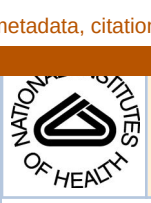

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Body mass index and risk of head and neck cancer by race: the Carolina Head and Neck Cancer Study

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Abstract

Purpose—Most studies, primarily conducted in populations of European ancestry, reported increased risk of head and neck cancer (HNC) associated with leanness (body mass index (BMI) <18.5 kg/m^2) and decreased for overweight or obesity (25.0-<30.0 and >30 kg/m^2, respectively), compared to normal weight (18.5-<25.0 kg/m^2).

Methods—The Carolina Head and Neck Cancer Study is a population-based, racially diverse case-control study of 1,289 incident HNC cases (330 African-Americans) and 1,361 controls (261 African-Americans). Odds ratios (ORs) and 95% confidence intervals (CIs) were estimated for associations between BMI one year pre-diagnosis and HNC risk stratified by race and adjusted for age, sex, smoking, alcohol, and education.

Results—Multiplicative interaction between BMI and race was evident (p_{int}=0.00007). Compared to normal weight, ORs for leanness were increased for African-Americans (OR=3.91, 95% CI 0.72-21.17) and whites (1.48, 0.60-3.65). For overweight and obesity, ORs were decreased in African-Americans (0.51, 0.32-0.83 and 0.47, 0.28-0.79, respectively), but not whites. The increased risk associated with leanness was greater for smokers than non-smokers (p_{int}=0.02).

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Conclusions—These data, which require replication, suggest that leanness is associated with increased HNC risk among African-Americans to a greater extent than whites and overweight and obesity is associated with decreased HNC risk only among African-Americans.

Keywords

epidemiology; case-control studies; cancer of the head and neck; race

Purpose

In the United States, an estimated 52,610 incident cases of oral cavity, pharynx, and larynx cancer – collectively, head and neck cancer (HNC) – and 11,500 associated deaths will occur in 2012 [1]. Between 2000 and 2009, the age-adjusted HNC incidence rate for African-Americans was 12% higher than for whites (16.0 vs. 14.3 per 100,000) [2]. During this time, the age-adjusted mortality rate of HNC for African-Americans was also 57% higher than for whites (5.8 vs. 3.7 per 100,000) [3]. These differences were more pronounced in males, where African-Americans have a 22% higher incidence and an 86% higher mortality of HNC than whites [2, 3].

In North America and Europe, approximately 75% of HNC are attributed to tobacco and alcohol consumption [4-6]. Most of the racial variation has been attributed to differences in the prevalence of exposure to alcohol and tobacco use [7]. However, there might be other risk factors, such as human papillomavirus (HPV) infection or socioeconomic status (SES), which would help explain the racial disparities. A subset of HNCs, specifically cancers of the oropharynx, has been attributed to HPV. These cancers have weaker associations with smoking and drinking and are more likely to be African-American [8]. Additionally, associations between cancer risk and SES vary by race [9, 10].

Body size, as measured by body mass index (BMI, kg/m^2), is another factor that might influence HNC risk and explain racial disparities. The majority of studies have found that being underweight (<18.5 kg/m^2) is associated with a higher HNC risk than normal weight (18.5-<25.0 kg/m^2). While in most studies, overweight (25.0-<30.0 kg/m^2) and obesity (>30 kg/m^2) are associated with a reduced HNC risk, compared to normal weight [11-20]. Conversely, a recent prospective study reported no association between BMI and HNC [21]. However, most of these studies were conducted in populations of European ancestry and did not assess the BMI-HNC relationship among other racial groups. The Carolina Head and Neck Cancer Study (CHANCE) is a large racially diverse population-based study that will allow us to estimate the effects of body size by race on HNC risk.

Methods

The CHANCE study is a population-based, case-control study of incident squamous cell carcinoma of the head and neck [22, 23]. Data were collected between January 1, 2002 and February 28, 2006 in a 46 county region of North Carolina. Cases were aged 20-80 and newly diagnosed with a first primary invasive squamous cell carcinoma of head and neck cancer, including larynx (ICD-O-3 topography codes C32.0-C32.9) and pharynx and oral cavity (C0.00-C14.8). Controls, without previous HNC diagnosis, were frequency matched

to cases using random sampling with stratification by age, race, and sex. The study was approved by the Institutional Review Board at the University of North Carolina at Chapel Hill and all participating institutions.

Participant interviews consisted of a structured questionnaire that assessed demographics and exposure to potential HNC risk factors. Participants self-reported height and weight one year prior to diagnosis. BMI, calculated as weight in kilograms (kg) divided by height in meters (m) squared (kg/m^2), was categorized according to World Health Organization definitions: underweight (<18.5), normal weight (18.5-24.9), overweight (25.0-29.9), and obese (≥30.0) [24].

Individual terms for the matching factors and pairwise product terms of age [20-49 (referent), 50-54, 55-59, 60-64, 65-69, 70-74, 75-80], sex [female (referent), male], and race [white (referent), African-American] were included in all models. Additional covariates were determined by a directed acyclic graph and if they altered the effect estimate by ten percent or more [25]. Confounders included in the full models were education level [high school or less (referent), some college/vocational training, college degree/post-graduate], duration of smoking [never use (referent), 0.5-10, 11-20, 21-30, 31-40, and 41 or more years of cigarette smoking], and lifetime alcohol consumption [never consumed alcohol (referent) and drinkers were divided into quartiles based on milliliters of ethanol consumption in the controls].

Odds ratios (OR) and 95% confidence intervals (CI) for the association between BMI and HNC risk were obtained using unconditional logistical regression and stratified by race. Effect measure modification was evaluated by testing for deviation from a multiplicative interaction model, using the likelihood ratio test to compare the fit of models with and without the interaction term. The association between BMI and HNC risk by race was evaluated by tumor site (including presumed HPV-related HNC sites [8]), smoking status, and sex. P-values for linear trend were calculated using a continuous BMI variable. All p-values are 2-sided and were considered statistically significant if $p<0.05$. In order to better describe the relationship between BMI and risk of HNC by race, we used restricted quadratic splines. For this analysis, the referent BMI was 21.75 (midpoint of normal weight) and knots were set at 18.5, 25.0, and 30.0 kg/m^2. Data analyses were conducted using SAS Institute Inc. software 9.2 (Cary, NC).

Results

The eligible CHANCE study sample consisted of 1,389 cases and 1,396 controls. For this analysis, subjects with lip cancer diagnosis (21 cases) or proxy interview (51 cases, 17 controls) were excluded. The analysis was restricted to individuals self-described as “white” or “African-American” due to the small number of “other” race (excluding 28 cases, 18 controls). The final dataset for this analysis included 1,289 cases (959 white, 330 African-American) and 1,361 controls (1,100 white, 261 African-American). Study characteristics are presented in Table 1.

Results from the analysis of BMI and HNC risk by race are presented in Table 2 (for HNC site stratifications, see Supplemental Tables S1 and S2). A statistically-significant multiplicative interaction between BMI and race was found [p=0.00007, χ^2=21.72 (d.f.=3)]. Compared to normal weight, underweight was associated with an increased HNC risk for whites and African-Americans (OR=1.48, 95% CI: 0.60-3.65; 3.91, 0.72-21.17, respectively). Although the effect estimates were imprecise, the increased risk associated with leanness was 2.6 times greater for African-Americans than whites. Among African-Americans, overweight and obesity were associated with a decreased HNC risk (0.51, 0.32-0.83; 0.47, 0.28-0.79, respectively). However, among whites, obesity was associated with an increased HNC risk (1.34, 1.02-1.76). The p-value for linear trend among whites was p=0.02 and among African-Americans was p=0.01. Leanness in whites was associated with an increased risk of presumed non-HPV-related HNC (1.61, 0.65-3.99) but not HPV-related HNC (0.45, 0.04-4.51). Leanness in African-Americans was associated with an increased risk of both presumed HPV-related and non-HPV-related HNC (5.64, 0.80-39.96; 4.12, 0.67-25.35, respectively), but these estimates are limited by small numbers for African-Americans.

A statistically-significant multiplicative interaction between BMI and smoking was found [p=0.02, χ^2=10.32 (d.f.=3)]. For both whites and African-Americans, the risk, albeit imprecise, associated with leanness was greater for smokers than non-smokers. As we would expect, the effect estimates were similar between presumed HPV-related HNC and nonsmokers. For both whites and African-Americans, there was no difference in the risk associated with leanness by sex [p=0.3, χ^2=4.06 (d.f.=3)].

The restricted quadratic spline graphs are presented in Figure 1 for whites and African-Americans. The splines show a similar trend to the categorical analysis of BMI: leanness was associated with increased risk of HNC in African-Americans and whites and overweight and obesity was associated with decreased risk of HNC in African-Americans but not whites.

Discussion

In this study, we determined that leanness was associated with increased overall HNC risk in both African-Americans and whites, compared to normal weight. Although imprecise, the effect estimate was higher in African-Americans than in whites. However, leanness was associated with decreased HNC risk in HPV-associated sites in whites. Compared to normal weight, a decreased HNC risk was associated with overweight and obesity in African-Americans, but not whites.

The results of lean individuals were similar to a pooled case-control study from the International Head and Neck Cancer Epidemiology (INHANCE) Consortium [20]. Compared to normal weight people in the study, lean individuals had an increased risk of HNC. However, this study found a decreased HNC risk associated with overweight and obesity, compared to normal weight [20]. These results were consistent with our findings for African-Americans but quite different for whites, which is notable given that the INHANCE study was 75% non-Hispanic whites. Given the difference in the minimally-adjusted and

multivariate-adjusted estimates in our study, it is possible that we were better able to adjust for race-specific confounding by smoking in our analyses compared to studies in the pooled analysis. Additionally, these study differences might also be due to shifts in biased reports of weight over time or across countries [26], but this is unclear.

In our study, the increased risk associated with leanness was greater for smokers than non-smokers. This is similar to the INHANCE study that found ever tobacco users had greater increased risk associated with leanness than never tobacco users [20]. The INHANCE study also found that the decreased risk of HNC associated with overweight and obesity was limited to tobacco users [20], which is similar to our results. Furthermore, in another analysis of INHANCE data by Lubin et al. [27], increased risk associated with leanness was greater for women than men for the HPV-associated site of oropharynx but not of non-HPV related sites [27]. Our results, although not statistically significant, are consistent with this observation.

Our results may be due to uncontrolled factors, including residual confounding by smoking, which was the strongest confounder of the BMI-HNC relationship. However, we examined multiple means of adjustment for smoking, including usual lifetime cigarette smoking intensity, pack-years of cigarette smoking, and cigar and pipe smoking. We also examined adjustment for cigarette smoking intensity and duration in the model at the same time and, none of the adjustments resulted in materially different estimates than adjustment for cigarette smoking duration alone. However, adjusting for smoking in the multivariate model attenuated the results for whites more than African-Americans. Therefore, potential residual confounding by smoking could account for some of the differences by race. The racial differences could also be due to the fact that smoking status varies by race and BMI. For instance, the percentage of participants that were never smokers and underweight and normal weight among whites or African-Americans were 0.4 and 29.6%, and 1.8 and 14.9%, respectively. In this study, African-Americans were less educated and heavier drinkers. We explored different adjustments for these covariates in our analysis, but there is still potential for residual confounding by these factors.

This study is the first to assess the association between leanness and HNC risk in a large, racially diverse, population-based study. However, study limitations included self-reported height and weight at baseline for the one year prior to diagnosis and 42.7% of cases diagnosed at an advanced stage. Therefore, there is potential that the cases' weights reported for one year prior to interview might be influenced by disease symptoms. African-Americans were also diagnosed at later stages of HNC (46.7% diagnosed at advanced stage vs. 40.7% in whites), which may have affected their self-report of weight. However, stratifying the estimates by stage did not yield materially different estimates. Additionally, the INHANCE study found underweight individuals had an increased HNC risk at reference and 2-5 years before reference (2.13, 1.75-2.58; 1.56, 0.80-3.02, respectively), but not at 20-30 years of age (0.91, 0.72-1.15) [20]. Thus, being lean over a lifetime is not associated with HNC risk or there is misreporting of weight at ages 20-30. Finally, we are limited in our interpretation of some estimates due to small sample size, especially for African-Americans and underweight participants.

In summary, we report that the associations between BMI and HNC risk vary by race, HPV-related HNC site, and smoking. These differences may be causal or alternatively explained by uncontrolled confounding. This is the first epidemiologic study to determine the association between BMI and HNC risk by race; therefore, further research needs to be conducted on the association between BMI and HNC risk before definite conclusions can be made about racial disparities in HNC incidence and mortality in the United States.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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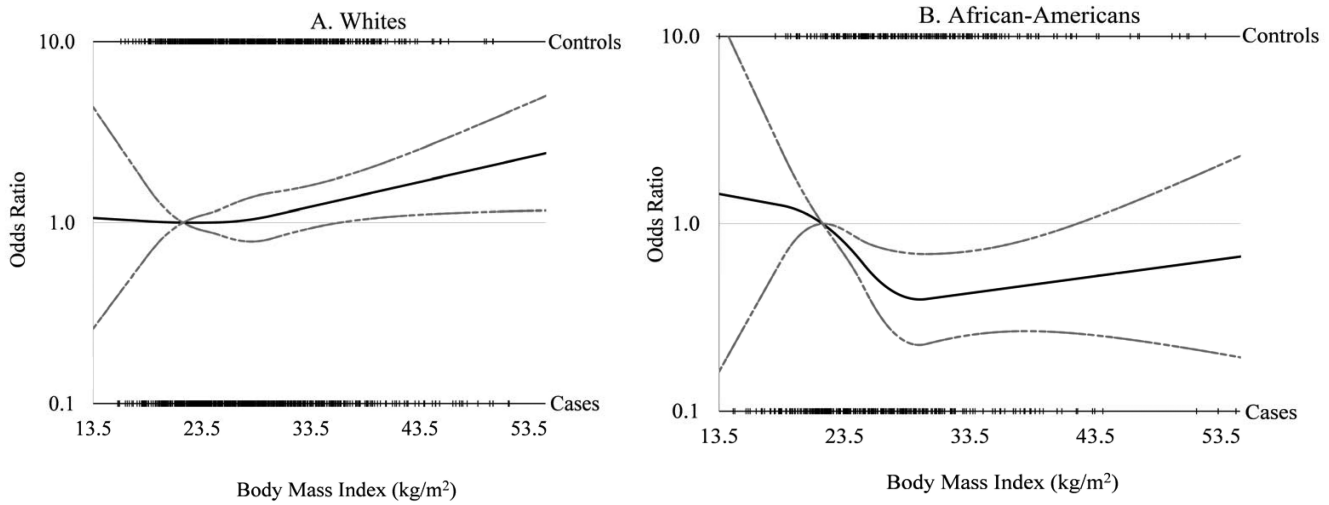
Abbreviations

HNC	head and neck cancer
BMI	body mass index
HPV	human papilloma virus
CHANCE	Carolina Head and Neck Study
OR	odds ratio
CI	confidence interval
INHANCE	International Head and Neck Cancer Epidemiology

References

1. American Cancer Society. Cancer Facts & Figures 2012. American Cancer Society; Atlanta: 2012.
2. Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Incidence - SEER 9 Regs Research Data, Nov 2011 Sub, Vintage 2009 Pops (1973-2009) , National Cancer Institute, DCCPS, Surveillance Research Program, Surveillance Systems Branch, released April 2012, based on the November 2011 submission.
3. Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) SEER*Stat Database: Mortality - All COD, Aggregated With State, Total U.S. (1969-2009) , National Cancer Institute, DCCPS, Surveillance Research Program, Surveillance Systems Branch, released April 2012. Underlying mortality data provided by NCHS (www.cdc.gov/nchs).
4. Negri E, et al. Attributable risk for oral cancer in northern Italy. Cancer Epidemiol Biomarkers Prev. 1993; 2(3):189–93. [PubMed: 8318870]
5. Hashibe M, et al. Alcohol drinking in never users of tobacco, cigarette smoking in never drinkers, and the risk of head and neck cancer: pooled analysis in the International Head and Neck Cancer Epidemiology Consortium. J Natl Cancer Inst. 2007; 99(10):777–89. [PubMed: 17505073]
6. Blot, WJ.; M.J.; Devasa, SS.; Fraumeni, JF, Jr.. Cancers of the oral cavity and pharynx. In: Schottenfeld D, FJ., Jr., editor. Cancer Epidemiology and Prevention. Oxford University Press; New York: 1996.
7. Day GL, et al. Racial differences in risk of oral and pharyngeal cancer: alcohol, tobacco, and other determinants. J Natl Cancer Inst. 1993; 85(6):465–73. [PubMed: 8445674]

8. Chaturvedi AK, et al. Incidence trends for human papillomavirus-related and -unrelated oral squamous cell carcinomas in the United States. Journal of clinical oncology : official journal of the American Society of Clinical Oncology. 2008; 26(4):612–9. [PubMed: 18235120]
9. Yin D, et al. Does socioeconomic disparity in cancer incidence vary across racial/ethnic groups? Cancer causes & control : CCC. 2010; 21(10):1721–30. [PubMed: 20567897]
10. Colevas AD. Population-based evaluation of incidence trends in oropharyngeal cancer focusing on socioeconomic status, sex, and race/ethnicity. Head & neck. 2013
11. Franceschi S, et al. Leanness as early marker of cancer of the oral cavity and pharynx. Ann Oncol. 2001; 12(3):331–6. [PubMed: 11332144]
12. Hashibe M, et al. Body mass index, tobacco chewing, alcohol drinking and the risk of oral submucous fibrosis in Kerala, India. Cancer Causes Control. 2002; 13(1):55–64. [PubMed: 11899118]
13. Nieto A, et al. Lifetime body mass index and risk of oral cavity and oropharyngeal cancer by smoking and drinking habits. Br J Cancer. 2003; 89(9):1667–71. [PubMed: 14583768]
14. Nieto A, et al. BMI throughout life, intake of vitamin supplements and oral cancer in Spain. IARC Sci Publ. 2002; 156:259–61. [PubMed: 12484183]
15. D'Avanzo B, et al. Anthropometric measures and risk of cancers of the upper digestive and respiratory tract. Nutr Cancer. 1996; 26(2):219–27. [PubMed: 8875559]
16. Garavello W, et al. Body size and laryngeal cancer risk. Ann Oncol. 2006; 17(9):1459–63. [PubMed: 16873426]
17. Kabat GC, Chang CJ, Wynder EL. The role of tobacco, alcohol use, and body mass index in oral and pharyngeal cancer. Int J Epidemiol. 1994; 23(6):1137–44. [PubMed: 7721514]
18. Kreimer AR, et al. Diet and body mass, and oral and oropharyngeal squamous cell carcinomas: analysis from the IARC multinational case-control study. Int J Cancer. 2006; 118(9):2293–7. [PubMed: 16331628]
19. Rodriguez T, et al. Risk factors for oral and pharyngeal cancer in young adults. Oral Oncol. 2004; 40(2):207–13. [PubMed: 14693246]
20. Gaudet MM, et al. Body mass index and risk of head and neck cancer in a pooled analysis of case-control studies in the International Head and Neck Cancer Epidemiology (INHANCE) Consortium. Int J Epidemiol. 2010
21. Gaudet MM, et al. Prospective studies of body mass index with head and neck cancer incidence and mortality. Cancer epidemiology, biomarkers & prevention : a publication of the American Association for Cancer Research, cosponsored by the American Society of Preventive Oncology. 2012; 21(3):497–503.
22. Divaris K, et al. Oral health and risk for head and neck squamous cell carcinoma: the Carolina Head and Neck Cancer Study. Cancer Causes Control. 2010; 21(4):567–75. [PubMed: 20049634]
23. Stingone JA, et al. Racial differences in the relationship between tobacco, alcohol, and squamous cell carcinoma of the head and neck. Cancer causes & control : CCC. 2012
24. Report of a WHO consultation on obesity. Obesity: preventing and managing the global epidemic. World Health Organization; Geneva: 1998.
25. Rothman, KJ.; Greenland, S.; Lash, TL. Modern epidemiology. 3rd ed. Vol. x. Wolters Kluwer Health/Lippincott Williams & Wilkins; Philadelphia: 2008. p. 758
26. Gorber SC, Tremblay MS. The bias in self-reported obesity from 1976 to 2005: a Canada-US comparison. Obesity. 2010; 18(2):354–61. [PubMed: 19556977]
27. Lubin JH, et al. An examination of male and female odds ratios by BMI, cigarette smoking, and alcohol consumption for cancers of the oral cavity, pharynx, and larynx in pooled data from 15 case-control studies. Cancer causes & control : CCC. 2011; 22(9):1217–31. [PubMed: 21744095]



[a]Adjusted for age (20-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-80), sex, education (high school or less, some college/vocational training, college degree/post-graduate), lifetime alcohol consumption (0, <29,036.5; 29,036.5-154,359.5; 154,359.6-488,460.0; ≥488,460.1 ml of ethanol), and cigarette smoking duration (0, 0.5-10, 11-20, 21-30, 31-40, and ≥41 years of cigarette smoking).

Figure 1.

Restricted quadratic spline graph of the multivariate adjusted[a] odds ratios (represented by the solid line) and 95% confidence intervals (represented by the dotted lines) of head and neck cancer for A) whites and B) African-Americans (case-control data points denoted on the axes) in the Carolina Head and Neck Cancer study.

Table 1

Characteristics of the Carolina Head and Neck Cancer (2002-2006) study subjects.

	Whites (N=2,059)				African-Americans (N=591)			
	Cases N=959		Controls N=1,100		Cases N=330		Controls N=261	
	n	%	n	%	n	%	n	%
Age (years)								
20-49	170	17.7	120	10.9	83	25.2	36	13.8
50-54	133	13.9	113	10.3	67	20.3	47	18.0
55-59	155	16.2	158	14.4	61	18.5	48	18.4
60-64	164	17.1	159	14.5	53	16.1	46	17.6
65-69	140	14.6	198	18.0	34	10.3	43	16.5
70-74	119	12.4	204	18.5	22	6.7	23	8.8
75-80	78	8.1	148	13.5	10	3.0	18	6.9
Sex								
Female	238	24.8	338	30.7	67	20.3	78	29.9
Male	721	75.2	762	69.3	263	79.7	183	70.1
Education								
High School or less	508	53.0	405	36.8	290	87.9	135	51.7
Some College	273	28.5	338	30.7	34	10.3	68	26.1
College/post-graduate	178	18.6	357	32.5	6	1.8	58	22.2
Body Mass Index (kg/m^2)								
<18.5	26	2.7	9	0.8	22	6.7	2	0.8
18.5-24.9	327	34.1	341	31.0	157	47.7	65	25.0
25.0-29.9	343	35.8	455	41.4	89	27.1	95	36.5
≥30.0	263	27.4	294	26.8	61	18.5	98	37.7
Missing	0		1		1		1	
Alcohol Consumption (ml of ethanol)								
None	111	12.3	235	22.1	10	3.3	54	21.4
<29,036.5	83	9.2	214	20.1	8	2.6	43	17.1
29,036.5-154,359.5	129	14.3	208	19.5	21	6.8	48	19.0
154,359.6-488,460.0	157	17.4	213	20.0	43	14.0	45	17.9
≥488,460.1	420	46.7	194	18.2	225	73.3	62	24.6
Missing	59		36		23		9	
Smoking Duration (years)								
None	156	16.3	420	38.3	14	4.3	101	38.7
0.5-10	53	5.5	124	11.3	6	1.8	25	9.6
11-20	57	6.0	121	11.0	12	3.7	32	12.3
21-30	121	12.6	133	12.1	70	21.3	39	14.9
31-40	216	22.6	127	11.6	114	34.8	31	11.9
≥41	354	37.0	172	15.7	112	34.1	33	12.6
Missing	2		3		2		0	

	Whites (N=2,059)				African-Americans (N=591)			
	Cases N=959		Controls N=1,100		Cases N=330		Controls N=261	
	n	%	n	%	n	%	n	%
Smoking Intensity (cigarettes/day)								
None	156	16.3	420	38.2	14	4.2	101	38.7
1-15	113	11.8	229	20.8	98	29.7	89	34.1
16-25	261	27.3	246	22.4	116	35.2	51	19.5
26-35	170	17.8	82	7.5	46	13.9	14	5.4
≥36	256	26.8	123	11.2	56	17.0	6	2.3
Missing	3		0		0		0	
Site								
Oral Cavity	136	14.2			47	14.2		
Oropharynx	269	28.1			80	24.2		
Hypopharynx	35	3.6			24	7.3		
NOS	181	18.9			56	17.0		
Larynx	338	35.2			123	37.3		
HPV Association								
HPV Site[a]	254	26.5			72	21.8		
Non-HPV Site	705	73.5			258	78.2		
TNM Stage								
I	230	24.0			61	18.5		
II	176	18.4			59	17.9		
III	163	17.0			56	17.0		
IVA	294	30.7			103	31.2		
IVB	92	9.6			46	13.9		
IVC	4	0.4			5	1.5		

[a]HNC sites that have been associated with HPV are cancers of the base on tongue, NOS; lingual tonsil; tonsil; oropharynx; and Waldeyer's ring [8].

Table 2

Minimally and fully adjusted odds ratios (95% confidence intervals) of head and neck cancer in the Carolina Head and Neck Cancer Study (2002-2006) for body mass index, overall and stratified by presumed HPV-related cancer site and smoking status.

	Overall HNC, minimal model[a]			Overall HNC, full model[b,c]			HPV-associated Sites[b]		Non-HPV-Associated Sites[b]		Never Tobacco Smokers[b,d]			Ever Tobacco Smokers[b,d]		
	No. of controls[a]	No. of cases	OR (95% CI)	No. of controls[b]	No. of cases	OR (95% CI)	No. of cases	OR (95% CI)	No. of cases	OR (95% CI)	Controls	No. of cases		Controls	No. of cases	
Whites																
<18.5 kg/m^2	9	26	3.44 (1.55, 7.60)	9	23	1.48 (0.60, 3.65)	1	0.45 (0.04, 4.51)	22	1.61 (0.65, 3.99)	2	0	Not Reported	7	23	1.42 (0.56, 3.61)
18.5-24.9 kg/m^2	341	327	Referent	332	302	Referent	64	Referent	238	Referent	135	32	Referent	197	270	Referent
25.0-29.9 kg/m^2	455	343	0.77 (0.62, 0.95)	441	320	1.05 (0.82, 1.35)	89	1.24 (0.84, 1.85)	231	0.96 (0.73, 1.26)	167	53	1.36 (0.83, 2.25)	274	267	0.98 (0.74, 1.30)
≥30.0 kg/m^2	294	263	0.87 (0.69, 1.10)	280	254	1.34 (1.02, 1.76)	83	1.66 (1.10, 2.51)	171	1.16 (0.86, 1.57)	106	68	2.28 (1.38, 3.76)	174	186	1.10 (0.81, 1.50)
p for trend			0.1			0.02		0.04		0.2			0.002			0.5
African-Americans																
<18.5 kg/m^2	2	22	4.03 (0.91, 17.88)	2	19	3.91 (0.72, 21.17)	6	5.64 (0.80, 39.96)	13	4.12 (0.67, 25.35)	1	1	2.84 (0.18, 45.39)	1	18	5.06 (0.61, 41.78)
18.5-24.9 kg/m^2	65	157	Referent	62	150	Referent	36	Referent	114	Referent	15	2	Referent	47	148	Referent
25.0-29.9 kg/m^2	95	89	0.39 (0.26, 0.60)	92	81	0.51 (0.32, 0.83)	19	0.42 (0.20, 0.88)	62	0.51 (0.30, 0.86)	36	6	0.69 (0.35, 1.37)	56	75	0.50 (0.30, 0.82)
≥30.0 kg/m^2	98	61	0.28 (0.18, 0.44)	95	57	0.47 (0.28, 0.79)	17	0.20 (0.08, 0.51)	50	0.56 (0.32, 0.96)	46	4	0.85 (0.42, 1.70)	49	53	0.41 (0.24, 0.70)
p for trend			<0.0001			0.01		0.0003		0.1			0.4			0.003

[a] Adjusted for age (20-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-80) and sex.

[b] Adjusted for age (20-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-80), sex, education (high school or less, some college/vocational training, college degree/post-graduate), lifetime alcohol consumption (0, <29,036.5; 29,036.5-154,359.5; 154,359.6-488,460.0; ≥488,460.1 ml of ethanol), and cigarette smoking duration (0, 0.5-10, 11-20, 21-30, 31-40, and ≥41 years of cigarette smoking).

[c] p for interaction (between BMI and race)=0.00007, X^2=21.72 (d.f.=3).

[d] p for interaction (between BMI and smoking)=0.02, χ^2=10.32 (d.f.=3).