

# An Evaluation of the Academic Stress' Affect on Periodontal Tissues

## SUMMARY

**Background/Aim:** It has been discussed over the years whether emotional stress might be a risk factor for periodontal diseases. The correlation between periodontal disease and stress can still not be explained. Our aim was to evaluate the effects of academic stress on gingival tissues in a prospective design. **Material and Methods:** The study population consisted of randomly selected 40 dental students. Clinical examinations of plaque (PI), gingival (GI) and sulcus bleeding (SBI) indices, probing pocket depth (PPD) and gingival crevicular fluid flow rate were performed along with State-Trait Anxiety Inventory at three different periods of the school year. Measurements were recorded one month before the finals (1<sup>st</sup> period), during the final exams (2<sup>nd</sup> period), and two months after the final exams (3<sup>rd</sup> period). **Results:** The changes in mean values of all parameters except plaque and pocket depth between the final and control terms were statistically significant. There was a significant correlation between gingival index and stress, plaque, pocket depth, and sulcus bleeding indices at 1<sup>st</sup> period. There was a significant correlation between gingival index and crevicular fluid at 2<sup>nd</sup> period. There was a significant correlation between gingival index and plaque, and sulcus bleeding indices at 3<sup>rd</sup> period. **Conclusions:** The present results support the hypothesis that academic stress is a significant risk factor for gingival and periodontal inflammation.

**Key words:** Gingivitis, Periodontal Diseases, Emotional Stress

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## Introduction

The hypotheses on periodontal diseases by Socransky and Haffejee, stimulated researches on host related factors causing the increase in periodontal destruction<sup>1</sup>. Stress, which effects individuals' susceptibility to the disease, is one of these factors. A life full of stress was shown to be a risk factor for periodontal diseases<sup>2-5</sup>. The increase in both acute and chronic oral symptoms was found to be related with stressful life conditions<sup>6</sup>. Stressful life events and negative emotions may modulate several physiological systems, as the endocrine and the immune system, causing the health changes. The association between stress and disease is especially evident for infectious diseases, inflammatory conditions, and impaired wound healing<sup>7,8</sup>.

The influence of stress on the immune system has been well established, and a possible influence on a chronic inflammatory disease like periodontitis appears probable<sup>9</sup>. Many researchers have been investigating the relationship between inflammatory periodontal disease and psychological factors for years<sup>10-13</sup>. Specific periodontal conditions associated with psychosocial variables involve chronic periodontitis, necrotizing ulcerative gingivitis, chronic and experimental gingivitis. Regarding the adults, the contribution of psychosocial factors to heightened gingivitis expression may relate to the stress-associated increase in plaque accumulation. In the reported gingivitis studies, the medical students were subjected to a major exam, as it increase levels of stress. An acute condition that relates to high levels of emotional stress may be

considered as one of the factors that modulates the clinical expression of plaque-induced gingivitis<sup>7</sup>.

Dentistry is regarded as a high-stress profession and often dental school is where stress begins. The main areas causing stress in undergraduate dental students include academic and clinical work, personal relations and environment<sup>14</sup>. Studies have showed high levels of anxiety among dental students. Students who had a major exam had significantly more plaque and gingival inflammation than the students who did not take any exams<sup>5</sup>.

Academic examinations are one of the most frequently used manipulations in stress research<sup>15</sup>. The objectives of the present study was to evaluate the academic stress determined using psychiatric questionnaires, at three different periods of a term and probable effect of out coming differences on periodontal tissues.

## Material and Methods

The study population composed of randomly selected 40 fourth grade dental students. Exclusion criteria were receiving periodontal therapy and/or antibiotics within last six months, endocrine or immune disorders, use of psychotherapeutic implementations, pregnancy and using calcium antagonists or anticonvulsants.

In order to evaluate the effect of academic stress on periodontal tissues, measurements were recorded one month before the finals (1<sup>st</sup> period), during the final exams (2<sup>nd</sup> period), two months after the final exams (3<sup>rd</sup> period) (at the beginning of the school year without any exams). Clinical examinations included periodontal charting and gingival crevicular fluid (GCF) collection, which were followed by a psychiatric questionnaire. At each period, the students were asked to fill out the questionnaire, and then by the help of a Williams periodontal probe, plaque index (PI), gingival index (GI), probing pocket depth (PPD), and sulcus bleeding index (SBI) measurements were performed at the Ramfjord teeth. Gingival Crevicular Fluid (GCF) samples were collected from vestibular aspects of right and left maxillary canine teeth according to the method described by Brill<sup>16</sup>. Prior to GCF sampling the respective teeth were dried by isolation with cotton rolls and air stream. Paper strips were inserted into the gingival crevice until resistance was felt and these were kept in place for a minute. Following this, the strips were dipped in alcoholic dilution of ninhydrine 0,2% and kept in the dark for at least 24 h. The two long edges of the strips were measured with a digital compass and the mean value was calculated.

State-Trait Anxiety Inventory by Spielberger *et al.* was used for determining emotional stress levels of the individuals<sup>17</sup>. This is a self-evaluation questionnaire

composed of short expressions. The two-stage psychiatric test was used in our study. First was "State Anxiety Test" which implies individual's present stress condition and the second was "Trait Anxiety Test" which informs us about the general stress status.

The study design was approved by local ethical review committee for human subjects, it was conducted in accordance with the Helsinki Declaration of 1975, as revised in 2000 and all participants provided written informed consent.

## Statistical analysis

Data analyses were performed by means of statistical software. ANOVA was used to determine significance between the differences in mean changes of all parameters for each individual at each period (SPSS, IBM, Chicago, IL. USA). The relationships between all parameters of each period were evaluated using Pearson's Correlation Coefficient for each individual.

## Results

At each measurement time, there were significant differences between the values of all parameters and these differences caused differences in the arithmetic averages of parameters.

Statistical analysis revealed that the differences in arithmetical averages between measurement periods (P) were statistically important ( $p < 0,001$ ). Moreover, the difference between P1-P2, and P2-P3 was statistically significant ( $p < 0,001$ ).

The changes in mean Trait Anxiety Test at 3 periods were also statistically significant ( $p < 0,001$ ). The comparison of S1-S2 and S2-S3 among each other also indicated statistically significant differences (S: Stress;  $p < 0,001$ ).

The amount of GCF increased from 1<sup>st</sup> to 2<sup>nd</sup> period, decreased from 2<sup>nd</sup> to 3<sup>rd</sup> period and this was statistically significant for both situations ( $p < 0,001$ ). The alterations in mean values at 3 periods were also statistically significant ( $p < 0,001$ ).

Table 1. Relationships between parameters according to Pearson Correlation Coefficient at 1st period

	SAT	TAT	GCF	PI	GI	PPD	BOP
SAT	1,00	-0,47**	-0,27	0,16	-0,11	0,13	0,03
TAT	-0,47**	1,00	0,12	0,26	0,37*	0,59	0,24
GCF	-0,27	0,12	1,00	0,15	0,06	0,01	0,06
PI	0,16	0,26	0,15	1,00	0,45**	0,22	0,28
GI	-0,11	0,37*	0,06	0,45**	1,00	0,40**	0,60**
PPD	0,13	0,06	0,10	0,22	0,40**	1,00	0,38*
BOP	0,03	0,24	0,06	0,28	0,60**	0,38*	1,00

\*Correlation significance level 0,05

\*\* Correlation significance level 0,01

According to the results of ANOVA, the changes in PI were not significant ( $p < 0,001$ ). However, the outcomes of testing at 10 % risk level were significant ( $p < 0,10$ ). The differences in PI from 1<sup>st</sup> period to 2<sup>nd</sup> and from 2<sup>nd</sup> to 3<sup>rd</sup> were not statistically significant as well ( $p < 0,001$ ).

The alterations in mean GI scores were significant between 3 time intervals. The differences in mean values of each period were significant ( $p < 0,001$ ). The comparison of time intervals among themselves also showed significant differences ( $p < 0,001$ ).

The differences in SBI values were also statistically significant ( $p < 0,001$ ). The changes between 3 periods for SBI were significant ( $p < 0,001$ ).

Pearson’s Correlation Coefficient was used to define the probable relationship between parameters that were measured at 3 different periods. The correlation between each parameter at 1<sup>st</sup> period was negative and statistically significant ( $p < 0,01$ ). There was a positive and significant relation between GI1 and S1 ( $p < 0,05$ ). There was a fairly strong linear relationship between PI1 and GI1 ( $p < 0,01$ ). The relationships between PPD1 and GI1; SBI1 and GI1; and PPD1 and SBI1 were also statistically significant ( $p < 0,01$ ;  $p < 0,01$  and  $p < 0,05$  respectively).

The measurements at 2<sup>nd</sup> period revealed significant relationships between GCF-PI, GI-PI, GI-SBI, PI-SBI, and SBI-PPD ( $p < 0,01$ ). The correlations between GI-GCF, PPD-GCF, PPD-PI, PPD-GI, and SBI-PI were at  $p < 0,05$  significance level. At 3<sup>rd</sup> period the only significant relationships were between GI-PI and GI-SBI ( $p < 0,01$ ;  $p < 0,05$  respectively).

Table 2. Relationships between parameters according to Pearson Correlation Coefficient at 2nd period

	SAT	TAT	GCF	PI	GI	PPD	BOP
SAT	1,00	-0,10	0,06	-0,05	-0,05	-0,10	0,00
TAT	-0,10	1,00	0,01	-0,24	-0,92	0,03	0,11
GCF	0,06	0,01	1,00	0,41**	0,40*	0,32*	0,28
PI	-0,05	-0,02	0,41**	1,00	0,62**	0,40*	0,42*
GI	-0,05	-0,09	0,40*	0,62**	1,00	0,37*	0,55**
PPD	-0,10	0,03	0,32*	0,40*	0,37*	1,00	0,54**
BOP	0,00	0,11	0,28	0,42**	0,55**	0,54**	1,00

\*Correlation significance level 0,05

\*\* Correlation significance level 0,01

Table 3. Relationships between parameters according to Pearson Correlation Coefficient at 3rd period

	SAT	TAT	GCF	PI	GI	PPD	BOP
SAT	1,00	-0,17	-	0,01	0,04	0,19	-0,03
TAT	0,17	1,00	0,03	-0,25	-0,24	0,05	0,01
GCF	-	0,03	1,00	0,02	-0,29	0,31	-0,13
PI	0,01	-0,25	0,02	1,00	0,62**	0,20	0,23
GI	0,04	-0,24	-0,29	0,62**	1,00	0,06	0,37*
PPD	0,19	0,06	0,31	0,20	0,06	1,00	0,12
BOP	-0,03	0,01	-0,13	0,23	0,37*	0,12	1,00

\*Correlation significance level 0,05

\*\* Correlation significance level 0,01

## Discussion

In the present study we aimed to measure the stress levels of same individuals at different periods since responses against stressful events differ due to agents like personality and environmental factors. Thus, we could compare the responses of same individual at different time intervals and evaluate their effects on gingival tissues. Finals period was considered the most stressful time period. Therefore we aimed to compare academic stress at finals period with two different periods.

Statistical analyses revealed that, for State Anxiety Test, the changes being highly significant between time periods, the students’ conditional stress was more than other two periods during finals (Figure 1). The first measurements’ being higher than third was considered to be related with the fact that it was end of the educational year and the students were tired. These changes reflected that high conditional anxiety values detected during stressful times decreased when stress diminished. However, since the individuals in our study were exposed to a long term stress, such as finals period, their general stress levels were also affected and changes in Trait Anxiety values were significant. The raise in stress levels indicated with the SAT during examination period was in accordance with other studies<sup>5,14,18,19</sup>.

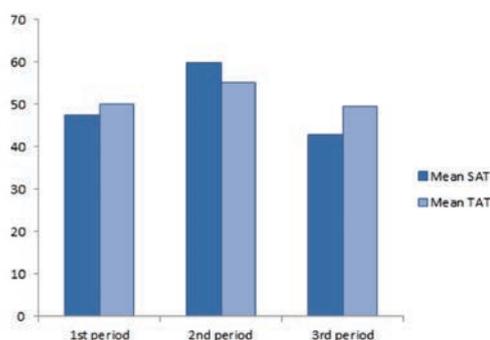


Figure 1. Alterations in mean State and Trait Test scores

In the present study, statistically significant changes were observed for all parameters except PI and PPD. The change in PI scores was not significant at  $p < 0,001$  levels. Likewise, the changes in PI among time periods were not significant ( $p < 0,001$ ). The non-significant results on PI were in accordance with Deinzer *et al.* report on medical school students<sup>8,20</sup>.

The differences in changes of GI values between time periods were statistically significant ( $p < 0,001$ ) and evaluation of time periods among themselves revealed that changes were statistically significant ( $p < 0,001$ ). Mean SBI values between time periods were also significant ( $p < 0,001$ ). The correlation between stress measures and GI and SBI did not indicate significance, like other

parameters. This alteration in gingival inflammation was also in accordance with Deinzer *et al*<sup>20</sup>.

There was, although statistically not important, a negative correlation between State and Trait Anxiety at all three measurement periods. This may be a result of individual responding less to an acute stress at the time of a general stress<sup>21</sup>. Deinzer *et al.* evaluated the changes in IL-1 levels at stressful time intervals and reported that being exposed to emotional stress at certain time points rather than constantly, increases IL-1 $\beta$  release more. Thus, IL-1 secretion increases more when conditional anxiety occurs one after another rather than when a constant anxiety is present.

The other results of correlation analysis indicated that, at 1<sup>st</sup> interval there was a positive correlation between GI and S, PI, PPD, SBI ( $p < 0,05$  for S;  $p < 0,01$  for PI, PPD and SBI). At 2<sup>nd</sup> interval there was a linear relation between GI and GCF, PI, PPD, SBI. At 3<sup>rd</sup> interval, however, there was a positive correlation only between GI and PI, and GI and SBI.

The correlation analysis between changes in stress measures and alterations in other parameters indicated a linear relation between first stress and GI measurements; but there were no correlations between other parameters. On the other hand, the changes seen in stress measures were observed in GI in the same way, and this raises the thought of a probable relationship between stress and GI. Since this was not a linear relationship, it could not be observed in statistical analyses. The similarity between the changes in State and Trait Anxiety at three time periods and the alterations in parameters indicating gingival inflammation, e.g. GI, SBI, and GCF, and the statistical significance among these leads us to consider that there may be a relationship between all these parameters. The changes in stress measures are observed at these parameters in a similar way. For instance, the alteration in State and Trait Anxiety measures from 1<sup>st</sup> to 2<sup>nd</sup> period was statistically significant and there were increases in GI, SBI, and GCF as well. An increase in PI and PPD also existed but this was not statistically significant. Moreover, from 2<sup>nd</sup> period, which was the one with highest stress scores, to 3<sup>rd</sup> period similar to the decrease in stress levels, the decrease in other parameters were also statistically significant. Depending on the data above, we consider that there may be a relationship between stress and gingival inflammation<sup>5,10</sup>.

Waschul *et al.* aimed to analyze whether the effects of emotional stress and experimental gingivitis on interleukin-1b (Il-1b) described before are compensated by concomitant increases in Il-1 receptor antagonist (Il-1ra), and whether gender differences exist in the Il-1 responses. They studied 13 medical students participating in a major academic exam and 14 medical students without academic stress refrained from oral hygiene in two quadrants. Neither stress nor experimental gingivitis exhibited significant effects on Il-1ra from weekly crevicular fluid samples. Similar results were found with respect to bleeding on probing. They stated that gender

should be monitored in studies on periodontal responses to pathogens and stress plays a role in this response<sup>22</sup>. In the present study, comparisons between genders were not performed.

The presence of statistically significant differences between mean GI scores and absence of this significance between PI scores indicates the changes caused by stress at cellular level, but not lack of oral hygiene. The increase in GI scores at stressful periods may be due to this reason.

The local psychological effect of stress is also being discussed. It is accepted that stress influences gingival microcirculation and temperature; but further studies are required. Both factors cause the condition to become convenient for periodontitis progression<sup>23-25</sup>.

The changes in periodontal pocket depth of three periods were not statistically significant. Previous studies have shown that chronic stress causes serious destruction in long term<sup>26</sup>. According to text books, for stress to cause destruction in tissues, a weak point must be present at that organ<sup>27,28</sup>. The individuals in our study were relatively periodontally healthy and this minimized the risk. Moreover, the time of stress exposure being short and inflammation being at the lowest level reduced the attachment loss-induced pocket depth increase. Since ours was a short-term study, the slight increase in probing pocket depth during the exams period may be due to the edema resulting from the increase in gingival index.

The responses of students against same types of stress at same intensity during the same time period differed. Personality plays a major role in the host response against stress. Trombelli *et al.* hypothesized that personality traits, coping style, stress, perception of stress and social support could modify a gingival inflammatory response to the new plaque accumulation; and found that psychological variables related to stress susceptibility/resistance and the current level of stress do not have an influence to clinical parameters of gingival inflammation during experimental gingivitis, and they do not contribute to the differences in individual susceptibility to plaque-induced gingivitis<sup>7</sup>.

Emotional stress is a risk indicator for periodontal diseases and should be determined before and during treatment<sup>9</sup>. Studies also have demonstrated the changes at cellular and organ level along with indicating that pathological changes do not necessarily occur at each individual. No pathological changes might occur against even a stress of high intensity. This also implies the importance of individual factors.

## Conclusions

The present results support the hypothesis that academic stress is a significant risk factor for gingival and periodontal inflammation.

## References

1. Socransky SS and Haffeje AD. Microbial mechanisms in the pathogenesis of destructive periodontal diseases; a critical assessment. *J Periodontol Res*, 1991;26:195-212.
2. Cohen-Cole S, Cogen R, Stevens A, Kirk K, Gaitan E, Hain J, et al. Psycho-social, endocrine and immune factors in acute ulcerative gingivitis (trench mouth). *Psychosom Med*, 1981;43: 91.
3. Arowojolu MO, Onyeano CO, Dosumu EB, Idaboh GK. Effect of academic stress on periodontal health in Nigerians. *Odontostomatol Trop*, 2006;29:9-13.
4. Becker R, Karp CL, Becker W, Berg L. Personality differences and stressful life events. Differences between treated periodontal patients with and without maintenance. *J Clin Periodontol*, 1988;15:49-52.
5. Johannsen A, Bjurshamar N, Gustafsson A. the influence of academic stress on gingival inflammation. *Int J Dent Hygiene*, 2010;8:22-27.
6. Mercenes WS, Croucher R, Marmot MG. The relationship between self-reported oral symptoms and life events. *J Dent Res*, 1992;72:702.
7. Trombelli L, Scapoli C, Tatakis DN, Grassi L. Modulation of clinical expression of plaque-induced gingivitis: effects of personality traits, social support and stress. *J Clin Periodontol*, 2005;32:1143-1150.
8. Deinzer R, Granrath N, Spahl M, Linz S, Waschul B, Herforth A. Stress, oral health behavior and clinical outcome. *Br J Health Psychol*, 2005;10:269-283.
9. Hildebrand HC, Epstein J, Larjava H. The influence of psychological stress on periodontal disease. *J West Soc Periodontol Periodontol Abstr*, 2000;48:69-77.
10. Breivik T, Thrane PS, Murison R, Gjerme P. Emotional stress effects on immunity, gingivitis, and periodontitis. *Eur J Oral Sci*, 1996;104:327-334.
11. Baker EG, Crook GH, Schwabacher ED. Personality correlates of periodontal disease. *J Dent Res*, 1961;40:396-409.
12. Clarke NG, Hirsch RS. Personal risk factor for generalized periodontitis. *J Clin Periodontol*, 1995;22:136-145.
13. Monteiro da Silva AM, Newman HN, Oakley DA. Psychological factors in inflammatory periodontal disease. *J Clin Periodontol*, 1995;22:516-523.
14. Muirhead V and Locker D. Canadian dental students' perceptions of stress. *JCDA*, 2007;73:323-e.
15. Bosch JA, de Geus EE, Ring C, Nieuw Amerongen AV, Stowell JR. academic examinations and immunity: Academic stress or examination stress. *Psychosom Med*, 2004;66:625-626.
16. Griffiths GS. Formation, collection and significance of gingival crevice fluid. *Periodontol* 2000, 2003;31:32-42.
17. Spielberger CD, Gorsuch RL, Luchene RE. Manual for state-trait anxiety inventory. Palo Alto, California: California Consulting Psychologist's Press, 1970.
18. Murphy L, Denis R, Ward CP, Tartar JL. Academic stress differentially perceived stress, salivary cortisol, and immune globulin-A in undergraduate students. *Stress*, 2010;13:366-371.
19. Naumova EA, Sandulescu T, Al Khatib P, Thie M, Lee WK, Zimmer S, et al. Acute short-term mental stress does not influence salivary flow rate dynamics. *PLoS One*, 2012;7:e51323.
20. Deinzer R, Rutermaun S, Mobes O, Herfort A. Increase in gingival inflammation under academic stress. *J Clin Periodontol*, 1998;25:431-433.
21. Deinzer R, Kottmann W, Förster P, Herforth A, Stiller-Winkler R, Idel H. After effects of stress on crevicular interleukin-1 $\beta$ . *J Clin Periodontol*, 2000;27:74-77.
22. Waschul B, Herforth A, Stiller-Winkler R, Idel H, Granrath N, Deinzer R. Effects of plaque, psychological stress, and gender on crevicular Il-1 and Il-1ra secretion. *J Clin Periodontol*, 2003;30:238-248.
23. Alexander MB, Damoulis PD. The role of cytokines in the pathogenesis of periodontal disease. *Curr Opin Periodontol*, 1994;39-53.
24. Ebersole JL, Cappelli D, Steffen MJ. Characteristics and utilization of antibody measurements in clinical studies of periodontal disease. *J Periodontol*, 1992;63:1110-1116.
25. Lamster IB. The host response in gingival crevicular fluid: potential applications in periodontitis clinical trials. *J Periodontol*, 1992;63:1117-1123.
26. Linden GJ, Mullually BH, Freeman K. Stress and progression of periodontal disease. *J Clin Periodontol*, 1996;23:675-680.
27. Bilge M. Hormonlar Bilimi. Istanbul: Istanbul University Cerrahpasa Medical School Publications, 1975:102-178.
28. Ganong WF. Review of Medical Physiology 17th ed. Volume 1. Istanbul: Baris Kitapevi, 1995;121:434.

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