Research Article

Cancer Epidemiology, Biomarkers & Prevention

Weight Cycling and Risk of Endometrial Cancer

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Abstract

Background: Obesity, as measured by body mass index (BMI), is an established risk factor for endometrial cancer in postmenopausal women. Weight cycling, which consists of repeated cycles of weight loss followed by regain, occurs frequently in overweight and obese women. It is unclear whether weight cycling is associated with risk of endometrial cancer independent of BMI.

Methods: This analysis included 38,148 postmenopausal women enrolled in the Cancer Prevention Study II Nutrition Cohort, of whom 559 were diagnosed with endometrial cancer between enrollment in 1992 and June 30, 2007. Number of lifetime weight cycles was determined from questions on the baseline questionnaire asking how many times 10 or more pounds were intentionally lost and later regained. Multivariable-adjusted hazard rate ratios (RR) and 95% confidence intervals (CI) were estimated using Cox proportional hazards regression.

Results: Weight cycling was positively associated with endometrial cancer incidence (RR, 2.13; 95% CI, 1.63–2.78 for 10+ weight cycles vs. no weight cycles; $P_{trend} < 0.0001$). However, after adjustment for BMI in 1992, this association was null (RR, 1.05; 95% CI, 0.77–1.42; $P_{trend} = 0.82$). Weight cycling was not associated with endometrial cancer in analyses stratified by BMI or by weight change after adjustment for BMI.

Conclusions: After adjustment for BMI, weight cycling was not associated with the risk of endometrial cancer.

Impact: These findings suggest that weight loss with subsequent regain is unlikely to increase risk of endometrial cancer. Therefore, weight loss for better health should be encouraged. *Cancer Epidemiol Biomarkers Prev; 21(5); 747–52. ©2012 AACR.*

Introduction

Endometrial cancer is the most common gynecologic cancer and the fourth most common cancer in women (1). The American Cancer Society predicts that 47,130 new cases and 8,010 deaths from endometrial cancer will occur in the United States in 2012. Most risk factors for endometrial cancer increase exposure to estrogen that is unopposed by progesterone. Adipose tissue converts androstenedione to estrone and is a major source of circulating estrogen in postmenopausal women. Accordingly, overweight and obesity are key risk factors for endometrial cancer (2) and are estimated to be responsible for 57% of all cases of this cancer in the United States (3).

Attempted weight loss is common among overweight and obese American women: 55% to 60% of women with a body mass index (BMI) $\geq$25 and <30 kg/m^2 and 63% to 73% of women with a BMI $\geq$30 kg/m^2 report trying to lose weight (4, 5). However, most weight loss is not maintained (6, 7). Repeated cycles of weight loss followed by regain result in weight cycling. Comparing studies of weight cycling is difficult because the definition of a weight cycle varies considerably in the amount of weight lost and regained, the period over which the loss and regain occurs, and whether the weight loss is intentional or unintentional. This lack of a standardized definition may contribute to inconsistencies in the findings from weight cycling studies.

The prevalence of weight cycling among postmenopausal women has been estimated to be about 41% by a study in which weight cycling was defined by the pattern of weight change at specific times during life (8) and between 27% (9) and 57% (10) by studies of weight cycling initiated by intentional weight loss. Women who weight cycle have more upper body subcutaneous adipose tissue (11) and visceral fat (12) than women who do not weight cycle. A recent study of weight regain after intentional weight loss in 78 postmenopausal women found that fat mass was regained to a greater degree than lean mass by the women who regained $\geq$2 kg (13), suggesting that women who lose then regain weight could experience increases in adipose tissue even without a net increase in BMI. However, a smaller study of 24 women found no evidence of an imbalance in the regain of fat and lean mass (14). The possibility that weight cycling may result in more adipose tissue raises the question of whether this influences risk of endometrial cancer independent of BMI.

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doi: 10.1158/1055-9965.EPI-12-0038

The association between weight cycling and endometrial cancer has been previously investigated in one prospective cohort (15) and 2 case–control studies (16, 17). No association was found in the Iowa Women's Health Study (15), a large prospective study of postmenopausal women with 176 endometrial cancer cases. In one of the case–control studies (16), weight cycling was associated with increased risk of endometrial cancer, but this association was attenuated and not statistically significant after adjustment for recent weight. In the second, somewhat larger case–control study, a modestly increased risk was found [OR, 1.27; 95% confidence interval (CI), 1.00–1.61] even after adjustment for recent weight (17). In these studies, the definition of weight cycling varied in the magnitude and timing of weight change, the range in number of weight cycles, and whether weight cycling was initiated by intentional weight loss or not.

Herein, we investigated the association of weight cycling with the risk of endometrial cancer using 38,148 postmenopausal women participants in the American Cancer Society Cancer Prevention Study II (CPS-II) Nutrition Cohort with 15 years of follow-up (1992–2007). With a large number of endometrial cancer cases, information on weight history, and high prevalence of weight cycling (10), this prospective cohort study of cancer incidence allowed for investigation of association of 3 levels of weight cycling with endometrial cancer independent of BMI.

Materials and Methods

Study population

Subjects for this study were participants in the CPS-II Nutrition Cohort, a prospective study of cancer incidence and mortality among 86,404 men and 97,786 women. The Nutrition Cohort, which is described in detail elsewhere (18), was initiated in 1992 as a subcohort of CPS-II, a prospective study of cancer mortality involving approximately 1.2 million Americans which began in 1982. Participants in the Nutrition Cohort were recruited from CPS-II members who resided in 21 states and were predominantly between the ages of 50 and 74 years. At baseline, participants completed a self-administered questionnaire that included demographic, medical, dietary, and lifestyle information. Follow-up questionnaires were sent to all living Nutrition Cohort members in 1997 and every 2 years thereafter to update exposure information and to ascertain newly diagnosed cancers. The response rates on all of the follow-up questionnaires, among those cohort participants who were mailed surveys, were at least 91%. For the present study, follow-up was from the date of receipt of the baseline survey in 1992/1993 until the date of diagnosis of endometrial cancer, death, date of the last returned survey, or June 30, 2007, whichever came first. All aspects of the CPS-II Nutrition Cohort study are approved by the Emory University Institutional Review Board (Atlanta, GA).

Women were excluded from this analysis if they were lost to follow-up ($n = 3,122$), reported any prevalent cancer other than nonmelanoma skin cancer in either 1982 ($n = 6,411$) or 1992 ($n = 6,680$), or whose self-report of endometrial cancer during the first follow-up interval (1992–1997) was not verified ($n = 45$). Women who reported endometrial cancer during later follow-up that could not be verified were censored on the date of the last questionnaire on which they did not report endometrial cancer. Also excluded was one woman with an uncertain diagnosis date (self-report > 180 days before the verified diagnosis date) and women who reported a BMI < 18.5 or >50 kg/m^2 or missing BMI ($n = 4,255$). To eliminate women who possibly misinterpreted the weight cycling questions, we excluded those with missing information on number of times they purposely lost 10 pounds or more or number of times regained weight that was purposefully lost ($n = 1,869$), who reported never purposefully losing 10 pounds or more but reported 10 or more pounds as the most weight ever purposefully lost ($n = 937$), and whose response to the number of times weight was purposely lost and number of time regained differed by more than 2 ($n = 3,011$). Women who were premenopausal or whose menopausal status was unknown ($n = 3,862$) and who reported that their uterus had been removed or had unknown uterine status at baseline ($n = 26,419$) were also excluded. Finally, to avoid the powerful influence of estrogen-only postmenopausal hormone (PMH) use on endometrial cancer risk, women who reported use of estrogen-only and those for whom the type of hormone use was unknown in 1992 ($n = 3,062$) were also excluded from the analysis. Women who did not return 2 consecutive surveys were censored at the date of the return of their last survey and women who underwent hysterectomy after enrollment were censored at the date of receipt of the questionnaire they reported this. The final analytic cohort for this study was 38,148 women.

Identification of endometrial cancer cases

We identified and verified 559 cases of endometrial cancer diagnosed between enrollment in 1992/1993 and June 30, 2007, for this analysis. This included 550 cases identified through self-report on the follow-up questionnaires and subsequently verified through medical records ($n = 379$) or linkage with state cancer registries ($n = 171$; ref. 19). Additional endometrial cancer cases were identified through linkage with the National Death Index ($n = 9$; ref. 20).

Identification of weight cyclers

Women in the CPS-II Nutrition cohort were defined as weight cyclers on the basis of their response to 2 questions about intentional weight loss and regain on the 1992 baseline questionnaire. The first question asked "How many times in your life have you purposefully lost 10 pounds or more?" The second question asked "How many time in your life have you regained as much as 10 pounds that you previously had lost?" A write-in answer was required with spaces for 2 digits provided. Thus, answers could range from 0 to 99. A weight cycle was defined to

include both a purposeful loss and a regain and cohort participants were classified by the number of weight cycles they reported. These criteria resulted in the identification of 17,253 women as noncyclers and 20,895 women as weight cyclers. The total number of weight cycles reported was used to further classify the weight cyclers into 3 groups of 1 to 4, 5 to 9, and 10 or more weight cycles.

Statistical analysis

Cox proportional hazards regression (21) was used to calculate hazard rate ratios (RR) and corresponding 95% CIs for the association of weight cycling with endometrial cancer incidence. *P* values for linear trend were estimated by modeling number of weight cycles as a categorical variable, with the median value assigned for each category.

All Cox models were stratified on the exact year of age in 1992. Additional covariates included in the multivariable adjusted models were family history of endometrial cancer (yes, no), alcohol consumption (not current drinker, <1 drink/wk, 1–6 drinks/wk, 1 drink/d, ≥2 drinks/d, missing), smoking status (never, former <20 years since quit, former ≥20 years since quit, current <40 years, current ≥40 years, ever/unclassifiable, missing), BMI in 1992 (continuous), history of diabetes (no, yes), total energy intake (quintiles, missing), physical activity in metabolic equivalents (METs: <8, 8–<17.5, 17.5–<31.5, ≥31.5 h/wk, missing), combinations of number of live births (nulliparous, 1–2, 3+, unknown), age at first birth (<25, 25–29, 30+, unknown), age at menarche (<12, 12, 13, 14+ years, missing), age at menopause (<50, 50–54, 55+ years), PMH use [never, current estrogen + progesterone (E + P), former E + P, other], and oral contraceptive use (never, ever, missing).

Results

Weight cycling was common among women in the CPS-II Nutrition Cohort. Of the 20,895 women (54.8% of the total cohort) who reported intentionally losing and regaining at least 10 pounds (4.5 kg) one or more times, 66.3% weight cycled 1 to 4 times, 16.3% weight cycled 5 to 9 times, and 17.4% weight cycled 10 or more times. Compared with noncyclers, weight cyclers were more likely to be younger, to have a higher BMI at 18 years, to have gained more weight during adulthood, and to have a higher BMI and to be obese at enrollment (Table 1). They were also slightly more likely to have given birth before the age of 25 years, have a family history of endometrial cancer, have used oral contraceptives, have never used PMH, and to be former smokers rather than never or current smokers. Weight cyclers were also more likely to have a higher energy intake and to consume less alcohol than noncyclers. For all of these characteristics, differences between weight cyclers and noncyclers increased with number of weight cycles.

In age-adjusted models, there was a statistically significant positive association between weight cycling and risk of endometrial cancer (Table 2). These associations were only slightly attenuated after adjustment for common risk factors (alcohol use, smoking, physical activity, family history of endometrial cancer, diabetes, number of live births/age at first birth, age at menarche, age at menopause, hormone replacement therapy use, oral contraceptive use, and total energy intake) other than BMI. However, after further adjustment for BMI at baseline, weight cycling was not associated with endometrial cancer risk (RR, 1.05; 95% CI, 0.77–1.42 for ≥10 weight cycles vs. noncycler; $P_{trend} = 0.82$).

In analyses stratified by BMI, weight cycling at any level was not associated with endometrial cancer risk among women who were lean (BMI = 18.5–<25 kg/m^2), overweight (BMI = 25–<30 kg/m^2), or obese (BMI = 30–50 kg/m^2; Table 3). The association between weight cycling and endometrial cancer was also investigated in analyses stratified by adult weight change. The association remained null for all levels of weight cycling for all weight change categories (data not shown).

No association was observed for any level of weight cycling in analyses limited to either ever or never smokers (data not shown). In addition, no association was observed in a sensitivity analysis excluding the first 2 years of follow-up.

Discussion

Weight cycling was not associated with increased risk of endometrial cancer independent of BMI in this large prospective cohort study. The positive association observed in the multivariable-adjusted analysis was eliminated after adjustment for BMI. No association between weight cycling and endometrial cancer was observed regardless of BMI at baseline or weight change during adulthood. Therefore, while weight cyclers may have a higher risk of endometrial cancer than noncyclers because they tend to have higher BMIs, weight cycling itself does not increase risk of this cancer in postmenopausal women.

Our findings are consistent with those of the Iowa Women's Health Study, the only other prospective study of weight cycling and endometrial cancer (15). In that study, with 176 endometrial cancer cases, French and colleagues (15) found that loss and regain of 10% of body weight was not associated with endometrial cancer risk after adjustment for BMI (RR, 0.95; 95% CI, 0.52–1.75). However, in a case–control study of 740 cases and 2,342 controls, Trentham-Dietz and colleagues (17) found that weight cycling, defined as having lost 20 pounds and regained at least 10 pounds in 1 year, was positively associated with risk of endometrial cancer (OR, 1.27; 95% CI, 1.00–1.61) even after adjustment for BMI. That study included both premenopausal and postmenopausal women. In a smaller case–control study of 403 cases and 297 controls, Swanson and colleagues (16) found that having lost and regained 20 pounds 5 or more times was positively associated with the increased risk of endometrial cancer, although the association was not statistically significant after adjustment for BMI (OR, 1.6; 95% CI, 0.8–2.9). All 3 of these studies defined weight cycling

Table 1. Age-standardized frequencies of selected characteristics of women in the CPS-II Nutrition Cohort, by weight cycling status in 1992

	Number of weight cycles			
Variable	**Noncycler (N = 17,253)**	**1–4 (N = 13,856)**	**5–9 (N = 3,414)**	**10+ (N = 3,625)**
Age in 1992[a]	63.6	62.4	61.6	61.1
Race				
White	97.6	98.0	98.4	98.3
Black	1.0	1.1	1.0	1.1
Other/missing	1.4	0.9	0.6	0.6
Education				
<High school	4.3	4.4	4.4	4.8
High school graduate	31.3	31.6	31.5	30.0
Some college	28.6	30.4	31.0	33.4
≥College graduate	35.2	32.9	32.6	31.2
Missing	0.6	0.7	0.5	0.6
Smoking status				
Never	56.0	53.5	51.8	48.2
Current	10.0	7.8	7.3	7.3
Former	32.6	37.4	39.5	43.3
Missing	1.4	1.3	1.4	1.2
Diabetes				
Yes	4.0	6.5	8.6	10.7
Number live births, age at first birth				
Nulliparous	7.9	7.5	7.1	7.8
1–2, <25 y	13.7	14.0	14.4	14.7
1–2, 25–29 y	13.3	11.9	12.3	10.7
1–2, 30+ y	6.3	6.0	5.2	4.9
3+, < 25 y	36.4	38.8	40.9	42.5
3+, 25–29 y	17.1	16.7	15.2	14.4
3+, 30+ y	2.8	2.8	2.5	2.5
Unknown/missing	2.5	2.3	2.4	2.5
Age at menarche, y	12.9	12.7	12.5	12.4
Age at menopause, y	51.2	51.2	51.3	51.2
Family history of endometrial cancer				
Yes	2.5	2.8	2.5	3.7
Oral contraceptive use				
Never	64.1	63.1	63.0	62.5
Ever	34.8	35.7	35.9	36.7
Unknown/missing	1.1	1.2	1.1	0.8
PMH use				
Never	57.3	58.8	58.9	60.9
Current	20.4	17.5	15.7	15.9
Former	18.7	20.2	22.2	19.9
Other/Unknown	3.6	3.5	3.2	3.2
BMI at age 18,[a] kg/m^2	19.9	20.9	21.8	22.6
BMI in 1982,[a] kg/m^2	22.5	25.1	27.0	28.3
BMI in 1992,[a] kg/m^2	23.3	26.4	28.7	30.3
BMI ≥ 30 kg/m^2 in 1992	3.8	16.2	33.7	46.9
Wt Δ 18 to age in 1992,[a] lbs	20.0	32.5	41.1	45.8
Wt Δ 18 to age in 1982,[a] lbs	15.2	24.5	31.1	33.9
Wt Δ 1982–1992,[a] lbs	4.7	7.9	10.1	11.9
Energy,[a] kcal/d	1,359	1,363	1,386	1,404
Exercise,[a] METS/h	12.3	11.8	11.5	12.0
Alcohol,[a] g/d	5.5	4.7	4.4	4.1

NOTE: Standardized to the age distribution of the women in the cohort. Values are presented as percentages unless otherwise noted.
Abbreviation: METS, physical activity in metabolic equivalents.
[a]Values are presented as means.

Table 2. Risk of incident endometrial cancer and weight cycling among women in the CPS-II Nutrition Cohort, 1992–2007

No. of weight cycles	Cases/person-years	RR[a] (95% CI)	RR[b] (95% CI)	RR[c] (95% CI)
Noncycler	182/202,184	1.00 (ref)	1.00 (ref)	1.00 (ref)
1–4	219/163,947	1.52 (1.25–1.85)	1.45 (1.19–1.77)	1.07 (0.87–1.32)
5–9	73/39,992	2.08 (1.58–2.74)	1.95 (1.48–2.58)	1.14 (0.85–1.53)
10+	85/42,203	2.33 (1.80–3.02)	2.13 (1.63–2.78)	1.05 (0.77–1.42)
P_{trend}		<0.0001	<0.0001	0.82

[a]Age-adjusted model.
[b]Base model stratified by age includes alcohol use, smoking, physical activity, family history of endometrial cancer, diabetes, number of live births/age at first birth, age at menarche, age at menopause, hormone replacement therapy use, oral contraceptive use, and total energy intake.
[c]Full model stratified by age includes covariates in base model, BMI in 1992 (continuous).

differently, which may contribute to the differing results among studies. However, the possibility that residual confounding by BMI also contributes should be considered. Both case–control studies (16, 17) adjusted for BMI using quartiles among controls, whereas our study and the Iowa Women's Health study adjusted for BMI using a continuous variable.

If weight cycling leads to the replacement of lean body mass with fat mass, then it may be expected to increase the risk of endometrial cancer in postmenopausal women. However, whether the balance of lean body mass and fat mass is really changed by weight cycling remains unclear because previous studies provided conflicting results (13, 14). Our finding of no increased risk associated with weight cycling could suggest either that repeated cycles of weight loss and regain do not change the ratio of fat to lean body mass or that any changes in this balance that occur are not sufficient to affect endometrial cancer risk. Additional studies of the influence of weight cycling on body composition are needed to determine if either of these possibilities is correct.

This study has several strengths, including its large study population and its prospective design. Detailed information on numerous covariates was used to analyze the association of weight cycling with endometrial cancer risk independent of these factors. Importantly, this study included more weight cyclers, both with and without endometrial cancer, than the previous studies (14–16), which allows for investigation of a history of 10 or more weight cycles, which was not done in previous studies (15–17).

Limitations of this study include the fact that the information on weight cycling was both self-reported and recalled. While this may result in some misclassification, there is no reason to expect it to be differential with respect to endometrial cancer risk. No information was collected on either the magnitude or the length of the weight cycles. Therefore, whether weight cycles of different magnitudes or duration might differentially affect risk for endometrial cancer cannot be determined. In addition, the questions used to determine weight cycling did not exclude postpregnancy weight loss. Whether a weight cycle which

Table 3. Risk of incident endometrial cancer and weight cycling by BMI in 1992 among women in the CPS-II Nutrition Cohort, 1992–2007

	BMI in 1992, kg/m^2					
	18.5–<25		25–<30		30–50	
No. of weight cycles	Cases	RR[a] (95% CI)	Cases	RR[a] (95% CI)	Cases	RR[a] (95% CI)
Noncycler	117	1.00 (ref)	48	1.00 (ref)	17	1.00 (ref)
1–4	56	1.09 (0.77–1.52)	87	0.99 (0.69–1.42)	76	1.32 (0.78–2.25)
5–9	7	1.01 (0.46–2.21)	19	0.87 (0.50–1.50)	47	1.52 (0.86–2.68)
10+	5	0.82 (0.33–2.05)	25	1.27 (0.77–2.12)	55	1.17 (0.67–2.05)
P_{trend}		0.82		0.41		0.85

[a]Model stratified by age includes alcohol use, smoking, physical activity, family history of endometrial cancer, diabetes, number of live births/age at first birth, age at menarche, age at menopause, hormone replacement therapy use, oral contraceptive use, total energy intake, and BMI in 1992 (continuous).

includes postpregnancy weight loss differs from those with purposeful weight loss for other reasons is not clear. Finally, the timing of the weight cycling was not ascertained, precluding investigation of the association between this behavior during different periods of life and endometrial cancer risk.

In summary, the results of this study suggest that cycles of intentional weight loss followed by regain do not contribute to risk of endometrial cancer independently of BMI. However, failure to maintain weight loss followed by net weight gain likely increases risk of various adverse health outcomes. Therefore, while overweight and obese women should be encouraged to try to lose weight, increased efforts should be made to help them maintain their weight loss.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interests were disclosed.

Authors' Contributions

Conception and design: V.L. Stevens, A.V. Patel, M.M. Gaudet, S.M. Gapstur.

Development of methodology: V.L. Stevens.

Acquisition of data (provided animals, acquired and managed patients, provided facilities, etc.): A.V. Patel, S.M. Gapstur.

Analysis and interpretation of data (e.g., statistical analysis, biostatistics, computational analysis): V.L. Stevens, E.J. Jacobs, J. Sun, M.L. McCullough, M.M. Gaudet, L.R. Teras, S.M. Gapstur.

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Acknowledgments

The authors thank the contribution to this study from central cancer registries supported through the CDC National Program of Cancer Registries and cancer registries supported by the NCI Surveillance, Epidemiology and End Results program.

Grant Support

The support for the creation, maintenance and updating of the CPS-II Nutrition Cohort is from the American Cancer Society.

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Received January 13, 2012; revised February 16, 2012; accepted February 16, 2012; published OnlineFirst February 23, 2012.

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