

Relative Weight at Age 12 and Risk of Postmenopausal Breast Cancer

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Abstract

Background: Early adolescent weight may affect the risk of postmenopausal breast cancer, and this association may be modified by a family history of breast or ovarian cancer in a first-degree relative, and/or estrogen (ER) and progesterone (PR) receptor status of the disease.

Methods: Relative weight at age 12 years (above, below, or average weight compared with peers) and family history were ascertained using a mailed questionnaire in 1986, in the Iowa Women's Health Study, a prospective cohort study of postmenopausal women. Incident breast cancer cases (including ER and PR status) were identified using the Iowa Surveillance, Epidemiology, and End Results Cancer Registry. Relative risks (RR) and 95% confidence intervals (95% CI) were estimated using Cox proportional hazards regression, and were adjusted for breast cancer risk factors, including body mass index at age 18 years and body mass index at study baseline.

Results: Through 2003, 2,503 cases of postmenopausal breast cancer were identified among 35,941 women in the analytic cohort. Compared with women with

average weight at age 12 years, there was no association of below average weight with risk of breast cancer (RR, 1.02; 95% CI, 0.92-1.13), whereas women with above average weight had a lower risk (RR, 0.85; 95% CI, 0.74-0.98). There was no evidence of an interaction between weight at age 12 years and family history ($P = 0.44$). The inverse association of above average weight with risk of breast cancer was strongest for PR- tumors (RR, 0.62; 95% CI, 0.43-0.89), intermediate for ER+ (RR, 0.80; 95% CI, 0.67-0.96) and ER- (RR, 0.77; 95% CI, 0.50-1.19) tumors, and weakest for PR+ tumors (RR, 0.90; 95% CI, 0.74-1.09). These associations were not modified by a family history (all $P > 0.18$). In a joint ER/PR analyses, the strongest inverse association with above average weight at age 12 years was seen for ER+/PR- (RR, 0.49; 95% CI, 0.29-0.85).

Conclusion: Above average weight at age 12 years was inversely associated with risk of postmenopausal breast cancer, and was not modified by a family history of the disease. The inverse association was strongest for ER+/PR- tumors. (Cancer Epidemiol Biomarkers Prev 2008;17(2):374-8)

Introduction

Breast cancer is the most common noncutaneous cancer among women and the second leading cause of cancer death among women in the United States (1). Most studies suggest that higher adult body mass index (BMI) is associated with elevated risk of postmenopausal breast cancer, presumably due to increased estrogen levels produced by excess adipose tissue (2, 3). However, among premenopausal women, where the major source of endogenous estrogens is the ovary, higher BMI has consistently been inversely associated with the risk of premenopausal breast cancer. Whereas the mechanism underlying this observation is not known, lower estrogen

levels among obese premenopausal women has been suggested (2, 4-6).

In contrast, greater BMI in later adolescence (age 15-18 years) has been inversely associated with both premenopausal (4, 7-13) and postmenopausal (4, 8-10, 14-16) breast cancer risk, although there are exceptions for premenopausal (17) and postmenopausal (7, 11, 17) breast cancer risk. More limited data suggest that this inverse association extends into earlier adolescence (ages 9-14 years) as well for premenopausal (8, 10, 12, 18-20) and postmenopausal (8, 10, 18, 19, 21, 22) breast cancer risk, although there are a few exceptions (17, 23). Early life obesity may also be modified by a family history of breast cancer (21, 24, 25). Finally, risk factors may differ for biological subtypes of breast cancer defined by estrogen and progesterone receptor (ER/PR) status (26, 27), but this has not been evaluated for early adolescent weight.

We evaluated the association of relative weight at age 12 years with postmenopausal breast cancer risk in the Iowa Women's Health Study. In addition, we investigated whether these associations differ by family history of breast cancer and/or biological subtypes of breast cancer defined by ER/PR status.

Received 4/29/07; revised 10/29/07; accepted 11/15/07.

Grant support: R01 CA39742.

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doi:10.1158/1055-9965.EPI-07-0389

Materials and Methods

The Iowa Women's Health Study is a prospective cohort study of women ages 55 to 69 years at study baseline in 1986 (28). Briefly, a mailed survey was returned by 41,836 women in 1986 from a random sample of women with a valid Iowa driver's license. The baseline survey included questions on a variety of potential breast cancer risk factors, including medical and family history, anthropometrics, reproductive factors, and lifestyle characteristics. Participants were also asked: "Think back to when you were in 6th grade or about the age of 12. Would you say at that time your weight was: below average for your age and height, about average for your age and height, or above average for your age and height?"

Breast cancers and their ER/PR status were ascertained through linkage to the State Health Registry of Iowa, part of the Surveillance, Epidemiology, and End Results program (29). On an annual basis, cohort members were linked to the registry based on name (first, last, maiden), zip code, birth date, and social security number. Deaths were identified through follow-up surveys, annual linkage to Iowa death certificates, and linkage to the National Death Index.

For this analysis, we excluded women if they reported any of the following on the baseline survey: being premenopausal ($n = 569$), having a history of any cancer other than skin cancer ($n = 3830$), or ever having had a total or partial mastectomy ($n = 1,884$). An additional 1,164 women were excluded due to missing data on

relative weight at age 12 years. This left a total of 35,941 women in the analysis (exclusions were not mutually exclusive).

Each woman accrued person-years of follow-up from the completion of the baseline questionnaire until a diagnosis of breast cancer, death, or emigration from Iowa; if none of these occurred, cohort members were censored on December 31, 2003. Relative risks (RR) and 95% confidence intervals (95% CI) were estimated using Cox regression, controlling for potential confounding factors as included in Table 1. Incidence was modeled as a function of age (30). Initially, we examined the overall association of weight at age 12 years with breast cancer risk. Formal assessments of risk were assessed using tests for trend, calculated by ordering the three-level weight variable, and including it in the Cox proportional hazards model as a linear term.

We then investigated whether a family history of (a) breast or (b) breast or ovarian cancer in a first-degree relative modified the association between weight at age 12 years and risk of breast cancer. Results were similar, so we report results for a family history of breast or ovarian cancer to facilitate comparison with a previously published study (21). Formal tests of interaction were carried out by including the main effects of weight at age 12 years (ordinal) and family history, and testing the statistical significance of the corresponding interaction term. Finally, we evaluated whether these associations varied by breast cancer subtypes defined by ER and PR status. In these receptor-specific analyses, events not of

Table 1. Distribution of breast cancer risk factors by relative weight at age 12 y, Iowa Women's Health Study, 1986

	Relative weight for age and height in 6th grade or about the age of 12		
	Below average ($n = 8,082$)	Average ($n = 23,127$)	Above average ($n = 4,732$)
		Mean \pm SD	
Age at study entry (y)	61.7 \pm 4.2	61.8 \pm 4.2	61.2 \pm 4.2
Age of first menstruation (y)	13.3 \pm 1.6	12.8 \pm 1.4	12.3 \pm 1.4
Age at menopause (y)	47.6 \pm 6.4	47.6 \pm 6.4	47.8 \pm 6.4
BMI at age 18 (kg/m^2)	19.6 \pm 2.3	21.6 \pm 2.5	25.2 \pm 4.1
BMI in 1986 (kg/m^2)	25.8 \pm 4.6	26.9 \pm 4.8	29.6 \pm 6.1
		Percent distribution (%)	
BMI at age 18 (kg/m^2)			
<18.0	30.0	6.6	2.2
18.0-24.9	67.9	85.6	53.6
25.0-29.9	1.9	7.0	32.0
30.0+	0.3	0.8	12.2
BMI in 1986 (kg/m^2)			
<18.0	1.6	0.9	0.4
18.0-24.9	47.3	38.7	23.6
25.0-29.9	35.6	37.9	34.7
30.0+	15.5	22.5	41.4
Education greater than high school	41.0	37.1	44.5
Family history of breast or ovarian cancer in a first-degree relative	14.0	13.6	12.8
Any live births	90.3	91.5	89.3
Age at first live birth <20 y	18.5	20.6	18.4
Ever used oral contraceptives	20.6	17.9	18.7
Ever used hormone replacement therapy	42.2	36.8	35.9
Did not drink alcohol in 1986	56.7	55.8	58.8
Never smoker	68.4	66.0	60.6
Physical activity index			
Low	47.3	47.1	48.5
Medium	27.4	27.9	26.2
High	25.3	25.0	25.3

Table 2. Multivariate-adjusted RRs of postmenopausal breast cancer by relative weight at age 12 y, overall and stratified by first-degree family history of breast or ovarian cancer, and by breast cancer subtype based on ER and PR status

Type of breast cancer	Relative weight at age 12	All			No family history			Family history		
		Cases	RR (95% CI)	<i>P</i> trend	Cases	RR (95% CI)	<i>P</i> trend	Cases	RR (95% CI)	<i>P</i> trend
All	Below	596	1.02 (0.92-1.13)	0.08	465	0.99 (0.88-1.11)	0.02	112	1.10 (0.87-1.38)	0.05
	Average	1635	1.00 (reference)		1309	1.00 (reference)		282	1.00 (reference)	
	Above	272	0.85 (0.74-0.98)		220	0.86 (0.73-1.01)		43	0.84 (0.60-1.17)	
<i>P</i> interaction (family history and weight 12) = 0.44										
ER+	Below	383	1.01 (0.89-1.16)	0.08	296	0.95 (0.82-1.11)	0.06	72	1.25 (0.94-1.67)	0.03
	Average	1055	1.00 (reference)		859	1.00 (reference)		165	1.00 (reference)	
	Above	166	0.80 (0.67-0.96)		137	0.82 (0.68-1.00)		24	0.77 (0.49-1.22)	
<i>P</i> interaction (family history and weight 12) = 0.21										
ER–	Below	76	1.10 (0.81-1.48)	0.20	62	1.17 (0.84-1.63)	0.11	12	0.78 (0.38-1.60)	0.71
	Average	190	1.00 (reference)		147	1.00 (reference)		35	1.00 (reference)	
	Above	31	0.77 (0.50-1.19)		26	0.81 (0.50-1.30)		5	0.81 (0.31-2.09)	
<i>P</i> interaction (family history and weight 12) = 0.73										
PR+	Below	315	1.00 (0.86-1.16)	0.48	240	0.94 (0.80-1.11)	0.31	63	1.27 (0.93-1.74)	0.06
	Average	868	1.00 (reference)		701	1.00 (reference)		139	1.00 (reference)	
	Above	149	0.90 (0.74-1.09)		122	0.91 (0.73-1.12)		23	0.94 (0.59-1.48)	
<i>P</i> interaction (family history and weight 12) = 0.19										
PR–	Below	123	1.08 (0.85-1.37)	0.24	100	1.08 (0.83-1.40)	0.02	18	0.91 (0.52-1.59)	0.45
	Average	328	1.00 (reference)		264	1.00 (reference)		54	1.00 (reference)	
	Above	42	0.62 (0.43-0.89)		36	0.69 (0.47-1.01)		6	0.53 (0.21-1.34)	
<i>P</i> interaction (family history and weight 12) = 0.81										

NOTE: Cox proportional hazards regression analysis was used, accounting for age, education status, age at menopause, age at menarche, parity, age at first birth BMI at age 18 y, BMI in 1986, oral contraceptive use, hormone replacement therapy, smoking status, alcohol use, and physical activity level.

that specific cancer type were considered censored observations. All analyses were done on SAS 8.02 (SAS Institute, Inc.) and Splus 7.0.6 (Mathsoft, Inc.) software systems.

Results

Of the 35,941 women in the analytic cohort, 22.5% reported below average weight at age 12 years, 64.3% reported average weight, and 13.2% reported above average weight. Table 1 reports breast cancer risk factors by relative weight at age 12 years. Compared with women reporting below average weight, women reporting above average weight had an earlier age at first menstrual cycle (13.3 versus 12.3 years); higher BMI at age 18 years (19.6 versus 25.2 kg/m²), and at study baseline in 1986 (25.8 versus 29.6 kg/m²); lower use of hormone replacement therapy (42.2% versus 35.9%); and were never smokers (68.4% versus 60.6%). Other breast cancer risk factors were similar across categories of relative weight at age 12 years.

During 548,567 person-years of follow-up (through 2003), 2,503 breast cancers were identified. The mean age at diagnosis was 71.4 years (SD 6.2 years). ER status was available on 1,901 (75.9%) of the cases, and 84.4% of these cases were ER+. PR status was available on 1,825 (72.9%) of the cases, and 73.0% of these cases were PR+.

The association of weight at age 12 years with breast cancer risk is reported in Table 2. Compared with women with average weight at age 12 years, there was no association of below average weight with risk of breast cancer, whereas women with above average weight had

a lower risk of breast cancer (RR, 0.85; 95% CI, 0.74-0.98) after adjustment for multiple risk factors including BMI at age 18 years and BMI at study baseline. As shown in Table 1, although there was tracking of weight over the life course, this was not absolute. For example, of women reporting above average weight at age 12 years, 44% were overweight or obese at age 18 years (but 56% had a BMI of <25) and 76% were overweight or obese at study baseline (but 24% had a BMI of <25). As shown in Fig. 1, results were unchanged when we stratified on BMI at age 18 years (adjusting for baseline BMI) or BMI at baseline (adjusting for BMI at age 18 years).

The inverse association of weight at age 12 years with breast cancer risk was not modified by a family history of breast or ovarian cancer in a first-degree relative (*P* for interaction = 0.44; Table 2). Similar results were seen if we restricted to a first-degree relative with breast cancer only (data not shown).

The inverse association of above average weight at age 12 years with risk of breast cancer was observed for all subtypes defined by ER and PR status, and was strongest for PR– tumors (RR, 0.62), intermediate for ER+ (RR, 0.80) and ER– (RR, 0.77) tumors, and weakest for PR+ tumors (RR, 0.90; Table 2). These associations were not modified by a family history of breast or ovarian cancer (all *P* > 0.18). In an analysis where ER and PR were jointly evaluated (not reported in the table), an inverse association with above average weight was seen for ER+PR+ (RR, 0.91; 95% CI, 0.75-1.10), ER+PR– (RR, 0.49; 95% CI, 0.29-0.85), ER–PR+ (RR, 0.74; 95% CI, 0.24-2.26), and ER–PR– (RR, 0.77; 95% CI, 0.47-1.26). There was insufficient sample size to evaluate the interaction of these subtypes by a family history of breast or ovarian cancer.

Discussion

We confirmed an inverse association between relative weight at age 12 years and risk of postmenopausal breast cancer after adjustment for a wide variety of breast cancer risk factors, including BMI at age 18 years and BMI at study baseline. However, we were not able to confirm the interaction between relative weight at age 12 years and family history of breast cancer or breast or ovarian cancer we had reported previously in a different population (21). We also found inverse associations for relative weight at age 12 years with all breast cancer subtypes defined by ER and PR status, and this was strongest for ER+PR- tumors.

Strengths of this study include the prospective cohort design; excellent follow-up of the cohort; case identification using a Surveillance, Epidemiology, and End Results cancer registry; ability to assess multiple potential confounders; and assessment of associations for breast cancer subtypes defined by ER and PR status. There are also limitations. Relative weight at age 12 years was self-reported and involved recalling a relative weight more than 4 to 6 decades in the past. Indeed, this is why we did not collect actual weight. Recall of childhood and adolescent body build by elderly subjects has been shown to have reasonable validity (31, 32) and any bias introduced is expected to attenuate associations.

Furthermore, we observed expected associations of relative weight at age 12 years with age of first menstruation, which provided some internal consistency for our measure. Information about ER/PR status of breast cancer was obtained through multiple laboratories involved with Surveillance, Epidemiology, and End Results, rather than a single reference laboratory. However, the availability of receptor status and the ER/PR distribution in our study (ER+ 64.1%, ER- 11.9%, PR+ 53.2%, and PR- 19.7%) was similar to that reported by other studies (27, 33).

Most studies have reported that greater weight or obesity in early adolescence (age 9-14 years) is associated

with a lower risk of postmenopausal breast cancer (8, 10, 18, 19, 21, 22), although there are exceptions (17, 23). This association seems to be independent of adult obesity (which is positively associated with postmenopausal breast cancer risk), particularly as shown in our multivariate-adjusted analyses and analyses stratified on adult BMI. However, we were not able to confirm results of a prior study that found a strong interaction ($P \leq 0.001$) between relative weight at age 12 years with family history on the risk of postmenopausal breast cancer (21). The latter study, conducted among a historical cohort of 426 families of breast cancer probands diagnosed between 1944 and 1952, found that women with above average weight at age 12 years had a lower risk of breast cancer if they had no family history of breast cancer [odds ratio (OR), 0.75; 95% CI, 0.26-2.16], whereas women with a family history had a greatly increased risk of breast cancer (OR, 4.25; 95% CI, 1.71-10.5). These latter findings, different from our study results, could be due to difference in the genetic risk, as the study was based on a historical cohort of families of breast cancer probands with a higher genetic risk of breast cancer, whereas our study was based on a population cohort of average genetic risk.

We found that the inverse association of relative weight at age 12 years with postmenopausal breast cancer risk was apparent for all tumor subtypes defined by ER or PR status, although results were strongest for ER+/PR- tumors. To our knowledge, no studies have evaluated the association between early adolescent obesity and postmenopausal breast cancer by ER/PR tumor subtype.

The biological mechanisms for a putative inverse relationship between relative weight at age 12 years and postmenopausal breast cancer risk is not known. In a 7-year longitudinal study of 286 girls initially ages 8 to 9 years, adiposity was associated with higher circulating concentrations of DHEA sulfate and lower concentrations of sex hormone-binding globulin; however, there were no consistent associations for circulating levels of estrogen or progesterone (34). The lack of an association for estrogen would suggest that early adolescent obesity is not likely to influence postmenopausal breast cancer risk through estrogen signaling, consistent with the similar association for ER+ (RR, 0.80) and ER- (RR, 0.77) tumors in our study. We observed larger differences for PR+ (RR, 0.90) versus PR- (RR, 0.62) tumors, however, which is inconsistent with the finding of no differences in progesterone concentrations noted above. Obese adolescent and preadolescent girls also have elevated levels of insulin and insulin-like growth factor I, and this leads to impaired ovarian steroid metabolism and anovulation (35).

Fewer ovulatory cycles are expected to protect against breast cancer (36, 37), although one recent study found that the inverse relationship between adult BMI and premenopausal breast cancer incidence was not likely to be explained by menstrual cycle characteristics of the women (38). However, the effect of fewer ovulatory cycles could be more pronounced during the time frame before first full-term pregnancy, and particularly in adolescence, due to the greater susceptibility to carcinogens of undifferentiated breast tissue (39, 40). There may also be other aspects of the hormonal milieu associated with obesity in the early teenage years that protects against breast cancer, and this requires further evaluation (34, 35).

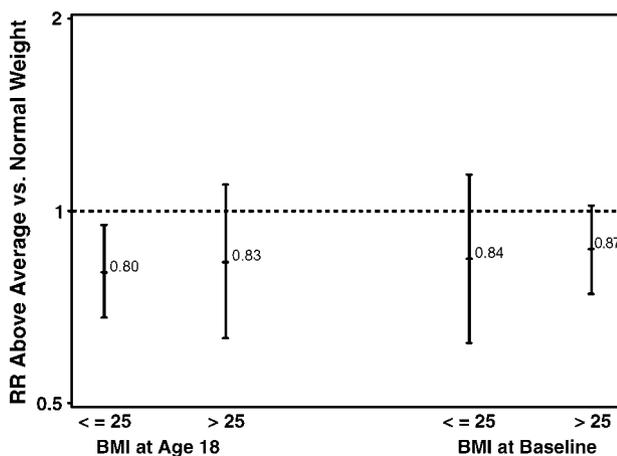


Figure 1. Multivariate-adjusted RRs and 95% CIs of postmenopausal breast cancer for above average weight at age 12 y (compared with average weight), stratified by BMI at age 18 y (including adjustment for BMI at study baseline) and study baseline in 1986 (including adjustment for BMI at age 18 y).

We confirmed an inverse association between relative weight at age 12 years and risk of postmenopausal breast cancer, and this was independent of adult BMI. The inverse association was strongest for ER+/PR– tumors. We did not find any interaction between relative weight at age 12 years and family history of cancer as suggested in previous studies.

References

- Jemal A, Siegel R, Ward E, et al. Cancer statistics, 2007. *CA Cancer J Clin* 2007;57:43–66.
- Key TJ, Appleby PN, Reeves GK, et al. Body mass index, serum sex hormones, and breast cancer risk in postmenopausal women. *J Natl Cancer Inst* 2003;95:1218–26.
- 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.
- Huang Z, Hankinson SE, Colditz GA, et al. Dual effects of weight and weight gain on breast cancer risk. *JAMA* 1997;278:1407–11.
- Twoogor SS, Eliassen AH, Missmer SA, et al. Birthweight and body size throughout life in relation to sex hormones and prolactin concentrations in premenopausal women. *Cancer Epidemiol Biomarkers Prev* 2006;15:2494–501.
- Ursin G, Longnecker MP, Haile RW, et al. A meta-analysis of body mass index and risk of premenopausal breast cancer. *Epidemiology* 1995;6:137–41.
- Choi NW, Howe GR, Miller AB, et al. An epidemiologic study of breast cancer. *Am J Epidemiol* 1978;107:510–21.
- Hislop TG, Coldman AJ, Elwood JM, et al. Childhood and recent eating patterns and risk of breast cancer. *Cancer Detect Prev* 1986;9:47–58.
- Chu SY, Lee NC, Wingo PA, et al. The relationship between body mass and breast cancer among women enrolled in the Cancer and Steroid Hormone Study. *J Clin Epidemiol* 1991;44:1197–206.
- Brinton LA, Swanson CA. Height and weight at various ages and risk of breast cancer. *Ann Epidemiol* 1992;2:597–609.
- Trentham-Dietz A, Newcomb PA, Storer BE, et al. Body size and risk of breast cancer. *Am J Epidemiol* 1997;145:1011–9.
- 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.
- Sanderson M, Shu XO, Jin F, et al. Weight at birth and adolescence and premenopausal breast cancer risk in a low-risk population. *Br J Cancer* 2002;86:84–88.
- Folsom AR, Kaye SA, Prineas RJ, et al. Increased incidence of carcinoma of the breast associated with abdominal adiposity in postmenopausal women. *Am J Epidemiol* 1990;131:794–803.
- Magnusson C, Baron J, Persson I, et al. Body size in different periods of life and breast cancer risk in post-menopausal women. *Int J Cancer* 1998;76:29–34.
- 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.
- Pryor M, Slattery ML, Robison LM, et al. Adolescent diet and breast cancer in Utah. *Cancer Res* 1989;49:2161–7.
- Le Marchand L, Kolonel LN, Earle ME, et al. Body size at different periods of life and breast cancer risk. *Am J Epidemiol* 1988;128:137–52.
- Berkey CS, Frazier AL, Gardner JD, et al. Adolescence and breast carcinoma risk. *Cancer* 1999;85:2400–9.
- Baer HJ, Colditz GA, Rosner B, et al. Body fatness during childhood and adolescence and incidence of breast cancer in premenopausal women: a prospective cohort study. *Breast Cancer Res* 2005;7:R314–25.
- Cerhan JR, Grabrick DM, Vierkant RA, et al. Interaction of adolescent anthropometric characteristics and family history on breast cancer risk in a Historical Cohort Study of 426 families (USA). *Cancer Causes Control* 2004;15:1–9.
- Ahlgren M, Melbye M, Wohlfahrt J, et al. Growth patterns and the risk of breast cancer in women. *N Engl J Med* 2004;351:1619–26.
- Franceschi S, Favero A, La Vecchia C, et al. Body size indices and breast cancer risk before and after menopause. *Int J Cancer* 1996;67:181–6.
- Ursin G, Paganini-Hill A, Siemiatycki J, et al. Early adult body weight, body mass index, and premenopausal bilateral breast cancer: data from a case-control study. *Breast Cancer Res Treat* 1994;33:75–82.
- Magnusson C, Colditz G, Rosner B, et al. Association of family history and other risk factors with breast cancer risk (Sweden). *Cancer Causes Control* 1998;9:259–67.
- Potter JD, Cerhan JR, Sellers TA, et al. Progesterone and estrogen receptors and mammary neoplasia in the Iowa Women's Health Study: how many kinds of breast cancer are there? *Cancer Epidemiol Biomarkers Prev* 1995;4:319–26.
- Colditz GA, Rosner BA, Chen WY, et al. Risk factors for breast cancer according to estrogen and progesterone receptor status. *J Natl Cancer Inst* 2004;96:218–28.
- Bisgard KM, Folsom AR, Hong CP, et al. Mortality and cancer rates in nonrespondents to a prospective cohort study of older women: 5-year follow-up. *Am J Epidemiol* 1994;139:990–1000.
- Ries LAG, Eisner MP, Kosary CL, et al. SEER cancer statistics review, 1975–2002. Bethesda (MD): National Cancer Institute; 2005.
- Korn EL, Graubard BI, Midthune D. Time-to-event analysis of longitudinal follow-up of a survey: choice of the time-scale. *Am J Epidemiol* 1997;145:72–80.
- Must A, Willett WC, Dietz WH. Remote recall of childhood height, weight, and body build by elderly subjects. *Am J Epidemiol* 1993;138:56–64.
- 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.
- Grann VR, Troxel AB, Zojwalla NJ, et al. Hormone receptor status and survival in a population-based cohort of patients with breast carcinoma. *Cancer* 2005;103:2241–51.
- Baer HJ, Colditz GA, Willett WC, et al. Adiposity and sex hormones in girls. *Cancer Epidemiol Biomarkers Prev* 2007;16:1880–8.
- Stoll BA. Teenage obesity in relation to breast cancer risk. *Int J Obes Relat Metab Disord* 1998;22:1035–40.
- Key TJ, Pike MC. The role of oestrogens and progestagens in the epidemiology and prevention of breast cancer. *Eur J Cancer Clin Oncol* 1988;24:29–43.
- Henderson BE, Ross RK, Judd HL, et al. Do regular ovulatory cycles increase breast cancer risk? *Cancer* 1985;56:1206–8.
- 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.
- Russo J, Hu YF, Silva ID, et al. Cancer risk related to mammary gland structure and development. *Microsc Res Tech* 2001;52:204–23.
- 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.

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Cancer Epidemiol Biomarkers Prev 2008;17:374-378.

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