Published in final edited form as:

Ann Allergy Asthma Immunol. 2014 April ; 112(4): 333–338.e1. doi:10.1016/j.anai.2013.12.025.

Depressive symptoms and the incidence of adult-onset asthma in African American women

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Abstract

Background—Some evidence suggests that depression may increase the risk of adult-onset asthma. No data are available for African American women, in whom the prevalence of depression and asthma is high.

Objective—To conduct prospective analyses of the relation of depressive symptoms to asthma incidence in the Black Women's Health Study, a prospective cohort of US black women followed since 1995 with mailed biennial questionnaires.

Methods—Of 31,848 participants followed from 1999 to 2011, 771 reported incident asthma. Depressive symptoms were ascertained on 1999 and 2005 follow-up questionnaires with the Center for Epidemiological Studies–Depression Scale (CES-D). Participants rated the frequency of 20 symptoms. A score was calculated by summing the responses to all questions. Cox regression models were used to derive incidence rate ratios and 95% confidence intervals for 4 categories of the CES-D score in relation to incident asthma, adjusted for body mass index, smoking, and other covariates.

Results—The multivariable incidence rate ratio in the highest category of CES-D score (≥ 33) compared with the lowest (<16) was 2.08 (95% confidence interval 1.58–2.74), with a significant trend ($P < .0001$). The incidence rate ratio was higher in women who took antidepressants, were current or former smokers, were not obese, and were at least 40 years old, although there were no statistically significant interactions.

Conclusion—A positive association was observed between CES-D score and the incidence of adult-onset asthma. If the hypothesis is confirmed, depression could contribute substantially to the burden of asthma in adults.

Introduction

Asthma and depression are significant burdens on the health of African American women, with estimated prevalences of 11.6%¹ (current asthma) and 13.1%² (lifetime prevalence of major depressive disorder). Depression commonly coexists with asthma and is associated

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Disclosures: Authors have nothing to disclose.

Supplementary Data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.anai.2013.12.025>.

with more severe asthma and poorer asthma management.^{3–5} Some data suggest that depressed or sad moods elicited under laboratory conditions can produce pulmonary effects consistent with decreased airway function, but overall the evidence is weak.^{6–8} Stress is a known trigger of asthma exacerbation in patients with asthma⁹; it has been hypothesized that stress effects on the immune and autonomic nervous systems are also relevant to the development of asthma.^{10,11} Major depressive disorder leads to overproduction of corticotropin-releasing hormone and hyperactivity of the hypothalamic–pituitary–adrenal axis,^{12,13} hallmarks of stress.

Although the influence of maternal and perinatal stress on asthma onset in the young child has received most study,¹¹ some epidemiologic evidence suggests that increased risk of adult-onset asthma is associated with stress, including perceived stress,¹⁴ war-related stress,¹⁵ abuse during childhood,¹⁶ and experiences of racism.¹⁷ Three prospective cohort studies—the Canadian National Population Health Survey,¹⁸ the National Health and Nutrition Examination Survey (NHANES),¹⁹ and a German cohort study²⁰—reported that the risk of new-onset asthma was increased in adults with a history of depression by 24%²⁰ to approximately 200%.^{18,19} No such data are available in an African American population.

The purpose of the present analysis was to assess the association between depressive symptoms and adult-onset asthma in a prospective cohort study of African American women, the Black Women’s Health Study (BWHS). In previous work, the authors found that high body mass index (BMI),²¹ experiences of child abuse,¹⁶ and experiences of racism¹⁷ were associated with an increased incidence of adult-onset asthma.

Methods

The Black Women’s Health Study

The BWHS is a prospective cohort study established in 1995, when 59,000 African American women 21 through 69 years old enrolled by completing health questionnaires.²² The baseline questionnaire elicited information on demographic and lifestyle factors, reproductive history, and medical conditions. The cohort is followed biennially by mailed questionnaire to update exposures and ascertain incident disease. Follow-up of the original cohort through 7 completed questionnaire cycles is 80%. The study protocol was approved by the institutional review board of the Boston University School of Medicine (Boston, Massachusetts). Participants indicated consent by completing and returning the questionnaires.

Diagnosis of Asthma

On all questionnaires from 1999 through 2011, participants were asked whether they had been diagnosed with asthma and whether they used “inhalers or pills” for asthma at least 3 days per week. Incident asthma was defined as a first diagnosis of asthma with concurrent use of asthma medication. During follow-up from 1999 through 2011, there were 771 women who met the case criteria. In a subset of 43 women who met the case definition and who gave permission to contact their physicians, 39 (91%) were confirmed by the physician as having asthma.

Ascertainment of Depressive Symptoms

Depressive symptoms were ascertained by the Center for Epidemiological Studies–Depression Scale (CES-D),²³ which was included in the 1999 and 2005 follow-up questionnaires. Participants rated 20 symptoms (eg, “I felt depressed,” “I was happy,” “I felt lonely”) on a 4-point scale ranging from “rarely or none of the time” to “most or all of the time,” scored from 0 to 3 (symptoms listed in eTable 1). Responses were summed to create a

composite score ranging from 0 (no depressive symptoms) to 60 (highest score on all symptoms). Although the CES-D score cannot be used to identify clinical depression, its validity as a screening tool for detecting depressive symptoms has been demonstrated.^{23,24} A high correlation has been found between the CES-D and the Major Depression Inventory (Spearman $\rho = 0.86$).²⁵ The CES-D measures depressive symptoms across 4 domains: depressed affect, positive affect, somatic complaints/activity inhibition, and interpersonal difficulties.²³ In confirmatory factor analyses in the BWHS, the same factors were extracted from the CES-D scale as in the white population in which it was developed,²⁶ demonstrating its validity for measuring depressive symptoms in African American women. The authors also verified in the BWHS that the scale is appropriate for use regardless of the health of the participant.²⁷

Ascertainment of Covariates

Data on weight and height were obtained at baseline, and weight was updated on each subsequent follow-up questionnaire. Data on smoking, alcohol consumption, hours per week spent in vigorous physical activity, and use of female hormone supplements were ascertained on most questionnaires. In 1997 respondents were asked about exposure to secondhand tobacco smoke at 0 to 10 years of age (home), 11 to 20 years of age (home), 21 to 30 years of age (home and work), 31 to 40 years of age (home and work), and currently (home and work), with exposure defined as being "in the same room with a smoker for at least an hour a day for 12 consecutive months or more." Various follow-up questionnaires also obtained information on years of education, asthma in the participant's mother or father, household income, health insurance and having a physician, sleep apnea, abuse as a child,¹⁶ antidepressant use, coping skills (assessed with a short form of the Brief COPE Scale²⁸), and perceptions of racism¹⁷ (assessed with questions adapted from an instrument developed by Williams et al²⁹).

Analytic Cohort

Follow-up for the present analysis was 1999 through 2011. Of 48,846 participants who returned the 1999 questionnaire and at least 1 follow-up questionnaire, the authors excluded 9,663 who did not answer at least 1 of the 1999 CES-D questions, 4,494 with prevalent asthma, 840 who reported asthma as a child, 753 who reported an asthma diagnosis but no concurrent use of asthma medication, 1,160 who reported use of asthma medication in the absence of an asthma diagnosis, 65 who met the case definition but disconfirmed the asthma diagnosis on a supplemental questionnaire, and 23 women with lung cancer, leaving 31,848 participants.

Women excluded because they did not answer all the 1999 CES-D questions were older and had less education than women who answered all the 1999 CES-D questions (mean ages 46 and 42 years and 27% and 16% with no more than a high school education, respectively). The 2 groups were similar in asthma incidence during follow-up (2.5% of excluded and 2.6% of included participants), BMI in 1999 (mean BMI 29 kg/m² for the 2 groups), and proportion who were current smokers (14% of included and 15% of excluded participants).

Statistical Methods

Cox proportional hazards models were used to estimate incidence rate ratios (IRRs) and 95% confidence intervals (CI) for asthma incidence associated with 4 categories of CES-D score: lower than 16, 16 to 22, 23 to 32, and at least 33. A score of at least 16 is customarily used to identify individuals at high risk of depression.^{23,30} The authors created the 3 highest categories to identify groups with increasing likelihood of depression. Participants contributed person time from 1999 until diagnosis of asthma, death, loss to follow-up, or end of follow-up, whichever came first. This study presents a model adjusted for age and

questionnaire cycle and a multivariable model adjusted for those factors plus BMI (kilograms per meter squared; <25, 25–29, 30–34, 35–39, or 40), menopausal female hormone use (never, <5 years, of 5 years), presence of sleep apnea (yes or no), household income (\$25,000, \$25,001–\$50,000, \$50,001–\$100,00, or >\$100,000), and pack-years of smoking (never smoker, 1–4, 5–14, 15–24, or 25). Missing values were modeled as separate categories. With the exception of sleep apnea and income, the variables varied by time. The 1999 CES-D score was assigned to follow up from 1999 to 2003 and the 2005 CES-D score was assigned to follow up from 2005 to 2011. The 1999 CES-D score was carried forward for women missing the 2005 CES-D score ($n = 11,371$). Addition of the following variables to the model did not materially change the IRRs: parental history of asthma, education, having a regular physician, having health insurance, secondhand smoke exposure at various ages, and experiences of abuse as a child and of racism as an adult (associated with asthma incidence and depressive symptoms in the BWHS^{16,17}). Control for BMI and pack-years of smoking using baseline values rather than time-varying values (which could have been influenced by depressive symptoms) did not change the results.

The authors conducted analyses stratified by age, BMI, smoking status, and coping score (lower than vs at least the median). Because coping scores were obtained in 2005, that stratified analysis used follow-up only from 2005 through 2011. The authors tested for interaction between the stratifying variables and CES-D score by conducting likelihood ratio tests on models with and without interaction terms.

Results

Compared with women in the lowest category of depressive symptoms (Table 1), those with higher levels were younger, heavier, had fewer years of education and lower income, and engaged in fewer hours of vigorous exercise. Women with higher levels also were more likely to smoke, to have been exposed to secondhand smoke at all ages, to report sleep apnea, to have used female hormone supplements, and were less likely to have a regular physician and health insurance. All covariates were significantly associated with CES-D score ($P < .001$), with the exception of parental history of asthma ($P = .26$).

There was a positive relation between CES-D score and asthma incidence (Table 2). The IRR adjusted only for age and calendar time was 2.59 (95% CI 1.98–3.40) for the highest compared with the lowest category of CES-D score, with a significant trend ($P < .0001$). The association was slightly attenuated after controlling for multiple confounding factors: the IRR in the highest category was 2.08 (95% CI 1.58–2.74, P for trend $< .0001$). Results from an analysis confined to women who had CES-D scores for 1999 and 2005 ($n = 20,477$) were almost identical to the main results (data not shown).

The authors assessed the joint effect of CES-D score (<16 vs 16) and use of antidepressant medication (yes vs no; Table 3). Twelve percent of participants had used an antidepressant during follow-up: 9% of those with a 1999 CES-D score lower than 16 and 20% of those with a CES-D score of at least 16. Compared with women with a CES-D score lower than 16 who did not take antidepressants (reference), the multivariable IRR for women with a CES-D score of at least 16 who used antidepressants was 2.77 (95% CI 2.05–3.75). The IRR also was significantly increased, although less so, for women with a CES-D score lower than 16 who used antidepressants and for women with a CES-D score of at least 16 who did not take antidepressants. There was no evidence of statistical interaction between antidepressant use and CES-D score (P for interaction = .61).

In analyses stratified by smoking (Table 4), the multivariable IRRs for the CES-D category of at least 33 vs lower than 16 were 2.73 (95% CI 1.85–4.04) in current and former smokers

and 1.68 (95% CI 1.12–2.50) in never smokers. The IRRs for the same comparison were 2.74 (95% CI 1.82–4.12) in women with a BMI lower than 30 kg/m² and 1.68 (95% CI 1.15–2.45) in obese women. IRRs were similar in normal-weight (BMI <25 kg/m²) and overweight (BMI 25–<30 kg/m²) women (data not shown). IRRs for the highest vs lowest CES-D categories were 2.35 (95% CI 1.71–3.22) in women at least 40 years old and 1.48 (95% CI 0.84–2.62) in younger women. IRRs were 1.98 (95% CI 1.28–3.05) in those who scored below the median on the coping scale (poorer coping skills) and 1.42 (95% CI 0.92–2.21) in those who scored at or above the median. There was no evidence of statistical interaction between CES-D score and any of the stratifying variables (Table 4).

In sensitivity analyses, a more stringent asthma case definition was used that included only cases who reported use of a preventive asthma medication and specified the medication name (eg, fluticasone; n = 521): the results were similar to the main results, with a multivariable IRR of 1.96 (95% CI 1.38–2.78) for a CES-D category of at least 33 vs lower than 16. In a second sensitivity analysis, the case definition was expanded to include all physician-diagnosed asthma regardless of medication use (n = 1,456): the IRR for the same comparison was 1.69 (95% CI 1.36–2.10). The authors also conducted a lagged analysis in which the 1999 CES-D score was the exposure and cases diagnosed during the first 4 years of follow-up were excluded. The IRRs for CES-D categories 16 to 22, 23 to 32, and at least 33 were 1.13 (95% CI 0.86–1.49), 1.06 (95% CI 0.75–1.51), and 1.73 (95% CI 1.16–2.60), respectively (*P* for trend < .0001).

Discussion

In this large follow-up study of African American women, there was a positive association between CES-D score and incidence of adult-onset asthma. The association was present in all strata of age, BMI, and smoking. The IRRs were highest in women who reported antidepressant use, which may indicate the presence of depression or of more severe depression.

The overall findings are consistent with positive results from 3 previous prospective studies of depressive symptoms and asthma.^{18–20} In the NHANES, depressive symptoms were assessed at baseline (NHANES I, 1982–1984) using a 4-item “cheerful versus depressed” scale and classified as low, medium, or high.¹⁹ Over an average of 9.4 years of follow-up, 181 of 5,231 adults reported new physician-diagnosed asthma. The multivariable relative risk for the high vs low depressive category was 2.08 (95% CI 1.14–3.79) in nonsmokers, but there was no association in smokers. The Canadian National Population Health Survey included 15,254 participants older than 12 years interviewed in 1994 and followed every 2 years until 2002.¹⁸ The Composite International Diagnostic Interview Short Form (2 screening questions followed by 7 questions for those who screened positive) was used to assess past-year major depressive episodes at every interview, and presence of major depression was modeled as varying by time. Over 8 years of follow-up, the hazard ratio for incident asthma in those reporting major depression was 1.7 (95% CI 1.2–2.4) after adjusting for sex, age, and health care use. During the first 2 years of follow-up, 283 cases occurred, but the total number of cases during the 8 years of follow-up was not given. In the most recent study,²⁰ 5,114 German adults 40 to 65 years old completed a baseline questionnaire in the early 1990s, including a 16-question depression scale, and newly diagnosed asthma was ascertained on a second questionnaire administered 8.5 years later. There was a nonsignificant 65% increase in the multivariable relative risk for the highest of 3 categories of depressive symptoms, based on 63 total cases. When analyzed as a continuous *z* score, the relative risk per 1-SD increment in the depressive symptom score was 1.24 (95% CI 1.02–1.50).

The present finding of a positive association in smokers and nonsmokers conflicts with the findings from the NHANES, in which a positive association was observed only in nonsmokers.¹⁹ The other 2 studies did not stratify by smoking status and none of the 3 stratified by BMI. The present observation of a stronger association between CES-D score and asthma in nonobese compared with obese women might reflect the lower background risk of asthma in nonobese women.²¹ A weak association in women with more coping skills may reflect the amelioration of stress effects by better coping strategies. Given that lung function decreases with age,³¹ the stronger association observed in older women may reflect a greater sensitivity of the aging lung to stress. However, none of the observed variations across strata were statistically significant, and apparent differences may be chance variation.

The present study was larger than the previous follow-up studies, providing excellent statistical power. The authors also controlled for some important potential confounding factors. The authors were not able to control for occupational or other environmental exposures that might be relevant, including indoor and outdoor air contaminants, and they did not have information on the presence of allergy or rhinitis. The ascertainment of depressive symptoms before asthma diagnosis precluded recall bias. Diagnostic bias might have contributed to the present findings if people with high levels of depressive symptoms are more likely to see a physician and then to be diagnosed with asthma than people with fewer depressive symptoms. The authors attempted to address this possibility by conducting a lagged analysis in which cases diagnosed in the first 4 years of follow-up were excluded. The IRRs were decreased by approximately 20%, compatible with some degree of detection bias. However, having symptoms is not the same as physician-diagnosed depression that would bring a person into the medical system; indeed, women in the highest CES-D category at baseline were less likely to report that they had a regular physician and health insurance (Table 1). Also, results were unchanged in an analysis using a stricter case definition that might have indicated the presence of more severe disease that would be diagnosed regardless of depressive symptoms.

The validity of the CES-D as a screening tool for detecting depressive symptoms has been demonstrated,^{23,24} as has the validity of this tool within the BWHS.²⁷ There is a high correlation between the CES-D and the Major Depression Inventory.²⁵

A limitation of the study was reliance on self-reported physician-diagnosed asthma. However, self-report is the standard method of identifying asthma cases in large national cohort studies^{32–36} because it is infeasible to examine all cases to verify asthma or to examine all noncases to identify undiagnosed asthma. In a small validation study of 43 women in the BWHS who met the present case definition and who gave the authors permission to contact their physicians, 39 (91%) were confirmed by their physicians as having asthma. In addition, overall results were consistent when a more restrictive case definition was used.

The authors hypothesized that stress might be a pathway linking depression and asthma. Psychological stress leads to neuroendocrine alterations of the hypothalamic–pituitary–adrenal axis and of the sympathetic and adrenomedullary systems, alterations that in turn appear to have immune system effects that could increase allergy and airway inflammation.¹⁰ Chronic stress is associated with increased basal cortisol concentration, which has been reported to affect antigen presentation³⁷ and bias the immune system toward T-helper type 2 cytokine responses.³⁸ In addition, central nervous system effects of stress may adversely affect autonomic nervous system control of airway smooth muscle tone, and stress also may lead directly to neurogenic airway inflammation through the effects of the nonadrenergic noncholinergic nervous system and tachykinins such as substance P.^{10,38} Although a stress or depression link is plausible, it is speculative for at least 2 reasons. First,

most of the data indicating a biological role for stress in asthma incidence pertain to prenatal and early-life stressors and their effect on asthma development in young children;¹ whether these mechanisms are relevant to stress experienced as adults and to adult-onset asthma is unknown. Second, whether depression causes significant chronic stress is not clear. Abnormalities in stress hormones (eg, elevated plasma cortisol levels) have been observed in some groups of patients with depression but are apparently not typical for all patients with depression.³⁹

In conclusion, the present results support the hypothesis that depression increases the risk of adult-onset asthma. Depression and asthma are substantial public health burdens on the US population, and the incidence and prevalence of the 2 conditions are higher in women than in men.^{40,41} If the hypothesis is correct, depression could contribute substantially to the burden of asthma in adults. Additional population-based research and laboratory and clinical studies explicating the mechanisms by which stress increases asthma risk might indicate subgroups at high risk for asthma and generate new therapies for asthma prevention.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding: This work was funded by grants from the National Heart, Lung, and Blood Institute (R01 HL107314) and the National Cancer Institute (R01 CA058420).

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Table 1

Age-adjusted^b characteristics of Black Women's Health Study participants by category of 1999 CES-D score in 1999 (unless otherwise noted)

	CES-D score in 1999 ^a			
	<16 (n = 23,212)	16–22 (n = 4,730)	23–32 (n = 2,745)	33 (n = 1,161)
Characteristic, mean (SD)				
Age (y) ^b	43.1 (10.7)	40.6 (9.7)	39.8 (9.2)	39.3 (8.8)
BMI (kg/m ²)	28.4 (6.5)	29.6 (7.0)	29.9 (7.3)	30.5 (8.0)
Pack-years of smoking in smokers	11.2 (12.0)	11.8 (13.0)	13.2 (13.1)	14.9 (13.4)
Characteristic, %				
Current/former smoker	32	38	40	43
Exposed to secondhand smoke (1997)				
Age 0–10 y	46	47	47	52
Age 11–20 y	45	47	49	52
Age 21–30 y	46	51	52	52
Age 31–40 y	36	41	43	46
12 y of education (1995)	14	19	21	25
Income \$50,000 (2003)	33	40	44	51
5 h/wk of vigorous exercise	9	7	6	5
Presence of sleep apnea (2001)	1	3	3	5
Female hormone supplement use	22	23	25	27
Had regular physician (1997)	82	80	79	77
Had health insurance (1997)	88	86	84	81
Parental history of asthma (1997)	6	7	7	7

Abbreviations: BMI, body mass index; CES-D, Center for Epidemiologic Studies–Depression Scale.

^a Pearson correlation coefficients for the correlation of the continuous CES-D score and continuous covariates and χ^2 associations for categorical variables and the 4-category CES-D score were significant at the $P < .001$ level with the exception of parental history of asthma ($P = .26$).

^b Age adjusted in 5-year age categories.

Table 2

CES-D score in relation to asthma incidence, Black Women's Health Study 1999 to 2011

CES-D score	Cases/person-years	Basic IRR ^a (95% CI)	Multivariable IRR ^b (95% CI)
<16	482/251,448	1.0	1.0
16–22	147/48,022	1.59 (1.32–1.91)	1.43 (1.19–1.72)
23–32	83/28,343	1.56 (1.24–1.97)	1.37 (1.08–1.73)
33	59/11,970	2.59 (1.98–3.40)	2.08 (1.58–2.74)
<i>P</i> for trend		<.0001	<.0001

Abbreviations: CES-D, Center for Epidemiologic Studies–Depression Scale; CI, confidence interval; IRR, incidence rate ratio.

^a Adjusted for age in 1-year increments and calendar time in 2-year increments.^b Also adjusted for body mass index (<25, 25–29, 30–34, 35–39, ≥40 kg/m²), female hormone use (never, <5 years, ≥5 years, unknown), presence of sleep apnea (yes, no, unknown), income (<\$25,000, \$25,001–\$50,000, \$50,001–\$100,000, >\$100,000, missing), and pack-years of smoking (never smoker, 1–4, 5–14, 15–24, ≥25, missing).

Table 3Joint CES-D score and use of antidepressants in relation to incidence of asthma, 1999 to 2011^a

Antidepressant use	CES-D score	Cases/person-years	Basic IRR ^b (95% CI)	Multivariable IRR ^c (95% CI)
No	<16	456/244,169	1.0	1.0
	16	240/81,071	1.59 (1.36–1.87)	1.42 (1.21–1.66)
Yes	<16	26/7,279	1.97 (1.33–2.93)	1.73 (1.16–2.58)
	16	49/7,264	3.54 (2.64–4.76)	2.77 (2.05–3.75)

Abbreviations: CES-D, Center for Epidemiologic Studies–Depression Scale; CI, confidence interval; IRR, incidence rate ratio.

^a CES-D score updated in 2005 and antidepressant use updated every questionnaire cycle.^b Adjusted for age in 1-year increments and calendar time in 2-year increments.^c Also adjusted for body mass index (<25, 25–29, 30–34, 35–39, 40 kg/m², missing), female hormone use (never, <5 years, 5 years, unknown), presence of sleep apnea (yes, no, unknown), income (<\$25,000, \$25,001–\$50,000, \$50,001–\$100,000, >\$100,000, missing), and pack-years of smoking (never smoker, 1–4, 5–14, 15–24, 25, missing).

Table 4

CES-D score in relation to asthma incidence by strata of smoking, BMI, age, and coping score, Black Women's Health Study 1999 to 2011

CES-D score	Cases/person-years	Multivariable IRR ^a (95% CI)	P for interaction
Never smokers			
<16	301/168,424	1.0	.10
16–22	73/30,716	1.19 (0.92–1.54)	
23–32	47/18,067	1.29 (0.95–1.76)	
33	27/7,386	1.68 (1.12–2.50)	
P for trend		.0036	
Current and former smokers			
<16	180/82,802	1.0	
16–22	74/17,246	1.83 (1.39–2.41)	
23–32	36/10,246	1.50 (1.04–2.16)	
33	32/4,578	2.73 (1.85–4.04)	
P for trend		<.0001	
BMI <30 kg/m ²			
<16	235/158,631	1.0	.22
16–22	53/27,053	1.25 (0.93–1.69)	
23–32	38/15,476	1.55 (1.09–2.19)	
33	27/6,158	2.74 (1.82–4.12)	
P for trend		<.0001	
BMI ≥30 kg/m ²			
<16	247/92,816	1.0	
16–22	94/20,970	1.55 (1.22–1.97)	
23–32	45/12,867	1.26 (0.91–1.74)	
33	32/5,812	1.68 (1.15–2.45)	
P for trend		.0008	
Age <40 y			
<16	116/65,249	1.0	.25
16–22	39/15,755	1.23 (0.85–1.78)	
23–32	21/9,636	1.06 (0.66–1.69)	
33	14/4,288	1.48 (0.84–2.62)	
P for trend		.2087	
Age ≥40 y			
<16	366/186,199	1.0	
16–22	108/32,268	1.51 (1.21–1.87)	
23–32	62/18,707	1.48 (1.13–1.95)	
33	45/7,683	2.35 (1.71–3.22)	
P for trend		<.0001	
Coping score lower than median (fewer coping skills) ^b			
<16	46/38,128	1.0	.19

CES-D score	Cases/person-years	Multivariable IRR ^a (95% CI)	P for interaction
16	43/15,784	1.98 (1.28–3.05)	
Coping score at least median ^b			
<16	70/50,062	1.0	
16	30/12,829	1.42 (0.92–2.21)	

Abbreviations: BMI, body mass index; CES-D, Center for Epidemiologic Studies– Depression Scale; CI, confidence interval; IRR, incidence rate ratio.

^a Adjusted for age, calendar time, body mass index (<25, 25–29, 30–34, 35–39, 40 kg/m², missing), female hormone use (never, <5 years, 5 years, unknown), presence of sleep apnea (yes, no, unknown), income (< \$25,000, \$25,001–\$50,000, \$50,001–\$100,000, >\$100,000, missing), and pack-years of smoking (never smoker, 1–4, 5–14, 15–24, 25, missing).

^b Follow-up 2005 to 2011.