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RESEARCH ARTICLE

Tobacco Exposure and Cervical Cancer: Smoking-Attributable Mortality in GBD 2021 and Serum Cotinine Associations in NHANES 1999-2018

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Abstract

Background: Persistent high-risk human papillomavirus (HPV) infection is the principal cause of cervical cancer, but modifiable cofactors such as tobacco exposure may influence HPV persistence and disease progression. This study integrated GBD 2021 smoking-attributable mortality estimates with NHANES 1999–2018 biomarker data to assess the population- and individual-level relevance of tobacco exposure to cervical cancer prevention. **Materials and Methods:** GBD 2021 estimates were used to summarize female cervical cancer deaths attributable to smoking and unsafe sex globally and in China in 2021. NHANES analyses included nonpregnant women aged ≥20 years with serum cotinine data in the fasting analytic sample (n=9,899; 141 self-reported cervical cancer cases). Smoking status and serum cotinine levels were analyzed using survey-weighted logistic regression with 20 multiple imputations. **Results:** In GBD 2021, smoking accounted for an estimated 22,614 global female cervical cancer deaths (95% uncertainty interval [UI], 13,558–33,567), representing 7.6% (95% UI, 4.5–11.2) of deaths. In China, smoking accounted for 3,267 deaths (95% UI, 1,751–5,442), or 6.6% (95% UI, 3.6–10.0). In NHANES, current smokers had higher odds of cervical cancer than nonsmokers (adjusted odds ratio [OR], 2.97; 95% confidence interval [CI], 1.79–4.93). Each doubling of serum cotinine was associated with higher odds of cervical cancer (OR, 1.09; 95% CI, 1.06–1.13), and cotinine ≥3.00 ng/mL was associated with higher odds than cotinine <0.05 ng/mL (OR, 3.16; 95% CI, 1.94–5.16). **Conclusion:** Smoking contributed a measurable proportion of cervical cancer mortality in GBD 2021, while current smoking and higher serum cotinine levels were associated with self-reported cervical cancer in NHANES. These cross-sectional associations do not establish causality but support considering tobacco cessation and reduction of smoke exposure as complementary components of cervical cancer prevention alongside HPV vaccination, screening, and timely treatment.

Keywords: cervical cancer; smoking; tobacco exposure; serum cotinine; NHANES; Global Burden of Disease; prevention

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Introduction

Cervical cancer remains one of the leading malignancies affecting women worldwide. The World Health Organization estimated approximately 660,000 new cases and 350,000 deaths in 2022, with the greatest incidence and mortality burden concentrated in low- and middle-income settings [1]. Persistent infection with oncogenic human papillomavirus (HPV) is the principal causal factor in most cervical cancers, but cofactors influence HPV persistence and progression to invasive disease [2]. Tobacco smoking is one such modifiable cofactor and is listed among the factors that increase the risk of cervical cancer progression [1- 3].

From a cancer prevention perspective, the relevant question is whether tobacco exposure contributes meaningfully to the cervical cancer burden in addition to the established HPV pathway. Smoking cessation cannot substitute for HPV vaccination or cervical screening, but tobacco-control interventions can be incorporated into prevention services that already reach women through vaccination, screening, and primary

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care. APJCP studies have emphasized the importance of HPV vaccination, screening access, and awareness of modifiable cervical cancer risk factors, including tobacco use [10- 12]. Quantifying smoking-attributable mortality and examining biomarker-based associations may therefore help define the complementary role of tobacco control in comprehensive cervical cancer prevention.

A recent analysis of NHANES 1999–2018 combined with Mendelian randomization reported positive associations between current smoking, serum cotinine, and cervical cancer [9]. Because the NHANES component of the present study overlaps with that data source, the added contribution here is the explicit comparison of individual-level biomarker associations with GBD 2021 smoking-attributable cervical cancer mortality. This population-burden context is intended to clarify the potential prevention relevance of tobacco exposure rather than to present the NHANES association as independent evidence from a wholly distinct population.

Materials and Methods

Study Design

This study combined an ecological descriptive analysis of GBD 2021 risk-attributable cervical cancer mortality with a cross-sectional analysis of NHANES 1999–2018. The analytic objective was to determine whether population-level tobacco-attributable mortality and individual-level biomarker associations provide convergent evidence supporting tobacco exposure as a relevant target for cervical cancer prevention.

GBD 2021 Data

GBD 2021 risk-attributable estimates for female cervical cancer were extracted from local IHME GBD results files [4,5]. We summarized deaths among females of all ages in 2021 for the cause of cervical cancer and the risk factors smoking, tobacco, and unsafe sex. Metrics included the number and percentage of deaths and the death rate, with 95% uncertainty intervals. The manuscript emphasizes smoking and unsafe sex because smoking represents the tobacco-specific exposure of interest, whereas unsafe sex is the dominant GBD risk category for attribution of HPV-mediated cervical cancer.

NHANES Data Source and Participants

NHANES is a nationally representative survey of the civilian, noninstitutionalized U.S. population that combines interviews, physical examinations, and laboratory testing [6]. We analyzed continuous NHANES cycles from 1999–2000 through 2017–2018. The initial merged dataset included 101,316 participants. Eligibility was restricted to participants aged ≥20 years, nonpregnant female participants, and those with the exposure and outcome information required for the fasting analytic sample. The final primary analytic sample included 9,899 women, with 141 self-reported cervical cancer cases. A complete-case sample (n=7,722; 116 cases) and a broader sensitivity sample analyzed using Mobile Examination Center (MEC) examination weights and excluding fasting-only laboratory covariates (n=17,104; 254 cases) were used in sensitivity analyses. No a priori power calculation was performed because all eligible NHANES participants from the selected cycles were included.

Outcome, Exposures, and Covariates

The outcome was self-reported history of cervical cancer derived from NHANES questionnaire data. Participants who reported a previous diagnosis of cervical cancer were classified as cases. Because NHANES is cross-sectional and the timing of cancer diagnosis relative to tobacco exposure cannot be established from these public-use files, all analyses were interpreted as associations rather than causal incidence estimates.

Smoking exposure was assessed in two ways. First, self-reported smoking status was categorized as nonsmokers, former smokers, and current smokers. Second, serum cotinine concentration was used as an objective biomarker of recent nicotine exposure. Cotinine was modeled both continuously after log2 transformation and categorically as <0.05 ng/mL, 0.05–2.99 ng/mL, and ≥3.00 ng/mL. The fully adjusted model included age, race/ethnicity, marital status, family income-to-poverty ratio, education, body mass index, waist circumference, alcohol use, fasting glucose, insulin, glycated hemoglobin, albumin, creatinine, hypertension, and diabetes.

Statistical Analysis

NHANES analyses accounted for the complex survey design using strata, primary sampling units, and combined fasting subsample weights in accordance with NHANES analytic guidance [7]. The primary analysis used WTSAF20YR weights constructed from fasting subsample weights across cycles (1999–2002 WTSAF4YR divided by 5; 2003–2018 WTSAF2YR divided by 10). Missing covariate data were handled using multiple imputation by chained equations with 20 imputed datasets and 20 iterations. Survey-weighted logistic regression was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs). All statistical tests were two-sided, and $P<0.05$ was considered statistically significant. Trend tests modeled smoking and cotinine categories as ordinal variables. Restricted cubic spline analysis of log2-transformed serum cotinine was performed in the first imputed dataset using survey-weighted logistic regression and Wald tests for overall and nonlinear associations. Sensitivity analyses included complete-case models, models excluding laboratory covariates, and models using Mobile Examination Center (MEC) examination weights in the broader sample without fasting-only laboratory covariates. Analyses were conducted in R version 4.5.0 using the survey, mice, tableone, dplyr, tidyr, and ggplot2 packages.

Ethics Statement

NHANES protocols were reviewed and approved by the National Center for Health Statistics Ethics Review Board or predecessor review bodies, and documented informed consent was obtained from participants [8]. The current analysis used publicly available, deidentified NHANES files and publicly available GBD estimates;

therefore, no additional ethics approval or individual participant consent was required for this secondary analysis.

Results

GBD 2021 Cervical Cancer Mortality Attributable to Risk Factors

Table 1 presents GBD 2021 cervical cancer deaths attributable to selected risk factors. GBD 2021 estimated that smoking accounted for 22,614 female cervical cancer deaths globally in 2021 (95% UI, 13,558–33,567), corresponding to 7.6% of cervical cancer deaths (95% UI, 4.5–11.2). In China, smoking accounted for 3,267 deaths (95% UI, 1,751–5,442), corresponding to 6.6% of cervical cancer deaths (95% UI, 3.6–10.0). Unsafe sex, the GBD risk category most directly aligned with HPV-mediated cervical carcinogenesis, accounted for substantially higher absolute mortality estimates: 296,597 global deaths (95% UI, 271,681–321,145) and 49,821 deaths in China (95% UI, 36,573–64,084).

NHANES Participant Flow and Baseline Characteristics

The NHANES analytic dataset was derived from 101,316 participants merged across survey cycles from 1999–2018. After restriction to adults aged ≥20 years (n=55,081), exclusion of pregnant participants (n=53,540), restriction to female participants (n=27,067), and application of the eligibility criteria for the fasting core analytic sample, 9,899 women remained, including 141 cervical cancer cases. Weighted baseline tables indicated differences across smoking and cotinine categories in age, body size, race/ethnicity, education, marital status, income, alcohol use, and selected metabolic characteristics.

The weighted prevalence of self-reported cervical cancer was 1.1% among nonsmokers, 2.0% among former smokers, and 3.8% among current smokers. By serum cotinine category, weighted cervical cancer prevalence was 1.1% for <0.05 ng/mL, 1.9% for 0.05–2.99 ng/mL, and 3.5% for ≥3.00 ng/mL.

Smoking Status and Serum Cotinine

Table 2 presents the adjusted NHANES associations of smoking status and serum cotinine with self-reported cervical cancer. After full adjustment, the association remained statistically significant for current smokers (OR, 2.97; 95% CI, 1.79–4.93; P<0.0001), whereas the estimate for former smokers was attenuated and was not statistically significant (OR, 1.60; 95% CI, 0.84–3.06; P=0.1533). The ordinal smoking trend was statistically significant (OR per category, 1.72; 95% CI, 1.33–2.23; P<0.0001).

Serum cotinine was positively associated with self-reported cervical cancer. In the fully adjusted model, each doubling of serum cotinine was associated with higher odds of cervical cancer (OR, 1.09; 95% CI, 1.06–1.13; P<0.0001). Compared with cotinine <0.05 ng/mL, cotinine 0.05–2.99 ng/mL showed a borderline association (OR, 1.88; 95% CI, 0.99–3.58; P=0.0544), whereas cotinine ≥3.00 ng/mL was associated with higher odds of cervical cancer (OR, 3.16; 95% CI, 1.94–5.16; P<0.0001). The cotinine category trend was statistically significant (OR per category, 1.78; 95% CI, 1.40–2.26; P<0.0001). The

Table 1. GBD 2021 Cervical Cancer Deaths Attributable to Selected Risk Factors.

Location	Risk factor	Deaths, n (95% UI)	Percent, % (95% UI)	Rate (95% UI)
Global	Smoking	22,614 (13,558–33,567)	7.6 (4.5–11.2)	NR
Global	Unsafe sex	296,597 (271,681–321,145)	NR	NR
China	Smoking	3,267 (1,751–5,442)	6.6 (3.6–10.0)	NR
China	Unsafe sex	49,821 (36,573–64,084)	NR	NR

Abbreviation: UI, uncertainty interval; NR, not reported.

Note: Values shown are those reported in the manuscript Results section. Rates and percentages not reported in the submitted manuscript text are indicated as NR rather than being reconstructed or inferred.

Table 2. Adjusted NHANES Associations of Smoking Status and Serum Cotinine with Self-reported Cervical Cancer.

Analysis	Exposure	N	Events	OR (95% CI)	P value
Smoking status	Former smokers	9,899	141	1.602 (0.839–3.058)	0.1533
Smoking status	Current smokers	9,899	141	2.974 (1.794–4.929)	<0.0001
Cotinine, continuous	Log2-transformed serum cotinine	9,899	141	1.091 (1.056–1.127)	<0.0001
Cotinine, category	0.05–2.99 ng/mL	9,899	141	1.880 (0.988–3.576)	0.0544
Cotinine, category	≥3.00 ng/mL	9,899	141	3.163 (1.940–5.156)	<0.0001
Smoking status trend	Ordinal trend	—	—	1.723 (1.332–2.230)	<0.0001
Cotinine category trend	Ordinal trend	—	—	1.776 (1.399–2.255)	<0.0001

Abbreviations: CI, confidence interval; NHANES, National Health and Nutrition Examination Survey; OR, odds ratio.

Notes: The fully adjusted model included age, race/ethnicity, marital status, income-to-poverty ratio, education, body mass index, waist circumference, alcohol use, glucose, insulin, glycated hemoglobin (HbA1c), albumin, creatinine, hypertension, and diabetes. For smoking status, nonsmokers were the reference category. For cotinine categories, <0.05 ng/mL was the reference category. The continuous cotinine estimate represents the change in odds associated with each doubling of serum cotinine. Ordinal trend models treated the smoking-status and cotinine categories as ordered variables.

Table 3. Selected Sensitivity Analyses of the Associations between Tobacco Exposure and Self-reported Cervical Cancer.

Analysis	Exposure	N	Events	OR (95% CI)	P value
Complete-case: smoking	Current smokers	7,722	116	3.203 (1.775–5.780)	<0.001
Complete-case: cotinine, continuous	Log2-transformed serum cotinine	7,722	116	1.090 (1.052–1.129)	<0.0001
Complete-case: cotinine, category	≥3.00 ng/mL	7,722	116	3.182 (1.867–5.421)	<0.0001
Excluding laboratory covariates: smoking	Current smokers	9,899	141	2.953 (1.765–4.942)	<0.0001
Excluding laboratory covariates: cotinine, continuous	Log2-transformed serum cotinine	9,899	141	1.090 (1.054–1.127)	<0.0001
Excluding laboratory covariates: cotinine, category	≥3.00 ng/mL	9,899	141	3.128 (1.899–5.154)	<0.0001
MEC examination-weighted, excluding laboratory covariates: smoking	Current smokers	17,104	254	4.768 (3.068–7.408)	<0.0001
MEC examination-weighted, excluding laboratory covariates: cotinine, continuous	Log2-transformed serum cotinine	17,104	254	1.108 (1.076–1.141)	<0.0001
MEC examination-weighted, excluding laboratory covariates: cotinine, category	≥3.00 ng/mL	17,104	254	3.623 (2.396–5.480)	<0.0001

Abbreviations: CI, confidence interval; MEC, mobile examination center; OR, odds ratio.

Notes: The complete-case analysis included participants with complete data for the variables included in the corresponding model. The models excluding laboratory covariates omitted glucose, insulin, glycated hemoglobin, albumin, and creatinine. The MEC examination-weighted sensitivity analysis used the broader sample with Mobile Examination Center examination weights and excluded fasting-only laboratory covariates. For categorical cotinine analyses, <0.05 ng/mL was the reference category. The continuous cotinine estimate represents the change in odds associated with each doubling of serum cotinine.

restricted cubic spline analysis is shown in Figure 1 and supported an overall association between serum cotinine and cervical cancer ($P=1.16\times10^{-5}$), with no evidence of nonlinearity (P=0.1537).

Sensitivity Analyses

Sensitivity analyses are summarized in Table 3 and were broadly consistent with the primary findings. In complete-case models, current smoking was associated with higher odds of cervical cancer (OR, 3.20; 95% CI, 1.78–5.78), each doubling of cotinine remained associated with higher odds (OR, 1.09; 95% CI, 1.05–1.13), and cotinine ≥3.00 ng/mL remained associated with higher odds (OR, 3.18; 95% CI, 1.87–5.42). Models excluding laboratory covariates produced estimates close to those of the primary analysis. In the broader MEC examination-weighted analysis without fasting-only laboratory covariates, the estimates were stronger, particularly for current smoking (OR, 4.768; 95% CI, 3.068–7.408) compared with the primary analysis (OR, 2.974; 95% CI, 1.794–4.929); the estimate for cotinine ≥3.00 ng/mL was also higher (OR, 3.623 vs 3.163).

Discussion

This study integrated global burden estimates with U.S. individual-level survey data to evaluate tobacco exposure in relation to cervical cancer. GBD 2021 estimated that smoking accounted for approximately 22,600 female cervical cancer deaths globally and 3,300 deaths in China in 2021, representing a smaller burden than the unsafe-sex risk category but a potentially preventable contributor. In NHANES 1999–2018, both self-reported current smoking and serum cotinine were associated with higher odds of self-reported cervical cancer after adjustment for sociodemographic, behavioral, metabolic, and clinical covariates.

The findings provide two complementary perspectives. At the population level, GBD 2021 attributes a measurable proportion of cervical cancer mortality to smoking. At the individual level, current smoking and higher serum cotinine were associated with a history of cervical cancer in NHANES. These findings should not be interpreted as equivalent forms of causal evidence: GBD estimates

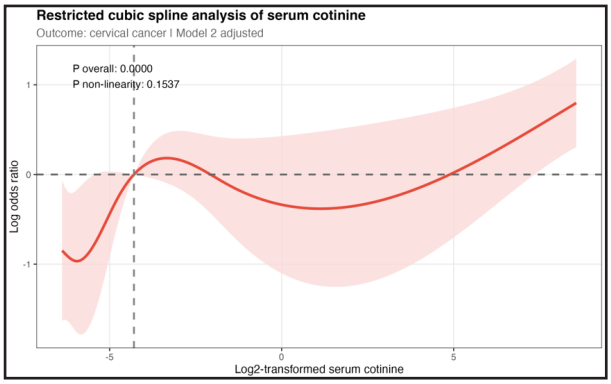


Figure 1. Restricted Cubic Spline Analysis of Log2-transformed Serum Cotinine and Self-reported Cervical Cancer in the Fully Adjusted NHANES Model. The plot shows the log odds ratio relative to the reference cotinine value, and the shaded band represents the 95% confidence interval. The overall association was statistically significant ($P=1.16\times10^{-5}$), with no evidence of a nonlinear association (P=0.1537).

are modeled population-attributable estimates, whereas NHANES is cross-sectional. Nevertheless, the results are compatible with a prevention framework in which tobacco cessation complements, rather than competes with, HPV vaccination and cervical screening. APJCP and APJCB publications similarly emphasize integrated prevention through HPV vaccination, screening, early detection, and risk-factor awareness [10- 14].

These findings are consistent with epidemiologic and mechanistic literature describing smoking as a cofactor in HPV persistence, cervical precancer, and invasive cervical cancer [2, 3]. They are also directionally consistent with the recent NHANES and Mendelian randomization analysis by Su et al. [9]. Importantly, the present NHANES analysis should be interpreted in light of that overlap rather than as an independent replication. Its principal extension is the integration of the biomarker association with GBD 2021 risk-attributable mortality estimates [4, 5]. Studies in APJCP have identified tobacco use as a recognized cervical cancer risk factor in prevention-focused populations [12], while APJCB studies continue to underscore the central etiologic role of HPV and the importance of screening and early detection [13, 14].

Several biological and behavioral pathways may help explain the observed association. Tobacco smoke contains carcinogenic compounds that can reach cervical tissue and may contribute to local DNA damage, oxidative stress, and impaired immune responses. Smoking may thereby reduce clearance of oncogenic HPV infection or facilitate disease progression in persistently infected women [2, 3]. At the population level, tobacco exposure may also cluster with socioeconomic disadvantage, reduced screening access, and other health behaviors. These pathways are biologically plausible, but they cannot be directly tested with the present cross-sectional NHANES analysis.

Strengths of this study include the use of two complementary data sources, objective biomarker-based assessment of tobacco exposure, nationally representative NHANES survey weighting, multiple imputation, trend testing, restricted cubic spline analysis, and sensitivity analyses. The broader MEC examination-weighted analysis produced stronger estimates than the primary fasting-subsample analysis, particularly for current smoking. Although the direction of association was unchanged, the difference in magnitude indicates that the effect estimates were sensitive to differences in the analytic sample, survey weights, and covariate specification. Several limitations should temper interpretation. NHANES is cross-sectional, and the temporal sequence between tobacco exposure and cervical cancer diagnosis cannot be determined. Cervical cancer history was self-reported and may be affected by recall or classification error. HPV infection, HPV vaccination, cervical screening history, histology, stage, treatment, time since diagnosis, and detailed sexual behavior variables were not available or were not consistently harmonized in the analytic files. Serum cotinine reflects recent nicotine exposure and may not capture lifetime smoking intensity. Residual confounding by socioeconomic factors, healthcare access, HPV-related factors, or secondhand smoke is possible. GBD estimates depend on modeling assumptions regarding risk exposure, relative risks, and counterfactual attribution; the unsafe-sex category should be interpreted as a modeled risk-attributable pathway rather than a direct individual-level exposure measure.

In conclusion, GBD 2021 estimates indicate a measurable smoking-attributable cervical cancer mortality burden, and NHANES analyses show associations between current smoking, higher serum cotinine, and self-reported cervical cancer. Because NHANES is cross-sectional and lacks consistent information on HPV status, screening history, and timing of diagnosis, these results do not establish that current tobacco exposure caused the reported cancers. Nevertheless, they support evaluating tobacco cessation and reduction of smoke exposure as complementary elements of cervical cancer prevention alongside HPV vaccination, screening, and equitable access to treatment [10- 14]. Prospective studies incorporating HPV status, lifetime tobacco exposure, screening history, and timing of diagnosis are needed to better define causal pathways and potential intervention targets.

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