J Clin Periodontol 2005; 32: 1226–1235 doi: 10.1111/j.1600-051X.2005.00852.x



Journal of Clinical Periodontology

# The influence of stress and anxiety on the response of non-surgical periodontal treatment

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*Vettore M, Quintanilha RS, Monteiro da Silva AM, Lamarca GA, Leão ATT. The influence of stress and anxiety on the response of non-surgical periodontal treatment. J Clin Periodontol 2005; 32: 1226–1235. doi: 10.1111/j.1600-051X.2005.00852.x.*


***Abstract***

**Aim:** The aim of this study was to evaluate the influence of stress and anxiety on the response to non-surgical periodontal treatment (NPT) in patients with chronic periodontitis.

**Method:** Sixty-six patients (mean age 46.1 ± 8 years) were assigned to three groups: control group, probing pocket depth (PPD) ⩽4 mm, $n = 20$; T1, at least four sites with PPD ⩾4 and ⩽6 mm, $n = 26$; and T2, at least four sites with PPD >6 mm, $n = 20$. Stress, state anxiety (SA) and trait anxiety (TA) and plaque index (PI), gingival index, PPD and clinical attachment level (CAL) were recorded at baseline and 3 months after NPT.

**Results:** TA scores were different among groups at baseline and after NPT. TA was related to periodontitis at baseline and after NPT. PI was associated with the SA at baseline. The reduction of frequency of CAL >6 mm was correlated with TA after adjusting for confounders. Stressed subjects did not show reduction of frequency of PPD >6 mm (T1), CAL 4–6 mm and CAL >6 mm (T2).

**Conclusions:** The data suggest an influence of trait of anxiety and stress on the response to NPT.

Key words: anxiety; chronic periodontits; periodontal treatment; psychosocial factors; stress

Accepted for publication 18 August 2005

Periodontal diseases are characterized by a bacterial challenge that can lead to periodontal attachment loss, bone loss and, ultimately, possible tooth loss. Epidemiological studies related to periodontal diseases search for both determinants of its occurrence and progression, as well as for preventive strategies and effective treatments. Several observational studies were conducted, and recognized risk factors are well known for periodontal tissues breakdown (Grossi et al. 1994, Page & Beck 1997).

Specific subgingival microorganisms clustered in microbial complexes are considered determinant agents for periodontal disease onset and progression, as well as for refractory/recurrent periodontitis (Offenbacher 1996, Colombo et al. 1998, Socransky et al. 1998). In addition, behavioural risk factors (e.g. tobacco use, oral cleanliness) and systemic disorders (e.g. uncontrolled diabetes mellitus patients) play an important role in the pathogenesis of periodontitis (Shlossman et al. 1990, Barbour et al. 1997).

The most common form of periodontal therapy in subjects with chronic periodontitis includes instruction for dental biofilm self-control, scaling and root planing. Periodontal healing based on surrogate endpoints such as probing pocket depth (PPD) reduction, probing clinical attachment level (CAL) maintenance and suppression of gingival inflammation is usually obtained when non-surgical periodontal treatment (NPT) is conducted (Badersten et al. 1981, 1984, Nordland et al. 1987). Similarly, the effects of NPT on major disease endpoints such as tooth loss, edentulism or quality of life confirm its benefits (Hujoel et al. 2000). Recognized risk factors for periodontal disease onset and progression have also been associated with poor response to conventional periodontal treatment (Hempton & Leone 1997).

Recently, researchers have tested the hypothesis that psychosocial factors can contribute to periodontitis. Several psychological disorders have been shown to be associated with chronic and aggressive periodontitis, as well as with progression of periodontal disease (Monteiro da Silva et al. 1996, Genco et al. 1999, Vettore et al. 2003).

Current research has focused on optimal ways of delivering non-surgical periodontal therapy but relatively little is known about patient factors that affect the variability in outcome of this impor-

tant treatment modality (Kinane 2005, Koshy et al. 2005, Wennstrom et al. 2005). To date, few studies reported the influence of psychosocial factors on periodontal healing after periodontal treatment (Axtelius et al. 1998, Wimmer et al. 2004). Patients responding less well to periodontal treatment had more psychosocial strain and a more passive-dependent personality (Axtelius et al. 1998). Inadequate coping behaviours including passive coping strategies were more pronounced in cases of poor response to NPT (Wimmer et al. 2004).

In a previous study, patients with high levels of trait anxiety (TA) had a higher frequency of moderate CAL (4–6 mm) and moderate PPD (4–6 mm) (Vettore et al. 2003). The aim of the present study was to assess the possible influence of stress and anxiety on the response to NPT in patients with different levels of chronic periodontitis.

## Material and Methods

In this case–control, longitudinal, double-blind study, stress and anxiety in patients with different levels of chronic periodontitis before and after NPT were assessed. After approval of the Federal University of Rio de Janeiro (UFRJ) Ethics Committee, individuals attending the Clinical Dentistry Department of the Dental School of the UFRJ were invited to take part in this study. The inclusion criteria consisted of participants being over 35 years of age and presenting at least 50% of dental surfaces with plaque index (PI) $\geqslant 2$ (Silness & Löe 1964). The exclusion criteria included patients presenting acute necrotizing ulcerative gingivitis, acute necrotizing ulcerative periodontitis, systemic conditions associated with periodontal disease, pregnancy and those who received periodontal therapy during the last 6 months. In addition, patients taking drugs that could affect the progression or treatment of periodontal diseases were excluded.

A pilot study including 15 patients with at least four sites with PPD $>4.0$ mm was conducted to calibrate the examiners, and to test the understanding and the layout of the stress and anxiety questionnaires.

In the main study, patients considered suitable were assigned to one of the following three groups in accordance with their PPD levels: the control group (C) had less than four sites with PPD $\leqslant 4.0$ mm, Test group 1 (T1) had at least four sites with PPD $\geqslant 4.0$ and $\leqslant 6.0$ mm and Test group 2 (T2) had at least four sites with PPD $>6$ mm.

After signing an informed consent consisting of the study aims, procedure and the voluntary character of their participation, medical history and socioeconomic data were collected. Socioeconomic data included the following: age, gender, employment status, marital status, schooling, familial income, smoking history, number of cigarettes smoked per day and alcoholic drink consumption. Subsequently, three self-reported questionnaires were used to evaluate stress and anxiety. All patients were examined for periodontal clinical parameters by two previously calibrated examiners. Those presenting periodontal disease were submitted to NPT. Periodontal clinical measurements and stress and anxiety assessment were also registered 3 months after NPT for all subjects.

### Psychosocial measurements

Psychological measures to assess stress and anxiety included three psychometric instruments. The Stress Symptoms Inventory (SSI) (Lipp & Guevara 1994) aims to detect whether a patient presents a clinical stress syndrome. Patients were asked to indicate whether a number of physical and psychological stress symptoms had occurred recently (involving the last day, week and month). The SSI is based on Selye's concepts of the General Adaptation Syndrome (Selye 1963). There are 53 items divided into three sections. Each of them corresponds to one of Selye's stress stages: alarm reaction, stage of resistance and stage of exhaustion. If somebody is clinically stressed, it is also possible to identify the stress phase.

Anxiety was assessed with the Spielberger State-Trait Anxiety Inventory (STAI) adapted to the Brazilian population by Biaggio et al. (1977). This inventory consists of two self-report scales. Each of them has 20 items followed by a four-point scale. These self-report scales measure two different dimensions of anxiety: state anxiety (SA) and TA. The TA scale requires that subjects describe the way they generally feel. The SA scale asks respondents to indicate how they feel at a specific moment in time. The range of possible score varies from a minimum score of 20 to a maximum score of 80 on both scales.

### Clinical measurements

After all questionnaires were completed, patients underwent a clinical examination. Two calibrated examiners assessed PI, Gingival Index (GI) (Löe 1967), PPD and CAL. These were recorded at six sites per tooth (mesiobuccal, buccal, distobuccal, distolingual, lingual and mesiolingual) at all teeth excluding third molars. PPD and CAL measures were taken as the average of two previous measures. Such measurements were recorded to the nearest millimetre using a North Carolina periodontal probe (Hu-Friedy, Chicago, IL, USA).

### Periodontal therapy

Periodontal therapy consisted initially of instruction in self-care of plaque control for all participants. Patients with PPD less than 4.0 mm were submitted to supragingival scaling, coronal polishing and topical fluoride gel when necessary. Patients with periodontal disease also received scaling and root planing in all sites with PPD $>3$ mm. Scaling and root planing were conducted using Gracey curettes no 1–2, 7–8, 11–12, 13–14 (Hu-Friedy). The removal of retentive factors for dental plaque accumulation including overhanging restorations and carious cavities was performed according to need. NPT was completed in at most four appointments of 1 h sessions. Local and systemic antibiotics were not administered to any patient. In addition, surgical procedures for periodontal disease treatment were not performed.

Periodontal clinical examination and treatment procedures were performed by two clinicians, in such a way that one of them treated half of the patients who had been previously examined by the other one and vice versa. The clinicians were blinded to patients' stress and anxiety status during the whole period of the study. The scaling and root planing procedures were appraised by a third periodontist in each session.

After the completion of the NPT, all patients had supportive monthly periodontal therapy, which consisted of oral hygiene instructions, coronal polishing and supragingival scaling when necessary.

### Follow-up assessment

Three months after the last session of periodontal therapy, all subjects were

invited for re-assessment on stress, anxiety and periodontal clinical parameters. A similar protocol was used to obtain measures at baseline and at the 3-month follow-up appointment, given that socioeconomic data were not collected at follow-up assessment. The periodontal examinations at baseline and 3 months after NPT were performed by the same examiner. Those patients who had taken antibiotics or reported systemic conditions that could affect periodontal disease during the course of the study were excluded.

#### Statistical methods

Data were analysed using SPSS 10.0 (Statistical Package for Social Sciences for Windows, SPSS Inc., Chicago, IL, USA). The significance level established for all analysis was 5% ($p \leqslant 0.05$).

Socioeconomic data were computed for each participant from data provided in the baseline questionnaire. The three groups were compared with respect to age by a Kruskal–Wallis test and for the remaining socioeconomic data, $\chi^2$ tests were performed.

Clinical parameters were registered and averaged for each patient in the three groups. Differences among clinical parameters were examined in sites subset according to PPD and CAL categories of $<4$ mm (shallow), 4–6 mm (moderate) and $>6$ mm (deep). Significance of differences among the three groups before and after periodontal therapy was verified by Kruskal–Wallis tests. Comparisons within each group between baseline and after periodontal therapy were made using the Wilcoxon signed-rank test.

The significance of differences in the frequency of participants with clinical stress among the three groups was examined using the $\chi^2$ test. Kruskal–Wallis tests were used to compare the three groups in terms of SA and TA. The stress and anxiety comparisons among groups were conducted at baseline and 3 months after NPT. Possible associations between psychosocial factors and periodontal parameters were examined by non-parametric Spearman's linear correlation coefficients at baseline and 3 months after NPT. Internal consistencies for the SSI and for the two scales of the STAI were evaluated by the Cronbach's $\alpha$ coefficient at baseline and 3 months after NPT.

The influence of psychosocial factors on periodontal status was assessed using two strategies. The frequency of PPD and CAL categories of $<4$ mm (shallow), 4–6 mm (moderate) and $>6$ mm (deep) were compared between baseline and after NPT for stressed and non-stressed patients in each group by the Wilcoxon signed-rank test.

The differences of PPD and CAL frequency $\geqslant 4$, 4–6, $>6$ mm between initial and 3 months after NPT were computed to assess the reduction of periodontal disease clinical parameters. Univariate analysis of covariance was carried out on the reduction of PPD and CAL frequencies (dependent variables) with all psychosocial measures (independent variables) adjusting for dental plaque and number of cigarettes (covariates).

## Results

A clinical calibration for periodontal parameters was performed in the pilot study for the two examiners involved Kappa measure and Intra-class Correlation Coefficient of agreement findings for CAL and PPD intra- and inter-examiner were over 0.71. The results have been described previously (Vettore et al. 2003).

Of the 85 originally selected participants, a total of 66 were considered for the final analysis. Sixteen patients were excluded, seven used antibiotics during the course of the study and 12 failed to return for the follow-up appointment.

#### Socioeconomic data

The demographic and socioeconomic characteristics of all subjects are summarized in Table 1. There were no significantly statistical differences among three groups. Statistical analysis comparing marital status, number of cigarettes/day between smokers and alcoholic drink consumption among groups could not be performed because of the small number of subjects in some cells.

Although not significant, a smaller percentage of smokers were found in the control group. In addition, the number of cigarettes smoked/day was greater in patients with periodontitis.

#### Clinical parameters

The clinical parameters data at baseline and 3 months after NPT for the three

*Table 1.* Demographic and socioeconomic characteristics of participants in the three groups

| Demographic and socioeconomic characteristics of subjects | Control ($N = 20$) | Test group 1 ($N = 26$) | Test group 2 ($N = 20$) | $p$ |
|---|---|---|---|---|
| Age (mean ± SD)* | 45.9 ± 8.3 | 46.4 ± 8.9 | 46.1 ± 7.8 | 0.96 |
| Gender (%)† | | | | 0.14 |
| Male | 45 | 73.1 | 55 | |
| Female | 55 | 26.9 | 45 | |
| Employment status (%)‡ | | | | – |
| Unemployed | 5 | 15 | 15 | |
| Employed | 85 | 73 | 75 | |
| Retired | 10 | 12 | 10 | |
| Marital status (%)† | | | | 0.36 |
| Unmarried/divorced/widowed | 75 | 57.7 | 55 | |
| Married/mate | 25 | 42.3 | 45 | |
| Schooling (%)† | | | | 0.93 |
| ⩽8 years | 55 | 50 | 50 | |
| >8 years | 45 | 50 | 50 | |
| Household income in minimum salaries (%)† | | | | 0.93 |
| <3 | 25 | 30.7 | 20 | |
| 3–6 | 35 | 30.8 | 35 | |
| >6 | 40 | 38.5 | 45 | |
| Smokers (%)† | 30 | 42 | 55 | 0.28 |
| No of cigarettes/day between smokers (%)‡ | | | | – |
| 1–10 | 83 | 36 | 55 | |
| 11–20 | 17 | 55 | 27 | |
| >20 | 0 | 9 | 18 | |
| Alcoholic drink consumption (%)‡ | | | | – |
| Less than two glasses per week | 80 | 70 | 76.9 | |
| One glass almost every day | 15 | 25 | 19.2 | |
| Two or more glasses per day | 5 | 5 | 3.8 | |

*Kruskal–Wallis test.
†$\chi^2$ test.
‡Statistical test could not be performed.

groups are presented in Tables 2–4. All periodontal measures were significantly different among the three groups at both baseline and 3 months after NPT analysis, with the exception of PI. The analysis of CAL and PPD periodontal sites categories of $<4$, 4–6 and $>6$ mm showed a gradual increase of deeper PPD and CAL frequencies, when the three groups were compared. Frequencies of deeper PPD and CAL were higher in groups with more periodontal disease. Bleeding on probing (BOP) was also related to poorer periodontal condition ($p<0.01$).

A significant reduction in the frequencies of moderate (4–6 mm) and deep ($>6$ mm) PPD and CAL categories was observed 3 months after NPT for group T2 ($p<0.01$). Similarly, moderate (4–6 mm) and deep ($>6$ mm) PPD categories had reduced 3 months after NPT for group T1. The percentage of sites with visible dental plaque and BOP dropped significantly in all groups.

### *Psychosocial variables findings*

The SSI and STAI understanding and layout were tested and adjusted when necessary in a pilot study (Vettore et al. 2003).

### *Internal consistency of the SSI and STAI*

The number of psychosocial items in the subscales that comprise the SSI, State-Anxiety Inventory (SAI) and Trait-Anxiety Inventory (TAI) were 53, 20 and 20, respectively. Results for the internal consistency of the items within each scale were computed at baseline and 3 months after NPT. Baseline Cronbach's coefficients for SSI, SAI and TAI were 0.77, 0.86 and 0.76, respectively. Three months after NPT, the coefficients were 0.85, 0.87 and 0.84, respectively.

### *Analysis among groups of participants with different levels of periodontal disease and psychosocial factors*

Figure 1 shows the prevalence of subjects with clinical stress at baseline and 3 months after NPT for the three groups. Group T1 showed the highest frequency of patients with clinical stress at both assessment times. The frequency of stressed patients 3 months after NPT increased in control and T1 groups from 19.1–29.4% to 47.0–58.9%, respectively. Among stressed patients, participants in the alarm stage at baseline were detected in the control group (2.4%) and 3 months after NPT in group T1 (5.9%). No participant was in the stage of exhaustion. No significant differences for clinical stress among groups

*Table 2.* Mean percentages (± SD) of clinical attachment level in the three groups at baseline and after therapy and according to the stress status

| Clinical attachment level | Control group | | Test group 1 | | Test group 2 | | $p^{**}$ |
|---|---|---|---|---|---|---|---|
| | baseline† | after NPT | baseline† | after NPT | baseline† | after NPT | |
| $<4$ mm | | | | | | | |
| All subjects | 90.6 ± 9.6 | 90.0 ± 12.5 | 69.0 ± 16.6 | 75.0 ± 16.7 | 31.9 ± 20.2 | 46.5 ± 25.0 | $<0.01$ |
| | | | * | | * | | |
| Non-stressed | 88.7 ± 11.2 | 89.4 ± 14.2 | 73.9 ± 17.4 | 79.9 ± 11.2 | 32.3 ± 20.8 | 48.1 ± 26.9 | $<0.01$ |
| | | | | | * | | |
| Stressed | 95.2 ± 3.5 | 92.1 ± 8.4 | 63.5 ± 14.4 | 69.9 ± 20.8 | 30.9 ± 20.7 | 42.4 ± 21.5 | $<0.01$ |
| | | | | | * | | |
| 4–6 mm | | | | | | | |
| All subjects | 9.0 ± 0.9 | 9.0 ± 10.8 | 27.4 ± 15.2 | 22.0 ± 14.2 | 47.3 ± 15.2 | 39.0 ± 19.0 | $<0.01$ |
| | | | | | * | | |
| Non-stressed | 10.8 ± 10.2 | 9.7 ± 11.9 | 23.2 ± 17.7 | 18.4 ± 10.7 | 45.6 ± 16.7 | 35.7 ± 18.6 | $<0.01$ |
| | | | | | * | | |
| Stressed | 4.5 ± 3.0 | 7.7 ± 8.4 | 32.3 ± 12.2 | 26.2 ± 17.0 | 52.5 ± 19.6 | 49.2 ± 19.1 | $<0.01$ |
| $>6$ mm | | | | | | | |
| All subjects | 0.5 ± 1.0 | 0.7 ± 2.0 | 3.5 ± 5.2 | 2.7 ± 4.5 | 20.7 ± 15.4 | 14.2 ± 16.7 | $<0.01$ |
| | | | | | * | | |
| Non-stressed | 0.5 ± 1.2 | 1.0 ± 2.5 | 2.9 ± 3.5 | 1.7 ± 2.4 | 22.1 ± 17.5 | 16.2 ± 18.8 | $<0.01$ |
| | | | | | * | | |
| Stressed | 0.3 ± 0.7 | 0.2 ± 0.4 | 4.2 ± 6.8 | 3.9 ± 6.1 | 16.6 ± 5.9 | 8.4 ± 6.6 | $<0.01$ |

Control group, all subjects, $N=20$; non-stressed, $N=14$; stressed, $N=6$.
Test group 1: all subjects, $N=26$; non-stressed, $N=14$; stressed, $N=12$.
Test group 2: all subjects, $N=20$; non-stressed, $N=15$; stressed, $N=5$.
**$p$ refers to the Kruskal–Wallis test for comparison among groups at baseline and after non-surgical therapy.
*$p<0.01$ refers to Wilcoxon's test for comparison within groups at baseline and after non-surgical therapy.
†There were no significant differences between stressed and non-stressed patients on the baseline data for all periodontal clinical parameters.

*Table 3.* Mean percentages (± SD) of periodontal pocket depth in the three groups at baseline and after therapy and according to the stress status

| Periodontal pocket depth | Control group | | Test group 1 | | Test group 2 | | $p^{**}$ |
|---|---|---|---|---|---|---|---|
| | baseline[†] | after NPT | baseline[†] | after NPT | baseline[†] | after NPT | |
| < 4 mm | | | | | | | |
| All subjects | 97.0 ± 5.2 | 96.6 ± 6.7 | 77.7 ± 12.7 | 87.0 ± 13.7 * | 43.7 ± 20.2 | 65.2 ± 19.0 * | < 0.01 |
| Non-stressed | 96.5 ± 6.2 | 97.4 ± 6.2 | 81.6 ± 11.9 | 89.8 ± 10.3 * | 44.2 ± 20.3 | 66.1 ± 19.8 * | < 0.01 |
| Stressed | 98.1 ± 1.9 | 94.9 ± 8.1 | 73.3 ± 12.6 | 83.7 ± 16.9 * | 42.5 ± 22.4 | 62.7 ± 17.6 * | < 0.01 |
| 4–6 mm | | | | | | | |
| All subjects | 3.0 ± 6.5 | 3.2 ± 6.7 | 20.6 ± 11.7 | 12.4 ± 12.8 * | 42.0 ± 17.6 | 27.6 ± 13.2 * | < 0.01 |
| Non-stressed | 3.5 ± 6.2 | 2.6 ± 6.3 | 17.3 ± 11.6 | 9.8 ± 9.9 * | 40.4 ± 16.3 | 25.6 ± 12.4 * | < 0.01 |
| Stressed | 1.9 ± 1.9 | 4.8 ± 8.3 | 24.6 ± 11.2 | 15.4 ± 15.6 * | 46.2 ± 22.8 | 33.8 ± 15.1 | < 0.01 |
| > 6 mm | | | | | | | |
| All subjects | 0.0 ± 0.0 | 0.1 ± 0.3 | 1.6 ± 2.7 | 0.6 ± 1.3 * | 14.4 ± 10.2 | 7.0 ± 9.7 * | < 0.01 |
| Non-stressed | 0.0 ± 0.0 | 0.1 ± 0.2 | 1.1 ± 1.9 | 0.4 ± 1.2 * | 15.5 ± 11.6 | 8.3 ± 10.9 * | < 0.01 |
| Stressed | 0.0 ± 0.0 | 0.3 ± 0.4 | 2.1 ± 3.4 | 0.9 ± 1.6 | 11.2 ± 3.4 | 3.5 ± 4.1 * | < 0.01 |

Control group, all subjects, $N = 20$; non-stressed, $N = 14$; stressed, $N = 6$.
Test group 1: all subjects, $N = 26$; non-stressed, $N = 14$; stressed, $N = 12$.
Test group 2: all subjects, $N = 20$; non-stressed, $N = 15$; stressed, $N = 5$.
*$p < 0.01$ refers to Wilcoxon's test for comparison within groups at baseline and after non-surgical therapy.
NS, not significant at the level of $p = 0.05$.
[†]There were no significant differences between stressed and non-stressed patients on the baseline data for all periodontal clinical parameters.
**$p$ refers to the Kruskal–Wallis test for comparison among groups at baseline and after non-surgical therapy.

were found at baseline and 3 months after NPT.

The average mean scores for TA among the three groups were statistically different at baseline and 3 months after NPT ($p < 0.05$). The mean baseline scores for TA for the three groups, control, T1 and T2, were 38.4, 40.5 and 45.3, respectively. After NPT, the mean scores were 38.3, 41.7 and 47.2. No significant differences among the three groups were found for mean scores of SA before and after NPT.

### *Correlation analysis between psychosocial factors and periodontal clinical measures*

Table 5 presents the non-parametric Spearman linear correlations between TA and periodontal clinical measures. Since TA seems stable over a short period of time, only baseline scores for TA were analysed. Significant associations were observed between PPD and CAL frequencies $\geqslant 4.0$ and 4–6 mm and high scores of TA in baseline and 3 months after NPT ($p < 0.05$). Frequencies of deeper PPD at baseline and frequencies of deeper CAL 3 months after NPT were also found to be significantly associated with high scores of anxiety trait ($p < 0.05$). High scores of SA at baseline were significantly associated with visible dental plaque ($p < 0.05$).

#### Periodontal clinical parameters changed from baseline to 3 months after NPT in stressed and non-stressed subjects

The comparisons of periodontal parameters between baseline and 3 months after NPT of patients with stress in both assessment times ($N = 8$), and without diagnoses of clinical stress ($N = 29$) are shown in Tables 2–4. Baseline PPD and CAL frequencies $< 4.0$ and $\geqslant 4.0$ mm were similar between participants without stress and those with stress, as well as visible dental plaque and BOP. Decreases in PPD and CAL frequencies $\geqslant 4.0$ mm were significant in both groups of participants: those with or without stress ($p < 0.05$). Significant reductions of visible dental plaque and BOP were also observed in both groups.

### *Univariate analysis of covariance between reduction of PPD and CAL frequencies, socioeconomic data and psychosocial factors*

Univariate analysis of covariance was performed on the reduction of PPD and CAL frequencies $> 4$, 4–6 and $> 6$ mm

*Table 4.* Mean percentages ( ± SD) of plaque index and gingival index in the three groups at baseline and after therapy and according to the stress status

| Clinical Parameters | Control group | | Test group 1 | | Test group 2 | | $p^{**}$ |
|---|---|---|---|---|---|---|---|
| | baseline | after NPT | baseline | after NPT | baseline | after NPT | |
| PI ⩾ 2 | | | | | | | |
| All subjects | 56.5 ± 19.4 | 11.4 ± 16.5 * | 51.0 ± 21.0 | 20.3 ± 20.9 * | 59.3 ± 17.1 | 20.0 ± 20.3 * | NS |
| Non-stressed | 58.3 ± 19.8 | 12.2 ± 19.2 * | 47.5 ± 22.1 | 21.0 ± 22.2 * | 59.8 ± 17.4 | 15.9 ± 16.1 * | <0.01 |
| Stressed | 52.7 ± 19.7 | 9.8 ± 9.1 * | 55.5 ± 19.2 | 19.6 ± 20.3 * | 58.1 ± 18.3 | 32.4 ± 28.5 * | <0.01 |
| Gingival index BOP | | | | | | | |
| All subjects | 6.4 ± 22.6 | 11.0 ± 20.0 * | 39.3 ± 29.0 | 23.0 ± 27.0 * | 67.5 ± 23.5 | 43.3 ± 24.4 * | <0.01 |
| Non-stressed | 15.3 ± 24.1 | 9.5 ± 19.3 * | 29.0 ± 22.2 | 14.0 ± 15.3 * | 67.1 ± 21.5 | 41.6 ± 23.0 * | <0.01 |
| Stressed | 19.2 ± 20.7 | 14.8 ± 23.5 | 51.4 ± 32.3 | 34.1 ± 34.1 * | 69.9 ± 31.8 | 48.7 ± 32.4 * | <0.01 |

Control group, all subjects, $N = 20$, non-stressed, $N = 14$; stressed, $N = 6$.
Test group 1: all subjects, $N = 26$, non-stressed, $N = 14$; stressed, $N = 12$.
Test group 2: all subjects, $N = 20$, non-stressed, $N = 15$; stressed, $N = 5$.
$^{**}p$ refers to the Kruskal–Wallis test for comparison among groups at baseline and after non-surgical therapy.
$^{*}p < 0.01$ refers to Wilcoxon's test for comparison within groups at baseline and after non-surgical therapy.
NS, not significant at the level of $p = 0.05$.
PI, plaque index; BOP, bleeding on probing.

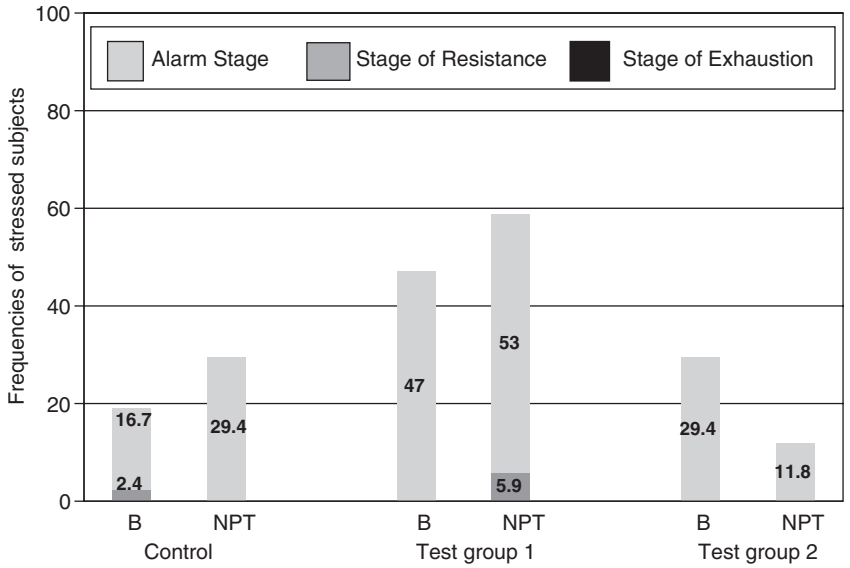


*Fig. 1.* Frequency subjects with stress in the groups at baseline (B) and 3 months after non-surgical therapy (NPT). Control group, less than four sites with probing pocket depth (PPD) ⩽4 mm; Group 1, at least four sites with PPD ⩾4 and ⩽6 mm; Group 2, at least four sites with PPD ⩾6 mm. There were no significantly statistical differences among the three groups. (Baseline: $p = 0.18$; 3 months after NPT: $p = 0.69$; $\chi^2$ test.)

with all psychosocial measures (Table 6). Scores of TA were statistically associated with the reduction of deeper CAL frequencies (>6 mm) ($p = 0.011$), reduction of deeper PPD frequencies (>6 mm) ($p = 0.030$) and reduction of PPD frequencies >4.0 mm ($p = 0.026$). The association between reduction of deeper CAL frequencies (>6 mm) and scores of TA remained statistically significant after adjusting for dental plaque and number of cigarettes ($p = 0.011$).

## Discussion

The findings in the present study demonstrated an influence of stress and TA on periodontal healing after non-surgical periodontal therapy. Significantly higher TA scores were observed for subjects with chronic periodontitis at baseline and 3 months after NPT. These results support the hypothesis that psychosocial factors can contribute to periodontal disease aetiology and also affect periodontal status after periodontal treatment.

The search for behavioural and psychosocial risk indicators for periodontal disease remains an important field in periodontology. Early evidence in this was obtained in studies that showed that acute necrotizing ulcerative gingivitis incidence was strongly predicted by endocrine imbalances caused by stress

*Table 5.* Correlation matrix (Spearman coefficient) between the total score of trait anxiety and periodontal clinical parameters at baseline and 3 months after non-surgical periodontal therapy (NPT)

| Clinical parameters | Trait anxiety | |
|---|---|---|
| | baseline | 3 months after NPT |
| Periodontal pocket depth (%) | | |
| ⩾4 mm | 0.305 ($p = 0.013$)* | 0.275 ($p = 0.026$)* |
| 4–6 mm | 0.306 ($p = 0.012$)* | 0.276 ($p = 0.025$)* |
| >6 mm | 0.300 ($p = 0.015$)* | 0.239 ($p = 0.053$) |
| Clinical attachment loss (%) | | |
| ⩾4 mm | 0.304 ($p = 0.013$)* | 0.288 ($p = 0.019$)* |
| 4–6 mm | 0.289 ($p = 0.018$)* | 0.294 ($p = 0.017$)* |
| >6 mm | 0.224 ($p = 0.070$) | 0.269 ($p = 0.029$)* |

*$p < 0.05$.

*Table 6.* Univariate analysis of variance on the reduction of clinical attachment level frequencies >6 mm

| Source | Mean of squares | *df* | *F* | *p*-value |
|---|---|---|---|---|
| Anxiety | 154.534 | 1 | 7.664 | 0.011 |
| Dental plaque | 28.096 | 1 | 1.393 | 0.249 |
| Number of cigarettes | 0.341 | 1 | 0.017 | 0.898 |
| Error | 483.902 | 24 | | |

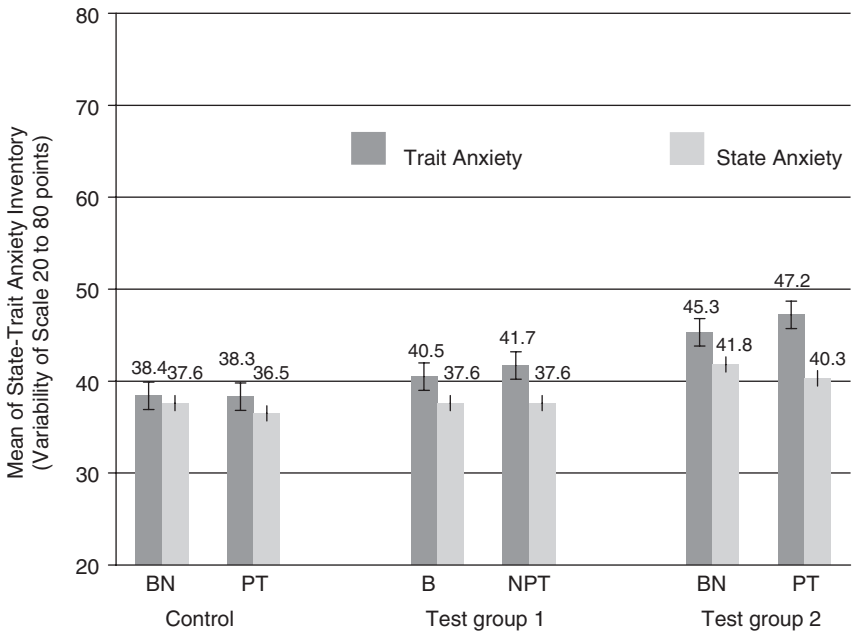


*Fig. 2.* Mean scores of State-Trait Anxiety Inventory at baseline (B) and 3 months after non-surgical therapy (NPT). Control group, less than four sites with probing pocket depth (PPD) ⩽4 mm; Group 1, at least four sites with PPD ⩾4 and ⩽6 mm; Group 2, at least four sites with PPD ⩾6 mm. There were significantly statistical difference among groups for trait-anxiety. (Trait-Anxiety: B: $p = 0.02$; NPT: $p = 0.05$; State-Anxiety: B: $p = 0.23$; NPT: $p = 0.53$; Kruskal–Wallis.)

situations (Shannon et al. 1969, Maupin & Bell 1975, Cogen et al. 1983, Cohen-Cole et al, 1983, Stevens et al. 1984). In the last two decades, several investigations have been carried out on associations between psychosocial factors and periodontal illness (Marcenes & Sheiham 1992, Monteiro da Silva et al. 1996, Moss et al. 1996, Croucher et al. 1997, Genco et al. 1999, Vettore et al. 2003, Solis et al. 2004). The methodological approaches used in those studies are quite different, which may be responsible for the conflicting results between some studies. Such differences involve the type of psychosocial variable analysed, the questionnaire used for its assessment, the type of periodontal disease investigated, the parameters used for periodontal status evaluation and adequate control for potential confounders.

The scientific evidence of the relationship between anxiety and periodontal disease has been demonstrated in a previous paper when the frequency of moderate CAL and moderate PPD were found to be significantly associated with higher trait-anxiety scores after adjusting for socioeconomic data and cigarette consumption (Vettore et al. 2003). In the present investigation, there was a significant difference of TA scores among groups with different levels of chronic periodontitis. There is a dose–response effect between the averaged mean TA scores and chronic periodontitis (Fig. 2).

Other studies did not find differences for anxiety when groups with different levels of periodontal disease were compared (Monteiro da Silva et al. 1996, Moss et al. 1996, Genco et al. 1999, Solis et al. 2004). Possible explanations for the different findings include methodological issues. The psychometric instrument used to assess anxiety in Genco and co-worker's study was the Hopkins-Symptom Checklist 90-revise (SCL-90-R), which involves nine symptom dimensions of psychological and somatic symptom patterns (Genco et al. 1999). Of the 53 items in this scale, only six are used to evaluate the anxiety. In common with Genco and co-workers' study, Moss and colleagues used a similar questionnaire to assess anxiety. They used an abbreviated version of the SCL-90-R that provides an assessment of psychological symptoms in nine areas including anxiety (Moss et al. 1996).

However, despite using the same psychometric instrument used in the present investigation to assess TA, early studies did not detect differences of anxiety mean scores between groups with and without periodontal disease (Monteiro da Silva et al. 1996, Solis et al. 2004). In an overall overview of the levels of TA in such studies, the anxiety scores in all studies were similar and cannot be responsible for the differences in the findings between the present study and others.

Nonetheless, the clinical criteria of diagnostic used to periodontal disease were quite different. In contrast with the present study, chronic periodontitis was considered when subjects were over 35 years of age and presented horizontal bone loss (Monteiro da Silva et al. 1996). Conventional periodontal clinical parameters to assess periodontal disease including PPD, clinical attachment loss and BOP were not used for periodontitis diagnostic. In another study, the cases were patients with ''established periodontitis'', according to Machtei et al. (1992) (Solis et al. 2004). The severity and distribution of periodontal disease in patients at Solis and co-worker's study might have been different from the present investigation. The non-differential misclassification bias on periodontal disease assessment might have occurred in these studies, and could also have affected the association between anxiety and periodontal disease.

The frequency of subjects with clinical stress was similar among groups at baseline and 3 months after non-surgical periodontal therapy. This finding is in accordance with previous studies on the association between stress and chronic periodontitis (Monteiro da Silva et al. 1996, Moss et al. 1996). Conversely, in other investigations groups that had higher scores for stress showed more severe periodontal disease (Marcenes & Sheiham 1992, Croucher et al. 1997, Genco et al. 1999, Hugoson et al. 2002, Wimmer et al. 2002).

The differences in the above-mentioned results may be explained by the stress model used in the studies. Stress is a complex process by which an organism responds to certain environmental or psychological events, called stressors, that pose a challenge or danger to the organism (Gatchel et al. 1989). The SSI (Lipp & Guevara 1994) used in the present study is based on the General Adaptation Syndrome proposed by Selye (1956). General Adaptation Syndrome consists of three stages of response: alarm reaction, stage of resistance and exhaustion. The alarm stage starts when the organism becomes aware of a stressor or the presence of a noxious stimulus. This reaction is considered universal for all human beings. The stage of resistance and exhaustion involves coping mechanisms and adaptive reserves' consumption, and personality and environmental variables can affect the stress process (Bartlett 1998).

Environmental variables related to stress include work-related mental demand, marital quality and socioeconomic status, which in turn were associated with periodontal status (Marcenes & Sheiham 1992). Marital status (widow/widower) and external locus of control significantly increased the risk of severe periodontal disease (Hugoson et al. 2001).

Current theories on the evolution of psychological mechanisms as a part of the stress process focus on appraisal and contextual cues (Lazarus & Folkman 1984). The elicitation of stress responses vary; while some divergent responses are based on biological predispositions, many others are explained in terms of differences between stressors, contexts in which they occur and perceived abilities to cope with them. Styles of coping with stress as well as psychological supports and assets will affect the stressor responses and the ultimate consequences of exposure to them (Kanner et al. 1981).

In this context, Genco et al. (1999) detected a greater risk of severe attachment loss in subjects with high emotion-focused coping and more financial strain. In addition, inadequate stress behaviour strategies (defensive coping) were strongly associated with the risk for severe periodontal disease (Wimmer et al. 2002).

The efficacy of the periodontal therapy observed in the present study is in agreement with data observed in longitudinal studies (Badersten et al. 1981, 1984, Nordland et al. 1987, Claffey et al. 1988). Improvements in periodontal clinical parameters after scaling and root planing followed by instruction in oral hygiene included reduction of the percentage of sites with deep and moderate PPD and CAL. Large amounts of dental plaque comprised the inclusion criterion for participants, and it was the only clinical parameter similar for the three groups at baseline. As dental plaque accumulation was high in all patients, it was possible to establish reliable associations between dental biofilm and psychosocial factors. Plaque levels were associated with anxiety status before periodontal treatment. This suggests that those who were more anxious may have not cleaned teeth so well. This can be associated with health self-care and internal locus of control (Monteiro da Silva et al. 1995). The decrease in plaque levels 3 months after NPT was significant for all groups. The analysis of the influence of stress and anxiety on periodontal healing was performed by controlling this confounding variable. In addition, The General Linear Model test was conducted to control for other possible confounding variables, such as number of cigarettes.

Few studies tried to demonstrate a possible influence of psychosocial factors on periodontal status after anti-infectious periodontal therapy (Axtelius et al. 1998, Wimmer et al. 2005). Passive coping strategies were more pronounced in cases of poor response to a non-surgical periodontal treatment. In addition, active coping strategies were associated with a favourable course of periodontal treatment (Wimmer et al. 2005). Another investigation compared psychological variables between patients responding well to periodontal treatment against those classified as responding less well to periodontal treatment. On one hand, the group responding well to periodontal treatment displayed a more resilient personality and possibly a less stressful psychosocial situation in the past. On the other, the group with patients responding less well to periodontal treatment had more psychological strains and a more passive-dependent personality (Axtelius et al. 1998).

The present study supports the hypothesis that psychosocial factors must be considered an important variable in the response to periodontal therapy. The reduction of severe CAL frequencies was significantly predicted by TA scores. This is the first study that shows the link between anxiety and periodontal healing. Socioeconomic characteristics were not significantly different among the three groups, although a tendency on the variability was observed. The three groups' sample size may have influenced these results. More than half of the subjects involved were employed and married and reported low alcohol consumption. The main variables were statistically controlled through general linear model during the analysis of the relationship of PPD and CAL measurement reduction after NPT and TA scores. The present study showed significantly positive correlations between TA and frequencies of clinical parameters for periodontal disease. Moreover, TA remained associated with periodontal healing after controlling for confounders. The periodontal healing was associated with the only psychosocial factor that represents a characteristic of personality and, therefore, has more temporal stability.

Stress did influence the periodontal healing after non-surgical periodontal

treatment. The findings in the present investigation are in accordance with previous studies, despite the lack of standardization in the model to assess stress among studies. To analyse the influence of stress on the response of non-surgical periodontal therapy, subjects who completed the periodontal treatment were grouped into ''non-stressed patients'' and ''stressed patients'' groups. The reduction of PPD and CAL frequencies 4–6 and $>6$ mm was not observed in stressed subjects in different groups with periodontal disease. On the other hand, non-stressed patient groups showed significant decreases in all PPD and CAL frequencies 4–6 and $>6$ mm. This lack of reduction in clinical parameters observed in the stressed group may either be owing to an influence of stress in periodontal healing or else the sample size of this group.

Stress and TA prediction for poor response to periodontal therapy may be explained by a similar model used to link the social environmental and psychological features that may trigger physiological processes leading to disease susceptibility. Immune system modifications (Rogers et al. 1979), crevicular interleukin-1 alterations (Deinzer et al. 1999), changes in gingival circulation (Manhold et al. 1971), alteration in salivary flow and components (Gupta 1966) and/or endocrine changes (Davis & Jenkins 1962) are possible mediating mechanisms involved in that process.

Knowledge concerning the possible influence of psychosocial factors on periodontal healing is scarce. More studies are needed to clarify the real effect of stress and anxiety on periodontal therapy response. In the future, new possible influence of psychological interventions on periodontal disease control therapies should be considered since behavioural and social components are already associated with periodontal disease.

## Acknowledgements

The authors are grateful to Dr. Eduardo Feres, Professor of Periodontology at Federal University of Rio de Janeiro, and to Dr. Ronnir Raggio for their assistance with statistical analyses.

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