

The effect of non-surgical periodontal therapy on peroxidase activity in diabetic patients: a case–control pilot study

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Gonçalves D, Correa FOB, Khalil NM, de Faria Oliveira OMM, Orrico SRP. The effect of non-surgical periodontal therapy on peroxidase activity in diabetic patients: a case–control pilot study. *J Clin Periodontol* 2008; 35: 799–806. doi: 10.1111/j.1600-051X.2008.01289.x.

Abstract

Objective: To evaluate the effect of periodontal therapy on clinical parameters as well as on total salivary peroxidase (TSP) activity and myeloperoxidase (MPO) activity in the gingival crevicular fluid (GCF) of patients with type 2 diabetes mellitus (DM2) and of systemically healthy individuals.

Material and Methods: Twenty DM2 subjects with inadequate metabolic control (test group) and 20 systemically healthy individuals (control group), both groups with chronic periodontitis, were enrolled. Periodontal clinical parameters, namely periodontal probing depth (PD), clinical attachment level (CAL), visible plaque index (VPI), bleeding on probing (BOP), gingival bleeding index (GBI) and presence of suppuration (SUP), as well as TSP activity and GCF MPO activity, were assessed before and 3 months after non-surgical periodontal therapy.

Results: At baseline and 3 months post-treatment, the test group presented a higher percentage of sites with VPI and BOP ($p < 0.01$). MPO activity in the GCF presented lower values ($p < 0.05$) for the test group at both baseline and the post-treatment period. The periodontal treatment resulted in a significant improvement of most clinical and enzymatic parameters for both groups ($p < 0.05$).

Conclusions: In both groups, the periodontal therapy was effective in improving most clinical parameters and in reducing salivary and GCF enzymatic activity. The diabetic individuals presented lower MPO activity in the GCF.

Key words: diabetes mellitus; glycaemic control; myeloperoxidase; salivary peroxidase; periodontal disease/therapy

Accepted for publication 6 June 2008

Conflict of interest and source of funding statement

The authors declare that they have no conflict of interests.

This study was supported in part by grants from the Coordination for Postgraduate Courses in Higher Education (CAPES, Brazil), by the São Paulo State Research Foundation (FAPESP, Brazil; Process #04/08142-0) and by the Foundation for the Development of UNESP (FUNDU-NESP, Brazil; Process #00006/05-DFP).

Immunoinflammatory reaction represents a host's response to microbiota and its products. In sites with active periodontal disease, it is clinically manifested by a series of alterations, including an increased flow of gingival crevicular fluid (GCF) (Kinane & Lappin 2001). For this reason, there has currently been a great interest in the diagnostic potential of GCF.

Saliva and GCF have several substances associated to the host's defence,

and their increased levels may be related to the periodontal status, which might allow for identifying and monitoring patients with potential periodontitis progression (Lamster 1997, Armitage 2004). The salivary peroxidase system is one of the most important non-immunological defence systems in saliva. It consists of salivary peroxidase (SPO), originating from the parotid glands; leucocyte-derived myeloperoxidase (MPO), originating from the GCF; and

other components (Tenovuo & Pruitt 1984).

SPO contributes to the maintenance of oral health in several ways. This enzyme has a bactericidal action that protects the teeth and oral mucosa by regulating the oral microbial species and their number (Mandel 1989). It also prevents the accumulation of toxic products from hydrogen peroxide (H₂O₂) and inactivates several cariogenic and mutagenic compounds (Tenovuo & Pruitt 1984, O'Brien 2000). SPO activity increases in the presence of gingival inflammation, returning to normal levels when oral health is restored (Smith et al. 1984).

Another important enzyme, MPO, may be detected both in saliva and in the GCF. It is the most abundant protein in the primary neutrophil granules, and it is also found in monocytes, although in a lesser amount. MPO catalyses the conversion of H₂O₂ and chloride (Cl⁻) into hypochlorous acid (HOCl), which is a potent oxidant produced by neutrophils, with a strong anti-microbial activity (O'Brien 2000). The relative contribution of MPO to total salivary peroxidase (TSP) activity depends on the presence of periodontal or oral inflammation (Smith et al. 1984).

GCF MPO may have great potential as a periodontal disease marker (Karhuvaara et al. 1990, Smith et al. 1992, Jentsch et al. 2004, Aras et al. 2007, Borges et al. 2007). It has been demonstrated that there is an increase in the MPO level in the GCF in systemically healthy patients with periodontitis (Smith et al. 1986, Cao & Smith 1989, Yamalik et al. 2000, Wei et al. 2004). Both conventional (Smith et al. 1986, Wolff et al. 1988) and surgical periodontal treatment (Buchmann et al. 2002, Jentsch et al. 2004) may produce a significant decrease in MPO activity in the GCF.

Several factors may act to modify the host's response to aggressive agents and hence alter periodontitis expression and progression. Diabetes mellitus has been recognized as an important risk factor for periodontitis (Shlossman et al. 1990, Safkan-Seppala & Ainamo 1992, Löe 1993, Lalla et al. 2007a). The greater severity and prevalence of periodontal disease in diabetic subjects may be related to GCF alterations, vascularization of periodontal tissues, host's immunoinflammatory response, collagen metabolism and genetic patterns (Manouchehr-Pour & Bissada 1983, Oliver & Tervonen

1994, Mealey & Oates 2006). Studies evaluating the immunoinflammatory response in diabetic subjects have shown greater cytokine secretion associated to periodontitis progression in these patients (Salvi et al. 1998, Engebretson et al. 2006, Navarro-Sanchez et al. 2007). Although several non-immunological markers from GCF may be evaluated (Armitage 2004), β -glucuronidase, aspartate aminotransferase, elastase and metalloproteinases (MMP-8 and MMP-9) have been the markers assessed most often in diabetic subjects (Smith et al. 1996, Alpagot et al. 2001, Engebretson et al. 2006, Kumar et al. 2006). Regarding peroxidases, studies evaluating the saliva of patients with type 1 and type 2 diabetes mellitus (DM1 and DM2) *versus* healthy individuals have reported greater SPO (Tenovuo et al. 1986, Guven et al. 1996, Dodds et al. 2000) and MPO (Dodds et al. 2000) activities in the diabetic subjects. Other authors have not found differences in SPO (Aren et al. 2003) or MPO (Tenovuo et al. 1986) activity in this group of patients.

Few studies have addressed these non-specific defence mechanisms in diabetic individuals, and there is a lack of investigations pertaining to the effect of periodontal treatment on these enzymatic parameters. Therefore, the purpose of this study was to evaluate the effect of non-surgical periodontal therapy on clinical parameters as well as on TSP activity and MPO activity in the GCF of DM2 patients with inadequate metabolic control and in systemically healthy individuals, both groups with chronic periodontitis.

Material and Methods

Patients

Patients selected for entry to the study were recruited from the Clinic of Periodontics for Diabetic Patients of the Department of Diagnosis and Surgery of the School of Dentistry of Araraquara, São Paulo State University, Brazil between January 2005 and July 2006.

The sample size was calculated based on previous information from a pilot study recently conducted by our research group, using data relative to the mean difference and standard deviation (SD) between the experimental periods (baseline and 3 months after periodontal treatment) for the clinical and enzymatic parameters of the control

and test groups (unpublished data). It was estimated that with a minimum of 20 subjects *per* group, significant differences in the clinical parameters, TSP activity and GCF MPO activity (primary outcome parameter) would be detected between the study periods (baseline and 3 months after non-surgical periodontal therapy), with 80% statistical power and 95% confidence interval.

The study population was composed of 40 individuals of both sexes aged 30–60 years and presenting chronic periodontitis (AAP 1999). All subjects enrolled in the study presented at baseline a minimum of 15 natural teeth (excluding third molars) and at least four teeth with one or more sites exhibiting periodontal probing depth (PD) ≥ 5 mm, clinical attachment level (CAL) ≥ 4 mm, visible plaque and bleeding on probing (BOP). The following exclusion criteria were considered for both groups: history of antibiotic therapy within the previous 6 months and anti-inflammatory drugs within the previous 3 months; pregnancy or use of contraceptives or any other form of hormone; current smoker or former smoker for < 5 years; and periodontal treatment within the previous 12 months. The study was conducted in full accordance with the applicable ethical principles, including the World Medical Association Declaration of Helsinki, and was independently reviewed and approved by the Ethics in Human Research Committee of the School of Dentistry of Araraquara (UNESP) (Protocol number 85/04). Study purposes and procedures were fully explained to the participants, and written informed consent was obtained from all of them.

Two groups with specific criteria were formed. The *test group* was composed of 20 DM2 patients (ADA 2005) (eight men and 12 women; mean age = 45.80 ± 6.01 years) with inadequate metabolic control (glycated haemoglobin test HbA_{1c} $\geq 7\%$) (UKPDS 1998) and mean diabetes duration of 9.85 ± 7.10 years; the *control group* was composed of 20 systemically healthy patients (10 men and 10 women; mean age = 43.65 ± 6.01 years) without diabetes, as confirmed by a fasting glycaemia test result (≤ 100 mg/dl).

All DM2 patients were under the supervision of an endocrinologist, with no alteration in the diabetes treatment in the last year before the study. Fifty-five per cent of the patients were receiving oral hypoglycaemic agents or dietary control only; 15% were undergoing

insulin therapy and 30% were treated with a combination of insulin and oral hypoglycaemic agents. Seventy per cent of the patients had at least one diabetes-related complication (e.g. hypertension, retinopathy, neuropathy or nephropathy). All the DM2 patients were authorized by their physician to undergo periodontal treatment.

Individuals who required antibiotic prophylaxis before periodontal examination were excluded.

Intra-oral clinical evaluation

All patients were subjected to an intra-oral clinical examination by a single trained calibrated examiner ($\kappa = 0.90$, data not shown).

The following clinical parameters were evaluated in six sites of all teeth, except for the third molars: visible plaque index (VPI) (Ainamo & Bay 1975), gingival bleeding index (GBI) (Ainamo & Bay 1975), PD, CAL, BOP and presence of suppuration (SUP), using the manual periodontal probe from the University of North Carolina (Hu-Friedy[®], Chicago, IL, USA). Clinical examinations were performed at baseline and 3 months after completion of the periodontal treatment.

Saliva collection

Patients were instructed not to consume any type of food within at least 1 h before saliva collection. All collections were performed in the morning. Approximately 2 ml of non-stimulated saliva were collected and stored in labelled 1.5-ml Eppendorf tubes. The samples were centrifuged for 30 min. at $10,000 \times g$ and 4°C and thereafter frozen (-20°C) for analysis of the enzymatic activity after 24 h.

GCF collection

In each individual, four sites with $\text{PD} \geq 5 \text{ mm}$, $\text{CAL} \geq 4 \text{ mm}$ and BOP were randomly chosen in different non-adjacent teeth for GCF collection. The sites to be sampled were isolated with cotton rolls and gently air-dried. Stagnated GCF samples were collected with paper strips (PerioPaper, ProFlow Inc., Amityville, NY, USA) introduced into the periodontal pocket and left for 30 s. GCF sample volume was measured with a calibrated Periotron 8000 equipment (Proflow Inc.). The samples were stored individually in labelled

1.5-ml Eppendorf tubes containing potassium phosphate buffer (0.1 M, pH. 7.0) with cetyltrimethylammonium bromide (3 mM) and kept at 4°C . The laboratorial processing was undertaken within 24 h after collection. Samples visibly contaminated with blood were discarded.

Enzymatic assays

The method described by Nishioka et al. (2003) was adapted to the study conditions for determination of TSP activity and MPO activity in the GCF. The enzymatic activity was determined spectrophotometrically, using guaiacol for analysis of TSP activity and monochlorodimedon (MCD) for analysis of MPO activity.

For analysis of TSP activity, saliva (50 μl), guaiacol (30 mM) and H_2O_2 (1 mM) in potassium phosphate buffer (0.1 M, pH. 7.0) were used. Guaiacol oxidation was evaluated by absorbance increase at the 470 nm wavelength during 120 s. The initial rate of reaction was determined by absorbance variation in seconds. Reactions were performed at 37°C .

GCF MPO activity was determined by the formation of its product, HOCl. For such a purpose, MCD was used, which is converted into dichlorodimedon by the action of HOCl, with a decrease in absorbance at 290 nm wavelength due to MCD chlorination. The kinetic assays were undertaken using 50 μl of GCF diluted in a potassium phosphate buffer (0.1 M) of pH 7.0, with NaCl (140 mM), H_2O_2 (1 mM) and MCD (60 μM), followed by reaction at the 290 nm wavelength. Absorbance decrease was recorded during 120 s at 37°C . The initial rate was determined by the absorbance range in seconds.

The spectrophotometric assays were undertaken in a UV-visible spectrophotometer (8453, Agilent Technologies Inc., Life Sciences and Chemical Analysis, Santa Clara, CA, USA) coupled to a workstation. The readings were performed with a thermostated cell at 37°C in a quartz cuvette with a 1 cm optical pathway. All assays were made in triplicate.

TSP and MPO enzymatic activity units (AUs) were determined based on the following definition: one enzymatic AU equals the amount of enzyme that causes a variation of 0.001 in the absorbance at 470 nm (TSP) or 290 nm (MPO) wavelength under assay condi-

tions. To calculate AU for both TSP and MPO, the values of initial rate of reaction (v_0) obtained from the graphs ($\Delta A_{470 \text{ nm}}$ or $\Delta A_{290 \text{ nm}}$ versus reaction time in seconds) were used.

Glycaemic control

The glycaemic control was evaluated by the concentration of glycated haemoglobin A1c (% HbA1c) using high-performance liquid chromatography (DiaSTAT Hemoglobin A1c Analyzer System, BioRad Laboratories, Hercules, CA, USA). This analysis was performed at baseline and 3 months after periodontal therapy.

Periodontal treatment

After all collections had been done, non-surgical periodontal therapy was performed, comprised of scaling and root planning, oral hygiene instructions and biofilm control. After scaling and root planing, a professional plaque control programme was performed twice a month during 3 months, consisting of supragingival plaque removal and reconstruction of oral hygiene procedures. Thereafter, all patients were clinically re-examined, and new saliva and GCF samples were collected to evaluate the effect of the non-surgical periodontal therapy on the previously assessed clinical and enzymatic parameters.

Statistical analysis

Data analysis was performed using BioStat 3.0 software (Optical Digital Optical Technology, Belém, PA, Brazil), considering the individual as a study unit.

The clinical and enzymatic data were analysed statistically by non-parametric statistical methods. Wilcoxon (intra-group comparison) and Mann-Whitney (intergroup comparison) tests as well as Spearman's correlation were used. A significance level was set at 5%, and a 95% confidence interval was established. The tests were based on the median values with variability measures (25% and 75% quartiles).

For VPI, GBI, BOP and SUP data, the percentage of positive sites was obtained *per* patient, and thereafter a mean value was calculated for the group. For distribution of PD and CAL frequencies, measured in millimetres, the per cent frequency was first obtained *per* patient, and thereafter a mean value

was calculated for the group. GCF volume was expressed as the median value of collected volume (μl).

For analysis of enzymatic activity in the GCF, two values were obtained: enzymatic activity *per site* (AU/site) and enzymatic activity *per μl* of collected GCF sample (AU/ μl). For AU/site, a median value of enzymatic activity in the four sites of each individual was first obtained, and then a median value was calculated for the group. For AU/ μl , the value of enzymatic activity in each site was divided by the volume of GCF collected *per site*, and thereafter a median value was obtained for each individual and for the group.

The changes of clinical and enzymatic parameters 3 months after treatment were evaluated in both groups.

Results

All patients completed all phases of this study. No drop out occurred.

Clinical findings

Medians and quartiles of the clinical parameters recorded for both groups at baseline and 3 months post-treatment are summarized in Tables 1 and 2. At baseline, the test group presented significantly higher per cent values for VPI ($p < 0.001$) and BOP ($p < 0.001$) compared with the control group. The other clinical parameters did not differ significantly between the groups.

In both groups, the non-surgical periodontal treatment promoted a significant reduction in the percentage of sites with VPI, GBI, BOP, PD between 4 and 6 mm, PD ≥ 7 mm and CAL ≥ 7 mm, while the percentage of sites with CAL between 4 and 6 mm increased significantly, probably due to the decrease in the percentage of sites with CAL ≤ 7 mm.

Intergroup comparison at 3 months revealed a higher percentage of sites with VPI ($p < 0.001$), GBI ($p < 0.001$) and BOP ($p = 0.002$) in the test group.

The test group showed a significantly lower percentage of reduction for VPI, GB and BOP compared with the control group (Table 3).

In the test group, the periodontal treatment resulted in a non-significant decrease in the HbA1c ($p = 0.7937$) values from baseline (mean \pm SD: 9.43 ± 1.80) to the 3-month interval (mean \pm SD: 9.03 ± 1.94).

Table 1. Medians and quartiles (25–75%) of the clinical and enzymatic parameters for the control ($n = 20$) and test ($n = 20$) groups at baseline

Clinical and enzymatic parameters	Control group	Test group
VPI (%)*	65.20 (51.16–78.69)	85.30 (81.02–94.80)
GBI (%)	38.90 (21.79–52.43)	40.90 (31.76–61.60)
BOP (%)*	80.90 (68.00–89.16)	94.72 (83.10–100.00)
PD ≤ 3 mm (%)	63.50 (48.00–72.25)	50.50 (25.25–70.25)
PD 4–6 mm (%)	30.50 (22.50–39.25)	40.50 (25.75–52.25)
PD ≥ 7 mm (%)	5.50 (3.00–10.00)	9.50 (2.00–18.00)
CAL ≤ 3 mm (%)	44.50 (30.32–50.75)	18.69 (11.17–46.87)
CAL 4–6 mm (%)	37.59 (35.32–49.13)	44.93 (36.70–50.38)
CAL ≥ 7 mm (%)	17.00 (11.91–21.35)	22.41 (9.12–41.87)
SUP (%)	6.00 (2.00–10.50)	9.50 (4.25–17.25)
TSP activity (AU) ($\times 10^{-3}$)	7.10 (4.60–13.80)	11.00 (3.80–24.70)
MPO activity/site (AU)*	2.41 (1.17–3.46)	0.47 (0.29–0.91)
MPO activity/ μl (AU/ μl)*	0.04 (0.02–0.07)	0.009 (0.006–0.01)
GCF volume (μl)	0.86 (0.68–0.93)	0.77 (0.65–0.96)

*Statistically significant difference between the control and test groups at baseline (Mann–Whitney *U*-test; $\alpha = 5\%$).

VPI, visible plaque index; GBI, gingival bleeding index; BOP, bleeding on probing; PD, probing depth; CAL, clinical attachment level; SUP, presence of suppuration; TSP activity, total salivary peroxidase activity; MPO/site (AU), myeloperoxidase activity in the gingival crevicular fluid (GCF) *per site*; MPO/ μl (AU), myeloperoxidase activity in the GCF *per μl* of collected fluid.

Table 2. Medians and quartiles (25–75%) of the clinical and enzymatic parameters for the control ($n = 20$) and test ($n = 20$) groups at 3 months after periodontal treatment

Clinical and enzymatic parameters	Control group	Test group
VPI (%)*	7.50 (5.88–10.45) ^A	15.00 (10.43–17.80) ^A
GBI (%)*	3.55 (0.00–4.22) ^A	8.10 (5.88–10.26) ^A
BOP (%)*	12.72 (8.62–19.63) ^A	25.60 (19.34–29.45) ^A
PD ≤ 3 mm (%)	90.50 (87.70–96.00) ^A	85.50 (75.25–95.00) ^A
PD 4–6 mm (%)	8.50 (4.00–11.25) ^A	13.00 (5.00–23.25) ^A
PD ≥ 7 mm (%)	0.00 (0.00–0.00) ^A	0.50 (0.00–1.25) ^A
CAL ≤ 3 mm (%)	39.73 (33.68–50.88)	26.58 (13.67–51.01)
CAL 4–6 mm (%)	53.67 (46.61–57.79) ^A	55.11 (40.03–64.77) ^A
CAL ≥ 7 mm (%)	6.24 (1.99–10.42) ^A	10.18 (2.38–19.70) ^A
SUP (%)	0.00 (0.00–0.00) ^A	0.00 (0.00–1.00) ^A
TSP activity (AU) ($\times 10^{-3}$)	3.70 (2.80–6.60) ^A	4.90 (3.90–6.40) ^A
MPO activity/site (AU)*	0.54 (0.29–1.05) ^A	0.24 (0.19–0.36) ^A
MPO activity/ μl (AU/ μl)*	0.01 (0.005–0.02) ^A	0.004 (0.003–0.007) ^A
GCF volume (μl)	0.33 (0.20–0.47) ^A	0.34 (0.21–0.55) ^A

*Statistically significant difference between the control and test groups at 3 months after periodontal treatment (Mann–Whitney *U*-test; $\alpha = 5\%$).

Superscript letter A indicates statistically significant difference between baseline and the 3-month period (Wilcoxon's test; $\alpha = 5\%$).

VPI, visible plaque index; GBI, gingival bleeding index; BOP, bleeding on probing; PD, probing depth; CAL, clinical attachment level; SUP, presence of suppuration; TSP activity, total salivary peroxidase activity; MPO/site (AU), myeloperoxidase activity in the gingival crevicular fluid (GCF) *per site*; MPO/ μl (AU), myeloperoxidase activity in the GCF *per μl* of collected fluid.

The periodontal treatment had no adverse effect in the patients of either group.

Biochemical analyses

There were no statistically significant differences ($p > 0.05$) between the groups with respect to salivary enzymatic activity at either baseline or

3 months. Analysis of enzymatic activity in the GCF showed significantly lower values for the test group ($p < 0.05$) at baseline (MPO activity/site – control group: 2.82 ± 2.47 and test group: 0.84 ± 0.95) and at 3 months (MPO activity/site – control group: 0.94 ± 0.95 and test group: 0.29 ± 0.15). In both groups, the periodontal treatment was effective in reducing the enzymatic

Table 3. Medians and quartiles (25–75%) for the changes of clinical and enzymatic parameters from baseline to 3 months after periodontal treatment for the control ($n = 20$) and test ($n = 20$) groups

Clinical and enzymatic parameters Δ 3 months at baseline	Control group	Test group
VPI (%)*	-86.52 (-91.36/-83.81)	-83.41 (-87.06/-80.04)
GBI (%)*	-91.42 (-100.00/-88.02)	-81.65 (-87.69/-70.15)
BOP (%)*	-82.21 (-90.58/-71.68)	-72.31 (-78.98/-63.42)
PD \leq 3 mm (%)	40.18 (27.34/79.34)	65.19 (30.30/184.29)
PD 4–6 mm (%)	-74.94 (-84.59/-49.84)	-68.42 (-77.34/-51.34)
PD \geq 7 mm (%)	-100.00 (-100.00/-100.00)	-98.21 (-100.00/-74.43)
CAL \leq 3 mm (%)	-2.24 (-16.30/11.21)	0.63 (-5.36/25.52)
CAL 4–6 mm (%)	28.50 (9.17/39.38)	18.06 (-1.10/36.62)
CAL \geq 7 mm (%)	-60.67 (-87.50/-35.29)	-51.52 (-68.82/-33.08)
SUP (%)	-100.00 (-100.00/-100.00)	-100.00 (-100.00/-92.66)
TSP activity (AU) ($\times 10^{-3}$)	-2.38 (-9.91/0.26)	-6.82 (-14.92/1.00)
MPO activity/site (AU)*	-1.16 (-2.53/-0.66)	-0.23 (-0.64/-0.03)
MPO activity/ μ l (AU/ μ l)*	-0.02 (-0.05/-0.01)	-0.01 (-0.01/0.00)
GCF volume (μ l)	-0.41 (-0.61/-0.25)	-0.34 (-0.56/-0.20)
HbA1c (%)	-	-1.71 (-12.18 / 5.63)

*Statistically significant difference between the control and test groups (Mann–Whitney U -test; $\alpha = 5\%$).

VPI, visible plaque index; GBI, gingival bleeding index; BOP, bleeding on probing; PD, probing depth; CAL, clinical attachment level; SUP, presence of suppuration; TSP activity, total salivary peroxidase activity; MPO/site (AU), myeloperoxidase activity in the gingival crevicular fluid (GCF) per site; MPO/ μ l (AU), myeloperoxidase activity in the GCF per μ l of collected fluid; HbA1c, glycated hemoglobin A1c.

activity in saliva and in the GCF ($p < 0.05$) (Tables 1 and 2).

The mean differences (\pm SD) of MPO activity/site between the experimental periods (baseline and 3 months post-treatment) were -1.88 ± 1.90 for the control group and -0.55 ± 0.83 for the test group.

The test group showed a significantly lower reduction of MPO activity compared with the control group (Table 3).

For the control group, there was a positive correlation between TSP activity and the presence of teeth with SUP ($r = 0.7466$; $p = 0.0001$). The test group did not present a correlation for any of the evaluated parameters.

Discussion

The present study evaluated the effect of non-surgical periodontal therapy on clinical parameters and on TSP activity and GCF MPO activity in DM2 patients with inadequate metabolic control compared with systemically healthy individuals.

The DM2 patients presented significantly higher VPI and BOP values at baseline. These results are consistent with those of other studies that found a higher risk to periodontal disease in DM1 and DM2 diabetic subjects compared with non-diabetic individuals

(Bridges et al. 1996, Guven et al. 1996, Aren et al. 2003, Lalla et al. 2007a) and a positive association between poor metabolic control and increased susceptibility to periodontitis (Novaes et al. 1996, Taylor et al. 1998, Jansson et al. 2006, Lim et al. 2007).

In both groups, the conventional periodontal treatment improved most evaluated clinical parameters. These findings corroborate those of previous studies, which used conventional periodontal therapy alone in diabetic individuals and observed improvement only in the clinical parameters, without significant alteration in glycaemic control (Smith et al. 1996, Christgau et al. 1998, Janket et al. 2005). Other studies (Kiran et al. 2005, Faria-Almeida et al. 2006, Navarro-Sanchez et al. 2007) have demonstrated a significant improvement both in clinical parameters and in glycaemic control.

While a considerable improvement in the clinical parameters was evident 3 months after periodontal treatment, a higher percentage of sites with gingival bleeding and BOP was observed in the test group. In addition, there was a significantly lower percentage of reduction for VPI, GBI and BOP in the test group compared with the control group. It is likely that patients' inadequate metabolic control contributed to these results. In addition, higher VPI values

were found in the test group after treatment, in spite of the rigorous periodontal monitoring programme to which the patients were submitted. This finding may be related to the fact that diabetic subjects with inadequate metabolic control may be less compliant with oral self-care (Karjalainen et al. 1994) and maintenance of a good oral health status (Syrjala et al. 1999).

Diabetic individuals present a deficiency in the defence mechanisms against infectious agents (Manouchehr-Pour et al. 1981, Sawant 1993). Studies investigating immunological defence mechanisms have demonstrated an increase in the production of individual cytokines and pro-inflammatory mediators (Salvi et al. 1997, Engebretson et al. 2006, Kumar et al. 2006, Navarro-Sanchez et al. 2007). However, there are few studies addressing the non-immunological defence mechanisms in individuals with inadequate metabolic control of diabetes mellitus.

In the present study, there was no significant difference in TSP activity between the test and control groups at baseline and 3 months post-treatment. These results are consistent with those of Aren et al. (2003), who investigated whether periodontal destruction and alterations in the salivary status were related to the duration of diabetes in children with insulin-dependent DM1 compared with healthy controls. Guven et al. (1996) reported a greater SPO activity in patients with DM1 compared with healthy individuals, which has also been observed by Tenovuo et al. (1986), although without report of metabolic control. Regarding salivary MPO activity, Tenovuo et al. (1986) have not found differences between DM1 and healthy individuals, while Dodds et al. (2000) have reported a greater concentration of SPO and MPO in patients with poorly controlled diabetes mellitus. Nevertheless, there are some methodological differences between the present study and the above-mentioned investigations. We evaluated enzyme activity rather than its concentration, as did some of those authors, and in the present study the diabetic group was formed exclusively by DM2 patients, unlike in some of those studies.

In the present study, TSP activity in the control group was positively correlated to the presence of SUP and might be the cause of the larger number of polymorphonuclear neutrophils (PMN) in saliva via gingival exudation.

GCF MPO activity was significantly lower in the test group compared with the healthy controls. To our knowledge, this is the first study to investigate MPO activity in the GCF and the effect of periodontal treatment on the activity of this enzyme in the gingival fluid of individuals with diabetes mellitus.

Several studies (Cao & Smith 1989, Karhuvaara et al. 1990, Smith et al. 1992; Wolff et al. 1997; Yamalik et al. 2000, Wei et al. 2004, Borges et al. 2007) have demonstrated an increase in MPO levels and/or activity in the GCF of patients with periodontitis. However, these evaluations have been performed in systemically healthy individuals.

The findings of the present study may be supported by the results of previous studies (Sato et al. 1992, 1997, Uchimura et al. 1999), which reported lower MPO activity in the leucocytes of DM2 patients compared with healthy controls. Lalla et al. (2006) also observed a lower concentration of MPO in the serum of individuals with DM1 compared with healthy controls, although not significant, statistically. On the other hand, Wykretowicz et al. (1993) detected low intra-cellular MPO activity in DM2 patients, whereas Lanza (1998) reported that diabetes mellitus is a clinical condition in which secondary MPO deficiency might occur. Regarding metabolic control, Sato et al. (1992) and Kemonia et al. (1985) observed a decrease in MPO activity in the blood of patients with poorly controlled diabetes mellitus, suggesting that the improvement in metabolic control did not alter the enzymatic activity (Kemonia et al. 1985).

PMN plays an important role in the maintenance of periodontal health against bacterial challenge. Nevertheless, the occurrence of dysfunctions in these cells with respect to chemotaxis, adherence and phagocytosis has been observed in diabetic subjects (Manouchehr-Pour et al. 1981, Marhoffer et al. 1992, Sawant 1993, Delamair et al. 1997, Gustke et al. 1998), which may be related to the inadequate metabolic control of diabetes mellitus (Wierusz-Wysocka et al. 1993).

MPO originates from the primary neutrophil granule, and the evaluation of its activity has been used in studies that investigate PMN function (Okouchi et al. 2002, 2004). Therefore, it may be hypothesized that the lower MPO activity observed in the GCF of patients with inadequate metabolic control of diabetes

might be derived from a deficit in the primary defence mechanism of these individuals in response to an aggressive agent.

In both groups, the periodontal treatment significantly reduced TSP activity and GCF MPO activity. These results are consistent with those of a previous study, in which TSP activity increased in the presence of experimental gingivitis in healthy individuals, and decreased after oral hygiene habits restarted (Smith et al. 1984). In addition, the reduction of MPO activity was significantly lower in the test group compared with the control group.

Regarding GCF MPO activity, several studies (Smith et al. 1986, Wolff et al. 1988, Buchmann et al. 2002, Jentsch et al. 2004) have demonstrated that periodontal treatment produces a significant decrease of MPO activity and levels in the GCF of systemically healthy individuals. Lalla et al. (2007b), on the other hand, did not find significant differences in the plasmatic concentration of MPO in diabetic patients submitted to periodontal therapy. However, different from our methodology, those authors evaluated the plasmatic concentration of MPO in patients with DM1 and DM2, with a wide range in metabolic control.

Within the limitations of this study, it may be concluded that TSP activity was similar for DM2 individuals with inadequate metabolic control and for systemically healthy individuals. However, an important finding was the lower GCF MPO activity in the diabetic group. The periodontal treatment was effective in reducing infection, with consequent improvement of most clinical and enzymatic parameters evaluated in both groups. To the best of our knowledge, this is the first study to demonstrate the decrease of TSP activity and GCF MPO activity after periodontal therapy in diabetic individuals, which indicates that, in spite of their inadequate metabolic control, these patients may respond favourably to periodontal treatment.

Although the present study reached significant outcomes, one of its limitations was the small sample size due to the use of restrictive inclusion and exclusion criteria in an attempt to minimize the occurrence of confounding factors. Owing to the lack of studies assessing non-specific defence mechanisms in diabetic patients with inadequate metabolic control, further research should be conducted to evaluate these

parameters in larger samples in order to elucidate the response to aggressive agents in these patients.

Acknowledgements

The authors wish to thank Prof. Dr. Iguatemy Lourenço Brunetti and Prof. Dr. José Silvio Govone.

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Clinical Relevance

Scientific rationale for the study: The increased prevalence and severity of periodontitis in diabetic subjects have been demonstrated. However, few studies have addressed the non-immunological defence mechanisms in individuals with inadequate metabolic control of diabetes. This study

investigated the effect of periodontal therapy on the periodontal status and peroxidase activity in diabetic subjects with inadequate metabolic control compared with systemically healthy individuals.

Principal findings: Periodontal therapy was effective in reducing infection, with improvement of most clinical

and enzymatic parameters evaluated in both groups.

Practical implications: Diabetic patients with periodontitis, even with inadequate metabolic control, might respond favourably to non-surgical periodontal therapy.