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Obesity, weight gain, and ovarian cancer risk in African American women

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Abstract

Although there is growing evidence that higher adiposity increases ovarian cancer risk, little is known about its impact in African American (AA) women, the racial/ethnic group with the highest prevalence of obesity. We evaluated the impact of body mass index (BMI) 1 year before diagnosis and weight gain since age 18 years on ovarian cancer risk in a population-based case-control study in AA women in 11 geographical areas in the US. Cases (n = 492) and age and site matched controls (n = 696) were identified through rapid case ascertainment and random-digit-dialing, respectively. Information was collected on demographic and lifestyle factors, including self-reported height, weight at age 18 and weight 1 year before diagnosis/interview. Multivariable logistic regression was used to compute odds ratios (OR) and 95% confidence intervals (CI), adjusting for potential covariates. Obese women had elevated ovarian cancer risk, particularly for BMI ≥ 40 kg/m^2 compared to BMI <25 (OR = 1.72, 95% CI: 1.12–2.66; p for trend: 0.03). There was also a strong association with weight gain since age 18 (OR: 1.52; 95% CI: 1.07–2.16; p for

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trend: 0.02) comparing the highest to lowest quartile. In stratified analyses by menopausal status, the association with BMI and weight gain was limited to postmenopausal women, with a 15% (95% CI: 1.05–1.23) increase in risk per 5 kg/m^2 of BMI and 6% (95% CI: 1.01–1.10) increase in risk per 5 kg of weight gain. Excluding hormone therapy users essentially did not change results. Obesity and excessive adult weight gain may increase ovarian cancer risk in post-menopausal AA women.

Keywords

obesity; weight gain; ovarian cancer; African American

Ovarian cancer is the most lethal gynecologic cancer in the United States.[1] This is due in part to the difficulties in early detection because symptoms tend be nonspecific and the lack of effective screening tools.[1] For this reason, identifying modifiable risk factors is particularly important. Compared to whites, African American (AA) women have lower risk of being diagnosed with ovarian cancer, but they tend to present with more aggressive disease and to experience worse survival.[2] Little is known about the epidemiology of ovarian cancer in AA women, but there is suggestive evidence that there may be some differences in risk profiles for this group.[3]

There is growing evidence that obesity increases ovarian cancer risk but the association varies by tumor subtype, menopausal status and hormone therapy use.[4] Little is known about these associations in AA women, a group that is most affected by obesity than any other racial/ethnic group in the United States [e.g., prevalence of obesity (BMI ≥ 30 kg/m^2) is 56.6% in AA women and 32.8% in white women].[5] To our knowledge, only two small case-control studies[3,6] with fewer than 115 cases evaluated the association in AA women and found no significant association, but statistical power was limited in both studies. We evaluated the impact of BMI and weight gain since age 18 years on ovarian cancer risk in AA women in a new study specifically designed to investigate risk factors for ovarian cancer in AA women.

Material and Methods

The African American Cancer Epidemiology Study (AACES), an ongoing population-based case-control study conducted in 11 sites in the United States (Alabama, Georgia, Illinois, Louisiana, Michigan, New Jersey, North Carolina, Ohio, South Carolina, Tennessee, Texas), which has been described in detail elsewhere.[7,8] In brief, eligible cases included all AA women aged 20–79 years newly diagnosed with histologically confirmed invasive epithelial ovarian cancer. They were identified since December 1, 2010 by state cancer registries, SEER (Surveillance, Epidemiology, and End Results) registries, or gynecologic oncology departments through rapid case ascertainment at participating sites. AA controls were identified since May 2011 using random digit dialing, matched to cases by 5-year age category and state of residence. Women with a previous diagnosis of ovarian cancer or with bilateral oophorectomy were excluded. Among those who could be contacted, 66.5% of potential cases and 72% of potential controls agreed to participate in the main telephone interview.[7]

Data collection was carried out by administering a computer-assisted telephone interview to collect information on demographic characteristics, anthropometric factors, reproductive history, medical history, hormone and medication use, family history of cancer, lifestyle characteristics (e.g., smoking, physical activity). In an effort to increase participation, those not willing to complete the full interview were offered the option of completing a short version of the questionnaire, which included all the key covariates and anthropometric factors needed for this analysis. Anthropometric factors included self-reported height and weight one year before diagnosis (cases) or interview (controls) and weight at age 18 years. Here we include cases and controls who completed the study by January 2015, totaling 512 cases and 722 controls. We excluded 20 cases and 26 controls for having missing data for self-reported height and weight 1 year before diagnosis/interview or covariates needed for this analysis resulting in 492 cases and 696 controls. Of those, 40 cases and 17 controls completed the short questionnaire. The study was approved by the Institutional Review Board at each site and all participants provided informed consent.

Statistical analyses

Distribution of key demographic variables and known or suspected risk factors for ovarian cancer were compared using χ^2 tests. Body mass index (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. There were only 14 underweight (BMI < 18.5) women (4 cases and 10 controls) and therefore we combined them with the normal weight group (BMI 18.5–24.9). Excluding underweight women did not change results.

Weight gain was computed as recent weight (1 year before) minus weight at age 18 years. Percent weight gain was calculated as recent weight minus weight at age 18 years divided by weight at age 18. Quartiles were used for weight gain and percent weight gain, with cutpoints based on the distribution of all controls combined. The same cutpoints were used for pre- and postmenopausal women to be able to compare estimates across menopausal status.

Multivariable unconditional logistic regression was used to estimate odds ratios (OR) and 95% confidence intervals (CI) after adjusting for potential covariates. The first model adjusted for age, geographic region (south- and mid-Atlantic, south central, midwest), and education (high school or less, some post-high school training, college or graduate degree). Additional covariates considered for inclusion in model 2 are the variables shown in Table 1. Variables retained in model 2 were those in model 1 plus those changing the effect estimate by >10% and included parity (0, 1–2, >2), oral contraceptive use (never, <60 months, ≥60 months), age at menarche (<12, 12–13, >13), menopause status (pre-, post-menopause), tubal ligation (no, yes), and first-degree family history of breast/ovarian cancer (no, yes). Weight gain was further adjusted for weight at age 18. *p* values for trend were computed by including the median in each quartile as a continuous variable in regression models. Stratified analyses by menopausal status were conducted with statistical significance for interaction obtained via the likelihood ratio test. Analyses in postmenopausal women were repeated after excluding hormone therapy users to assess possible effect modification. The

number of users was too small to conduct separate analyses on them. Analyses were also conducted by histologic subtypes and tested for heterogeneity. All statistical analyses were performed using Stata (version 13.1; StataCorp LP).

Results

The distribution of demographics and other factors known or suspected to affect ovarian cancer risk for cases and controls is shown in Table 1. Cases tended to be older and less educated than controls. The distribution of other risk factors was in the expected direction (e.g., less likely to have children, use oral contraceptive or to have had a tubal ligation and more likely to have a family history of breast or ovarian cancers). As shown in Table 2, cases were also more likely to be obese and to have gained weight since age 18 years. Overall, obesity was highly prevalent in this population: 62% of the cases and 56% of the controls were obese (BMI ≥ 30).

After adjusting for relevant covariates, we found an association between BMI one year before diagnosis and ovarian cancer risk (Table 2), particularly for those with a BMI ≥ 40 (OR: 1.72; 95% CI: 1.12–2.66) with a p for trend of 0.03, compared to a BMI < 25. BMI at age 18 was not associated with ovarian cancer risk. However, weight gain since that age increased risk, with those in the highest quartile of weight gain (corresponding to gaining 36.3 kg or more) having 52% higher risk of developing ovarian cancer, compared to the lowest quartile (OR: 1.52; 95% CI: 1.07–2.16). We estimated a 4% increased risk per 5 kg of weight gain since age 18 (95% CI: 1.00–1.07). For this analysis we excluded women who reported having lost weight since age 18 (13 cases and 32 controls). Results for percent weight gain were virtually the same (data not shown).

In stratified analyses by menopausal status (Table 3), the association with both BMI 1 year before diagnosis and weight gain since age 18 was limited to postmenopausal women (p for interaction: 0.04 and 0.09, respectively). Among them, there was a significant dose-response relationship with BMI (p for trend = 0.004), with those with a BMI ≥ 40 having an OR of 1.94 (95% CI: 1.14–3.30), corresponding to 15% increase in risk per 5 units of BMI (OR: 1.15; 95% CI: 1.05–1.26). For weight gain, risk more than doubled for those in the highest quartile compared to the lowest (p for trend 0.001), with an estimated 6% increase in risk per 5 kg of weight gain since age 18 (OR: 1.06; 95% CI: 1.01–1.10). We repeated analyses excluding postmenopausal hormone therapy users and results were very similar (Table 3). We also evaluated the impact of BMI and weight gain by histologic subtype (Table 4) and found similar magnitude of associations across subtypes and no significant heterogeneity.

Discussion

In the first thorough evaluation of the association between BMI and ovarian cancer risk in AA women, we found that body fatness had a strong impact on increasing ovarian cancer risk in AA women. The evidence was consistent for both a current measure of BMI as well as adult weight gain. These associations were largely confined to postmenopausal women.

Several mechanisms have been proposed to explain the obesity-cancer link, including insulin resistance, increased steroid hormone bioavailability from adipose tissue, systemic

inflammation, and altered adipokine (leptin and adiponectin) pathophysiology.[9] Adipokines are particularly relevant given the central role of inflammation on ovarian cancer etiology:[10,11] circulating leptin is proportional to the amount of body fat and a potent inflammatory agent, whereas adiponectin is inversely related to adiposity and has potent antiinflammatory activity.[9] Biomarkers of systemic inflammation, such as C-reactive protein (CRP), tumor necrosis factor (TNF), interleukin-1β (IL-1β), IL-6 and IL-18 have also been shown to increase as adiposity increases.[9] Leptin and some of these pro-inflammatory cytokines are also associated with immune suppression.[12] Therefore, obesity leads to a state of chronic inflammation and immune suppression which favors carcinogenesis and tumor growth and progression.

There is growing evidence that obesity increases the risk of ovarian cancer with several recent pooled analyses[13–15] and meta-analyses[4,16–18] consistently reporting an association. However, studies included in these reports were largely from white populations and these analyses included few AA women. The only two studies[3,6] that evaluated the association in AA women were based in small numbers of AA participants (<115 cases), which did not allow definite conclusions. In the North Carolina Ovarian Cancer Study,[3] compared to a BMI <25, BMI ≥ 35 (highest category evaluated) was associated with an OR of 1.04 (95% CI: 0.75–1.45) in whites and 1.62 (95% CI: 0.79–3.35) in AA women.[3] The study included only 111 AA cases and 189 AA controls, which did not allow the evaluation of the association by menopausal status, hormone therapy use, or histologic subtype. Nevertheless, it suggested a stronger association in AA compared to white women. The other study was conducted in the Delaware Valley (including contiguous counties in eastern Pennsylvania, southern New Jersey, and Delaware) and reported no association with BMI six months before diagnosis.[6] Only 84 AA cases and 204 AA controls were included in this analysis and the ascertainment period may have been too close to diagnosis, as the cases may have started to lose weight caused by the disease.

In the Ovarian Cancer Association Consortium (OCAC), which pooled 15 case-control studies and, therefore, results may be more comparable to our study (based on study design), the summary OR for BMI ≥ 40 was 1.22 (95% CI: 1.05–1.41), with a 4% increase per 5 kg/m^2 (95% CI: 1.00–1.08). In our study, we found stronger estimates for the same level and increment of BMI in AA women, compared to OCAC, which pools data from studies including mostly white women. This raises the possibility that excess adiposity may have a larger impact in AA women, which is biologically plausible as AA women have been shown to have important differences in body composition and obesity-related biomarkers, compared to white women. AA women tend to have more lean mass and lower fat mass for the same level of BMI,[19] and less visceral fat and more subcutaneous adipose tissue for a given amount of body fat.[20] However, AA women are more likely to be insulin resistant than whites at similar levels of adiposity[21,22] and to have higher levels of inflammatory markers (CRP and leptin) and lower levels of adiponectin.[23]

In addition to BMI, we evaluated the impact of weight gain since age 18, which may be a better measure of adiposity as it represents body fatness accumulated over the years, in contrast to BMI which is a mixture of lean mass and fat mass.[24] In agreement with our study, a recent meta-analysis of prospective studies reported an increased risk of ovarian

cancer associated with postmenopausal weight gain, with an estimate of 13% increased risk per 5 kg of weight gain (95% CI: 1.03–1.23).[24] To our knowledge only the North Carolina Ovarian Cancer Study[25] has evaluated weight gain in AA women in an analysis including only 79 cases and 86 controls and found no association. However, the study lacked power to detect an association.

Previous studies have suggested a stronger association of BMI with ovarian cancer in premenopausal women,[13–15] but in contrast in our study the association with obesity and weight gain was limited to postmenopausal women. While the reasons for these findings are unknown, it is possible that the impact of excess adiposity becomes more detrimental with longer years of exposure, particularly at the high levels of obesity in this population. It may also relate to differences in body composition. For example, the menopausal transition is characterized by increases in total body fat and visceral fat,[26] which in turn promotes inflammation, oxidative stress, cytokines and adipokines.[27] Weight gain during menopause tends to increase abdominal fatness and insulin resistance.[28] Furthermore, the elevated testosterone and insulin levels in obese women are particularly higher in postmenopausal women, and both hormones have been implicated in ovarian cancer etiology.[16]

Hormone therapy use has been shown to be an effect modifier of the association of adiposity with ovarian cancer and other cancers (breast, endometrium), with the association being stronger or limited to users.[9] However, in our study in AA women, results essentially did not change after excluding hormone therapy users. Interestingly, similar findings were observed in a recent consortium of four studies of breast cancer in AA women evaluating the impact of obesity on breast cancer risk: results were unaffected after excluding hormone therapy users.[29] This could perhaps be explained by different prevalence of use and formulations in AA women, who are less likely to be users overall, and because of the high hysterectomy rates in this population, they tend to use estrogen only formulations.[30] Over 80% of our study population reported no use of hormone therapy.

Previous pooled data analyses suggested different association with BMI for the different histologic subtypes.[15,31] For example, in OCAC,[15] a higher BMI did not have an impact on high grade invasive serous tumors, while increasing risk for low grade invasive serous, as well as for other invasive tumors, including mucinous and endometrioid, and borderline tumors. In contrast, in our study in AA women risk estimates were very similar across histologic subtypes. However, our analyses were based on small numbers (e.g., 25 mucinous and 60 endometrioid), limiting our statistical power to detect differences in risk.

Additional limitations of our study should be mentioned, including inherent limitations of case-control studies, such as selection bias and recall bias. Similar to other population-based studies, maintaining a high participation rate was challenging. However, there is some reassuring evidence showing that declines in participation rates in epidemiologic studies may not substantially influence point estimates of exposure-disease associations.[32] Furthermore, while the potential for selection bias can never totally discarded, the fact that the distribution of risk factors for ovarian cancer among cases and controls was in the expected direction gives us reassurance about the validity of our findings. Recall bias is also a possibility if cases tended to report their weight and height differently than controls.

However, the association of obesity with ovarian cancer is not widely known. One potential concern is the use of self-report of weight and height, given the known tendency for subjects to overreport their height and underreport weight, which results in random underestimation of BMI.[33] However, studies have shown a strong correlation (>0.9) between self-reported and measured weight and height.[34–37] Moreover, a recent meta-analysis showed that the summary estimates for ovarian cancer risk were similar for studies that used self-reported and measured BMI.[18]

An important strength of our study is that it was designed specifically to evaluate risk factors of ovarian cancer in AA and represents the largest study to date assessing the impact of obesity and weight gain during adulthood on ovarian cancer risk in AA women. Through the unique infrastructure of AACES we were able to recruit women throughout a large geographical area, which allows representation of a wider range of BMI than have been included in other studies.

Obesity is a major burden and a critical issue that disproportionately affects AA women, given its high prevalence (56.6% in the United States)[5] and metabolic and health consequences, such as metabolic syndrome, type 2 diabetes, hypertension, cardiovascular disease[38] and cancer.[39] There is also growing evidence that obesity has a detrimental effect on survival after a cancer diagnosis,[40] including ovarian cancer.[41,42] Therefore, weight control is critical to reduce the mortality gap in this population. Our study adds to this evidence and suggests that maintaining a healthy weight through adulthood may also reduce ovarian cancer risk in AA women.

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Abbreviations

AA	African American
AACES	African American Cancer Epidemiology Study
BMI	body mass index

CI	confidence intervals
CRP	C-reactive protein
IL-1β	interleukin-1β
OCAC	Ovarian Cancer Association Consortium
OR	odds ratio
TNF	tumor necrosis factor
WHO	World Health Organization

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What's new?

While obesity can raise a woman's risk for certain types of cancer, menopausal status and hormone therapy significantly influence those associations. Moreover, in the case of ovarian cancer, racial/ethnic associations may also be at play, though links with adiposity remain obscure for African American women, a group disproportionately affected by obesity in the United States. Here, analyses of data from the U.S. population-based African American Cancer Epidemiology Study reveal a strong association between high adiposity and increased ovarian cancer risk, particularly among postmenopausal women. The findings highlight the importance of weight control in reducing ovarian cancer risk among African American women.

Table 1

Selected characteristics of African American women participating in AACES

	Cases (*n* = 492)		Controls (*n* = 696)		
	n	**%**	***n***	**%**	***p* value[1]**
Age					
<50	110	22.4	193	27.7	0.005
50–59	168	34.2	263	37.8	
≥60	214	43.5	240	34.5	
Education					
High school or less	224	45.5	256	36.8	0.01
Some post-high school training	151	30.7	252	36.2	
College or graduate degree	117	23.8	188	27.0	
Region[2]					
South- and mid-Atlantic	271	55.1	366	52.6	0.04
South central	134	27.2	166	23.9	
Midwest	87	17.7	164	23.6	
Parity					
0	92	18.7	93	13.4	0.04
1–2	207	42.1	313	45.0	
>2	193	39.2	290	41.7	
Smoking status					
Never	280	56.9	405	58.2	0.66
Past/current	212	43.1	291	41.8	
Oral contraceptive use					
Never	151	30.7	141	20.3	<0.001
<60 months	192	39.0	315	45.3	
≥60 months	149	30.3	240	34.5	
Use of hormone therapy					
Never	389	79.1	579	83.2	0.09

	Cases (*n* = 492)		Controls (*n* = 696)		
	n	**%**	***n***	**%**	***p* value[1]**
Ever	100	20.3	115	16.5	
Missing	3	0.6	2	0.3	
Age at menarche					
<12	110	22.4	182	26.2	0.30
12–13	257	52.2	339	48.7	
>13	125	25.4	175	25.1	
Menopause status					
Premenopausal	133	27.0	215	30.9	0.15
Postmenopausal	359	73.0	481	69.1	
Tubal Ligation					
No	328	66.7	421	60.5	0.03
Yes	164	33.3	275	39.5	
Hysterectomy					
No	373	75.8	544	78.2	0.34
Yes	119	24.2	152	21.8	
Diabetes					
No	381	77.4	536	77.0	0.86
Yes	111	22.6	160	23.0	
Family history of breast/ovarian cancer (first-degree relative)					
No	364	74.0	567	81.5	0.002
Yes	128	26.0	129	18.5	

[1]Chi-square tests for *p*-value.

[2]South- and mid-Atlantic includes GA, NC, SC, NJ; South central includes AL, TN, LA, TX; and Midwest includes IL, MI, OH.

Table 2

Association of anthropometric factors and ovarian cancer risk in AACES

	Cases (*n* = 492)		Controls (*n* = 696)					
	n	**%**	***n***	**%**	**OR1**	**95% CI**	**OR2**	**95% CI**
BMI 1 year before (kg/m^2)								
<25	65	13.2	124	17.8	1.00	Ref	1.00	Ref
25–<30	123	25.0	183	26.3	1.19	0.81, 1.75	1.29	0.87, 1.91
30–<35	135	27.4	182	26.2	1.35	0.92, 1.97	1.38	0.94, 2.04
35–<40	80	16.3	112	16.1	1.29	0.84, 1.96	1.32	0.86, 2.03
≥40	89	18.1	95	13.7	1.69	1.10, 2.58	1.72	1.12, 2.66
p for trend						0.02		0.03
Per 5 kg/m^2					1.10	1.02, 1.17	1.09	1.01, 1.17
BMI at age 18 (kg/m^2) [1]								
<18.5	94	19.8	129	18.6	1.08	0.79, 1.47	1.07	0.78, 1.47
18.5–<25	276	58.0	417	60.3	1.00	Ref	1.00	Ref
25–<30	67	14.1	100	14.5	1.08	0.76, 1.54	1.04	0.72, 1.49
≥30	39	8.2	46	6.7	1.37	0.86, 2.19	1.20	0.74, 1.94
p for trend						0.34		0.68
Per 5 kg/m^2					1.03	0.92, 1.15	0.99	0.88, 1.11
Weight gain (kg) [1]								
Q1 (0–13.5)	98	21.2	168	25.5	1.00	Ref	1.00	Ref
Q2 (13.6–23.9)	115	24.8	175	26.5	1.10	0.76, 1.59	1.20	0.84, 1.71
Q3 (24.0–36.2)	103	22.2	158	23.9	0.98	0.67, 1.43	1.11	0.77, 1.60
Q4 (≥36.3)	147	31.8	159	24.1	1.47	1.03, 2.11	1.52	1.07, 2.16
p for trend						0.02		0.02
Per 5 kg					1.04	1.00, 1.07	1.04	1.00, 1.07

OR, Odds Ratio; CI, Confidence Interval.

OR1, Adjusted for age, education (high school or less, some post-high school training, college or graduate degree), region (south- and mid-Atlantic, South central, Midwest).

OR2, Further adjusted for parity (0, 1–2, >2), oral contraceptive use (never, <60 mo, ≥60 mo), age at menarche (<12, 12–13, >13, menopause status (pre, post-menopausal), tubal ligation (yes, no), family history of breast/ovarian cancer (yes, no). Weight gain and percent weight gain further adjusted for weight at age 18.

[1] Sixteen cases and four controls had missing data for weight at age 18 and weight gain.

Table 3

Associations of BMI, weight gain and ovarian cancer risk by menopausal status[1]

	Cases		Controls					
	n	%	*n*	%	OR1	95% CI	OR2	95% CI
Premenopausal women								
BMI 1 year before (kg/m^2)								
<25	23	17.3	45	20.9	1.00	Ref	1.00	Ref
25–<30	33	24.8	45	20.9	1.49	0.73, 3.02	1.91	0.91, 4.04
30–<35	37	27.8	50	23.3	1.51	0.76, 3.00	1.40	0.67, 2.90
35–<40	19	14.3	43	20.0	0.77	0.36, 1.66	0.70	0.31, 1.57
≥40	21	15.8	32	14.9	1.21	0.55, 2.64	1.28	0.56, 2.92
p for trend						0.72		0.57
Per 5 kg/m^2					0.99	0.88, 1.11	0.97	0.86, 1.10
Weight gain (kg)[2]								
Q1 (0–13.5)	43	34.7	63	31.0	1.00	Ref	1.00	Ref
Q2 (13.6–23.9)	27	21.8	55	27.1	0.63	0.33, 1.18	0.80	0.41, 1.58
Q3 (24.0–36.2)	27	21.8	35	17.2	0.91	0.47, 1.79	1.07	0.53, 2.19
Q4 (≥36.3)	27	21.8	50	24.6	0.64	0.33, 1.23	0.73	0.37, 1.47
p for trend						0.29		0.48
Per 5 kg					0.98	0.92, 1.05	0.99	0.93, 1.06
Postmenopausal women								
BMI 1 year before (kg/m^2)								
<25	42	11.7	79	16.4	1.00	Ref	1.00	Ref
25–<30	90	25.1	138	28.7	1.15	0.72, 1.83	1.17	0.73, 1.89
30–<35	98	27.3	132	27.4	1.35	0.85, 2.14	1.37	0.85, 2.19
35–<40	61	17.0	69	14.4	1.60	0.96, 2.68	1.65	0.97, 2.78
≥40	68	18.9	63	13.1	1.96	1.17, 3.27	1.94	1.14, 3.30
p for trend						0.003		0.004
Per 5 kg/m^2					1.16	1.06, 1.27	1.15	1.05, 1.26

	Cases		Controls					
	n	%	*n*	%	OR1	95% CI	OR2	95% CI
Weight gain (kg)[2]								
Q1 (0–13.5)	55	16.2	105	23.0	1.00	Ref	1.00	Ref
Q2 (13.6–23.9)	88	26.0	120	26.3	1.40	0.91, 2.16	1.50	0.97, 2.34
Q3 (24.0–36.2)	76	22.4	123	26.9	1.17	0.75, 1.81	1.24	0.79, 1.94
Q4 (≥36.3)	120	35.4	109	23.8	2.08	1.36, 3.17	2.10	1.37, 3.23
p for trend						0.001		0.001
Per 5 kg					1.06	1.02, 1.10	1.06	1.01, 1.10
Postmenopausal non-HT users								
BMI 1 year before (kg/m^2)								
<25	28	10.5	60	16.0	1.00	Ref	1.00	Ref
25–<30	65	24.4	106	28.3	1.23	0.71, 2.14	1.24	0.70, 2.18
30–<35	73	27.4	102	27.2	1.47	0.85, 2.53	1.45	0.83, 2.53
35–<40	48	18.1	54	14.4	1.81	0.99, 3.30	1.79	0.97, 3.31
≥40	52	19.6	53	14.1	1.96	1.08, 3.56	1.84	0.99, 3.41
p for trend						0.009		0.02
Per 5 kg/m^2					1.18	1.07, 1.31	1.16	1.05, 1.29
Weight gain (kg)[2]								
Q1 (0–13.5)	35	14.1	85	23.9	1.00	Ref	1.00	Ref
Q2 (13.6–23.9)	65	26.1	88	24.8	1.79	1.07, 3.00	1.93	1.14, 3.28
Q3 (24.0–36.2)	57	22.9	98	27.6	1.36	0.81, 2.29	1.44	0.85, 2.46
Q4 (≥36.3)	92	36.9	84	23.7	2.56	1.56, 4.22	2.54	1.52, 4.24
p for trend						0.001		0.001
Per 5 kg					1.07	1.02, 1.12	1.06	1.01, 1.12

OR: Odds Ratio; CI: Confidence Interval.

OR1: Adjusted for age, education (high school or less, some post-high school training, college or graduate degree), region (south- and mid-Atlantic, South central, Midwest). OR2: Further adjusted for parity (0, 1–2, >2), oral contraceptive use (never, <60 mo, ≥60 mo), age at menarche (<12, 12–13, >13, menopause status (pre, post-menopausal), tubal ligation (yes, no), family history of breast/ovarian cancer (yes, no). Weight gain further adjusted for weight at age 18.

[1] *p* for interaction between menopausal status and BMI 1 year before or weight gain is 0.04 or 0.09 respectively.

[2] 16 cases and 4 controls had missing data for weight at age 18 and weight gain.

Table 4

Associations of BMI and ovarian cancer risk by histologic subtype in AACES (cases *vs.* all controls)

	BMI, per 5 kg/m^2				Weight Gain, per 5 kg[3]			
	Cases *n*	**OR**	**95% CI**	**p_{het}**	**Cases *n***	**OR**	**95% CI**	**p_{het}**
All cases	492	1.09	1.01, 1.17	–	463	1.04	1.00, 1.07	–
Serous	279	1.10	1.01, 1.20	0.78	267	1.05	1.01, 1.09	0.78
High-grade serous only[1]	225	1.08	0.98, 1.19		216	1.04	1.00, 1.09	
Endometrioid	60	1.10	0.94, 1.29		54	1.04	0.96, 1.13	
Mucinous	25	0.93	0.72, 1.20		23	0.97	0.87, 1.09	
Others[2]	99	1.12	0.99, 1.27		91	1.04	0.97, 1.10	

OR: Odds Ratio; CI: Confidence Interval. Adjusted for age, education (high school or less, some post-high school training, college or graduate degree), region (south- and mid-Atlantic, South central, Midwest), parity (0, 1–2, >2), oral contraceptive use (never, <60 mo, ≥60 mo), age at menarche (<12, 12–13, >13, menopause status (pre, post-menopausal), tubal ligation (yes, no), family history of breast/ovarian cancer (yes, no).

[1] Serious cases with unknown grade were excluded ($n = 39$).

[2] Others include clear cell ($n = 10$), mixed ($n = 17$), NOS ($n = 63$) and others ($n = 9$). Those with missing histology ($n = 29$) were excluded.

[3] Sixteen cases and four controls had missing data for weight at age 18 and weight gain.