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Clinical and microbiological effects of mechanical instrumentation and local antimicrobials during periodontal supportive therapy in aggressive periodontitis patients: smoker *versus* non-smoker patients

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Abstract

Aim: To compare the clinical and microbiological effects of ultrasonic mechanical instrumentation (UMI) associated to home-care use of amine fluoride/stannous fluoride (AmF/SnF_2)-containing mouthrinse and toothpaste in smoker and non-smoker patients affected by generalized aggressive periodontitis (G-AgP) during a recall session of supportive periodontal therapy (SPT).

Material and Methods: Thirteen smokers and 25 non-smokers G-AgP patients enrolled in an SPT programme received a single session of UMI associated with home-care use of AmF/SnF_2-containing mouthrinse and toothpaste. Clinical and microbiological parameters were assessed pre-treatment, at 6 and 12 weeks post-treatment.

Results: In both groups, UMI plus AmF/SnF_2-implemented oral hygiene use determined a significant decrease of total bacterial counts, with non-smokers exhibiting a lower count compared with smokers at 12 weeks. No significant differences were observed between smokers and non-smokers in the counts of total pathogens and red complex species at each observation interval. Clinically, a significant reduction of supragingival plaque, gingival inflammation and probing pocket depth was similarly observed in both groups.

Conclusions: A combined mechanical/chemical plaque control approach based on UMI and the use of AmF/SnF_2 agents resulted in the reduction of supragingival plaque deposits, gingival inflammation and subgingival periodontal pathogens in G-AgP patients during SPT, with no substantial difference between smokers and non-smokers.

Key words: aggressive periodontitis; amine-stannous fluoride; supportive periodontal therapy; ultrasonic instrumentation

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Conflict of interest and source of funding statement

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The main objective of supportive periodontal therapy (SPT) for periodontitis patients is to maintain a microflora compatible with periodontal health at the completion of the active phase of

treatment. Professional supra- and subgingival plaque control represents the elective approach for the prevention of the recurrence of periodontitis lesions (Kristoffersen & Meyer 1983). In this respect, ultrasonic mechanical instrumentation (UMI) is an effective method to control the supra- and subgingival microbial infection (Thornton & Garnick 1982, Breininger et al. 1987) as well as the major risk indicators for periodontal disease, such as increased probing pocket depths (PPD) and bleeding on probing scores (Greenstein 2000, Drisko 2001, Hallmon & Rees 2003).

Recolonization of the subgingival area after professional instrumentation may be affected by the effectiveness of self-performed plaque control regimen adopted by the patient. The adjunctive daily use of an antimicrobial agent to mechanical plaque control may result in a retarded subgingival recolonization (Quirynen et al. 1995, 2000, Bollen et al. 1998). In this respect, we have demonstrated previously that the use of an amine fluoride/stannous fluoride (AmF/SnF_2)-containing mouthrinse as an adjunct to conventional mechanical oral hygiene procedures is effective in controlling the amount of supragingival plaque deposits and related gingival inflammation during SPT in generalized aggressive periodontitis (G-AgP) patients (Guarnelli et al. 2004). We have also shown that UMI associated with the home-based use of AmF/SnF_2-containing mouthrinse and toothpaste determines a significant reduction of supragingival plaque and gingival inflammation in patients affected by G-AgP. Consistently, we have observed a substantial change in the total subgingival bacterial load and a significant reduction of subgingival bacterial pathogens in deep periodontal pockets (Guarnelli et al. 2008). Our results support the use of stringent oral hygiene protocols supplemented by the use of antimicrobial agents to control early subgingival recolonization by periodontal pathogens.

Several studies demonstrated a detrimental effect of smoking on clinical and microbiological parameters, which relate to treatment response following non-surgical therapy (Grossi et al. 1997, Haffajee et al. 1997, Renvert et al. 1998, van Winkelhoff et al. 2001, van der Velden et al. 2003, Darby et al. 2005, Labriola et al. 2005, Heasman et al. 2006, Johnson & Guthmiller 2007). At present, however, limited data are available on patients affected by G-AgP. At the microbiological level, a significantly higher decrease in some periodontal pathogenic species was observed following non-surgical treatment in non-smoker G-AgP patients compared with smoker G-AgP patients. Non-smoker G-AgP patients had also significantly greater reduction in PPD than the respective smokers (Darby et al. 2005). In a recent study, Hughes et al. (2006b) observed a significant association of smoking with a poor response to non-surgical treatment in terms of probing depth reduction and clinical attachment gain. When smoker and non-smoker AgP patients were compared, a higher prevalence of non-responder AgP patients (i.e. patients in whom 30% of their deep sites did not show probing depth reduction) were found in smokers than non-smokers (Hughes et al. 2006a).

The observed diminished treatment response in smoker AgP may call for a more stringent professional and home-based protocol to enhance the level of supra- and subgingival plaque control. It may be speculated that such protocol may incorporate the adjunctive use of an antimicrobial therapy in addition to mechanical instrumentation, particularly when maintaining individuals who are highly susceptible to destructive periodontal diseases such as AgP patients (Guarnelli et al. 2004, 2008). In other words, the use of specific plaque-control strategies supplemented by antimicrobial agents in addition to conventional plaque removal procedures could result beneficial in the SPT of AgP patients, especially when additional risk factors, such as smoking, are present.

The present clinical trial was designed to compare the clinical and microbiological effects of UMI associated to home-care use of AmF/SnF_2-containing mouthrinse and toothpaste in smoker and non-smoker patients affected by G-AgP during a recall session of SPT.

Material and Methods

Study population

Patients were selected among those seeking periodontal care at the Research Centre for the Study of Periodontal and Peri-Implant Diseases, University of Ferrara. The study was conducted between January 2008 (first subject in) and June 2009 (last subject out).

Patients were included if they fulfilled the following inclusion criteria: (i) diagnosis of G-AgP according to the definition by Tonetti & Mombelli (1999); (ii) current smoker ($\geq$ 5 cigarettes per day) or non-smoker (never smoked); (iii) systemically healthy; (iv) active (non-surgical, surgical) phase of periodontal therapy completed; (v) manual or mechanical instrumentation at least 3 months before the experimental phase; (vi) at least one site with PPD $\geqslant$ 5 mm per each quadrant; and (vii) able and willing to provide informed consent and to ensure compliance throughout the study.

Patients were excluded from the study if they met any of the following exclusion criteria: pregnancy or lactation; physical or mental handicap that could interfere with adequate oral hygiene performance; systemic and/or topical steroidal and non-steroidal anti-inflammatory drugs and local and/or topical antimicrobials during the last 6 weeks before the study; systemic and/or topical antibiotics during the last 3 months before and during the study; fixed or removable orthodontic devices; diagnosis of pathologies affecting soft tissues of the oral cavity, excluding G-AgP; previous adverse events or documented allergy following use of oral hygiene products such as chlorhexidine and AmF/SnF_2 mouthrinse or toothpaste; and conditions requiring prophylactic antibiotic coverage before invasive dental procedures.

Experimental procedures

The experimental design and procedures are illustrated in Fig. 1. At day 0 (baseline), each patient received a single session of full-mouth UMI by means of a piezoelectric ultrasonic device (Piezosteril 5; Castellini S.p.A., Castel Maggiore, Bologna, Italy) with periodontal fine tips (Perio Slim Tip; EMS S.p.A., Milan, Italy) followed by supragingival polishing with non-fluoridated prophylaxis paste. At the end of the UMI session, patients were given a toothbrush, inter-proximal cleaning devices, an AmF/SnF_2-containing mouthrinse and toothpaste (Meridol® mouthrinse/toothpaste, GABA International AG, Therwil, Switzerland). Personalized oral hygiene instructions (OHI) were verbally provided. The use of AmF/SnF_2-containing mouthrinse and toothpaste was prescribed for 12 weeks. The mouthrinse was prescribed at 10 ml twice daily (after morning and evening toothbrushing) for 60 s. The toothpaste was prescribed three times a day (morning, afternoon and night).

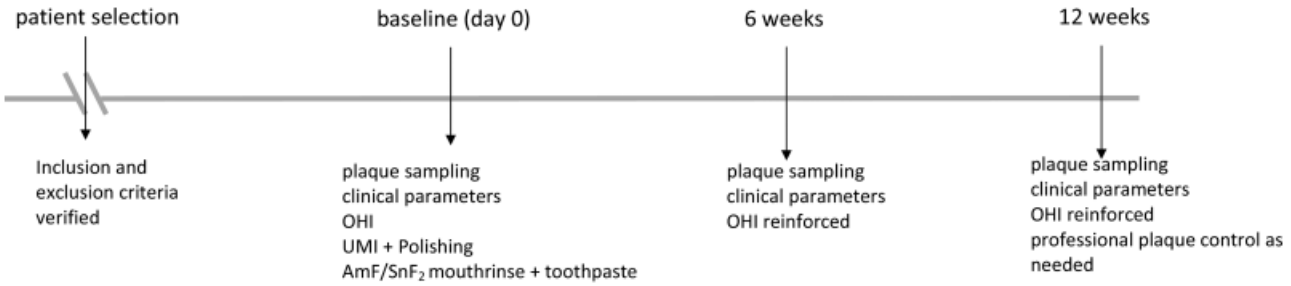

Fig. 1. Experimental design and procedures. UMI, ultrasonic mechanical instrumentation; OHI, oral hygiene instructions; AmF/SnF_2, amine fluoride/stannous fluoride.

At week 6 and 12 weeks following the UMI session, OHI were reinforced. At week 12, periodontal debridement and prophylaxis were provided as needed for plaque/calculus/stain elimination (Fig. 1).

Subgingival plaque sampling and microbiological analysis

Immediately before the UMI session (baseline) as well as after 6 and 12 weeks, subgingival plaque samples were collected at four sites, one for each quadrant, with PPD $\geqslant$ 5 mm. Sites were selected at baseline and kept consistent at 6 and 12 weeks. At these sites, PPD was recorded at each observation interval by means of a standard periodontal probe (UNC 15, Hu Friedy, Chicago, IL, USA) with a manual pressure of approximately 25 g.

Plaque samples were collected according to a standardized procedure (Meridol® Perio Diagnostics, GABA International AG). First, sites to be sampled were isolated with cotton rolls and dried with air to minimize saliva contamination. A sterile paper point was inserted subgingivally at each site and left in place for 15 s. Then, the samples were pooled, immediately transferred into a sterile transport tube and sent to a specialized laboratory (Carpegen GmbH, Münster, Germany).

Real-time PCR was used for detection and quantification of total bacterial counts (TBC), as well as the counts of periopathogenic bacteria such as *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola*, *Fusobacterium nucleatum* and *Prevotella intermedia*. The level of detection was set at 10^2 bacteria/plaque sample. The microbiological analysis was performed blinded as to study design, smoking status and observation interval.

Clinical recordings

At baseline as well at week 6 and 12, the following periodontal parameters were recorded in the following chronological order:

a. Gingival index (GI) (Löe & Silness 1963): GI was recorded at six sites (mesio-buccal, buccal, disto-buccal, mesio-lingual, lingual, disto-lingual) on the following teeth: maxillary right first molar, maxillary right central incisor, maxillary left first premolar, mandibular left first molar, mandibular left first central incisor, mandibular right first premolar. When one of these teeth was absent, the adjacent distal tooth was used for recordings (Guarnelli et al. 2004, 2008).
b. Plaque index (PlI) (Turesky et al. 1970): A disclosing agent (Red Cote®, Butler, Montvale, NJ, USA) was used to visualize supragingival plaque deposits. PlI was recorded at the same teeth and sites where GI was recorded.

Statistical analysis

Data were analysed using JMP v.7 (SAS Institute Inc., Cary, NC, USA) and MedCalc v.11, MedCalc Software, Mariakerke, Belgium). The patient was regarded as the statistical unit; therefore, the clinical recordings assessed in the representative (for GI and PlI) or sampled (for PPD) sites for each patient were averaged to obtain patient-based values. The four pooled paper points as collected at each observation interval were processed together to give patient-based bacterial counts. Before the analysis, bacterial counts were transformed into logarithms (base 10). In each patient, total pathogens (TP) were calculated by adding the counts of *A. actinomycetemcomitans*, *P. gingivalis*, *T. forsythia*, *T. denticola*, *F. nucleatum* and *P. intermedia* while ''red complex species'' (RC) by adding the counts of *P. gingivalis*, *T. forsythia* and *T. denticola*.

Kolmogorov–Smirnov goodness-of-fit tests were computed for each variable to assess whether the variables were distributed normally. Because variables were not distributed normally, all data from clinical parameters and microbiological analysis were expressed as median and inter-quartile range. To test the effect of ''time'' and ''smoking status'' on response variables, Friedman's test was used. Post hoc multiple comparisons were performed to explore intra- and inter-group differences (Newman–Keuls Student's test). Mann–Whitney's test was performed to explore inter-group differences. The level of significance was set at 5%. The minimum difference, in terms of reduction of baseline value with respect to 12 weeks, that would be needed (with 80% statistical power and $\alpha = 0.05$) for groups to result statistically significant was calculated: 0.395 for PlI reduction; 0.28 for GI reduction; 0.40 (log base 10) for TBC; 0.48 (log base 10) for TP; and 0.69 (log base 10) for RC species.

Results

Study population

Thirteen smokers (mean age: 37.4 $\pm$ 3.5 years; 13 females) and 25 non-smokers (mean age: 36.4 $\pm$ 4.1 years; nine males, 16 females) were included in the study and completed the experimental phase showing optimal compliance with clinical procedures. The mean cigarette consumption for smokers was 14.8 $\pm$ 9.9 cigarettes/day. No variation in the smoking status was recorded

during the study. Smokers and non-smokers were significantly different for gender distribution ($p = 0.02$).

PlI

Table 1 shows the PlI scores in smokers and non-smokers at each observation interval. At baseline, PlI was similar in smokers and non-smokers. During the experimental phase, statistically significant changes in PlI occurred in both smokers and non-smokers ($F = 5.22$, $p = 0.013$, and $F = 6.49$, $p = 0.003$, respectively). In both groups, PlI was significantly lower at 6 weeks compared with baseline, and significantly higher at 12 weeks compared with 6 weeks. Twelve-week PlI was significantly lower than baseline value only in non-smokers. No significant differences in PlI were observed between groups at 6 and 12 weeks.

Table 1. Plaque index (PlI) as assessed at six selected teeth in smokers and non-smokers [median; inter-quartile range (IR)]

	Smokers ($n = 13$) PlI		Non-smokers ($n = 25$) PlI		p-Value for inter-group comparison (Mann–Whitney U-test)
	median	IR	median	IR	
Baseline	1.6	1.09–1.95	1.9	1.18–2.18	0.325
6 weeks	1.1^{*}	0.38–1.42	1.1^{φ}	0.57–1.56	0.758
12 weeks	$1.4^{\#}$	0.49–1.88	$1.2^{\varphi,\psi}$	0.95–1.57	0.579

*Significantly different from baseline value ($p = 0.013$).
#Significantly different from 6-week value ($p = 0.013$).
φSignificantly different from baseline value ($p < 0.001$).
ψSignificantly different from 6-week value ($p = 0.009$).

GI

Statistics for GI are summarized in Table 2. At baseline, GI was 1.0 (0.48–1.22) and 0.9 (0.62–1.10) in smokers and non-smokers, respectively ($p = 0.622$). Statistically significant changes in GI occurred in both smokers and non-smokers throughout the study ($F = 4.30$, $p = 0.025$, and $F = 22.41$, $p < 0.001$, respectively). In both groups, GI was significantly lower at 6 weeks compared with baseline, while it was significantly increased at 12 weeks compared with 6 weeks. Twelve-week GI was significantly lower compared with baseline value only in non-smokers. No significant differences in GI were observed between groups at 6 and 12 weeks.

Table 2. Gingival index (GI) as assessed at six selected teeth in smokers and non-smokers group [median; inter-quartile range (IR)]

	Smokers ($n = 13$) GI		Non-smokers ($n = 25$) GI		p-Value for inter-group comparison (Mann–Whitney U-test)
	median	IR	median	IR	
Baseline	1.0	0.48–1.22	0.9	0.62–1.10	0.622
6 weeks	0.4^{*}	0.17–0.78	0.5^{φ}	0.10–0.74	0.841
12 weeks	$0.8^{\#}$	0.27–1.07	$0.6^{\psi,\theta}$	0.17–0.92	0.712

*Significantly different from baseline value ($p = 0.025$).
#Significantly different from 6-week value ($p = 0.007$).
φSignificantly different from baseline value ($p < 0.001$).
ψSignificantly different from 6-week value ($p < 0.001$).
θSignificantly different from baseline value ($p = 0.006$).

PPD at sampled sites

Descriptive statistics of PPD at sites sampled for microbiological analysis are summarized in Table 3. During the experimental phase, a statistically significant decrease in PPD occurred in non-smokers ($F = 9.73$, $p < 0.001$), but not in smokers. In non-smokers, PPD was significantly lower at 6 and 12 weeks compared with baseline.

When groups were compared, a significantly lower PPD was observed in non-smokers at 6 weeks.

Table 3. Probing pocket depth (PPD) as assessed at four microbiologically sampled sites in smokers and non-smokers [median; inter-quartile range (IR)]

	Smokers ($n = 13$) PPD		Non-smokers ($n = 25$) PPD		p-Value for inter-group comparison (Mann–Whitney U-test)
	median	IR	median	IR	
Baseline	6.0	5.25–6.75	5.5	5.25–5.50	0.068
6 weeks	5.5	5.13–6.38	5.0^{φ}	4.13–5.38	0.010
12 weeks	5.5	4.63–6.00	5.0^{φ}	4.13–5.50	0.228

φSignificantly different from baseline value ($p < 0.001$).

TBC

TBC significantly decreased over time in smokers and non-smokers ($F = 4.90$, $p = 0.016$, and $F = 5.53$, $p = 0.007$, respectively) (Fig. 2). In non-smokers, TBC was significantly higher at baseline than 6 ($p = 0.011$) and 12 weeks ($p = 0.001$).

When groups were compared, a significantly lower TBC was observed in non-smokers at 12 weeks ($p = 0.043$).

TP

In both groups, treatment resulted in a significant reduction of TP throughout the study ($F = 6.78$, $p = 0.005$, and $F = 3.27$, $p = 0.047$, respectively) (Fig. 3). In both smokers and non-smokers, TP was significantly higher at baseline than 6 ($p = 0.022$ for smokers, and $p = 0.046$ for non-smokers) and 12 weeks ($p = 0.027$ for smokers, and $p = 0.006$ for non-smokers).

No significant difference in TP was found between smokers and non-smokers at each observation interval.

RC

A significant change in RC was observed only in smokers ($F = 7.12$,

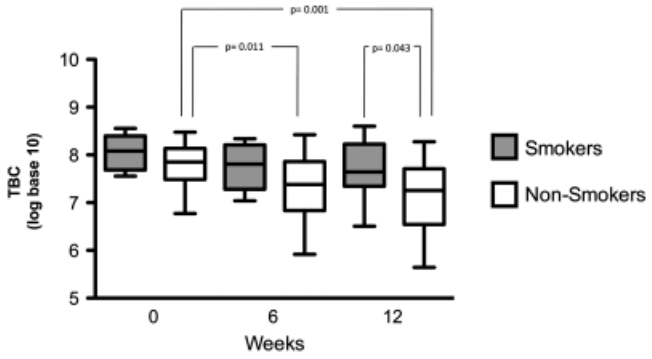

Fig. 2. Total bacterial count (TBC; logarithms base 10) in smokers ($n = 13$) and non-smokers ($n = 25$) assessed at four sampled sites $\geqslant 5$ mm at baseline (week 0), 6 and 12 weeks (Box–Whisker plot).

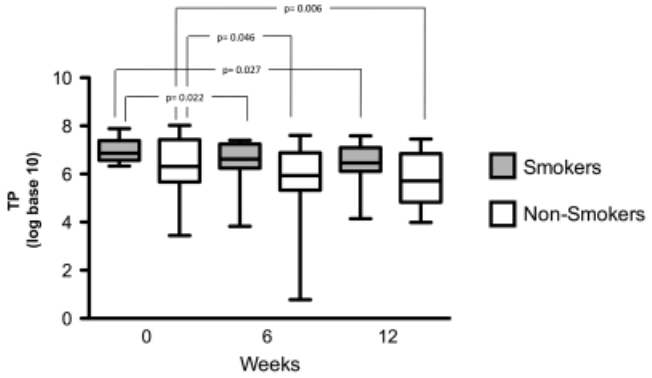

Fig. 3. Total pathogens (TP; logarithms base 10) (counts for *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola*, *Fusobacterium nucleatum*, *Prevotella intermedia*) in smokers ($n = 13$) and non-smokers ($n = 25$) assessed at four sampled sites $\geqslant 5$ mm at baseline (week 0), 6 and 12 weeks (Box–Whisker plot).

$p = 0.004$) (Fig. 4). In smokers, RC was significantly reduced from baseline to both 6 ($p = 0.022$) and 12 weeks ($p = 0.027$). In non-smokers, 12 week RC was significantly lower than baseline value ($p = 0.008$).

No significant difference in RC was found between smokers and non-smokers at each observation interval.

A. actinomycetemcomitans

A. actinomycetemcomitans was only detected in a small number of patients at each observation interval. Low prevalence prevented any statistical comparison both within and between groups.

Discussion

The present clinical trial was designed to compare the clinical and microbiological effects of UMI associated to home-care use of AmF/SnF_2-containing mouthrinse and toothpaste in smoker and non-smoker patients affected by G-AgP during a recall session of SPT. Thirteen smoker and 25 non-smoker G-AgP patients enrolled in an SPT programme received a single session of UMI associated with the use of AmF/SnF_2-containing mouthrinse and toothpaste. In both groups, PlI and GI showed a significant reduction at 6 weeks with respect to baseline; however, 12-week values were significantly lower than baseline only in non-smokers. Treatment also resulted in a significant decrease of TBC, TP and RC, with non-smokers exhibiting a lower TBC compared with smokers at 12 weeks. No significant differences in PlI, GI, TP and RC species were observed between smokers and non-smokers at each observation interval.

In our material, a significant reduction in both supragingival plaque and associated gingival inflammation was obtained at 6 weeks after treatment for both smokers and non-smokers. These results may be ascribed to the combined effect of the professional plaque removal and the stringent patient-performed regimen aimed at supragingival plaque control. Consistently with these observations, we have demonstrated previously that UMI either with or without additional professional use of CHX may provide a significant reduction of PlI and GI during SPT in G-AgP patients (Guarnelli et al. 2008). In addition, we also observed that the home-based use of an AmF/SnF_2-containing mouthrinse following conventional mechanical oral hygiene procedures in G-AgP patients was more effective in controlling the extent of supragingival plaque and gingivitis compared with a placebo (Guarnelli et al. 2004). These results support the validity of professional plaque removal supplemented by the mechanical/chemical self-performed regimen in the control of supragingival plaque deposits and related gingival inflammation during SPT in G-AgP patients.

Although PlI and GI showed a rebound towards baseline values from 6 to 12 weeks in both groups, 12-week PlI and GI remained significantly lower than baseline only in non-smokers. Interestingly, when a similar professional and self-performed plaque control approach was used in a G-AgP cohort, including smoker and non-smoker patients, PlI and GI were significantly decreased from baseline to 3 weeks and remained similarly low up to 12 weeks post-treatment (Guarnelli et al. 2008). Overall, these findings seem to suggest that smoking may negatively affect the rate of supragingival plaque accumulation as well as the severity of plaque-associated gingival inflammation. However, previous studies failed to show a detrimental effect of smoking on the rate and/or amount of de novo plaque accumulation on tooth surfaces (Swenson 1979, Bergström 1981, MacGregor et al. 1985, Giannopoulou et al. 2003, Salvi et al. 2005). In addition, clinical studies reported limited differences in plaque accumulation and gingival inflammation between smokers and non-smokers at 2–6 months following subgingival instrumentation (Grossi et al. 1997, Renvert et al. 1998, Darby et al. 2005). Therefore, in view of these contrasting results, the need for a more stringent supragingival plaque control regimens in smokers, either implemented with the use of antimicrobials or not, remains to be clarified further.

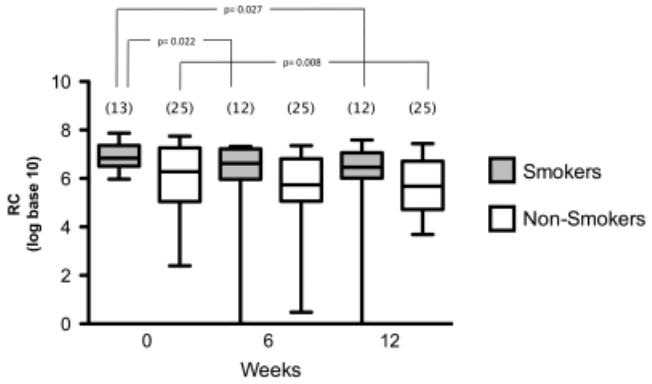

Fig. 4. ''Red complex species'' (RC; logarithms base 10) (counts for *Porphyromonas gingivalis*, *Tannerella forsythia* and *Treponema denticola*) in smokers ($n = 13$) and non-smokers ($n = 25$) assessed at four sampled sites $\geqslant 5$ mm at baseline (week 0), 6 and 12 weeks (Box–Whisker plot). The number in parenthesis represents the number of patients who were positive for at least one of the three bacterial species.

As a result of thorough subgingival UMI, a significant decrease in TBC was observed throughout the study in both smokers and non-smokers. However, 6- and 12-week counts were significantly lower than baseline only in non-smokers. The reduction in TBC up to 12 weeks post-treatment was consistent with our previous findings (Guarnelli et al. 2008), but contrast with those by Salvi et al. (2005). They evaluated the microbiological effects of supragingival polishing and reinstitution of self-performed oral hygiene procedures after 21 days of experimentally induced plaque accumulation in smoker and non-smoker subjects without signs of destructive periodontal disease. After 2 weeks of mechanical plaque control, total DNA probe counts were not significantly abated within either smoker and non-smoker group, without any significant intergroup differences in total DNA counts after treatment (Salvi et al. 2005). Differences between study populations (G-AgP patients *versus* experimental gingivitis patients) may partly account for the discrepancy between studies.

Twelve-week TBC was significantly lower in non-smokers compared with smokers. This finding seems to indicate a faster re-growth of the entire microbial complex in smokers compared with non-smokers. To date, limited evidence is available regarding the influence of smoking on subgingival bacterial recolonization after mechanical debridement. However, previous reports in untreated periodontitis patients showed that, although professional subgingival instrumentation significantly decreased the TBC, smoking did not affect the recolonization of periodontal pockets as assessed at 1 and 2 weeks after treatment (Rhemrev et al. 2006). Differences between our results and those reported previously may partly be explained by the timing selected for microbiological evaluations, the periodontal status of the patient cohorts and the method for microbial sampling and analysis. In this respect, it should be noted that previous studies demonstrated that the residual PPD may influence the prevalence of some bacterial species within weeks following treatment (Magnusson et al. 1984). Therefore, differences in PPD observed between groups at 6 weeks, with smokers exhibiting significantly deeper pockets than non-smokers, may have influenced per se the pattern and rate of subgingival bacterial re-growth after treatment in smokers *versus* non-smokers.

A reduction of TP as well as RC was recorded throughout the study, with no significant difference between smokers and non-smokers at each observation interval. Consistently, previous studies failed to find significant differences in the prevalence of periodontal pathogens between smokers and non-smokers, both in the general population (Boström et al. 2001, Natto et al. 2005) and among patients affected by generalized early-onset periodontitis (Darby et al. 2000). Contrasting evidence, however, is at present available with regard to the effect of smoking on the presence and counts of periodontal pathogens in smokers and non-smokers after professional root instrumentation. While in some studies subgingival pathogens were found to be more difficult to eradicate or reduce in smokers compared with non-smokers (Grossi et al. 1997, Haffajee et al. 1997, Renvert et al. 1998, van Winkelhoff et al. 2001, van der Velden et al. 2003, Darby et al. 2005), other studies reported that mechanical plaque removal decreased the levels of some pathogenic bacterial species similarly in smokers and non-smokers (Preber et al. 1995, Rhemrev et al. 2006). It should be considered that all these studies dealing with the effect of smoking on the microbiological effect of the subgingival instrumentation present with a substantial level of heterogeneity with respect to the patient-performed and professional plaque control regimen adopted.

In the present study, smokers and non-smoker G-AgP patients showed an uneven gender distribution, with smokers having a higher prevalence of females compared to non-smokers. Although this may have introduced a bias in evaluating the effect of the treatment, to the best of our knowledge no evidence are at present available showing whether and to what extent gender may affect the clinical and microbial outcomes of UMI of G-AgP patients under maintenance.

In conclusion, the results of the present study demonstrated that a combined mechanical/chemical plaque control approach based on UMI and the use of AmF/SnF_2 – oral hygiene agents resulted in the reduction of supragingival plaque deposits, gingival inflammation and subgingival periodontal pathogens in G-AgP patients during SPT, with no substantial difference between smokers and non-smokers. Further studies are needed to assess whether and to what extent the additional use of antimicrobials to mechanical oral hygiene procedures may be of some benefit in smoker G-AgP patients during maintenance.

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Clinical Relevance

Scientific rationale: Limited data are available on the effect of smoking status on the microbiological and clinical outcomes of SPT in G-AgP patients.

Principal findings: UMI plus AmF/SnF_2-implemented oral hygiene was similarly effective in reducing plaque accumulation, periodontal pathogens and gingival inflammation in smokers and non-smoker G-AgP patients.

Practical implications: A stringent professional and patient-based plaque control regimen seems to be valuable for a maintenance programme of patients who are highly susceptible to destructive periodontal disease.