Journal of Clinical Periodontology
Official Journal of The European Federation of Periodontology
Founded by the British, Dutch, French, German, Scandinavian, and Swiss Societies of Periodontology

J Clin Periodontol 2008; 35: 576–583 doi: 10.1111/j.1600-051X.2008.01236.x

Journal of Clinical Periodontology

Clinical changes in periodontium during pregnancy and post-partum

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Gürsoy M, Pajukanta R, Sorsa T, Könönen E. Clinical changes in periodontium during pregnancy and post-partum. J Clin Periodontol 2008; 35: 576–583. doi: 10.1111/j.1600-051X.2008.01236.x.

Abstract

Background and Aim: Pregnancy has been presented to increase susceptibility to gingival inflammation. It is unclear whether pregnancy gingivitis exposes or proceeds to periodontitis. We examined longitudinally the severity of periodontal changes during pregnancy and post-partum, and compared the findings with an age-matched group of non-pregnant women.

Material and Methods: Thirty generally healthy, non-smoking women at an early phase of their pregnancy and 24 non-pregnant women as controls were recruited. The pregnant group was examined three times during pregnancy and twice during post-partum, and the non-pregnant group three times, once per subsequent month. At each visit, visible plaque index (VPI), bleeding on probing (BOP), probing pocket depth (PPD), and clinical attachment level (CAL) were measured from six sites per tooth.

Results: In the pregnant group, BOP and PPD increased simultaneously without relation to plaque between the first and second trimesters, and thereafter decreased during subsequent visits. No changes were detected in CAL during the study period. In the non-pregnant group, BOP stayed invariable during the follow-up and correlated with the amount of plaque. Neither periodontal pocket formation nor significant changes in attachment levels were observed.

Conclusion: Based on this study, changes in clinical parameters during pregnancy are reversible, indicating that pregnancy gingivitis does not predispose or proceed to periodontitis.

Key words: bleeding on probing; clinical attachment level; gingivitis; post-partum; pregnancy; probing pocket depth

Accepted for publication 25 February 2008

Periodontal health in pregnant women has been a topic of interest during the last four decades; however, the information available is, at least in part, controversial. Increased female hormone levels during pregnancy are suggested to be related to the increased susceptibility to gingival inflammation without a certain association with the amount of plaque (Silness & Löe 1964, Hugoson 1970, O'Neil 1979a, Raber-Durlacher et al. 1994), but reports on oral debris correlation have been reported as well (Cohen et al. 1969, Samant et al. 1976). On the other hand, one study failed to demonstrate any correlation between increased female hormones and increasing gingival inflammation (Jonsson et al. 1988). Perhaps the most common opinion among dental clinicians is that healthy gingiva stays usually unaffected, and thus pregnancy itself does not cause gingivitis, but dental plaque or calculus is required for initiation of gingival changes during pregnancy (Arafat 1974).

Periodontal changes associated with pregnancy have been studied in various ethnic populations (Malisa et al. 1993, Muramatsu & Takaesu 1994, Tilakaratne et al. 2000). The prevalence of pregnancy gingivitis varies widely, ranging from around 35% (Hasson 1960, Chaikin 1977) up to 100% (Löe & Silness 1963). Gingival bleeding tendency has been found to increase significantly from the first to the third trimesters (Löe & Silness 1963, Hugoson 1970, Tilakaratne et al. 2000,

Conflict of interest and source of funding statement

The authors declare that they have no conflict of interests.
This study was supported by research grants from the Finnish Dental Society Apollonia and the Finnish Women Dentists' Association (M. G.) as well as by grants from the Finnish Academy and the HUCH-EVO (TYH 5306, TYH 7114, and TI 020 Y0002). The primary author was the recipient of the year 2006 Colgate Palmolive's grant delivered by the Finnish Dental Society Apollonia.

Taani et al. 2003) along with simultaneous increase in oestrogen and progesterone levels, and the highest bleeding scores appearing between the first and second trimesters (Cohen et al. 1969, Samant et al. 1976, O'Neil 1979a). Furthermore, the severity of gingivitis has been described to increase significantly during the second and third trimesters (Samant et al. 1976), and gingival inflammation to decrease spontaneously 3 months after delivery (Löe & Silness 1963, Cohen et al. 1971, Tilakaratne et al. 2000).

Not only gingivitis but also increased periodontal pocket depths have been detected during pregnancy (Miyazaki et al. 1991, Raber-Durlacher et al. 1994, Taani et al. 2003). Nonetheless, it is still unclear whether pregnancy affects periodontal attachment levels or not, mainly because in most studies carried out on pregnant women the clinical attachment level (CAL) measurements were not included in the study protocol. When it was taken into account, no statistically significant differences could be assessed with respect to the mean probing attachment level (Raber-Durlacher et al. 1994, Taani et al. 2003) or the loss of attachment between pregnant and non-pregnant women (Tilakaratne et al. 2000). Certain levels of periodontal attachment loss, which do not completely revert after delivery, may occur (Cohen et al. 1969). In type-1 diabetes mellitus patients, pregnancy seems to increase periodontal destruction seen as significantly greater probing pocket depth (PPD) values and loss of CALs (Guthmiller et al. 2001).

Most data related to pregnancy gingivitis are from cross-sectional studies, making it difficult to evaluate the relationship between pregnancy and periodontal changes. In addition, the data comparison is complicated by the great variation in the study material and measurements. The heterogeneity in study populations, different clinical measurements used, and numbers of teeth examined, including certain index teeth only or all teeth with/without the third molars, is evident and complicates the comparative evaluation of earlier observations.

With this background, as part of a longitudinal comprehensive study series on pregnancy-related changes in the periodontium, the aim of the present study was to examine clinical periodontal changes during pregnancy and post-partum. The clinical parameters measured after lactation were compared with the clinical parameters of a matched group of non-pregnant women.

Material and Methods

The clinical examinations were performed between October 2002 and October 2006 in a dental clinic of the Kerava Municipal Health Care Center, Kerava, Finland. The research protocol was reviewed and approved by the Helsinki University Central Hospital Obstetrics and Gynecology Ethics Committee. All subjects were informed of the purpose and objectives of this study, and their consent was obtained.

Patient selection

Pregnant study group (Pr)

Thirty volunteers were recruited among pregnant women in a pre-natal clinic of the Kerava Municipal Health Care Center. The inclusion criteria were being at 10 ± 1 weeks of pregnancy, between 24–36 years of age, periodontally healthy (no history of previous periodontitis, presence of deepened pockets, profuse gingival inflammation, or poor oral hygiene), and a non-smoker or former smoker. Exclusion criteria were using systemic or topical anti-microbial/anti-inflammatory therapy within the previous 3 months, having a history of systemic disease, and poor oral hygiene and/or deep caries lesions, and/or remnant roots. According to the research protocol (Fig. 1), the women in the pregnant (Pr) group were asked to make three visits during pregnancy (Pr Ex I: at 12–14 weeks; Pr Ex II: at 25–27 weeks; and Pr Ex III: at 34–38 weeks of pregnancy) and two visits during post-partum (Pr Ex IV; at 4–6 weeks after delivery; and Pr Ex V: after lactation).

Non-pregnant control group (N-Pr)

Twenty-four volunteers for the non-pregnant control group (N-Pr) were selected from subjects who were asking for an appointment for the dental examination in the same dental clinic. They had the same inclusion and exclusion criteria as the Pr group except for being pregnant or lactating. In this group, non-smoking status was not strictly required, but recommended. We did not want to make a cross-sectional data collection for the

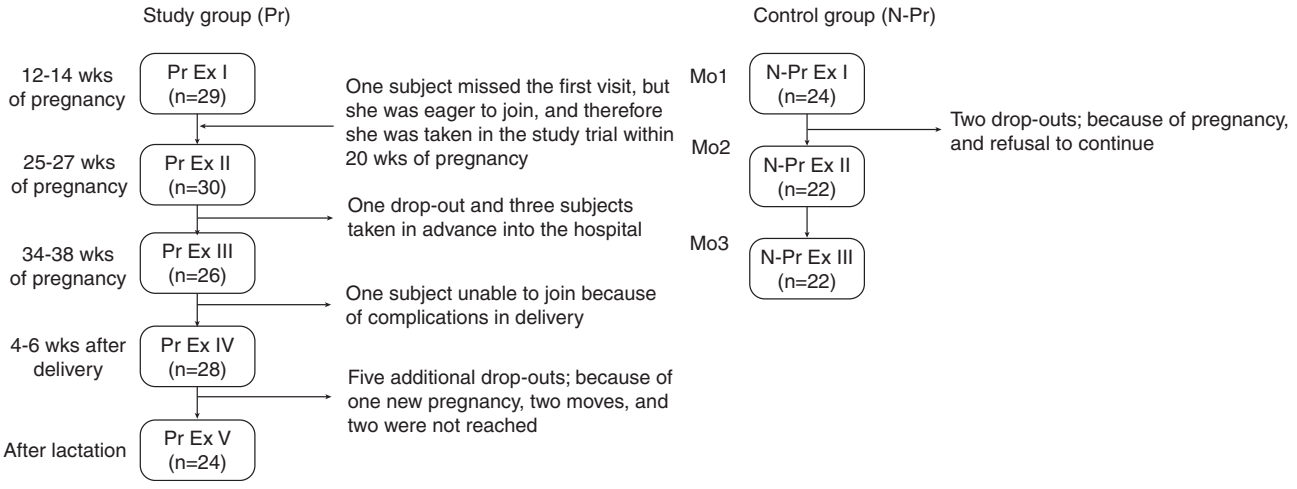

Fig. 1. Flow diagram presenting the derivation of the 30 pregnant women and the 24 non-pregnant women for this longitudinal study. Pr, pregnant study group; N-Pr, non-pregnant control group.

control group, because there might be acute changes in their periodontal clinical parameters. Therefore, according to the research protocol (Fig. 1), the N-Pr group was asked to make three visits (N-Pr Ex I, N-Pr Ex II, and N-Pr Ex III), once per subsequent month, to get stable mean values. Each visit was scheduled to be performed around the same time of the subject's menstrual cycle, excluding the menstruation days.

Initial procedures

All subjects in both groups filled a detailed questionnaire on their age, educational and professional levels, marital status, previous pregnancies, medical history, and previous dental care. Initial scaling and polishing procedures, when needed, were performed to reduce pre-existing gingival inflammation. The subjects received general oral hygiene instructions and were instructed not to rinse or brush with any anti-microbial compound, such as chlorhexidine or triclosan, but recommended to use regular toothpaste during the study trial.

Clinical measurements

All clinical measurements were generated by the same dentist (M. G.) from six sites (mesiobuccal, buccal, distobuccal, distolingual, lingual, and mesiolingual) per tooth, including third molars. During every visit, the periodontal status was identified using the following values: (A) visible plaque index (VPI) using a 0–1 measure, where 0 indicates no visible plaque and 1 the presence of dental plaque, (B) bleeding on probing (BOP) using a 0–2 measure, where 0 indicates no bleeding, 1 the presence of bleeding as a single point or thin line, and 2 the presence of profuse bleeding as an immediate flow, (C) PPD measured from the gingival margin to the most apical penetration point of the probe, and (D) CAL as the distance between the cemento-enamel junction and the base of the gingival sulcus. For measuring and recording BOP, each quadrant was individually probed separately starting with the quadrant's facial sites, and then continuing with the quadrant's oral sites. Probing and measuring BOP, PPD, and CAL were performed using a WHO probe (LM-Instruments Oy, Parainen, Finland) with a ball-tip end diameter of 0.5 mm. During the follow-up, the previous measurements were not available to the examiner.

Statistical analyses

For descriptive analyses, mean values and standard deviations were calculated based on the subject as the unit. Statistical analysis was performed using SPSS 14.0 (SPSS Inc., Chicago, IL, USA). Statistical differences within the groups were identified with the Friedman test followed by the Wilcoxon signed ranks test for comparing the differences between the follow-up visits. Furthermore, the Mann–Whitney test was used for the comparison between the groups. p-values <0.05 were considered as statistically significant.

Results

Out of the 30 participants in the Pr group, 24 (80%) completed the study period and 21 (70%) took part in all five visits. Reasons for non-participation at some visits were: failure to keep scheduled appointments because of earlier intake to the hospital ($n = 3$) or prolonged stay in hospital due to complication in delivery ($n = 1$), the examiner's inability to reach the participant ($n = 2$), having moved to other cities ($n = 2$), and new pregnancy ($n = 1$). Out of the 24 participants in the N-Pr group, 22 (92%) took part in all three visits. Reasons for two drop-outs were pregnancy ($n = 1$) and refusal to continue ($n = 1$). Figure 1 shows the derivation of the availability of the two groups for the present longitudinal study.

General characteristics

Various characteristics of the participants in the Pr and N-Pr groups are described in Table 1. The majority of the participants in the Pr group (77%) and in the N-Pr group (75%) had received 12–15 years of formal education, including comprehensive school with vocational school, upper secondary school, or polytechnic school grades. The mean age ± SD in the Pr group was 29.3 ± 2.8 years and in the N-Pr group was 30.4 ± 3.1 years. The mean number ± SD of erupted teeth per individual was 28.5 ± 1.4 and 28.6 ± 1.8, respectively.

Table 2 describes variables in general health and oral health behaviours of the subjects in the Pr and N-Pr groups. Half of the participants in the Pr group had allergy, and 83% were non-smokers and 17% former smokers, whereas the corresponding percentages among the participants in the N-Pr group were 29%, 71%, and 17%. Three subjects (13%) in the N-Pr group were smokers. The oral health care habits and regularity of visiting a dentist in the Pr and N-Pr groups matched well; ⩾90% of the subjects in both groups brushed their

Table 1. Various characteristics of the women in the pregnant (Pr) and non-pregnant (N-Pr) groups

	Pr group ($n = 30$), n (%)	N-Pr group ($n = 24$), n (%)
Age (years)		
Mean ± SD	29.3 ± 2.8	30.4 ± 3.1
Minimum	24	25
Maximum	35	36
Education level		
Basic	0 (0)	4 (17)
Secondary	18 (60)	10 (42)
Higher	12 (40)	10 (42)
Marital status		
Single/divorced	2 (7)	2 (8)
Cohabitant	11 (36)	14 (58)
Married	17 (56)	8 (33)
Employment status		
Working full time	23 (77)	13 (54)
Working partial time	1 (3)	2 (8)
Student	2 (7)	2 (8)
On child-care leave	3 (10)	6 (25)
Unemployed	1 (3)	1 (4)
Number of teeth		
Mean ± SD	28.5 ± 1.4	28.6 ± 1.8
Minimum	25	24
Maximum	32	32

N-Pr, non-pregnant control group; Pr, pregnant study group; SD, standard deviation.

Table 2. General and oral health behaviours for women in the pregnant (Pr) and non-pregnant (N-Pr) groups

	Pr group ($n = 30$), n (%)	N-Pr group ($n = 24$), n (%)
Allergies		
Yes	15 (50)	7 (29)
Smoking		
Non-smoker	25 (83)	17 (71)
Smoker	0 (0)	3 (13)
Former smoker	5 (17)	4 (17)
Tooth brushing frequency		
Once/day	3 (10)	1 (4)
Twice/day	27 (90)	22 (92)
More than twice/day	0 (0)	1 (4)
Tooth brushing		
Manual	23 (77)	16 (67)
Powered brush	7 (23)	8 (33)
Interdental cleaning		
Daily	2 (7)	9 (38)
Once or twice/week	10 (33)	5 (21)
Less than weekly	14 (47)	10 (42)
Never	4 (13)	0 (0)
Regular dental checkups		
Yes	23 (77)	20 (83)
Last dental checkup		
<1 year ago	8 (27)	7 (29)
1–2 years ago	15 (50)	13 (54)
>2 years ago	7 (23)	4 (17)

N-Pr, non-pregnant control group; Pr, pregnant study group.

Table 3. Various pregnancy-related characteristics of the women in the pregnant (Pr) and non-pregnant (N-Pr) groups

	Pr group ($n = 30$), n (%)	N-Pr group ($n = 24$), n (%)
Pregnant		
Yes	30 (100)	0 (0)
Previous pregnancies		
Yes	20 (67)	14 (58)
Pregnancy gingivitis during previous pregnancies		
Yes	5 (25)	7 (50)
Mode of delivery		
Vaginal delivery	28 (97)	–
Caesarean section	1 (3)	–
Pregnancy outcome		
Normal	24 (83)	–
Breech presentation	1 (3)	–
Pre-eclampsia	1 (3)	–
Premature birth <37 weeks	2 (7)	–
Stillbirth	1 (3)	–
Gestational age (in weeks)		
Mean $\pm$ SD	40.1 ± 2.1	–
Minimum	34	–
Maximum	42	–
Time of lactation (in weeks)		
Mean $\pm$ SD	38.7 ± 19.2	–
Minimum	8	–
Maximum	88	–

N-Pr, non-pregnant control group; Pr, pregnant study group; SD, standard deviation.

teeth twice a day and $>75\%$ of the subjects had regular dental checkups.

Table 3 describes pregnancy-related characteristics. More than half of the pregnant and non-pregnant control women had had one or more previous pregnancies. Over 80% of the women in the Pr group had a normal pregnancy outcome, with the mean gestational age of 40.1 ± 2.1 weeks. The mean weight of the newborns was 3605.4 ± 719.9 g and mean length was 50.1 ± 3.1 cm (data not shown).

Clinical parameters

The mean percentages of VPI and BOP, the latter including the total amount and the severity of gingival bleeding, in the Pr and N-Pr groups at every visit are presented in Fig. 2. In the Pr group, VPI decreased during the follow-up period but the percentages were always higher in relation to the corresponding mean values in the N-Pr group. The mean percentages of BOP were constantly higher in pregnant women than in non-pregnant women. Despite the significant ($p<0.01$) decrease in the VPI percentages in the Pr group between Pr Ex I and Pr Ex II, the highest BOP values ($p<0.01$) were recorded during the second trimester (Pr Ex II). A transient increase ($p<0.001$) in the severity of gingival bleeding, shown as profuse bleeding, was recorded during Pr Ex II as well. From the third trimester to the last post-partum visit, the mean BOP values decreased significantly, and during Pr Ex V the mean BOP percentages were similar to those in the N-Pr group (Fig. 2). The variation in the proportions of BOP in the Pr group is described in detail in Table 4.

The number of periodontal pockets (PPD $\geqslant 4$ mm) in pregnant women increased significantly ($p<0.001$) between Pr Ex I and Pr Ex II (Fig. 3). Most periodontal pockets found in the Pr group appeared in interproximal sites and in posterior regions. The peak of pocket formation occurred during the second trimester when 90% of the participants had gingival pockets (Table 4). After the second trimester, the number of pockets decreased again, visit by visit ($p = 0.08$, $p<0.001$, and $p<0.05$), and only 8% of the participants had one to two pockets during the last visit (Fig. 3 and Table 4). No gingival pockets over 4 mm were found during the follow-up. At the Pr Ex V visit, two women (7%) were found to have 1 mm of gingival recession buccally altogether in three teeth. No real loss in CAL was discovered during the follow-up. In the N-Pr group, CAL and PPD values, measured at three visits, remained stable.

One pyogenic granuloma, epulis gravidarum, was found (frequency 1:30; 3%) in a 30-year-old woman during the 20th week of pregnancy. The

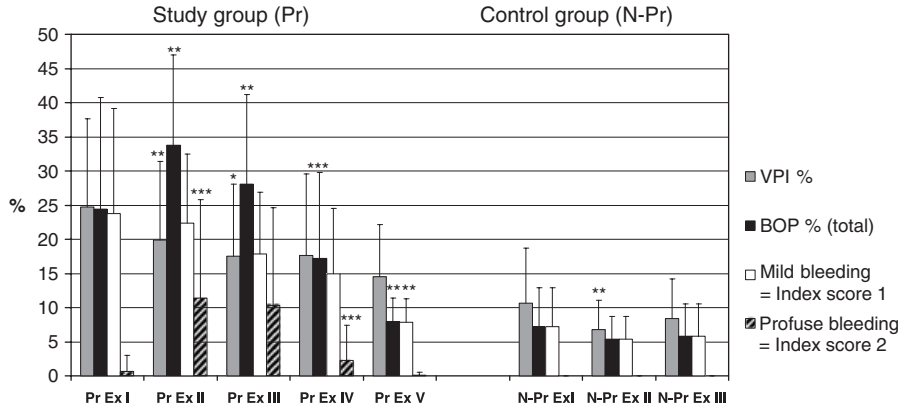

Fig. 2. Mean percentages of visible plaque index (VPI) and bleeding on probing (BOP) measured from six sites per tooth in pregnant (Pr) women at five visits and in non-pregnant (N-Pr) women at three visits. BOP is shown as the total amount of bleeding and according to the severity of gingival bleeding ($^{*}p<0.05$, $^{**}p<0.01$, $^{***}p<0.001$).

Table 4. Variation in the proportions of total bleeding on probing (BOP) (including Index score 1 and 2) and probing pocket depth (PPD) $\geqslant$4 mm in the pregnant (Pr) group during the study

Visits (number of women)	Number of women with BOP			Number (%) of women with PPD $\geqslant$4 mm, *n* (%)	Number of pockets per woman	
	BOP<20%, *n* (%)	BOP 20–31%, *n* (%)	BOP>31%, *n* (%)		mean	range
Pr Ex I (*n* = 29)	15 (52)	4 (14)	10 (35)	2 (7)	2.5	(1–4)
Pr Ex II (*n* = 30)	5 (17)	9 (30)	16 (53)	27 (90)	12.9	(1–48)
Pr Ex III (*n* = 26)	6 (23)	11 (42)	9 (35)	20 (77)	10.7	(1–42)
Pr Ex IV (*n* = 28)	21 (75)	3 (11)	4 (14)	7 (25)	8.6	(4–13)
Pr Ex V (*n* = 24)	24 (100)	–	–	2 (8)	1.5	(1–2)

BOP, bleeding on probing; PPD, probing pocket depth; Pr, pregnant study group.

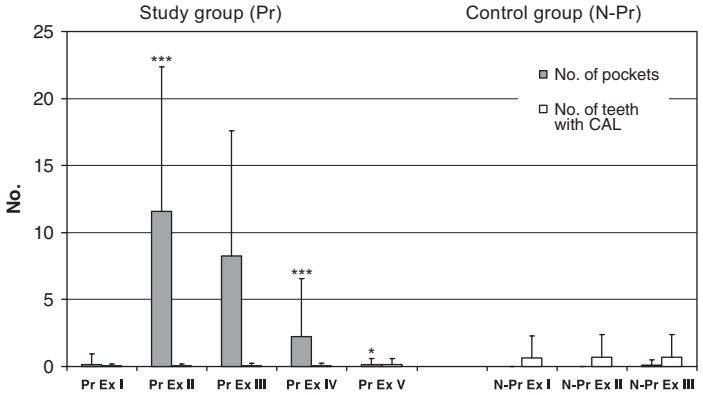

Fig. 3. Mean number of gingival pockets (probing pocket depth $\geqslant$4 mm) and teeth with loss of clinical attachment level (CAL) measured from six sites per tooth in pregnant (Pr) women at five visits and in non-pregnant (N-Pr) women at three visits ($^{*}p<0.05$, $^{***}p<0.001$).

granuloma, deep red in colour with small fibrin spots, was 7 mm in diameter, and was located with a short pedunculated base on the labial site of gingival papilla between the lower right central and lateral incisor.

Discussion

The results of the present longitudinal study confirm and further extend earlier reports and clinicians' observations that periodontal tissues are more susceptible to inflammation during pregnancy. Both the tendency and the severity of gingival bleeding and also PPD scores increased significantly in pregnant women, but returned to the same level as found in their matched non-pregnant controls after lactation. The present study showed a significant difference related to gingival inflammation between the periodontal conditions during pregnancy and post-partum.

The longitudinal study design with several follow-up visits is eventually the major strength of our study. The pregnant and non-pregnant women were recruited for this study from the same health care centre, and they represented similar study populations according to their age, ethnicity, socio-economical status, and oral care habits. The age range was limited to 24–36 years in order to have steady periodontal conditions without hormonal fluctuations other than pregnancy or emergence of periodontal disease, and none of the participants in either group had periodontitis. Therefore, the potential influence of these variables on their periodontal condition was avoided, thus increasing the strength of the results. In 2003–2004, the mean age for all parturients in Finland was around 30 years and for first-time mothers nearly 28 years (Vuori & Gissler 2004), which is in line with the mean age of the present study population. The recruitment of pregnant women for the study was somewhat difficult, mainly because of the demand of commitment for a long follow-up period. In recruiting the control group, the lack of common interest resulted in smaller group size than that in the Pr group. Because of

difficulties in recruiting participants to the control group, non-smoking status was not strictly required, but recommended. Three women in the control group smoked, but none >10 cigarettes/day. At the initial screening, one of the inclusion criteria was that they were periodontally healthy. After all, the number of participants in both groups was reasonable and comparable with that in previous longitudinal clinical studies, where the group size had varied within 16–47 participants (Cohen et al. 1969, O'Neil 1979a, b, Tilakaratne et al. 2000).

All clinical measurements during each visit were carried out by the same investigator in order to minimize the source of errors. In this study, we chose the WHO probe for clinical measurements because of its availability in the dental clinic. It has proven to be useful in measuring not only BOP but also PPD values, the light probe weight minimizing the potential misreading by overprobing (Ainamo et al. 1982). Before starting the study, the investigator was already familiar with using the WHO probe, which also increased the reliability of the results. While Osborn et al. (1990) compared the standard and the constant force (Florida Disc Probe) periodontal probes, the changeability between two examiners proved to be commonly higher than intraexaminer changeability. The only considered disadvantage of using the WHO probe in our study was the limited scale for CAL measurements.

In the present study, the greatest peak in the plaque scores appeared during the first trimester of pregnancy, then decreasing for each visit, the lowest VPI values being recorded after lactation. This finding differs from most previous reports. There may be minor effects, if any, due to the Hawthorne effect, because scaling and oral health instructions were given only once (during the recruitment), and the VPI values decreased for each visit during the 2-year follow-up. Instead, probably the main reason for this is due to various measurements for determining oral deposits. Silness & Löe (1964) reported in their cross-sectional study that the plaque index, measured from certain index teeth only, increased from the second month up to the eighth month of pregnancy. In contrast, in an experimental study by Raber-Durlacher et al. (1994), the plaque index appeared to be nearly identical at all phases during pregnancy and post-partum. Similar results were also reported from two longitudinal studies where the plaque index was measured from all teeth (Cohen et al. 1969, Tilakaratne et al. 2000). One explanation for high VPI percentages during the first trimester might be the nausea and vomiting during the first weeks of pregnancy. Most women in the Pr group reported that tooth brushing was nearly impossible, especially in premolar and molar areas because of the pregnancy-related nausea. Also, Taani et al. (2003) reported a significant connection between pregnancy-related vomiting and increased gingival inflammation in their cross-sectional study, and speculated that the main reason for this was impaired capability for proper brushing.

BOP is commonly used for measuring gingival inflammation, and the method has been proved to be a useful criterion when evaluating the absence or progression of periodontal disease (Lang et al. 1986, 1990, Joss et al. 1994). The present study revealed that the pregnant women exerted clearly elevated levels of gingivitis compared with the non-pregnant women. Our study confirmed that major changes in clinical parameters occur during the second trimester, appearing as pregnancy gingivitis. Notably, this condition developed regardless of the amount of plaque.

According to previous longitudinal follow-up studies (Lang et al. 1986, 1990, Joss et al. 1994), periodontal patients with a frequent mean value of BOP $\leqslant 20\%$ have diminished incidence for periodontal disease progress, whereas higher mean values of BOP correlate to increased levels of attachment loss and PPDs. Based on this observation, it authorized us to speculate on whether increased gingival bleeding during pregnancy is able to affect CALs. In the Pr group, the degree and severity of gingivitis was especially high during the second and third trimesters. A similar phenomenon was reported by Samant et al. (1976) in their cross-sectional study. In the Pr group, the increased mean BOP values ($\geqslant 30\%$) during pregnancy were associated with the simultaneous, considerable increase in PPD. Furthermore, the decrease in mean BOP values ($\leqslant 20\%$) during post-partum was associated with diminished prevalence of periodontal pockets. These results are in agreement with earlier studies by Lang and colleagues (1986, 1994), the only difference was that we did not find any correlation between gingival bleeding and CAL.

Most periodontal pockets found in the Pr group appeared in interproximal sites and in posterior regions, also seen in earlier studies (Löe & Silness 1963, Cohen et al. 1971). Our findings support the suggestion of Miyazaki et al. (1991) that the periodontal pocket formation during pregnancy occurs mainly due to gingival swelling rather than periodontal tissue breakdown, because the CALs among pregnant women remained unchanged and were comparable to those in the periodontally healthy non-pregnant group. Indeed, our study is in line with previous studies (Raber-Durlacher et al. 1994, Tilakaratne et al. 2000, Taani et al. 2003) showing that CALs remain unchanged during pregnancy. Overall, not a single site in the Pr group exerted characteristics to progress or transform to periodontitis during the follow-up.

The highest level of gingival inflammation together with elevated pocket depths was observed during the second and third trimesters, then reducing after delivery and lactation close to the levels seen in the control group. Therefore, we suggest that the gingival inflammatory changes detected in the Pr group are associated with a factor dependent on pregnancy. One potential explanation could be due to elevated levels of circulating oestrogen and progesterone, even though O'Neil (1979b) failed to prove the direct correlation between these hormonal elevations and increased gingival inflammation. However, elevated circulating progesterone levels have been suggested to contribute to enhanced gingival vascular permeability resulting in enhanced gingival exudates (Lindhe et al. 1969, Hugoson 1970). Sex hormones, especially progesterone but also oestradiol in higher concentrations, can increase the prostaglandin E_2 production by lipopolysaccharide-stimulated human monocytes (Miyagi et al. 1993), which is considered to increase the inflammatory reaction. Furthermore, a defensive neutrophil chemotactic responsiveness can be disturbed by elevated progesterone levels (Miyagi et al. 1992). In saliva and other oral fluid samples (mouth rinses and gingival crevicular fluid), the levels and especially the degree of activation of matrix metalloproteinases (MMPs) can differentiate periodontitis patients

from gingivitis patients (Lee et al. 1995, Sorsa et al. 2006). In our preliminary report (Latva-aho et al. 2005) on salivary levels of MMP-9 (gelatinase B) among these pregnant women, we found that the levels are consistent and stable during pregnancy, and also that the degree of activation of pro-MMP-9 remains unchanged. These salivary biomarker findings further support the conjecture that the periodontium of the pregnant women in the present study was affected by gingivitis but hardly by periodontitis. Also, a resolution of gingival inflammation and related elevated pocket depths occurred after delivery and lactation.

Our longitudinal data suggest that pregnancy does not necessarily result in irreversible periodontal breakdown but in reversible gingivitis without any loss of periodontal attachment.

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Clinical Relevance

Scientific rationale for the study: Periodontal changes were examined longitudinally during pregnancy and post-partum and compared with age-matched non-pregnant women.

Principal findings: Clinical indices demonstrated that pregnancy can cause reversible gingivitis without any loss of periodontal attachment.
Practical implications: Inflammatory changes during pregnancy are reversible, indicating that pregnancy gingivitis seems not to predispose or proceed to periodontitis.