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The natural history of periodontal attachment loss during the third and fourth decades of life

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Abstract

Aim: To describe changes in the occurrence of periodontal attachment loss (AL) through ages 26, 32 and 38 in a complete birth cohort.

Materials and Methods: Systematic periodontal examinations conducted at ages 26, 32 and 38 in a longstanding New Zealand cohort study (N = 1037). Periodontitis extent data were used to assign participants to periodontitis trajectories using group-based trajectory analysis.

Results: Eight hundred and thirty-one individuals were periodontally examined at all three ages; the prevalence and extent of AL increased as the cohort aged. Between 26 and 32, one in nine participants had 1+ sites showing new or progressing AL; that proportion almost doubled between ages 32 and 38. Four periodontitis trajectory groups were identified, comprising 55.2%, 31.5%, 10.7% and 2.5% of the cohort; these were termed the "Very low", "Low", "Moderately increasing" and "Markedly increasing" trajectory groups respectively. Those who had smoked tobacco at all ages from 15 through 38 were at higher risk of being in the "Moderately increasing" or "Markedly increasing" trajectory groups. There was a similar risk gradient for those who were in the highest 20% of cannabis usage.

Conclusions: Periodontitis commences relatively early in adulthood, and its progression accelerates with age, particularly among smokers.

Key words: cannabis; longitudinal studies; periodontal diseases; risk factors; smoking

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Although periodontitis is largely painless, individuals with advanced disease may suffer the direct effects on their quality of life (Cunha-Cruz et al. 2007; Needleman et al. 2004). Its greatest burden may be only now becoming apparent as evidence mounts for associations with inflammation-driven systemic disease (D'Aiuto et al. 2005; Cullinan et al. 2009; Kuo et al. 2008). Periodontitis may also be more prevalent than is generally assumed (Krustrup and Petersen, 2006; AIHW Dental Statistics and Research Unit, 2007; Bourgeois et al. 2007; Holtfreter et al. 2009; Ministry of Health, 2010; Eke et al. 2012).

Much research has focused on periodontitis prevalence in middle-aged and older populations, often with an emphasis on complications such as tooth loss. Putative risk factors that may be part of the causal chain have also been identified, along with more peripheral risk markers. However, understanding of

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the natural history of the disorder in young adults as they move through their third and fourth decades towards middle age is lacking. The scarcity of comprehensive longitudinal data gathered for this age group means that it remains currently unclear just how early in the life course the pathway to poor periodontal health begins. A shortage of prospective cohort studies of population-based samples means that little is known of its natural history through the life course.

A notable exception is the Dunedin Multidisciplinary Health and Development Study (DMHDS) (Silva and Stanton, 1996), a prospective study of a complete birth cohort. We have previously reported both descriptive (at ages 26 and 32) and longitudinal findings (changes from age 26 to 32) on periodontitis occurrence in that cohort (Thomson et al. 2000; Thomson et al. 2006); the findings have underlined the importance of risk factors such as dental restorations (Broadbent et al. 2006), tobacco smoking (Thomson et al. 2007), cannabis smoking (Thomson et al. 2008) and persistent poor oral hygiene (Broadbent et al. 2011). The recently completed age-38 periodontal assessments afford unprecedented data from three ages during a critical life-course epoch about which little is known. Efficiently describing longitudinal changes in periodontal attachment loss (AL) over three ages is a challenge; we are not aware of it having been carried out in any previous epidemiological study. The aim of this study was to describe changes in the occurrence of periodontal AL, and to evaluate risk factors for unfavourable AL progression, through ages 26, 32 and 38.

Materials and Methods

The DMHDS is a longitudinal study of a cohort of children who were born at the Queen Mary Hospital, Dunedin, New Zealand between 1st April 1972 and 31st March 1973 (Silva and Stanton, 1996). Perinatal data were obtained and the sample for the longitudinal study was defined at age 3 years. This initially comprised 1037 children assessed within a month of their third birthdays and again at ages 5, 7, 9, 11, 13, 15, 18, 21, 26, 32 and 38 (when 961 or 95% of the living cohort were assessed). Over 90% of the cohort self-identified as being of European origin.

The various assessments (for example, oral health, mental health, cardiovascular health) are presented as standardized modules in counterbalanced order, with each conducted by a different examiner kept blind to all other Study data. The Otago Research Ethics Committee granted ethics approval for each assessment phase. Study members gave informed consent before participating.

Socio-economic status (SES) was measured in childhood using standard New Zealand occupationally-based indices (Elley and Irving, 1985; Irving and Elley, 1977) which employ a 6-category classification (where, for example, a doctor scores "1" and a labourer scores "6"). Participants were classified (using occupation at age 32) as having low (groups 5 and 6), medium (groups 3 and 4) or high (groups 1 and 2) SES. The childhood SES measure we used here is based on both parents, and is the mean of the SES from the assessments at ages 0, 3, 5, 7, 9, 11, 13 and 15.

Clinical assessments

Periodontal examinations were conducted at ages 26, 32 and 38, with half-mouth examinations at age 26, but full-mouth examinations at ages 32 and 38. Third molars and implants were not included in the periodontal examinations. At age 26, dental examinations were conducted by three examiners who had been previously calibrated and who examined 85%, 10% and 5% (respectively) of the cohort. Periodontal measurements were made in only two quadrants (quadrants 1 and 3 for those whose study ID number was odd; quadrants two and four for those with an even ID number; the mix of odd and even ID numbers was approximately 50:50) because of time constraints. Three sites (mesiobuccal, buccal and distolingual) per tooth were examined, and gingival recession (GR; the distance in millimetres from the cemento-enamel junction to the gingival margin) and probing depth (PD; the distance from the gingival margin to the base of the pocket) were recorded, using an NIDR probe (the Hu-Friedy PCP-2; Rotterdam, the Netherlands). Midbuccal measurements for molars were made at the midpoint of the mesial root. All measurements were rounded down to the nearest whole millimetre at the time of recording. Where the gingival margin was situated more than 1 mm coronally to the cemento-enamel junction, a negative value for GR was recorded. Periodontal measurements were not conducted on Study members who reported a history of cardiac valvular anomalies or rheumatic fever. At ages 32 and 38, the clinical procedures were identical, except that a full-mouth examination was now possible. Two examiners were used at age 32, and they examined 53% and 47% of Study members respectively; the second examiner was the same one who had conducted the bulk of the examinations at age 26. At age 38, three examiners were used; these comprised the two who had undertaken the age-32 examinations; they undertook 58% and 39% of the age-38 examinations, respectively, and the third examiner completed 3% of them.

Tobacco smoking status was determined using data from ages 15, 18, 21, 26, 32 and 38. At age 15, it was determined with the question "Have you smoked in the last 4 weeks?". At age 18, we used "Have you been smoking every day for the last month?". At ages 21, 26, 32 and 38, we used "Have you smoked every day for 1 month or more of the previous 12 months?". Those data were used to allocate participants to smoking exposure groups based on their smoking status (smoker or non-smoker) at each of those ages.

Chronic cannabis smoking during the life course was determined by asking participants at each of ages 18, 21, 26, 32 and 38 how many times they had used cannabis in the previous year. The exposure measure employed here uses the mean of the usage over the four ages to be 32 (for which Cronbach's alpha was 0.79). This enabled identification of participants in the highest 20% of exposure (with a mean of 41 or more occasions of cannabis use during the previous year; described in Thomson et al. 2008). The usual method of

smoking cannabis in New Zealand is not to mix it with tobacco.

Examiner reliability

Three examiners were used at age 38, and they examined 354, 31 and 527 Study members respectively. Replicate periodontal examinations were not possible during the assessments because of time constraints (due to the busy assessment day undergone by Study members). However, replicate examinations were conducted on a separate sample of adults during the Dunedin Study's age-38 data collection, giving data for 672 periodontal sites measured by the three examiners twice each. Intra-examiner reliability was high (accepting 1 mm variation in measurements as being normal measurement error). Intra-examiner reliability coefficients for absolute agreement of the site-level periodontal measurements (three sites per tooth) pooled for the three examiners (with the individual examiner intra-examiner reliability coefficients in brackets) were as follows: 0.95 (0.99, 0.92 and 0.94) for GR; 0.73 (0.73, 0.69 and 0.80) for PD; and 0.71 (0.71, 0.68 and 0.79) for combined attachment loss (CAL), while the coefficient for the prevalence of 1+ sites with 4+ mm CAL was 0.75 (0.99, 0.74 and 0.97). However, a 1 mm difference within and between examiners is normal measurement error in the measurement of GR, PD and CAL (Goodson 1986). When considering intra-examiner reliability pooled for all three examiners, only 2.6% of measurements differed by more than 1 mm (2.2%, 2.5% and 3.0% for examiners 1, 2 and 3 respectively). No measurements differed by more than 2 mm for any of the three examiners. Inter-examiner reliability was also high. Of all the sites assessed for CAL, examiner one differed from examiner two by more than 1 mm in 5.4% of measurements and by more than 2 mm in only 0.8% of measurements. Examiner one differed from examiner three by more than 1 mm in 3.2% of measurements, and by more than 2 mm in only one measurement (0.1%). Examiner two differed from examiner three by more than 1 mm in 5.0% of measurements, and by more than 2 mm in only 0.7% of measurements. Our examiner reliability was similar to that reported by Goodson (1986). At age 32, two examiners were used (W.M.T. and J.M.B.), and they examined 437 and 495 Study members respectively; examiner reliability data for the age-32 examinations have been reported previously (Thomson et al. 2006) and were found to be acceptable. At age 26, 84% of participants were examined by W.M.T. Calibration data for those age-26 assessments were not collected.

Data analysis and case definitions

Analyses were undertaken in SPSS (version 20; IBM Corporation, Armonk, NY, USA). For each age, the CAL for each site was computed by summing the GR and PD measurements. The changes in CAL between 26 and 32 (and between 32 and 38) were determined for each site by subtracting the later age's CAL from that of the earlier one.

A previously non-diseased site (one with 3 mm or less CAL) that increased by at least 3 mm to result in a periodontal pocket with CAL of at least 4 mm was classified as having incident disease, whereas one showing progression was defined as having CAL of 4+ mm at age 26 that had increased by at least 3 mm by age 32 (or CAL of 4+ mm at age 32 that had increased by at least 3 mm by age 38). An incident case was someone with 1+ sites experiencing incident disease or progression.

Differences in proportions were tested for statistical significance using Chi-square tests for cross-sectional comparisons, and McNemar tests for longitudinal ones. Similarly, differences in means were tested for statistical significance using (as appropriate) Mann–Whitney *U*-tests (or Kruskal–Wallis tests) or Wilcoxon tests, as appropriate. A p value <0.05 was considered statistically significant.

Group-based trajectory modelling – a useful tool for characterizing developmental trajectories (Nagin and Odgers, 2010) – was used to identify periodontitis experience trajectories. Group-based trajectory modelling assumes that individual differences in trajectories can be summarized by a finite set of different polynomial functions of age; these group trajectories are identified through maximum likelihood estimation. We have used it previously with dental caries experience (Broadbent et al. 2008) and plaque scores (Broadbent et al. 2011). The response variable (the extent of 4 + mm CAL; that is, the percentage of measured sites with 4+ mm CAL) was modelled using the zero-inflated Poisson distribution, and, through maximum likelihood estimation, the model of best fit was identified as a four-group one (using Stata IC 10.0 for Windows; StataCorp, College Station, Tx, USA). The likelihood that individual Study members were members of these groups was estimated through group-based trajectory modelling (Jones et al. 2001). Group membership (the dependent variable) was modelled using a multinomial logistic regression model jointly with the estimation of the trajectories themselves. To avoid classification bias, the relationship of the independent variables with trajectory group membership was estimated based on the probability that an individual with a specific profile (such as smoker, or low SES) belongs to a specific trajectory group (Nagin, 2005).

Results

Of the 961 participants assessed at age 38, periodontal data were available for 895 (93.1%), of whom 444 (49.6%) were female. Over 90% of those examined clinically at age 38 had been periodontally examined at all three ages (Table 1), with no systematic sex differences in the proportions examined. There were significant SES differences, with higher proportions of low-SES participants not periodontally examined at ages 26 and 38, and across all three ages (at least where childhood SES is concerned). There were no significant differences at any age in the proportion who were current tobacco smokers. Subsequent analyses in this report are restricted to the 831 individuals who were periodontally examined at all three ages.

The number of teeth remaining by age 38 ranged from 6 to 32, and the mean number of teeth present was 27.5 (SD, 3.0). Some 259 (31.2%) had one or more teeth missing due to caries, and 815 (98.1%)

Table 1. Attrition analysis: numbers of the 916 dentally examined at 38 who were periodontally examined at ages 26, 32 and 38, by sex and childhood socio-economic status (SES; brackets contain column percentages unless otherwise indicated)

	Periodontally examined at							
	Age 26		Age 32		Age 38		All 3 ages	
	Yes	No	Yes	No	Yes	No	Yes	No
Number*	861 (94.0)	55 (6.0)	868 (94.8)	48 (5.2)	895 (97.7)	21 (2.3)	831 (90.7)	85 (9.3)
Sex								
Male	433 (50.3)	27 (49.1)	442 (50.9)	18 (37.5)	451 (50.4)	9 (42.9)	423 (50.9)	37 (43.5)
Female	428 (49.7)	28 (50.9)	426 (49.1)	30 (62.5)	444 (49.6)	12 (57.1)	408 (49.1)	48 (56.5)
Childhood SES								
High	148 (17.3)	6 (11.1)†	148 (17.1)	6 (12.5)	153 (17.2)	1 (4.8)†	144 (17.4)	10 (11.9)†
Medium	551 (64.3)	29 (53.7)	551 (63.8)	29 (60.4)	569 (63.9)	11 (52.1)	532 (64.3)	48 (57.1)
Low	158 (18.4)	19 (35.2)	164 (19.0)	13 (27.1)	168 (18.9)	9 (42.9)	151 (18.3)	26 (31.0)
Adult SES (age 32)								
High	151 (17.6)	8 (15.7)	151 (17.4)	8 (19.0)	159 (17.9)	0 (0.0)†	148 (17.8)	11 (13.9)
Medium	460 (53.6)	22 (43.1)	464 (53.5)	18 (42.5)	472 (53.1)	10 (47.6)	445 (53.5)	37 (46.8)
Low	248 (28.9)	21 (41.2)	253 (29.1)	16 (38.1)	258 (29.0)	11 (52.4)	238 (28.6)	31 (39.3)
Smoking status at that age								
Non-smoker	536 (62.3)	31 (56.4)	592 (68.2)	33 (68.8)	670 (74.9)	12 (57.1)	660 (79.4)	63 (74.1)
Current smoker	325 (37.7)	24 (43.6)	276 (31.8)	15 (31.2)	225 (25.1)	9 (42.9)	171 (20.6)	22 (25.9)

*Row percentages
†$P < 0.05$ (χ^2 test)

had retained 21 or more teeth, with 514 (61.8%) having 28 or more teeth. Implants were present in 14 individuals (1.6%), of whom 11 had one implant, two persons had two and one had three.

Summary data on periodontal status are presented by age in Table 2, determined using both half-mouth (for all ages, in order to enable longitudinal comparisons) and full-mouth analyses (for ages 32 and 38 only). The mean number of periodontally-assessed sites decreased with age. The prevalence and extent of CAL increased as the cohort aged, whereas the severity (mean CAL) did not show such a marked change.

Between ages 26 and 32, about one in nine participants were incident cases (that is, they had 1+ sites showing new or progressing AL); that proportion almost doubled between ages 32 and 38 (Table 3). Similarly, the mean proportion of sites with CAL increases was greater between 32 and 38 than it was between 26 and 32, and the mean CAL increased more during that period. Overall, one in nine sites (on average) showed a CAL increase by 3+ mm between ages 26 and 32. Without exception, the changes observed between ages 32 and 38 were greater than those between 26 and 32. Changes were greater among molars and anterior teeth (canines and incisors) than premolars.

During both periods, there was considerably more new AL than progression (Table 4). The proportion of the cohort experiencing new AL between ages 32 and 38 was almost twice that experiencing it between 26 and 32; the proportion experiencing progression increased fivefold, but those rates were relatively low in absolute terms. Almost without exception, the changes observed between ages 32 and 38 were greater than those between 26 and 32; the exception was the observed progression in the molars, where the proportion of sites showing it between ages 32 and 38 was smaller than that observed between 26 and 32.

The outcome of the growth trajectory modelling using the extent of

Table 2. Summary data on periodontal status at ages 26, 32 and 38 (half-mouth and full-mouth data; all differences are statistically significant)

	Age 26	Age 32	Age 38
Half-mouth data			
Number of sites assessed			
Mean (SD*; range)	40.4 (2.5; 27–42)	40.0 (2.9; 21–42)	39.4 (3.9; 9–42)
Prevalence: number of participants with 1+ sites with			
4 + mm CAL (%)	144 (17.3)	173 (20.8)	281 (33.8)
5 + mm CAL (%)	26 (3.1)	64 (7.7)	141 (17.0)
6 + mm CAL (%)	7 (0.8)	27 (3.2)	73 (8.8)
Extent: mean percentage of sites with			
4 + mm CAL (SD; range)	0.9 (3.2; 0–37)	1.8 (5.7; 0–71)	4.8 (13.0; 0–100)
5 + mm CAL (SD; range)	0.2 (1.2; 0–25)	0.5 (13.0; 0–50)	2.2 (9.3; 0–100)
Severity: mean CAL (SD; range)	1.5 (0.3; 0.5–3.1)	1.4 (0.4; 0.5–4.7)	1.6 (0.7; 0.2–8.5)
Full-mouth data†			
Number of sites assessed (SD)			
Mean (SD; range)	–	80.2 (5.6; 42–84)	78.8 (7.5; 18–84)
Prevalence: no of participants with 1+ sites with			
4 + mm CAL (%)	–	225 (27.1)	358 (43.1)
5 + mm CAL (%)	–	93 (11.2)	185 (22.3)
6 + mm CAL (%)	–	42 (5.1)	94 (11.3)
Extent: mean percentage of sites with			
4 + mm CAL (SD; range)	–	1.8 (5.4; 0–57)	4.8 (12.9; 0–100)
5 + mm CAL (SD; range)	–	0.5 (2.5; 0–46)	2.2 (9.3; 0–100)
Severity: mean CAL (SD; range)	–	1.4 (0.4; 0.6–4.6)	1.6 (0.7; 0.3–8.4)

*Standard deviation
†Half-mouth examinations undertaken at age 26 due to time constraints

Table 3. Summary data on changes in the occurrence of periodontal attachment loss from ages 26 through 38, overall and by tooth type (half-mouth data; differences statistically significant unless otherwise indicated)

	Between ages	
	26 and 32	32 and 38
All teeth combined		
Number of people (%) with 1+ sites		
CAL increasing by 3 + mm	100 (12.0)	184 (22.1)
CAL increasing by 4 + mm	25 (3.0)	77 (9.3)
CAL increasing by 5 + mm	10 (1.2)	34 (4.1)
Mean percentage of sites* (SD) with		
CAL increasing by 3 + mm	4.3 (4.2)	9.3 (14.2)
CAL increasing by 4 + mm	4.3 (3.8)	9.7 (14.6)
CAL increasing by 5 + mm	3.6 (2.2)	9.6 (14.5)
Mean change in CAL overall (SD)	−0.1 (0.3)	0.2 (0.5)
Mean change in CAL restricted† (SD)	0.3 (0.4)	0.7 (0.8)
Molars only		
Number of people (%) with 1+ sites		
CAL increasing by 3 + mm	71 (8.5)	108 (13.0)
CAL increasing by 4 + mm	17 (2.0)	35 (4.2)
CAL increasing by 5 + mm	7 (0.8)	16 (1.9)‡
Mean percentage of sites* (sd) with		
CAL increasing by 3 + mm	3.4 (2.3)	5.2 (4.6)
CAL increasing by 4 + mm	3.3 (1.8)	5.4 (5.0)
CAL increasing by 5 + mm	3.2 (1.8)	5.2 (6.4)
Mean change in CAL overall (SD)	−0.0 (0.4)	0.2 (0.6)
Mean change in CAL restricted† (SD)	0.6 (0.6)	1.1 (1.0)
Premolars only		
Number of people (%) with 1+ sites		
CAL increasing by 3 + mm	15 (1.8)	70 (8.4)
CAL increasing by 4 + mm	5 (0.6)	28 (3.4)
CAL increasing by 5 + mm	2 (0.2)	10 (1.2)
Mean percentage of sites* (SD) with		
CAL increasing by 3 + mm	4.1 (2.9)	5.0 (4.2)
CAL increasing by 4 + mm	4.4 (4.5)	5.4 (4.3)
CAL increasing by 5 + mm	5.4 (4.2)	5.6 (3.2)
Mean change in CAL overall (SD)	−0.1 (0.4)	0.2 (0.6)
Mean change in CAL restricted† (SD)	0.9 (0.7)	1.2 (0.9)
Anteriors only		
Number of people (%) with 1+ sites		
CAL increasing by 3 + mm	40 (4.8)	104 (12.5)
CAL increasing by 4 + mm	8 (1.0)	54 (6.5)
CAL increasing by 5 + mm	3 (0.4)	24 (2.9)
Mean percentage of sites* (SD) with		
CAL increasing by 3 + mm	4.0 (2.3)	7.7 (10.2)
CAL increasing by 4 + mm	3.8 (1.2)	7.5 (9.3)
CAL increasing by 5 + mm	2.9 (0.5)	7.8 (8.2)
Mean change in CAL overall (SD)	−0.1 (0.4)	0.2 (0.6)
Mean change in CAL restricted† (SD)	0.6 (0.5)	1.0 (1.0)

*Among incident cases (those with 1+ sites with that particular increase in CAL
†To those with 1+ sites with a 3 + mm increase in CAL
‡P > 0.05 (χ^2 test)
CAL, combined attachment loss.

4+ mm CAL is presented in Fig. 1. A 4-group solution was the most parsimonious [with a bayesian information criterion (BIC) of −4886.71; 3- and 5-group solutions gave BICs of −5601.96 and −4898.93 respectively]. The 4 trajectory groups comprised 55.2%, 31.5%, 10.7% and 2.5% of the cohort; these were termed the "Very low", "Low", "Moderately increasing" and "Markedly increasing" trajectory groups respectively. The latter two groups showed greater increases in the extent of AL after age 32; this was particularly marked in the "Markedly increasing" group. The Wald test was used to test the parameters of the four groups in the trajectory model. The intercepts of all four groups were significantly different from one another ($\chi^2 = 15.5$, df = 3, $p = 0.0014$), as were the slopes of the three groups with linear functions ($\chi^2 = 126.5$, df = 2, $p < 0.0001$) in the model (the "no disease" group was a constant, and thus had no slope).

Higher proportions of males and those of low SES were in the more severe trajectory groups (Table 5), which also had higher proportions of current smokers; the "Very low" group comprised two-thirds of those who had never smoked, but just over half of the former smokers, and fewer than one-third of the current smokers. Only one-quarter of those who had smoked since age 15 were in the "Very low" group; among those who had not, it was well over half. Those who were in the highest cannabis smoking exposure group were disproportionately represented in the two highest trajectory groups.

The multivariate model for trajectory group membership (Table 6) showed a gradient in relative risk by smoking status, whereby those who had smoked tobacco at all ages from 15 through 38 were at higher risk of being in the "Moderately increasing" or "Markedly increasing" trajectory groups. There was a similar risk gradient for those who were in the highest 20% of cannabis usage.

Discussion

This study aimed to describe changes in the occurrence of periodontal AL through ages 26, 32 and 38 among members of a birth cohort followed since birth. It found that the prevalence and extent of AL increased with age, with greater changes between ages 32 and 38 than between 26 and 32, and more new AL than progressing AL. Four trajectories of periodontitis experience were able to be characterized, and longer term smokers and those of low SES were more likely to be in the two groups with the least favourable trajectories.

Before discussing the findings, it is appropriate to consider the limitations and strengths of the study. The first limitation to be considered is representativeness: although the sample is a complete birth cohort with exceptional follow-up rates overall, there were some systematic SES differences in those who were periodontally examined. Given that the study findings highlight low SES as a risk

Table 4. Summary data on new and progressing AL by 3 + mm (half-mouth data; differences statistically significant unless otherwise indicated)

	Between ages	
	26 and 32	32 and 38
All teeth combined		
Number of people with new AL (%)	100 (12.0)	183 (22.0)
Mean percentage of sites with new AL* (SD)	4.3 (3.5)	8.4 (11.9)
Number of people with progressing AL* (%)	5 (0.6)	25 (3.0)
Mean percentage of progressing sites* (SD)	5.8 (6.2)	7.5 (10.4)
Molars only		
Number of people with new AL (%)	71 (8.5)	108 (13.0)
Mean percentage of sites with new AL* (SD)	10.8 (5.3)	20.0 (18.6)
Number of people with progressing AL* (%)	4 (0.5)	14 (1.7)
Mean percentage of progressing sites* (SD)	18.1 (12.1)	13.3 (5.4)
Premolars only		
Number of people with new AL (%)	15 (1.8)	70 (8.4)
Mean percentage of sites with new AL* (SD)	16.5 (11.3)	18.1 (16.3)
Number of people with progressing AL* (%)	1 (0.1)	5 (0.6)†
Mean percentage of progressing sites* (SD)	8.3 (—)	17.2 (11.4)
Anteriors only		
Number of people with new AL (%)	40 (4.8)	104 (12.5)
Mean percentage of sites with new AL* (SD)	8.3 (4.6)	13.7 (15.4)
Number of people with progressing AL* (%)	2 (0.2)	13 (1.6)
Mean percentage of progressing sites* (SD)	8.3 (3.9)	15.4 (18.3)

*Among those experiencing it
†$P > 0.05$; McNemar test
AL, attachment loss.

factor for AL through the 20s and 30s, it is possible that the disproportionate loss of low-SES individuals has resulted in a degree of underestimation of the association's strength. The other major concern is the use of partial recording protocols for the periodontal examinations. There are two issues here: (1) for the examinations at 26, 32 and 38, we measured periodontal AL at only three sites (instead of six) per tooth; and (2) we examined the changes over time using half-mouth data. A recent study of the effect of partial recording protocols on estimates of periodontal disease prevalence found that the particular three-site combination used in this study was associated with the least misclassification relative to estimates from the use of all six sites per tooth (Susin et al. 2005), but caution should be exercised in interpreting findings from any periodontal study which has not used full-mouth recordings from six sites per tooth (Papapanou, 2012). Resource and time constraints meant that we had to use half-mouth periodontal examinations in the age-26 assessments; accordingly, the longitudinal analyses of changes over time through ages 26, 32 and 38 had to be limited to the same half-mouth sites, and that will inevitably have resulted in underestimation (which is apparent from the half- and full-mouth data in Table 2). We have not presented separate data on GR and PD in this report, chiefly because it would considerably complicate the data presentation (but we intend to identify an effective method of doing so and to report those data at a later date). Finally, we were unable to determine whether any participant had periodontal surgery by age 38 (which could possibly have eliminated periodontal pocketing), but the proportion who have

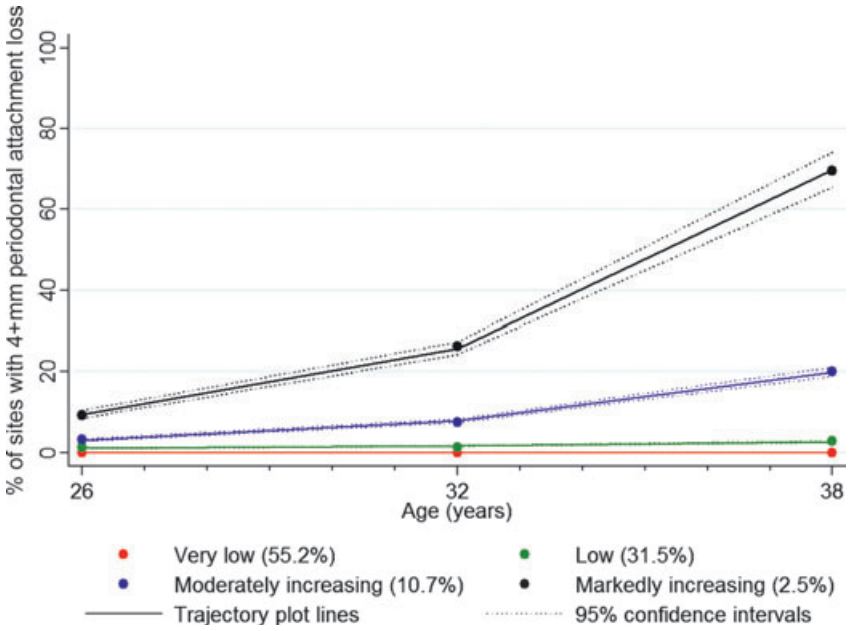

Fig. 1. Trajectories of periodontitis experience [based on the percentage of sites at each age with 4 + mm combined attachment loss (CAL)]

Table 5. Membership of periodontitis experience trajectory groups, by sociodemographic characteristics and smoking status (brackets contain row percentages unless otherwise indicated)

	Periodontitis experience trajectory group			
	Very low	Low	Moderately increasing	Markedly increasing
Number	459 (55.2)	262 (31.5)	89 (10.7)	21 (2.5)
Sex				
Male	206 (48.7)	152 (35.9)	52 (12.3)	13 (3.1)*
Female	253 (62.0)	110 (27.0)	37 (9.1)	8 (2.0)
Childhood SES				
High	84 (58.3)	44 (30.6)	15 (10.4)	1 (0.7)*
Medium	319 (60.0)	158 (29.7)	44 (8.3)	11 (2.1)
Low	54 (35.8)	59 (39.1)	29 (19.2)	9 (6.0)
Adult SES (at age 32)				
High	99 (66.9)	39 (26.4)	10 (6.8)	0 (0.0)*
Medium	255 (57.3)	144 (32.4)	38 (8.5)	8 (1.8)
Low	105 (44.1)	79 (33.2)	41 (17.2)	13 (5.5)
Tobacco smoking status at age 38				
Never	268 (67.7)	108 (27.3)	19 (4.8)	1 (0.3)*
Former	129 (56.1)	77 (33.5)	20 (8.7)	4 (1.7)
Current	62 (30.2)	77 (37.6)	50 (24.4)	16 (7.8)
Tobacco smoker at ages 15, 21, 26, 32 and 38				
No	438 (58.6)	233 (31.1)	67 (9.0)	10 (1.3)
Yes	21 (25.3)	29 (34.9)	22 (26.5)	11 (13.3)
Cannabis smoking from ages 18 to 38				
Highest 20%	17 (25.4)	22 (32.8)	21 (31.3)	7 (10.4)
The remainder	442 (57.9)	240 (31.4)	68 (8.9)	14 (1.8)

$P < 0.05$ (χ^2 test)

Table 6. Outcome of multivariate analysis of periodontitis trajectory group membership (estimates are relative risks; brackets contain 95% confidence intervals)

Independent variables*	Very low relative risk (95% CI)	Low relative risk (95% CI)	Moderately increasing relative risk (95% CI)	Markedly increasing relative risk (95% CI)
Female	1.0	0.5 (0.4, 0.7)‡	0.6 (0.4, 1.0)†	0.5 (0.2, 1.1)
Low adult SES	1.0	1.8 (1.1, 2.9)†	2.1 (1.0, 4.7)	8.8 (1.1, 71.2)†
Medium adult SES	1.0	1.4 (0.9, 2.1)	1.1 (0.5, 2.4)	2.5 (0.3, 20.9)
Smoked tobacco at all ages from 15 to 38	1.0	1.9 (1.5, 2.5)‡	3.8 (2.6, 5.7)‡	7.4 (3.9, 14.1)‡
Highest 20% of cannabis users	1.0	1.2 (0.6, 2.3)	3.5 (1.7, 7.2)‡	2.9 (1.1, 8.0)†

*Reference categories: male (for females); high adult SES (for low and medium SES); other smokers and non-smokers; and those not in the highest 20% of cannabis users.
†$P < 0.05$
‡$P < 0.001$
SES, Socio-economic status.

had it is likely to be very low. Where the study's strengths are concerned, it remains the only study which is producing information on the natural history of periodontal AL in a population sample of adults as they age through their 20s and 30s. The longitudinal data set enables the use of novel analytical approaches such as growth trajectory modelling.

Turning to the findings, perhaps the most noteworthy are (1) the observation that the rate of periodontal destruction was greater between 32 and 38 than it was between 26 and 32, and (2) the characterization of trajectories of the extent of periodontal AL. The apparent acceleration in periodontal AL in the mid-to-late 30s was considerable, with a doubling of the proportion of sites showing AL, especially among the anterior teeth. Given the strong association observed between chronic smoking and periodontitis, it is likely that this acceleration largely represents the cumulative effects of both chronic smoking and low socio-economic status.

As far as we are aware, this is the first report of the characterization of trajectories of the extent of periodontal AL. Leaving aside the caveats arising from the use of partial recording protocols in their identification (Papapanou, 2012), the trajectory groups appear to be valid (Fig. 2), with marked gradients across them in terms of accumulated AL by age 38, determined with the full-mouth periodontal data. The practical relevance of such trajectories remains to be seen, although it is clear that they can be useful in enabling further understanding of the longer term effects of smoking (and they may be useful health education tools in that respect). Undetectable in the cohort as yet, periodontitis-associated incremental tooth loss is likely to be a feature of the oral status of the "Moderately increasing" and "Markedly increasing" trajectory groups as the Study members age.

Undertaking analyses of periodontal data at three time points is difficult, necessitating the computation of CAL for each site at each age, determining the changes in CAL between ages, and then deriving person-level measures from the site-level data. There is scope to explore the utility of multilevel modelling in future analyses (including determination of the effects of inter-proximal restorations), but applying that to longitudinal periodontal data is likely to be challenging.

The likely public health implications of periodontal disease have been realized only of late, and the need for a greater understanding of how the disorder develops over time has become crucial. Little research to date has investigated the transition between periodontal health and manifest disease. Indeed, this is the period in which preventive intervention is likely to have the greatest impact and be the most cost-effective. Our study has bridged the gap between these two health states, highlighting the detrimental impact of smoking on the development of periodontitis, and confirming the

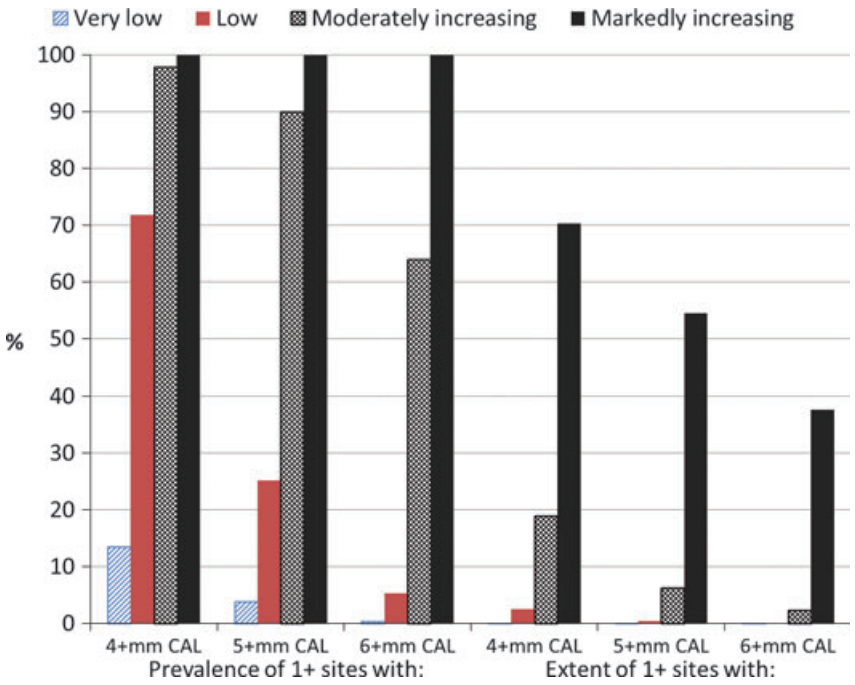

Fig. 2. Prevalence and extent of periodontitis at age 38 (full-mouth data), by periodontitis experience trajectory group

greater risk experienced by those in the lowest SES groups. In addition, the use of group-based trajectory modelling converted our complex data to a form easily understood by non-technical audiences. This is an important advantage when discussing findings with general practitioners, public health funders, politicians and the public. The strength of association with smoking (whether tobacco or cannabis) means that the greatest population gains in periodontal health are likely to come from the eradication of smoking.

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Clinical Relevance

Scientific rationale for the study: Little is known of the natural history of periodontal AL in young adults as they move through their third and fourth decades towards middle age.

Principal findings: The prevalence and extent of AL increased with age, with greater changes between ages 32 and 38 than between 26 and 32, and more new AL than progressing AL. Four trajectories of periodontitis experience were able to be characterized, and longer term smokers and those of low SES were more likely to be in the less favourable trajectories.

Practical implications: The early commencement of periodontal AL and its accelerating progression with age, particularly among chronic smokers of tobacco or cannabis, mean that clinicians need to screen for it in young adults, and public health efforts to eliminate smoking are more urgent than ever.