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Co-Occurring Obesity and Smoking Among U.S. Women of Reproductive Age: Associations with Educational Attainment and Health Biomarkers and Outcomes

Drina Vurbic, PhD^{1,2,3}, Valerie S. Harder, PhD^{2,3,4}, Ryan R. Redner, PhD^{1,2}, Alexa A. Lopez, PhD^{1,2,3}, Julie K. Phillips, MD^{1,5}, and Stephen T. Higgins, PhD^{1,2,3}

¹Vermont Center on Behavior and Health, University of Vermont

²Department of Psychiatry, University of Vermont

³Department of Psychology, University of Vermont

⁴Department of Pediatrics, University of Vermont

⁵Department of Obstetrics, Gynecology and Reproductive Sciences, University of Vermont

Abstract

Introduction—Obesity and smoking are independently associated with socioeconomic disadvantage and adverse health effects in women of reproductive age and their children, but little is known about co-occurring obesity and smoking. The purpose of this study was to investigate relationships between co-occurring obesity and smoking, socioeconomic status, and health biomarkers and outcomes in a nationally representative sample.

Methods—Data from non-pregnant women of reproductive age were obtained from the U.S. National Health and Nutrition Examination Surveys reported between 2007-2010. Linear and logistic regressions were used to examine associations between obesity and smoking alone and in combination with educational attainment and a range of health biomarkers and outcomes.

Results—Prevalence of co-occurring obesity and smoking was 8.1% (~4.1 million U.S. women of reproductive age) and increased as an inverse function of educational attainment, with the least educated women being 11.6 times more likely to be obese smokers than the most educated. Compared to women with neither condition, obese smokers had significantly poorer cardiovascular and glycemic biomarker profiles, and higher rates of menstrual irregularity, hysterectomy, oophorectomy, physical limitations, and depression. Obese smokers also had significantly worse high-density lipoprotein (HDL) cholesterol levels, physical mobility, and depression scores than those with obesity or smoking alone.

Correspondent: Stephen T. Higgins, Vermont Center on Behavior and Health, University of Vermont, UHC Campus, Rm. 3100B Old Hall, 1 S. Prospect St, Burlington, VT 05401, Phone: 802-656-9614, Stephen.Higgins@uvm.edu.

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COMPETING INTERESTS

The authors have no conflicts of interest to report.

Conclusions—Co-occurring obesity and smoking is highly associated with low educational attainment, a marker of socioeconomic disadvantage, and a broad range of adverse health biomarkers and outcomes. Interventions specifically targeting co-occurring obesity and smoking are likely necessary in efforts to reduce health disparities among disadvantaged women and their children.

Keywords

cigarette smoking; obesity; co-occurring smoking-obesity; body mass index; women's health; cardiovascular disease; diabetes; reproductive health; physical mobility; depression; education

Obesity and smoking are the two leading causes of preventable morbidity and mortality in U.S. and many other industrialized countries and independently linked to many of the same adverse outcomes in women, including reproductive health problems, heart disease, type-2 diabetes, cancer, impaired physical mobility, and depression (Abunassar et al., 2012; Hennekens & Andreotti, 2013; Jordan et al., 2006; LaCroix et al., 1993; Leitzmann et al., 2009; Østbye et al., 2002). Obesity and smoking are each overrepresented among socioeconomically disadvantaged women (CDC, 2011; Chilcoat, 2009; Wadden et al., 2002) and likely contributors to the unsettling trends towards increasing inverse associations between educational attainment and mortality risk among U.S. women (Montez & Zajacova, 2013).

While each condition is strongly associated with serious adverse health outcomes in women, relatively few studies have been reported on their co-occurrence. We know of only one prior study estimating prevalence of co-occurrence in a nationally representative sample of U.S. adult women in 2002, with prevalence estimated at 4.2% (Healton et al., 2006). Prevalence rates in women of reproductive age were not reported nor were relationships to health examined in that study. The few studies that are available examining associations with health suggest that co-occurring obesity and smoking poses greater risks than either alone, but much remains to be learned (Akbartabartoori et al., 2006). For example, answers to such basic questions as whether combined risks are additive or synergistic remain unclear. The only study we are aware of examining risks of co-occurring obesity and smoking on reproductive health was one that we reported examining breastfeeding rates in a clinical sample (Vurbic et al., 2013). The probability of breastfeeding varied in a graded additive manner, being lowest among those with co-occurring obesity and smoking, intermediate among those with obesity or smoking alone, and greatest among non-obese non-smokers (Vurbic et al., 2013). Considering the current U.S. and global obesity epidemic and that cigarette smoking prevalence in the U.S. is decreasing at a slower rate among women than men, these are not minor gaps.

The purpose of the present study was to begin learning more about the potential impact of co-occurring obesity and smoking on women's health. The present study used data from the National Health and Nutrition Examination Surveys (NHANES) to investigate U.S. prevalence rates, and associations with socioeconomic status, health biomarkers, and health outcomes associated with co-occurring obesity and smoking in women of reproductive age. We focused on educational attainment as a socioeconomic predictor because it is strongly

linked to disparities in women's health (Higgins & Chilcoat, 2009; Meara et al., 2008). We examined associations between co-occurring obesity and smoking with cardiovascular and glycemic biomarkers to extend to women of reproductive age what has been observed on these health indicators in other populations (Akbarabartoori et al., 2006). We focused on gynecological/reproductive health outcomes, physical functioning, and depression because they are strongly associated with obesity and smoking alone, the impact of co-occurring obesity and smoking on these outcomes has not been previously reported, and these outcomes are of considerable importance to the health, economic, and social stability of women and their children.

METHOD

Data Source and Study Sample

This study was conducted using data from the National Health and Nutrition Examination Survey (NHANES). NHANES is a continuous, nationally representative health survey of the U.S. civilian non-institutionalized population that uses a stratified multistage probability design and is reported in two-year cycles. We selected non-pregnant women between 25-49 years for whom there were data for lab-measured height and weight, and self-reported smoking status across two survey cycles, 2007–2008 and 2009–2010 to increase statistical power. The higher-than-usual lower age limit for women of reproductive age was chosen because setting the limit at age 25 increases the likelihood that women will have completed their education. We used a slightly higher-than-usual upper limit (49 vs. 44 years) to assure adequate sample sizes given the higher-than-usual lower limit. Educational attainment was selected to represent socioeconomic status because of its robust association with a wide range of health-related risk factors and outcomes (Cutler & Lleras-Muney, 2010). Strong associations between educational attainment and smoking in women have been widely reported (e.g., Higgins & Chilcoat, 2009), and associations with obesity in women have been reported as well including in previous reports using the NHANES (Ogden et al., 2010).

Each survey included a household interview and clinical examination. Demographic data and information regarding pregnancy and smoking status were collected during the former. Participants were classified as current smokers if they reported smoking 100 cigarettes lifetime and in past 30 days. Height and weight were obtained during the physical examination. Obesity was defined as BMI (kg/m^2) ≥ 30 .

Blood specimens were analyzed for cardiovascular (high-density lipoprotein [HDL] cholesterol, low-density lipoprotein [LDL] cholesterol, and triglyceride) and glycemic (blood glycohemoglobin [A1C], fasting plasma glucose, 2-h glucose [oral glucose tolerance test], and serum insulin) biomarkers (see CDC, 2010). HDL cholesterol and glycohemoglobin levels, which do not require fasting, were available for the full sample while biomarkers that require fasting (e.g., triglycerides) were only collected in a randomly selected subset of participants.

Gynecological/reproductive health, physical functioning, and depression were assessed by questionnaires administered to the full sample. Gynecological/reproductive function was assessed by three items assessing whether women had (a) at least one menstrual period in

the past 12 months (not including bleeding caused by medical conditions, hormone therapy, or surgery), (b) a hysterectomy (including partial removal of the uterus) or (c) bilateral oophorectomy (removal of both ovaries). Women who reported absence of a menstrual period related to pregnancy or breastfeeding were excluded from analyses of that item. Physical functioning was assessed using the validated and widely used physical functioning questionnaire (e.g., Vasquez et al., 2014), focusing on two items related to economic independence. The items inquired whether, as a result of a chronic health condition, respondents were limited in the work they were able to do or required use of special healthcare equipment. For depression, the validated Patient Health Questionnaire (PHQ-9; Martin et al., 2006) was used, which includes nine questions about frequency of depressive symptoms over the previous two weeks. Responses on each item correspond to a score ranging from 0 ("Not at all") to 3 ("Nearly every day"), which are added together to obtain a final score ranging from 0 to 27 and corresponding to mild (5-9), moderate (10-14), moderately severe (15-19), and severe depression (20-27).

Statistical Methods

Simple statistical tests (t-tests and chi-squared) were used as a first look to compare socio-demographic characteristics across obese smokers, non-obese non-smokers, obese only, and smokers only. Associations between educational attainment (college graduates as the reference group) and being an obese smoker, smoker only, or obese only, each compared to all others, were tested using logistic regression. Linear regression was conducted to estimate average differences in continuous outcomes comparing each pairing of the indicator variables for obesity and smoking status. Similarly, logistic regression was conducted to estimate odds of binary outcomes comparing each pairing of indicator variables for obesity and smoking status. Looking into a possible interaction between smoking and obesity, linear and logistic regression models were tested with main effects of smoking and obesity with a smoking by obesity interaction term. All regressions controlled for the potential confounding effects of age, education, race, and marital status.

Statistical software STATA version 12.1 was used for all analyses. The complex sampling design was taken into account by using the survey commands in STATA. NHANES provided weighting, stratum, and probability sampling unit variables that took into account unequal probabilities of selection resulting from the sample design, nonresponse, and planned oversampling of certain subgroups. Standard errors were computed using the jackknife repeated replication method, and weighted means, proportions, coefficients, odds ratios (OR), and 95% confidence intervals (CI) were reported. When comparing obese smokers vs. smokers only on two cholesterol (LDL cholesterol, triglyceride) and three glycemic (glycohemoglobin, fasting glucose, 2-hour fasting glucose) biomarkers, only the weighting and probability sampling unit designations were used. The stratum designation was not used due to only having one probability-sampling unit (minimum of two required) in one of the 31 strata. The unweighted sample sizes in these four models were all less than 400 individuals, resulting in the occurrence of a single probability-sampling unit when subjects were stratified into 31 groups. Excluding the stratum designation from our weighting procedure results in larger standard error, larger CIs, and, therefore, more conservative weighted effect estimates. Statistical significance was $p < .05$ in all analyses.

RESULTS

Participant Characteristics

Participants were 2,477 non-pregnant women between the ages of 25-49 years, with 201 women in the obese smokers category, 406 in smokers only (smokers who are not obese), 644 in obese only (obese non-smokers), and 1,226 in the neither obese or smoker category. Among our weighted sample, 8.1% were obese smokers, representing 4,139,065 women in the US population, 16.4% current smokers only, representing 8,323,895 women, 26.0% were obese only, representing 13,195,178 women, and the remaining 49.5% were neither obese nor smokers, representing 25,190,332 women, for a total weighted population of 50,848,470. Overall prevalence of obesity in this study sample was 34.1% (obese smokers + obese only) and current smoking was 24.5% (obese smokers + smokers only).

Unadjusted comparisons of socio-demographic characteristics shown in Table 1. Weighted mean BMI was 24.2 ± 0.12 (jackknife standard error) for non-obese and 36.9 ± 0.22 for obese women. Among smokers, weighted average number of cigarettes/day in the past 30 days was 12.4 ± 0.49 . Obese smokers did not differ in BMI compared to obese only or smoking rates compared to smoker only.

Prevalence of Co-Occurring Obesity and Smoking by Educational Attainment

An a priori study goal was to investigate associations of co-occurring obesity and smoking with educational attainment as a marker socioeconomic status. Educational attainment had a linear, inverse relationship with co-occurring smoking and obesity (Figure). Compared to college-educated women, odds of being an obese smoker was 11.59 (CI = 5.43 – 24.73, $p < .0005$) times greater for those with less than a high school diploma or GED, 8.54 (CI = 4.00 – 18.20, $p < .0005$) times greater for those with a high school diploma, and 5.36 (CI = 2.58 – 11.12, $p < .0005$) times greater for those with some college or an associate's degree. Similar relationships between educational attainment and being obese only or a smoker only were observed as well, although the functions were less graded especially obese only. Compared to college graduates, odds of being obese only was 1.55 (CI = 1.17 – 2.05, $p = .003$) times greater for those with less than a high school diploma or GED, 1.37 (CI = 1.00 – 1.87, $p = .047$) times greater for those with a high school diploma, and 1.87 (CI = 1.32 – 2.66, $p = .001$) times greater for those with some college or an associate's degree. Compared to college graduates, odds of being a smoker only was 3.43 (CI = 2.07 – 5.65, $p < .001$) times greater for those with less than a high school diploma or GED, 3.82 (CI = 2.52 – 5.77, $p < .001$) times greater for those with a high school diploma, and 2.25 (CI = 1.52 – 3.32, $p < .001$) times greater for those with some college or an associate's degree.

Associations with Health Biomarkers and Outcomes

Investigating associations with a broad range of health biomarkers and outcomes was the other a priori study goal. As is detailed below, obese smokers had poorer biomarker levels and health outcomes compared to those with neither condition as well as a pattern of elevated risk compared to those with obesity or smoking alone.

Cardiovascular biomarkers—Obesity and smoking were associated with lower mean HDL cholesterol levels (mg/dL) when present alone and in combination compared to non-obese non-smokers (Table 2). Lowest levels were seen among obese smokers (Coefficient [Coeff]: -15.71 , $p < .0005$, 95% CI: $-18.36 - -13.05$), followed by obese-only (Coeff: -11.30 , $p < .0005$, 95% CI: $-12.76 - -9.84$), and then smokers-only (Coeff: -6.25 , $p < .0005$, 95% CI: $-9.27 - -3.22$). Obese smokers also had lower levels of HDL than smokers-only (Coeff: -11.20 , $p < .0005$, 95% CI: $-14.48 - -7.92$) and obese-only (Coeff: -4.62 , $p < .0005$, 95% CI: $-6.80 - -2.45$), and obese-only had lower levels than smokers-only (Coeff: -5.67 , $p < .0005$, 95% CI: $-8.28 - -3.06$).

LDL cholesterol levels (mg/dL) were elevated among obese women, with the largest differences seen between obese smokers compared to non-obese non-smokers (Coeff: 12.94 , $p = .006$, 95% CI: $4.03 - 21.84$) and obese smokers compared to smokers-only (Coeff: 13.30 , $p = .016$, 95% CI: $6.00 - 20.60$). LDL cholesterol levels were also elevated among obese-only compared to non-obese non-smokers (Coeff: 6.70 , $p = .035$, 95% CI: $0.51 - 12.90$). There were no other significant differences.

Triglyceride levels (mg/dL) also followed a similar pattern (Table 2), with obese smokers having the largest average difference from non-obese non-smokers (Coeff: 60.73 , $p < .0005$, 95% CI: $37.85 - 83.60$), followed by obese-only (Coeff: 42.83 , $p < .0005$, 95% CI: $33.29 - 52.37$), and then smokers-only (Coeff: 28.67 , $p < .0005$, 95% CI: $15.46 - 41.88$). Results for obese smokers compared to smokers only were also significant (Coeff: 37.66 , $p = .001$, 95% CI: $33.63 - 41.69$). There were no other significant differences.

Glycemic biomarkers—Obesity, smoking, and their co-occurrence were significantly associated with differences in glycemic biomarkers compared to non-obese non-smokers, but differences were attributable to obesity (Table 2). Compared to non-obese non-smokers, comparable elevations in mean glycohemoglobin levels (%) were seen among obese smokers (Coeff: $.39$, $p < .0005$, 95% CI: $.26 - .52$) and obese-only (Coeff: $.39$, $p < .0005$, 95% CI: $.29 - .50$), and smaller elevations among smokers-only (Coeff: $.09$, $p = .003$, 95% CI: $.04 - .15$). Glycohemoglobin levels were also higher among obese smokers vs. smokers-only (Coeff: $.31$, $p < .0005$, 95% CI: $.19 - .42$) and obese-only vs. smokers-only (Coeff: $.31$, $p < .0005$, 95% CI: $.19 - .42$). There were no other significant differences.

Comparable elevations in mean fasting glucose (mg/dL) levels were seen in obese smokers (Coeff: 9.02 , $p < .0005$, 95% CI: $4.59 - 13.46$) and obese-only (Coeff: 11.30 , $p < .0005$, 95% CI: $7.65 - 14.96$) compared to non-obese non-smokers, and obese-only was associated with higher mean fasting glucose levels compared to smokers-only (Coeff: 12.58 , $p < .0005$, 95% CI: $7.48 - 17.67$). There were no other significant differences.

Similarly, 2-hour fasting glucose (mg/dL) was on average 14.75 units higher ($p < .003$, 95% CI: $5.31 - 24.19$) among obese smokers, and 23.88 units higher ($p < .0005$, 95% CI: $16.23 - 31.52$) among obese-only compared to non-obese nonsmokers. Two-hour fasting glucose was also on average higher among obese smokers compared to smoker-only (Coeff: 21.07 , $p = .003$, 95% CI: $15.83 - 26.30$). There were no other significant differences.

Finally, insulin (uU/mL) levels were elevated among obese smokers (Coeff: 9.26, $p < .0005$, 95% CI: 5.99 – 12.52) and obese-only (Coeff: 9.70, $p < .0005$, 95% CI: 8.01 – 11.37) compared to non-obese nonsmokers. Insulin levels also differed when comparing obese smokers to smoker-only (Coeff: 8.94, $p = .005$, 95% CI = 6.20 – 11.67) and when comparing obese-only to smokers-only (Coeff: 9.84, $p < .0005$, 95% CI: 7.80 – 11.87). There were no other significant differences.

Reproductive health—Obese smokers on average showed the largest elevations in the proportion of women with reproductive problems compared to non-obese nonsmokers, followed by obese-only and then smokers-only, although obese smokers did not differ significantly from the obese-only or smokers-only groups nor did the latter two groups differ significantly from each other (Table 3).

Compared to non-obese nonsmokers, the proportion of women with menstrual irregularity was significantly elevated only in obese smokers (Odds Ratio (OR) = 1.76, $p = .038$, 95% CI: 1.03 – 2.99). No other comparisons yielded significant differences.

Compared to non-obese nonsmokers, the proportion of women with hysterectomies was 2.46 times greater among obese smokers ($p = .008$, 95% CI: 1.28 – 4.73) and 1.67 times greater among obese-only ($p = .028$, 95% CI: 1.06 – 2.63). No other comparisons were significantly different.

Compared to non-obese nonsmokers, the proportion of women with oophorectomies was significantly elevated in obese smokers (OR = 3.78, $p < .001$, 95% CI: 1.86 – 7.64), obese-only (OR = 2.61, $p = .003$, 95% CI: 1.44 – 4.74), and smokers-only (OR = 2.73, $p = .013$, 95% CI: 1.25 – 5.94). No other comparisons were significantly different.

Physical functioning—The proportion of women reporting work limitations was elevated in obese smokers, obese-only, and smokers-only compared to non-obese nonsmokers (Table 4), with obese smokers showing the greatest odds of limitations (OR = 3.71, $p < .0005$, 95% CI: 1.93 – 7.14), followed by smokers-only (OR = 2.56, $p < .001$, 95% CI: 1.48 – 4.42), and then obese-only (OR = 2.00, $p = .015$, 95% CI: 1.16 – 3.45). Odds of work limitations were also 1.79 times greater in obese smokers compared to obese-only ($p < .032$, 95% CI: 1.06 – 3.04). Other differences were not significant.

Compared to non-obese nonsmokers, a larger proportion of obese smokers also reported needing special healthcare equipment (OR = 12.68, $p < .0005$, 95% CI: 3.94 – 40.82), followed by obese-only (OR = 5.32, $p = .001$, 95% CI: 2.00 – 14.13), and then smokers-only (OR = 4.05, $p = .011$, 95% CI: 1.41 – 11.61). A larger proportion of obese smokers also reported needing special equipment than smokers-only (OR = 2.87, $p = .037$, CI: 1.07 – 7.68). No other differences were significant.

Depression scores—Compared to non-obese nonsmokers, average depression scores were highest among obese smokers (Coeff: 3.13, $p < .0005$, 95% CI: 2.18 – 4.08), smokers-only (Coeff: 1.62, $p = .001$, 95% CI: 0.75 – 2.49), and obese-only (Coeff: 0.95, $p < .0005$, 95% CI: 0.46 – 1.44) (Table 5). Obese smokers also had mean depression scores that were

significantly higher than obese-only (Coeff: 2.20, $p < .0005$, 95% CI: 1.23 - 3.16), and smokers-only (Coeff: 1.42; $p = .023$, 95% CI: 0.21 – 2.63). The comparison between smokers-only and obese-only was not significant.

Smoking x obese interaction—There were no interaction effects of smoking and obesity in any models ($ps > .05$). That is, the associations between smoking and outcomes did not differ among obese and non-obese strata, and the associations between obesity and outcomes did not differ among smokers and non-smokers strata.

DISCUSSION

The present study extends the relatively small literature on co-occurring obesity and smoking in other populations to a nationally representative sample of U.S. women of reproductive age. This condition impacts approximately 8.1% of U.S. women of reproductive age (~4.1 million) and is overrepresented among socioeconomically disadvantaged women. This socioeconomic disparity is large, with the least educated women being 11.6 times more likely to be obese smokers than the most educated, and an almost certain contributor to the problems of health disparities considering the wide-ranging associations with adverse health biomarkers and outcomes observed. The flatter functions relating educational attainment to the odds of obesity alone versus smoking alone or co-occurring obesity and smoking likely reflect the more recent onset of the obesity epidemic compared to cigarette smoking and associated greater diffusion of knowledge about the health risks associated with smoking into the U.S. general population. As a general rule of thumb, education gradients in the odds of health-related risk behaviors and associated outcomes become increasingly steep as knowledge regarding the potential risks involved are diffused into the general population (Link & Phelan, 1995; 2009).

The adverse biomarkers and outcomes noted among those with co-occurring obesity and smoking generally trended towards exceeding levels seen with obesity or smoking alone, with significant differences noted for at least one of the cardiovascular biomarkers (HDL cholesterol—the one biomarker assessed in the entire sample), and two measures of physical limitations and depression scores (also assessed in the entire sample). In these cases the elevations associated with co-occurrence compared to either risk factor alone were additive or slightly less than additive. We saw no evidence of synergistic increases in risk.

The 8.1% prevalence rate of co-occurring obesity and smoking among U.S. women of childbearing age is almost twice the 4.2% rate reported in 2002 across U.S. adult women 18 years of age (Healton et al., 2006), likely influenced in part by the different age ranges examined but also increases over time in prevalence of obesity among more disadvantaged women who are also more likely to smoke (Robinson et al., 2014). The results complement those of previous studies that have underscored the combined adverse effects of co-occurring obesity and smoking on cardiovascular biomarkers (Akbarabartoori et al., 2006) and breastfeeding (Vurbic et al., 2013), and contribute new knowledge on co-occurring obesity and smoking by characterizing the relationship with socioeconomic disadvantage among women of reproductive age and demonstrating previously unreported associations with gynecological/reproductive health outcomes, physical functioning, and depression.

Interestingly, there was no evidence suggesting a combined effect on glycemic control in the present study. Both obese smokers and obese-only groups had similarly elevated levels on of the glycemic measures compared to non-obese groups. The fact that smoking added no discernible increase in risk across the glycemic measures when co-occurring with obesity was unexpected considering that smoking alone was associated with a small but significant elevation in these measures and other studies have shown a dose-response relationship between smoking and risk for diabetes (Willi et al., 2007). Worth considering is that the age range in the present study was restricted to women who were relatively young (25-49), we controlled for the potentially confounding influence of sociodemographic characteristics (e.g., educational attainment) not always controlled for in prior studies, and the level of smoking was not particularly high (40% of smokers reported smoking < 10 cigarettes/day).

Regarding measures of reproductive health, obesity and smoking each independently affected menstrual function. Both conditions favor a hormonal environment of excess androgens and hyperinsulinemia causing chronic anovulation, oligo- or amenorrhea, or even menorrhagia and anemia (ASRM, 2008; Grossman & Nakajima, 2006). While the rate of menstrual irregularity, hysterectomy, and oophorectomy with co-occurring obesity and smoking did not reach significance compared to either condition alone in the present study, the pattern was in that direction. The present study is limited in the gynecologic outcomes reported, and in that indication for hysterectomy or oophorectomy was unavailable, but further investigation appears warranted considering the serious potential consequences of these outcomes. Menorrhagia and anemia may lead to missed days of work, lower productivity, and need for iron replacement therapy or even blood transfusion, while oligo- or amenorrhea in reproductive aged women affects fertility, leading to an increase in assisted-reproductive therapies. Those undergoing hysterectomy or oophorectomy no longer have child-bearing potential, raising concerns about emotional impacts (Darwish et al., 2014; Wang et al., 2014). Oophorectomy induces surgical menopause, which is associated with menopausal symptoms, often need for hormone-replacement therapy, and an increased risk of cardiovascular disease (Colditz et al., 1987). Finally, although cancer is rare in this age group, obesity is a risk factor for both endometrial and ovarian cancer (Olsen et al., 2007; Cramer, 2012). Smoking has varied effects on cancer risk, actually providing protection against endometrial cancer, with the effect on ovarian cancer being indeterminate, especially in reproductive-aged women (Jordan, 2006; Hunn & Rodriguez, 2012). Further examination into the effects of co-occurring obesity and smoking on reproductive tract cancer risk could provide important information.

Considering the relatively young age of the women studied, it is disconcerting that 22% of those with co-occurring obesity and smoking reported physical limitations in ability to work, more than a 4-fold increase above levels seen among those with neither condition and 2.3- and 1.5-fold increases above levels among those with obesity or smoking only, respectively. Prior studies have reported increases in disability in middle-aged (Østbye et al., 2002) and elderly (LaCroix et al., 1993) populations associated with obesity and smoking alone. The present results demonstrate the same in this younger population and still further increases with co-occurring obesity and smoking.

Depression is among the world's leading causes of disability in women and thus is a critically important outcome to examine when considering the health impact of co-occurring obesity and smoking in women (e.g., Arterburn et al., 2012; Murray & Lopez, 1996). While mean depression scores among those with co-occurring obesity and smoking in the present study were in the mild range, they nevertheless exceeded scores seen among those with obesity or smoking alone, two well established independent risk factors for depression (e.g., Arterburn et al., 2012; Farr et al., 2011). Moreover, even mild depression in women of reproductive age is associated with significant increases in risk for chronic disease (Farr et al., 2011), adverse birth outcomes in pregnant women (Grote et al., 2010), and psychopathology among their children (Goodman et al., 2011).

There are several limitations to this study. First, the data are from an observational cross-sectional study and thus cannot support causal inferences. That point notwithstanding, to our knowledge this is only the second study reported on co-occurring obesity and smoking in women of reproductive age and the results provided offer an empirical basis for formulating future experimental studies that are capable of supporting causal inferences (e.g., effects of smoking cessation alone, weight reduction alone, and combined smoking cessation and weight reduction on biomarker levels). Second, our adjustment in the upper and lower age brackets of reproductive age may limit direct comparisons of estimates to those reported in studies using more conventional brackets. As mentioned in the Methods section, we chose an upper age limit of 49 years to increase the statistical power of the study given the use of a higher than usual lower age limit. This differs from most studies of reproductive aged women, which use 44 years as the upper limit. Although the average age of menopause in US women is 51 years, menopausal transition with irregular menstrual cycles begins on average at age 47. Thus, by including women in menopause transition, rates of reproductive outcomes may be influenced. However, the average age of women was similar across the subgroups examined, therefore we see no reason why outcomes would vary across groups simply by increasing the upper limit of the age included. Moreover, when we reanalyzed results excluding women in this older age bracket, it had minimal influence on estimates of the magnitude of the associations and in no instance changed their direction. Third, we excluded pregnant women from this study, a subgroup in whom obesity and smoking alone are known to increase risk for pregnancy complications and adverse birth outcomes. Extending this line of research to that especially vulnerable population represents an important future research direction. Fourth, the weighted participant characteristics of certain subgroups (e.g., college graduate obese-smokers) were based on relatively small samples, which seems unlikely to alter the nature of the relationships reported, but may change estimates of absolute levels. Fifth, we did not examine how heaviness (cigs day) or duration (pack years) of smoking or type (visceral vs. subcutaneous) or severity (class I, II, III) of obesity might factor into the observed associations, which are important topics for future investigation. Lastly, we examined only one marker of socioeconomic status (educational attainment) in the present study. How other socioeconomic markers (e.g., income, occupation) relate to prevalence of co-occurring obesity and smoking warrants investigation.

Overall, the health patterns reported here are concerning on several levels. First, they demonstrate that obese women of reproductive age who smoke are vulnerable to a broader

range of adverse health biomarkers and outcomes than those with neither problem, and in a number of instances also those with either problem alone. Considering the staggering costs associated with either obesity or smoking alone, their increasing rate of co-occurrence has the potential to add further strain on what are already unmanageable individual and societal health care costs. The individual-level impact is especially concerning considering that the present study also demonstrates that this burden is disproportionately borne by those with fewer resources. Second, little research has been done to examine which intervention strategies might yield the greatest improvements in health for this population. Although there is some evidence to suggest that health risks faced by obese smokers may be mitigated by removing one condition (e.g., smoking cessation), risks may remain elevated beyond the level associated with the other condition (Freedman et al., 2006). Further reductions in risk may thus require that both conditions be effectively treated through a combination of smoking cessation and weight loss. Unfortunately, we know of few studies systematically exploring this possibility. In addition to treatments, devising effective prevention interventions simultaneously targeting smoking and weight gain in disadvantaged girls should be a priority as well. Studies investigating pathways linking socioeconomic status, risk for co-occurring obesity and smoking, and adverse health outcomes and markers may inform such treatment and prevention efforts.

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Highlights

Strong associations between education and co-occurring obesity and smoking in women.

Co-occurring obesity and smoking and concerning health status in women.

Combined weight management and smoking cessation interventions may be needed.

Figure.

Weighted percentages of women who were obese smokers, smokers only, or obese only by level of educational attainment (< high school diploma, high school diploma/ GED, some college / associates degree, bachelor's degree). Smokers reported smoking > 100 cigarettes lifetime and currently smoking; obesity defined as BMI (kg/m^2) ≥ 30 .

Table 1

Weighted demographic characteristics of non-pregnant women age 25-49 years in the 2007-08 and 2008-2010 National Health and Nutrition Examination Surveys.

	Non-obese Non-smoker	Obese Only	Smoker Only	Obese + Smoker
Age (yrs)	37.3 (0.19) ^a	38.0 (0.40) ^a	37.2 (0.11) ^a	37.6 (0.08) ^a
High school graduate or lower education (%)	27.4 ^a	38.1 ^b	54.0 ^c	63.3 ^c
Caucasian (%)	65.4 ^a	54.2 ^b	77.0 ^c	62.9 ^{a,b}
Married (%)	67.0 ^a	60.0 ^b	42.0 ^c	36.3 ^c
Cigarettes/day	-	-	14.2 (0.10) ^a	13.7 (0.23) ^a
Body Mass Index	24.2 (0.14) ^a	37.0 (0.27) ^b	24.1 (0.37) ^a	36.5 (0.10) ^b

Note: Numbers in parentheses are jackknife standard errors.

Values that do not share a superscript letter differ from each other at $p < .05$ in unadjusted comparisons.

Table 2

Weighted mean levels of cardiovascular and glycemic biomarkers

	Non-obese Non-smoker	Obese only	Smoker only	Obese + Smoker
HDL cholesterol	62.56±0.44 ^a	50.62±0.6 ^b	55.57±0.96 ^c	45.06±0.93 ^d
LDL cholesterol	107.36±1.53 ^a	116.06±2.44 ^{b,c}	115.41±3.22 ^{a,b}	124.50±3.48 ^c
Triglycerides	83.52±1.65 ^a	128.13±4.84 ^{b,c}	118.06±8.79 ^b	148.75±7.57 ^c
Glycohemoglobin (A1C)	5.26±0.02 ^a	5.73±0.05 ^{b,d}	5.36±0.04 ^c	5.69±0.09 ^d
Fasting glucose	93.32±0.61 ^a	105.66±1.75 ^{b,c}	94.16±1.23 ^a	104.28±2.71 ^{b,c}
2-h glucose tolerance test	101.13±1.78 ^a	127.09±2.93 ^{b,c}	100.93±2.80 ^a	122.76±4.79 ^{b,c}
Insulin	8.53±0.41 ^a	18.42±0.66 ^{b,c}	9.19±0.76 ^a	18.96±1.26 ^{b,c}

Note: Values represent weighted means for HDL and LDL cholesterol (mg/dL), triglycerides (mg/dL), glycohemoglobin (%), fasting glucose (mg/dL), two-hour glucose (mg/dL), and insulin (uU/mL) (±jackknife standard errors). Values that do not share a superscript letter differ from each other at $p < .05$ in adjusted comparisons.

Table 3

Weighted proportions of women with gynecological/reproductive problems

	Non-obese Non-smoker	Obese only	Smoker only	Obese + Smoker
Menstrual irregularity	.115 ^a	.173 ^a	.171 ^a	.217 ^b
Hysterectomy	.067 ^a	.127 ^b	.119 ^{a,b}	.164 ^b
Oophorectomy	.017 ^a	.051 ^b	.053 ^b	.078 ^b

Note: Values that do not share a superscript letter differ from each other at $p < .05$ in adjusted comparisons.

Table 4

Weighted proportions of women endorsing physical limitations

	Non- obese Non- smoker	Obese only	Smoker Only	Obese + Smoker
Work limitations	.040 ^a	.095 ^b	.149 ^{b,c}	.217 ^{c,d}
Requires use of special healthcare equipment	.007 ^a	.037 ^{b,c}	.030 ^b	.087 ^c

Note: Values represent weighted proportions. Values that do not share a superscript letter differ from each other at $p < .05$ in adjusted comparisons.

Table 5

Weighted mean depression scores

	Non-obese Non-smoker	Obese only	Smoker only	Obese + Smoker
Depression score	2.91 ± 0.14 ^a	4.10 ± 0.19 ^b	5.07 ± 0.32 ^b	6.83 ± 0.44 ^c

Note: Values represent weighted mean scores for PHQ depression screener ($\pm$ jackknife standard errors). Values that do not share a superscript letter differ from each other at $p < .05$ in adjusted comparisons.