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Dental biofilm, gingivitis and interleukin-1 adjacent to approximal sites of a bonded ceramic

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Abstract

Aim: The aim of this study was to investigate in vivo the influence of aged, resin-bonded, ceramic restorations on approximal dental biofilm formation and gingival inflammatory response, associated with and without customary oral hygiene.

Material and Methods: In a cross-sectional and in a 10-day experimental gingivitis study, Quigley–Hein plaque index, gingival index (GI), crevicular fluid and its levels of interleukin (IL)-1α, -1β and receptor antagonist were measured at appoximal surfaces of leucite-reinforced bonded ceramic coverages, resin composite restorations and enamel and compared intra-individually in 17 participants.

Results: No differences were found between the ceramic, composite and enamel regarding plaque index, GI, levels of IL-1α, -1β and the receptor antagonist. Throughout, higher crevicular fluid amounts were observed at ceramic sites compared with the enamel ($p < 0.05$). In the experimental gingivitis, plaque index, GI, crevicular fluid and its IL-1α levels increased significantly.

Conclusion: The need for optimal oral hygiene and professional preventive oral health care does not seem to be reduced with regard to approximal surfaces of aged, resin-bonded, leucite-reinforced ceramic restorations in comparison with those of a hybrid, resin composite and enamel.

Key words: biofilm; ceramics; composites; gingivitis; gingival crevicular fluid; IL-1; plaque

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The dental biofilm or dental plaque is composed of various bacterial species embedded in a matrix of bacterial products and host-derived factors (Marsh 2004). This biofilm stimulates the gingival inflammatory response. The growth of the biofilm result in an enhancement of the gingival crevicular fluid (GCF; Löe & Holm-Pedersen 1965) and subsequent clinical signs of gingivitis (Löe et al. 1965). One of the major mediators of the inflammatory response is interleukin-1 (IL-1). IL-1α and -1β are two structurally related proteins. The IL-1 activity is controlled by binding of the IL-1 receptor antagonist (IL-1ra) to the IL-1 receptor (Dinarello & Wolff 1993). Experimental gingivitis models with ceased oral hygiene have demonstrated enhanced amounts of IL-1β in GCF, before the onset of the clinical signs of gingivitis (Zhang et al. 2002).

The demand for dental materials with an esthetic appearance has increased. Currently, resin composites are bonded micro-mechanically in a wide range of cavities conditioned by a tissue-preserving technique. However, a major disadvantage is their polymerization shrinkage (Davidson et al. 1984), and concern exists regarding their toxicity (Bouillaguet et al. 2002). Dental ceramics are considered to be chemically stable with a high biocompatibility (Schuster et al. 1996). The specific properties of ceramics are hardness, porosity and brittleness. Early-fired ceramics had a low fracture resistance, which was improved in leucite-reinforced feldspathic porcelains. Ceramic onlays/crowns of a leucite-reinforced ceramic, luted with a dentin bonding adhesive system and a resin composite material, showed satisfactory clinical

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performance (van Dijken et al. 2001). It has been reported that dental ceramics show less biofilm accumulation (Savitt et al. 1987, Jensen et al. 1990). However, dental restorations on approximal tooth surfaces can be a risk factor for localized periodontal attachment loss (Broadbent et al. 2006). The effects of bonded dental ceramic restorations on the adjacent GCF-levels and its content of IL-1 have not been described previously in the literature.

The aim of the present study was to investigate in vivo the influence of aged, resin-bonded, leucite-reinforced ceramic restorations on appoximal dental biofilm formation and gingival inflammatory response, associated with and without customary oral hygiene.

The hypothesis was that there is less biofilm accumulation and concomitantly less gingival inflammatory response at approximal sites of the ceramic restorations compared with enamel.

Material and Methods

Individuals at their yearly recalls of a clinical follow-up of bonded ceramic onlays/crowns (van Dijken et al. 2001) were requested to participate in the present study. Ther inclusion criteria were a leucite-reinforced enamel/dentin-bonded ceramic coverage (IPS Empress, Ivoclar, Schaan, Liechtenstein) with an approximal surface extended to the gingiva, and a non-restored approximal enamel surface in the same dental arch. No caries lesion or adjacent approximal restoration of restorative material other than the tested material was allowed. Surfaces with an adjacent probing pocket depth exceeding 3 mm were not included. The exclusion criteria were use of antibiotics, anti-inflammatory drugs and/or oral anti-microbial agents within the preceding 3 months. The individuals gave informed consent to participate. The ethics committee of the Umeå University approved the study design, which conforms to the guidelines of the World Medical Association Declaration of Helsinki.

In 17 participants (11 females and six males, mean age 54.0 years, range 43–85), intra-individually comparisons were made between one approximal surface of the bonded ceramic restoration (4–7 years old) and one of enamel. In a subgroup, including 11 of these participants, an additional approximal surface of a Class II mid-filled, hybrid resin composite restoration (>1-year old) was also compared. Two sets of the three surfaces, ceramic, composite and enamel, were compared in one individual. In all, 18 sets of ceramic and enamel were compared, 12 of which also included composite. The intra-oral distribution of the included surfaces is shown in Table 1. The mean number of teeth was 28 (14–31). The presence of plaque (Lenox & Kopczyck 1973) varied between 20% and 50% of all cervical tooth surfaces in 15 participants, and was below 20% of all cervical tooth surfaces in two participants. The gingival bleeding on marginal probing at buccal, mesial, distal and lingual sites (Ainamo & Bay 1975) varied between 5% and 15% of all cervical sites in four participants and was below 5% in 13 participants. Four participants were smokers.

Table 1. Intra-oral distribution between different tooth types of the included, approximal leucite-reinforced bonded ceramic, resin composite and enamel surfaces

	Ceramic	Composite	Enamel
Molars	15 (10)	2	4 (2)
Pre-molars	3 (2)	8	11 (7)
Canines	0	1	3
Incisors	0	1	0
Number of surfaces	18 (12)	12	18 (12)

Eighteen ceramic and enamel surfaces were compared, 12 of which were also compared with a resin composite surface.

Study design

The main outlines of the method have been described in detail (Konradsson & van Dijken 2002, 2005) and will be depicted here more briefly. The effects of the ceramic restorations on approximal biofilm formation and of the adjacent gingival inflammatory response were investigated. Influences associated with customary oral hygiene, i.e. the effect of oral hygiene regimes on the biofilm removal from the surfaces, were evaluated at a single point in a cross-sectional study. Then, influences associated with abstained oral hygiene were evaluated in an experimental gingivitis study for 10 days with neglected oral hygiene, in order to initiate gingivitis. After measurement, the opposite teeth in the dental arch were polished, except on day 0 (baseline). Before the experimental gingivitis study, gingival health had to be established. If needed, a period of intense oral hygiene was performed until plaque and gingivitis indices approached zero.

Clinical parameters

In the cross-sectional study and on days 0 (baseline), 3, 7 and 10 of the experimental gingivitis study, at one approximal site of each of the ceramic, composite and enamel surfaces, the parameters were measured as follows: sampling and determination of the GCF volumes. Gingival index (GI; Löe 1967) and a modified Quigley–Hein plaque index (QHI; Turesky et al. 1970) where healthy gingival (GI = 0) and gingival redness and swelling (GI = 1) were estimated initially and gingival bleeding (GI = 2) was recorded after measuring the plaque index.

GCF and IL-1

The sites to be sampled were isolated with cotton rolls and saliva was removed by carefully spraying water. Each site to be collected was gently dried with an air syringe. A paper strip (Periopaper®; Proflow Inc., Amityville, NY, USA) was placed in the orifice of the gingival crevice for 10 s. The fluid volume was determined with a calibrated Periotron 8000 (Proflow, Amityville, NY, USA). The absorbing part of the strip was cutoff and transferred to a coded plastic tube. The sample was eluted twice by adding 50 μl 0.9% sodium chloride and was centrifuged at 2060 $\times$ *g* and 5°C for 20 min. Then, 100 μl 0.9% sodium chloride was added and the supernatant was stored frozen at -80°C (Rasmussen et al. 2000). The concentrations of IL-1α, -1β and IL-1ra were quantified with enzyme-linked immunosorbent assays (ELISAs, Quantikine, R&D Systems Europe, Abingdon, Oxon, UK) in accordance with the instructions provided by the manufacturer. The detection levels of the assays of IL-1α and -1β are 1 pg/ml and of the assay of IL-1ra 14 pg/ml. To calculate the levels of IL-1α, -1β and -1ra in the GCF, the dilution factors and the collected volume were taken into account.

Statistics

The data were processed in SPSS (Statistical Package for Social Sciences, version 13.0). A paired *t*-test was used to analyse the differences of the mean GCF volumes. The IL-1 concentrations were analysed by the Kolmogorov–Smirnov goodness-of-fit test for normality. The IL-1 data were not normally distributed. Wilcoxon's signed-rank test and Exact test (Monte Carlo) were used for intra-individual comparisons. The *p*-value was set at <0.05 to indicate statistical significance.

Results

Among the 17 participants included in the initial cross-sectional study, two participants (one female and one male) refused to participate in the experimental gingivitis study, which comprised the remaining 15 participants. The QHI, GI and the GCF volumes in the cross-sectional study, and on days 0 and on 10 of the experimental gingivitis study are shown in Table 2. Each of these parameters increased significantly between days 0 and 10 of the experimental gingivitis study at the sites of ceramic restorations ($p<0.001$), composite restorations ($p<0.009$) and enamel control surfaces ($p<0.002$). No significant differences were found between the surfaces regarding plaque and gingival indices. Sites adjacent to ceramic surfaces showed a significantly higher amount of GCF compared with enamel in both the cross-sectional study ($p=0.017$), and days 0 ($p=0.019$) and 10 of the experimental gingivitis study ($p=0.016$). The GCF levels of IL-1α, -1β and -1ra of the compared ceramic and enamel sites in the cross-sectional study, and on days 0 and 10 of the experimental gingivitis, are shown in Fig. 1a–c, respectively. A few extreme outlier values were observed. An increase of IL-1α at ceramic sites ($p=0.024$), composite sites ($p=0.034$) and enamel sites ($p=0.028$) was shown from day 0 to day 10. The IL-1 levels during the whole study period of the sets including resin composites are described in Table 3. An increase of IL-1ra at composite sites ($p=0.027$) between day 0 and day 10 was showed. No significant differences in the levels of IL-1α, -1β and -1ra were found between the ceramic, resin composite and enamel surfaces.

Table 2. The means of modified Quigley-Hein plaque index (QHI), gingival index according to Löe (GI), and gingival crevicular fluid samples (GCF; μl/10 s; standard deviation) adjacent to approximal surfaces of a leucite-reinforced ceramic, resin composite and enamel in the cross sectional study (CSS) and on days 0 and 10 of the experimental gingivitis study

	Ceramic	Composite	Enamel	*n*
CSS				
QHI	0.94	–	1.06	18
	1.1	0.83	1.4	12
GI	0.61	–	0.28	18
	0.75	0.5	0.42	12
GCF	0.49 (0.10)*	–	0.22 (0.23)*	18
	0.49 (0.41)	0.32 (0.36)	0.24 (0.23)	12
Day 0				
QHI	0.33	–	0.43	16
	0.1	0.1	0.1	12
GI	0	–	0	16
	0	0	0	12
GCF	0.30 (0.33)*	–	0.13 (0.19)*	16
	0.23 (0.25)	0.18 (0.26)	0.19 (0.16)	12
Day 10				
QHI	2.62	–	3.06	16
	2.67	3.33	3.17	12
GI	1.00	–	0.93	16
	1.00	1.08	0.83	12
GCF	0.82 (0.41)*	–	0.49 (0.38)*	16
	0.78 (0.34)	0.51 (0.54)	0.44 (0.37)	12

*$p<0.05$.
n = number of sets of the intra-individually compared surfaces of ceramic and enamel ($n=18$ in CSS, $n=16$ on day 0 and 10), also including the evaluated composite subgroup ($n=12$).

Discussion

With customary oral hygiene, the biofilm extension on the aged, bonded ceramic surfaces was comparable with that on enamel surfaces, and also with abstained oral hygiene, the amount of biofilm was not influenced by the ceramic material. This supports the statement that a non-material-dependent developed biofilm exists in the oral cavity (Hannig 1999).

Ceramic materials have been reported to be biocompatible and with a lower bacterial adhesion compared with enamel. Less amount of biofilm was accumulated in vivo, on cast glass–ceramic crowns than on contra-lateral teeth (Savitt et al. 1987, Jensen et al. 1990). Some clinical follow-ups of bonded dental ceramics, compared with contra-lateral teeth/surfaces, have not indicated higher biofilm formation on and gingival bleeding adjacent to restorations of a glass–ceramic (Sjögren et al. 1999), of approximal surfaces of glass–ceramic inlays (Stenberg & Matsson 1993) and of a leucite-reinforced ceramic (Tidehag & Gunne 1995). The last mentioned ceramic was also evaluated in the present study. A clinically healthy gingiva did not change for the worse at 4-year-old glass–ceramic crowns (Kelsey et al. 1995). In comparison with the plaque indices used in those evaluations, the QHI utilized in the present study is more detailed in depicting the biofilm extension on the surface. The index can therefore contribute to revealing small variations between the surfaces. However, this index is still limited to the visible part of the approximal tooth surfaces.

In the experimental gingivitis study, the biofilm, GCF amounts and clinical signs of gingivitis increased significantly irrespective of the underlying surface material. The concomitant gingival response to an increase of accumulated and matured biofilm is well known (Löe & Holm-Pedersen 1965, Löe et al. 1965, Theilade et al. 1966). At the end of the experimental gingivitis, most of the sites showed no bleeding. This is not surprising, as the experimental gingivitis period did not exceed 10 days. The length of the experimental gingivitis period was based on previous studies (van Dijken et al. 1987, van Dijken & Sjöström 1991, 1998, Konradsson & van Dijken 2002, 2005). There are differences in the gingival response to dental biofilm between low and high responders (Trombelli et al. 2004). After neglected oral hygiene, manifest gingivitis can

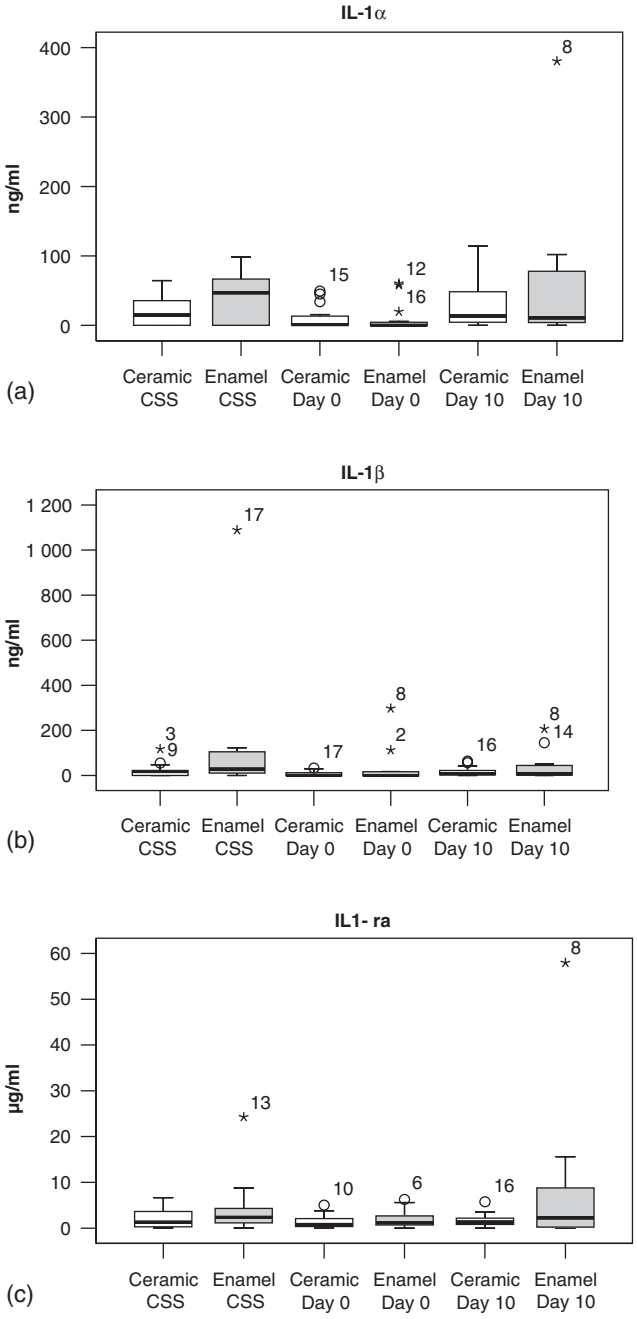

Fig. 1. Boxplots of the concentrations of (a) interleukin (IL)-1α (ng/ml; $n = 16$), (b) IL-1β (ng/ml, $n = 16$) and (c) IL-1 receptor antagonist (ra) (μl/ml, $n = 15$) in samples of gingival crevicular fluid (GCF) adjacent to approximal surfaces of resin-bonded, leucite-reinforced ceramic restorations and enamel in the cross-sectional study and on days 0 and 10 of the experimental gingivitis study. n is the number of intra-individually compared surfaces, possibly to be continuously followed throughout the whole study period. The median, 25th and 75th percentiles are showed in boxplots. Whiskers include the minimum or maximum, except outliers (o) and extreme outliers (*), which are marked with their participant codes.

occur within 2 weeks, by 21 days, or not occur within a 3-week period, as reported by Wiedemann et al. 1979 (Tatakis & Trombelli 2004). The initial low percentage of gingival bleeding as compared with the percentage of plaque might be an indication of participants with a low response to plaque-induced gingivitis. Moreover, the opposite arch was polished after measuring to support the compliance. This might have decreased the rate of dental biofilm formation with a subsequent delay of the development of gingivitis (Quirynen & Steenberghe 1989). The benefit of a further length of the experimental gingivitis can be discussed; however, the primary aim was to compare the materials under the prevailing conditions at initiation of gingivitis, not to evaluate the development of gingivitis per se.

In the present study, higher amounts of the GCF were collected at the sites of ceramic compared with enamel despite there being no differences in the presence of an apparent biofilm. Neither were there any differences observed in the levels of IL-1α, -1β and -1ra between the ceramic and enamel sites. It can be assumed that the higher GCF amounts sampled were due to stimulation by biofilm on the non-visible part of the approximal surface. Most of the bonded, extensive ceramic restorations were placed in molars, while the enamel included surfaces were located on pre-molars. In comparison with the pre-molars, molars have an extended approximal contact area and ''col'', where some biofilm can be hidden, inaccessible to oral hygiene (Takai 1980). Another reason for possible biofilm accumulation can be that, at the time of luting and polishing of ceramic crowns, iatrogenic defects can be established on the approximal surfaces (Pallesen & van Dijken 2000). Over time, wear may also cause marginal defects (Kramer & Frankenberger 2000) and a deteriorated ceramic surface (Kramer & Frankenberger 2005). This in turn results in an increased surface roughness, which can facilitate the biofilm formation (Quirynen et al. 1990) or influence the biofilm removal. The biofilm formation can also be affected by the surface energy to the material (Quirynen et al. 1990, Kawai et al. 2000). Less dental biofilm formation on glazed ceramic surfaces than on non-glazed ceramic surfaces has been reported (Castellani et al. 1996). In the present study, aged ceramic and resin composite surfaces were studied as representative for most of the reconstructions in function. Probably, those aged surfaces have an increased surface roughness compared with their initial high gloss at baseline. However, in the clinical follow-up, including the current restorations, the tendency towards rougher surfaces and detoriated marginal adaptation was not shown to be significant on the ceramic coverages (van Dijken et al. 2001). The ceramic restorations evaluated were exposed for

Table 3. The median concentration (range) of interleukin (IL) -1α (ng/ml), IL-1β (ng/ml) and IL-1 receptor antagonist (ra; μg/ml) in samples of gingival crevicular fluid adjacent to ceramic, resin composite and enamel in the cross-sectional study (CSS) and on days 0, 3, 7 and 10 of the experimental gingivitis study

	Ceramic	*n*	Composite	*n*	Enamel	*n*
CSS						
IL-1α	15.6 (0–64.6)	11	2.7 (0–273.0)	11	57.8 (0–98.3)	11
IL-1β	9.5 (0–55.4)	9	2.6 (0–154.9)	9	27.8 (0–123.0)	9
IL-1 ra	1.3 (0–6.7)	11	1.4 (0–14.3)	11	2.4 (0–24.3)	11
Day 0						
IL-1α	2.5 (0–49.3)	12	0 (0–131.6)	12	0 (0–60.5)	12
IL-1β	0 (0–29.5)	9	4.9 (0–62.5)	9	0 (0–17.5)	9
IL-1 ra	1.3 (0–5.0)	10	0.7 (0–3.1)	10	1.0 (0–6.3)	10
Day 3						
IL-1α	11.0 (0–102.0)	12	2.0 (0–154.8)	12	0 (0–80.7)	12
IL-1β	9.8 (0–56.5)	9	0 (0–89.1)	9	10.7 (0–399.3)	9
IL-1 ra	0.7 (0.1–15.3)	10	0.8 (0.2–11.1)	10	1.0 (0–16.1)	10
Day 7						
IL-1α	17.0 (0–118.2)	12	12.9 (0–75.4)	12	9.4 (0–105.3)	12
IL-1β	25.4 (0–56.7)	9	8.5 (0–175.7)	9	18.9 (0–101.4)	9
IL-1 ra	1.4 (0.1–8.3)	10	2.0 (0.1-7.0)	10	1.4 (0.1–9.8)	10
Day 10						
IL-1α	19.4 (1.0–113.9)	12	17.3 (0–109.0)	12	10.8 (0.9–102.0)	12
IL-1β	8.8 (0–63.7)	9	15.6 (5.4–89.6)	9	2.5 (0–145.2)	9
IL-1 ra	1.8 (0.1–5.7)	10	3.4 (0.5–7.1)	10	5.4 (0–15.6)	10

The digit zero (0) represents all values below the detection levels (IL-1α, IL-1β, <1 pg/ml; IL-1ra <14 pg/ml) of the assays.

the oral environment for several years, eliminating the possible initial effects of unbound monomers of the luting cement on bacterial growth (Ferracane 1994).

When comparing ceramics with resin composites in the present study, no obvious differences in biofilm formation or signs of gingival inflammation including the IL-1 levels were demonstrated. Nor were there any significant differences observed between the composite and the enamel. The number of available composite surfaces to be compared was, however, limited, which complicated any findings of small differences.

In vitro, lower bacterial adhesion was observed on ceramic surfaces than on the resin composite. The biofilm adhesion was reduced on the composite, but not on ceramic discs after polishing (Kawai & Urano 2001). After 3 days of biofilm formation in an intra-individual approach, less biofilm was accumulated on the ceramic than on the resin composite and enamel (Hahn et al. 1993). Comparisons were also made within each participant in the present study to enable elimination of inter-individual variations with regard to biofilm formation (Simonsson et al. 1987) to response on plaque-induced gingivitis (Trombelli et al. 2004), and to underlying inflammatory mechanisms (Scapoli et al. 2005).

In a previous study of cervical restorations on smooth tooth surfaces, no difficulties in removing deposits from composite surfaces were noticed with customary oral hygiene procedures. However, after 10-day biofilm formation, more biofilm was accumulated on the composite than on the enamel. Differences were not found in the clinical signs of gingival inflammation and GCF (Konradsson & van Dijken 2002). As opposed to that study, the present study evaluated approximal surfaces, which could contribute to the differences in the results.

IL-1α increased over a 7-day period of biofilm formation adjacent to cervical surfaces of resin composite (Konradsson & van Dijken 2005). The increase of IL-1α was also found in the present study of 10-day biofilm formation adjacent to ceramic, composite and enamel surfaces. No significant enhancement of IL-1β was shown.

In a study of Zhang et al. (2002), still, the amounts of IL-1β were increased after 3 days of biofilm accumulation; the concentration was essentially maintained at a constant level. However, Heasman et al. (1993) showed an abrupt increase of the IL-1β concentrations after 1 week of biofilm accumulation. During 28 days of an experimental gingivitis study, the IL-1ra levels could not be associated with gingival inflammation (Waschul et al. 2003), although it was demonstrated by others (Rawlinson et al. 2000, Holmlund et al. 2004).

In conclusion, the results of this study indicate that dental biofilm formation, clinical signs of gingivitis and IL-1 levels did not differ between approximal surfaces of leucite-reinforced, bonded ceramic coverages, Class II restorations of hybrid resin composites and enamel, either with customary or with neglected oral hygiene. Higher levels of GCF were observed adjacent to the ceramic than adjacent to the enamel. Despite the high biocompatibility of dental ceramics considered, the need for optimal oral hygiene and professional preventive oral health care does not seem to be reduced with regard to approximal surfaces of resin-bonded, leucite-reinforced ceramic restorations in comparison with those of a resin composite and an enamel.

References

Ainamo, J. & Bay, I. (1975) Problems and proposals for recording gingivitis and plaque. *International Dental Journal* **25**, 229–235.

Bouillaguet, S., Shaw, L., Gonzalez, L., Wataha, J. C. & Krejci, I. (2002) Long-term cytotoxicity of resin-based dental restorative materials. *Journal of Oral Rehabilitation* **29**, 7–13.

Broadbent, J. M., Williams, K. B., Thomson, W. M. & Williams, S. M. (2006) Dental restorations: a risk factor for periodontal attachment loss? *Journal of Clinical Periodontology* **33**, 803–810.

Castellani, D., Bechelli, C., Tiscione, E., Lo Nostro, A. & Pierloni, M. D. (1996) *In vivo* plaque formation on cast ceramic (Dicor) and conventional ceramic. *International Journal of Prosthodontics* **9**, 459–465.

Davidson, C. L., de Gee, A. J. & Feilzer, A. J. (1984) The competition between the composite-dentin bond strength and the polymerisation contraction stress. *Journal of Dental Research* **63**, 1396–1399.

van Dijken, J. W. V., Hasselrot, L., Örmin, A. & Olofsson, A. L. (2001) Restorations with extensive dentin/enamel-bonded ceramic coverage. A 5-year follow-up. *European Journal of Oral Sciences* **109**, 222–229.

van Dijken, J. W. V. & Sjöström, S. (1991) The effect of glass ionomer cement and composite resin fillings on marginal gingiva. *Journal of Clinical Periodontology* **18**, 200–203.

van Dijken, J. W. V. & Sjöström, S. (1998) Development of gingivitis around aged restorations of composite. *Clinical Oral Investigations* **2**, 180–183.

van Dijken, J. W. V., Sjöström, S. & Wing, K. (1987) The effect of different types of composite resin fillings on marginal gingiva. *Journal of Clinical Periodontology* **14**, 185–189.

Dinarello, C. A. & Wolff, S. M. (1993) The role of interleukin-1 in disease. *The New England Journal of Medicine* **328**, 106–113.

Ferracane, J. L. (1994) Elution of leachable components from composites. *Journal of Oral Rehabilitation* **21**, 441–452.

Hahn, R., Weiger, R., Netuschil, L. & Brüch, M. (1993) Microbial accumulation and vitality on different restorative materials. *Dental Materials* **9**, 312–316.

Hannig, M. (1999) Transmission electron microscopy study or early plaque formation on dental materials *in vivo*. *European Journal of Oral Sciences* **107**, 55–64.

Heasman, P. A., Collins, J. G. & Offenbacher, S. (1993) Changes in crevicular fluid levels of interleukin-1β, leukotriene B_4, prostaglandin E_2, thromboxane B_2 and tumor necrosis factor α in experimental gingivitis in humans. *Journal of Periodontal Research* **28**, 241–247.

Holmlund, A., Hänström, L. & Lerner, U. (2004) Bone resorbing activity and cytokine levels in gingival crevicular fluid before and after treatment of periodontal disease. *Journal of Clinical Periodontology* **31**, 475–482.

Jensen, O. E., Schultes, A. M., Handelman, S. L. & Proskin, H. M. (1990) Plaque retention on Dicor crowns and gingival health evaluated over a 4-year period. *The International Journal of Periodontics & Restorative Dentistry* **10**, 454–463.

Kawai, K. & Urano, M. (2001) Adherence of plaque components to different restorative materials. *Operative Dentistry* **26**, 396–400.

Kawai, K., Urano, M. & Ebisu, S. (2000) Effect of surface roughness of porcelain on adhesion of bacteria and their synthesizing glucans. *Journal of Prosthetic Dentistry* **83**, 664–667.

Kelsey, W. P., Cavel, T., Blankenau, R. J., Barkmeier, W. W., Wilwerding, T. M. & Latta, M. A. (1995) 4-year clinical study of castable ceramic crowns. *American Journal of Dentistry* **8**, 259–262.

Konradsson, K. & van Dijken, J. W. V. (2002) Effect of a novel ceramic filling material on plaque formation and marginal gingiva. *Acta Odontologica Scandinavica* **60**, 370–374.

Konradsson, K. & van Dijken, J. W. V. (2005) Interleukin-1 levels in gingival crevicular fluid adjacent to restorations of calcium aluminate cement and resin composite. *Journal of Clinical Periodontology* **32**, 462–466.

Kramer, N. & Frankenberger, R. (2000) Leucite-reinforced glass ceramic inlays after six years: wear of luting composites. *Operative Dentistry* **25**, 466–472.

Kramer, N. & Frankenberger, R. (2005) Clinical performance of bonded leucite-reinforced glass ceramic inlays and onlays after eight years. *Dental Materials* **21**, 262–271.

Lenox, J. A. & Kopczyck, R. A. (1973) A clinical system for scoring a patient's oral hygiene performance. *Journal of American Dental Association* **86**, 849–852.

Löe, H. (1967) The gingival index, the plaque index and the retention index systems. *Journal of Periodontology* **38**, 610–616.

Löe, H. & Holm-Pedersen, P. (1965) Absence and presence of fluid from normal and inflamed gingivae. *Periodontics* **3**, 171–177.

Löe, H., Theilade, E. & Jensen, S. B. (1965) Experimental gingivitis in man. *Journal of Periodontology* **36**, 177–187.

Marsh, P. D. (2004) Dental plaque as a microbial biofilm. *Caries Research* **38**, 204–211.

Pallesen, U. & van Dijken, J. W. V. (2000) An 8-year evaluation of sintered ceramic and glass ceramic inlays processed by the Cerec CAD/CAM system. *European Journal of Oral Sciences* **108**, 239–246.

Quirynen, M., Marechal, M., Busscher, H. J., Weerkamp, A. H., Darius, P. L. & van Steenberghe, D. (1990) The influence of surface free energy and surface roughness on early plaque formation. An in vivo study in man. *Journal of Periodontology* **17**, 138–144.

Quirynen, M. & van Steenberghe, D. (1989) Is early plaque growth rate constant with time? *Journal of Clinical Periodontology* **16**, 278–283.

Rasmussen, L., Hänström, L. & Lerner, U. H. (2000) Characterization of bone resorbing activity in gingival crevicular fluid from patients with periodontitis. *Journal of Clinical Periodontology* **27**, 41–52.

Rawlinson, A., Dalati, M. H. N., Rahman, S., Walsh, T. F. & Fairclough, A. L. (2000) Interleukin-1 and IL-1 receptor antagonist in gingival crevicular fluid. *Journal of Clinical Periodontology* **27**, 738–743.

Savitt, E. D., Malament, K. A., Socransky, S. S., Melcer, A. J. & Backman, K. J. (1987) Effects on colonization of oral microbiota by a cast glass–ceramic restoration. *The International Journal of Periodontics & Restorative Dentistry* **2**, 23–35.

Scapoli, C., Tatakis, D. N., Mamolini, E. & Trombelli, L. (2005) Modulation of clinical expression of plaque-induced gingivitis interleukin-1 gene cluster polymorphisms. *Journal of Periodontology* **76**, 49–56.

Schuster, G. S., Lefebre, C. A., Wataha, J. C. & White, S. N. (1996) Biocompatibility of posterior restorative materials. *Journal of the California Dental Association* **24**, 17–31, review.

Simonsson, T., Ronstrom, A., Rundegren, J. & Birkhed, D. (1987) Rate of plaque formation–some clinical and biochemical characteristics of ''heavy'' and ''light'' plaque formers. *Scandinavian Journal of Dental Research* **95**, 97–103.

Sjögren, G., Lantto, R. & Tillberg, A. (1999) Clinical evaluation of all-ceramic crowns (Dicor) in general practice. *The Journal of Prosthetic Dentistry* **81**, 277–284.

Stenberg, R. & Matsson, L. (1993) Clinical evaluation of glass ceramic inlays (Dicor). *Acta Odontologica Scandinavica* **51**, 91–97.

Takai, H. H. (1980) The interdental space. *Dental Clinics of North America* **24**, 169–176.

Tatakis, D. N. & Trombelli, L. (2004) Modulation of clinical expression of plaque-induced gingivitis. I. Background review and rationale. *Journal of Clinical Periodontology* **31**, 229–238.

Theilade, E., Wright, H., Jensen, S. B. & Löe, H. (1966) Experimental gingivitis in man. II. *Journal of Periodontal Research* **1**, 1–13.

Tidehag, P. & Gunne, J. (1995) A 2-year clinical follow-up study of IPS Empress ceramic inlays. *The International Journal of Prosthodontics* **8**, 456–460.

Trombelli, L., Tatakis, D. N., Scapoli, C., Bottega, S., Orlandini, E. & Tosi, M. (2004) Modulation of clinical expression of plaque-induced gingivitis. II. Identification of ''high-responder'' and ''low-responder'' subjects. *Journal of Clinical Periodontology* **31**, 239–252.

Turesky, S., Gilmore, N. & Glickman, I. (1970) Reduced plaque formation by the chloromethyl analogue of vitamin C. *Annals of Periodontology* **41**, 41–43.

Waschul, B., Herforth, A., Stiller-Wrinkler, R., Idel, H., Granrath, N. & Deinzer, R. (2003) Effects of plaque, psychological stress and gender on crevicular IL-1 β and IL-ra secretion. *Journal of Clinical Periodontology* **30**, 238–248.

Wiedemann, W., Lahrsow, J. & Naujoks, R. (1979) The effect of periodontal resistance on experimental gingivitis. *Deutsche Zahnarztliche Zeitschrift* **34**, 6–9.

Zhang, J., Kashket, S. & Lingström, P. (2002) Evidence for the early onset of gingival inflammation following short-term plaque accumulation. *Journal of Clinical Periodontology* **29**, 1082–1085.

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Clinical Relevance

Scientific rationale for the study: Dental ceramics are considered to be biocompatible and not to favour plaque accumulation. However, they are often luted with a resin composite and approximal restorations can be a local, periodontal risk factor.

Principal findings: Dental biofilm formation, clinical signs of gingivitis and IL-1 levels at sites of a resin-bonded ceramic restorations were similar to those of resin composite and an enamel.

Practical implications: Lack of effects on biofilm formation and gingival response indicating that the choice of ceramics as a restorative material should rely on other factors. Optimal oral hygiene to maintain the adjacent gingival health is required on approximal surfaces of bonded leucite-reinforced ceramic restorations.