49	35/45	0.42	0.22–0.79	0.47	0.23–0.93
Heaviest/much heavier	6/24	0.23	0.09–0.60	0.23	0.08–0.63	5/11	1.18	0.05–0.62	0.18	0.04–0.79
Relative weight at 15 or 16[*a]										
Thinnest/much thinner	50/46	1.18	0.73–1.89	1.11	0.67–1.83	17/14	0.92	0.37–2.30	0.75	0.29–1.93
Somewhat thinner	73/64	1.19	0.78–1.79	1.15	0.75–1.77	53/28	1.52	0.81–2.82	1.34	0.70–2.56
About the same	201/198	REF		REF		96/87	REF		REF	
Somewhat heavier	41/50	0.94	0.58–1.53	0.87	0.51–1.48	30/36	0.59	0.31–1.16	0.74	0.35–1.54
Heaviest/much heavier	7/20	0.37	0.15–0.93	0.39	0.14–1.09	7/7	0.61	0.18–2.08	0.93	0.23–3.81
BMI at age 20 yrs.[b]										
<25	279/286	REF		REF		183/148	REF		REF	
25–29.9	46/51	0.99	0.63–1.56	1.25	0.76–2.06	12/10	0.86	0.32–2.27	1.32	0.47–3.71
≥30	14/23	0.68	0.33–1.38	1.05	0.47–2.35	3/11	0.06	0.01–0.40	0.12	0.02–0.82
p for trend			0.37		0.60			0.004		0.09
Weight gain since age 20, kg[c]										
Q 1	55/68	REF		REF		41/32	REF		REF	
Q 2	81/86	1.17	0.71–1.92	1.21	0.73–2.01	35/30	0.74	0.33–1.66	0.90	0.39–2.06
Q 3	83/90	1.20	0.73–1.96	1.29	0.75–2.21	51/40	0.69	0.33–1.45	0.94	0.42–2.13
Q 4	99/96	1.21	0.74–1.95	1.38	0.72–2.64	55/46	0.63	0.30–1.30	1.26	0.43–3.69
p for trend			0.52		0.37			0.25		0.59

[*] compared to peers

OR1: Adjusted for age, ethnicity, country of origin, education, family history of breast cancer, history of benign breast disease, age at menarche, age at menopause, parity, breastfeeding status, age at first birth, oral contraceptive use.

OR2:

[a] further adjusted for BMI at age 20;

[b] further adjusted for height and weight at menarche;

[c] further adjusted for current BMI