Chemokine RANTES in gingival crevicular fluid of adult patients with periodontitis


Abstract

Background, aims: This study presents the first evidence on the presence of the chemokine RANTES in the gingival fluid crevicular (GCF) of patients with periodontitis. RANTES is a chemokine that selectively attracts and activates macrophages and lymphocytes. Leucocytes play a critical rôle in the host response to the subgingival microflora.

Method: In this study, the presence de RANTES in GCF was determined in samples obtained from adult patients with periodontitis and from control subjects with clinically healthy gingiva. GCF was collected from different probing depths (<3 mm, 4–6 mm, >6 mm) (n=72); and active (n=12) and inactive sites (n=12). An active site was defined as attachment loss >2 mm, as determined by sequential probing and the tolerance method. GFC was collected for 30 s using Periopaper® strips, and RANTES was quantified by ELISA.

Results: The presence of RANTES was detected exclusively in the group of patients with periodontitis, presenting a total amount of 40.43±16 pg and a concentration 67.80±41 pg/ml. RANTES concentration was significantly higher in probing depth <3 mm than in probing depth >6 mm (87.24 versus 51.87, p=0.014). Total amount and concentration in the GCF samples from active sites were higher that in inactive sites (p>0.05).

Conclusions: The finding that RANTES is found only in patients with periodontitis, may represent a general feature of chronic inflammatory in peri-odontal diseases. Finally, RANTES may be implicated in the biological mechanisms underlying the pathogenesis and progression of periodontal disease.

Key words: periodontitis; chemokines; RANTES; gingival crevicular fluid

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amino acid (CXC). In contrast, the C family has a single NH2-terminal cysteine residue and CXC family has these cysteines separated by 3 intervening amino acids (Baggiolini 1998, Ward et al. 1998).

5 receptors for CXC chemokines and 8 for CC chemokines have already been characterized (Murphy 1996). Among each family, most receptors recognize more than one chemokine, and certain chemokines interact with more than one receptor, reflecting that redundancy and versatility are important features of the chemokine system. Chemokine receptors are selectively expressed in leukocytes. Thus, IL-8, a CXC chemokine, is a chemoattractant for neutrophils, that express CXC receptors (Baggiolini et al. 1997). RANTES, a member of CC chemokines, activates monocytes, eosinophils and basophil leukocytes (Murphy 1996), inducing chemotaxis and the release of other cell mediators (Baggiolini & Dahinden 1994). Both families of chemokine receptors, CXC and CC, are present in activated T-lymphocytes (Bonecchi et al. 1998, Qin et al. 1998).

Different chemokines have been previously implicated in CIPD. Several authors have described the presence of IL-8 chemokine in gingival crevicular fluid (GCF) (Mathur et al. 1996, Payne et al. 1993, Tsai et al. 1995) and in association with β-glucuronidase, a marker of the presence of PMN leukocytes (Chung et al. 1997). Among the members of the CC subfamily, only macrophage inflammatory protein-1 (MCP-1) has been directly implicated in CIPD. MCP-1, an active chemoattractant of monocytes/macrophages, has been detected in human GCF and inflammatory gingival tissue (Hanazawa et al. 1993, Tonetti et al. 1994, Xiaohui et al. 1993). However, to our knowledge, RANTES, has not been associated with CIPD. RANTES interacts with CCR3 and CCR5 chemokine receptors, which are present in monocytes, eosinophils, basophil leukocytes and activated T-cells (Bacon & Schall 1996, Dairaghi & Schall 1996, Greaves et al. 1998, Schall & Bacon 1994, Strietter et al. 1996).

Moreover, an interesting feature of RANTES in the study of CIPD is supported by recent data demonstrating that CCR5, is expressed almost exclusively by T-helper type 1 (Th1) cells (Loetscher et al. 1998). RANTES is an efficient chemoattractant of Th1 cells, inducing their dose response transmigration, whereas Th2 cells are not attracted by this chemokine (Siveke & Hamann 1998).

The aim of our study was to determine the presence of RANTES in GCF samples from adult patients with periodontitis. Therefore, RANTES, a specific chemoattractant of macrophages and lymphocytes, may be involved in the recruitment of inflammatory cells from towards periodontal tissues.

Materials and Methods

Patients

Patients for this study were selected from Primary Attention Service, Faculty of Odontology, Universidad Complutense de Madrid. Criteria for entry were a minimum of 14 natural teeth, excluding 3rd molars, and including at least 10 posterior teeth. Patients with chronic inflammatory periodontal disease (CIPD) had moderate to advanced periodontal disease (at least 5–6 teeth had sites with probing depth >6 mm and with attachment loss ≥3 mm and extensive radiographic bone loss), and had received no treatment at the time of examination. Subjects did not suffer from systemic illness and they had not received antibiotics or non-steroid anti-inflammatory therapy in the 6-month period prior to the study. The control group was selected from normal volunteers with no evidence of periodontal disease. Patients were monitored over a period of 4 months from the beginning of the study until at least two sites showed activity, determined by >2 mm attachment loss. The protocol was explained to all patients and Institutional Review Board-approved informed consents were signed. Within 2 weeks of the detection of disease activity all patients were provided with periodontal treatment.

Clinical measurement

Prior to the beginning of the study, all subjects received a supragingival prophylaxis to remove gross calculus and allow probing access. All teeth, with the exception of third molars were scored for probing depth and clinical attachment level. A 2nd measurement of the attachment level and probing depth was taken within 7 days of the first measurement. They were obtained from 6 sites per tooth every 2 months, by a single calibrated investigator. Measurements were made at the mesiobuccal, buccal, distobuccal, distolingual, lingual and mesiolingual positions. Dichotomous measurement of supragingival plaque accumulation (PI) and bleeding on probing (BOP) were also made at 6 sites per tooth 1× every 2 months. Attachment level (AL) and pocket depth (PD) measurements were taken with two models of the Florida Probe (Florida Probe Corporation, Gainesville, Fl). The Florida Disk Probe was used for relative attachment level recordings and the pocket depth probe was used to make probing depth recording. Disease activity was defined by the tolerance method (Haffajee et al. 1983). The active sites exhibited attachment loss >2 mm during the following 2-month period, considering the site threshold. Population and subject thresholds were not considered because no differences were observed.

Collection of gingival crevicular fluid (GCF)

If patients met entry criteria, GCF samples were collected from selected sites in all patients according to probing depth groups: <3 mm (n=24), 4 to 6 mm (n=24), or >6 mm (n=24) and following a simple aleatory sampling. From each patient, 6 samples were collected at the beginning of the study (baseline) (n=36 samples) and 6 samples two months later (n=36 samples) and they were always obtained from active and inactive sites. GCF was collected from active sites by the time that attachment loss was >2 mm. Simultaneously, GCF was collected from inactive sites, which were defined as those sites showing similar probing depth as active sites but in the absence of attachment loss. GCF samples in control groups were collected in the mesiobuccal gingival sulci at teeth 16 and 26 (n=12 per group).

After isolating the tooth with a cotton roll, supragingival plaque was removed with curettas (Hu Friedy, Gracey, USA), avoiding touching the marginal gingiva. The crevicular site was then gently dried with an air syringe. GCF was collected with filter paper strips Periopaper® (ProFlow, Amityville, New York). Strips were placed into the sulcus/pocket until mild resistance was felt and left in place for 30 s. Strips contaminated by saliva or blood were excluded from the sampled
group. A calibrated Periotron-6000® (ProFlow, Amityville, New York) was used for volume determination of the strips. The Periopaper® strips were then immediately placed inside a sterile vial and stored at −70°C until analysis.

**Analysis of GCF**

Following collection of GCF, the volume of the sample on the Periopaper® strips was measured using a calibrated Periotron-6000®. A standard curve correlating digital readout to volume was constructed for each calibration with standard human serum. Each volume was applied 3 times to a Periopaper strip. The Periopaper® strips were then immersed in 0.18 M sulfite, and the corresponding periotron units were recorded. No re-calibration of the Periotron-6000® was necessary throughout the study period. The readings from the Periotron-6000® were converted to an actual volume (µl) by reference to the standard curve.

After GCF collection, strips were placed in eppendorf vials with 50 µl of phosphate buffered saline with 0.05% Tween-20 (PBS-T). GCF was extracted by centrifugation at 10,000 g for 5 min at 4°C (Heraeus SEPA TECH Biofuge 17RS), and the procedure was repeated three times (Chung et al. 1997).

**Quantification of RANTES**

Aliquots of each GCF sample were assayed by an enzyme linked immunosorbent assay (ELISA) to determine the levels of RANTES using matched antibody pairs and according to the manufacturer’s recommendations (ENDOGEN Inc., Cambridge, USA). Briefly, plates (F16 Maxisorp Loose, Nunc A/S Roskilde, Denmark) were coated with the anti-human monoclonal RANTES antibody (M-421B-E) overnight at 4°C. Plates were blocked with PBS 4% BSA and washed 3×. 10 µl of GCF samples in 90 µl PBS-T were added to the plate in duplicate and incubated 1 h at room temperature (RT). 100 µl of appropriate diluted biotin-labeled antibody (M-420B-B) was added to each well, covered and incubated for 1 h at room temperature (RT). Plates were washed 3× and incubated with 100 µl HRP-conjugated Streptavidin (ENDOGEN) 1:32,000 for 30 min at RT. After extensive washing, 100 µl TMB (ENDOGEN) substrate solution was added. The reaction was stopped after 30 min by the addition of 50 µl de 0.18 M sulfuric acid, and color measured at 450 nm using an automated microplate spectrophotometer ( Labsystems Multiskan, BICHROMATIC, UK). RANTES concentration in the samples was calculated with a standard curve (15–1000 pg) obtained with recombinant RANTES chemokine (ENDOGEN Inc., Cambridge, USA). Values below 15 pg were not considered. RANTES concentration was calculated according to the following formula: RANTES concentration (pg/µl)=total RANTES (pg)/volume (µl).

**Statistical methods**

The clinical parameters as well as the amounts and concentrations of RANTES at healthy and diseased sites were calculated as subject means±standard deviation. The unpaired Student t-test was used to analyze differences in clinical and biochemical parameters between patients from periodontitis and control group. Differences in clinical and biochemical parameters inside each group were also analyzed with the unpaired Student t-test. The significance (α=0.05) of differences was assessed using the Turkey test. The correlation of RANTES levels with clinical parameters, probing depth and degree of

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**Table 1. Clinical characteristics (mean±SD) from periodontitis and control groups**

<table>
<thead>
<tr>
<th></th>
<th>Periodontitis group</th>
<th>Control groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=6)</td>
<td>(n=6)</td>
</tr>
<tr>
<td>age (years)</td>
<td>47.16±11.15</td>
<td>40.16±3.71</td>
</tr>
<tr>
<td>% males</td>
<td>33.33</td>
<td>33.33</td>
</tr>
<tr>
<td>mean probing depth (mm)</td>
<td>3.17±0.53</td>
<td>–</td>
</tr>
<tr>
<td>mean attachment level (mm)</td>
<td>3.6±1.15</td>
<td>–</td>
</tr>
<tr>
<td>mean probing depth at active sites (mm)</td>
<td>4.97±0.43</td>
<td>–</td>
</tr>
<tr>
<td>% sites with plaque</td>
<td>81.50±11.11</td>
<td>45.40±8.7</td>
</tr>
<tr>
<td>% sites with BOP</td>
<td>56.28±15.7</td>
<td>–</td>
</tr>
<tr>
<td>GCF volume (µl)</td>
<td>0.72±0.33</td>
<td>0.26±0.10*</td>
</tr>
</tbody>
</table>

* p-value=0.0001.

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**Table 2. RANTES in gingival crevicular fluid from periodontitis and control groups (means±SD)**

<table>
<thead>
<tr>
<th></th>
<th>Periodontitis group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=36)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 months</td>
<td>range</td>
</tr>
<tr>
<td>RANTES (pg)*</td>
<td>42.02±20.6</td>
<td>38.67±9.7</td>
</tr>
<tr>
<td>RANTES (pg/µl)**</td>
<td>64.86±39.3</td>
<td>70.90±44.7</td>
</tr>
<tr>
<td>GCF (µl)</td>
<td>0.74±0.3</td>
<td>0.70±0.3</td>
</tr>
</tbody>
</table>

* Total amount of RANTES (pg). ** Concentration of RANTES (pg/µl). bkg under detection level. nd, not done.

**Table 3. RANTES in GCF from periodontitis group according to probing depth (mean±SD)**

<table>
<thead>
<tr>
<th>Probing depth (mm)</th>
<th>Total amount (pg)</th>
<th>Concentration (pg/µl)</th>
<th>GCF (µl)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>baseline</td>
<td>2 months</td>
<td>total</td>
</tr>
<tr>
<td>&lt;3</td>
<td>42.11±28.2</td>
<td>30.46±17.3</td>
<td>39.68±21.8</td>
</tr>
<tr>
<td>4 a 6</td>
<td>38.68±20.0</td>
<td>42.68±11.5</td>
<td>42.45±13.9</td>
</tr>
<tr>
<td>&gt;6</td>
<td>41.68±16.0</td>
<td>29.98±14.2</td>
<td>39.07±12.1</td>
</tr>
</tbody>
</table>

* p-value=0.014. * p-value=0.012. * p-value=0.0005.
activity in healthy and diseased subjects was examined using Pearson’s correlation.

Results

The clinical characteristics of patients included in this study are grouped in Table 1. 4 males and 8 females were studied, with age range 35–67 years old (mean age $47.16 \pm 11.05$ for periodontitis group; mean age $40.16 \pm 3.71$ for control group). No statistically significant differences in age or gender existed between the two groups. As expected, significantly lower amounts of GCF were obtained from control subjects as compared with periodontitis patients ($p$-value=$0.0001$).

Mean variations of total amount and concentration of RANTES in the periodontitis group and in the control group at the beginning of the study and after two months, are shown in Table 2. RANTES in GCF was analyzed in 72 samples from patients with periodontal disease. In this group mean values of $40.43 \pm 16.3$ and an estimated concentration of $67.80 \pm 41.8$ pg/μl were obtained. In the control group, all samples tested had RANTES values below detection levels ($<15.62$ pg).

Interestingly, total amount of RANTES was independent of the probing depth. As shown in Table 3 and Fig. 1A, no significant differences were observed between sites with $<3$ mm, 4–6 mm and $>6$ mm in any of the patients analyzed. RANTES concentration in GCF at sites with $<3$ mm probing depth was significantly higher ($87.24$ pg/μl) than that observed at sites with $>6$ mm ($51.87$ pg/μl; $p=0.014$) (Table 3 and Fig. 1B).

However, the volume of GCF recovered with the periopaper is directly related to the probing depth in the periodontal pockets; as shown in Table 3, a volume of $0.50$ μl GCF was obtained from probing depth $<3$ mm, $0.75$ μl from periodontal pockets with probing depth of 4–6 mm and $0.90$ μl from $>6$ mm. The volume variation observed between the different probing depths was statistically significant ($p<0.05$).

Therefore, considering that the total amount of RANTES remains fairly constant and is independent of probing depth, whereas GCF volume increases with probing depth, RANTES concentration in GCF at sites with $<3$ mm probing depth was significantly higher ($87.24$ pg/μl) than that obtained at sites with $>6$ mm ($51.87$ pg/μl; $p=0.014$) (Table 3).

As shown in Table 4, the amount of RANTES measured in an active site showed no significant variation ($p>0.05$) as compared with inactive sites presenting similar probing depth. Active sites showed no significant increase in RANTES concentration ($49.64$ pg/μl) compared with inactive sites ($47.53$ pg/μl; $p>0.05$). Moreover, GCF volume in active sites (1.03 μl) was not significantly higher than in inactive sites (0.83 μl). Consequently, active disease could not be correlated with the presence of RANTES.

Because of the difference between total amount and concentration, the relationship between the GCF volume and these two parameters were examined. Table 2 and Table 3 showed the GCF volume increased in subjects with periodontitis with probing depths from $<3$ mm to $>6$ mm. Table 4 showed that GCF values were reduced in inactives sites. A negative correlation ($r=-0.647$, $p<0.05$) between the total amount of RANTES and the GCF volume was found. Similarly, positive correlation was found between concentration of RANTES and the GCF volume ($r=0.678$, $p<0.05$). The results of our study found no correlation between levels of RANTES with clinical parameters.

Discussion

This study examined the total amount and concentration of the chemokine RANTES in GCF of adult patients with chronic inflammatory periodontal disease. Our data demonstrates that chemokine RANTES is present in GCF of patients with periodontitis and is undetectable in healthy subjects. RANTES is a member of a superfamily of proinflammatory cytokines designates chemokines implicated in selective attraction of different leukocyte subsets (Baggioiini 1998, Ward et al. 1998). RANTES belongs to a subfamily of chemokines characterized by conservation of the firsts two adjacent cystines in the primary protein structure (Schall & Bacon 1994, Schall et al. 1990). It is, a 68 amino acid protein, originally identified as a T-cell specific gene (Schall et al. 1988). Subsequent studies demonstrate that RANTES is more broadly expressed than originally thought, and is inducible in a variety of tissues by specific stimuli (Schall et al. 1988). CC chemokines are considered to promote inflammation by the selective chemotraction of specific subsets of haematopoietic cells; RANTES in particular is a chemoattractant of eosinophils (Kame-

### Table 4. RANTES in GCF from periodontitis group in active and inactive sites

<table>
<thead>
<tr>
<th>Site designation</th>
<th>No. observations</th>
<th>Total amount (pg)</th>
<th>Concentration (pg/μl)</th>
<th>GCF (μl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>active</td>
<td>12</td>
<td>47.30±14.51</td>
<td>49.64±19.06</td>
<td>1.03±0.30</td>
</tr>
<tr>
<td>inactive</td>
<td>12</td>
<td>37.55±14.25</td>
<td>47.53±17.71</td>
<td>0.83±0.21</td>
</tr>
</tbody>
</table>

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**Fig. 1.** (A) Presence of RANTES in GCF obtained from different sites of periodontitis patients and according to probing depth. GCF from each site was evaluated by ELISA and probing depth evaluated as previously described. (B) RANTES concentration measured in GCF obtained from each site in patients with periodontitis and according to probing depth. RANTES concentration was calculated according to the formula described in Material and Methods.
ability to determine whether mechanisms may be explained because the impossi-
ment loss, considered as a marker of ac-
tion of mediators, such as
ting on RANTES concentration.
3 mm was signifi-
yness was not significant (p>0.05).
Our data demonstrate that sulci <3 mm contains high concentration of RANT-
chological activity of mediators, such as cytokines, whose function depends on
binds to cell surface receptors, is closely related to local concentrations.
In fact, it has been reported that, in vitro, different responses are obtained de-
concentration below 100 nM RANTES induces a predominant chemotactic re-
concentration above 100 nM induce T-cell activation, as oc-
curs with antigen stimulation of T cells (Dairaghi et al. 1998). This suggest that
both types of chemokine response, de-
concentration could play an independent role in the
progression of periodontitis.

In the present study, we were unable to establish an association between levels of RANTES and probing attachment loss, considered as a marker of activ-
ty progression. However, episodic periodontal probing attachment loss may be associated with variations in the supracrestal inflammatory cell popula-
tions where significantly more mast cells, monocytes/macrophages and plasma cells are present in activity sites as compared with inactive sites (Zappa et al. 1991). In part, our observations may be explained because the impossibility to determine whether mechanisms underlying attachment loss were active at the time of sampling, which was per-
formed at 2-month intervals.

In our study, the correlation between the levels of RANTES and clinical para-
eters was determined in the sampled sites, and although total amount and concentration in GCF obtained from inflamed sites was much greater than that from healthy sites, our results showed no correlation of RANTES with clinical parameters. Examination of gingival tissue for bleeding following probing or the presence of suppuration are indi-
cators of the degree of inflammation. However, these clinical parameters are subject to the variability inherent in clinical evaluation, therefore, the lack of re-
lationship between RANTES in GCF and clinical parameters could be ex-
plained by this fact. In our study, we have observed a significant decrease in RANTES concentration in GCF of probing depth >6 mm compared to probing depth <3 mm. However, con-
sidering that the volume of GCF pro-
duced in sites with >6 mm was signifi-
cantly higher than in sites with probing depth <3 mm. It explains the lower RANTES concentration detected in sites with higher probing depth.

RANTES is produced locally at inflam-
atory sites, it binds to activated endothelium (Pattison et al. 1994), and is capable of attracting monocytes (Wiedermann et al. 1995). Monocytes/ macrophages play a central role in mo-
ibilizing the host defense mechanisms against bacterial infection, because they are involved both in the initial responses as antigen-presenting cells and in the ef-
ector phase as inflammatory, tumoricid-
al and microbicidal cells. Early studies (Attstrom 1970, Sinden & Walker 1979) have shown that monocytes markedly infiltrate periodontal tissues in adult periodontal patients. Monocytes/macrophages produce multiple regul-
atory factors such as inflammatory cyto-
kines and growth factors, and also re-
lease arachidonic acid metabolites, oxy-
gen radicals, and proteases. Considering the multifunctional abili-
ties of monocytes/macrophages, these cells could be involved in initiation and develop-
ment of the inflammatory reac-
tions and alveolar bone loss observed in adult periodontal disease. Therefore, analysis of the mechanism that induce monocyte recruitment into periodontal tissues represent an important step to-
ward understanding the pathogenesis of this disease.

On the one hand, RANTES is an effi-
cient chemoattractant for Th1 cells (but not for Th2 cells), inducing a dose response transmigration of Th1 (Siveke & Hamann 1998). Th1 and Th2 cells define 2 forms of the specific CD4+Th cell-mediated immune re-
sponse based on their differential cyto-
kine secretion (Mosmann & Coffman 1989). Th1 or Th2 cell cytokines have been detected in periodontal diseases by several investigators (Ishikawa et al. 1997). However, diverse periodontal pathogens cause different periodontal disease, and furthermore variable host response are observed among patients or even during the various stages of the dis-
ease. Recently, CCR5, the receptor for RANTES and MIP-1α and β chemokin-
es, has been reported to be preferentially expressed during human Th1 responses (Loetscher et al. 1998, Qin et al. 1998). Moreover, activated T cells, expressing CCR3 and CCR5, are specifically at-
tracted by RANTES, MCP-1 and MIP-
1β chemokines, which were describe re-
ported to be ligands for these receptors (Qin et al. 1998). Thus, several findings suggest the existence of dynamic pro-
grams in the differentiation/activation process of human Th1 and Th2 cells. The understanding of genetic and environ-
mental mechanisms responsible for these associations may provide new insights into the functional regulatory of the spec-
ific effector cells.

Several authors have previously de-
scribed the presence of IL-8 and MCP-1 chemokines in GCF (Chung et al. 1997, Mathur et al. 1996, Murphy 1996, Tonetti et al. 1994). We have determined that higher levels of RANTES are found in GCF from patients with CIPD as compared to healthy subjects. Considering that gingival inflammation develops in parallel to increasing infiltrating of monocytes/macrophages and lymphoid cells, it suggests that in CIPD, the migration and accumulation of these cells in inflammatory loci might be related to the release of chemokines, such as RANTES, providing a potential mech-
anism to account for the recruitment of inflammatory cells observed in bac-
terially induced inflammatory processes in human gingiva.

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We thank Iciar Lazaro for critical
Das Chemokin RANTES in der Sulkussilik-
sigkeit erwachsener Parodontitispatienten

Einleitung: Diese Studie präsentiert die ersten
Beweise für das Vorkommen des Chemokins
RANTES in der Sulkussilikisität (SF) von
Patienten mit Parodontitis. RANTES ist ein
Chemokin, das Makrophagen und Lympho-
zyten selektiv anzieht und aktiviert. Leuko-
zyten spielen eine wichtige Rolle in der Wirt-
sabwehr gegenüber der subgingivalen Mikro-
flora.

Methoden: In dieser Studie wurde RANTES in
SF untersucht, die erwachsenen Parodon-
titispatienten und Kontrollprobanden mit
klinisch gesunder Gingiva entnommen wur-
den. SF wurde an Stellen mit unterschiedli-
chen Sondierungstiefen (ST < 3 mm, 4–6 mm, > 6 mm; n = 72) und an aktiven (n = 12)
sowie inaktiven (n = 12) Stellen gewonnen.
Stellen wurden als aktiv betrachtet, wenn ein
Attachementverlust von > 2 mm vorlag, der
durch sequentielle Messungen und die Tole-
ranzmethode bestimmt worden war. SF wur-
de mittels Periopaper-Streifen über 30 Se-
kunden gesammelt und RANTES mittels ELISA quantifiziert.

Ergebnisse: RANTES konnte lediglich in der
Gruppe der Parodontitispatienten nachgewei-
sen werden mit einer Gesamtmenge von
40.43 ± 16 pg und einer Konzentration von
67.80 ± 41 pg/mL. Die RANTES-Konzentra-
tion war an Stellen mit ST < 3 mm signifikant
höher als an solchen mit ST > 6 mm (87.24 zu
51.87 pg, p = 0.014). Die Gesamtmenge und
Konzentration von RANTES in SF-Pro-
ben von aktiven Stellen unterschied sich nicht
signifikant von inaktiven Stellen.

Schlußfolgerungen: Die Beobachtung, daß
RANTES ausschließlich bei Parodontitispa-
tienten gefunden wurde, könnte darauf hin-
deuten, daß es sich dabei um ein generelles
Charakteristikum chronischer Entzündung
bei marginaler Parodontitis handels. RAN-
TES könnte eine Rolle in den biologischen
Mechanismen der Pathogenese und Progres-
son der Parodontitis spielen.

Résumé

La chimio kinase RANTES (Regulated on Acti-
vation, Normal T cell Expressed and Secre-
ted) dans le fluide gingival de patients adultes
atteints de parodontite

Cette étude présente la première preuve de la
présence de la chimio kinase RANTES dans le
fluide gingival de patients atteints de para-
donite. RANTES est une chimio kinase qui at-
tire et active sélectivement les macrophages et
les lymphocytes. Les leucocytes jouent un rôle
critique dans la réponse de l’hôte à la
microflore sous gingivale. Dans cette étude,
la présence de RANTES dans le fluide gingi-
val fut déterminée à partir d’échantillons ob-
tenus chez des patients adultes atteints de pa-
ronodite, et chez des sujets contrôles présent-
tant une gencive cliniquement saine. Le fluide
gingival fut collecté dans des sites ayant des
profondeurs au sondage différences (< 3 mm,
4–6 mm, > 6 mm; n = 72) et dans des sites
actifs (n = 12) ou inactifs (n = 12). On définis-
sait les sites actifs comme présentant une per-
te d’attache > 2 mm, déterminée par des
sondages répétés et une méthode de tolérance.
Le fluide gingival fut récolté pendant 30 s
avec des bandelettes de Periopaper et RAN-
TES fut quantifié par ELISA. La présence de
RANTES fut détectée exclusivement dans
le groupe des patients atteints de parodonti-
te, en quantité total de 40.43 ± 16 pg et une
concentration de 67.80 ± 41 pg/mL. La concen-
tration de RANTES était significativement
plus élevée dans les poches < 3 mm que dans
les poches > 6 mm (87.24 contre 51.87, p = 0.014).
La quantité total et la concentration de RANTES dans les échantillons de fluide
gingival des sites actifs étaient plus élevée que
dans des sites inactifs (p = 0.05). Le fait que
RANTES soit retrouvée seulement chez les
patients présentant une parodontite, peut
représenter un tableau général d’inflammation
chronique au cours des maladies parodontale-
es. Enfin, RANTES peut être impliquée dans
les mécanismes biologiques de la patho-
génie et de la progression des maladies para-
donatales.

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