J Clin Periodontol 2005; 32: 492–498 doi: 10.1111/j.1600-051X.2005.00703.x

Journal of Clinical Periodontology

Clinical risk factors associated with incidence and progression of periodontal conditions in pregnant women

Kevin L. Moss, James D. Beck and Steven Offenbacher

Department of Dental Ecology, School of Dentistry, University of North Carolina Chapel Hill, Chapel Hill, NC, USA

Moss KL, Beck JD, Offenbacher S. Clinical risk factors associated with incidence and progression of periodontal conditions in pregnant women. J Clin Periodontol 2005; 32: 492–498. doi: 10.1111/j.1600-051X.2005.00703.x. © Blackwell Munksgaard, 2005.

Abstract

Objectives: Few large studies have investigated the progression of periodontal conditions during pregnancy in a comprehensive manner. This study aimed to identify clinical factors that were predictive of incidence/progression of periodontal measures in pregnant women adjusting for relevant predictors.

Material and Methods: Periodontal examinations were conducted on 891 pregnant women prior to 26 weeks gestational age and within 48 h after delivery. Gingivitis/periodontitis incidence/progression (GPIP) was defined as four plus sites with 2+ mm increase in probing depth (PD) that resulted in PD of at least 4 mm at delivery. Multivariable models including relevant clinical variables and significant covariates were developed.

Results: While several clinical measures were significantly associated with the outcome, having ⩾10% of sites with bleeding on probing (BOP) and four plus sites with PD ⩾4 mm (PD4) were the best two predictors of GPIP (odds ratio (OR) = 2.8, 95% confidence interval (CI) = 1.8–4.2; OR = 2.0, 95% CI = 1.4–2.9, respectively), adjusting for maternal race, age, enrollment weight, smoking during pregnancy, marital status, food stamp eligibility, and private health insurance. Multivariable models assessed the impact of BOP on the PD4–GPIP relationship. PD4 was significant in the presence of BOP (low BOP OR = 1.3, 95% CI = 0.5–3.3; high BOP OR = 3.0, 95% CI = 2.2–4.3).

Conclusions: Enrollment BOP and PD4 were significant predictors of PD in pregnant women, however; PD4 is only a predictor with BOP.

Key words: bleeding on probing; disease incidence; disease progression; pregnancy; probing depth; risk factors

Accepted for publication 8 September 2004

There is an established literature on relationships between pregnancy and periodontal conditions (Ziskin et al. 1933, Maier & Orban 1949, Loe & Silness 1963, Silness & Loe 1964, 1966, Cohen et al. 1969, Cohen et al. 1971, Machuca et al. 1999, Tilakaratne et al. 2000, Laine 2002, Taani et al. 2003). For example, it is thought that pregnancy does not cause periodontal disease but may exacerbate pre-existing periodontal conditions (Laine 2002) and it has been shown that periodontal pockets increase in parallel with the increase in the stage of pregnancy (Taani et al. 2003). Thus, there is some agreement that women who are pregnant have a decline in periodontal health, as compared with women who are not pregnant. There also is a growing literature on the relationship between periodontal disease in pregnant women and premature birth, which includes low-birth weight for gestational age and foetal growth restriction (Offenbacher et al. 1996, 1998, 2001, Jeffcoat et al. 2001, 2003, Mitchell-Lewis et al. 2001, Davenport et al. 2002, Lopez et al. 2002, Boggess et al. 2003, Dasanayake et al. 2003). This more recent literature adds a different dimension to the potential consequences of increased levels of periodontal disease during pregnancy, which has led to a renewed interest among dental professionals in treating and preventing exacerbation of periodontal conditions during pregnancy.

Recent literature also has attempted to distinguish potential conceptual differences between defining periodontal disease when it is the object of oral treatment (i.e. a tooth or periodontal-

based therapeutic outcome) *versus* defining periodontal disease as it represents a potential exposure for another systemic condition (Beck & Offenbacher 2002). For example, when considering periodontal disease as an outcome, there is usually clinical interest in distinguishing between the destructive forms of the disease, periodontitis, from less destructive forms, i.e., gingivitis, or among types of periodontitis (aggressive *versus* chronic). This diagnosis aides in defining severity, prognosis and treatment options, as it relates to tooth and oral-centric outcomes. However, when considering periodontal disease as a systemic exposure, investigators must ascertain those aspects of periodontitis (i.e. signs and symptoms that convey risk for having the systemic outcome of interest, which may or may not differ from tooth-based outcomes).

We have reported that changes in periodontal probing depths (PDs) during pregnancy are related to premature births and to foetal growth restriction (having a weight for gestational age in the lowest 10%). Thus it is of clinical interest to assess individuals risk for PD change in order to treat and prevent that change during pregnancy. The aims of this study are to provide some information that is basic to assessing this risk by (a) to describing the patterns of increasing pocket depth measures at the site level according to baseline periodontal status and (b) determining at the person level, which baseline periodontal status measures are associated with incidence/progression of pocket depth, adjusting for relevant socio-demographic and behavioural characteristics.

Materials and Methods

This investigation is a part of the prospective study oral conditions and pregnancy (OCAP) aimed to examine the role of oral and vaginal infections and maternal cytokine responses on the incidence of pre-maturity and growth restriction. The OCAP study was carried out on pregnant women who were patients at the Duke University Hospital, Department of Obstetrics during the period December 1997–July 2001. The Duke University Medical Study Institutional Review Committee for Human Subjects approved this protocol. The methods for this project are described in more detail in a previous publication (Lieff et al. 2004) and are briefly presented here.

Subjects

Exclusion criteria included gestation greater than 26 weeks, multifoetal gestation (twins or greater), chronic hypertension, HIV/AIDS, pre-gestational diabetes, heart murmur, mitral valve prolapse or history of Phen-fen use without documentation of a clear echocardiogram, any medical conditions requiring antibiotic prophylaxis for dental treatment, age less than 18 years of age unaccompanied by a legal guardian and non-English speaking. Included in these analyses were the 891 subjects who had both a complete antenatal and postpartum dental examination completed. Subjects' ages ranged from 14 to 46 years.

Periodontal examinations

Full-mouth periodontal exams were performed at enrollment prior to 24 weeks gestational age and were generally repeated within 48 h of delivery and always by 72 h. Exams included all teeth present in the mouth (including third molars). A modified gingival index (Loe & Silness 1963 at one site per tooth), plaque score (Silness & Loe 1964) (one site per tooth), periodontal pocket depth (six sites per tooth), gingival recession (six sites per tooth) and bleeding on probing (BOP) (six sites per tooth). pocket depth and gingival recession scores were measured with a UNC-15 probe and rounded down to the nearest millimetre. The examinations were conducted by research dental hygienists who were trained and calibrated initially and at 1 year intervals during the study. Calibrations were conducted chair side and in hospital beds to replicate study conditions. Inter- and Intra-class correlations were calculated at each calibration session and were >0.9 for each examiner. weighted κ scores were above 85% and considered nearly perfect (Landis & Koch 1977).

Covariates

A combination of questionnaires and patient chart abstractions were administered to collect socio-demographic and behavioural variables that may influence change in periodontal status during pregnancy. Potential covariates included maternal race (African American, Caucasian and Other), maternal age in years, maternal weight in pounds at enrollment, being a first time mom (yes, no), having a previous pre-term baby (<37 week gestational age (yes, no)), smoke during pregnancy (yes, no), use of alcohol during pregnancy (yes, no), use of elicit drugs during pregnancy (yes, no), married (yes, no), income eligible to receive women and infant care (WIC) services or food stamps (yes, no), medical insurance (yes, no), and treated for sexually transmitted disease during pregnancy (yes, no). Bivariate analyses on race revealed that the ''Other'' group was small. Since Caucasians and ''Other'' had similar rates of incidence/progression, we combined the two groups to create a dichotomous variable for race containing African American and Other categories.

Outcome variable

A change in periodontal status during pregnancy occurs in two types of periodontal sites in the mouth – sites with previous evidence of disease and periodontally normal sites. In this study, a previously non-diseased site that increases at least 2 mm resulting in a periodontal pocket that was at least 4 mm postpartum was classified as having incident disease. A site exhibiting progression was defined as a site with a pocket at least 4 mm at baseline that increased an additional 2 mm during pregnancy. An individual exhibiting gingivitis/periodontitis incidence or progression (GPIP) was defined as having four plus sites with 2+ mm increase in PD that resulted in PD of at least 4 mm at delivery.

Figure 1 describes how the Incidence and Progression components were derived. Periodontal sites that had baseline scores of 0 or 1 mm, but did not change enough to have at least a 4 mm pocket were not counted as incident sites (A cells). Sites with 2 or 3 mm pockets at baseline that progressed to at least 4 mm at follow-up were considered to be Incident sites because their baseline scores were not considered diseased (B cells). Diseased sites at baseline (4 or 5 mm pockets) that increased at follow-up were considered to have progressed (C cells).

Statistical analyses

Both a person-based and a periodontal site-based data set were created for these analyses. Data were analyzed using SAS (v8.0, Research Triangle Park, NC, USA). Site-based analyses consisted

Baseline PD / PD Increase	0mm (n=7919)	1mm (n=70,021)	2mm (n=47,026)	3mm (n=13,500)	4mm (n=4,101)	5+mm (n=1,076)
2mm	A 571 (7.21%)	A 2193 (3.13%)	B 1358 (2.89%)	B 426 (3.16%)	C 37 (0.90%)	C 0 (0.0%)
3mm	A 51 (0.64%)	B 227 (0.32%)	B 248 (0.53%)	B 33 (0.24%)	C 6 (0.15%)	C 0 (0.0%)
4+mm	B 3 (0.04%)	B 22 (0.03%)	B 23 (0.05%)	B 7 (0.05%)	C 1 (0.01%)	C 0 (0.0%)

A Not Included in incidence/progression since resulting probing depth is less than 4mm

B Incidence

C Progression

B + C

Fig. 1. Incidence/progression of pocket depth by baseline probing depth and amount of progression, $N = 143{,}643$ sites.

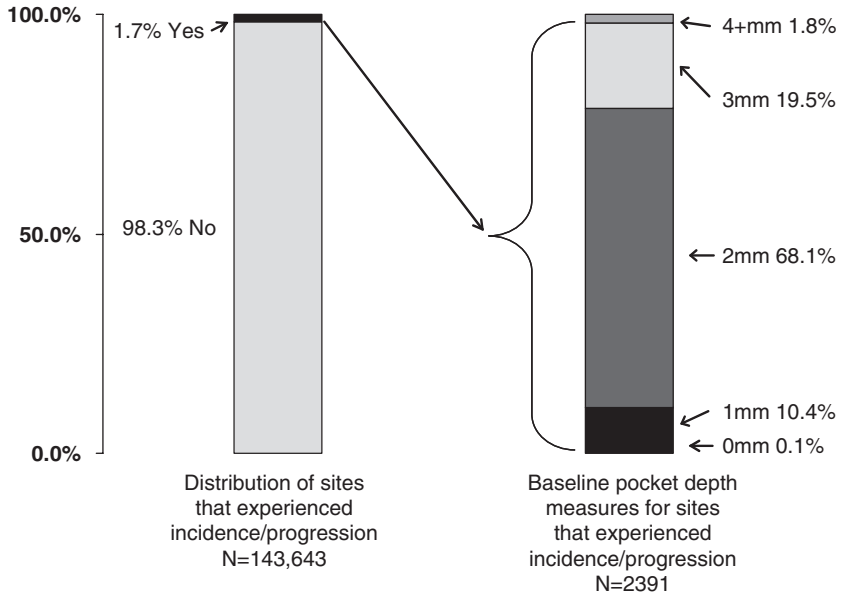

Fig. 2. Percent of all sites that experienced incidence/progression and baseline probing depth measures for those sites with incidence/progression.

entirely of frequency distributions that described GPIP patterns. For analyses stratified by other variables, chi-squared tests were used for dichotomous variables, and *t*-tests and GLM models with *post hoc* comparisons were used for variables with multiple categories. For person-based analyses, chi-squared and *t*-tests were used to test bivariate relationships between baseline measures and the outcome variable. Unconditional logistic models were used to derive the odds ratios (ORs) and 95% confidence intervals (CIs). Potential covariates were eligible for inclusion in the multivariable model based on significant a bivariate association with the outcome. *p*-Values $\leqslant 0.05$ were considered significant. Covariates that were bivariately statistically significant were included in the final model. Interactions between covariates and the association between the periodontal clinical variable and incidence/progression were evaluated. Any statistically significant interaction resulted in stratification of the model on that variable. Proc Logistic with the C statistic was used to estimate the area under an ROC curve.

Results

Overall, the number of sites that experienced GPIP was relatively small (2391% or 1.7% of all sites); however, the number of people with one or more sites that have GPIP of pockets was larger (46.0%).

Site-based analysis

As shown in the left histogram in Fig. 1, a total of 143,643 sites were examined antepartum and again postpartum. Of those sites, 2391 experienced incidence/progression during pregnancy (1.7%). The right side of Fig. 2 focuses only on sites that had incidence/progression. Here we see that sites with 2 mm sulcus depths comprised 68.1% of sites that experienced GPIP and sites with 3 mm sulcus depths accounted for another 19.5%. The vast majority of sites that changed were previously non-diseased sites (98.2%) in that the PD was 3 mm or less initially and therefore were incident events, rather than disease progression (1.8%). It also appears that BOP significantly increases the risk of incident events at each level of antenatal pocket depth, except for 0 mm sulcus depth where no sites bled (Fig. 3). However disease progression (4 mm pocket depth) simply exhibited a positive trend in relation to BOP. Figure 4 is composed of two analyses and indicates that GPIP occurs more often on premolars and molars than on anterior teeth and is more likely to occur on inter-proximal sites.

Person-based analyses

An individual exhibiting incidence/progression was defined as having four plus sites with 2+ mm increase in PD that resulted in PD of at least 4 mm at delivery. Since this definition does not contain a measure of destructive periodontitis (attachment loss or bone loss), we describe the changes that occurred as GPIP of their current periodontal status, rather than strictly as periodontal disease or periodontitis progression.

Bivariate analyses (Table 1) showed that 26.2% of women experienced a worsening of their periodontal status. Women who experienced GPIP of their periodontal status were more likely to be younger, weighed more, be African American, smoke tobacco, be unmarried, be WIC or food stamp eligible, and not have medical insurance. Clinical characteristics associated with GPIP included having 10% or more of their sites BOP, being in the top quartile of BOP, be a periodontal case at baseline (four plus sites with pocket depth

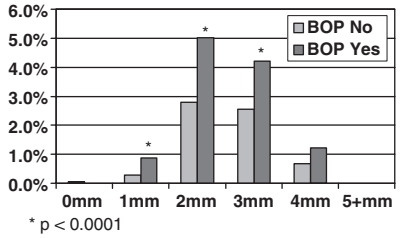

Fig. 3. Percentage of sites that experienced incidence/progression by baseline pocket depth and bleeding on probing, $N = 143{,}643$ sites.

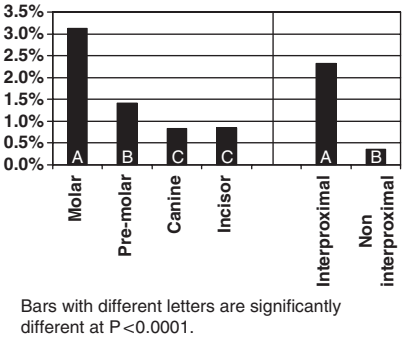

Fig. 4. Tooth type and location of sites that experienced incidence/progression, $N = 143{,}643$ sites.

Table 1. Baseline characteristics of OCAP subjects who experienced incidence/progression of their periodontal status

Variable	Periodontal incidence/progression?		
	no (<4 sites) $n = 658$ (73.9%)	yes (4+ sites) $n = 233$ (26.2%)	*p*-value
Maternal age (years, mean+SD)	28.7 (6.6)	27.1 (6.4)	<0.01
Maternal weight (pounds, mean+SD)	160.2 (43.5)	167.2 (45.2)	<0.05
African American	253 (61.9%)	156 (38.1%)	
Caucasian	367 (84.4%)	68 (15.6%)	
Other race	38 (80.9%)	9 (19.2%)	<0.0001
Smoke during pregnancy	93 (63.7%)	53 (36.3%)	
Did not smoke	565 (75.8%)	180 (24.2%)	<0.01
Alcohol	117 (79.1%)	31 (21.0%)	
No alcohol	541 (72.8%)	202 (27.2%)	NS
Illicit drugs	26 (66.7%)	13 (33.3%)	
No drugs	632 (74.2%)	220 (25.8%)	NS
Not married	281 (65.5%)	148 (34.5%)	
Married	377 (81.6%)	85 (18.4)%	<0.0001
WIC or food stamp eligibility	96 (59.3%)	66 (40.7%)	
No WIC or food stamp eligibility	562 (77.1%)	167 (22.9%)	<0.0001
No medical insurance	302 (65.4%)	160 (34.6%)	
Medical insurance	356 (83.0%)	73 (17.0%)	<0.0001
First birth	380 (72.4%)	145 (27.6%)	
Mulitiparous	278 (76.0%)	88 (24.0%)	NS
Previous pre-term delivery	103 (69.6%)	45 (30.4%)	
No previous pre-term delivery	555 (74.7%)	188 (25.3%)	NS
STD	92 (74.8%)	31 (25.2%)	
No STD	566 (73.7%)	202 (26.3%)	NS
Extent BOP ⩾10%	347 (64.1%)	194 (35.9%)	
Extent BOP <10%	311 (88.9%)	39 (11.1%)	<0.0001
Extent BOP quartile (75–100%)	114 (50.7%)	111 (49.3%)	
Extent BOP quartile (50–75%)	157 (71.0%)	64 (29.0%)	
Extent BOP quartile (25–50%)	185 (83.7%)	36 (16.3%)	
Extent BOP quartile (0–25%)	202 (90.2%)	22 (9.8%)	<0.0001
PD case (4+ sites w/PD ⩾4 mm)	158 (55.2%)	128 (44.8%)	
PD non-case (<4 sites w/PD ⩾4 mm)	500 (82.6%)	105 (17.4%)	<0.0001
PD group quartile (75–100%)	109 (49.5%)	111 (50.5%)	
PD group quartile (50–75%)	167 (73.2%)	61 (26.8%)	
PD group quartile (0–50%)	382 (86.2%)	61 (13.8%)	<0.0001

OCAP, oral conditions and pregnancy; WIC, women and infant care; STD, sexually transmitted disease; BOP, bleeding on probing; PD, probing depth.

⩾4 mm) and being in the top two quartiles of extent pocket depth ⩾4 mm. However, the last line of Table 1 shows that 50% of the women had no pockets of 4+ mm and 13.8% of them experienced incidence/progression.

We developed three unconditional logistic regression models of the potential clinical exposures and GPIP. In the first model (Table 2a), the clinical variable was extent of BOP at baseline, which was dichotomized at 10% of sites that bled. This variable was strongly related to incidence/progression, adjusting for other variables in the model (OR = 3.69, CI = 2.48–5.49). African American also had a significantly higher likelihood of experiencing incidence/progression. There were no significant interactions between any of the covariates and BOP. We also created groups based on quartiles of extent of BOP and used the lowest quartile as the reference group in order to examine the effect of increased extents of BOP on GPIP (analysis not shown). Adjusting for the same variables, quartiles of BOP showed increasing ORs for each quartile (Q1: referent group; Q2: OR = 1.48, CI = 0.82–2.67; Q3: OR = 2.87, CI = 1.64–5.03; Q4: OR = 6.94, CI = 4.01–12.00).

The clinical variable tested in the second model (Table 2b) was our pocket depth case definition: four plus sites with pocket depth ⩾4 mm at baseline. In this model, there was a significant interaction between race and pocket depth case status with African American race (OR = 3.48, CI = 2.08–5.84) and pocket depth case status (OR = 5.99, CI = 3.48–10.30) as significant main effects and the interaction between African Americans and PD case status (OR = 6.46, CI = 3.85–10.85) when compared with the Other race/not a PD case group. We then created two logistic models stratified by race. For African Americans the OR between pocket depth and GPIP was 1.9 (CI = 1.3–2.9) and for the ''Other'' group, the OR was 5.4 (CI = 3.1–9.5). We also created groups based on quartiles of extent of pockets ⩾4 mm (not shown). The lower two quartiles were combined since 49% of women had and extent score of zero. Adjusted ORs for quartiles 3 and 4 were 1.91 (CI = 1.27–2.89), 4.61 (CI = 3.08–6.92), respectively, indicating increasingly higher odds for GPIP for the third and fourth quartiles of pocket depth ⩾4 mm.

We then examined any potential interaction between BOP and PD case by creating a series of dummy variables with individuals having low BOP and low PD case as the referent group. Table 2c shows that both the high BOP–low PD and high BOP–high PD groups had significantly higher odds of experiencing GPIP than the referent group. Having a high level of pockets without high BOP is not significantly related to incidence/progression; however, there is a

Table 2. Unconditional logistic regression models of the associations between baseline clinical variables and incidence/progression of periodontal status

Variable	Odds ratio (lcl–ucl)
(a) Extent of baseline BOP ⩾10% and incidence/progression (n = 872)	
Extent BOP ⩾10%	3.69 (2.48–5.49)
Race (African American)	2.34 (1.57–3.48)
Maternal age (years) 5 years	0.99 (0.95–1.04)
Maternal weight (pounds) 10 lb	1.00 (0.96–1.03)
Smoke during pregnancy (yes)	1.40 (0.92–2.13)
Marital status (not married)	1.09 (0.71–1.68)
WIC or food stamps eligibile	1.34 (0.89–2.03)
Medical insurance (no)	1.03 (0.65–1.61)
(b) Baseline pocket depth case status and incidence/progression with race by case status interaction (n = 872)*	
Other race/not PD case	Referent
African American/not PD case	3.48 (2.08–5.84)
Other race/PD case	5.99 (3.48–10.30)
African American/PD case	6.46 (3.85–10.85)
Maternal age (years) 5 years	0.99 (0.93–1.05)
Maternal weight (pounds) 10 lb	1.00 (0.97–1.04)
Smoke during pregnancy (yes)	1.30 (0.85–1.98)
Marital status (not married)	1.12 (0.73–1.74)
WIC or food stamps eligible	1.22 (0.81–1.85)
Medical insurance (no)	1.15 (0.73–1.84)
(c) Interaction between baseline BOP and periodontal pocket case status (pocketing) and incidence/progression (n = 872)	
BOP low, pocketing low†	Referent
BOP low, pocketing high‡	2.37 (0.88–6.37)
BOP high, pocketing low§	2.93 (1.83–4.68)
BOP high, pocketing high¶	5.89 (3.69–9.40)
Race (African American)	2.08 (1.39–3.11)
Maternal age (years) 5 years	0.99 (0.94–1.05)
Maternal weight (pounds) 10 lb	1.00 (0.96–1.03)
Smoke during pregnancy (yes)	1.33 (0.87–2.03)
Marital status (not married)	1.12 (0.73–1.73)
WIC or food stamps eligible	1.26 (0.83–1.92)
Medical insurance (no)	1.02 (0.64–1.60)

*Pocket depth case = 4+ sites w/PD ⩾4 mm.
†Extent BOP <10% and <4 sites with pocket depth ⩾4 mm.
‡Extent BOP <10% and 4+ sites with pocket depth ⩾4 mm.
§Extent BOP ⩾10% and <4 sites with pocket depth ⩾4 mm.
¶Extent BOP ⩾10% and 4+ sites with pocket depth ⩾4 mm.
BOP, bleeding on probing; WIC, women and infant care; PD, probing depth.

positive trend (OR = 2.37, CI = 0.88–6.37). Conversely, individuals who have high levels of BOP with low level of pockets is related to GPIP (OR = 2.93, CI = 1.83–4.68). Having a high level of pockets and high BOP is the strongest of the relationships compared with having neither (OR = 5.89, CI = 3.69–9.40).

We also evaluated excess odds for GPIP associated with baseline gingival index and plaque scores. While both of these indices were related to incidence/progression, BOP and PD were better predictors when evaluated using the area under the ROC curve as an indicator. BOP was the best predictor with a C-statistic of 0.76, followed by PD at 0.74. The gingival index score was 0.70 with the C-statistic for plaque at 0.69.

Discussion

The outcome variable in this study was GPIP and it did not contain a measure of attachment loss or bone loss. Thus, it should not be thought of strictly as progression of periodontal disease or periodontitis. We emphasized changes in pocket depth because pockets theoretically are more relevant to systemic conditions and because once an individual is classified as having periodontitis, gingivitis is no longer emphasized. However, most study subjects who experienced GPIP also experienced attachment loss. For example, on average individuals who experienced GPIP also experienced 16.6% of their sites increase by one or more millimetres of attachment loss compared with 7.5% of sites in those not experiencing GPIP. Similarly, a 2+ mm change in attachment loss occurred in 6.1% of sites of those experiencing GPIP compared with 0.8% of sites of those not experiencing GPIP.

The evaluation of change in a clinical variable over time also should consider whether that change is ''true'' change or instead is likely because of intra-examiner measurement error. In order to provide a perspective on this issue, we used information from our examiner calibration training. If we consider the change in PD assessed by our gold standard examiner as ''true'' change, then we can compare change scores from each of our examiners with the ''true'' change. We found that the examiners differed by a mean of 0.03 mm from the gold standard examiner and the standard deviation was 0.8 mm. Thus, a change in PD of 2 m is about 2.5 times the examiners' variability (0.8) and thus a change of 2 mm is likely to be ''true'' change.

GPIP during pregnancy is much more likely to occur in sites that were not diseased at baseline (baseline pocket depths are <4 mm). However, at the person level, having four or more pockets of 4 mm or more elsewhere in the mouth increased the risk of incidence/progression. It appears that disease elsewhere in the mouth indicates that the individual is at increased risk for additional expression of disease. This pattern certainly implies that PD of 4 or more mm identify a subject at risk, but that all sites in the mouth should be followed in individuals with baseline disease. However, our findings also show that 13.8% (Table 1, last line) of pregnant women will exhibit incident disease during pregnancy without having existing periodontal pockets, indicating that monitoring pregnant women irrespective of their current periodontal status may be beneficial.

Existing pockets are not the only risk for GPIP. BOP at baseline is related to increased risk of GPIP at both the site level and the person level. At the person level, we also see a dose response type of pattern. This finding is consistent with BOP being a clinical indicator of an active disease process (Lang et al. 1986, 1990, Haffajee et al. 1991, Joss et al. 1994, Lang & Corbet 1995). Existing pockets ⩾4 mm combined with BOP increased the risk of GPIP of periodontal status for the mother (Table 2c). However we did not see a significant

relationship between existing pockets and GPIP in the presence of a low extent of BOP, likely because the disease process was not active at the time of the baseline exam. Conversely, BOP, even when there was a low level of pockets in the mouth (<four sites with 4+ mm), was significantly related to incidence/progression. Thus, existing PD as a predictor of incidence/progression appears to depend on the presence of BOP. This finding can be interpreted as being consistent with previous studies indicating that pocket depth and BOP were the best predictors of progression of periodontal disease with the caveat that those studies used attachment loss as their outcome, not pocket depth (a component of the attachment loss measure) (Lang et al. 1986, 1990, Goodson 1990, Haffajee et al. 1991).

Race was also a significant main effect in our models, indicating that African Americans were more likely to experience incidence/progression. Additional analyses (not shown) indicated that 47% of African Americans experienced GPIP compared with 20% for the ''Other'' group. In addition, 38% of African Americans had 4+ mm pockets at baseline compared with 16% for the ''Other'' group. However when we created two separate models of the association between baseline PD and incidence/progression, the association was weaker for African Americans (OR = 1.9, CI = 1.3–2.9) compared for the ''Other'' group (OR = 5.4, CI = 3.1–9.5). Thus, the race effect seen in the unstratified models appears to simply be because of African Americans having a greater overall incidence of periodontal conditions rather than baseline PD being a better predictor in African Americans. This observation is consistent with the increased incidence of periodontal disease progression in non-pregnant older African American adults, as previously reported (Beck et al. 1990, 1995).

Smoking was not significant in any of the models, even though it was significant as an unadjusted association in the bivariate analysis. However, smoking is known to have an effect on BOP and is a risk factor for periodontitis. Consequently, we conducted additional analyses of both the BOP – incidence/progression and the PD – incidence/progression relationships stratified by smoking status (not shown). The adjusted ORs and 95% confidence levels between high BOP and GPIP for non-smokers and smokers were 3.40 (CI = 2.20–5.33) and 5.40 (CI = 2.03–14.31), respectively. For high PD compared with low PD the associations with GPIP were 2.75 (CI = 2.20–5.33) in non-smokers and 2.87 (CI = 1.33–6.19) for smokers. Thus, it appears that while smoking results in lower BOP scores, those who exhibit high BOP scores have much greater odds of experiencing incidence progression. However, smoking status does not appear to greatly impact the PD – incidence/progression relationship.

The finding that the vast majority of periodontal activity during pregnancy was in the form of incident ''new'' disease rather than disease progression should not be surprising. Women in these age groups are not likely to have extensive disease, so it stands to reason that, if there is extensive disease activity during pregnancy, it is likely to involve healthy sites. This phenomenon also has been observed in studies of older populations containing men and women (Beck et al. 1995). Thus, when treating patients, following all sites, not just sites with pockets at baseline, is recommended.

The dental profession has long been concerned about maternal periodontal disease during pregnancy. Recent studies indicate that the consequences of periodontal disease activity during pregnancy may affect birth outcomes and that the level of disease activity does not necessarily have to result in attachment or bone loss. The findings from this study are consistent with earlier studies of attachment loss during pregnancy in that the extents of pocket depth and BOP early in pregnancy are indicative of PD changes that result in an increased number of pockets of 4 or more mm in depth to occur later in that pregnancy. Even if the newly developed periodontal pockets resolve after the pregnancy is over, it may be important to consider that preventing gingival inflammation during pregnancy may become a therapeutic goal. While it is known that periodontal treatment and preventive therapy can improve oral status during pregnancy, studies have not yet definitively demonstrated that these therapies will reduce adverse birth outcomes. Nevertheless, it may be useful to plan for such a contingency.

Acknowledgements

This study was funded by grant DE-012453 from the National Institutes of Health.

References

Beck, J. D., Koch, G. G. & Offenbacher, S. (1995) Incidence of attachment loss over 3 years in older adults – new and progressing lesions. *Community Dentistry and Oral Epidemiology* **23**, 291–296.

Beck, J., Koch, G. G., Rozier, R. & Tudor, G. (1990) Prevalence and risk indicators for periodontal attachment loss in a population of older community-dwelling blacks and whites. *Journal of Periodontology* **61**, 521–528.

Beck, J. & Offenbacher, S. (2002) Relationships among clinical measures of periodontal disease and their associations with systemic markers. *Annals of Periodontology* **7**, 79–89.

Boggess, K. A., Lieff, S., Murtha, A. P., Moss, K., Beck, J. & Offenbacher, S. (2003) Maternal periodontal disease is associated with an increased risk for preeclampsia. *Obsteric and Gynecology* **101**, 227–231.

Cohen, D., Friedman, L., Shapiro, J. & Kyle, G. (1969) A longitudinal investigation of periodontal changes during pregnancy. *Journal of Periodontology* **40**, 563–570.

Cohen, D., Shapiro, J., Friedman, L., Kyle, G. & Franklin, S. (1971) A longitudinal investigation of periodontal changes during pregnancy and fifteen months post-partum: part II. *Journal of Periodontology* **42**, 653–657.

Dasanayake, A. P., Russell, S., Boyd, D., Madianos, P. N., Forster, T. & Hill, E. (2003) Preterm low birth weight and periodontal disease among African Americans. *Dental Clinics of North America* **47**, 115–125, x–xi.

Davenport, E. S., Williams, C. E., Sterne, J. A., Murad, S., Sivapathasundram, V. & Curtis, M. A. (2002) Maternal periodontal disease and preterm low birthweight: case–control study. *Journal of Dental Research* **81**, 313–318.

Goodson, J. M. (1990) Selection of suitable indicators of periodontitis. In: Bader, J. (ed). *Risk Assessment in Dentistry*, pp. 69–74. Chapel Hill: University of North Carolina Dental Ecology.

Haffajee, A. D., Socransky, S. S., Lindhe, J., Kent, R. L., Okamoto, H. & Yoneyama, T. (1991) Clinical risk indicators for periodontal attachment loss. *Journal of Clinical Periodontology* **18**, 117–125.

Jeffcoat, M. K., Geurs, N. C., Reddy, M. S., Cliver, S. P., Goldberg, S. L. & Hauth, J. C. (2001) Periodontal infection and preterm birth: results of a prospective study. *Journal of the American Dental Association* **132**, 875–880.

Jeffcoat, M. K., Hauth, J. C., Geurs, N. C., Reddy, M. S., Cliver, S. P., Hodgkins, P. M. & Goldenberg, R. L. (2003) Periodontal disease and preterm birth: results of a pilot intervention study. *Journal of Periodontology* **74**, 1214–1218.

Joss, A., Adler, R. & Lang, N. P. (1994) Bleeding on probing. A parameter for monitoring periodontal conditions in clinical practice. *Journal of Clinical Periodontology* **21**, 402–408.

Laine, M. A. (2002) Effect of pregnancy on periodontal and dental health. *Acta Odontologica Scandanavica* **60**, 257–264.

Landis, J. R. & Koch, G. G. (1977) The measurement of observer agreement for categorical data. *Biometrics* **33**, 159–174.

Lang, N. P., Adler, R., Joss, A. & Nyman, S. (1990) Absence of bleeding on probing. An indicator of periodontal stability. *Journal of Clinical Periodontology* **17**, 714–721.

Lang, N. P. & Corbet, E. F. (1995) Periodontal diagnosis in daily practice. *Inernational Dental Journal* **45**, 3–15.

Lang, N. P., Joss, A., Orsanic, T., Gusberti, F. A. & Siegrist, B. E. (1986) Bleeding on probing. A predictor for the progression of periodontal disease? *Journal of Clinical Periodontology* **13**, 590–596.

Lieff, S., Boggess, K. A., Murtha, A. P., Jared, H. L., Madianos, P. N., Beck, J. & Offenbacher, S. (2004) The oral conditions in pregnancy study: periodontal status of a cohort of pregnant women. *Journal of Periodontology* **75**, 116–126.

Loe, H. & Silness, J. (1963) Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odontologica Scandanavica* **21**, 533–551.

Lopez, N. J., Smith, P. C. & Gutierrez, J. (2002) Periodontal therapy may reduce the risk of preterm low birth weight in women with periodontal disease: a randomized controlled trial. *Journal of Periodontology* **73**, 911–924.

Machuca, G., Khoshfeiz, O., Lacalle, J., Machuca, C. & Bullon, P. (1999) The influence of general health and socio-cultural variables on the periodontal condition of pregnant women. *Journal of Periodontology* **70**, 779–785.

Maier, A. & Orban, B. (1949) Gingivitis in pregnancy. *Oral Surgery* **2**, 334–373.

Mitchell-Lewis, D., Engebretson, S., Chan, J. M., Ib, L. & Papapanou, P. N. (2001) Periodontal infection and preterm birth: early findings from a cohort of young minority women in New York. *European Journal of Oral Sciences* **109**, 34–39.

Offenbacher, S., Katz, V., Fertik, G., Collins, J., Boyd, D., Maynor, G., McKaig, R. & Beck, J. (1996) Periodontal infection as a possible risk factor for preterm low birth weight. *Journal of Periodontology* **67**, 1103–1113.

Offenbacher, S., Lieff, S. & Beck, J. D. (1998) Periodontitis-associated pregnancy complications. *Prenatal and Neonatal Medicine* **3**, 82–85.

Offenbacher, S., Lieff, S., Boggess, K. A., Murtha, A. P., Madianos, P. N., Champagne, C. M., McKaig, R. G., Jared, H. L., Mauriello, S. M., Auten, R. L. Jr., Herbert, W. N. & Beck, J. D. (2001) Maternal periodontitis and prematurity. Part I: obstetric outcome of prematurity and growth restriction. *Annals of Periodontology* **6**, 164–174.

Silness, J. & Loe, H. (1964) Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. *Acta Odontologica Scandanavica* **22**, 121–135.

Silness, J. & Loe, H. (1966) Periodontal disease in pregnancy III. Response to local treatment. *Acta Odontologica Scandanavica* **24**, 747–759.

Taani, D. Q., Habashneh, R., Hammad, M. M. & Batieha, A. (2003) The periodontal status of pregnant women and its relationship with socio-demographic and clinical variables. *Journal of Oral Rehabilitation* **30**, 440–445.

Tilakaratne, A., Soory, M., Ranasinghe, A. W., Corea, S. M., Ekanayake, S. L. & de Silva, M. (2000) Periodontal disease status during pregnancy and 3 months post-partum, in a rural population of Sri-Lankan women. *Journal of Clinical Periodontology* **27**, 787–792.

Ziskin, D., Blackberg, S. & Stout, A. (1933) The gingivae during pregnancy. *Surgery Gynecology and Obstetrics* **57**, 719–726.

Address:
James D. Beck
Department of Dental Ecology
CB#7450
School of Dentistry
University of North Carolina
Chapel Hill, NC 27599
USA
E-mail: james_beck@unc.edu