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Modulation of clinical expression of plaque-induced gingivitis: effects of personality traits, social support and stress

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Abstract

Background: Studies have shown an association between an acute stressful event and gingivitis. However, the possible effects of personality traits associated with stress resistance/susceptibility and current level of stress on the clinical expression of plaque-induced inflammation remain to be examined. The aim of this study was to characterize the subject-based clinical behaviour of the gingiva during experimental gingivitis in relation to personality profile, psychological stress and coping behaviour.

Methods: Ninety-six systemically and periodontally healthy subjects (mean age: 23.6 ± 1.7 years), 46 males and 50 females, non-smokers, participated in a randomized, split-mouth, localized experimental gingivitis trial. Prior to the trial, subjects were asked to complete self-administered questionnaires evaluating personality traits (Hardiness scale and Courtauld Emotional Control Scale), subjective stress (Visual Analogue Scale – Total Distress), social support (Multidimensional Scale of Perceived Social Support, MSPSS) and life events (Life Experiences Survey (LES)). The influence of psychosocial factors was investigated in the overall population as well as in two sub-populations with different inflammatory response to plaque accumulation.

Results: No significant relationships were found between gingival inflammation variables and psychological measures. No significant differences were detected between subjects with different susceptibilties to plaque-associated gingivitis for any considered psychological variable. A significant association between plaque variables and LES (negative) or MSPSS (positive) was found; however, the variance explained by the model was low.

Conclusions: Differences in the current level of stress and psychosocial variables indicative of stress susceptibility do not account for variability in plaque accumulation and gingival inflammation during experimental gingivitis in young adults.

Key words: gingivitis; personality tests; psychological; social support; stress

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Stressful life events and negative emotions have been shown to modulate several physiological systems, including the endocrine and the immune system, leading to health changes (Kiecolt-Glaser et al. 2002, LeResche & Dworkin 2002). The association between stress and disease is particularly strong for infectious diseases, inflammatory conditions and impaired wound healing (Kiecolt-Glaser et al. 2002, LeResche & Dworkin 2002, Broadbent et al. 2003). Several disorders of the gastrointestinal system, such as gastric and duodenal ulcers (Holtmann et al. 1992, Levenstein 2000, Overmier & Murison 2000), inflammatory bowel disease (Robertson et al. 1989, Talal & Drossman 1995), irritable bowel syndrome (Locke et al. 2004), and periodontal diseases (da Silva et al. 1995, Breivik et al. 1996, LeResche & Dworkin 2002), have been associated with psychosocial stressors.

Specific periodontal conditions associated with psychosocial variables include chronic periodontitis (Green et al. 1986, Linden et al. 1996, Genco

et al. 1999, Pistorius et al. 2002, Wimmer et al. 2002), necrotizing ulcerative gingivitis (Shields 1977, Cohen-Cole et al. 1983, Horning & Cohen 1995), chronic and experimental gingivitis (Kurer et al. 1995, Minneman et al. 1995, Deinzer et al. 1998, Waschul et al. 2003). In adults, the reported contribution of psychosocial factors to heightened gingivitis expression (Deinzer et al. 1998) may relate to the stress-associated increase in plaque accumulation (Deinzer et al. 2001). In the reported gingivitis studies (Deinzer et al. 1998, 2001, Waschul et al. 2003), the test subjects (medical students) were exposed to a specific event (major exam), known to increase subjective levels of stress (Deinzer & Schüller 1998, Deinzer et al. 2001, Waschul et al. 2003). Therefore, an acute condition that relates to high levels of psychological stress can be considered as one of the factors that modulates the clinical expression of plaque-induced gingivitis (Tatakis & Trombelli 2004). However, the possible association of other psychosocial variables, such as personality traits, which are associated with either susceptibility or resistance to stress, with changes in the inflammatory response of the gingiva to de novo plaque accumulation, remains to be examined.

The aim of the present experimental gingivitis study was to characterize the subject-based clinical behaviour of the gingiva with respect to psychosocial variables related to the capacity to cope with stressful events as well as the perceived current level of stress. In particular, the impact of these psychosocial variables on gingival inflammation was comparatively analysed in two sub-populations presenting a significantly different gingival inflammatory response in the absence of any differences in plaque exposure (Trombelli et al. 2004a, b). We hypothesized that personality traits, coping style, stress, perception of stress and social support will modulate a subject's gingival inflammatory response to de novo plaque accumulation.

Materials and Methods

Experimental design and study population

The overall experimental design has been described previously (Tatakis & Trombelli 2004, Trombelli et al. 2004a, b), and the clinical analysis of the examined population, consisting of 96 systemically and periodontally healthy non-smokers, 46 males (mean age: 23.9 ± 1.7 years) and 50 females (mean age: 23.3 ± 1.6 years), has been detailed (Trombelli et al. 2004a, b). Briefly, a randomized split-mouth localized experimental gingivitis clinical trail was conducted in volunteers. In each subject, one maxillary quadrant was randomly assigned as ''test'' (experimental gingivitis) and the contralateral quadrant as ''control''. According to gingival crevicular fluid (GCF) values, as recorded on day 21 in test quadrants and standardized on the derived clinical parameter ''cumulative plaque exposure'', it was possible to discriminate two sets of individuals, defined as high responders (HRs; $n = 24$) and low responders (LRs; $n = 24$), with significantly different severity of gingivitis to similar amounts of plaque deposits. The HR group comprised 13 males and 11 females (mean age: 24.1 ± 1.6 years) and the LR group comprised 11 males and 13 females (mean age: 23.4 ± 1.9 years) (Trombelli et al. 2004a).

The study design was approved by the local ethical committee, and was found to conform to the requirements of the ''Declaration of Helsinki'' as adopted by the 18th World Medical Assembly in 1964 and subsequently revised (www.wma.net/e/policy/17-c_e.html). All participants provided written informed consent.

Clinical parameters

The following clinical parameters, defined in detail previously (Trombelli et al. 2004a), were obtained in the order listed below from the selected sites: gingival index (GI), plaque index (PlI), GCF volume, angulated bleeding score (AngBS) and the derived parameter ''cumulative plaque exposure'' (CPE) was calculated. CPE represents the area under the curve (AUC) of subject-specific PlI over a specific period of time (Trombelli et al. 2004a). All clinical parameters were recorded at days 0, 7, 14 and 21 by two trained and calibrated examiners with good to excellent intra- and inter-examiner agreement, as measured by the *k* coefficient (Trombelli et al. 2004a).

Psychological measures

One week before the beginning of the experimental gingivitis period (day 7), all subjects were asked to complete a series of self-report psychological questionnaires aimed at evaluating personality traits and coping styles (Hardiness scale and Courtauld Emotional Control Scale), the current level of emotional stress (Visual Analogue Scale), social support (Multidimensional Scale of Perceived Social Support (MSPSS)) and occurrence/subjective perception of stressful life events in the previous year (Life Experiences Survey (LES)). Subjects were assured that their answers would be held strictly confidential to help encourage complete and truthful self-reporting. All questionnaires have been previously used to evaluate Italian populations and showed acceptable levels of validity and internal consistency (Grassi et al. 2000, 2002).

The Hardiness Scale (Kobasa & Puccetti 1983, Kobasa et al. 1985) was used to examine the presence of personality traits that have been shown to be associated with a higher psychophysiological resistance (e.g. to stressful events). The scale is a 50-item questionnaire that evaluates on a 0–3 Likert-scale the individual's capacity to deal with stressful events in a competitive way (hard style) rather than in a fatalistic or resigned way. According to what is indicated (Kobasa & Puccetti 1983) and used in the Italian population (Costantini et al. 1997), the scoring system was based on computation of the scores of three sub-scales, namely Challenge (17 items: each item is given a score ranging from 0 to 3; for the subscale score the sum of raw scores is divided by 51), Commitment (16 items: each item is given a score ranging from 0 to 3; for the subscale score the sum of raw scores is divided by 48) and Control (17 items: each item is given a score ranging from 0 to 3; for the subscale score the sum of raw scores is divided by 51). To obtain the Hardiness total score, the scores from the three subscales are added, and the sum is multiplied by 100 and divided by 3. Higher scores correspond to a competitive way of coping with stressful events, indicating a better adjustment to both the psychophysiological concomitants of stress.

The Courtauld Emotional Control Scale (CEC-S) (Watson & Greer 1983) is a 21-item questionnaire examining on a 1–4 Likert scale the individual's tendency to show or repress the behavioural expression of his/her emotions. Three emotional reactions are investigated, specifically, anger (anger sub-scale,

seven items; score range: 7–28), anxiety (anxiety sub-scale, seven items; score range: 7–28) and sadness (depression sub-scale, seven items; score range: 7–28). A total CEC-S repression score is yielded by summing up the three subscales (range: 21–84). The CECS was used as it has been demonstrated that a tendency to repress and control one's own emotions, especially anger, is related to higher vulnerability to the psychophysiological effects of stress (Watson et al. 1984).

The MSPSS (Zimet et al. 1988, 1990) consists of a 12-item questionnaire identifying the level of support from interpersonal ties. A Total Social Support score ranges from 12 to 84, with higher scores corresponding to high support and better adjustment to stress (Bolger & Eckenrode 1991, Grassi et al. 2000). The MSPSS was used in order to gain a measure of a possible mediator between stress and psychophysiological response to stress, as suggested by previous studies (Zimmermann-Tansella et al. 1993, Shields 2004).

The LES (Sarason et al. 1978) was used to assess the number and impact of possible life events that might have occurred in the last year, as it has been demonstrated that a high level of chronic stress tends to reduce the individuals capacity to adequately respond, in psychobiological terms, to acute stressors (van Eck et al. 1996, Leserman et al. 1998, Spiegel & Sephton 2001, Tse & Bond 2004). The LES consists of a list of pre-codified 62 events (e.g. divorce, death of a family member, severe physical illness, significant change in leisure activity, etc.) plus possible relevant events that the subject can arbitrarily add to the list. The subjective impact of each event is rated on a seven-point Likert scale (from -3, extremely negative, to $+3$, extremely positive), the total score resulting from the algebraic sum of the positive or negative score assigned to each event (Impact Score). A negative Impact Score is indicative of a generally negative perception of events that are subjectively considered as stressful. Therefore, LES includes two subscales: the total number of life events that occurred and the Impact Score.

Finally, a 100 mm Visual Analogue Scale was used to evaluate the current level of stress, ranging from ''no stress'' to ''extreme stress'' perceived by the subject during the last week (VAS Total Distress, VAS-TD). The VAS was chosen for its easy and reliable use in measuring an acute stress condition in patients with different medical illnesses (Tanum & Malt 2001), including dental patients (Br 1999).

Statistical analysis

The subject was regarded as the statistical unit. For each clinical parameter, the recordings from the six selected sites for either test and control quadrants were added and divided by 6 to give the mean value for each subject. Therefore, for each clinical parameter at each observational period, the subject was represented by a single test and a single control value. Data were expressed by either median and inter-quartile range (IR) for non-parametric variables, or mean $\pm$ standard deviation (SD) for parametric variables.

For the Hardiness Scale and the CEC-S, the total score as well as the score from each single sub-scale were calculated. For the LES, the raw number of events and the Impact Score were determined (Sarason et al. 1985, Schuppel et al. 1996, Leserman et al. 1998). Reliability test (Cronbach α), Pearson's correlation test and ANOVA were used, when appropriate, on psychological measures.

A bivariate analysis of the relationship between psychological measures and clinical parameters, as assessed in test quadrant on day 21, was performed using Pearson's correlation test for parametric variables (PII, GCF, CPE, Hardiness scale and subscales, CEC scale and subscales, MSPSS scale) and Spearman's rank correlation test for non-parametric variables (GI, AngBS, LES subscales and VAS-TD). Multiple regression analysis was used to determine whether psychological measures could be independent predictors for plaque accumulation or gingival inflammatory variables. Specifically, multiple stepwise linear regression analysis was used for PlI, CPE and GCF as outcome variables, whereas the generalized linear model was applied to non-parametric outcome variables (GI and AngBS). When GCF, GI and AngBS were regarded as outcome variables, CPE was also entered into the regression model as a predictor.

Among the 96 volunteers, cluster analysis was used to identify different sub-populations with diverse psychological profile, characterized by significantly different levels of susceptibility to stress. The Euclidean distance of the six main psychological variables was used as a measure of distance: Hardiness total scores, CEC-S and MSPSS together with VAS-TD and the two LES subscales. An initial partition of the 96 subjects into three random clusters (clusters A–C) was iteratively improved by non-hierarchical disjunction cluster analysis with the k-means algorithm with the goal to (1) minimize variability within clusters and (2) maximize variability between clusters. ANOVA was applied to search for differences between clusters (Table 1). Therefore, we were able to identify two sub-populations showing the most extreme psychological profiles in terms of susceptibility to stress: one sub-population defined as low stress susceptible (LSS; Table 1, cluster A), and one sub-population defined as highly stress susceptible (HSS; Table 1, cluster C). The LSS sub-

Table 1. Psychological measures in clusters A–C

	Mean $\pm$ SD			*p*-value*
	cluster A ($N = 20$) (LSS)	cluster B ($N = 39$)	cluster C ($N = 37$) (HSS)	
Hardiness total	68.82 $\pm$ 9.79	69.83 $\pm$ 5.91	73.45 $\pm$ 5.96	**0.034**
CECS-Total	51.95 $\pm$ 13.26	50.77 $\pm$ 7.07	35.54 $\pm$ 5.22	**<0.001**
MSPSS-Total	47.45 $\pm$ 10.23	73.63 $\pm$ 7.12	73.78 $\pm$ 7.31	**<0.001**
	Median (IR)	Median (IR)	Median (IR)	*p*-value
VAS-TD	67.50 (40.0–80.0)	25.0 (15.0–45.0)	50.0 (35.0–70.0)	**0.002**
Number of life events	2.50 (1.0–6.0)	0.50 (0.0–2.0)	3.00 (1.0–6.0)	**0.001**
Impact Score	0.50 (-2.0–2.0)	0.00 (0.0–1.0)	3.00 (0.0–6.0)	**0.025**

*ANOVA was applied to search for difference between clusters; values in bold indicate statistical significance.

LSS, low stress-susceptible sub-population; HSS, highly stress-susceptible sub-population; CECS, Courtauld Emotional Control Scale; MSPSS, Multidimensional Scale of Perceived Social Support; VAS-TD, Visual Analogue Scale Total Distress.

population is characterized by a lower capacity to cope with stressful events (i.e. significantly lower Hardiness scale score), greater vulnerability to stressors (i.e. significantly higher CECS score) and a lower level of social support (i.e. significantly lower MSPSS total score) compared with the HSS sub-population.

To assess the effect of the current level of stress on plaque and gingivitis parameters, two sets of subjects were selected on the basis of upper and lower quartiles of the VAS-TD distribution: one set having a VAS-TD $\leqslant 20$, and one set having a VAS-TD $\geqslant 70$.

Comparisons between psychological measures in HR and LR groups, and between clinical parameters in different sets of subjects, were analysed by using the unpaired *t*-test and the Mann–Whitney *U*-test for parametric and non-parametric variables, respectively.

All analyses were performed with STATISTICA software version 5.5 (StatSoft, Italia s.r.l., Vigonza, Italy). The level of significance was set at 5%.

Table 2. Psychological measures in the study population

	N	Mean	Standard deviation	Cronbach's α-value
Challenge	94	0.59	0.09	0.55
Commitment	93	0.79	0.09	0.60
Control	84	0.75	0.08	0.56
Hardiness total	83	71.18	6.92	0.79
CECS-Anger	95	14.71	5.62	0.63
CECS-Anxiety	95	15.74	4.51	0.84
CECS-Depression	95	14.63	3.88	0.77
CECS-Total	95	45.07	11.09	0.84
MSPSS-Total	94	68.12	13.35	0.91

	N	Median	Interquartile range
VAS-TD	96	40.0	20.0–70.0
Number of life events	91	2.00	0.00–5.00
Impact Score	91	0.00	0.00–3.00

CECS, Courtauld Emotional Control Scale; MSPSS, Multidimensional Scale of Perceived Social Support; VAS-TD, Visual Analogue Scale Total Distress.

Table 3. Correlation analysis between psychological measures

	Hardiness total	CECS-Total	MSPSS-Total	VAS	Number of life events	Impact Score
Hardiness total	–					
CECS-Total	− 0.160 *N* = 82 *p* = 0.151	–				
MSPSS-Total	**0.360** ***N* = 81** ***p* = 0.001**	**− 0.284** ***N* = 93** ***p* = 0.006**	–			
VAS-TD	− 0.212 *N* = 83 *p* = 0.055	− 0.172 *N* = 95 *p* = 0.096	− 0.179 *N* = 94 *p* = 0.084	–		
Number of life events	**0.272** ***N* = 78** ***p* = 0.016**	**− 0.278** ***N* = 90** ***p* = 0.008**	− 0.029 *N* = 89 *p* = 0.786	**0.231** ***N* = 91** ***p* = 0.027**	–	
Impact Score	**0.312** ***N* = 78** ***p* = 0.005**	− 0.143 *N* = 90 *p* = 0.177	0.129 *N* = 89 *p* = 0.228	0.064 *N* = 91 *p* = 0.546	**0.403** ***N* = 91** ***p* < 0.001**	–

CECS, Courtauld Emotional Control Scale; MSPSS, Multidimensional Scale of Perceived Social Support; VAS-TD, Visual Analogue Scale Total Distress.
Statistically significant correlations are in bold

Results

Clinical parameters

Clinical parameters of plaque accumulation (PlI, CPE) and gingival inflammation (GI, AngBS, GCF) over time in both the overall population and HR/LR groups have been previously reported in detail (Trombelli et al. 2004a).

Psychological measures

Table 2 illustrates the scores for each psychological questionnaire as well as the number of subjects who completed all items included in each questionnaire sub-scale, thus providing suitable data for analysis. All the psychometric instruments showed acceptable levels of reliability, as shown by the Cronbach α values (Table 2). Furthermore, correlation analysis showed the expected strong positive association between subscales and the main scale variable for the Hardiness, CEC-S and LES variables (data not shown).

Bivariate analyses among main scales of psychological measures are summarized in Table 3. Significant positive correlations were found between Hardiness and both MSPSS and LES subscales; negative correlations were found between CEC-S and both MSPSS and the number of life events. The number of life events also positively associated with VAS-TD.

Relationship between psychological measures and clinical parameters

Only LES presents a significant correlation with both PlI – day 21 and CPE – day 21 in the test quadrant (Table 4). In particular, PlI is negatively associated with both the number of life events and the Impact Score (Spearman's $r_s = -0.26$ for both); CPE results negatively correlated only with the Impact Score (Spearman's $r_s = 0.21$).

Multiple regression analysis confirmed the negative association between plaque accumulation variables and the Impact Score observed in the bivariate analysis ($p = 0.029$ and 0.011 for CPE and PlI, respectively). The MSPSS variable was positively associated with the amount/rate of plaque deposits ($p = 0.019$ and 0.005 for CPE and PlI, respectively). However, even if these two variables result in a significant *p*-value, the variance explained from the model is quite low, around 40% for either CPE or PlI.

No significant relationships were found between gingival inflammation variables and psychological measures. However, CPE showed a significant positive association with GCF levels,

Table 4. Correlation analysis (p-value) between psychological measures and day 21 test quadrant clinical parameters

	PlI	CPE	GCF (μl)	GI	AngBS
Challenge	− 0.018 (0.867)	0.012 (0.909)	0.046 (0.662)	− 0.061 (0.556)	− 0.145 (0.164)
Commitment	− 0.098 (0.351)	− 0.046 (0.660)	0.105 (0.315)	0.055 (0.600)	− 0.027 (0.798)
Control	− 0.030 (0.790)	− 0.026 (0.812)	− 0.006 (0.958)	− 0.138 (0.210)	− 0.171 (0.119)
Hardiness total	− 0.059 (0.599)	− 0.036 (0.750)	0.026 (0.813)	− 0.045 (0.689)	− 0.142 (0.202)
CECS-Anger	0.053 (0.613)	− 0.020 (0.850)	0.006 (0.954)	0.089 (0.389)	0.129 (0.214)
CECS-Anxiety	− 0.095 (0.358)	− 0.122 (0.240)	0.034 (0.746)	0.073 (0.481)	− 0.023 (0.824)
CECS-Depression	− 0.045 (0.668)	− 0.172 (0.096)	0.010 (0.925)	− 0.078 (0.451)	− 0.088 (0.397)
CECS-Total	− 0.028 (0.789)	− 0.120 (0.248)	0.020 (0.846)	0.048 (0.646)	− 0.012 (0.907)
MSPSS-Total	0.087 (0.406)	0.083 (0.427)	0.079 (0.450)	− 0.051 (0.628)	− 0.097 (0.352)
VAS-TD	0.079 (0.446)	0.147 (0.152)	0.087 (0.399)	− 0.076 (0.462)	0.007 (0.945)
Number of life events	**− 0.255 (0.015)**	− 0.201 (0.056)	− 0.195 (0.063)	− 0.143 (0.177)	− 0.092 (0.387)
Impact Score	**− 0.258 (0.014)**	**− 0.213 (0.043)**	− 0.111 (0.295)	− 0.056 (0.597)	− 0.060 (0.575)

Values in bold indicate statistical significance.

CECS, Courtauld Emotional Control Scale; MSPSS, Multidimensional Scale of Perceived Social Support; VAS-TD, Visual Analogue Scale Total Distress; PlI, plaque index; CPE, cumulative plaque exposure; GCF, gingival crevicular fluid; GI, gingival index; AngBS, angulated bleeding score.

even when controlled for the effect of psychosocial variables ($p < 0.001$, $r^2 = 0.59$).

Comparison of psychological measures in subjects with different susceptibility to gingivitis

Psychological measures as assessed in HR and LR subjects are summarized in Table 5. No significant differences were detected between subjects with different susceptibility to plaque-associated gingivitis for any psychological variable considered.

Comparison of clinical parameters in subjects with different susceptibility/ resistance to stress

LSS sub-population (Table 1, cluster A) comprised 20 subjects, 12 males and eight females (mean age: 23.7 years), and the HSS sub-population (Table 1, cluster C) comprised 37 subjects, 13 males and 24 females (mean age: 23.4 years). No significant differences were found between LSS and HSS with respect to age and gender distribution ($p > 0.1$). When the clinical parameters of plaque accumulation and gingival inflammation were compared in LSS and HSS sub-populations, no significant differences were detected (Table 6).

Comparison of clinical parameters in subjects with different levels of stress

Twenty-seven subjects (18 males, nine females; mean age: 24.2 years) presented a VAS-TD $\leqslant 20$, and 29 subjects (14 males, 15 females; mean age: 23.2 years) presented a VAS-TD $\geqslant 70$. No significant differences were found in clinical parameters of plaque accumulation and gingival inflammation between groups ($p > 0.2$).

Table 5. Psychological measures assessed in low responder (LR) and high responder (HR) subjects

	N	LR: mean ± SD	N	HR: mean ± SD	t-test	df	p-value
Challenge	24	0.59 ± 0.108	23	0.60 ± 0.079	− 0.40	45	0.69
Commitment	24	0.78 ± 0.091	23	0.82 ± 0.065	− 1.74	45	0.09
Control	23	0.75 ± 0.084	19	0.76 ± 0.060	− 0.26	40	0.80
Hardiness total	23	70.70 ± 7.764	19	72.28 ± 5.437	− 0.74	40	0.46
CECS-Anger	23	16.04 ± 9.335	24	15.71 ± 3.342	0.17	45	0.87
CECS-Anxiety	23	16.26 ± 3.793	24	16.71 ± 4.319	− 0.38	45	0.71
CECS-Depression	23	15.00 ± 3.954	24	15.50 ± 3.799	− 0.44	45	0.66
CECS-Total	23	47.30 ± 13.792	24	47.92 ± 9.627	− 0.18	45	0.86
MSPSS-Total	24	67.63 ± 14.685	24	70.29 ± 12.526	− 0.68	46	0.50
	N	Median (IR)	N	Median (IR)	U-test	df	p-value
VAS-TD	24	45.0 (25.0–70.0)	24	45.0 (20.0–70.0)	− 0.13	46	0.89
Number of life events	23	2.00 (0.0–5.0)	23	1.00 (0.0–3.0)	0.85	44	0.39
Impact Score	23	0.00 (0.0–5.0)	23	0.00 (0.0–3.0)	− 0.31	44	0.75

CECS, Courtauld Emotional Control Scale; MSPSS, Multidimensional Scale of Perceived Social Support; VAS-TD, Visual Analogue Scale Total Distress.

Table 6. Clinical parameters assessed in clusters A and C

	Mean ± SD		p-value
	cluster A (LSS) ($N = 20$)	cluster C (HSS) ($N = 37$)	
PlI	1.63 ± 0.452	1.65 ± 0.300	0.779
CPE	26.37 ± 6.076	27.80 ± 4.637	0.325
GCF (μl)	0.32 ± 0.151	0.33 ± 0.099	0.826
	Median (IR)	Median (IR)	p-value
GI	0.50 (0.50–0.92)	0.67 (0.33–0.83)	0.547
AngBS	0.50 (0.17–1.17)	0.50 (0.17–0.83)	0.463

LSS, low stress susceptible sub-population; HSS, highly stress-susceptible sub-population; SD, standard deviation; IR, interquartile range; PlI, plaque index; CPE, cumulative plaque exposure; GCF, gingival crevicular fluid; GI, gingival index; AngBS, angulated bleeding score.

Discussion

The aim of the present randomized, split-mouth, controlled trial was to determine the effect of psychosocial variables on clinical parameters of plaque accumulation and gingival inflammation following a 21-day experimental gingivitis trial, and the association of psychosocial variables with individual susceptibility to plaque-induced gingivitis. Results from the present study indicate that psychological variables related to stress susceptibility/resistance and the current level of stress do not contribute to differences in clinical parameters of gingival inflammation during experimental gingivitis, and they do not account for differences in individual susceptibility to plaque-induced gingivitis as detected in LR and HR subjects. Moreover, the results indicate that only stressful life events and their subjective impact have a significant, albeit weak, correlation with plaque accumulation.

Recently, several studies have been published analysing the relationship between psychological stress and destructive periodontal disease, leading to the general conclusion that stress represents a risk factor for periodontitis (da Silva et al. 1995, Breivik et al. 1996, LeResche & Dworkin 2002). The exact mechanisms through which psychological stress might increase susceptibility to periodontitis remain undetermined, although both physiological, through the psycho-neuro-endocrine pathways of immune response regulation, and behavioural changes have been implicated (da Silva et al. 1995, Breivik et al. 1996, LeResche & Dworkin 2002). In this context, the evaluation of the impact of stress on modulating the clinical expression of plaque-induced gingival inflammation is clinically relevant when one considers that increased gingival inflammation has been shown to be a risk factor/indicator for periodontal breakdown at both the patient and the site level (Joss et al. 1994, Schätzle et al. 2003).

Previous reports have clearly shown that psychosocial stress can affect oral hygiene behaviour and can increase plaque accumulation (Deinzer et al. 2001). In the present study, we found no association between the current level of stress and either amount of plaque deposits or plaque accumulation rate. On the other hand, we observed a significant association between plaque variables and the subjective impact of stressful events (negative) or the level of social support (positive). The association of these psychological factors with plaque variables likely represents complex relationships, e.g., where psychological factors moderate host physiological aspects associated with plaque accumulation; such relationships remain to be elucidated. However, statistical analysis revealed that these psychological factors are only weak predictors for clinical parameters of plaque accumulation. Discrepancies between this and previous studies may be partly attributed to study design differences. In the study by Deinzer et al. (2001), subjects were not informed of the purpose of the study, i.e., to assess the effect of academic stress on oral hygiene, and no attempt was made to alter experimentally the oral hygiene habits of the participants during the study period. In contrast, subjects in the present study were asked to cease oral hygiene measures voluntarily in experimental quadrants, while oral hygiene instructions were weekly reinforced to ensure optimal plaque control in control quadrants (Trombelli et al. 2004a, b). Therefore, as in all controlled experimental gingivitis trials, plaque deposits observed in the present study may not be regarded as representative of spontaneous oral hygiene behaviour. As stress appears to affect plaque accumulation through changes in oral hygiene behaviour (Deinzer et al. 2001), the controlled nature of oral hygiene behaviour in an experimental gingivitis trial could explain the lack of a stress effect on plaque accumulation reported herein, a finding consistent with reports from other studies of this type (Deinzer et al. 1999, Waschul et al. 2003).

In our material, none of the clinical parameters of gingival inflammation were correlated with the current level of stress or the stress susceptibility/resistance of the individual. This result is consistent with other experimental gingivitis studies, where stress had no effect on either GCF levels (Deinzer et al. 1999) or bleeding indices (Deinzer et al. 1999, Waschul et al. 2003), although the possibility of a gender-specific effect of stress on bleeding indices may not yet be excluded (Waschul et al. 2003). It should be noted that there are significant methodological differences between these studies. In the present experimental gingivitis study, measures were taken to include only non-smokers and to eliminate or control for all other possible parameters known to contribute in modifying the inflammatory response of the gingiva to plaque accumulation, such as vitamin C nutritional status (Tatakis & Trombelli 2004). Furthermore, in the present study, the young adult subjects were not intentionally subjected to a stressful event, unlike the test group in previous studies where the stress–gingivitis relationship was investigated (Deinzer et al. 1999, Waschul et al. 2003).

As previously reported (Pengilly & Dowd 2000), some psychosocial measures, which relate to personality traits and perceived social support, were significantly inter-correlated. Based on this observation, two sub-populations of individuals (LSS and HSS, characterized by different personality traits and social support) were identified and analysed with respect to clinical parameters of plaque accumulation and gingival inflammation. The assumption was that the subjective tendency to repress the emotions, the level of social support and the capacity to cope with stressful events would somewhat determine the individual susceptibility or resistance to stressors and this, in turn, would exert an effect on gingival inflammatory response. However, when LSS and HSS sub-populations were compared, no differences in GCF volume or other gingival inflammatory indexes were detected (Table 6). These results do not support an association between susceptibility/resistance to stress and either the susceptibility to or the severity of plaque-induced gingivitis. In general, our study population reported scores in all the psychological measures (hardiness, emotional repression, social support) that were comparable with normative data of the Italian population (Costantini et al. 1997, Grassi et al. 1985, 2000). The current level of stress was low to moderate (median VAS-TD = 40), and the number of major stressful life events reported in the previous year was quite low. Even when the HSS sub-population was considered, the perceived stress and the number of stressful events were not in the highest range (Table 1). In such a context, the possible negative psychophysiological effects of chronic stress, as reported in other studies of subjects facing multiple daily stressors (Biondi et al. 1994), may have been underestimated. Nevertheless, the results of the present study indicate that either the susceptibility/

resistance to stress or the perceived level of stress at the time of induced gingival inflammation, in the absence of significant stressful events, contributes little, if any, to the observed differences in plaque accumulation and related gingivitis expression. In fact, the results of the multiple regression analysis confirmed here that, even accounting for possible differences in psychological variables, the exposure to supragingival plaque deposits (CPE) has a strong association with GCF levels, as reported previously (Trombelli et al. 2004a).

In the original report of this experimental gingivitis cohort, it was possible to identify two sets of individuals with different severity of gingival inflammation in response to a similar rate of plaque accumulation (i.e. HR and LR subjects) (Trombelli et al. 2004a). This identification was based on standardization of the volume of GCF, assessed at day 21 of the experimentally induced gingival inflammation, on the level of exposure to supragingival plaque (Trombelli et al. 2004a). GCF was selected because of its strong association to the aetiologic agent (plaque levels) and because it is the most objective and reliable indicator of the inflammatory status of the gingiva (evidence reviewed in Trombelli et al. 2004a, b). In the present study, no differences were found between HR and LR subjects in terms of either the current level of stress or personality traits indicative of stress susceptibility/resistance. Therefore, differences in susceptibility to plaque-induced inflammation, as detected in LR and HR subjects, cannot be ascribed to any of the above psychological parameters.

In summary, within the limitations of the present study, personality traits, social support and current levels of stress have limited impact on clinical parameters of plaque accumulation and gingival inflammation during experimental gingivitis in young adults. These psychological parameters do not account for differences in individual susceptibility to plaque-induced gingivitis as detected in LR and HR subjects.

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Clinical Relevance

Based on the increasing evidence supporting an association between stress and periodontal disease, we designed an experimental gingivitis study to characterize the subject-based clinical behaviour of the gingiva with respect to psychosocial variables related to the coping behaviour and the perceived current level of stress. After 21 days of plaque accumulation, none of the clinical parameters of gingival inflammation were correlated with the current level of stress or the stress susceptibility/resistance of the individual. It appears that personality traits, social support and current levels of stress have a limited impact on clinical parameters of experimentally induced gingival inflammation.