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Gingival crevicular fluid laminin-5 γ2-chain levels in periodontal disease

Gülnur Emingil[1], Heidi Kuula[2], Emma Pirilä[3], Gül Atilla[1] and Timo Sorsa[2]

[1]Department of Periodontology, Ege University, School of Dentistry, İzmır, Turkey; [2]Department of Oral and Maxillofacial Diseases, Institute of Dentistry, University of Helsinki, Helsinki University Central Hospital HUCH Helsinki, Finland; [3]Institute of Dentistry, University of Oulu, Oulu, Finland.

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Abstract

Aim: Our study aimed to examine the molecular forms and gingival crevicular fluid (GCF) levels of laminin-5 γ2-chain in patients with different periodontal disease, and compare the effects of *P.gingivalis* trypsin-like proteinase on intact laminin-5 γ2-chain species.

Methods: Eighteen patients with generalized aggressive periodontitis (G-AgP), 29 patients with chronic periodontitis (CP), 20 with gingivitis and 20 periodontally healthy subjects were included. Probing depth, clinical attachment loss, presence of bleeding on probing and plaque were recorded. Molecular forms and GCF laminin-5 γ2-chain levels and the effects of *P. gingivalis* trypsin-like proteinase on intact laminin-5 γ2-chain were analysed by computer-quantitated Western immunoblotting.

Results: Laminin-5 γ2-chain 40 and 70 kDa fragments could be detected in all groups, in varying levels.The CP group had elevated GCF laminin-5 γ2-chain fragment levels compared with the gingivitis and healthy groups ($p < 0.008$).The G-AgP group had GCF laminin-5 γ2-chain fragment levels similar to the gingivitis and healthy groups ($p > 0.008$). GCF laminin-5 γ2-chain fragments differed clearly from the multiple lower molecular size fragments of *P.gingivalis* trypsin-laminin-5 γ2-chain proteinases.

Conclusion: Increased GCF laminin-5 γ2-chain fragments in periodontitis sites with deep periodontal pocket suggest that these cleaved 40 and 70 kDa fragments could reflect the extent of the inflammatory reaction in CP.

Key words: gingival crevicular fluid/western immunoblotting; laminin-5 γ2-chain fragments; periodontal disease/pathogenesis

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The pathogenesis of periodontal disease involves the participation of numerous inflammatory and immune mediators released from the resident and recruiting inflammatory cells as well as from bacteria that contribute to periodontal tissue destruction by degrading extracellular matrix and basement membrane components (Birkedal-Hansen 1993, Madianos et al. 2005, Sanz & Quirynen 2005). Destructive mechanisms leading to the conversion of the junctional epithelium into an infected pocket epithelium play a crucial role in the progression of periodontal disease, but the precise mechanisms in this destructive process have not yet been established (Pöllänen et al. 2003).

Cell migration is an essential part of several physiological and pathogenic processes, including several chronic inflammatory conditions, tumour invasion and wound healing (Terranova & Lyall 1986, O'Toole 2001). Laminins, large molecular size glycoproteins and major components of the basement membrane have multiple structural and functional roles in cell differentiation, proliferation, adhesion and migration (Colognato & Yurchenco 2000, O'Toole 2001). It has been suggested that they are involved in the apical migration of epithelial cells during the development of periodontal pockets (Pöllänen et al. 2003). Intact laminin-5 is a well-known epithelial cell-derived adhesive protein localized to the anchoring filaments within the lamina lucida space of the basal membrane zone of the junctional and gingival epithelium (Hormia et al. 1998, Gürses et al. 1999). It is secreted by epithelial cells and composed of three polypeptide chains, α3, β3 and γ2, of which the γ2-chain is specific for ln-5 (Colognato & Yurchenco 2000). Laminin-5 can stimulate epithelial cell migration after having been cleaved by proteolytic enzymes, whereas intact laminin-5 serves to anchor epithelial cells on the basement membrane (Giannelli et al. 1997, O'Toole et al. 1997, Gagnoux-Palacios et al. 2001). Certain matrix metalloproteinases (MMPs) can process the laminin-5 γ2-chain, producing 40 and 70 kDa fragments (Heck et al. 1990, Mäkelä et al. 1999, Pirilä et al. 2001, 2003). The cleaved laminin-5 γ2-chain

can mediate inflammatory reactions by regulating cell adhesion, migration and proliferation of fibroblasts and epithelial cells, and the fragments also act as chemoattractants for leucocytes (Terranova & Lyall 1986, Giannelli et al. 1997). The presence of cleaved laminin-5 γ2-chain fragments in tissues and body fluids has been suggested to be important in active, physiological and pathological tissue remodelling and in epithelial induction (O'Toole et al. 1997, Gagnoux-Palacios et al. 2001, Kivelä-Rajamäki et al. 2003).

It was recently demonstrated that the presence of the elevated laminin-5 γ2-chain fragments in diseased peri-implant sulcular fluid could reflect basement membrane disruption caused by active inflammation (Kivelä-Rajamäki et al. 2003). Pirilä et al. (2001) have shown that human laminin-5 γ2-chain was co-localized with MMPs within the basement membrane area in the human gingiva at the sites of periodontal inflammation. Figueredo & Gustafsson (2000) examined laminins in gingival crevicular fluid (GCF) of patients with different periodontal disease and found elevated total laminin levels in GCF in periodontitis patients. However, in this study, laminin molecular forms were not identified. The aim of the present study was to investigate the presence of molecular forms, fragmentation and levels of laminin-5 γ2-chain in GCF samples from patients with different periodontal disease categories and to test whether GCF laminin-5 γ2-chain levels are correlated with clinical parameters. For comparison, we studied the effects of *Porphyromonas gingivalis* trypsin-proteinase (Sorsa et al. 1987) on intact laminin-5 γ2-chain.

Materials and Methods

Study population

A total of 87 subjects were included in this study. All consecutive subjects were recruited from the Department of Periodontology, School of Dentistry, Ege University, İzmir. The purpose of the study was completely explained to each subject before they entered the study, and informed consent was obtained from each subject. Complete medical and dental histories were taken from all subjects. All the patients were non-smokers, and had at least 20 teeth in the mouth. None of the subjects had a history of systemic disease or had received antibiotics or other medicines or periodontal treatment within the past 4 months. Patients with severe medical disorders, including diabetes mellitus and immunological disorders, and pregnant females were excluded from the study. The selection of the patients was made according to the clinical and radiographic criteria proposed by the 1999 International World Workshop for a Classification of Periodontal Disease and Conditions (Armitage 1999).

Generalized aggressive periodontitis group (G-AgP)

The G-AgP group included nine females and nine males, ranging in age from 19 to 36, with a mean age of 30 ± 12.4 years. These patients demonstrated a generalized pattern of severe destruction and clinical attachment loss (CAL) of ≥ 5 mm on eight or more teeth; at least three of these were teeth other than the central incisors or first molars.

Chronic periodontitis group (CP)

The CP group consisted of 13 females and 16 males between the ages of 37 and 63 (mean of 48.5 ± 7.6 years). They had moderate to severe alveolar bone loss and a CAL of ≥ 5 mm and probing depth (PD) of ≥ 6 mm in multiple sites of all four quadrants of the mouth, but with no evidence of rapid progression.

Gingivitis group

The gingivitis group included eight females and 12 males with varying degrees of gingival inflammation, but no signs of attachment loss were observed. These patients ranged in age from 15 to 53 (mean age 32.3 ± 11.3 years).

Healthy group

The healthy group consisted of 10 females and 10 males who exhibited PD <3 mm and no CAL, clinical inflammation, sulcular bleeding and radiographic evidence of bone loss (mean age 36.9 ± 13.9 years; range 22–62 years). These individuals were healthy volunteers from the Department of Periodontology.

Determination of periodontal status

At the screening stage, to determine the clinical periodontal status, all subjects had a clinical periodontal examination including the measurement of PD and CAL by one examiner (G.E). Dichotomous measurement of supragingival plaque accumulation and bleeding on probing were also recorded. All measurements were performed at six sites per tooth for the whole mouth.

Collection of GCF samples

After being selected for the study, subjects were recalled for GCF sampling. In the G-AgP and CP groups, GCF samples were collected from one approximal site of a tooth with ≥ 6 mm probing pocket depth. In the gingivitis group, GCF sampling was performed from one approximal site of a tooth with bleeding and ≥ 2 mm probing pocket depth. In the healthy group, GCF samples were collected from one approximal site of a tooth with ≤ 2 mm probing pocket depth. Before GCF sampling, the supragingival plaque was removed from the interproximal surfaces with a sterile curette; these surfaces were dried gently by an air syringe and were isolated by cotton rolls. GCF was sampled with filter paper. Paper strips were carefully inserted into the crevice until mild resistance was felt and were left there for 30 s (Lamster et al. 1985). Care was taken to avoid mechanical injury. Strips contaminated with blood were discarded (Cimasoni 1983). The absorbed GCF volume of each strip was determined by electronic impedance (Periotron 8000, ProFlow Inc., Amityville, NY, USA) and placed in a sterile eppendorff vial and held at -40°C until analysis. The readings from the Periotron 8000 were converted to an actual volume (μl) by reference into the standard curve.

Analysis of molecular forms and fragments of laminin-5 γ2-chain by Western immunoblotting

Western immunoblotting was used to detect molecular forms and fragments of laminin-5 γ2-chain in GCF utilizing specific polyclonal anti-laminin-5 γ2-chain antibodies (Kivelä-Rajamäki et al. 2003, Pirilä et al. 2003). Ten microlitre samples of GCF strips extracted (for 1 h at 22°C) in 50 μl 50 mM Tris-HCL, 0.2 M NaCl, 1.0 mM $CaCl_2$, pH 7.8, to be analysed for laminin-5 γ2-chain, were mixed with 5 μl of 2% β-mercaptoethanol and Laemmli's buffer and heated for 5 min. at 100°C, followed by protein separation on 8%

Table 1. Clinical parameters of the sampling sites in the study groups (mean ± SD)

	G-AgP	CP	Gingivitis	Healthy
PD (mm)	7.4 ± 2*	7.1 ± 0.8*	2.6 ± 0.5†	1.9 ± 0.8
CAL (mm)	7.5 ± 1.9*	8.2 ± 1.9*	–	–
% of sites with bleeding on probing	87.5 ± 34†	92 ± 28†	100†	0
% of sites with plaque	81.3 ± 40.3†	96.2 ± 19.6†	89.5 ± 31.5†	0
GCF (μl)	0.58 ± 0.26*	0.49 ± 0.16*	0.24 ± 0.07†	0.15 ± 0.06

*Significant difference from gingivitis and healthy groups (Kruskal–Wallis test, $p<0.0001$, Mann–Whitney *U*-test, $p<0.008$).
†Significant difference from healthy group (Kruskal–Wallis test, $p<0.0001$, Mann–Whitney *U*-test, $p<0.008$).
G-AgP, generalized aggressive periodontitis; CP, chronic periodontitis; PD, probing depth; CAL, clinical attachment loss; GCF, gingival crevicular fluid.

SDS-polyacrylamide gels. The protocol of the ECL™-Western blotting analysis system was according to the instructions of the manufacturer.* The Molecular Analyst® BioRad scanning program (image analysis system was version 1.4) was used to quantitate the immunoreactivities and molecular forms of laminin-5 γ2-chain from the Western immunoblots. Intact and cleaved laminin-5 γ2-chains were used as controls and data is expressed as densitometric units (Pirilä et al. 2003, Emingil et al. 2004b). Intact 100 and 140 kDa laminin-5 γ2-chain species were prepared as described by Pirilä et al. (2003). The *P.gingivalis* trypsin-like proteinase was purified as described by Sorsa et al. (1987). Five micrograms intact laminin-5 γ2-chain was incubated with buffer and partially purified 1 μg *P.gingivalis* trypsin-like proteinase for 1 and 5 h at 37°C. Resulting cleavage products were analysed by Western immunoblot (Pirilä et al. 2003) using the same antibody as for GCF analysis. Calculation of the laminin-5 γ2-chain concentration was performed by dividing the amount of laminin-5 γ2-chain (densitometric unit) by the volume of the sample.

Statistical analysis

Statistical analysis was performed using non-parametrical techniques. Comparisons were performed using the Kruskal–Wallis test. When there were significant differences ($p<0.05$), post hoc two-group comparisons were assessed with Bonferroni-corrected Mann–Whitney U tests, and *p*-values <0.008 were considered to be statistically significant. Spearman rank correlation analysis was used to analyse the correlations between GCF laminin-5 γ2-chain levels and clinical parameters, and $p<0.05$ was considered as significant. All data analysis was performed using a statistical package (Abacus Concepts Inc., Berkeley, CA, USA).

*Amersham Pharmacia Biotech, Buckinghamshire, UK.

Results

Clinical findings

The mean clinical data for the sampling areas are shown in Table 1.

PD and CAL

As expected, the mean PD scores of sampling sites in the G-AgP and CP groups were significantly higher than the healthy group ($p<0.008$). G-AgP and CP groups had similar PD scores, and they had higher scores compared with the gingivitis group ($p<0.008$). The mean CAL scores of sampling sites in the G-AgP and CP groups were significantly higher than the gingivitis and healthy groups ($p<0.008$). G-AgP and CP groups had similar CAL scores ($p>0.008$).

Percentage of sites with bleeding on probing and plaque

All patient groups had a significantly higher percentage of sites with bleeding on probing and plaque compared with the healthy group ($p<0.008$). The G-AgP, CP and gingivitis groups had a similar percentage of sites with bleeding and plaque ($p>0.008$).

Biochemical findings

Molecular forms, fragments and levels of laminin-5 γ2-chain in GCF

The distribution of GCF laminin-5 γ2-chain 40 and 70 kDa fragment levels is shown in Fig. 1. Significant differences were found between the groups studied ($p=0.0211$). Among the groups, the CP group had the highest GCF laminin-5 γ2-chain 40 and 70 kDa fragment levels compared with the gingivitis and healthy groups ($p=0.0065$ and 0.0052, respectively). GCF laminin-5 γ2 40 and 70 kDa fragment levels of CP group were similar to those of the G-AgP group ($p=0.0923$). G-AgP, gingivitis and healthy groups had similar levels of GCF laminin-5 γ2 40 and 70 kDa fragments ($p>0.008$) (Fig. 2).

When the data were expressed as concentration, significant differences in GCF laminin-5 γ2-chain concentration were found between the groups studied ($p<0.0006$). Among the groups, the healthy group had the highest GCF laminin-5 γ2-chain 40 and 70 kDa fragment concentration compared with the diseased groups. The healthy group had a significantly higher laminin-5 γ2-chain concentration compared with the G-AgP group ($p=0.0001$) (Fig. 3).

The correlation between laminin-5 γ2-chain levels and clinical parameters is presented in Table 2. Levels of laminin-5 γ2-chain 40 and 70 kDa fragment correlated positively with both CAL and percentage of sites with bleeding ($r=0.236$ and 0.272; $p<0.05$, respectively), clinical markers of periodontal disease. Laminin-5 γ2-chain 40 and 70 kDa fragment levels were slightly correlated with PD ($r=0.217$; $p=0.0533$). There was no correlation between the total amount of GCF laminin-5 γ2-chain 40 and 70 kDa fragment levels and the percentage of sites with plaque ($r=0.55$; $p=0.1730$). GCF volume also correlated with total levels of laminin-5 γ2-chain 40 and 70 kDa fragments ($r=0.258$; $p=0.0217$).

P.gingivalis trypsin-like proteinase produced multiple-size lower molecular weight fragments and short peptides from mature and unprocessed 100 and 140 kDa laminin-5 γ2-chain species (Fig. 1B, lanes 2 and 3) differing clearly from human MMPs that cleave laminin-

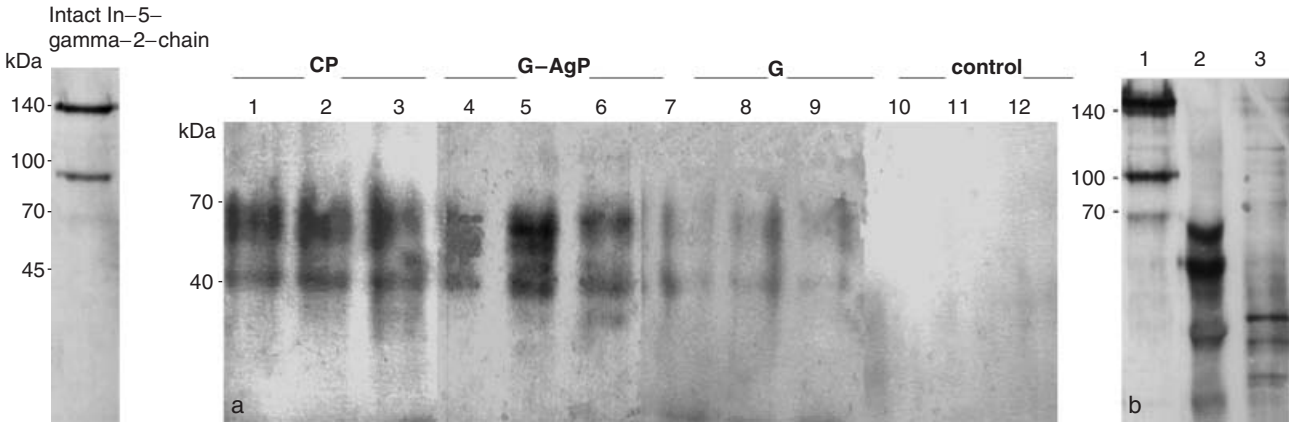

Fig. 1. A Western immunoblot of laminin-5 γ2-chain immunoreactivities in (a) gingival crevicular fluid (GCF) from chronic periodontitis (CP), generalized aggressive periodontitis (G-AgP), gingivitis and healthy group sites as well as the (b) effect of *P.gingivalis* trypsin-like proteinase on intact laminin-5 γ2-chain. Intact 140 and 100 kDa laminin-5 γ2-chains are shown as a control on the left. (a) CP, G-AgP, gingivitis and healthy represent three representative GCF samples from chronic periodontitis, aggressive periodontitis, gingivitis and healthy groups, respectively. (b) Intact 100 and 140 kDa laminin-5 γ2-chain species (5 μg) were exposed to buffer (lane 1) for 5 h and partially purified *P.gingivalis* trypsin-like proteinase (1 μg) for 1 h (lane 2) and 5 h (lane 3) at 37°C. Molecular weight markers are indicated on the left.

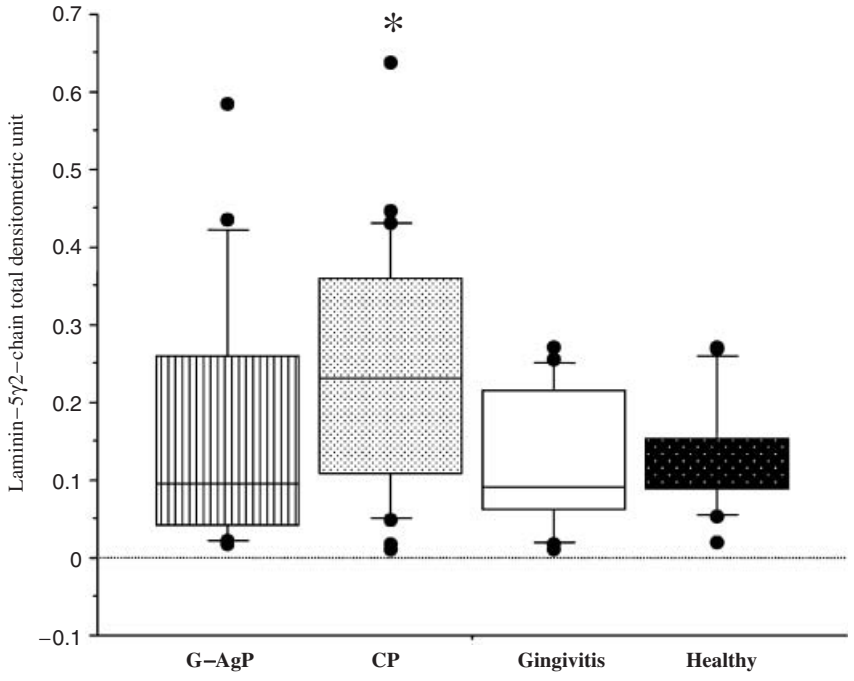

Fig. 2. Gingival crevicular fluid laminin-5 γ2-chain total amount of chronic periodontitis, generalized aggressive periodontitis , gingivitis and healthy groups. Box plots show medians, 25th and 75th percentiles as boxes, and 10th and 90th percentiles as whiskers. Outside values are shown as open circles. *Significantly different from the gingivitis and healthy group (Kruskal–Wallis test, $p<0.05$, Mann–Whitney *U*-test, $p<0.008$).

5 γ2-chain to 40 and 70 kDa fragments (Pirilä et al. 2003).

Discussion

In the present study, we investigated levels, molecular forms and fragmentation of laminin-5 γ2-chain in GCF of patients with different periodontal diseases to determine how these levels changed as the severity of periodontal disease increased. Laminin-5 γ2-chain was present in GCF predominantly as 40 and 70 kDa fragmented molecular species. Hardly any of the studied GCF samples showed intact laminin-5 γ2-chain 100 and 140 kDa species. Here, we used Western immunoblot together with computer-quantitation because, to the best of our knowledge, no ELISA-techniques exist to assess laminin-5 γ2-chain and, especially, its proteolytic fragments. The results demonstrated that total amounts of GCF laminin-5 γ2-chain were significantly elevated in patients with CP relative to other study groups. On the other hand, total amounts of GCF laminin-5 γ2-chain were similar among other groups studied. To the best of our knowledge, this is the first study that has examined total amounts of laminin-5 γ2-chain and fragmentation in GCF from patients with various periodontal disease categories.

It has generally been accepted that analysis of GCF constituents can provide information on the association between specific metabolic change and periodontal disease status (Lamster et al. 1985, Tsai et al. 1995). One important consideration in the analysis of host mediators in GCF is the method of presenting the data. Some researchers have stated that expression of GCF data as total amount per standardized sampling time is a more sensitive method than reporting them as concentration (Lamster et al. 1986, Hanioka et al. 2000). In the present study, we collected GCF samples for the same length of time and reported the data as total amount per sample as well as concentration.

In the healthy periodontium, intact junctional epithelium forms the first defence barrier against microbial invasion into periodontal tissues. When the host bacterium equilibrium is disturbed, bac-

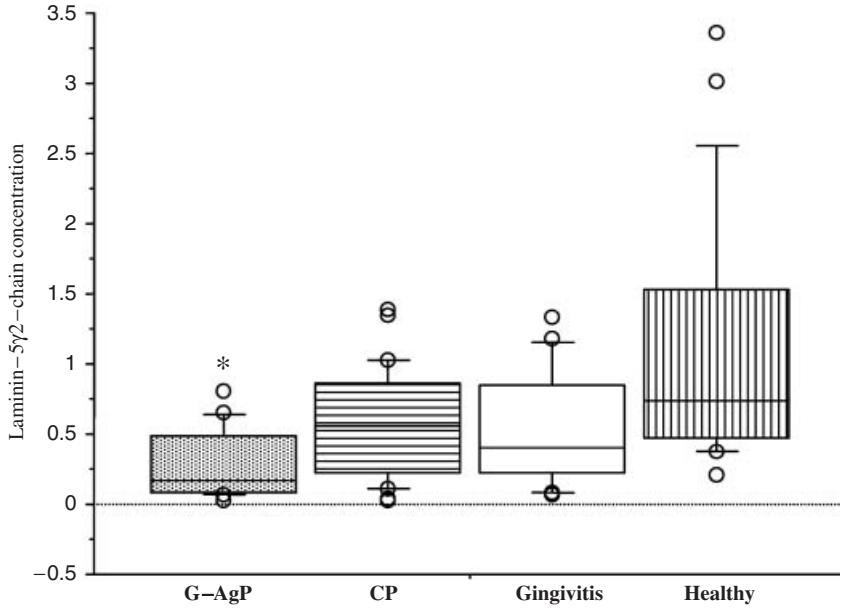

Fig. 3. Gingival crevicular fluid Ln-5 γ2-chain concentration of chronic periodontitis , generalized aggressive periodontitis , gingivitis and healthy groups. Box plots show medians, 25th and 75th percentiles as boxes, and 10th and 90th percentiles as whiskers. Outside values are shown as open circles. *Significantly different from healthy group (Kruskal–Wallis test, $p<0.05$, Mann–Whitney *U*- test, $p<0.008$).

Table 2. Correlations between laminin-5 γ2-chain levels and clinical parameters

Clinical parameters	Laminin-5 γ2-chain
Probing pocket depth	0.217
Clinical attachment loss	0.236‡
% of sites with bleeding	0.272‡
% of sites with plaque	0.155
GCF (μl)	0.258‡

‡$p<0.05$.

terial or host-derived factors trigger host defence mechanisms, which results in the degeneration of the dentoepithelial junction and, consequently, the conversion of the junctional epithelium into an infected pocket epithelium (Page & Schroeder 1976, Madianos et al. 2005). Our findings demonstrated the presence of laminin-5 γ2-chain fragments in GCF of healthy subjects, which could reflect the eventual existence of subclinical inflammation and turnover of the basement membrane (Page & Schroeder 1976, Birkedal-Hansen et al. 1993, Giannelli et al. 1997). The gingivitis group represented a group of subjects, who were characterized by the presence of gingival inflammation without periodontal-attachment loss. By having this group, we were able to compare how laminin-5 γ2-chain fragments are involved in the destructive process during the progression of periodontal disease.

Intact laminin-5, a major component of the internal basal lamina, provides significant adhesive properties between the tooth and the epithelium interface. However, after having been cleaved by MMPs, laminin-5 γ2-chain 40 and 70 kDa fragments (Giannelli et al. 1997, Pirilä et al. 2003) can stimulate epithelial cell migration, i.e., pocket formation (Gagnoux-Palacios et al. 2001, Pöllänen et al. 2003). In the present study, CP patients have significantly elevated GCF laminin-5 γ2-chain 40 and 70 kDa fragment levels when compared with gingivitis and periodontally healthy subjects. In our previous study (Emingil et al. 2004b), we have shown the presence of 45 and 70 kDa laminin-5 γ2-chain fragments in periodontitis patients' GCF, which could strongly suggest the proteolytic processing of the intact laminin-5 by MMPs in periodontal disease (Giannelli et al. 1997, Pirilä et al. 2001, Pirilä et al. 2003). Furthermore, the cleavage pattern of laminin-5 γ2-chain detected in GCF differs clearly from that produced by *P.gingivalis* trypsin-like proteinase, suggesting that human, not microbial, proteinases (MMPs) are predominantly responsible for it. Although *P.gingivalis* trypsin-like proteinase could initially process laminin-5 γ2-chain to 40 and 70 kDa fragments, further incubation with this enzyme always resulted in their fragmentation into small peptides; however, no such degradation could be detected in any GCF or peri-implant sulcular fluid samples studied (Kivelä-Rajamäki et al. 2003, Emingil et al. 2004b). The cleaved laminin-5 γ2-chain in GCF can originate from the degraded basement membrane between the junctional epithelium and connective tissue, because the junctional epithelium cells can express laminin-5 mRNA while in other parts of gingival epithelium the laminin-5 expression is less prominent (Hormia et al. 1998, Gürses et al. 1999). Figueredo & Gustafsson (2000) found elevated total laminin levels in GCF from periodontitis patients compared with gingivitis patients. They suggested activated neutrophils to be generating extensive destruction of the basement membrane. Especially, the neutrophil-derived MMP-8 species has been shown to correlate with the levels of laminin-5 γ2-chain 40 and 70 kDa fragments in GCF and peri-implant sulcular fluid before and after periodontal treatment (Kivelä-Rajamäki et al. 2003, Emingil et al. 2004b,c). In this regard, the present data support and further extend these previous findings that the presence of elevated laminin-5 γ2-chain 40 and 70 kDa fragments in GCF could reflect basement membrane disruption caused by the active ongoing inflammation at the dentogingival area. Elevated laminin-5 γ2-chain 40 and 70 kDa fragment levels in CP patients' GCF suggest that these fragments could play a role in the conversion of a gingival sulcus into an infected pocket epithelium during the destructive process in periodontal tissues. In other words, increased laminin-5 γ2-chain fragment levels could reflect the extent of the inflammatory reaction in CP.

It has been suggested that a specific response of junctional epithelial cells to microbial and host factors is an important factor in the pathogenesis of certain forms of periodontal disease (Pöllänen et al. 2003). AgP and CP, clinically distinct forms of periodontal diseases, have different aetiologies and rates of progression (Flemmig 1999, Tonetti & Mombelli 1999). Previous studies have shown that alteration in host defence cell functions (Takahashi et al. 2001, Emingil et al. 2001a), deregulated cytokine and inflammatory mediator levels (Emingil et al. 2001b, c, 2004a, 2005) and the presence of specific genetic risk factors (Hart 1996, Parkhill et al. 2000, Loos et al. 2005, Shapira et al. 2005)

could attribute to the pathogenesis of the aggressive form of periodontal disease. Numerous investigators have reported that AgP patients exhibit depressed or hyperactive neutrophil functions, while patients with CP do not have such abnormalities including chemotaxis (Altman et al. 1985, Van Dyke et al. 1988, Hart et al. 1994, Kantarci et al. 2003, Van Dyke & Serhan 2003). In the present study, G-AgP patients had laminin-5 γ2-chain fragment levels comparable to those found in gingivitis and healthy groups. There is also no difference in GCF laminin-5 γ2-chain fragment levels between aggressive and CP patients, although the amount of periodontal destruction and gingival inflammation in the sampling areas were comparable. As AgP has a more aggressive clinical course than the chronic form of periodontal disease (Tonetti & Mombelli 1999), one might expect to find more or similar GCF laminin-5 γ2-chain fragment levels in this group. However, this prediction was not verified by the biochemical findings of the present study. It is possible that the basement membranes in the G-AgP patients are degraded to such an extent that almost all laminin-5 γ2-chain fragments are lost. Another possibility is that as a result of the inflammation in G-AgP, epithelial cells do no longer express laminin-5 γ2-chain fragments but express other isoforms. It was also recently concluded that the aggressive and chronic forms of periodontitis have similar cellular composition (Berglundh & Donati 2005, Kinane & Attström 2005). Therefore, it is not surprising to find similar GCF laminin-5 γ2-chain fragment levels in both aggressive and chronic forms of periodontitis as well as in gingivitis and healthy sites.

Our present study has provided the first evidence about the presence of laminin-5 γ2-chain levels and fragments in GCF from different periodontal diseases. In conclusion, increased GCF laminin-5 γ2-chain fragments in periodontitis sites with deep periodontal pocket suggest that these cleaved fragments could reflect the extent of the inflammatory reaction in CP.

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Address:
Gülnur Emingil
Ege Üniversitesi
Dişhekimliği Fakültesi
Periodontoloji Anabilim Dalı
Bornova-35100, İzmir
Türkiye
E-mail: gemingil@yahoo.com

Clinical Relevance

Scientific rationale for study: Periodontal disease results from interactions between bacteria and host response. This study aimed to investigate the presence of molecular forms and gingival crevicular fluid (GCF) laminin-5 γ2-chain fragment levels in patients with different periodontal disease.

Principal findings: Chronic periodontitis patients had elevated GCF laminin-5 γ2-chain fragment levels compared with gingivitis and healthy groups. Aggressive periodontitis patients had similar GCF laminin-5 γ2-chain fragment levels to chronic periodontitis as well as gingivitis and healthy groups.

Practical implications: This study has provided the first evidence that GCF laminin-5 γ2-chain fragment levels might indicate the extent of the inflammatory reaction in chronic periodontitis.