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**ORIGINAL ARTICLE**

# Caries experience after periodontal treatment in aggressive and chronic periodontitis: Results of a 10-year follow-up

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**Abstract**

***Objective.*** To compare the increase of DMF-T and DMF-S in patients with aggressive periodontitis (AgP) and chronic periodontitis (ChP) after active periodontal therapy. ***Materials and methods.*** One hundred and thirty-six periodontally treated patients were re-examined after 10 years. Dental and periodontal status was assessed and patients' charts were screened for diagnosis, compliance to supportive periodontal treatment (SPT) and DMF-T/-S at baseline and re-examination. δDMF-T/-S was calculated and multi-level regression analyses were performed to identify factors contributing to increase of DMF-T/-S. ***Results.*** Thirty patients with AgP, 37 with moderate ChP and 69 with severe ChP could be included. δDMF-T between first visit and re-examination was 2.07 (SD = 2.51, range = 0–14 teeth), mean δDMF-S = 14.66 (SD = 14.54, range = 0–83 surfaces). Patients with AgP showed a similar increase in DMF-T/-S to those with ChP. Regression analysis identified compliance as the only factor significantly accounting for preventing an increase of DMF-S ($p = 0.017$). No factor had a significant impact on DMF-T. ***Conclusions.*** DMF-T and DMF-S developed similarly in periodontally-treated patients with AgP and ChP during a follow-up of 10 years. SPT showed a positive influence on avoiding decline in DMF-S in periodontally compromised patients. No significant impact was detected for all other studied factors.

**Key Words:** *aggressive periodontitis, DMF-T, DMF-S, periodontal disease, supportive periodontal therapy (SPT)*

## Introduction

Periodontitis as well as dental caries are common and highly prevalent chronic diseases [1], both with multifactorial pathogenesis caused by a complex interaction between environmental, etiological and host factors as well as genetic predisposition [2,3]. One of the main factors for dental caries and periodontitis is the bacterial colonization of tooth surfaces [4,5], whereby gram-negative bacteria such as Porphyromonas gingivalis or Aggregatibacter actinomycetemcomitans play an essential role in the development of periodontal diseases [6]. In caries development acid production of bacteria in the oral biofilm is a direct causative factor for demineralization of tooth surfaces. Under prolonged acidic conditions the amount of aciduric bacteria, e.g. *Streptococcus mutans*, *lactobazilli*, *Streptococcus sanguinis*, *Streptococcus oralis*, *Streptococcus gordonii*, *Streptococcus mitis*, *Actinomyces* and *Bifidobacteriae*, increases [7].

When periodontal treatment including subgingival debridement or periodontal surgery is conducted, a marked reduction of all periodontal pathogens can be observed and the composition of the bacterial colonization in the oral cavity shifts proportionally towards more gram-positive aerobic bacteria [8].

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In a study by Fine et al. [9], patients with juvenile periodontitis without periodontal therapy showed a lower number of proximal carious lesions than the periodontally healthy control group, which could be confirmed by Sioson et al. [10]. Furthermore, Al-Omari et al. [11] detected a significantly lower caries experience in patients with untreated aggressive periodontitis (AgP) than those with chronic periodontitis (ChP) in groups of age-matched individuals from 20–40 years. Whether this observation proceeds after periodontal treatment in people with different periodontal diagnoses is a subject of debate and to the best of our knowledge not studied so far.

The aim of this retrospective study was to detect if periodontally treated patients with AgP showed a different development of caries and thus elevation of DMF-T/-S (decayed/missed/filled teeth/surfaces) than ChP-patients over a period of 10 years after initial periodontal treatment.

## Materials and methods

All patients included in this retrospective study were treated at the Section of Periodontology at the Department of Conservative Dentistry, Clinic for Oral, Dental and Maxillofacial Disease at the University Hospital Heidelberg between 1993–2000. Clinical examinations were conducted at the start of periodontal therapy. All patients received active periodontal treatment (APT) consisting of anti-infective therapy with subgingival debridement under local anesthesia and, if required, periodontal surgery. Thereafter, patients were scheduled into supportive periodontal therapy (SPT).

After a follow-up time of 10 years all patients fulfilling the following inclusion criteria were re-examined by two investigators blinded for antecedent treatment:

- Existence of a generalized moderate (localized severe) chronic periodontitis (gm(ls)ChP), generalized severe chronic periodontitis (gsChP) or a localized/generalized aggressive periodontitis (l/gAgP),
- Periodontal treatment at the Section of Periodontology at the Department of Conservative Dentistry, Clinic for Oral, Dental and Maxillofacial Disease at the University Hospital Heidelberg 10 years ± 6 month before, and
- ≥ 18 years at re-examination.

Re-examination contained dental and periodontal status, scoring of Gingival Bleeding Index (GBI) [12] and Plaque Control Record (PCR) [13], as well as a test for Interleukin (IL)-1 composite genotype. Furthermore, patients were asked regarding their:

- smoking history (current, former and non-smokers according to Lang and Tonetti [14]),
- marital status (single, married, divorced/widowed),
- educational status (low (< 9 years in school), moderate (apprenticeship, college) and high (university degree)),
- Body weight and height for calculation of the Body-Mass-Index (BMI), and
- Dietary habits.

The study was approved by the Institutional Review Board for Human Studies of the Medical Faculty of Heidelberg University (Application# 331/2002 and 033/2009). All patients were informed on risks and benefits as well as procedures of the study. All gave written informed consent.

### *Evaluation of patients' charts*

The differences of DMF-T and DMF-S as main outcome variables of this study were determined by comparison of the dental status at baseline (examination at start of APT) and re-examination. Retrospectively, a baseline diagnosis (gm(ls)ChP, gsChP or gAgP) was assigned to each patient according to the classification of periodontal diseases [15,16]. Furthermore, regularity of SPT at the Section of Periodontology at the University Hospital Heidelberg was documented. A frequency of at least two visits per year was recommended. If someone had extended the recall interval once over 100% (i.e. returning after 13 months for SPT), the patient was considered as non-compliant, belonging to the irregular SPT group [17]. The mean value for GBI and PCR recorded in each SPT appointment was calculated and evaluation of patients' charts was accomplished by two examiners independently.

### *SPT*

Each SPT-session consisted of assessment of GBI and PCR, re-instruction and re-motivation to an effective individual plaque control, professional tooth cleaning as well as application of a fluoride gel. Twice a year, the dental status was obtained and probing pocket depth (PPD), vertical probing attachment level (PAL-V) and bleeding on probing (BOP) were surveyed at four or six sites per tooth. Sites exhibiting PPD of 4 mm and BOP as well as sites with PPD > 4 mm were scaled subgingivally. When initial caries were detected during SPT the patient was instructed to use fluoride gel once a week, if carious lesions were diagnosed a restorative treatment was recommended and took place at the University Hospital or the patient's family dentist.

### *Statistical analysis*

All data was entered into two data files by two individuals independently. Both data files were compared by subtraction of columns of identical variables.

Table I. Patient's characteristics.

| | Total | gm(ls)ChP | sChP | gAgP |
|---|---|---|---|---|
| | (*n* = 136) | (*n* = 37; 27.2%) | (*n* = 69; 50.7%) | (*n* = 30; 22.1%) |
| Age in years (mean, range) | 54.9 (26–77) | 59.0 ± 9.3 | 57.2 ± 9.6 | 42.5 ± 5.3 |
| Sex (female) | 87 (64.0%) | 20 (54.0%) | 43 (62.3%) | 24 (80.0%) |
| Smoking | | | | |
| Non-smoker | 57 (41.9%) | 19 (51.4%) | 27 (39.1%) | 11 (36.7%) |
| Former smoker | 47 (34.6%) | 12 (32.4%) | 25 (36.2%) | 10 (33.3%) |
| Smoker | 32 (23.5%) | 6 (16.2%) | 17 (24.7%) | 9 (30.0%) |
| Compliance | 72 (53.3%) | 20 (54.0%) | 37 (53.6%) | 15 (50.0%) |
| Educational status | | | | |
| High | 43 (31.6%) | 16 (43.2%) | 19 (27.5%) | 8 (26.7%) |
| Moderate | 65 (47.8%) | 13 (35.1%) | 32 (46.4%) | 20 (66.7%) |
| Low | 5 (3.7%) | 1 (2.7%) | 4 (5.8%) | 0 (0%) |
| No information | 23 (16.9%) | 7 (19.0%) | 14 (20.3%) | 2 (6.6%) |
| Familial status | | | | |
| Single | 9 (6.7%) | 1 (2.7%) | 4 (5.8%) | 4 (13.3%) |
| Married | 83 (61.0%) | 20 (54.0%) | 41 (59.4%) | 22 (73.3%) |
| Divorced/widowed | 20 (14.7%) | 8 (21.6%) | 10 (14.5%) | 2 (6.7%) |
| No information | 24 (17.6%) | 8 (21.6%) | 14 (20.3%) | 2 (6.7%) |
| IL-1 composite genotype (positive) | 44 (33.3%) | 12 (43.2%) | 28 (40.6%) | 4 (13.6%) |
| Mean GBI during follow-up (mean, range) | 7% (0–43%) | 7% | 8% | 4% |
| Mean PCR during follow-up (mean, range) | 25% (2–64%) | 26% | 25% | 23% |
| BMI (mean, range) | 24.5 (18.2–41.7) | 25.2 | 24.8 | 23.2 |

gm(ls)ChP, generalized moderate (localized severe) chronic periodontitis; gsChP, generalized severe chronic periodontitis; gAgP, generalized aggressive periodontitis; IL-1, Interleukin-1; GBI, Gingival Bleeding Index; PCR, Plaque Control Record; BMI, Body-Mass-Index.

If subtraction resulted in values different from null the entries were compared with the original charts and corrected.

The patient was looked upon as a statistical unit and change of DMF-T and DMF-S were defined as main outcome variables.

Descriptive statistics and logistic regression analysis was performed using a computer program (SPSS, Version 18, SPSS Inc., Chicago IL). Poisson regressions were modeled by an independent statistician (PR) using another program (SAS® version 9.1, SAS Institute, Cary, NC).

Using Poisson regression factors should be identified that influenced the dependent variable change of DMF-T and DMF-S. The following independent patient-related variables assessed at re-examination were entered into the model: sex, age, diagnosis at initiation of therapy (moderate (localized severe) chronic periodontitis vs severe chronic vs aggressive periodontitis), IL-1 polymorphism, nicotine consumption (current vs never and former smoking), compliance with recommended SPT attendance, status of oral hygiene according to mean GBI and PCR during SPT, education and marital status. After identification of dichotomous factors, means, standard deviations, medians and ranges for tooth loss of the respective groups were calculated. Third molars were excluded from analysis.

## Results

### *Patients*

One hundred and thirty-six patients (87 female) with a mean age of 54.9 years (range 26–77 years) were included in this study. Thirty-seven patients (27.2%) showed a gm(ls)ChP at baseline, 69 a gsChP (50.7%) and 30 a gAgP (22.1%). Seventy-two were compliant (53.3%), 32 smoked (23.5%) and 47 patients were former smokers (34.6%). Follow-up time was 10 years ± 6 months. Mean GBI during follow-up reached 7% (range 0–43%) and mean PCR 25% (range 2–64%); no differences in mean PCR were found between the two groups (AgP and ChP). Further characteristics such as BMI, IL-1- composite genotype or educational and familial status are given in Table I.

### *DMF-T*

Mean DMF-T at baseline was 16.27 (SD = 5.44, range = 5–28), at re-evaluation 18.34 (SD = 5.31,

Table II. DMF-T/DMF-S at start of APT and at re-examination as well as ÎDMF-T/ÎDMF-S of 10 years follow-up according to different periodontal diagnoses.

| | Total ($n$ = 136) | gm(ls)ChP ($n$ = 37) | sChP ($n$ = 69) | gAgP ($n$ = 30) |
|---|---|---|---|---|
| DMF-T at Baseline (mean ± SD, range) | 16.27 ± 5.44; 5–28 | 15.78 ± 5.63 | 17.62 ± 5.50 | 13.80 ± 4.07 |
| DMF-T at Re-examination (mean ± SD, range) | 18.34 ± 5.31; 5–28 | 17.81 ± 5.19 | 19.74 ± 5.30 | 15.77 ± 4.52 |
| Î DMF-T (mean ± SD, range) | 2.07 ± 2.51; 0–14 | 2.03 ± 2.77 | 2.12 ± 3.31 | 1.97 ± 2.17 |
| DMF-S at Baseline (mean ± SD, range) | 53.89 ± 28.08; 7–128 | 52.92 ± 26.96 | 62.38 ± 28.87 | 35.83 ± 17.54 |
| DMF-S at Re-examination (mean ± SD, range) | 68.55 ± 29.09; 13–128 | 64.97 ± 26.62 | 78.14 ± 28.53 | 50.90 ± 24.34 |
| Î DMF-S (mean ± SD, range) | 14.66 ± 14.54; 0–83 | 12.05 ± 12.27 | 15.76 ± 17.61 | 15.07 ± 15.17 |

gm(ls)ChP, generalized moderate (localized severe) chronic periodontitis; gsChP, generalized severe chronic periodontitis; gAgP, generalized aggressive periodontitis; DMF-T, decayed/missed/filled teeth; DMF-S, destroyed/missed/filled surfaces; SD, Standard deviation.

range = 5–28). Regarding DMF-T, a mean difference of 2.07 (SD = 2.51, range = 0–14) could be found during 10 years of follow-up (Table II). At baseline, patients with AgP showed a lower DMF-T compared to patients diagnosed with gm(ls)ChP or gsChP. However, the amount of destroyed, missing or filled teeth in patients with gm(ls)ChP, gsChP and gAgP developed similarly (mean ÎDMF-T = 2.03 ± 2.77, 2.12 ± 3.31 and 1.97 ± 2.17, see Figure 1) and no statistical significance for diagnosis could be detected ($p$ = 0.958). Further factors such as age, sex, mean PCR and mean GBI during follow-up or smoking failed to show a statistical significant influence on increase of DMF-T (Table III). The only factor contributing to an increase in DMF-T was compliance (1.65 ± 2.06 vs 2.84 ± 3.62 in non-compliers), although it slightly failed statistical significance ($p$ = 0.081).

*DMF-S*

During follow-up, the mean DMF-S increase augmented to 14.66 (SD = 14.54, range = 0–83) (Table II). As described for DMF-T, mean DMF-S at baseline was significantly lower in patients with AgP (35.83 ± 17.54) compared to those with ChP (gm(ls)ChP: 52.92 ± 26.96 and gsChP: 62.38 ± 28.87). During SPT, patients with gsChP presented the most severe increase of DMF-S with a mean rise of 15.76 ± 17.61 (Table II), but no statistically significant differences regarding increase of DMF-S could be found in patients with different periodontal diagnoses (Figure 2). On the other hand, compliance showed a statistically significant impact on DMF-S increase. In non-compliant patients, DMF-S increased significantly during follow-up compared to compliant patients (20.06 ± 19.83 vs 10.82 ± 9.45, $p$ = 0.038). As for DMF-T, no statistically significant influence on DMF-S increase could be detected for the factors sex, age, IL-1 composite genotype, mean PCR and mean GBI as well as educational and familial status during 10 years of follow-up (Table IV).

By excluding the 'M'-component, DF-S at baseline was 36.14 and at re-evaluation 39.80, resulting in a ÎDF-S of 3.66.

## Discussion

Former studies focusing on patients with juvenile periodontitis (according to the classification before 1999) observed that these patients carry a lower risk for proximal caries compared to healthy subjects in the same age group [9,10]. Furthermore, Al-Omari et al. [11] detected significantly less carious lesions or restorative treatment in patients aged 20–40 years with AgP than in those with ChP. In our study, a statistically significant difference between AgP and ChP-patients regarding DMF-T as well as DMF-S before APT could be confirmed in favor of AgP-patients.

However, it has not been investigated so far in which way caries experience develops in patients with AgP

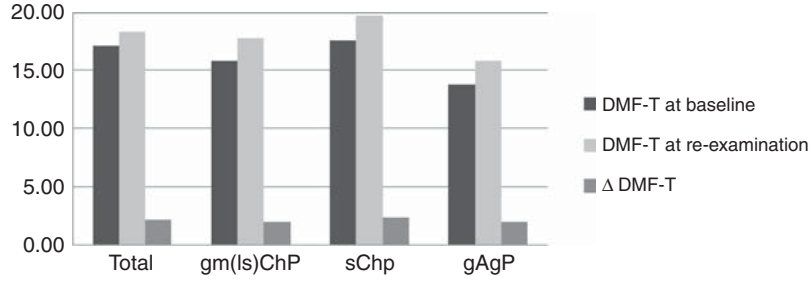


Figure 1. DMF-T at start of APT, at re-examination and δDMF-T of 10 years follow-up according to periodontal diagnoses.

Table III. Poisson regression analyses regarding different factors influencing ÎDMF-T after 10 years of follow-up.

| | Estimate | SE | *t* | *p* |
|---|---|---|---|---|
| Intercept | 20.542 | 35.482 | 0.58 | 0.5641 |
| Sex (female) | 0.6205 | 0.6418 | 0.97 | 0.3363 |
| Age | 0.0042 | 0.0344 | 0.12 | 0.9029 |
| Smoking | | | | |
| Non-smoker | 0.2606 | 0.7308 | 0.36 | 0.7223 |
| Former smoker | 0.3881 | 0.7388 | 0.53 | 0.6006 |
| Current smoker | 0 | — | — | — |
| Diagnosis | | | | |
| gm(ls)ChP | 0.0809 | 0.6530 | 0.12 | 0.9016 |
| gsChP | 0 | — | — | — |
| gAgP | 0.1961 | 0.8507 | 0.23 | 0.8182 |
| Compliance | 1.0401 | 0.5890 | 1.77 | 0.0809 |
| Mean GBI | 2.4404 | 5.5978 | 0.44 | 0.6639 |
| Mean PCR | 0.9384 | 3.1699 | 0.30 | 0.7679 |
| IL-1 composite genotype (negative) | 0.4167 | 0.6115 | 0.68 | 0.4973 |
| BMI | 0.0633 | 0.0892 | 0.71 | 0.4798 |
| Educational status | | | | |
| High | 0.1857 | 1.4671 | 0.13 | 0.8995 |
| Moderate | 0.4123 | 1.4683 | 0.28 | 0.7795 |
| Low | 0 | — | — | — |
| Familial status | | | | |
| Single | 1.2487 | 1.7431 | 0.72 | 0.4757 |
| Married | 0.1415 | 1.4445 | 0.10 | 0.9222 |
| Divorced/widowed | 0.3286 | 1.5613 | 0.21 | 0.8338 |

gm(ls)ChP, generalized moderate (localized severe) chronic periodontitis; gsChP, generalized severe chronic periodontitis; gAgP, generalized aggressive periodontitis; IL-1, Interleukin-1; GBI, Gingival Bleeding Index; PCR, Plaque Control Record; BMI, Body-Mass-Index.

compared to ChP after periodontal treatment. Therefore, we tried to detect the increase of DMF-T and DMF-S in patients with AgP and ChP after active periodontal treatment over a long-term period.

Consistent with the results reported by Al-Omari et al. [11], in our study patients with AgP showed significantly less DMF-T and DMF-S at baseline compared to patients with moderate or severe ChP. While the 542 subjects examined in the mentioned study were age-matched, our groups had different mean ages at baseline: patients with AgP were on average aged 32 years, whereas patients with moderate ChP were 49 and those with severe ChP 47 years of age on average. As seen in 'Deutsche Mundgesundheitsstudie IV' (DMS IV) [1], DMF-T as well as DMF-S increases significantly with age and, therefore, our results have to be interpreted with care, although they point in the same direction as the study noted above. Furthermore, by comparing data of 35–44 years old participants of the DMS IV [1] with a mean DMF-T of 14.5, slightly higher results can be found in our younger patients with AgP (mean age 32 years at baseline and a DMF-T of 13.8).

However, during the 10 years of follow-up, DMF-T and DMF-S increased similarly in all three groups and no statistical differences could be observed regarding the different diagnoses of AgP and ChP.

The only statistically significant factor minimizing the increase of DMF-S was compliance with SPT. The same tendency could be detected for DMF-T, but without statistical significance. In patients non-compliant with recommended SPT intervals, DMF-T increased almost 60% more (1.6-fold) than in compliant patients (ÎDMF-T of 1.65 in compliers vs ÎDMF-T of 2.84 in non-compliers). The fact that regularity of maintenance impedes not only further attachment and tooth loss but also deters caries experience is well known and has been demonstrated over long-term periods up to 30 years in patients with ChP [2,18,19]. Our results affirm these findings in patients with AgP for the first time. It can be concluded that, in AgP- as well as ChP-patients, regular maintenance contributed to a significantly improved dental status in general.

For other factors such as educational status, familial status, IL-1 composite genotype, BMI or mean GBI

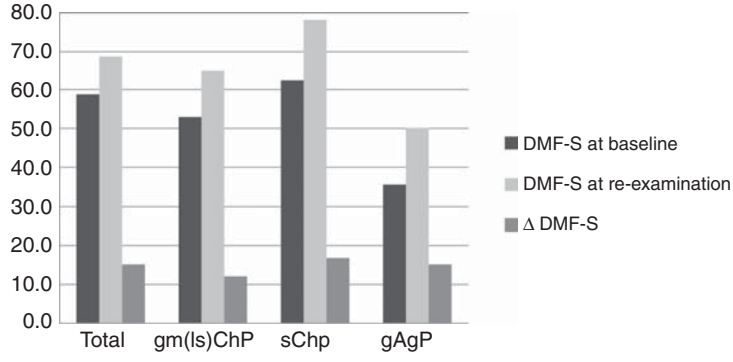


Figure 2. DMF-S at start of APT, at re-examination and δDMF-S of 10 years follow-up according to periodontal diagnoses.

Table IV. Poisson regression analyses regarding different factors influencing ÎDMF-S after 10 years of follow-up.

| | Estimate | SE | *t* | *p* |
|---|---|---|---|---|
| Intercept | 14.6031 | 192.525 | 0.76 | 0.4502 |
| Sex (female) | 0.4773 | 3.4822 | 0.14 | 0.8913 |
| Age | 0.0215 | 0.1864 | 0.12 | 0.9086 |
| Smoking | | | | |
| Non-smoker | 3.9246 | 3.9653 | 0.99 | 0.3250 |
| Former smoker | 2.9620 | 4.0085 | 0.74 | 0.4619 |
| Current smoker | 0 | — | — | — |
| Diagnosis | | | | |
| gm(ls)ChP | 3.0051 | 3.5431 | 0.85 | 0.3987 |
| gsChP | 2.6747 | 4.6157 | 0.58 | 0.5638 |
| gAgP | 0 | — | — | — |
| Compliance | 6.7498 | 3.1959 | 2.11 | 0.0376 |
| Mean GBI | 13.9279 | 30.3735 | 0.46 | 0.6477 |
| Mean PCR | 6.0486 | 17.1997 | 0.35 | 0.7259 |
| IL-1 composite genotype (negative) | 1.0210 | 3.3178 | 0.31 | 0.7590 |
| BMI | 0.0915 | 0.4837 | 0.19 | 0.8504 |
| Educational status | | | | |
| High | 2.5661 | 7.9606 | 0.32 | 0.7480 |
| Moderate | 4.6627 | 7.9670 | 0.59 | 0.5599 |
| Low | 0 | — | — | — |
| Familial status | | | | |
| Single | 9.3840 | 9.4579 | 0.99 | 0.3239 |
| Married | 3.4330 | 7.8378 | 0.44 | 0.6625 |
| Divorced/widowed | 1.0142 | 8.4714 | 0.12 | 0.9050 |

gm(ls)ChP, generalized moderate (localized severe) chronic periodontitis; gsChP, generalized severe chronic periodontitis; gAgP, generalized aggressive periodontitis; IL-1, Interleukin-1; GBI, Gingival Bleeding Index; PCR, Plaque Control Record; BMI, Body-Mass-Index.

no significant influence regarding increase of DMF-T or DMF-S could be observed after APT in patients with ChP and AgP. Furthermore, a higher mean PCR during SPT showed no relevance for developing an increased risk for caries. Caries is caused by dental biofilms. So why does PCR as a plaque index not correlate with increase of DMF? The PCR is scored to explain gingival inflammation. Thus, only bacterial deposits near the gingival margin are considered. In periodontally treated patients the gingival margin is likely to have receded apically. Inter-proximal and occlusal crown surfaces are not scored for plaque by PCR.

In a study by Al-Habashneh et al. [20] an elevated mean DMF-T was detected in smokers compared to non-smokers. This tendency could not be confirmed in our patients at baseline: smokers showed a mean DMF-T of 15.61 ± 5.87 and non-smokers of 16.11 ± 5.06, respectively. During maintenance, smoking did not exhibit a statistically significant effect on DMF-T or DMF-S increase, either.

Several limitations of the study have to be kept in mind: The possible over-estimation of caries when using the DMF-T has been criticized before, especially in periodontally compromised patients the 'missing' teeth could have been lost due to periodontal and not caries reasons [21,22]. Although sometimes discussed critically, the DMF-T and DMF-S still represent standard indexes for assessment of dental caries and were therefore used in this analysis [23]. Beside the possible over-estimation by using the DMF-T/-S index, the study groups were not distributed evenly according to mean age (younger patients in AgP-group) and initial diagnosis (AgP less prevalent than ChP) as mentioned above.

## Conclusion

During a follow-up time of 10 years DMF-T and DMF-S developed similarly in periodontally treated patients with AgP and ChP. Regularity of SPT proved a statistically significant positive effect on avoiding increase in DMF-S in periodontally compromised patients. For all other included factors no statistically significant impact could be demonstrated. Thus, a positive long-term influence not only on the periodontal but also on the dental status in general might be attributed to regular SPT.

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## References

[1] Micheelis W, Schiffner U. Vierte Deutsche Mundgesundheitsstudie (DMS IV). In Institut für deutsche Zahnärzte (IDZ Materialreihe Band 31). Köln: Deutscher Ärzte-Verlag; 2006.
[2] Chambrone LA, Chambrone L. Results of a 20-year oral hygiene and prevention program on caries and periodontal disease in children attended at a private periodontal practice. Int J Dent Hygiene 2011;9:155–8.
[3] Kinane DF, Bartold PM. Clinical relevance of the host response of periodontitis. Periodontol 2000 2007;43:278–93.
[4] Löe H, Theilade E, Jensen S. Experimental gingivitis in man. J Periodontol 1965;36:177–87.
[5] Von der Fehr F, Löe J, Theilade E. Experimental caries in man. Caries Res 1970;4:131–48.
[6] Sokransky SS, Haffajee AD, Cugini MA, Smith C, Kent RL. Jr. Microbial complexes in subgingival plaque. J Clin Periodontol 1998;25:134–44.
[7] Takahashi N, Nyvad B. The role of bacteria in the Caries Process: ecological perspectives. J Dent Res 2011;90:294–303.

[8] Haffajee AD, Teles RP, Sokransky SS. The effect of periodontal therapy on the composition of the subgingival microbiota. Periodontol 2000 2003;43:7–12.
[9] Fine DH, Goldberg D, Karol R. Caries levels in patients with juvenile periodontitis. J Periodontol 1984;55:242–6.
[10] Sioson PB, Furgang D, Steinberg LM, Fine DH. Proximal caries in juvenile periodontitis patients. J Periodontol 2000;71: 710–16.
[11] Al-Omari MA, Al-Habashneh R, Taani DQ. Dental caries experience in patients with aggressive periodontitis compared to those with chronic periodontitis. Quintessenz Int 2008;39: 847–51.
[12] Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. In Dent J 1975 25:229–35.
[13] O'Leary TJ, Drake RB, Naylor JE. The plaque control record. J Periodontol 1972;43:38.
[14] Lang NP, Tonetti MS. Periodontal risk assessment (PRA) for patients in supportive periodontal therapy (SPT). Oral Health Prevent Dent 2003;1:7–16.
[15] Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999; 4:1–6.
[16] Lang N, Bartold PM, Cullinan M, Jeffcoat M, Mombelli A, Murakami S, et al. Consensus report: aggressive periodontitis. Ann Periodontol 1999;4:53.
[17] Eickholz P, Kaltschmitt J, Berbig J, Reitmeir P, Pretzl B. Tooth loss after active periodontal therapy. 1: patient-related factors for risk, prognosis, and quality of outcome. J Clin Periodontol 2008;35:165–74.
[18] Axelsson P, Nyström B, Lindhe J. The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults. Results after 30 years of maintenance. J Clin Periodontol 2004;31:749–57.
[19] Chambrone LA, Chambrone L. Tooth loss in well maintained patients with chronic periodontitis during long-term supportive therapy in Brazil. J Clin Periodontol 2006;33:759–64.
[20] Al-Habashneh R, Al-Omari MAO, Taani DQ. Smoking and caries experience in subjects with various form of periodontal diseases from a teaching hospital clinic. Int J Dent Hygiene 2008;7:55–61.
[21] Lawrence HP, Beck JD, Hunt RJ, Koch GG. Adjustment of the M-component of the DMFS index for prevalence studies of older adults. Community Dent Oral Epidemiol 1996;24: 322–31.
[22] Mack F, Mojon P, Budtz-Jørgensen E, Kocher T, Splieth C, Schwahn C, et al. Caries and periodontal disease of the elderly in Pomerania, Germany: results of the Study of Health in Pomerania. Gerodontology 2003;21:27–36.
[23] World Health Organisation. Oral Health Surveys - basic methods. 4th ed. Geneva: World Health Organisation; 1997.