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Obesity and the Likelihood of Sexual Behavioral Risk Factors for HPV and Cervical Cancer

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Abstract

Obesity is associated with higher cervical cancer mortality, but its relationship with sexual behavioral risk factors that predispose women to human papilloma virus (HPV) and cervical cancer is unclear. We used data from 3,329 women participants, aged 20–59 years, of the 1999–2004 National Health and Nutrition Examination Survey, to analyze the relationship between BMI and age at first intercourse, number of sexual partners, condom use during sexual activity, history of sexually transmitted disease (STD), herpes simplex virus 2 (HSV-2) seropositivity, and HPV prevalence. BMI was not associated with the prevalence of HPV. Mildly obese women (BMI 30.0–34.9 kg/m²) were least likely to report a STD history (9% vs. 13% in normal weight) and ≥ 2 sexual partners in the previous year (8% vs. 13%) while overweight women (BMI 25.0–29.9 kg/m²) were least likely to report ≥ 10 lifetime partners; among those with multiple partners, BMI was not associated with sexual activity without condoms in the past month. After adjustment for age, race/ethnicity, and education, women with higher BMI were less likely to report sexual behavioral risk factors than normal-weight women; however, odds ratios were only significant for mildly obese women for reporting a STD history (0.74, 95% confidence interval 0.55–0.99) and having ≥ 2 sexual partners in the last year (0.57, 0.39–0.85). Higher BMI was not associated with HSV-2 seropositivity after adjustment. HPV and sexual behavioral risk factors for HPV and cervical cancer are not more prevalent in obese than normal-weight women and unlikely to account for higher-observed cervical cancer mortality in obese women.

Obesity is associated with a higher risk of cancer death including deaths from cervical cancer (1). Each year, almost 10,000 women in the United States are diagnosed with invasive cervical cancer and another 4,000 women die from the disease (2). Women who are moderately to severely obese are more than three times as likely to die from cervical cancer as women with normal body weight (1).

The excess mortality from cervical cancer associated with obesity is especially astounding given that cervical cancer is largely a preventable disease. Regular screening using the Papanicolaou test has been effective in reducing the incidence of this disease in developed countries (3,4). However, despite their higher cancer risk, evidence suggests that women with obesity are less likely to report timely screening for cervical cancer (5,6). Hence, lower adherence to screening recommendations may contribute to the higher risk of premature death from cervical cancer observed among obese women.

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DISCLOSURE

The authors declared no conflict of interest.

The human papilloma virus (HPV) is central to the development of cervical cancer (7) and has become increasingly prevalent (8). Some of these infections are transient but a fraction of these do lead to cervical changes that progress to cervical cancer if not detected and treated early. There are also several serotypes of HPV, some conferring higher risk of malignancy than others (9,10). HPV is transmitted through sexual activity, and several modifiable factors have been identified that place women at higher risk for contracting HPV and in turn lead to higher risk of cervical cancer. These factors include many ameliorable behaviors such as early onset of sexual activity, having multiple sexual partners, and previous sexually transmitted disease (STD) (11–16). Few studies have examined whether the psychosocial consequences of obesity impacts the sexual behaviors of obese individuals, particularly high-risk behaviors that might place women at risk for HPV infection and thus cervical cancer (17).

In this context, we examined the association between obesity and the prevalence of several cervical cancer risk factors reported by participants in the 1999–2004 National Health and Nutrition Examination Study (NHANES) to understand better the contribution of sexual behavioral risk factors to the higher-observed cervical cancer mortality among women with obesity.

METHODS

NHANES is a stratified multistage probability sample of the noninstitutionalized civilian population of the United States conducted by the Centers for Disease Control and Prevention and the National Center for Health Statistics (18). NHANES is designed to assess the health and nutritional status of adults and children and combines both interviews and physical examination. Beginning in 1999, the study was redesigned to be an ongoing continuous program. Data are released in 2-year cycles. Design weights and sampling information are provided so that analyses that account for the complex sampling design can be generalized to the US population.

As part of the interview, participants aged 20–59 years were asked a series of questions about their health and sexual behavior. We restricted our study sample to women participants in this age group ($n = 4,132$). Questions elicited whether women ever had sexual intercourse, the age at which they first had sexual intercourse, and the number of sexual partners in the past month, in the past year, and in their lifetime. Among women who reported sexual activity in the previous month ($n = 408$), they were asked the number of times they had intercourse without using a condom. Participants were also asked about having been diagnosed with a STD, namely genital herpes, genital warts, gonorrhea, and chlamydia (“Has a doctor ever told you that you had ...”). In addition, women were also asked about demographic information including age, race and ethnicity, and educational level.

All participants underwent physical and laboratory examinations in mobile examination units. Height and weight were measured, which we used to calculate BMI. We then categorized participants according to standard weight categories (19): normal weight or BMI of 18.5–24.9 kg/m² (served as our reference group), overweight or BMI of 25.0–29.9 kg/m², class I obesity or BMI 30.0–34.9 kg/m², and class II–III obesity or BMI 35.0 kg/m² and higher. We excluded women who were missing either height or weight ($n = 52$) as well as women who were underweight (BMI <18.5 kg/m²) ($n = 80$) or who were pregnant by history ($n = 661$) because body weight in these situations may not reflect stable baseline weights. Blood samples were collected and tested for herpes simplex virus 2 (HSV-2) antibodies in women aged 20–49 years. HSV-2 is the cause of most cases of genital herpes, a STD. A positive antibody test (seropositivity) indicates previous infection.

Female respondents to the 2003–2004 NHANES were also asked to submit self-collected cervicovaginal swabs. DNA was extracted using modifications of the QIAmp Mini Kit protocol within one month of sample collection, and HPV detection and typing was conducted as described elsewhere (8). Between January 2003 and April 2004, probes were performed for 27 HPV serotypes including those that were high risk (16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 68, 73, 82) and low risk (6, 11, 40, 42, 54, 55, 83, 84) for cervical cancer. After April, additional high- (67, 69, 70, IS39) and low-risk (61, 62, 64, 71, 72, 81, 89) serotypes were probed.

Data analysis

We used bivariable and multivariable statistical analytic methods to examine the relationship between BMI category and several risk factors of HPV infections and cervical cancer: age at first intercourse, having any history of STD (genital herpes, genital warts, gonorrhea, and chlamydia), number of lifetime partners (≥ 10 partners), whether or not women had two or more partners in the previous year. Among those with two or more partners in the previous year who were also sexually active in the preceding month, we examined the relationship between BMI and reporting not using condoms at least one or more times in the preceding month. Finally in the subgroup who submitted adequate cervicovaginal swabs, we studied the association between BMI and HPV infection. Models were adjusted for age, race/ethnicity, and education level. We weighted analyses to reflect US population estimates and used SUDAAN analytic software (20) to account for the complex stratified sampling scheme so that appropriate standard errors are derived.

RESULTS

Table 1 characterizes our study sample. Of 3,329 nonpregnant women aged 20–59 years who had a BMI of ≥ 18.5 kg/m², 39% were normal weight, 27% were overweight, and 34% were obese. Normal-weight women were significantly younger, were more likely to be non-Hispanic white, and completed more years of formal education.

Figure 1 characterizes women's sexual behavioral risk factors according to BMI before adjustment. Compared to normal-weight women, women with higher BMI were significantly less likely to have two or more sexual partners in the previous year. Obese women with a BMI between 30.0 and 34.9 kg/m² were the least likely to report a prior history of STD, $P = 0.04$; however, women with higher-than-normal BMI had higher rates of HSV-2 seropositivity. Age of intercourse, number of lifetime partners, and condom use did not significantly differ across BMI. BMI was also not significantly associated with active HPV prevalence; these results were consistent when we limited the outcome to include only high-risk HPV serotypes.

After adjustment for age, race and ethnicity, and educational level (Table 2), women with higher-than-normal BMI in general were less likely to report higher-risk sexual behaviors. These results, however, did not achieve statistical significance except in women with BMIs between 30.0 and 34.9 kg/m² who were less likely to report a history of STD or having two or more sexual partners in the previous year. HPV prevalence did not differ by BMI after adjustment.

DISCUSSION

Our study found that the prevalence of HPV did not vary by BMI. Women with a higher BMI were no more likely to report sexual behavioral risk factors that might predispose them to acquiring HPV infection and developing cervical cancer. Compared to normal-weight women, women with a higher BMI were as or less likely to report previous STD, had comparable numbers of lifetime partners, and were as or less likely to have two or more sexual partners in

the previous year; among those with multiple partners in the previous year, heavier women were no more likely to have sexual intercourse without condoms in the previous month. After adjusting for sociodemographic factors, heavier women were also no more likely than normal-weight women to demonstrate objective evidence of previous genital herpes infection.

Beyond its association with adverse health consequences, obesity has been shown to result in profound psychosocial consequences (21). Studies suggest that obese individuals are often viewed as less desirable dating and courtship partners (21–24) and have difficulty entering into romantic and marriage relationships (21,25–27). Obese women tend to marry later and marry less desirable partners even though they do not have lower marital aspirations than thinner women (28). However, few studies have examined whether the social stigma associated with obesity actually affects sexual behavior, particularly high-risk sexual behavior. A recent analysis by Nagelkerke *et al.* (17) using the 1999 and 2000 NHANES sample found that obese men reported fewer lifetime sexual partners than thinner men but did not find a similar relationship in women; however, their sample was only a third the size of ours. Nagelkerke *et al.* also examined the relationship between BMI and HSV-2 seropositivity, but because of limited sample size, could not examine obesity's relationship with other STDs or other high-risk sexual behaviors. Our study confirms the lack of relationship between obesity and the number of lifetime sexual partners in Nagelkerke *et al.*'s earlier work and extends this earlier work in a larger national sample to include other sexual behaviors such as the age of first intercourse, common STDs, and the likelihood of sexual activity without using a condom. We also found that the prevalence of HPV did not vary by BMI. The high prevalence of HPV in the general population (8) may have masked any variation by BMI.

Given our findings, higher death rates from cervical cancer in obese women compared to normal women are unlikely to be due to a higher underlying risk of HPV infection which is a necessary precursor to developing cervical cancer. Rather, higher mortality from cervical cancer among obese women likely reflects lower rates of screening documented by other studies (5,6). Although the absolute difference in screening rates are modest, we estimated previously (5) that a reduction of 3.5 and 6% in screening in overweight and obese women during a 3-year screening interval would lead to ~1,200 cases of missed or delayed diagnoses of invasive cervical cancer, highlighting the importance of targeting efforts at improved screening.

Although HPV infection is a necessary precursor to the development of cervical cancer, women who are exposed and become infected with HPV do not necessarily develop the disease. In many women, especially those who were exposed at younger ages, the infection becomes undetectable over time (29,30). Moreover, the cervical changes that are produced by HPV progress through several stages before malignancy results (30). Hence, cervical cancer can be avoided if these cervical changes are detected early enough and intervened upon through biopsy and colposcopy procedures before the lesions develop into cervical cancer (30). Whether obese women are less likely than thinner women to obtain proper follow-up evaluation and treatment once either HPV or HPV-associated cervical changes are detected has not been studied.

Our study should be interpreted in the context of its limitations. First, the cross-sectional and observational design prevents us from drawing causal inferences about the relationship between obesity and high-risk sexual behaviors. This is particularly the case in the outcome of prior behaviors and STDs because we have no information on respondents' BMI at the time of the past behaviors, although there is likely a strong correlation between past weight and current weight. Second, sexual behavioral characteristics were assessed exclusively by self-report and consequently there is likely under-reporting of socially undesirable behaviors such as high-risk sexual behaviors; the NHANES does, however, use an audio computer-assisted survey interviewer which has been shown to improve the quality of self-reported data on

socially undesirable behavior (31). Moreover, there is no evidence that any under-reporting should occur differentially by BMI and given that the estimates for high-risk behavior for higher-weight women were consistently lower than their normal-weight counterpart, it is unlikely that a strong enough weight-related reporting bias exists that would have altered our conclusions.

In summary, despite the higher risk of death from cervical cancer observed among women with higher BMI, obesity was not associated with a higher prevalence of HPV or with sexual behavioral risk factors that might predispose women to developing HPV infection and cervical cancer. Higher risk of death from cervical cancer among obese women are more likely to be due to previously described disparities in cancer screening than to differences in underlying behavioral risk factors.

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References

1. Jemal A, Siegel R, Ward E, et al. Cancer statistics. *CA Cancer J Clin* 2006;56:106–130. [PubMed: 16514137]
2. Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. *N Engl J Med* 2003;348:1625–1638. [PubMed: 12711737]
3. Janerich DT, Hadjimichael O, Schwartz PE, et al. The screening histories of women with invasive cervical cancer, Connecticut. *Am J Public Health* 1995;85:791–794. [PubMed: 7762711]
4. Sung HY, Kearney KA, Miller M, et al. Papanicolaou smear history and diagnosis of invasive cervical carcinoma among members of a large prepaid health plan. *Cancer* 2000;88:2283–2289. [PubMed: 10820350]
5. Wee CC, McCarthy EP, Davis RB, Phillips RS. Screening for cervical and breast cancer: is obesity an unrecognized barrier to preventive care? *Ann Intern Med* 2000;132:697–704. [PubMed: 10787362]
6. Wee CC, Phillips RS, McCarthy EP. BMI and cervical cancer screening among white, African-American, and Hispanic women in the United States. *Obes Res* 2005;13:1275–1280. [PubMed: 16076999]
7. Castle PE, Wacholder S, Lorincz AT, et al. A prospective study of high-grade cervical neoplasia risk among human papillomavirus-infected women. *J Natl Cancer Inst* 2002;94:1406–1414. [PubMed: 12237286]
8. Dunne EF, Unger ER, Sternberg M, et al. Prevalence of HPV infection among females in the United States. *JAMA* 2007;297:813–819. [PubMed: 17327523]
9. Walboomers JM, Jacobs MV, Manos MM, et al. Human papillomavirus is a necessary cause of invasive cervical cancer worldwide. *J Pathol* 1999;189:12–19. [PubMed: 10451482]
10. Bosch FX, de Sanjose S. *J Natl Cancer Inst Monogr*. Vol. 31. 2003. Chapter 1: human papillomavirus and cervical cancer-burden and assessment of causality; p. 3–13.
11. Castellsagué X, Bosch X, Muñoz N, et al. Male circumcision, penile human papillomavirus infection, and cervical cancer in female partners. *N Engl J Med* 2002;346:1105–1112. [PubMed: 11948269]
12. Anttila T, Saikku P, Koskela P, et al. Serotypes of *Chlamydia trachomatis* and risk for development of cervical squamous cell carcinoma. *JAMA* 2001;285:47–51. [PubMed: 11150108]
13. Wallin KL, Wiklund F, Luostarinen T, et al. A population-based prospective study of *Chlamydia trachomatis* infection and cervical carcinoma. *Int J Cancer* 2002;101:371–374. [PubMed: 12209962]

14. Hawes SE, Kiviat NB. Are genital infections and inflammation cofactors in the pathogenesis of invasive cervical cancer? *J Natl Cancer Inst* 2002;94:1592–1593. [PubMed: 12419778]
15. Castle PE, Wacholder S, Lorinc AT, et al. A prospective study of high-grade cervical neoplasia risk among human papillomavirus-infected women. *J Natl Cancer Inst* 2002;94:1406–1414. [PubMed: 12237286]
16. Appleby P, Beral V, Berrington de González A, et al. Carcinoma of the cervix and tobacco smoking: Collaborative reanalysis of individual data on 13,541 women with carcinoma of the cervix and 23,017 women without carcinoma of the cervix from 23 epidemiological studies. *Int J Cancer* 2006;118:1481–1495. [PubMed: 16206285]
17. Nagelkerke NJ, Bernsen RM, Sgaier SK, Jha P. Body mass index, sexual behaviour, and sexually transmitted infections: an analysis using the NHANES 1999–2000 data. *BMC Public Health* 2006;6:199. [PubMed: 16884541]
18. National Center for Health Statistics. NHANES current data files. <http://www.cdc.gov/nchs/about/major/nhanes/nhanes99-02.htm>
19. US Department of Health and Human Service. The practical guide—identification, evaluation, and treatment of overweight and obesity in adults. National Institutes of Health; Bethesda, MD: 2000. NIH publication no. 004084
20. Shah, BV.; Barnwell, BG.; Hunt, PN.; La Vange, LM. Release 5.50 with Addendum for SUDAAN Changes from 5.50 to 6.30. Research Triangle Institute; Research Triangle Park, NC: 1992. SUDAAN User's Manual.
21. Brownell, KD.; Puhl, R.; Schwartz, MB.; Rudd, L., editors. Weight Bias: Nature, Consequences, and Remedies. Vol. Chapter 11. Guilford Publications; New York: 2005.
22. Sobal J, Burszty M. Dating people with anorexia nervosa and bulimia nervosa: attitudes and beliefs of university students. *Women Health* 1998;27:73–88. [PubMed: 9698639]
23. Sobal J, Nicolopoulos V, Lee J. Attitudes about overweight and dating among secondary school students. *Int J Obes Relat Metab Disord* 1995;19:376–381. [PubMed: 7550520]
24. Venes AM, Krupka LR, Gerard RJ. Overweight/obese patients. *Practitioner* 1982;226:1102–1109. [PubMed: 7111142]
25. Fu H, Goldman N. Incorporating health into marriage choice models: demographic and sociological perspectives. *J Marriage Fam* 1996;58:740–758.
26. Garn SM, Sullivan TV, Hawthorne VM. Educational level, fatness, and fatness differences between husbands and wives. *Am J Clin Nutr* 1989;50:740–745. [PubMed: 2801578]
27. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. *N Engl J Med* 1993;329:1008–1012. [PubMed: 8366901]
28. Ball K, Crawford D, Kenardy J. Longitudinal relationships among overweight, life satisfaction, and aspirations in young women. *Obes Res* 2004;12:1019–1030. [PubMed: 15229343]
29. Moscicki AB, Shiboski S, Broering J, et al. The natural history of human papillomavirus infection as measured by repeated DNA testing in adolescent and young women. *J Pediatr* 1998;132:277–284. [PubMed: 9506641]
30. Franco EL, Villa LL, Sobrinho JP, et al. Epidemiology of acquisition and clearance of cervical human papillomavirus infection in women from a high-risk area for cervical cancer. *J Infect Dis* 1999;180:1415–1423. [PubMed: 10515798]
31. Rogers SM, Willis G, Al-Tayyib A, et al. Audio computer assisted interviewing to measure HIV risk behaviours in a clinic population. *Sex Transm Infect* 2005;81:501–507. [PubMed: 16326855]

Figure 1.

Prevalence of sexual behavioral risk factors across BMI.

*Sex without a condom was only assessed among those with ≥ 2 sexual partners in the previous year. **HPV is available for the 2003–2004 assessment only.

Table 1sample characteristics by BMI ($n = 3,329$)

BMI, kg/m²	18.5–24.9	25.0–29.9	30.0–34.9	≥35.0
Sample size (%)	1,135 (38.6)	929 (26.9)	659 (17.7)	606 (16.8)
Mean age [*] , years	38.1	41.5	41.6	42.9
Race/Ethnicity [*] , %				
White	79	68	65	66
African-American	6	12	17	20
Mexican-Hispanic	5	8	9	7
Other hispanic	4	7	8	6
Other race	5	4	1	1
Education [*] , %				
<High school	10	17	22	17
High school graduate	23	25	25	28
>High school	67	58	53	55

^{*} $P < 0.001$.

Table 2

odds ratio (95% confidence interval) for sexual behavioral risk factors

	≤16 years of age at first intercourse (<i>n</i> = 3,320)	≥10 Lifetime partners (<i>n</i> = 3,328)	≥2 Partners last year (<i>n</i> = 3,326)	Sex without condom in the past month (<i>n</i> = 331)	History of STD (<i>n</i> = 3,327)	HSV-2 seropositivity (<i>n</i> = 2,435)	HPV ^a (<i>n</i> = 990)
BMI, kg/m ²							
18.5–24.9	1.00	1.00	1.00	1.00	1.00	1.00	1.00
25.0–29.9	1.24 (0.98, 1.57)	0.79 (0.62, 1.01)	0.78 (0.57, 1.08)	0.93 (0.38, 2.26)	1.13 (0.82, 1.55)	1.06 (0.75–1.51)	0.99 (0.74, 1.33)
30.0–34.9	1.13 (0.87, 1.48)	0.88 (0.65, 1.20)	0.57 (0.39, 0.85)	0.66 (0.32, 1.37)	0.74 (0.55, 0.99)	0.98 (0.64–1.52)	1.34 (0.99, 1.81)
≥35.0	1.11 (0.84, 1.46)	0.95 (0.73, 1.23)	0.82 (0.56, 1.22)	0.82 (0.36, 1.86)	0.99 (0.65, 1.51)	0.93 (0.70–1.23)	0.78 (0.49, 1.23)

Results adjusted for age, race/ethnicity, and education.

^a Available for the 2003–2004 NHANES participants only.