Addiction

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Substance use and periodontal disease among Australian Aboriginal young adults

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ABSTRACT

Aim To investigate the effects of tobacco, marijuana, alcohol and petrol sniffing on periodontal disease among Australian Aboriginal young adults. **Design** Cross-sectional nested within a long-standing prospective longitudinal study. **Setting** Aboriginal communities in Australia's Northern Territory. **Participants** Members of the Aboriginal Birth Cohort study who were recruited from birth between January 1987 and March 1990 at the Royal Darwin Hospital, Northern Territory, Australia. Data were from wave III, when the mean age of participants was 18 years. **Measurements** Clinical dental examination and self-report questionnaire. **Findings** Of 425 participants with complete data, 26.6% had moderate/severe periodontal disease. There was elevated risk of periodontal disease associated with tobacco [prevalence ratio (PR) = 1.59, 95% CI = 1.06–2.40], marijuana (PR = 1.44, 95% CI = 1.05–1.97) and petrol sniffing (PR = 1.83, 95% CI = 1.08–3.11), but not alcohol (PR = 0.92, 95% CI = 0.67–1.27). Stratified analysis showed that the effect of marijuana persisted among tobacco users (PR = 1.47, 95% CI 1.03–2.11). It was not possible to isolate an independent effect of petrol sniffing because all petrol sniffers used both marijuana and tobacco, although among smokers of both substances, petrol sniffing was associated with an 11.8% increased prevalence of periodontal disease. **Conclusions** This is the first time that substance use has been linked with periodontal disease in a young Australian Aboriginal adult population, and the first time that petrol sniffing has been linked with periodontal disease in any population. The role of substance use in periodontal disease among this, and other, marginalized groups warrants further investigation.

Keywords Australian Aboriginal, periodontal disease, substance use, young adults.

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INTRODUCTION

At an international level little is known about the periodontal health of many marginalized groups, which include the socially disadvantaged, ethnic minorities, those living in rural-remote locations and substance users. It could be argued that many Australian Aboriginal young adults meet all four of these criteria with regard to marginalization, being a group that faces incredible social disadvantage at a population level [1], representing only 2.4% of the total Australian population in the 2006 Census [2]. Around two-thirds living in locations classified as 'rural' or 'remote' [2] and many being at risk of misusing substances such as tobacco [3], marijuana [4], alcohol [5] and petrol (gasoline) [6].

Periodontal disease is a chronic infectious condition caused by Gram-negative bacteria with a persistent host inflammatory reaction [7]. It has been suggested that life-course factors may influence periodontal disease [8], along with oral health-related self-care behaviours [9] and social determinants [10]. Periodontal disease can have a marked impact on quality of life [11] and has been associated with a range of conditions, including cardiovascular disease, stroke, diabetes and pulmonary disease [12].

There is undisputed evidence that tobacco smoking is a causative risk factor for periodontal disease [13–15]. The association occurs because the chemicals inhaled interfere with the body's ability to repair damaged tissues [16]. Because tobacco is a vasoconstrictor it cuts down on

the blood supply, thus limiting the flow of oxygen and nutrients to the tissue. Nicotine is additionally toxic to fibroblasts, which are responsible for manufacturing new connective tissue. The less fibroblasts work, the more rapid the progression of periodontal disease [17]. Marijuana use has also been linked with periodontal disease, with Thomson and colleagues [18] reporting that, after controlling for tobacco smoking and other important confounders in a prospective cohort of young adults, regular exposure to cannabis smoke was associated strongly with the prevalence and incidence of periodontal attachment loss by age 32 years. Alcohol misuse has been linked additionally with periodontal disease, with Khocht and colleagues [19] investigating the effects of alcohol and cocaine misuse on periodontal status in a group of alcohol-dependent patients. Their results suggested that persistent alcohol abuse increased periodontal disease development by heightening the loss of periodontal attachment through recession of gingival margins. No significant associations were found between cocaine use and attachment loss. We were unable to locate literature that reported any associations between petrol sniffing and periodontal disease. It is not unreasonable to speculate that such an association might exist, however, perhaps occurring systemically through the adverse effects of the toxic constituents of the inhalant on immune function and the inflammatory response, as well as through reducing peripheral blood flow. To the best of our knowledge, there have been no reports of the impact of substance use on the periodontal health of Indigenous Australians.

The aim of this study was to determine if substance misuse would be a risk indicator for periodontal disease among a cohort of Australian Aboriginal young adults, taking into account the possible confounding effects of socio-demographics, perinatal factors and oral health-related behaviours in an Australian Aboriginal context. Although conducted in an Australian setting, the research question has relevance among other marginalized groups around the world who are potentially at risk of periodontal disease through their substance use behaviours.

MATERIALS AND METHODS

Background

Participants were members of the Aboriginal Birth Cohort (ABC) study, a longitudinal investigation of health and behaviour in a birth cohort of Australian Aboriginals. Babies were eligible for enrolment if they were live-born singletons delivered at the Royal Darwin Hospital, Northern Territory, Australia between January 1987 and March 1990 to a mother recorded as Aboriginal. Of the mothers found and interviewed at baseline, 686 agreed to participate, accounting for 55% of potential recruits. There were no mean birth weight or gender ratio differences between those recruited and not recruited [20].

Follow-ups were carried out at mean ages 4, 11 and—most recently—18 years. The Human Research Ethics Committee of the Northern Territory Department of Health and Community Services and Menzies School of Health Research (including an Aboriginal subcommittee with absolute right of veto) granted ethics approval for each assessment phase. Study members gave informed consent before participating.

Periodontal measures

Dental examinations for periodontal assessment were conducted at the third follow-up (mean participant age = 18 years). Examinations were conducted by two calibrated dentists, both unaware of participants' substance use frequency or socio-economic position. Two sites (mesiobuccal and buccal) per tooth, excluding third molars, were examined. Probing depth (the distance from the probe tip to the gingival margin) and gingival recession (the distance from the gingival margin to the amelo–cemental junction) were recorded by a disposable probe with 2–3 mm markings. The combined attachment loss for each site was computed by summing gingival recession and probing depth. Mid-buccal measurements for molars were made at the mid-point of the mesial root. All measurements were rounded down to the nearest whole millimetre. The US Centres for Disease Control and Prevention and the American Academy of Periodontology definitions were used to describe moderate and severe periodontal disease, whereby moderate periodontal disease was defined as the presence of either two sites between adjacent teeth with 4 mm+ attachment loss, or at least two such sites with 5 mm+ pockets. Severe periodontal disease was classified as having at least two sites between adjacent teeth with 6 mm+ attachment loss and with at least one 5 mm+ pocket [21]. Repeat examinations for examiner reliability were not possible due to logistical and time constraints imposed by the study's multi-disciplinary nature.

Substance use measures

At mean age 18 years, study members were also asked to take part in a social and emotional wellbeing interview that included information pertaining to petrol sniffing, marijuana, tobacco and alcohol use. Participants were asked 'how much petrol do you sniff', 'how much marijuana do you smoke', 'how much tobacco do you smoke' and 'how much alcohol do you drink', with response options including (1) 'never or only tried it once'; (2) 'used to sniff (or smoke or drink), but not any more'; and (3) 'still sniff (or smoke or drink) sometimes'. For the pur-

poses of this analysis, response options (2) and (3) were combined to represent a 'case' of consuming petrol, marijuana, tobacco or alcohol, respectively. Option 2 was included in the case definition because of the young age of participants, meaning that any past substance use was likely to have been relatively recent. Interviewers were aware of the sensitive nature of the items (marijuana use being illegal, for example) and took the utmost care when asking questions, for example conducting interviews in a private settings, assuring participants that their answers would not 'get them in trouble' and having interviewers whose gender matched, where possible, that of the participant. One of the two dentists asked the substance use questions to female participants if the team psychologist was unavailable. In the instances where the examining dentist asked the substance use questions, it was always after the dental examinations and dental questionnaire data had been collected.

Covariates

Demographic

Age, sex and location were included. Location was dichotomized into 'regional', which included participants living in the three regional centres included in the study (with approximate population sizes of 121 000, 27 000 and 9800, respectively) [22] and 'rural/remote', which included participants living outside the regional jurisdictions.

Social

Education was defined as 'qualification status', with participant responses dichotomized into 'not currently studying' or 'currently studying'. Occupation was defined as 'welfare' (i.e. unemployment or various government welfare programmes) or 'job' (i.e. employment). Because conventional social determinant measures do not have the same meaning in an Australian Aboriginal context, particularly in remote communities where employment is scarce and education opportunities limited, the socioeconomic position of participants was also assessed using household size and car ownership. Household size was assessed by the question 'how many people stayed in your house last night?', while car ownership was measured by the question 'does someone in your house own a car?'. Household size was dichotomized into response options of 'four or less' and 'five or more'.

Perinatal factors

Perinatal factors included participant weight at birth, whether or not mother smoked tobacco at time of participants' birth and whether or not mother drank alcohol at time of participants' birth.

Oral health-related behaviours

Oral health-related behaviours included dental service utilization and toothbrush ownership and use.

Data analytical approach

Univariate and bivariate distributions of moderate or severe periodontal disease were determined, with stratified analysis undertaken to assess the effects of combinations of substance use risk indicators on the prevalence of periodontal disease. The potential for effect measure modification was also determined and differences between strata evaluated by examining the overlap of confidence intervals; there were no variables identified as effect measure modifiers. Correlation tests confirmed the existence of weak associations between items in a given group (Pearson's correlation coefficient range 0.1–0.4), with no variables needing to be excluded due to collinearity. The high prevalence of moderate or severe periodontal disease meant that odds ratios were poor indicators of relative frequency, so prevalence ratios (PR) were determined using Poisson regression modelling [23]. Poisson regression analysis was used to derive adjusted estimates for the dependent variable. Exposure variables were classified into substance use, demographic, socio-economic, perinatal, dental service utilization and oral health-related behaviour groups.

Adjusted PR were considered statistically significant when P values derived from the Wald statistic were ≤ 0.05. The final regression models for moderate or severe periodontal disease were constructed by removing covariates one at a time according to P value size. The degree of attenuation was calculated as the crude PR minus the adjusted PR, divided by the crude PR and multiplied by 100. Data were analysed using Intercooled STATA 8.

RESULTS

Of the original 686 ABC study participants, 618 were traced at a mean age of 18 years, of whom 27 were known to have died. Of the 591 available for examination, 121 participants were not examined but only 11 refused outright, and the remainder were not seen because of logistic reasons relating to poor weather, mobility of participants and single participants living in very remote locations. Basic anthropomorphic measures at a mean age of 18 years were obtained for 468 participants; a response rate of 73%. The numbers of participants undergoing procedures varied because some were seen by an incomplete study team, some had disabilities that prevented all measurements and others refused some procedures. Of the 468 for whom vital status was obtained, 442 agreed to be dentally examined and

provided complete information in a self-report dental questionnaire, which was 95% of the total number of participants examined at mean age 18 years, and 69% of those recruited at birth who were still alive. Substance use information was available for 425 (96%) of those individuals, and all subsequent analyses were limited to those 425 participants. There were no significant periodontal differences between those included and not included in the analysis.

Just over 26% of participants had moderate or severe periodontal disease (Table 1). The proportion of males was 48.2% and the age range was 16–20 years. Without adjusting for other risk indicators, there was an elevated risk of periodontal disease associated with tobacco, marijuana and petrol sniffing, although not with alcohol. Risk of periodontal disease was greater for males than females, and for those who did not own a toothbrush compared to their toothbrush-owning counterparts.

In stratified analysis, marijuana use was associated with a 1.5-fold increase in the prevalence of periodontal disease among tobacco smokers and the effect was statistically significant (Fig. 1). Among non-users of tobacco there were only 13 marijuana users, none of whom had periodontal disease. In contrast, the prevalence of periodontal disease was 20.9% among non-users of tobacco who did not use marijuana, although this was statistically non-significant. The small number of marijuana users in this stratum of participants who did not smoke tobacco contributed to the extremely wide 95% confidence interval (CI) for the PR, highlighting the unreliability of this result. It was not possible to determine if petrol sniffing was an independent risk indicator for periodontal disease, because all those who reported sniffing petrol also reported using both marijuana and tobacco. However, among participants who smoked both tobacco and marijuana, the prevalence of periodontal disease among petrol sniffers was 47.1% compared with 33.8% among non-petrol sniffers (PR = 1.39; 95% CI 0.80–2.43: results not tabulated).

Two models were used in multivariate modelling (Table 2). Model 1 was run for all participants and included risk indicators significant at a bivariate level. Being male and non-ownership of a toothbrush remained statistically significant in the final model, with the adjusted PR being attenuated by 9.3 and 11.1%, respectively, from the unadjusted estimates. In model 2, only participants who smoked tobacco were included. In this model, being male and non-ownership of a toothbrush also remained statistically significant, with the adjusted PR decreasing by 27.0 and 3.9%, respectively, from the unadjusted estimates. Although statistical significance was not reached, tobacco was close to being statistically significant in model 1 (PR = 1.39; 95% CI 0.92–2.08) and marijuana was close to being statistically significant in model 2 (PR 1.26; 95% CI 0.89–1.80).

DISCUSSION

This study set out to determine if substance use, as measured by use of tobacco, marijuana, alcohol and petrol, was a risk indicator for periodontal disease among a cohort of Australian Aboriginal young adults. At a bivariate level, tobacco, marijuana and petrol use were associated strongly with periodontal disease prevalence. In stratified analysis, marijuana was found to have an effect on periodontal health among tobacco smokers, but this ceased to be significant after adjusting for the more traditional periodontal disease risk indicators in multivariate modelling. While the findings support previous research indicating negative associations between the abuse of tobacco [15] and marijuana [18] with periodontal health, the link between petrol sniffing and periodontal disease has not been shown before. Although conducted in an Australian context, the findings have relevance for the international community.

It is important to bear in mind that the study was not designed at the outset to examine the relationship between substance use and periodontal disease. *Post-hoc* sample size calculations indicated that there was sufficient power to detect a PR of at least 1.75 for the effect of tobacco smoking on periodontal disease, while for marijuana the minimal detectable PR was 1.65; so it was unsurprising that, after adjusting for other risk indicators in the multivariate models, the smaller PR for tobacco smoking and marijuana were not statistically significant. Ideally we should have had a larger cohort, but this was not possible. In fact, this study represents the largest cohort of an Australian Indigenous population that has ever been assembled with this, in turn, being the largest study of periodontal disease in an Indigenous population. Despite the lack of statistical power, the findings are still useful and give a clear indication for future investigations. Participants in this study were young adults with relatively few years of exposure to the substances studied here. Probably a longer period of follow-up would yield greater power to unearth a statistically significant association if use of these substances were to persist, although that epidemiological discovery would come at considerable cost to the health of individuals in the study. Furthermore, it would be incorrect to dismiss the effect of these substances, and to presume that periodontal disease among this group can be rectified by addressing tooth brushing and male-specific risks. Instead, we believe that these findings call for action now to help young Indigenous adults reduce their reliance upon these substances and to tackle the upstream factors that create an environment of substance misuse, both in the

Table 1 Total counts, prevalences and prevalence ratios of Aboriginal Birth Cohort (ABC) participants with moderate/severe periodontal disease by substance use, socio-demographic, life-course and oral health-related behaviour variables (95% confidence interval).

	No. of people	*Prevalence of moderate or severe periodontal disease*	*Prevalence ratio*
Total	425	26.6 (22.4–30.8)	
Substance use			
Tobacco			
Yes	302	29.8 (24.7–34.9)	1.59 (1.06–2.40)*
No	123	18.7 (11.8–25.6)	
Marijuana			
Yes	169	32.5 (25.5–39.5)	1.44 (1.05–1.97)*
No	256	22.7 (17.6–27.8)	
Petrol sniffing			
Yes	17	47.1 (23.5–70.7)	1.83 (1.08–3.11)*
No	408	25.7 (21.5–29.9)	
Alcohol			
Yes	193	25.4 (19.3–31.5)	0.92 (0.67–1.27)
No	232	27.6 (21.9–33.3)	
Demographic			
Sex			
Male	205	35.1 (28.6–41.6)	1.88 (1.35–2.63)*
Female	220	18.6 (13.5–23.7)	
Age group (years)			
16–18	176	26.7 (20.2–33.2)	1.01 (0.73–1.39)
19–20	249	26.5 (21.0–32.0)	
Residential location			
Rural/remote	335	28.4 (23.6–33.2)	1.42 (0.91–2.22)
Regional	90	20.0 (11.8–28.2)	
Socio-economic			
Highest qualification			
Not currently studying	290	29.6 (24.4–34.8)	1.18 (0.85–1.63)
Currently studying	135	25.2 (17.9–32.5)	
Household income			
Welfare	373	27.1 (22.6–31.6)	1.17 (0.69–1.98)
Job	52	23.1 (11.7–34.5)	
Household size			
Five or more people	343	28.3 (23.6–33.0)	1.32 (0.74–2.35)
Four or less people	82	19.5 (11.0–28.0)	
Someone in house own car?			
Yes	233	27.9 (22.2–33.6)	1.11 (0.81–1.54)
No	192	25.0 (18.9–31.1)	
Perinatal			
Birth weight			
Greater than 2500 g	361	26.7 (22.2–31.2)	1.00 (0.64–1.56)
Less than 2500 g	64	26.6 (15.8–37.4)	
Mother smoked tobacco at birth?			
Yes	226	28.1 (22.3–33.9)	1.12 (0.81–1.53)
No	199	25.2 (19.2–31.2)	
Mother drank alcohol at birth?			
Yes	62	21.0 (10.9–31.1)	0.76 (0.46–1.27)
No	363	27.6 (23.0–32.2)	
Dental service utilisation			
Visited dentist before?			
No	27	37.0 (18.9–55.1)	1.43 (0.85–2.41)
Yes	398	25.9 (21.6–30.2)	
If yes, when was last visit?			
Aged 12 years or less	239	27.2 (21.6–32.8)	1.15 (0.81–1.62)
Aged 13 years or more	160	23.8 (17.2–30.4)	

Table 1 *Cont.*

	No. of people	*Prevalence of moderate or severe periodontal disease*	*Prevalence ratio*
Dental behaviour			
Toothbrush ownership			
No	127	42.5 (33.9–51.1)	2.15 (1.58–2.91)*
Yes	298	19.8 (15.3–24.3)	
If yes, did brush teeth yesterday?			
No	84	23.8 (14.7–32.9)	1.29 (0.80–2.07)
Yes	216	18.5 (13.3–23.7)	
If yes, what age when started to brush?			
When had big teeth	125	19.2 (12.3–26.1)	1.00 (0.62–1.62)
When had little teeth	162	19.1 (13.1–25.1)	

*$P < 0.05$.

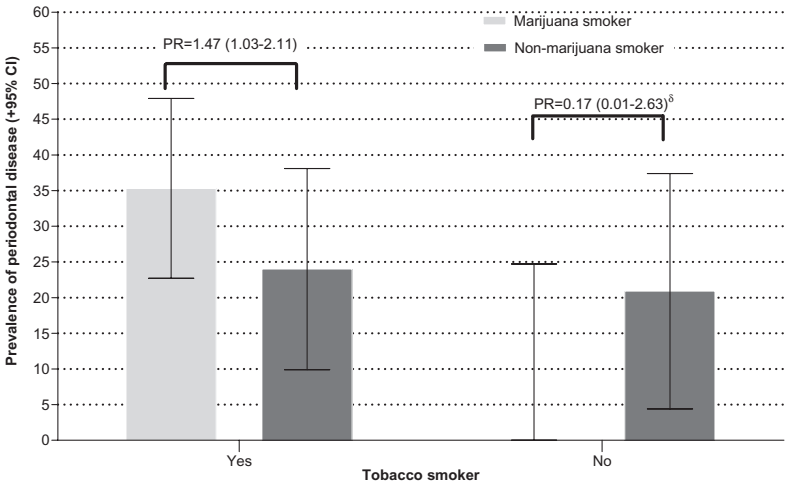

Figure 1 Periodontal disease prevalence by tobacco and marijuana smoking*. *Breslow–Day test for homogeneity; $P = 0.022$; δestimate of prevalence ratio (PR) used the logit estimation, based on calculating 0.5 in a cell containing 0; CI: confidence interval

Australian context and among other marginalized populations at risk of substance misuse at a global level.

It is important to examine other limitations of the investigation. Although the study is longitudinal in design, important periodontal disease indicators were collected only in the most recent phase. True causality can therefore not be determined in this cross-sectional analysis, although this should be possible in future data collection waves of the ABC study. The self-report nature of the socio-demographic, substance use and oral health behaviour-related information may have led to an underestimation of these factors. However, we took great care with interviewing and, in any case, non-differential under-reporting would have resulted in more conservative estimates of the socio-demographic, substance use and oral health behaviour-related associations with periodontal disease, meaning that our findings are unlikely to be spurious. Although the generalizability of the findings to the source population has not been established, Aboriginal people in Australia's National Survey of Adult Oral Health had markedly higher levels of periodontal disease than their non-Aboriginal counterparts [24]. Among the study's strengths are the high follow-up rates in each wave of the investigation, meaning that the prospective determination of substance exposure (and the length of time over which the exposure data are collected), and the use of data on periodontal disease incidence as well as prevalence, should be possible in future data collection waves. Although some of the classifications of demographic and social determinant data (education and employment, for example) were fairly crude, given the strength of the associations, the evidence seems sufficient to draw public attention to the potential role of substance use and periodontal disease among this and other marginalized groups at an international level, to stimulate further research and to call for appropriate public health measures.

The study's demonstration of an association between substance misuse and periodontal disease among young Aboriginal Australian adults supports the literature indicating that substance misuse is detrimental to periodontal tissues, and that public health measures to reduce the

Table 2 Adjusted prevalence ratios (PR) for moderate or severe periodontal disease among Aboriginal Birth Cohort (ABC) study participants.

	Model 1 All participants; n = 425	*Model 2 Tobacco smokers; n = 302*
	Adjusted PR (95% CI)	*Adjusted PR (95% CI)*
Demographic		
Sex		
Male	1.72 (1.23–2.40)*	1.48 (1.03–2.14)*
Female	Ref.	Ref.
Dental behaviour		
Toothbrush ownership		
No	1.93 (1.42–2.63)*	2.07 (1.47–2.92)*
Yes	Ref.	Ref.
Substance use		
Tobacco		
Yes	1.39 (0.92–2.08)	Not in model
No	Ref.	
Marijuana		
Yes	Not in model	1.26 (0.89–1.80)
No		Ref.

*$P < 0.05$. CI: confidence interval.

prevalence of substance misuse may, among other advantages, have periodontal benefits, for both this population and other marginalized groups at a global level. We were unable to find other data with which to compare the findings, but determining whether the association exists in other populations should be a priority for periodontal epidemiological research. The prevalence of periodontal disease in this marginalized population was high; more than three times that of the national estimates for 15–34-year-olds [24]. Given the associations of periodontal disease with diabetes and cardiovascular risk, and the amenability of both traditional and emerging risk factors, this is of public health importance both in Australia and elsewhere.

Since the 1980s, substance misuse has been prevalent among other Indigenous young adult populations including Native American Indians [25], Inuit Canadians [26] and New Zealand Maoris [27]. Our findings thus have relevance for other Indigenous populations at a global level. Although definitively establishing the periodontal effects of substance misuse should await confirmation in other populations, health promoters and practitioners would be prudent to take steps to raise awareness of the probability that those who misuse substances regularly may, among the other myriad health problems, be additionally causing damage to their periodontal tissues.

Declarations of interest

None.

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