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Smoking-attributable periodontal disease in the Australian adult population

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Abstract

Background: The extent to which periodontitis is attributable to smoking in Australia has not been examined.

Objectives: To investigate the smoking–periodontitis relationship and to estimate the public health impact of smoking on periodontitis in Australia.

Material and Methods: The National Survey of Adult Oral Health 2004–2006 collected nationally representative oral epidemiologic data for the Australian adult population. Examiners measured probing pocket depth (PPD) and gingival recession at three sites per tooth to compute clinical attachment level (CAL). Moderate-severe cases were defined as having: $\geqslant 2$ interproximal sites (not on same tooth) with $\geqslant 4$ mm CAL or with $\geqslant 5$ mm PPD. Smoking status was defined as never-, former- or current-smoker. Current-smokers were further classified into light-, moderate- or heavy-smoker using calculated pack-years. Age, sex and socioeconomic position were examined as potential confounders.

Results: Twenty-three per cent were former-smokers and 15% were current-smokers. Prevalence of periodontitis was 23%. In unadjusted analyses, former- and current-smokers had significantly higher periodontitis prevalence than never-smokers. Relative to non-smokers, adjusted prevalence ratios (95% confidence interval) for periodontitis were as follows: former-smokers: 1.22 (1.03–1.46), moderate-smokers: 1.63 (1.16–2.30); and heavy-smokers: 1.64 (1.27–2.12). The population attributable fraction of smoking for moderate-severe periodontitis was 32% (equivalent to 700,000 adults).

Conclusion: Smoking has a significant impact on periodontal health of the Australian adults.

Key words: Australia; epidemiology; periodontitis; population attributable fraction; smoking

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Smoking is a major risk factor for periodontitis (US Surgeon General's Report 2004, AAP 2005, Borrell & Papapanou 2005). Its effect on prevalence, extent and severity of periodontitis has been the topic of numerous epidemiological studies (Grossi et al. 1994, Haber 1994, Linden & Mullally 1994, Grossi et al. 1995, Martinez-Canut et al. 1995, Gelskey 1999, Obeid & Bercy 2000, Tomar & Asma 2000, Albandar & Rams 2002, Amarasena et al. 2002, Do et al. 2003, Hujoel et al. 2003, Van Dyke & Sheilesh 2005, Ojima et al. 2006, Thomson et al. 2007). Recent systematic reviews (Tonetti 1998, Bergstrom 2006) further strengthened the evidence that smoking is a risk factor for periodontitis. Taken together, these studies concur in finding smoking to be a causative risk factor for periodontitis. Less consistent is the reported

Conflict of interest and source of funding statement

The authors declare that they have no conflict of interests.

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magnitude of the hazard associated with smoking. Reported odds ratios (ORs) range from 2 to 14 reflecting variation in measurement of exposure and case definitions of the disease (Tonetti 1998, Bergstrom 2006). Furthermore, the observed magnitude of the effect of smoking on periodontal status can vary among populations due to differences in the distribution of risk factors for the disease. The population-impact of smoking on periodontitis also varies according to the frequency of exposure to tobacco smoke in populations.

Most studies of the impact of smoking on periodontal health have used relatively restricted sampling designs, such as patient groups, that limit their generalizability to the general population. Those studies provide ''proof-of-principle'' support for a potential aetiological association between smoking and periodontitis. Other studies have explored the relationship between smoking and periodontal disease using representative samples of population groups (Beck et al. 1994, Albandar et al. 2000, Amarasena et al. 2002, Do et al. 2003) and a small number of studies have used nationally representative samples (Tomar & Asma 2000, Ojima et al. 2006). Several studies used a longitudinal cohort design (Beck et al. 1994, Bergstrom et al. 2000, Thomson et al. 2007). In addition to providing evidence about risk factors for periodontitis, a few studies generated estimates of the population-impact of smoking on periodontal health (Tomar & Asma 2000, Thomson et al. 2007).

Criteria needed to draw causal inferences from observational studies include evidence of a biological gradient between degree of exposure and risk of disease, strength and consistency of association (Beck 1998, US Surgeon General's Report 2004). Evidence of observational studies needs to be evaluated against those criteria of causality.

This study aims to investigate the relationship between smoking and periodontitis, and to estimate the public health impact of smoking on periodontitis in the Australian adult population.

Material and Methods

Data for this study were from the 2004–2006 Australian National Survey of Adult Oral Health (NSAOH) (Slade et al. 2007). The target population was community-dwelling people aged 15 years or more. Study participants were selected at random using a multistage, stratified random sample selection procedure using a sampling frame compiled from listed telephone numbers. Information was collected by a computer-assisted telephone interview, an oral epidemiological examination and a self-administered questionnaire. The telephone interview collected information on sociodemographic characteristics and a number of health-related factors including smoking status. Dentate interviewees were invited to participate in an oral epidemiological examination that included periodontal assessment. A medical history check precluded those people for whom a periodontal examination was contraindicated. The examination was conducted under standardized clinical conditions by one of 30 trained and calibrated dentists. Two light sources were used throughout: an intra-oral battery-operated mirror light and standard dental clinical halogen light. No radiographs were taken. Following the examination, a 16-page questionnaire was mailed to all examined people, with up to three reminder letters. The questionnaire included questions about history of tobacco smoking. Full details of methods and participation in the study, together with descriptive findings, have been reported elsewhere (Slade et al. 2007). The examination protocol can be viewed at http://www.arcpoh.adelaide.edu.au/project/distribution/nsaoh_pdf%20files/NSAOH_ExamProtocol_v8.pdf.

The Human Research Ethics Committee of the University of Adelaide and the Ethics Committee of the Australian Institute of Health and Welfare reviewed and approved the project. Signed, informed consent was obtained from all participants.

Sociodemographic data

Participants were asked questions about their sex, age, and socioeconomic position. Educational attainment was classified as having nine or fewer *versus* 10 or more years of formal education. Two indicators of dental care affordability were used: private dental insurance status and the possession of a government concession card that entitled the holders to publicly subsidised health care. The former is expensive hence prohibitive for low-income families; the latter is means-tested.

Smoking data

During the telephone interview, adults were asked, ''Which of the following best describes your smoking status (includes cigarettes, cigars and pipes)''. Three response options were current smoker, former smoker or never smoked. In the questionnaire, current smokers were asked to estimate their daily number of cigarettes usually smoked and the number of years they had smoked. Former smokers indicated the number of years that they had smoked as well as time since they stopped smoking.

For current smokers, pack-years were calculated using a standard formula based on 20 cigarettes per pack (Grossi et al. 1994).

$$\text{pack-year} = \frac{\text{Number of cigarettes per day} \times \text{Number of years smoked}}{20}$$

Pack-years was used to classify current smokers into three groups: light-smokers >0 and $\leqslant 4.45$ pack-years; moderate-smokers: >4.45 and $\leqslant 15$ pack-years; and heavy-smokers: >15 pack-years.

Periodontal status

Periodontal status was evaluated at the clinical examination using a method modified from the examination manual used in the US National Health and Nutrition Examination Survey (NHANES) 2001 (CDC 2001). Periodontal pocket depth and gingival recession were measured using a periodontal probe with 2-mm bandings (Hu-Friedy PCP2, Hu Friedy, Chicago, IL, USA). A variation from the NHANES protocol was that measurements were made for all teeth except third molars, while only two random quadrants were examined in the NHANES. Three sites were assessed per tooth: mesio-buccal, mid-buccal and disto-buccal. All fractional millimetre measurements were rounded down to the nearest whole millimetre. Gingival recession (REC) was defined as the distance from the cemento-enamel junction (CEJ) to the free gingival margin. When the CEJ was subgingival, the value was recorded as negative; otherwise it was positive or zero. Probing pocket depth (PPD) was defined as the distance from the free gingival margin to the bottom of the periodontal crevice/pocket. Clinical attachment level (CAL) was calculated as sum of PPD and REC for each site during the data management stage.

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The extent of sites with periodontal disease was calculated using two measures: the proportion of sites that had PPD $\geqslant$4 mm and the proportion of sites that had CAL $\geqslant$4 mm. Population-level estimates of the proportion were calculated using the ratio procedure in the SUDAAN statistical computing program. This procedure estimates a population ratio with adjustment for complex sampling design of the survey.

Using criteria developed by a consensus panel convened by the US Centers for Disease Control and Prevention and the American Academy of Periodontology, moderate-severe cases of periodontitis were defined as people with: $\geqslant$2 interproximal sites (not on same tooth) with $\geqslant$4 mm CAL or $\geqslant$2 interproximal sites (not on same tooth) with $\geqslant$5 mm PPD (Page & Eke 2007). Severe cases of periodontitis was defined as people who had $\geqslant$2 interproximal sites (not on same tooth) with $\geqslant$6 mm CAL and $\geqslant$1 interproximal sites with $\geqslant$5 mm PPD (Page & Eke 2007).

Data analysis

The association of smoking status and other putative risk factors for periodontal status was examined in bivariate and age-stratified contingency analysis where the prevalence ratio (PR) estimate and its corresponding 95% confidence interval (CI) were calculated for each category of risk indicator relative to a reference group. PRs were calculated in preference to ORs because the prevalence of periodontal disease was relatively high (23%). Under these conditions, ORs are not a good approximation of relative difference in prevalence of the disease between the exposure groups. Multivariate regression models were created to compute adjusted estimates of the effect of smoking, controlling for other risk factors found to be significant in bivariate analyses. Because age was strongly associated with periodontitis and smoking, additional age-group-specific multivariate models were constructed to investigate potential modification by age of the smoking–periodontitis relationship. Multivariate modelling was undertaken using the *loglink* procedure in SUDAAN with robust variance estimation to estimate PRs and associated 95% CIs. This procedure fits Poisson's regression to cluster-correlated count data in forms other than proportions and has been shown to adequately estimate adjusted PRs (Barros & Hirakata 2003). In order to quantify the potential impact of smoking on population periodontal health, the population attributable fraction (PAF) was calculated. The PAF estimates the proportion of cases of a disease attributed to the exposure of interest in the population. The PAF is a function of both the strength of the association and the prevalence of the exposure in the population. We estimated PAF using multivariate logistic regression modelling (Bruzzi et al. 1985), a method used previously to estimate impact of smoking on periodontitis (Tomar & Asma 2000, Thomson et al. 2007).

For all analyses, SAS-callable SUDAAN software was used to adjust for the complex sampling design to produce nationally representative population estimates.

Results

There were a total of 3161 participants in the study. Twenty-three per cent of the population were former smokers and another 15% were current smokers (Table 1). The prevalence of current and former smoking was higher among males than among females. Compared with younger age groups, older adults were more likely to be former smokers but less likely to be current smokers. People who were eligible for public dental care were more likely to be current smokers than those who were ineligible, and the dentally uninsured people were more likely to be current smokers than the insured. However, the frequency of smoking did not differ by geographical location or educational attainment.

A total of 1.0% of all periodontal sites had PPD of 4+ mm while 3.5% of all periodontal sites had CAL exceeding that threshold (Table 2). Smoking status was significantly associated with both measures of extent of periodontitis. Heavy-smokers had 3.3% of sites with deep periodontal pocket as compared with only 0.7% among people who had never smoked. Almost 10% of periodontal sites among the heavy-smokers had loss of periodontal support of 4+ mm compared with fewer than 3% of sites among people who had never smoked.

Twenty-three per cent of the Australian adult population was classified as having moderate or severe periodontitis (Table 3). In unadjusted analysis, smoking was significantly associated with prevalence of the disease. Prevalence was greater by a factor of 1.6 for both former-smokers and moderate-smokers relative to people who had never smoked.

Table 1. Variation in smoking status among groups classified according to putative risk indicators

Risk indicator	Never smoker weighted % (95% CI)	Former smoker weighted % (95% CI)	Current smoker weighted % (95% CI)
All people	61.9 (59.5–64.4)	23.4 (21.3–25.4)	14.7 (12.8–16.6
Sex			
Female, $n = 1965$	66.9 (64.2–69.7)	20.5 (18.1–22.9)	12.6 (10.7–14.5)
Male, $n = 1196$	56.7 (52.6–60.7)	26.4 (23.0–29.8)	17.0 (13.7–20.3)
Age group (years)			
15–34, $n = 598$	72.8 (67.7–77.8)	12.3 (8.1–18.6)	15.0 (11.3–18.6)
35–54, $n = 1331$	56.1 (52.9–59.7)	26.2 (23.1–29.3)	17.7 (15.0–20.4)
55–64, $n = 692$	52.7 (47.6–57.8)	33.7 (29.0–38.5)	13.6 (8.9–18.2)
65+, $n = 540$	60.9 (55.5–66.4)	34.3 (29.0–39.5)	4.8 (2.7–6.9)
Residency			
Non-capital, $n = 1168$	62.2 (59.2–65.2)	23.0 (20.5–25.5)	14.7 (12.5–17.1)
Capital cities, $n = 1993$	61.5 (57.0–65.9)	24.0 (20.4–27.5)	14.6 (11.4–17.8)
Eligible for public dental care			
Eligible, $n = 914$	57.4 (52.3–62.5)	23.4 (20.9–25.8)	13.2 (11.2–15.3)
Ineligible, $n = 2243$	63.4 (60.5–66.3)	23.4 (19.6–27.3)	19.2 (15.1–23.2)
Private dental insurance			
Insured, $n = 1583$	64.2 (60.5–67.9)	25.0 (22.0–27.9)	10.9 (8.2–13.6)
Uninsured, $n = 1552$	59.0 (55.7–62.4)	22.1 (19.3–24.9)	18.9 (16.2–21.7)
School education			
Year 9 or less, $n = 348$	62.1 (54.4–69.8)	23.6 (21.4–25.8)	14.5 (12.5–16.5)
Year 10 or higher, $n = 2806$	61.9 (59.3–69.5)	21.4 (15.1–27.7)	16.5 (10.6–22.4)

CI: confidence intervals. Within columns, sub-groups are significantly different if 95% CI does not overlap. Analysis based on 3161 participants.

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Table 2. Extent of sites with PPD or CAL of 4+ mm among all people and groups classified by smoking status

	Extent of sites with PPD 4+ mm		Extent of sites with CAL 4+ mm	
	weighted %	95% CI	weighted %	95% CI
All people	1.0	0.8–1.2	3.5	3.2–3.9
Never smokers, $n = 2206$	0.7	0.5–0.8	2.6	2.3–2.9
Former smokers, $n = 842$	1.0	0.8–1.3	5.1	4.2–6.0
Light smokers, $n = 125$	1.1	0.3–1.9	2.3	1.1–3.5
Moderate smokers, $n = 141$	3.2	0.7–5.7	4.8	3.0–6.6
Heavy smokers, $n = 234$	3.3	1.7–4.8	9.7	6.8–12.6

Within columns, subgroups are significantly different when their 95% CI does not overlap.
CAL, clinical attachment level; PPD, probing pocket depth; CI, confidence intervals.

Table 3. Prevalence of moderate-severe periodontitis and prevalence ratios of association with risk indicators

Risk indicator	Moderate-severe periodontitis		
	prevalence (weighted %)	un-adjusted PR (95% CI)	adjusted PR (95% CI)
All people	22.9		
Smoking status			
Never smokers, $n = 2206$	18.5	1	1
Former smokers, $n = 842$	31.0	1.68 (1.40–2.00)	1.22 (1.03–1.46)
Light smokers, $n = 125$	15.6	0.84 (0.48–1.46)	1.17 (0.70–1.95)
Moderate smokers, $n = 141$	29.1	1.57 (1.10–2.24)	1.63 (1.16–2.30)
Heavy smokers, $n = 234$	46.6	2.52 (1.96–3.25)	1.64 (1.27–2.12)
Sex			
Female, $n = 1965$	18.2	1	1
Male, $n = 1196$	28.0	1.55 (1.32–1.81)	1.46 (1.27–1.67)
Age group (years)			
15–34, $n = 598$	7.5	1	1
35–54, $n = 1331$	22.2	2.95 (2.00–4.37)	2.76 (1.87–4.07)
55–64, $n = 692$	40.0	5.31 (3.60–7.83)	4.88 (3.30–7.21)
65+, $n = 540$	52.0	6.90 (4.78–9.98)	5.88 (3.95–8.76)
Residency			
Capital cities, $n = 1993$	22.1	1	1
Other areas, $n = 1168$	24.5	1.03 (0.97–1.09)	1.01 (0.86–1.19)
Eligible for public dental care			
No, $n = 2243$	19.6	1	1
Yes, $n = 914$	33.3	1.70 (1.44–2.00)	1.12 (0.95–1.33)
Private dental insurance			
Yes, $n = 1583$	18.8	1	1
No, $n = 1552$	27.9	1.49 (1.18–1.89)	1.44 (1.23–1.68)
School education			
Year 10 or higher, $n = 2806$	21.4	1	1
Year 9 or less, $n = 348$	37.7	1.76 (1.44–2.16)	1.19 (0.97–1.47)

Case of moderate-severe periodontitis defined as having: $\geqslant 2$ interproximal sites (not on same tooth) with $\geqslant 4$ mm CAL or $\geqslant 2$ interproximal sites with $\geqslant 5$ mm PPD (not on same tooth).
Prevalence: weighted estimates.
Un-adjusted PR: prevalence ratio estimated in bivariate analysis among $n = 3161$ participants.
Adjusted PR: prevalence ratio estimated in multivariate regression model adjusted for other variables among $n = 3161$ participants.
CI: confidence intervals. Significantly different from the reference if 95% CI does not include unity.
CAL, clinical attachment level; PPD, probing pocket depth.

Among current smokers, there was a positive association between pack-years of smoking and periodontitis prevalence. Almost half the heavy-smokers were periodontitis cases, while fewer than one-fifth of those who had never smoked were periodontitis cases. Prevalence of periodontitis was significantly higher among males than females and positively associated with age with over half of the 65+ year age group having the disease. Lower education attainment, lack of dental insurance and being eligible for public dental care were significantly related to greater prevalence of periodontitis.

In the multivariate analysis that adjusted for age and other risk indicators shown in Table 3, smoking persisted as a statistically significant risk indicator for moderate-severe periodontitis. Relative to people who had never smoked, the prevalence of periodontitis was greater by a factor of 1.2 among former-smokers and by a factor of 1.6 among moderate- and heavy-smokers (Table 3). This effect of smoking was significant after adjusting for other potential risk factors. Sex, age, smoking and dental insurance status were also significantly associated with moderate-severe periodontitis in this multivariate model.

Prevalence of severe periodontitis was 2.2% (Table 4) and was strongly associated with smoking status, age, eligibility for public dental care and insurance status in unadjusted analysis. There was a positive association between the prevalence of severe periodontitis with the severity of smoking status. The association between smoking and severe periodontitis persisted after adjustment for other factors in the multivariate model.

In age-stratified analysis, the effect of smoking on moderate-severe periodontitis was most obvious among 15–34-year-olds after adjusting for other factors (Table 5). In that age group, former- or heavy-smokers had significantly higher prevalence compared with those who never smoked. There was a clear gradient of smoking effect on moderate-severe periodontitis among current smokers. A biological gradient was also observed among 35–54-year-olds where former-smokers and heavy-smokers were significantly more likely to have periodontitis compared with never-smokers. The prevalence of periodontitis was high in the 55+-year-old group, among whom only moderate-smokers had significantly higher prevalence of periodontitis than the people who never smoked.

The estimated PAF of smoking (classified as both current and former smokers) was 32% (95% CI: 14–41) for moderate-severe periodontitis and 56% (95% CI: 28–68) for severe periodontitis (Table 6). This was equivalent to 700,000 (95% CI: 305,000–893,000) cases of moderate-severe periodontitis that were attributable to cigarette smoking in the Australian adult population.

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More than half the severe cases [109,000 (95% CI: 54,000–132,000)] would have been prevented if smoking was eliminated in the population. The PAF estimates of current smokers were higher than that of former smokers for periodontitis defined by either case definition.

Table 4. Prevalence of severe periodontitis and prevalence ratios of association with risk indicators

Risk indicator	Severe periodontitis		
	prevalence (weighted %)	un-adjusted PR (95% CI)	adjusted PR (95% CI)
All people	2.2		
Smoking status			
Never smokers, $n = 2206$	1.3	1	1
Former smokers, $n = 842$	3.0	2.27 (1.28–4.02)	1.60 (0.93–2.74)
Light smokers, $n = 125$	1.6	1.18 (0.33–4.21)	1.76 (0.49–6.41)
Moderate smokers, $n = 141$	5.8	4.34 (2.04–9.24)	4.72 (2.16–10.35)
Heavy smokers, $n = 234$	7.6	5.75 (2.42–13.70)	3.31 (1.39–7.88)
Sex			
Female, $n = 1965$	1.8	1	1
Male, $n = 1196$	2.6	1.42 (0.86–2.35)	1.23 (0.75–2.01)
Age group (years)			
15–34, $n = 598$	0.4	1	1
35–54, $n = 1331$	2.0	4.70 (1.19–18.63)	4.18 (1.07–16.33)
55–64, $n = 692$	5.3	12.56 (2.87–55.00)	11.08 (2.54–48.46)
65+, $n = 540$	4.6	10.91 (2.53–47.05)	8.86 (1.98–39.58)
Residency			
Capital cities, $n = 1993$	2.2	1	1
Other areas, $n = 1168$	2.2	0.96 (0.54–1.73)	0.83 (0.45–1.45)
Eligible for public dental care			
No, $n = 2243$	1.7	1	1
Yes, $n = 914$	3.8	2.20 (1.32–3.64)	1.33 (0.78–2.27)
Private dental insurance			
Yes, $n = 1583$	1.5	1	1
No, $n = 1552$	3.0	1.98 (1.24–3.13)	1.84 (1.11–3.07)
School education			
Year 10 or higher, $n = 2806$	2.0	1	1
Year 9 or less, $n = 348$	4.6	2.35 (1.35–4.10)	1.42 (0.74–2.73)

Case of severe periodontitis defined as having: $\geqslant 2$ interproximal sites (not on same tooth) with $\geqslant 6$ mm CAL and $\geqslant 1$ interproximal sites with PPD 5+ mm.

Un-adjusted PR: prevalence ratio estimated in bivariate analysis among $n = 3161$ participants.

Adjusted PR: prevalence ratio estimated in multivariate regression model adjusted for other variables among $n = 3161$ participants.

CI: confidence intervals. Significantly different from the reference if 95% CI does not include unity.

PR, prevalence ratios; CAL, clinical attachment level; PPD, probing pocket depth.

Discussion

Overview

This study is one of few to examine the relationship between smoking and periodontitis in a nationally representative population sample. The large and representative sample strengthened the validity of finding that smoking is a significant risk indicator for periodontitis. In addition to the strength and statistical significance of the association, there are two findings that support the likelihood of a causal role for the relationship. First, there was a clear evidence of a biological gradient within smokers and this was most apparent in the youngest and the middle-aged groups. We believe this is important, because there is much less tooth loss in younger age groups, a phenomenon that can mask the true history of periodontitis, which can be measured only on teeth that are present in the mouth. Second, the case definition of periodontitis used here is similar to other case definitions that are based on CAL (Beck et al. 1990, Tonetti & Claffey 2005). CAL represents a cumulative history of the disease, with the consequence that measuring its prevalence is analogous to measuring lifetime incidence. Virtually all smokers begin smoking when they are teenagers or young adults (Hill et al. 1999) – a stage of life when it is exceedingly unlikely that they would have developed periodontitis at the level used in the case definition used in our study. Hence, there are good grounds to believe that the smoking habit was initiated before development of periodontitis, which may be considered to be surrogate evidence of a temporal sequence between smoking and periodontitis. Furthermore,

Table 5. Adjusted prevalence ratios for moderate-severe periodontitis associated with smoking in three age groups

	15–34 years		35–54 years		55+ years	
	prevalence (w %)	PR (95% CI)	prevalence (w %)	PR (95% CI)	prevalence (w %)	PR (95% CI)
All people	7.5		22.2		45.4	
Never smokers	5.5	1	17.3	1	42.7	1
Former smokers	15.4	2.57 (1.18–5.61)	24.4	1.44 (1.07–1.96)	46.9	1.01 (0.86–1.19)
Light smokers	7.6	1.37 (0.42–4.53)	23.6	1.26 (0.61–2.59)	60.5	1.25 (0.74–2.12)
Moderate smokers	16.8	2.32 (0.90–5.95)	27.5	1.39 (0.82–2.35)	74.6	1.88 (1.35–2.62)
Heavy smokers	25.4	3.38 (1.23–9.25)	47.6	2.12 (1.44–3.13)	52.5	1.09 (0.81–1.48)

Case of moderate-severe periodontitis defined as having: $\geqslant 2$ interproximal sites (not on same tooth) with $\geqslant 4$ mm CAL or $\geqslant 2$ interproximal sites with $\geqslant 5$ mm PPD (not on same tooth).

PR: prevalence ratios relative to people who had never smoked; adjusted for sex, residency, eligibility for public dental care, insurance status and school attainment.

CI: confidence intervals. Significantly different from the reference if 95% CI do not include unity.

W%, weighted per cent of people; CAL, clinical attachment level; PPD, probing pocket depth; PR, prevalence ratios.

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Table 6. Estimated population attributable fraction and attributable cases of smoking for periodontitis in the Australian adult population

	Population attributable fraction (PAF) (95% CI)		Attributable cases, thousands people (95% CI)	
	moderate to severe periodontitis	severe periodontitis	moderate to severe periodontitis	severe periodontitis
Current smokers	21.3 (15.3–26.0)	31.7 (20.9–36.3)	460 (331–562)	62 (41–71)
Former smokers	11.1 (2.5–19.1)	24.5 (9.2–32.8)	240 (54–413)	48 (18–64)
All smokers	32.4 (14.1–41.3)	56.2 (28.0–68.1)	700 (305–893)	109 (54–132)

Case of periodontitis

Moderate to severe ⩾2 interproximal sites (not on same tooth) with ⩾4 mm CAL or ⩾2 interproximal sites with ⩾5 mm PPD (not on same tooth).

Severe ⩾2 interproximal sites (not on same tooth) with ⩾6 mm CAL and ⩾1 interproximal sites with PPD 5+ mm.

PAF (per cent of cases in the population attributable to exposure) of smoking was estimated based on logistic regression models for the cases of periodontitis, adjusted for sex, residency, eligibility for public dental care, insurance status and school attainment.

Attributable cases estimates (rounded to nearest thousand) were based on calculated PAF and the estimated total number of cases of periodontitis in the Australian adult population (*n* moderate to severe cases = 2,161,265; *n* severe cases = 194,295).

CI, confidence interval of estimates; CAL, clinical attachment level; PPD, probing pocket depth.

the smoking effect on periodontitis must be considered in both initiation of periodontitis (when temporal sequence is important) and progression of periodontitis (when temporal sequence may not be necessary) (Borrell & Papapanou 2005).

We used PR as a measure of effect because in cross-sectional research this better reflects incidence rate ratio, when the disease of interest is common (Greenland 1987, Osborn & Cattaruzza 1995, Thompson et al. 1998). However, most studies examining the smoking and periodontitis relationship have reported OR. Therefore, direct comparison with those studies was not readily possible. However, our estimates of ORs were comparable to those reported in the dental literature (data not shown).

We used the CDC case definition because our examination protocol developed in 2002/2003 was based on the NHANES protocol. Furthermore, the CDC case definition was used to report the prevalence of periodontitis in the NSAOH official report published in 2007. Further analysis using this case definition will expand the usefulness of the study findings. Also, except for the severe level, the two case definitions are conceptually similar by using clinical attachment level at interproximal sites. The two case definitions differ in the threshold in mm: 4+ mm for the CDC and 3+ mm for the European Workshop's case definition. Despite that, the CDC case definition was designed for use in population surveillance that was more applicable to the NSAOH.

Our case definition reflects both historical periodontal destruction (CAL) and more recent periodontal disease activity (PPD) (Borrell & Papapanou 2005, Page & Eke 2007). We considered accumulation of the disease with age as an important characteristic in evaluating the effect of smoking, which was also assessed using questions about lifetime exposure. This creates concordance between the lifetime nature of the exposure and an outcome that was measured across a lifetime. Our comparative analysis using the European Workshop's case definition (Tonetti & Claffey 2005) revealed similar patterns of association between smoking and periodontitis in our study sample. For example, the PRs of level two cases, defined as having 30+% of teeth with 5+ mm CAL (Tonetti & Claffey 2005), among the former- and current-smokers relative to those who never smoked were 3.6 and 5.8, respectively. Other studies have used different case definitions (Grossi et al. 1994, Gelskey et al. 1998, Tomar & Asma 2000, Thomson et al. 2007) that may obscure a direct comparison. However, regardless of case definition used, a strong and consistent effect of smoking was observed in all studies.

Cigarette smoking among Australian adults

In this study of Australians aged 15 years or more 15% were current smokers, which was lower than 21% reported for people aged 18 years or more in the National Health Survey (ABS 2001). The distribution of pack-years observed in NSAOH was similar to that reported in two other studies using the same formula (Grossi et al. 1994, Paidi et al. 1999).

Smoking as a risk indicator for periodontitis in Australian adults

This cross-sectional study provided strong evidence of smoking as an independent risk indicator for periodontitis in the Australian adult population. Our findings build on a body of evidence that smoking is causally associated with periodontitis.

Taken together, the evidence suggests that eliminating exposure to cigarettes would reduce the prevalence of periodontitis in this population. We estimated that elimination of cigarette smoking would reduce the number of moderate-severe cases by one-third, equivalent to approximately 700,000 adults. A large proportion of periodontitis cases in the population could be prevented if smoking was effectively targeted. To achieve this, anti-smoking campaigns smoking policies would be needed to prevent the uptake of smoking and to help current smokers to quit.

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Clinical Relevance

Scientific rationale for the study: The impact of smoking on the periodontal health of a population must be evaluated to fully understand its effect and to guide clinicians and policy makers appropriately.

Principal findings: The study provided strong evidence of smoking as an independent risk indicator for periodontitis in the Australian adult population. One-third of the moderate-severe periodontitis cases in the population could be prevented if smoking was eliminated.

Practical implications: Smoking cessation would attenuate the risk of periodontitis. Clinicians should urge potential smokers to avoid the habit and encourage smoking cessation among smokers.

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