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Original investigation

Relationships Between Smoking Behaviors and Cotinine Levels Among Two American Indian Populations With Distinct Smoking Patterns

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Abstract

Introduction: Smoking prevalence, cigarettes per day (CPD), and lung cancer incidence differ between Northern Plains (NP) and Southwest (SW) American Indian populations. We used cotinine as a biomarker of tobacco smoke exposure to biochemically characterize NP and SW smokers and nonsmokers and to investigate factors associated with variation in tobacco exposure.

Methods: American Indians ($N = 636$) were recruited from two different tribal populations (NP and SW) as part of a study conducted as part of the Collaborative to Improve Native Cancer Outcomes P50 project. For each participant, a questionnaire assessed smoking status, CPD, second-hand smoke exposure, and traditional ceremonial tobacco use; plasma and/or salivary cotinine was measured.

Results: Cotinine levels were (mean ± 95% confidence interval [CI]) 81.6 ± 14.1 and 21.3 ± 7.3 ng/ml among NP smokers and non-mokers, respectively, and 44.8 ± 14.4 and 9.8 ± 5.8 ng/ml among SW smokers and nonsmokers, respectively. Cotinine levels correlated with CPD in both populations ($p < .0001$). Cotinine ≥15 ng/ml was measured in 73.4% of NP smokers and 47.8% of SW smokers and in 19.0% of NP nonsmokers and 10.9% of SW nonsmokers. Ceremonial traditional tobacco use was associated with higher cotinine among NP smokers only ($p = 0.004$). Second-hand smoke exposure was associated with higher cotinine among NP non-smokers ($P < 0.02$). More second-hand smoke exposure was associated with smoking more CPD in both populations ($p = 0.03$–0.29). Linear regression modeling mirrored these findings.

Conclusions: High prevalence of smoking in the Northern Plains and high cotinine levels among nonsmokers in both regions highlights the tribal populations' risk for tobacco-related disease.

Implications: There is a high prevalence of smoking in Northern Plains American Indians. Among Northern Plains and Southwest nonsmokers, relatively high cotinine levels, representative of high tobacco exposure, suggest considerable exposure to second-hand smoke. It is critical to highlight the extent of second-hand smoke exposure among the Northern Plains and Southwest American Indians and to enhance efforts to initiate smoke-free policies in tribal communities, which are not subject to state-level polices.

Introduction

Commercial tobacco use, the leading cause of preventable disease and death in the United States, is more prevalent in adult American Indians and Alaska Natives (AI/ANs) than in other US populations. For cigarette smoking, prevalence and patterns of use vary substantially across tribal populations. The Northern Plains (NP) tribal members of South Dakota have a smoking prevalence of ~50% compared to ~14% among Southwest (SW) tribal members in Arizona.[1] The NP tribal members who smoke consume an average of 13 cigarettes per day (CPD), compared to 7 CPD in SW tribal populations.[1,2] As smoking is a risk factor for many health conditions, including lung cancer and cardiovascular disease, the distinctive rates of smoking reported for these two regions are consistent with tribal members' different rates of lung cancer incidence (104/100 000 in NP vs. 15/100 000 in SW) and mortality (94/100 000 in NP vs. 14/100 000 in SW).[3,4] Despite these smoking patterns, most AI/AN adults report a desire to quit smoking and have made quit attempts.[5–7]

AI/AN tribes differ in their consumption of commercial tobacco and use of traditional tobacco for spiritual and ceremonial purposes.[1,8,9] Ceremonial traditional tobacco generally does not contain nicotine, the major psychoactive component of tobacco.[10] However, nicotine-containing commercial tobacco can be used in combination with traditional tobacco, and this practice varies across tribes and age-groups.[11–13] The shift toward use of commercial tobacco for ceremonial purposes resulted, in part, from convenience of obtaining commercial compared to traditional tobacco and in response to commercial cigarette advertisements; for further information please see the Black Hills Center for American Indian Health videos (e.g., https://www.youtube.com/watch?v=l_gYL_oJxhg&spfreload=5, Accessed May 29, 2017).

Second-hand smoke inhalation, another potential source of nicotine and carcinogen exposure, can increase lung cancer risk by 20–30% in nonsmokers.[14] Smoke-free policies have been implemented in public places throughout the United States,[15] and as a result, rates of exposure to second-hand smoke and associated chemical additives have fallen substantially in most US communities.[16] However, second-hand exposure in tribal populations is poorly understood; studies of the effectiveness of smoke-free policies have not typically included residents of AI/AN reservations.[17,18] As sovereign nations, AI/AN tribes are not legally subject to state-level smoke-free policies.[12] Therefore, it is critical to establish the extent of second-hand smoke exposure among understudied NP and SW American Indians, particularly for efforts to initiate smoke-free policies in tribal communities.

American Indian populations in the NP and SW likely experience differential exposure to tobacco smoke through different sources, both actively and passively. This variation might affect their risk for tobacco-related disease. One way to quantify tobacco smoke exposure and lung cancer risk is to measure levels of cotinine, the primary metabolite of nicotine,[19] in smokers and nonsmokers. The utility of cotinine as a biomarker of tobacco smoke exposure derives from its relatively long half-life of ~16 hours.[20] Plasma cotinine levels are robustly associated with self-reported CPD and lung cancer risk among active smokers.[21,22] Cotinine levels above a given threshold have also been used to differentiate active smoking from second-hand tobacco smoke exposure; potential cutpoints range from 1 to 15 ng/ml of plasma cotinine.[23,24] Although cotinine >15 ng/ml has been used in the past as a cutpoint for active smoking,[23] recent work advocates a more stringent threshold of 3 ng/ml.[24] Nonetheless, the higher threshold is likely more relevant for assessing passive exposure in AI/AN communities, given the absence of smoke-free policies on most reservations. For the present investigation, we elected to assess the proportion of smokers and nonsmokers with cotinine ≥15 and 3 ng/ml.

No previous studies in NP or SW tribal communities have used biomarkers to investigate tobacco smoke exposure. Thus, the relationship of cotinine with smoking levels, as well as other potential sources of variability in tobacco smoke exposure, has never been explored in these communities. The aim of the present study was to assess cotinine levels and investigate factors associated with variation in cotinine among NP and SW smokers and nonsmokers, using survey measures of exposure. In particular, this study assessed commercial cigarette consumption, use of ceremonial traditional tobacco products, and potential sources of passive smoke exposure.

Methods

Study Design

The data presented here were collected for a cross-sectional study entitled "Topography and Genetics of Smoking and Nicotine Dependence in American Indians," one of five major research studies conducted by the Collaborative to Improve Native Cancer Outcomes P50 program project. To protect the confidentiality of the participating communities, geographic descriptors, consistent with previous publications and approval by tribal review boards, were assigned instead of tribal names. Participating tribal populations in each region (NP versus SW) were culturally and linguistically unrelated to those in the other region and had substantially different historical experiences. The SW group is urban, while the NP group resides predominantly on rural reservations. NP participants were recruited from a random subset of participants in an earlier study of community health.[25] The original study from which participants were resampled represented approximately one-third of all adults in the participating reservations and communities.[26] SW participants were recruited using respondent-driven sampling among American Indian friends and family members of tribal participants in an earlier randomized clinical trial in the greater Phoenix metropolitan area[27]; both NP and SW subsamples were stratified by sex and smoking status. The data presented stem from a secondary analysis of cotinine levels in the NP and SW tribal populations; therefore, a priori power analyses were not performed to calculate the minimum sample size required in each population.

Data Collection

From 2012 to 2014, all participants completed a questionnaire either via an interviewer or self-administered, which assessed, in part, demographics, tobacco use, nicotine dependence, and social influences on smoking. Biological samples (blood or saliva) were also collected from all participants. The majority of the participants provided a blood sample for the cotinine analysis (97%), with the remaining 3% providing a saliva sample. The individuals who provided saliva samples were from both tribal populations (N = 12 from NP, N = 9 from SW). There is generally strong agreement between saliva and plasma/blood measures of cotinine,[28] and the same cotinine cutpoint is suggested to differentiate active versus second-hand smoke exposure in saliva and plasma.[29] The final cohort comprised 636 American Indians aged 20–88 years, with 426 in the NP and 210 in the SW. Ethical approval for all study procedures was obtained from the review boards of the Great Plains Indian Health Service, the University of Toronto, the University of Washington, MedStar Health Research Institute,

and appropriate tribal entities. All participants provided informed consent.

Measures

Smoking status, CPD, ceremonial traditional tobacco use, and two measures of second-hand smoke exposure were assessed, as outlined in Supplementary Table 2. For smoking status, former and never smokers were aggregated in a single nonsmoking group, within each tribal population, distinguishing them from participants who reported current cigarette smoking. To determine the amount of current active tobacco consumption, participants reported either CPD or cigarettes per month (CPMo). Data on CPMo were divided by 30 for consistency with CPD data. Within each tribal population, and within smoking status groups, participants who formerly or never used ceremonial traditional tobacco were also aggregated in a single nontraditional tobacco user group, being separate from participants who reported current traditional tobacco use. Our two measures of second-hand smoke exposure were (1) allowing smoking in the home and (2) having friends who smoke. Each measure of second-hand smoke was analyzed independently. Either plasma or salivary cotinine was measured in biological samples collected from all participants using liquid chromatography tandem mass spectrometry; analytic methods have been previously described.[30]

Statistical Analysis

As these are two distinct American Indian populations and smoking patterns differ between the NP and the SW, we have chosen to analyze each tribal population separately, allowing us to determine independent relationships between cotinine levels and CPD, ceremonial traditional tobacco use, and second-hand tobacco smoke exposure in each tribal population. Cotinine levels and CPD were nonnormally distributed, indicating the use of nonparametric statistical tests. The correlations of cotinine levels and CPD within each tribal population were determined by Spearman correlations. Chi-square tests were used to determine differences between the proportion of smokers and nonsmokers who had mean cotinine levels equal to or higher than our predetermined cutpoints, 3 and 15 ng/ml, both of which have been used to differentiate second-hand from active smoke exposure. We used Mann–Whitney and Kruskal–Wallis tests to analyze the association of smoking status, traditional tobacco use, and our two measures of passive smoke exposure with cotinine levels and CPD in each smoking status group. We ran separate linear regression models for NP smokers, NP nonsmokers, SW smokers, and SW nonsmokers to calculate the percentage of variation in cotinine levels attributable to each variable in each group. Model variables included CPD (except for nonsmokers), traditional tobacco use, smoking in the home, and number of friends who smoked. Cotinine was the output variable. Analyses were conducted with GraphPad Prism (v6.0) and SPSS (v22), and statistical tests were considered significant for $p < .05$.

Results

Cotinine Levels in the NP

NP smokers had cotinine levels (mean ± 95% confidence interval [CI]) of 81.6 ± 14.1 ng/ml, and NP nonsmokers had cotinine levels of 21.3 ± 7.3 ng/ml (Figure 1). The latter value exceeds both cutpoints (3 and 15 ng/ml) used for second-hand smoke exposure.[23,29] Previous studies indicated that NP smokers consumed an average of 13 CPD.[2] However, smokers in the present study reported consuming ~7 CPD,

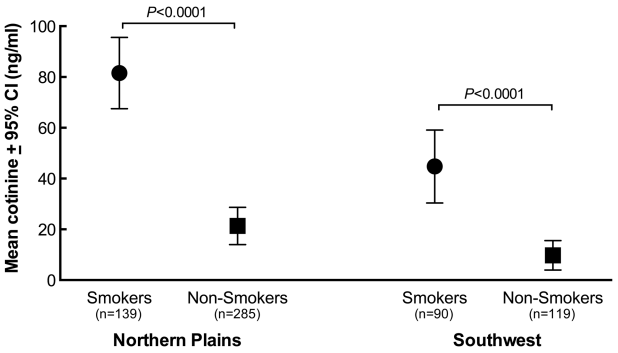

Figure 1. Association between self-reported smoking status and cotinine levels (ng/ml) among Northern Plains and Southwest smokers and nonsmokers. *P* values are based on Mann–Whitney tests. The number of participants included in each analysis was determined by available data. Two Northern Plains participants were excluded because they had no data on cotinine levels.

consistent with the relatively low cotinine levels observed among the smokers. This measure was positively correlated with cotinine levels among smokers ($n = 130$, Spearman $r = .50$, $p < .0001$; Figure 2). This result is consistent with previous findings in other racial and ethnic groups, including Caucasians[31] (Figure 2). CPD accounted for 19.01% of the variation in cotinine levels in our linear regression model (Table 1). Only 84% and 73% of NP smokers had cotinine levels ≥3 and 15 ng/ml, respectively, whereas a relatively high proportion of NP nonsmokers (28% and 19%) had cotinine levels ≥3 and 15 ng/ml (Figure 3). Thus, NP nonsmokers had substantial exposure to tobacco smoke, and NP smokers smoked at relatively low levels.

Association Between Cotinine Levels and Ceremonial Traditional Tobacco Use in the NP

We found no association between ceremonial traditional tobacco use and cotinine levels among NP nonsmokers ($p = .85$; Supplementary Figure 1a), suggesting that traditional tobacco was not a source of nicotine exposure. Nonetheless, cotinine levels were higher among NP smokers who used traditional tobacco than those who did not ($p = .004$; Supplementary Figure 1a). This finding might be explained by the fact that NP smokers who used traditional tobacco also smoked more commercial CPD than NP smokers who did not ($p = .13$; Supplementary Figure 1b).

In linear regression models, CPD ($p < .001$) and traditional tobacco use ($p = .03$) were significant independent predictors of cotinine levels among NP smokers (Table 1). However, a similar linear regression model indicated that traditional tobacco use was not a significant predictor of cotinine levels among NP nonsmokers ($p = .77$; Table 1).

Association Between Cotinine Levels and Passive Smoke Exposure in the NP

Supplementary Table 1 summarizes tribal measures of second-hand smoke exposure. We investigated the possibility that second-hand smoke exposure influenced cotinine levels in the NP population sample using two items in the participant survey: "Are your home's residents or visitors allowed to smoke in your home?" and "Of your closest three friends, how many of them smoke?" NP nonsmokers who allowed smoking in the home and had close friends who smoked had higher cotinine levels than those who did not (*p*

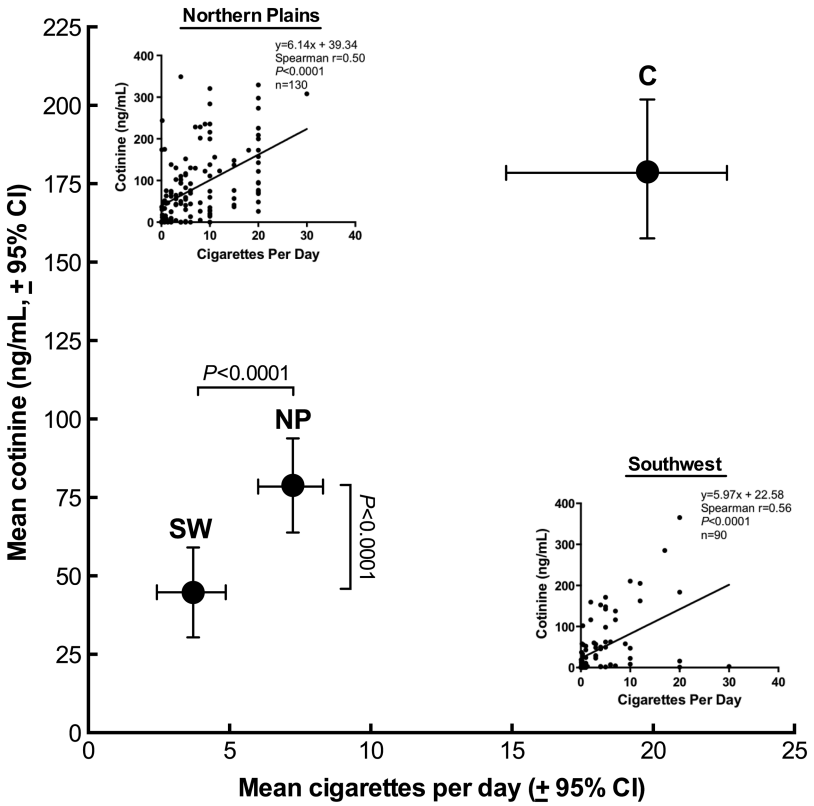

Figure 2. Association of cotinine levels (ng/ml) and number of cigarettes smoked per day among Northern Plains (NP), Southwest (SW), and Caucasian (C) smokers. Inset graphs depict the correlation and linear regression of cotinine and cigarettes smoked per day among NP and SW smokers. *P* and Spearman *r* values are based on Spearman correlation tests or Mann–Whitney tests. The number of participants included in each analysis was determined by available data. Nine NP smokers were excluded because they did not report number of cigarettes smoked per day. All error bars represent 95% confidence intervals (CI) except those referring to cigarettes smoked per day by Caucasians; these error bars represent the interquartile range. Caucasian data were taken from St. Helen et al.[31]

= .01 and $p < 0.0001$, respectively; Supplementary Figures 2a and 3a). Similar relationships between cotinine levels and second-hand smoke exposure were observed among NP smokers, although the differences were not significant ($p = 039$; Supplementary Figures 2a and 3a). NP smokers who allowed smoking in the home and had friends who smoked also consumed more CPD than those who did not ($p = .07$ and $p = .06$, respectively; Supplementary Figures 2b and 3b). Linear regression modeling suggested that, among NP nonsmokers, having friends who smoked was a significant independent predictor of elevated cotinine levels ($p = .02$; Table 1). However, neither indicator of passive smoke exposure had a significant effect on cotinine levels among NP smokers ($p > .45$; Table 1).

Cotinine Levels in the SW

Findings for the SW sample were similar to those for the NP. SW smokers had relatively low cotinine levels (mean ± 95% CI) of 44.8 ± 14.4 ng/ml, yet SW nonsmokers had mean cotinine levels above the more stringent cutpoint (3 ng/ml) for a nonactively-smoking population (9.8 ± 5.8 ng/ml; Figure 1). Although previous studies have reported that SW smokers consumed an average of 7 CPD,[2] SW smokers in the present study reported consuming ~4 CPD, consistent with the low cotinine levels observed. CPD was positively correlated with cotinine levels among smokers ($n = 90$, Spearman $r = .56$, $p < .0001$; Figure 2). Again, the relationship between cotinine and CPD was consistent with findings for other racial and ethnic groups[31] (Figure 2). CPD accounted for 22.0% of the variation in cotinine levels in our linear regression model (Table 1). Approximately 74% and 48% of SW smokers had cotinine levels ≥3 and 15 ng/ml, respectively, while 28% and 11% of SW nonsmokers had cotinine levels >3 and 15 ng/ml, respectively (Figure 3).

Association Between Cotinine Levels and Ceremonial Traditional Tobacco Use in the Southwest

In the SW sample, we found no association between cotinine levels and ceremonial traditional tobacco use among smokers or nonsmokers ($p = .68$ and $p = .53$, respectively; Supplementary Figure 1c). Unlike NP smokers, SW smokers who used traditional tobacco did not smoke more CPD than those who did not ($p = .44$; Supplementary Figure 1d). Linear regression modeling for SW smokers suggested that CPD was a significant independent predictor of cotinine levels ($p < .001$; Table 1), whereas for SW smokers and nonsmokers, traditional tobacco use was not a predictor of cotinine levels ($p = .21$ and $p = .88$, respectively; Table 1).

Association Between Cotinine Levels and Passive Smoke Exposure in the SW

Compared to household smoking in the general US population (17% in all households, 9% in households with no adult smokers, 54% in households with ≥1 adult smoker),[32] NP household smoking consisted of 30% in all households, 22% among nonsmokers, and 45% among smokers, and in the SW smoking in the home occurred in 16% of all households, 12% among nonsmokers, and 29% among smokers. We found no significant association between indicators of

Table 1. Linear regression analyses of cotinine levels (ng/ml) among Northern Plains and Southwestern smokers and nonsmokers

Variable	*B*	95% confidence interval for B	β	Variation in cotinine attributable to each variable[a]	*p*
Northern Plains smokers (n = 129 included in model)[b]					
Cigarettes per day	5.61	3.66 to 7.56	0.45	19.01%	**<.001**
Ceremonial traditional tobacco use	38.51	4.70 to 72.31	0.18	2.99%	**.03**
Residents and/or visitors smoke at home	9.88	−16.41 to 36.17	0.06	0.32%	.46
How many of 3 closest friends smoke (0 vs. 1–3)	30.01	−116.89 to 176.90	0.03	0.10%	.69
Northern Plains nonsmokers (n = 282 included in model)[c]					
Ceremonial traditional tobacco use	−2.04	−24.39 to 20.31	−0.01	0.01%	.86
Residents and/or visitors smoke at home	4.60	−13.12 to 22.32	0.03	0.09%	.61
How many of 3 closest friends smoke (0 vs. 1–3)	22.72	3.95 to 41.48	0.14	1.99%	**.02**
Southwestern smokers (n = 90 included in model)[d]					
Cigarettes per day	5.83	3.55 to 8.11	0.49	22.00%	**<.001**
Ceremonial traditional tobacco use	−21.17	−50.29 to 7.95	-0.14	1.77%	.15
Residents and/or visitors smoke at home	9.63	−21.65 to 40.93	0.06	0.32%	.54
How many of 3 closest friends smoke (0 vs. 1–3)	17.97	−21.70 to 57.63	0.09	0.69%	.37
Southwestern Nonsmokers (n = 120 included in model)[e]					
Ceremonial traditional tobacco use	−1.02	−17.80 to 15.76	-0.01	0.01%	.90
Residents and/or visitors smoke at home	−3.40	−14.64 to 7.85	-0.06	0.30%	.55
How many of 3 closest friends smoke (0 vs. 1–3)	−3.34	−15.17 to 8.49	-0.05	0.27%	.58

Bold values indicate factors that reached significance.

[a]The variation in cotinine levels attributable to each variable is determined by: (Part Correlation)^2 × 100

[b]NP smokers: R^2 = .272, p < .001. R^2 indicates the proportion of variance in cotinine levels (27.2%) explained by this model.

[c]NP non-smokers: R^2 = .022, p > .05. R^2 indicates the proportion of variance in cotinine levels (2.1%) explained by this model.

[d]SW smokers: R^2 = .275, p < .001. R^2 indicates the proportion of variance in cotinine levels (27.5%) explained by this model.

[e]SW non-smokers: R^2 = .006, p > .05. R^2 indicates the proportion of variance in cotinine levels (0.6%) explained by this model

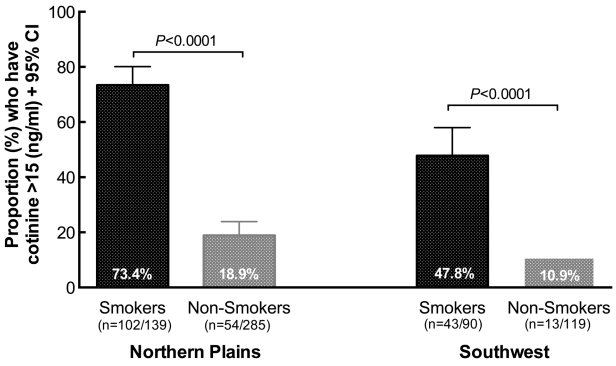

Figure 3. Proportion of Northern Plains and Southwest smokers and nonsmokers who had cotinine levels ≥15 ng/ml. Using 3 ng/ml as the cut point, these values were 84.2% and 28.2% in Northern Plains smokers and nonsmokers (p < .0001), and 74.4% and 27.7% in Southwest smokers and nonsmokers (p < .0001), respectively. The p values are based on chi-square tests. The number of participants included in each analysis was determined by available data. Two Northern Plains participants were excluded because they had no data on cotinine levels.

second-hand smoke exposure and cotinine levels among SW smokers or nonsmokers (smokers, p = .40 and p = .20, respectively; nonsmokers, p =.57 and p = .13, respectively; Supplementary Figures 2c and 3c). SW smokers who allowed smoking in the home and had friends who smoked also smoked more CPD than those who did not (p = .03 and p = .29, respectively; Supplementary Figures 2d and 3d). Measures of passive smoke exposure were not significant independent predictors of cotinine levels among smokers or nonsmokers, as indicated by linear regression analysis (p > .39; Table 1).

Discussion

Tribal Cotinine Levels, Cigarette Consumption, and Smoking Prevalence

We report several key findings related to tobacco smoke exposure among American Indian populations. NP and SW smokers exhibited relatively low average cotinine levels of 81.6 and 44.8 ng/ml, respectively, compared to 165–180 ng/ml among US smokers of Caucasian, African American, and Alaska Native descent.[31,33,34] Among smokers in both populations, cotinine levels were correlated with CPD (NP r = .50, SW r = .56), and CPD accounted for approximately the same degree of variation in cotinine levels (NP 19.01%, SW 22.00%), confirming previous reports of the association between cotinine levels and self-reported CPD.[22] The high prevalence of smoking in the NP tribal population (50%),[1] and the association between CPD and cotinine levels, suggest widespread exposure to nicotine and carcinogens across the NP population. For a nonsmoking population, NP nonsmokers had very high cotinine levels (21.3 ng/ml), with 28% at levels above the more stringent cutpoint (3 ng/ml), and 19% above the more modest cutpoint (15 ng/ml). In contrast, smoking prevalence was considerably lower in the SW tribal population, and SW nonsmokers had lower average cotinine levels (9.8 ng/ml). Nevertheless, this value still exceeded the more stringent cutpoint for second-hand smoke exposure.

Although CPD and cotinine levels were relatively low among smokers in both tribal populations, lung cancer incidence remains high in the NP.[3,4] This incidence is likely related to the high prevalence of smoking in this region[1] as well as to genetic factors.[35] Additionally, even light smokers are at elevated risk for lung cancer[36] as are nonsmokers exposed to second-hand smoke.[14] Therefore, we

suggest that a combination of risk factors contributes to the elevated lung cancer incidence and mortality rates reported for the NP.[3,4]

Traditional Tobacco Use

We found no association between ceremonial traditional tobacco use and variation in cotinine levels among SW smokers, SW nonsmokers, or NP nonsmokers. While a similar proportion of participants in each regional sample used traditional tobacco (NP 14%, SW 20%), such use is common only in the NP.[1] Our null result supports general knowledge of genuine ceremonial traditional tobacco, which does not contain nicotine and thus does not elevate cotinine levels. However, among NP smokers, traditional tobacco users had higher cotinine levels and reported smoking more CPD than nonusers, suggesting that elevated cotinine levels among NP smokers reflect more consumption of commercial cigarettes. Yet we also found that both CPD and traditional tobacco use independently contributed to variation in cotinine levels, indicating that higher commercial tobacco consumption does not fully account for elevated cotinine levels among NP smokers. Therefore, NP smokers may consume commercial and traditional tobacco in combination. This practice can occur in all age-groups, but studies have suggested that AI/AN adolescents may be a vulnerable population, although this was not investigated in the current study. Specifically, Forster et al.[11] demonstrated that 39% of AI/AN adolescents (aged 11–18 years) living in Minneapolis and St. Paul areas reported using commercial tobacco for ceremonial purposes, compared to 24% who use native tobacco for ceremony. Additionally, Unger et al.[13] showed that adolescents report observing both homegrown and commercial tobacco smoking at ceremonies and events. Smoking a mixture of commercial and traditional tobacco results in exposure to nicotine and tobacco-specific carcinogens, elevating the risk of lung cancer and cardiovascular disease, health disparities that are already present in the NP tribal population.[3,4,37,38] Accordingly, we advise community efforts to work with traditional healers and tribal leaders to educate tribal members on risks of blending commercial tobacco products with traditional tobacco.

Second-Hand Smoke Exposure

We observed higher cotinine levels among NP, but not SW, nonsmokers who allowed smoking in the home and had friends who smoked, suggesting that these two factors increase passive exposure to tobacco smoke. These results are consistent with previous findings that nonsmokers with friends who smoke have cotinine levels 1.5 times higher than nonsmokers without such friends.[39] The general absence of smoke-free policies in AI/AN communities mean that smokers and nonsmokers often share the same environments. For example, in the largely urban SW tribal population, participants benefited from smoke-free workplaces, whereas the reservation-based NP tribal populations did not. More second-hand smoke exposure among NP nonsmokers increases the community risk of lung cancer, stroke, and cardiovascular disease while also increasing symptoms of nicotine dependence among younger nonsmokers.[14,40,41] Additionally, second-hand smoke exposure is a negative predictor of smoking cessation among smokers.[42–44]

The same two factors also affected smokers in both regions,such that NP and SW smokers who allowed smoking in the home and had friends who smoked also exhibited higher cotinine levels and smoked more CPD than those who did not. Smoking more CPD likely accounts for these elevated cotinine levels, since CPD was a significant independent predictor of cotinine in our linear regression models, whereas passive smoke exposure was not.

Tailored Smoking Cessation Approaches

Culturally tailored, population-specific smoking cessation interventions may be beneficial in AI/AN communities. Interventions validated in heavy smokers are not necessarily effective for light smokers as observed in two unsuccessful clinical trials of smoking cessation therapy among African Americans light smokers where compliance was also low.[45,46] Thus, improving compliance, which increases the likelihood of successfully quitting,[47,48] and culturally tailoring cessation, such as incorporating AI/AN imagery in intervention materials, disseminating information on traditional tobacco and its spiritual use, and providing pharmacotherapy and counseling by AI/AN practitioners,[49–51] may be approaches which could be tested to enhance smoking cessation among these populations.

Limitations

This study has several limitations. First, there are limitations associated with using cotinine as a biomarker of tobacco smoke exposure. Although cotinine is superior to carbon monoxide as a biomarker of tobacco smoke exposure, primarily because of its specificity to nicotine,[29] measurement of total nicotine equivalents in urine is more sensitive than cotinine, especially among light or intermittent smokers. Additionally, cotinine levels in smokers and nonsmokers are sensitive only to tobacco smoke exposure occurring a few days before measurement. Therefore, our approach assessed only recent tobacco smoke exposure. Despite these concerns, we found a robust correlation between cotinine levels and self-reported CPD, suggesting that cotinine was a useful biomarker of tobacco smoke exposure for study purposes.

Second, although we assessed several measures of second-hand smoke exposure, this was not an exhaustive evaluation. Other potential sources of second-hand exposure should be addressed by future research in AI/AN communities. The scope of this study also did not include the assessment of other tobacco products, aside from commercial cigarettes and traditional tobacco, including noncombustibles. Noncombustibles, such as electronic cigarettes, were not in widespread use at the time of evaluation (2012–2014). Moreover, NP and SW cotinine levels were correlated with CPD, suggesting that tobacco exposure in these populations is associated with cigarette consumption, and not the use of other tobacco products, such as noncombustibles; however, the observed correlations do not preclude the association of cotinine levels with the use of other products. Additionally, we did not independently determine the composition of the forms of ceremonial traditional tobacco used by study participants. This study also did not assess the impact of body mass index on cotinine, which has previously exhibited a weak negative correlation.[52–54] Furthermore, sample sizes limited our statistical power. Analyses indicated that the direction of effects and the association between cotinine levels and tobacco smoke exposure were similar in the NP and SW samples. However, specific findings differed in significance, likely because of a lack of analytic power resulting from insufficient sample sizes, particularly in the SW. Additionally, weighting was not conducted to correct for possible clustering due to respondent-driven sampling. Finally, participants in the current study were individuals who had participated in an earlier large epidemiological study that was designed to characterize the lifestyle, dietary, environmental, and cultural factors associated with cancer in adult American Indians. It is possible that being involved in the EARTH

study increased participants' awareness of, and education on, the health risks of smoking, contributing to reduced smoking quantities, and thus lower than anticipated measures of CPD in the current study (NP 7 vs. 13, and SW 4 vs. 7 CPD) compared to EARTH.

Conclusions

Although overall cotinine levels were lower among these two tribal populations compared to smokers in other US ethnic and racial groups, smoking prevalence[1] and second-hand smoke exposure were high in both populations, especially the NP. Given the causal relationship of smoking and secondhand smoke inhalation with lung cancer and cardiovascular disease, our results suggest that these are at-risk tribal populations. In both tribal populations, our findings support the implementation of stricter indoor smoking bans and culturally tailored tobacco cessation programs.

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Disclosure Statement

RFT has consulted for Apotex and McNeil. The remaining authors declare no conflicts of interest.

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