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Effect of periodontal therapy on lung function: a twelve-month follow-up intervention study

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Abstract

Background Evidence suggest an inflammatory link between respiratory health and periodontitis. This study aims to evaluate the impact of periodontal therapy on lung function.

Methods Sixty-two never-smoking patients with mild periodontitis and without other medical conditions participated in this single-blind, prospective trial. Patients underwent periodontal therapy following an infection control approach. Lung function was measured using forced oscillation technique, assessing airway resistance and reactance, and spirometry evaluating FEV₁, FVC, and FEV₁/FVC. Lung function and fractional exhaled nitric oxide were assessed at baseline, three and six weeks, and every three months for a year. Periodontal parameters were recorded at baseline, six weeks, six and 12 months. Data were analysed using mixed-effects regression models.

Results Patients (mean age 36 years, 58% female) showed significant improvements in periodontal parameters ($p < 0.001$). Oscillometry revealed a significant decrease in airway resistance at 11 Hz and 19 Hz after six weeks, with further significant decreases throughout the study. Resistance at 5 Hz (R_5) consistently declined, reaching significance at three months ($p = 0.001$). By one year, R_5 , R_{11} , R_{19} , and R_{5-20} showed significant reductions (all $p < 0.05$). Airway reactance at 5 Hz became less negative at three months ($p = 0.002$), while the reactance area (AX) decreased significantly at six months ($p = 0.008$). No significant changes were observed in spirometry or fractional exhaled nitric oxide.

Conclusion A decrease in airway resistance was observed after periodontal therapy, underscoring its positive impact on small airway function. These findings suggest that oral infection control is valuable for respiratory health in young adults before chronic conditions establish.

Clinical trial registration The trial was registered at ClinicalTrials.gov (NCT04781153) on February 19, 2021, prior to participant enrolment.

Keywords Oral health, Periodontitis, Respiratory health, Small airway resistance, Lung function testing

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Background

Chronic respiratory diseases, leading causes of global morbidity and mortality [1], affect the airways and lungs. Periodontitis is a chronic, non-communicable disease that affects up to 50% of the global population [2]. It is initiated by accumulation and dysbiosis of dental biofilm, prompting an inflammatory host response that in turn leads to the destruction of tooth-supporting tissues [3].

Studies have shown associations between periodontitis and impaired lung health, including airflow obstruction [4], airflow limitation [5], and reduced spirometry indices like forced expiratory volume in the first second (FEV₁) [6]. The suggested biological pathways are pathogens entering the lungs through micro-aspiration or systemic dissemination, triggering airway inflammation [7] leading to poorer respiratory health outcomes [8–10]. Additionally, shared risk factors may also play a role in linking periodontitis to respiratory health [11, 12]. Research, primarily in patients suffering from chronic obstructive pulmonary disease (COPD), indicates that dental treatment can improve lung function and reduce COPD exacerbations [13–16].

To our knowledge, no prior study has investigated the effect of periodontal therapy on lung function in a never-smoking population without overt chronic lung disease with focus on mild periodontitis. This is the most common form of periodontitis, affecting approximately 50% of the global population. Therefore, the primary aim of this study was to evaluate whether removing dental biofilm and achieving oral infection control in cases of mild periodontitis can lead to improved lung function.

Methods

Study design

The present study is a single-blind, prospective, longitudinal clinical trial designed to evaluate change in lung function following periodontal therapy. Sample size was calculated based on the primary outcome of forced expiratory volume in the first second (FEV₁). Hypothesising that periodontal therapy would cause a minor change in FEV₁, we designed the study to detect an absolute difference of 120 ml in FEV₁. Assuming also that the standard deviation of the difference would be 150 ml, we calculated that a sample size of 52 was needed to have 80% power of observing a statistically significant result with a 5% level of significance. To account for 15% drop out within the population, we aimed to include 62 participants.

Flow-chart of enrolment, therapy, data collection and dropout reasons and timepoints are presented in Fig. 1. Following the active phase of periodontal therapy, patients were monitored at three-month intervals for one year. The trial was conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines to ensure

comprehensive and transparent reporting. Ethical approval was obtained from the Regional Committee for Medical and Health Research Ethics in Western Norway (approval no. #94605) and the trial was registered at ClinicalTrials.gov (NCT04781153) on February 19, 2021, prior to inclusion. All participants provided written informed consent.

Study participants

Recruitment of participants relied primarily on advertising through media channels, as well as through a review of eligible subjects from the university clinic's patient database. A total of 187 individuals were contacted by phone for assessment of inclusion and exclusion criteria, 129 individuals underwent a clinical examination of which 62 fulfilled the inclusion criteria and consented to study inclusion. The examination and treatment were performed at the Department of Clinical Dentistry, University of Bergen between April 2021 to June 2024. Details on pre-study tests are described in the supplement.

Inclusion criteria: (1) Never-smoking individuals aged between 25 and 45 years; (2) good general systemic health defined as no history of systemic diseases or medications likely to interfere with study outcomes; (3) non-severe periodontitis (Stage I-II); (4) high dental plaque- and bleeding percentage ($\geq 50\%$).

Exclusion criteria were: (1) current or former smoking. Smoking history was assessed during the screening process, and participants who reported any current or former smoking during the interview were excluded; (2) current use of moist tobacco; (3) chronic lung diseases based on self-reported medical history. Chronic lung diseases were defined as physician-diagnosed, long-term respiratory conditions such as asthma, chronic obstructive pulmonary disease, or other persistent pulmonary disorders requiring ongoing treatment or follow-up; (4) symptoms of pollen allergy; (5) pregnancy; (6) systemic antibiotics last six months; (7) subgingival scaling last six months; (8) regularly use of oral antiseptic mouth rinse; (9) any current medical condition which may mediate the association between exposure and outcome.

Figure 1. Showing enrolment, periodontal therapy, data collection, and dropout timepoints during the whole trial. Full data: comprised a full-mouth periodontal charting, oral hygiene assessment, lung function testing and questionnaire. Lung function and FeNO: Forced oscillation technique (FOT), spirometry, and Fractional exhaled nitric oxide (FeNO) testing. Active periodontal therapy (APT): supra- and subgingival biofilm removal with a full-mouth disinfection protocol. Supportive periodontal care (SPC): proactive and preventive measure aimed at maintaining the health of the periodontium after active treatment and ensuring periodontal biofilm

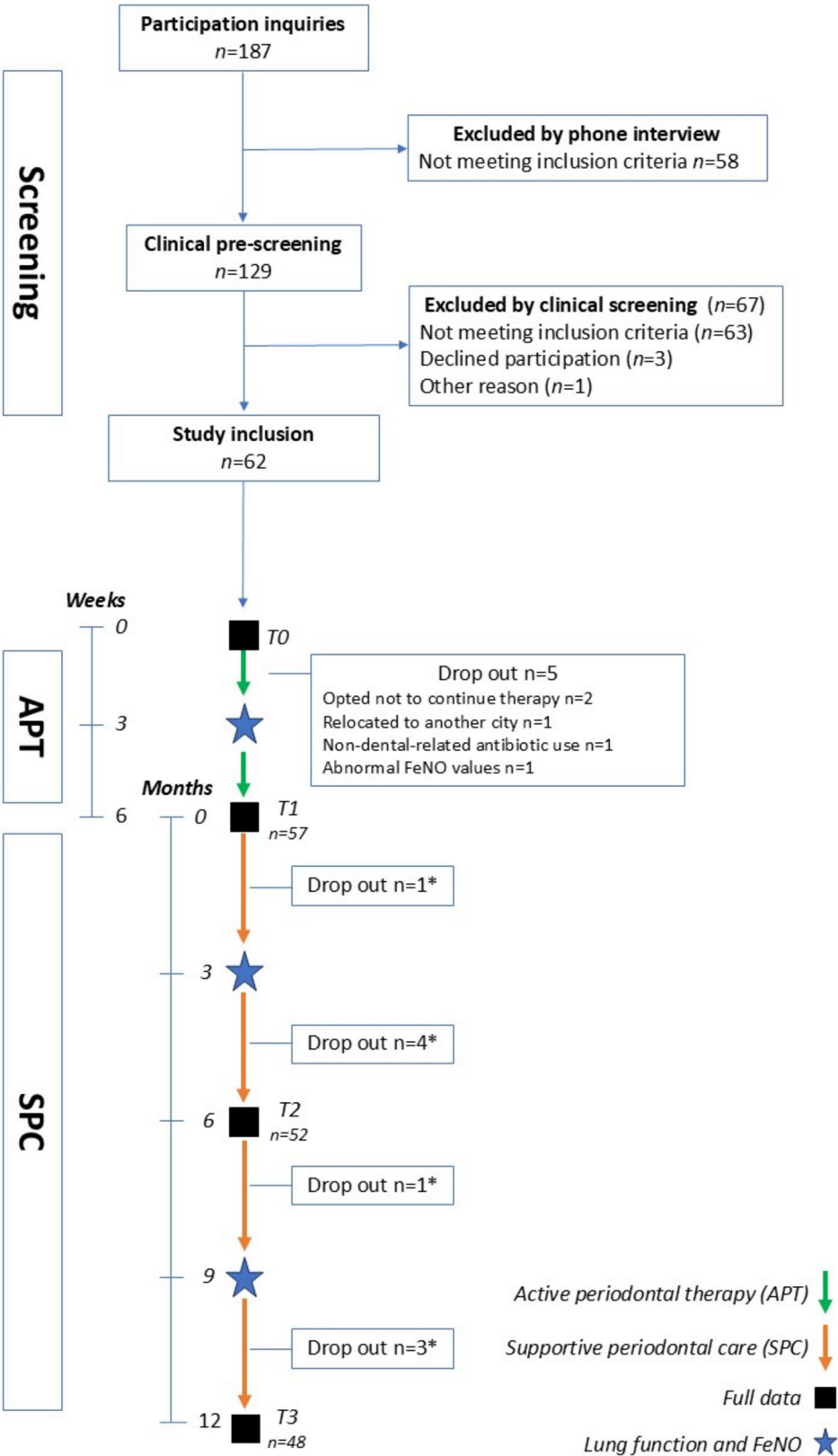


Fig. 1 Flow chart of enrolment, periodontal therapy, data collection, and dropout timepoints and reasons for dropout

and inflammation control. T0: baseline, T1: six-week follow-up, T2: six-month follow-up, T3: twelve-month follow-up. * patient citing an inability to adhere to the follow-up regimen.

Data collection

A full data collection comprised lung function measurements and assessment of levels of fraction of exhaled nitric oxide (FeNO), blood samples (serum), questionnaires, and a full-mouth periodontal charting. Lung function measurements, FeNO assessments, blood sample collection, and questionnaire completion were conducted on the same day, prior to the dental examination. The personnel assessing lung function were blinded to the participants' periodontitis status. The first author (AR) collected the periodontal parameters blinded to lung function results. Participants were un-blinded to all variables.

Full data collection occurred at four time points: baseline (T0), six weeks (T1), six months (T2), and twelve months (T3) following the active periodontal therapy. During the follow-up time points SPC1 (supportive periodontal care 1, three months post-therapy) and SPC2 (supportive periodontal care 2, nine months post-therapy), patients underwent lung function measurements, oral hygiene assessment, and a full-mouth dental biofilm removal. At each clinical examination, patients were also asked about any changes in their medical history and medication status.

Lung function and FeNO measurements

Lung function, the primary outcome, and FeNO data were collected by trained fieldworkers at Research Unit for Health Surveys (RUHS), University of Bergen. FeNO measurements were performed according to standardized methods [17], prior to lung function assessment.

Respiratory impedance was measured by the forced oscillation technique (FOT) using Thorasys Tremflo C-100 (Thorasys Medical Systems, Canada), prior to spirometry. During oscillometry testing, a stimulus is applied to the respiratory system at the mouth to measure the respiratory impedance in a passive manner [18]. Impedance is further split into its components of respiratory system resistance (Rrs) and reactance (Xrs). Rrs reflects airway diameter whereas Xrs reflects the elastic and inertive properties of the respiratory system [18]. Resistance from 5 to 20 Hz (R_5 , R_{11} , R_{19} , R_{20}), the reactance at 5 Hz (X_5), the resonant frequency (F_{res}) and the area under the reactance curve (AX) were calculated for each manoeuvre. To acquire the resistance of the distal airways, the difference between R_5 and R_{20} was calculated. The coefficient of variation (CoV) for R_5 was the main index of the reliability and repeatability of the impedance measurements, and measurements were

conducted according to the 2020 European Respiratory Society guideline [18]. Further details can be found in the supplement.

Spirometry was performed in line with the American Thoracic Society/European Respiratory Society recommendations [19] using *ndd EasyOne Spirometer*. No bronchodilator medication was given to the subjects. To obtain optimal flow-volume curves the participants performed at least three, but no more than eight manoeuvres. The highest recorded values of forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC) and the ratio of FEV_1 and FVC (FEV_1/FVC) were used in the analysis. Percent predicted values and z-scores were calculated from the 2012 Global Lung function Initiative (GLI) [20]. Based on the GLI 2012 recommendations for asymptomatic individuals, the lower limit of normal (LLN) was defined as the 2.5th percentile (z-score = -1.96). Normal lung function was defined as FEV_1 , FVC, and FEV_1/FVC z-scores all at or above the LLN. A restrictive spirometric pattern was defined as a preserved FEV_1/FVC ratio (\geq LLN) with a reduced FVC (z-score < -1.96).

Periodontal variables

Dental and medical data (e.g. use of medications) were recorded during the periodontal examination by a single examiner (AR). The radiographic examination included intraoral radiographs with bitewing, and if needed supplementary periapical radiographs. The clinical examination comprised a full-mouth registration of probing pocket depth (PD) and clinical attachment loss (CAL) using a periodontal probe with 1 mm grading (Hu-Friedy PCPUNC157). PD was recorded at six sites per tooth as the distance in mm from the gingival margin to the base of the pockets, and CAL as the distance in mm from the cementum–enamel junction to the depth of the pocket (third molars excluded). Clinical periodontal inflammation and dental biofilm were assessed at four sites per tooth and expressed as the percentage of sites exhibiting bleeding on probing (BoP) [21] and visible supragingival dental plaque (PI) [22], using a disclosing agent (Cura-prox, PCA 260). Periodontitis were diagnosed according to the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions [23].

Other patient related assessments

Anthropometric data, serum samples, and questionnaire data were collected during the clinical examinations at RUHS. Further details can be found in the supplement. Serum high-sensitive C-reactive protein (CRP) levels (mg/L) were analysed using a high-sensitivity immunoturbidimetric assay (Cobas 8000, Roche Diagnostics) in

the laboratory at Haukeland University Hospital, Bergen, Norway.

Periodontal therapy

Periodontal therapy was conducted (by AR) in a step-wise manner according to the S3-level clinical practice guidelines from the European Federation of Periodontology [24]. Participants started Step 1 therapy immediately after baseline data collection (T0), which included oral hygiene instructions, removal of supragingival biofilm and calculus, and the elimination of local biofilm-retentive factors. To enhance treatment compliance, each patient was provided with a comprehensive oral hygiene kit for home use (details provided in the supplement). Step 2 periodontal therapy followed Step 1 and involved a modified full-mouth disinfection and scaling (FDIS) intervention approach [25]. Step 2 therapy commenced no later than three weeks following Step 1.

During Step 2 therapy, patients received a full-mouth supra- and subgingival biofilm removal using an ultrasonic scaler (SonicFlex, Piezolux/Sonosoft tip no.10, KaVo, USA) as well as hand instruments (Hu-Friedy, Chicago, IL, USA) within 24 h. Local anaesthesia (Xylocain Dental 2% adrenalin, Dentsply) was administered, if needed. The intervention session was completed with a thorough cleansing with prophylactic paste (Prophy paste CCS RDA 170, Directa AB) and application of 1% chlorhexidine gel (Corsodyl, GlaxoSmithKline Consumer Healthcare, Denmark) in all sulci and pockets. The approximal sites were then flossed to carry the gel into the proximal contacts. At the end of the session, each patient was provided with a bottle of 0.2% chlorhexidine solution (Curaprox Perio Plus Forte CHX 0.2%) and a tube of 1% chlorhexidine gel (Corsodyl, GlaxoSmithKline Consumer Healthcare, Denmark) for home use. For a two-week period following therapy, participants were instructed to rinse with the chlorhexidine solution for one minute twice daily (morning and before bedtime) and to brush their tongues with chlorhexidine gel at bedtime. Between tooth brushing and chlorhexidine mouth wash, participants were instructed to rinse thoroughly with water for 30 s to avoid possible carryover interactions between toothpaste and chlorhexidine agents.

Statistical analyses

For the analysis of the periodontal parameters, the patient was the unit of observation, meaning that BoP, PI, PD, and CAL was represented as the whole-mouth averages. All individuals had lung function data collected at multiple time-points. To assess changes in the repeated measurements over time, linear mixed-effects models were used, with individual as a random effect. The random mixed effects models also adjust for data missing

at random (i.e. participants with loss to follow-up or drop-out).

Differences between groups over time were tested in the mixed effects model with the use of an interaction term between groups and time. P-values (two sided) less than 0.05 were considered statistically significant. All statistical analyses were conducted using Stata 17.0 (Stata Corporation, College Station, TX, USA).

Results

The baseline characteristics of the study population are summarized in Table 1. A total of 62 participants were included, with a mean age of 35.8 years, and a predominance of female participants (58%). Fifty-six participants (90%) had normal lung function based on the LLN threshold, while one (1.6%) showed an obstructive spirometric pattern and three (4.8%) showed a restrictive pattern (data not shown). The mildest form of periodontitis (Stage I) was diagnosed in 21% of participants, while 79% were diagnosed with Stage II. When stratified by sex, statistically significant differences were observed in baseline FEV₁ and FVC values, as well as in the absolute values of oscillometry variables R₅, R₁₁, and R₁₉. Of the initial cohort, 48 patients (77.4%) successfully completed the trial (Fig. 1). An attrition analysis was conducted to compare baseline characteristics between subjects who dropped out and those who completed the trial (T0 to T3). No statistically significant differences were observed in lung function measurements between the two groups (Table S1). No adverse events were reported throughout the study.

Table 1. Data are presented as *n* (%) or mean and standard deviations (SD). FEV₁/FVC is unitless and has the formula FEV₁(L)/FVC (L). Percent predicted values were calculated from the 2012 Global Lung function Initiative. P-values are calculated by t-test (continuous variables) and Chi-square tests (categorical variables). Abbreviations: FEV₁: forced expiratory volume in the first second, FVC: forced vital capacity, R₅: airway resistance measured at 5 Hz, R₁₁: airway resistance measured at 11 Hz, R₁₉: airway resistance measured at 19 Hz. BoP: bleeding on probing, PI: plaque index, PD: periodontal pocket depth, CAL: clinical attachment loss.

Periodontal parameters

Periodontal therapy significantly reduced the mean values of BoP, PI, PD and CAL (all *p* < 0.001) throughout the trial, compared to baseline measurements (Table 2). No statistically significant differences in the periodontal parameters were observed between sex (Figure S1).

Table 2. Periodontal variables from baseline (T0) to twelve-month follow-up (T3). Data are presented as mean and standard error (SE). Abbreviations; BoP: bleeding on probing, PI: plaque index, PD: periodontal

Table 1 Baseline characteristics of study population stratified by sex

Baseline characteristics of study population		Sex		p-value
Variables	Total	Female	Male	
Participants	n = 62	36 (58%)	26 (42%)	
Mean age (SD)	35.8 (6.2)	36.1 (6.0)	35.3 (6.5)	0.64
Married or cohabitant	38 (63.3%)	22 (57.9%)	16 (42.1%)	0.66
Education at University Level	51 (85.0%)	32 (62.8%)	19 (37.2%)	0.56
Employed	50 (83.3%)	30 (40%)	20 (60%)	0.73
Mean body mass index in kg/m ² (SD)	27.4 (5.1)	27.5 (5.3)	27.3 (5.1)	0.89
Mean height in cm (SD)	172 (9.9)	165.6 (6.3)	181.2 (5.9)	< 0.001
Mean weight in kg (SD)	81.5 (18.7)	75.7 (17.3)	89.6 (17.7)	0.003
Severity of periodontitis				0.77
Stage I	13 (21.0%)	8 (61.5%)	5 (38.5%)	
Stage II	49 (79.0%)	28 (57.1%)	21 (42.9%)	
Exercise				0.38
Once a week or less	19 (32.2%)	9 (47.4%)	10 (52.6%)	
2–3 times per week	25 (42.4%)	17 (68%)	8 (32%)	
Almost every day	15 (25.4%)	9 (60%)	6 (40%)	
Absolute values of spirometry variables				
Mean FEV ₁ (SD)	3.55 (0.77)	3.1 (0.44)	4.2 (0.62)	< 0.001
Mean FVC (SD)	4.53 (1.02)	3.9 (0.60)	5.4 (0.90)	< 0.001
Mean FEV ₁ /FVC (SD)	0.79 (0.06)	0.79 (0.06)	0.78 (0.05)	0.58
%predicted spirometry variables				
Mean %predicted FEV ₁ (SD)	94.3 (10.8)	95.5 (10.8)	92.6 (10.9)	0.30
Mean %predicted FVC (SD)	98.6 (11.4)	100.2 (10.7)	96.4 (12.1)	0.18
Absolute values of oscillometry variables				
Mean R _s (SD)	3.22 (1.10)	3.53 (1.12)	2.80 (0.90)	0.006
Mean R ₁₁ (SD)	3.00 (0.84)	3.30 (0.85)	2.60 (0.70)	0.001
Mean R ₁₉ (SD)	2.78 (0.72)	3.10 (0.70)	2.40 (0.52)	< 0.001
%predicted oscillometry variables				
Mean R _s %predicted (SD)	102.85 (26.44)	103.2 (29.2)	102.4 (22.3)	0.91
Mean R ₁₁ %predicted (SD)	102.37 (23.47)	102.9 (25.8)	101.6 (19.9)	0.84
Mean R ₁₉ %predicted (SD)	93.10 (19.40)	94.3 (20.7)	91.4 (17.5)	0.58
Fraction of Exhaled Nitric Oxide (FeNO)				
Mean FeNO in parts per billion (SD)	19.01 (14.35)	18.2 (16.5)	20.1 (10.9)	0.61
Periodontal variables				
Mean BoP in % (SD)	63.01 (1.62)	62.8 (14.1)	63.5 (13.7)	0.84
Mean PI in % (SD)	62.0 (1.90)	61.8 (10.4)	62.4 (10.2)	0.82
Mean PD in mm (SD)	2.48 (0.02)	2.47 (0.20)	2.48 (0.18)	0.86
Mean CAL in mm (SD)	2.70 (0.03)	2.68 (0.20)	2.73 (0.18)	0.39
Mean high-sensitive C-reactive protein (mg/L)	1.70 (2.34)	1.67 (1.96)	1.71 (2.87)	0.94

Table 2 Periodontal variables from baseline (T0) to twelve-month follow-up (T3)

	T0	T1	p-value	T2	p-value	T3	p-value
	n = 62	n = 57		n = 52		n = 48	
Periodontal variables							
BoP in % (SE)	63.10 (1.62)	25.00 (1.68)	< 0.001	28.55 (1.73)	< 0.001	30.01 (1.78)	< 0.001
PI in % (SE)	62.00 (1.90)	22.15 (1.96)	< 0.001	23.06 (2.04)	< 0.001	27.30 (2.11)	< 0.001
PD in mm (SE)	2.48 (0.02)	2.14 (0.02)	< 0.001	2.15 (0.02)	< 0.001	2.16 (0.02)	< 0.001
CAL in mm (SE)	2.70 (0.03)	2.56 (0.03)	< 0.001	2.53 (0.03)	< 0.001	2.44 (0.03)	< 0.001

pocket depth, CAL: clinical attachment loss. Results from a mixed-effects model indicate the statistical significance of differences in means between timepoints, with baseline (T0) as the reference.

Lung function and FeNO measurements following periodontal therapy

The mean absolute values for lung function and FeNO measurements from baseline (T0) to twelve-month follow-up (T3) are presented in Table 3. Oscillometry testing revealed a decrease in airway resistance across all

Table 3 Absolute lung function measurements by spirometry and forced oscillation technique from baseline (T0) to twelve-month follow-up (T3)

Variables	T0 n=62	T1 n=57	p-value	SPC1 n=55	p-value	T2 n=52	p-value	SPC2 n=50	p-value	T3 n=48	p-value
Spirometry											
FEV ₁ in L (SE)	3.55 (0.09)	3.53 (0.09)	0.35	3.54 (0.09)	0.62	3.55 (0.09)	0.92	3.55 (0.09)	0.86	3.54 (0.09)	0.74
FVC in L (SE)	4.53 (0.13)	4.53 (0.13)	0.86	4.54 (0.13)	0.68	4.54 (0.13)	0.63	4.55 (0.13)	0.29	4.55 (0.13)	0.43
FEV ₁ /FVC (SE)	0.786 (0.008)	0.781 (0.008)	0.07	0.783 (0.008)	0.2	0.785 (0.008)	0.55	0.784 (0.008)	0.37	0.782 (0.008)	0.16
Oscillometry											
R ₅ (SE)	3.22 (0.13)	3.10 (0.13)	0.1	2.97 (0.13)	0.001	2.98 (0.13)	0.001	3.00 (0.13)	0.002	2.92 (0.13)	<0.001
R ₁₁ (SE)	3.00 (0.10)	2.86 (0.10)	0.01	2.77 (0.10)	<0.001	2.77 (0.10)	<0.001	2.77 (0.10)	<0.001	2.75 (0.10)	<0.001
R ₁₉ (SE)	2.78 (0.08)	2.63 (0.08)	0.005	2.59 (0.08)	<0.001	2.60 (0.08)	0.001	2.57 (0.08)	<0.001	2.59 (0.08)	0.001
R ₅ -R ₂₀ (SE)	0.45 (0.07)	0.47 (0.08)	0.63	0.39 (0.08)	0.19	0.37 (0.08)	0.11	0.43 (0.08)	0.6	0.33 (0.08)	0.02
F _{res} (SE)	16.73 (0.60)	16.90 (0.61)	0.72	16.31 (0.62)	0.3	16.04 (0.62)	0.09	16.57 (0.62)	0.69	16.18 (0.63)	0.19
X ₅ (SE)	-1.29 (0.06)	-1.24 (0.06)	0.26	-1.15 (0.06)	0.002	-1.14 (0.06)	0.001	-1.12 (0.06)	<0.001	-1.12 (0.06)	<0.001
AX (SE)	7.66 (0.73)	7.84 (0.75)	0.71	6.86 (0.75)	0.1	6.33 (0.76)	0.008	6.87 (0.76)	0.11	6.30 (0.77)	0.008
FeNO (SE)	19.01 (1.62)	18.34 (1.62)	0.525	18.16 (1.65)	0.424	18.12 (1.67)	0.413	19.00 (1.68)	0.986	18.41 (1.70)	0.602

frequencies (Fig. 2). A statistically significant reduction in resistance was noted at 11 Hz and 19 Hz after six weeks (T1), with further decreases throughout the trial. Airway resistance at 5 Hz (R₅) showed a consistent decline at all timepoints, reaching statistical significance three months after T1 (SPC1). One year following therapy, measurements of R₅, R₁₁, and R₁₉ decreased by 9.3%, 8.3%, and 6.8%, respectively (all $p < 0.05$). Additionally, there was a statistically significant reduction in R₅₋₂₀ at T3 ($p = 0.02$). Airway reactance at 5 Hz (X₅) showed improvements (less negative values) at all timepoints compared to baseline, reaching statistical significance at SPC1. Correspondingly, the reactance area (AX) showed an overall reduction from T0 to T3, with a statistically significant decrease six months after therapy (T2) (Fig. 2). No statistically significant changes were observed in FEV₁, FVC, FEV₁/FVC, or FeNO levels following periodontal therapy ($p > 0.05$) (Fig. 3).

Figure 2. Graphs showing marginal means with 95% confidence intervals. Abbreviations: R₅: airway resistance measured at 5 Hz, R₁₁: airway resistance measured at 11 Hz, R₁₉: airway resistance measured at 19 Hz, R₅₋₂₀: the difference in airway resistance measured at 20 Hz and 5 Hz, X₅: reactance measured at 5 Hz, AX: reactance area. T0: baseline, 3 W: three-week control, T1: follow-up at six weeks, SPC1: supportive periodontal care 1, conducted three months after therapy, T2: six months follow-up, SPC2: supportive periodontal care 2, conducted nine months after therapy, T3: twelve-month follow-up. * indicating $p < 0.05$.

Figure 3. Graphs showing marginal means with 95% confidence intervals. Abbreviations: FEV₁: forced expiratory volume in the first second, FVC: forced vital capacity. FEV₁/FVC is unitless and has the formula FEV₁(L)/FVC (L), FeNO: fraction of exhaled nitric oxide, T0: baseline, 3 W: three-week control, T1: follow-up at six weeks,

SPC1: supportive periodontal care 1, conducted three months after therapy, T2: six months follow-up, SPC2: supportive periodontal care 2, conducted nine months after therapy, T3: twelve-month follow-up.

Table 3. Absolute measurements by spirometry, forced oscillation technique and FeNO from baseline (T0) to T3. Results from mixed-effects model presented as mean and standard error (SE). P-values from mixed-effects model indicate the statistical significance of differences in means between timepoints, with baseline (T0) as the reference. Abbreviations: FEV₁: forced expiratory volume in the first second, FVC: forced vital capacity, R₅: airway resistance measured at 5 Hz, R₁₁: airway resistance measured at 11 Hz, R₁₉: airway resistance measured at 19 Hz, R₅₋₂₀: the difference in airway resistance measured at 20 Hz and 5 Hz, F_{res}: resonance frequency measured in Hz, X₅: reactance measured at 5 Hz, AX: reactance area. FEV₁/FVC is unitless and has the formula FEV₁(L)/FVC (L). T0: baseline, T1: follow-up at six weeks, SPC1: supportive periodontal care 1 conducted three months after therapy, T2: six months follow-up, SPC2: supportive periodontal care conducted nine months after therapy, T3: twelve-month follow-up. FeNO: fraction of exhaled nitric oxide has the unit parts per billion. Variables R₅, R₁₁, R₁₉, R₅₋₂₀, X₅, and AX have the unit cm H₂O.L⁻¹. s⁻¹.

Additional analyses of lung function outcomes and C-reactive protein

Out of the 373 R₅-observations collected across all participants and time points (baseline to T3), 98.7% exhibited a coefficient of variation (CoV) below 15%. At baseline, one participant had an R₅ CoV exceeding 15%. Participants were stratified into two groups based on their baseline CoV: those with a CoV ≤ 10% and those with a CoV > 10%. An interaction analysis was performed to assess the

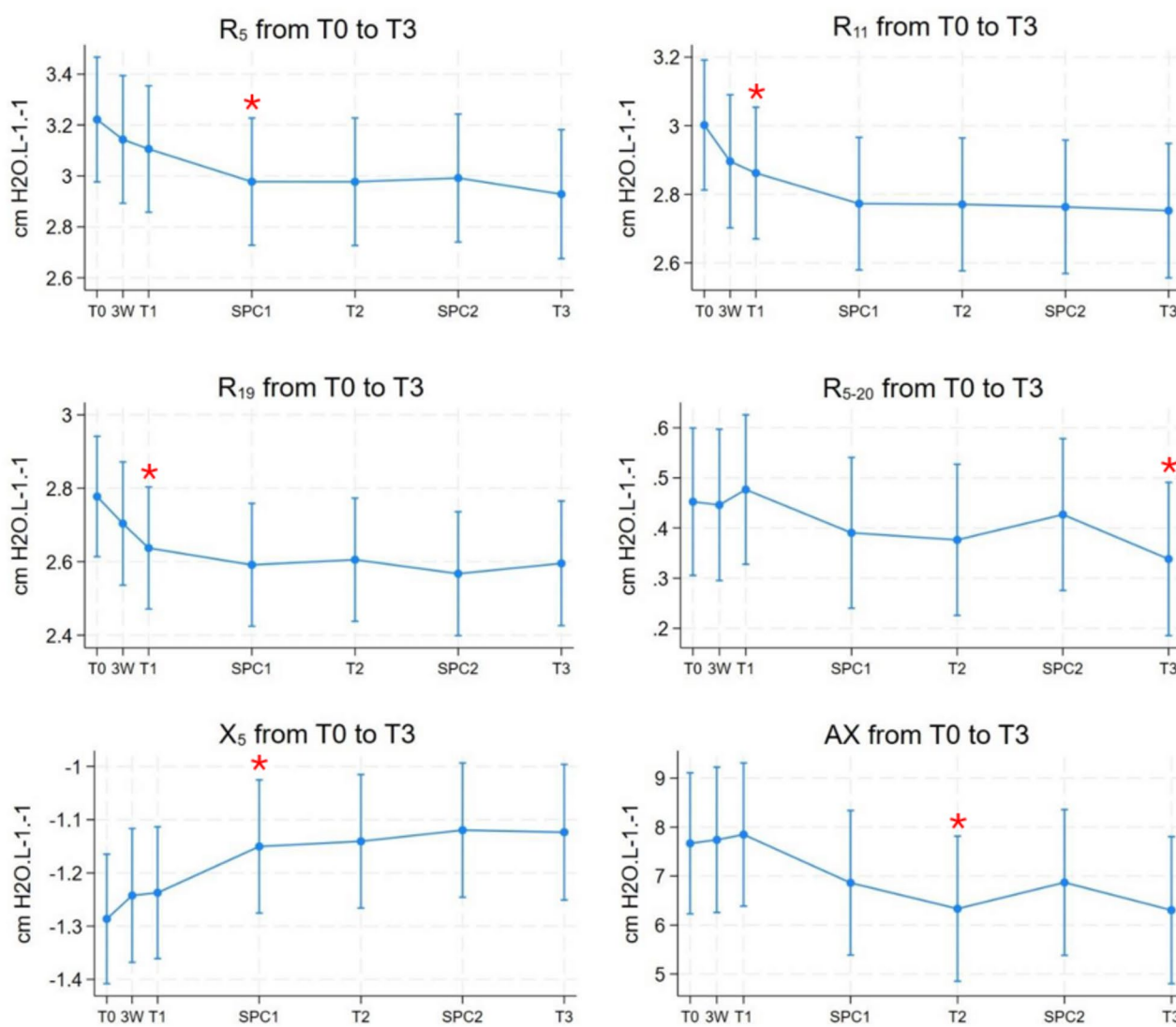


Fig. 2 Graphs showing change in oscillometry variables from baseline (T0) to T3

treatment effect between these groups. The interaction term was not statistically significant for either resistance or reactance measurements (Figure S2). We stratified participants based on periodontal diagnosis (stage I or II) and evaluated potential differences in oscillometry and spirometry outcomes (Figure S3). The interaction analysis showed no statistically significant differences in either the oscillometry or spirometry variables between the strata ($p > 0.05$).

No significant reduction in CRP levels was observed at T1, and no further significant changes were detected throughout the trial period ($p > 0.05$) (Figure S4). However, when participants were stratified by baseline CRP levels above or below the median (0.85 mg/L), a statistically significant difference in treatment effects was observed for R_5 , X_5 , and AX (all $p < 0.05$) between these

strata. No significant effect was found for R_{11} or R_{19} (all $p > 0.05$) (Figure S5).

Discussion

In this novel study addressing the effect of periodontal biofilm control on lung function in healthy individuals, we could find an improvement in respiratory resistance and reactance, but no significant differences in spirometry. Improvements in respiratory resistance were evident as early as six weeks after treatment, with continued enhancement throughout the one-year follow-up period. The observed reduction in resistance, despite the absence of significant changes in spirometry, may be explained by the higher sensitivity of oscillometry in detecting early or subtle changes in lung function.

Multiple studies in general populations have shown an association between periodontitis and lower levels of

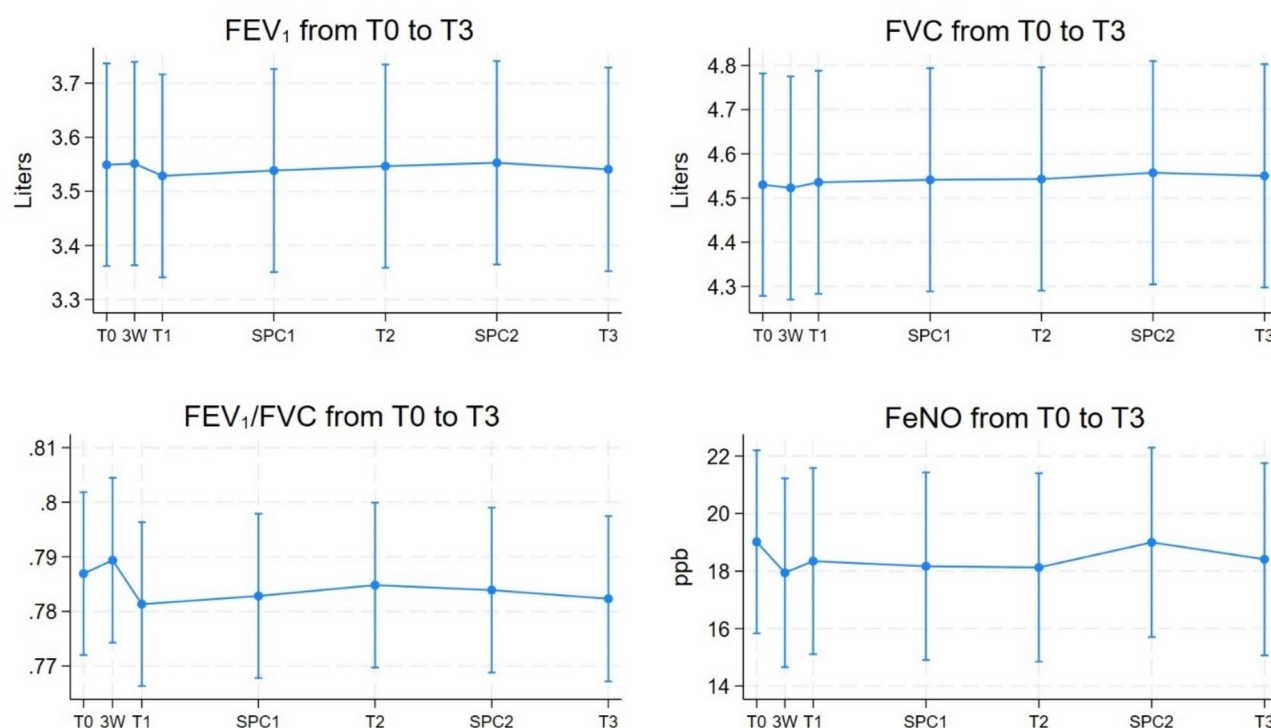


Fig. 3 Graphs showing change in FEV₁, FVC, FEV₁/FVC and levels of FeNO from baseline (T0) to T3

FEV₁ [6], increased risk of airflow obstruction [4], and reduced lung volumes and airflow limitation [5]. A recent systematic review by Molina et al. found positive associations between periodontitis and several lung disorders [26]. In addition to the epidemiological evidence, a small number of intervention studies have shown beneficial effects of periodontal interventions on individuals suffering from COPD [13–15]. Even though those intervention studies have been on COPD, the findings suggest that oral health is closely correlated with key indicators of lung disease, including exacerbations and lung function.

Studies have demonstrated that oscillometry is more effective in detecting early or subtle changes in lung function, particularly in small airways, as seen in conditions like asthma and COPD [27]. Oscillometry parameters may detect airflow obstruction in the peripheral airways before changes become evident in traditional spirometry, making it a more sensitive tool for capturing early changes [28]. The Rrs-parameters measure respiratory resistance at different frequencies, with R₅ reflecting both central and peripheral airway resistance, and R₁₉ primarily assessing central airway resistance. As such, FOT may be better suited for identifying gradual improvements in smaller airways. Additionally, the interaction analysis revealed that individuals with higher baseline airway resistance benefited more from periodontal treatment. This finding underscores the potential importance of integrating periodontal treatment into the management

of patients with pre-existing airway obstructions, or those at risk.

Periodontitis, characterized by oral microbiota dysbiosis, has been linked to chronic respiratory diseases due to the close anatomical proximity of the oral and respiratory tracts and the risk of microaspiration of pathogens [29]. The most common route by which the oral cavity may influence pulmonary function is the aspiration of saliva containing oral bacteria into the lung [7, 30]. In addition to direct aspiration, pulmonary pathogens can colonize the dental biofilm supporting the idea that the oral cavity may act as a reservoir for pathogens associated with respiratory diseases, particularly in high-risk patients [31]. Elevated systemic host inflammatory response are linked to both periodontitis [10] and reduced lung function [9]. Moreover, released inflammatory cytokines from periodontal tissues may alter the respiratory epithelium, promoting respiratory pathogen growth and increasing susceptibility to lower airway infections [32]. Oxidative stress, a common feature of both periodontitis and lung diseases such as COPD, may play a role in the shared pathophysiology of these conditions. It contributes to lung tissue damage, impairs repair mechanisms, and exacerbates respiratory dysfunction, further linking oral and respiratory health [33].

Periodontal treatment has been shown to reduce systemic inflammation in otherwise systemically healthy patients [34]. In our study, we observed an overall

non-significant decrease in CRP levels from baseline to the one-year follow-up (T3). This contrasts with previous studies that reported significant reductions in CRP following periodontal therapy, likely due to differences in study populations; prior studies included individuals with more severe periodontitis and higher baseline CRP levels than our cohort [34, 35]. Notably, our data indicate that patients with baseline CRP levels above the median experienced more pronounced benefits from treatment, with significant improvements in R_5 , X_5 , and AX measurements compared to patients with a CRP below median. By enhancing periodontal health and reducing systemic inflammation, it is plausible that inflammatory processes in the respiratory system may also be mitigated.

Chlorhexidine (CHX), a common antimicrobial in dental care, has demonstrated clinical benefits in one-stage full-mouth disinfection compared to traditional tooth cleaning methods [36]. The use of CHX targets not only periodontal pockets but also other bacterial reservoirs, such as the tongue and oral mucosa, thereby reducing the risk of recontamination in treated areas. In this study, periodontal treatment positively impacted periodontal health, consistent with previous findings on non-surgical therapy using the FDIS approach [37]. However, concerns exist about CHX's potential to disrupt the oral microbiome by reducing beneficial bacteria and promoting less favourable ones [38], which may create a more acidic and harmful oral environment [39], along with possible systemic effects [40]. Despite this, adherence to the treatment protocol was excellent, and the risk of CHX-related adverse effects was considered very low.

Strengths and limitations

The study possesses several strengths. Its longitudinal investigator-masked design, coupled with close follow-up throughout the intervention period, enables the comprehensive evaluation of the airway resistance before, during, and after the intervention. By targeting a population without lung disease but mild forms of periodontitis, elevated gingival inflammation, and high levels of dental biofilm, the study broadens the relevance of its findings. This focus enhances the generalizability of the results, as these periodontal conditions are widespread affecting many who do not have underlying respiratory disease. The positive outcomes observed in the present study could indicate benefits with wide-ranging implications for public health, supporting the role of periodontal care as a potential preventive measure for respiratory health across the general population.

Further strengths of this study include the use of FOT-measurements, which provide additional insight into airway function. Respiratory oscillometry measures identify functional abnormalities in symptomatic subjects despite normal airflow by spirometry and have been

shown to correlate with clinical symptoms [41]. Moreover, by including healthy, never-smoking individuals aged 25–45, who have reached maximum lung capacity [42], minimizing potential confounding effects of age-related lung function decline and factors such as smoking which is a shared risk factor for both periodontitis and respiratory disease [43]. There were no observed differences in the participants' physical exercise frequency or duration throughout the trial, suggesting that any potential effects on lung health outcomes remained consistent during the intervention period (data not shown). None of the patients reported using medications, such as inhalers or bronchodilators, during the follow-up period.

The study has several limitations that warrant consideration. First, the lack of a control group complicates the attribution of improvements in airway resistance solely to the intervention. All patients received periodontal therapy, and no untreated control group was included. This decision was grounded in ethical considerations regarding the progressive nature of periodontitis. Withholding treatment would have posed significant ethical concerns, particularly as standard periodontal therapies are well-established in clinical practice and withholding treatment could potentially harm participants. Instead, a pre-post study design was employed, where participants acted as their own controls, and baseline measures were used to evaluate the effects of the therapy on lung function. This approach allowed us to balance the ethical obligation to provide necessary care with the scientific aim of exploring the link between periodontal therapy and respiratory outcomes.

Another limitation of the study is the lack of established thresholds for minimal clinically important difference (MCID) for oscillometry parameters in healthy individuals. Although our findings of significant improvements in R_{rs} and X_{rs} over 12 months following periodontal therapy fall below the MCID thresholds for asthmatic patients [44], it is important to emphasize that our study focused on systemically healthy individuals without overt respiratory diseases. Nevertheless, the observed improvements, though modest compared to thresholds for diseased populations, may still indicate meaningful physiological benefits, especially if the trends continue beyond 12 months.

Unrecorded external factors may have contributed to the observed changes, complicating the interpretation of results. Changes in lifestyle behaviours such as diet, which were not part of the data collection, could introduce confounding effects. Additionally, selection bias may be present as participants who volunteered may systematically differ from non-participants, potentially affecting generalizability. Moreover, the onset of the COVID-19 pandemic presented logistical challenges, including appointment rescheduling due to only mild

respiratory symptoms among participants. On the other hand, strict adherence to infection control measures ensured that respiratory infections did not impact lung function assessments during the study.

Within the limitations of the present study, our longitudinal investigator-masked clinical trial demonstrated a statistically significant decrease in airway resistance following periodontal therapy. These findings underscore the potential importance of periodontal biofilm control in managing respiratory health. However, the absence of a control group limits our ability to establish causal relationships between periodontal therapy and respiratory health outcomes. Therefore, caution is warranted in interpreting these findings as generalizable to broader populations. Future large-scale randomized controlled trials, incorporating both short-term and long-term follow-up periods, are warranted to further elucidate the impact of periodontitis prevention and treatment on respiratory outcomes.

Abbreviations

AX	Reactance area
BoP	Bleeding on probing
CAL	Clinical attachment loss
CHX	Chlorhexidine
CoV	Coefficient of variance
CRP	High-sensitive C-reactive protein
COPD	Chronic obstructive pulmonary disease
FDIS	Full-mouth disinfection and scaling
FeNO	Fraction of exhaled nitric oxide
FEV ₁	Forced expiratory volume in the first second
FOT	Forced oscillation technique
Fres	The resonant frequency
FVC	Forced vital capacity
MCID	Minimal clinically important difference
PD	Probing pocket depth
PI	Plaque index
Rrs	Respiratory system resistance
RUHS	Research Unit for Health Surveys
Xrs	Respiratory system reactance
R ₅	The resistance measured at 5 Hz
R ₁₁	The resistance measured at 11 Hz
R ₁₉	The resistance measured at 19 Hz
R ₂₀	The resistance measured at 20 Hz
SPC	1 supportive periodontal care 1, three months post-therapy
SPC2	Supportive periodontal care 2, nine months post-therapy
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology
X ₅	The reactance measured at 5 Hz

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12931-025-03246-1>.

Supplementary Material 1

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of charge to the study participants. Finally, we are profoundly grateful to the BORALIS participants for their dedication and involvement in this research.

Author contributions

All listed authors met the International Committee of Medical Journal Editors recommended four requirements for authorship. RBJ and DFB conceived of and designed the study. AR was responsible for the dental examinations and periodontal therapy. AR, RBJ, DFB, AM and SAL acquired, analysed, and interpreted the data. AR and SAL were responsible for the data management and the statistical analysis. DFB supervised the dental clinical procedures throughout the trial. AR, RBJ, DFB, AM and SAL created the first draft of the manuscript. AR, RBJ, DFB, AM, SAL and JH provided crucial revision for important intellectual content and gave final approval of the manuscript.

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Data availability

Due to Norwegian ethical and legal restrictions, all the data underlying the findings in our study cannot be made publicly available. Request for data access can be directed to the corresponding author.

Declarations

Ethics approval and consent to participate

Ethical approval was obtained from the Regional Committee for Medical and Health Research Ethics in Western Norway (approval no. #94605) and the trial was registered at ClinicalTrials.gov (NCT04781153) prior to inclusion. All participants provided written informed consent.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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