Prevalence and progression of untreated periodontal disease in a young Indonesian population
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LONGITUDINAL EVALUATION OF THE DEVELOPMENT OF PERIODONTAL DESTRUCTION IN SPOUSES

CHAPTER 6

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During recent decades, it has become evident that severe periodontitis concentrates in a relatively small part of the population (Löe 1986, Truin et al. 1989, Burt 1990). One explanation for this phenomenon may be that some individuals are genetically predisposed to periodontitis. This concept of genetic predisposition was first studied in families of patients with juvenile periodontitis (JP). The results of most of these studies show that over 50% of the siblings of JP patients also suffer from periodontitis (Benjamin & Baer 1967, Melnick et al. 1976, Ohtonen et al. 1983, Spector et al. 1985, Boughman et al. 1992, Marazita et al. 1994). Recent data suggest that periodontitis not only aggregates in JP families, but also in families with other forms of periodontitis (Van der Velden et al. 1993, Petit et al. 1994). On top of that, twin studies revealed a strong genetic background for periodontitis (Michalowicz et al. 1991, Corey et al. 1993).

The present study deals with a population from a remote village in Indonesia deprived of regular dental care and showing a relatively high prevalence of periodontal disease. Previous research showed that in this population, a significant sibship effect existed for loss of attachment (Van der Velden et al. 1993), thus supporting the observation that periodontitis aggregates in families. Apart from a genetic predisposition for periodontitis, it has also been documented that environmental factors influence the development of periodontitis Wolf et al. 1994). Besides plaque, it is proposed that the development of periodontitis depends on the presence of a critical number of 1 or more putative periodontal pathogens (Slots & Listgarten 1988). It has been shown that transmission of Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis between parents and children (Alaluussua et al.1991, Könönen et al.1992, Petit et al. 1993a, DiRienzo et al.1994) and between spouses (Petit et al. 1993b, Saarela et al 1993, Van Steenbergen et al. 1993, DiRienzo et al.1994) can occur. The latter may have consequences for the periodontal condition of a subject who marries a periodontitis patient. For example, when a periodontitis patient harbouring A. actinomycetemcomitans and P. gingivalis marries a spouse with poor oral hygiene and gingivitis, transmission of these bacteria from the patient to the spouse can occur. As a consequence, the gingivitis may develop into
periodontitis. The implication of such a phenomenon is that after some time the periodontal condition of the spouses of a couple would resemble each other. This hypothesis is supported by the results of a recent study, which showed that spouses of severe periodontitis patients had a worse periodontal condition in comparison to spouses of periodontally healthy subjects (Von Troil-Lindèn et al. 1995). However, these data are obtained from a cross-sectional study. To substantiate the hypothesis that the periodontal condition of a spouse is influenced by that of the partner, the periodontal condition of young married couples in the Indonesian study population was evaluated longitudinally. The basic assumption was that if spouses would influence each others periodontal condition, the significant differences in clinical status as observed in the previous examination in 1987 would have decreased after 7 years of cohabitation. The purpose of the present study was to investigate whether changes had occurred in the differences in periodontal condition between spouses.

MATERIAL AND METHODS

This study is based on data of a longitudinal investigation on the onset and progression of periodontal destruction in a population deprived from regular dental care. For this longitudinal study, a remote village with about 2000 inhabitants at the Malabar tea estate on Western Java, Indonesia was selected. In 1987, all subjects available, who reported to be between 15-25 years of age, were studied clinically. 7 years later, in 1994, the clinical assessment was repeated in the subjects of this group still available for investigation. The culture of the subgingival microbiota was performed in all subjects.

Clinical examination
Both in 1987 and 1994, the following clinical parameters were recorded interproximally from the buccal aspect of all teeth.
- Plaque (Silness & Löe 1964)
- Calculus (Björby & Löe 1967)
- Probing depth (PD) using a force controlled probe (Brodontic, 240 N/cm²)
- Bleeding on probing using the force controlled probe and scored as:
0) no bleeding;
1) point bleeding within 30 s;
2) immediate, overt bleeding.

Loss of Attachment (LA) assessed by subtracting the distance between the gingival margin and the cemento-enamel junction (GM-CEJ) from the recorded probing depth or, in case of gingival recession adding the GM-CEJ value to the probing depth measurement. The GM-CEJ distance was evaluated using a Hu-Friedy® probe with a Williams calibration.

All measurements were rounded off to the nearest mm.

**Microbiological examination in 1994**

After the clinical examination was completed, in each quadrant the deepest site with the maximum loss of attachment was selected for microbiological evaluation. After careful removal of the supragingival plaque by means of a curette a pooled subgingival plaque sample was taken, using 2 sterile paperpoints per pocket. Subsequently the 8 paperpoints were suspended in 1.8 ml RTF and processed within 30 minutes. Specimens were vortexed for 30 s at the maximum setting and serially diluted in RTF in 10-fold steps. Aliquots of 0.1 ml appropriate dilutions were plated onto 5% horse blood agar plates (Oxoid no. 2) supplemented with haemin (5 mg/L) and menadione (1 mg/ml). TSBV plates were inoculated for the selective isolation of *A. actinomycetemcomitans* (Slots 1982). For transportation to the Netherlands the blood agar plates were anaerobically incubated in jars using an anaerobic system (BBL, Gaspak, Becton Dickinson, Cockeysville, USA). The TSBV plates were incubated in jars using a CO₂-generating system (BBL, Gaspak, Becton Dickinson, Cockeysville, USA). The jars were stored in boxes, which were kept at 37°C. 3 days were spent on sampling and plating and subsequent transportation to Amsterdam took 4 days. Immediately upon arrival in the laboratory blood agar plates were incubated in 80% - N₂, 10% - H₂ and 10% - CO₂ at 37°C for up to 14 days. TSBV plates were incubated in air + 5% CO₂ at 37°C for 7 days. The total number of colony forming units and the number of dark-pigmented colonies were counted on blood agar plates. Representative pigmented colonies were purified and identified using standard techniques including Gram stain, fermentation of glucose, production of indole from tryptophan, agglutination of 3% sheep erythrocytes. *A. actinomycetemcomitans* was identified on the basis
of the specific colony morphology on TSBV agar (star-like inner structure) and a positive catalase reaction and production of specific enzymes (Slots 1981).

**Statistical analysis**

The mean values of the clinical parameters for each subject were computed at each examination. For the microbiological analysis the presence or absence of a specific bacterium in a subject was used. Statistical significance for any possible differences between spouses was tested using the Wilcoxon test. An unbalanced repeated measures model analysis (BMDP5V) was carried out to study within-couple influences. $P$-values $\leq 0.05$ were considered statistically significant.

**RESULTS**

In 1987, 255 subjects were examined, of which 169 could be located in 1994. From the initial 32 married couples, 23 couples could be re-examined in 1994. The data of the present study were based on 23 married couples in whom the periodontal condition could be evaluated longitudinally for a period of 7 years. Evaluation of the demographic characteristics showed that in 1994 on the average the couples were married for at least 7 years (mean 10 years, range 7-14 years). In 1994 the mean age of the subjects was 29.1 years (range 23-32); males were older than the females, 30.2 and 28.1 years respectively ($p = 0.0005$). Table 1 shows the clinical condition of males and females both in 1987 and in 1994. Over this period of time the amount of plaque and bleeding on probing increased in both sexes, whereas the mean PD and the % of sites with a PD $\geq 5$ mm remained virtually unchanged. For males and females the amount of LA increased. In terms of mean LA, the males already had more destruction in 1987 than the females. This difference even more pronounced in 1994; the males not only had a higher mean LA, but also showed a higher % of sites with LA of $\geq 2$ mm.

In order to assess a possible influence of the periodontal condition of a spouse on that of the partner, the patient material was classified in the following way. In each couple, the partner with the highest score for mean LA in 1994 was

Table 1. Mean values (SD) of the clinical parameters in males and females: plaque index (PI),
classified as the diseased proband and the partner as the spouse. As a result, 5 females and 18 males were classified as the diseased proband. Table 2 presents the clinical condition of the diseased probands and their spouses in 1987 and 1994. It can be seen that in 1994, apart from the mean LA, the diseased probands had a higher % of sites with LA ≥ 2 mm as well as sites with a PD ≥ 5 mm. In 1987, the diseased probands, as defined by the situation in 1994, already had a worse periodontal condition compared to that of their spouses. Both the diseased probands and the spouses showed a decline in the periodontal condition between 1987 and 1994. Further analysis was carried out by computing the mean difference of the clinical parameters in 1987 and in 1994 for each couple.

Table 3 shows that the difference in mean LA and the % of sites with LA ≥ 2 mm increased. To study the within-couple influences, a repeated measures model analysis was carried out, using age gender and plaque score as covariates. This analysis showed that for loss of attachment, the variance of the differences between spouses is explained by the variance between couples. This result was found for both data of 1987 and 1994. It implies that differences occurring between spouses are of the same magnitude as differences occurring between all members of the population. This does not change between 1987 and 1994.
Table 2. Mean values (SD) of the clinical parameters in diseased probands and the spouses: plaque index (PI), bleeding on probing (BOP), pocket depth (PD) and loss of attachment (LA)

<table>
<thead>
<tr>
<th></th>
<th>1987</th>
<th>1994</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diseased probands</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PI</td>
<td>1.04 (0.49)</td>
<td>1.22 (0.41)</td>
<td>NS</td>
</tr>
<tr>
<td>BOP</td>
<td>0.75 (0.42)</td>
<td>1.12 (0.44)</td>
<td>0.0002</td>
</tr>
<tr>
<td>PD (mm)</td>
<td>3.20 (0.65)</td>
<td>3.31 (0.53)</td>
<td>NS</td>
</tr>
<tr>
<td>%sites PD ≥5 mm</td>
<td>11.00 (19.80)</td>
<td>10.41 (12.83)</td>
<td>NS</td>
</tr>
<tr>
<td>LA (mm)</td>
<td>0.38 (0.29)</td>
<td>0.90 (0.50)</td>
<td>&lt;0.00005</td>
</tr>
<tr>
<td>%sites LA ≥2 mm</td>
<td>9.58 (10.93)</td>
<td>20.72 (15.84)</td>
<td>0.0005</td>
</tr>
<tr>
<td><strong>Spouses</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PI</td>
<td>0.90 (0.41)</td>
<td>1.07 (0.33)</td>
<td>0.026</td>
</tr>
<tr>
<td>BOP</td>
<td>0.71 (0.25)</td>
<td>1.12 (0.34)</td>
<td>0.0001</td>
</tr>
<tr>
<td>PD (mm)</td>
<td>3.14 (0.20)</td>
<td>3.14 (0.40)</td>
<td>NS</td>
</tr>
<tr>
<td>%sites PD ≥5 mm</td>
<td>4.10 (3.37)</td>
<td>5.82 (10.74)</td>
<td>NS</td>
</tr>
<tr>
<td>LA (mm)</td>
<td>0.17 (0.10)</td>
<td>0.43 (0.23)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>%sites LA ≥2 mm</td>
<td>3.07 (2.41)</td>
<td>6.57 (7.65)</td>
<td>NS</td>
</tr>
</tbody>
</table>

1 significantly different from b: p = 0.001.
2 significantly different from c: p = 0.0006.
3 significantly different from d: p = 0.03.
4 significantly different from e: p < 0.0001.
5 significantly different from f: p = 0.0003.

Table 3. Mean differences (SD) of the clinical parameters between the diseased proband and the spouse of a couple: plaque index (PI), bleeding on probing (BOP), pocket depth (PD) and loss of attachment (LA)

<table>
<thead>
<tr>
<th></th>
<th>1987</th>
<th>1994</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PI</strong></td>
<td>0.13 (0.72)</td>
<td>0.31 (0.63)</td>
<td>NS</td>
</tr>
<tr>
<td><strong>BOP</strong></td>
<td>0.04 (0.61)</td>
<td>0.01 (0.50)</td>
<td>NS</td>
</tr>
<tr>
<td><strong>PD (mm)</strong></td>
<td>0.05 (0.74)</td>
<td>0.16 (0.58)</td>
<td>NS</td>
</tr>
<tr>
<td>%sites PD ≥5 mm</td>
<td>6.81 (20.72)</td>
<td>4.58 (15.84)</td>
<td>NS</td>
</tr>
<tr>
<td><strong>LA (mm)</strong></td>
<td>0.21 (0.25)</td>
<td>0.48 (0.49)</td>
<td>0.01</td>
</tr>
<tr>
<td>%sites LA ≥2 mm</td>
<td>6.51 (10.07)</td>
<td>14.15 (15.82)</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Microbiological evaluation revealed a relatively high subgingival prevalence of *A.*
actinomycetemcomitans (50%), P. gingivalis (67%) and Prevotella intermedia (61%) (Table 4). Statistical analysis showed that males harboured A. actinomycetemcomitans more often than females. Table 4 also presents the prevalence of these bacteria in the diseased probands and spouses. No differences were found between the diseased probands and the spouses. Table 4 also shows that the 23 A. actinomycetemcomitans positive subjects include 2 positive couples. Within the 31 P. gingivalis and 28 P. intermedia positive subjects 9 and 7 couples, respectively, were present.

Table 4. Prevalence of micro-organisms in 1994

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
<th>Diseased proband</th>
<th>Spouse</th>
<th>Couples 1 partner positive</th>
<th>Couples both partners positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. actinomycetemcomitans</td>
<td>16$^a$</td>
<td>7$^b$</td>
<td>13</td>
<td>10</td>
<td>19</td>
<td>2</td>
</tr>
<tr>
<td>P. gingivalis</td>
<td>15</td>
<td>16</td>
<td>17</td>
<td>14</td>
<td>22</td>
<td>9</td>
</tr>
<tr>
<td>P. intermedia</td>
<td>14</td>
<td>14</td>
<td>12</td>
<td>16</td>
<td>21</td>
<td>7</td>
</tr>
</tbody>
</table>

$^a$ significantly different from $^b$: $p = 0.02$.

To illustrate the lack of influences within couples, the individual data are presented of the diseased subjects with the worst periodontal condition and their partners (Table 5). This condition was defined as having 1 or more sites showing a PD $\geq$ 5 mm in conjunction with LA $\geq$ 4 mm. This condition was present in 10 out of the 46 subjects, of which only 2 subjects were in one couple (couple 2). All other spouses showed a more favourable periodontal
Table 5. Individual clinical and microbiological data of the subjects with the worst periodontal condition and their spouses. Both spouses of couple 2 belonged to the group with the worst periodontal condition. Presence (+) or absence (-) of *A. actinomycetemcomitans* (Aa), *P. gingivalis* (Pg) and *P. intermedia* (Pi) is indicated. Plaque index (Pil), bleeding on probing (BOP), pocket depth (PD) and loss of attachment (LA)

<table>
<thead>
<tr>
<th>Couple No.</th>
<th>Timespan relationship</th>
<th>Gender</th>
<th>Mean %PD≥5</th>
<th>Mean %LA≥2</th>
<th>LAmx</th>
<th>Aa</th>
<th>Pg</th>
<th>Pi</th>
<th>Spouse</th>
<th>Gender</th>
<th>Mean %PD≥5</th>
<th>Mean %LA≥2</th>
<th>LAmx</th>
<th>Aa</th>
<th>Pg</th>
<th>Pi</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10</td>
<td>F</td>
<td>1.8</td>
<td>5.4</td>
<td>7</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>M</td>
<td>15.3</td>
<td>11.5</td>
<td>2</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>13</td>
<td>M</td>
<td>6.0</td>
<td>32.0</td>
<td>4</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>M</td>
<td>7.2</td>
<td>16.4</td>
<td>3</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>14</td>
<td>F</td>
<td>6.3</td>
<td>17.0</td>
<td>4</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>F</td>
<td>51.8</td>
<td>14.3</td>
<td>3</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>+</td>
<td>-</td>
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<tr>
<td>5</td>
<td>11</td>
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<td>9.8</td>
<td>39.2</td>
<td>4</td>
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<td>-</td>
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<td>0</td>
<td>1</td>
<td>1</td>
<td>-</td>
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<td>+</td>
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<tr>
<td>6</td>
<td>12</td>
<td>M</td>
<td>10.7</td>
<td>21.4</td>
<td>4</td>
<td>-</td>
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<td>5.3</td>
<td>1.8</td>
<td>2</td>
<td>-</td>
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<td>-</td>
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<tr>
<td>7</td>
<td>7</td>
<td>M</td>
<td>26.1</td>
<td>27.5</td>
<td>5</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>F</td>
<td>16.1</td>
<td>21.4</td>
<td>3</td>
<td>-</td>
<td>+</td>
<td>+</td>
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<tr>
<td>8</td>
<td>12</td>
<td>M</td>
<td>36.9</td>
<td>43.5</td>
<td>5</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>M</td>
<td>1.8</td>
<td>1.8</td>
<td>1</td>
<td>+</td>
<td>-</td>
<td>+</td>
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<td>+</td>
<td>-</td>
<td>-</td>
<td>M</td>
<td>1.8</td>
<td>1.8</td>
<td>1</td>
<td>+</td>
<td>-</td>
<td>+</td>
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</table>
DEVELOPMENT OF PERIODONTITIS IN SPOUSES

condition. The most extreme situation was observed in couple 9. This couple consisted of the diseased subject with the most extensive periodontal destruction and the spouse with minimal periodontal breakdown. The diseased subject was positive for *P. gingivalis* and negative for *A. actinomycetemcomitans* and *P. intermedia* whereas the opposite was found in the spouse.

**DISCUSSION**

The main objective of the present study was to investigate the effect of cohabitation of spouses on the periodontal condition of each other. Recently it has been proposed that spouses of periodontitis patients have elevated risk of periodontitis due to transmission of putative periodontal pathogens from the diseased spouse to the partner (Von Troil-Lindèn et al. 1995). Several studies have now shown that transmission of periodontal bacteria between spouse can occur (Petit et al. 1993b, Saarela et al. 1993, Van Steenbergen et al. 1993, DiRienzo et al. 1994). However, information regarding the frequency of transmission remains limited. In the present study 16 males and 7 females harboured *A. actinomycetemcomitans* forming 19 couples in which 1 of the 2 subjects was positive and 2 couples in which both subjects were positive. This indicates that transmission of *A. actinomycetemcomitans*, i.e. isolation of the same clonal type from both spouses may have occurred in at the most 10% of the cases. In 22 couples 1 of the 2 subjects was colonized by *P. gingivalis* forming 9 positive couples. This demonstrates that transmission of *P. gingivalis* may have occurred in at most 41% of the cases. In the studies of Petit et al. (1993b), Saarela et al. (1993) and Van Steenbergen et al. (1993) it has been shown that *A. actinomycetemcomitans* or *P. gingivalis* isolates from spouses are not necessarily from the same clonal type. Therefore, the actual frequency of transmission of *A. actinomycetemcomitans* and *P. gingivalis* in the present study will be lower than the previously mentioned 10% and 41%. However, the date of the present study and the above-mentioned studies suggest that *P. gingivalis* seems to be more readily transmitted between spouses than *A. actinomycetemcomitans*.

The important question remains whether transmission of *A. actinomycetemcomitans*, *P. gingivalis* and other periodontal pathogens affects
the periodontal condition of individuals who become colonised. The results of
the present study fail to show that the periodontal condition of a spouse is
influenced by that of the partner, since the differences in periodontal condition
increased, instead of decreased, over time.

These results are in contrast to those of Von Troil-Lindèn et al. (1995). These
authors found that spouses of patients with advanced periodontitis have a
worse periodontal status than spouses of a control group who were married
with periodontally healthy subjects. Several factors may explain the differences
between the results of their study and the present results. For instance, this
could be the result of either a different periodontal condition of the diseased
probands, a longer duration of marriage (26 years compared to 10 years in the
present study) or differences in socio-economic circumstances. On the other
hand, due to the sample size and the degree of matching of the diseased and
the control group, the study of Von Troil-Lindèn et al. (1995) has its limitations.
The data of the present study are derived from a young population with a high
prevalence of periodontal destruction, living in a remote village deprived from
regular dental care. The baseline examination of 1987 was carried out in 255
subjects and included 32 married couples. An analysis of these data failed to
show a significant effect of marriage on the periodontal condition of the
spouses (data not shown). However, at that time, the couples were married for
only 3 years. In contrast, the evaluation of the effect of sibling relationship on
the periodontal condition showed a significant sibship effect for plaque, calculus
and loss of attachment (Van der Velden et al. 1993). This finding supported the
concept that periodontitis aggregates in families. The present longitudinal study
failed to demonstrate that 10 years of cohabitation has an effect on the
periodontal condition of spouses. In general, this result questions the
importance of a periodontally diseased subject as the source for the spread of
periodontitis.
REFERENCES


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