Periodontal Health and Adverse Pregnancy Outcome in 3,576 Turkish Women

H. Uslu Toygar,* G. Seydaoglu,† S. Kurklu,‡ E. Guzeldemir,* and N. Arpak§

Background: Preterm low birth weight (PLBW) is a problem encountered worldwide. The many causes of low birth weight (LBW) and premature or preterm birth (PTB) vary among developing/industrialized countries. Few studies have been published, in part because of the paucity of subjects and the ethnic heterogeneity of the populations. Our goals were to correlate maternal periodontal disease with birth outcomes in a Turkish population and evaluate maternal periodontal health.

Methods: This study consisted of 3,576 Turkish women who gave birth within 24 hours of the onset of labor. The adjusted odds ratio was generated from various logistic regression models.

Results: Codes from the Community Periodontal Index of Treatment Needs (CPITN) were evaluated according to treatment need (TN). The TN-I rate was 24.2% (N = 866), the TN-II rate was 73.5% (N = 2,628), and the TN-III rate was 2.3% (N = 82). There were 566 (15.8%) adverse birth outcomes; the PTB with LBW rate was 4.2%; the PTB with normal birth weight (NBW) rate was 8.3%; the in term birth with LBW rate was 3.3%, and the in term birth with NBW rate was 84.2%. The overall PTB rate was 12.5% (N = 447), and the LBW rate was 7.5% (N = 269). The mean birth weight and weeks of gestation decreased as the CPITN level increased ($P < 0.001$ for both). The LBW rate was 4.6% in the TN-I group, 8.3% in the TN-II group, and 14.6% in the TN-III group. TPTB rates were 10.5%, 12.7%, and 26.8%, respectively, whereas adverse birth outcome rates were 12.0%, 16.6%, and 30.5%, respectively ($P < 0.01$ for all). TN-II and TN-III were independent risk factors for LBW, PTB, and abnormal births in regression analyses.

Conclusion: Maternal periodontal disease may be a risk factor for an adverse pregnancy outcome. *J Periodontol 2007;78:2081-2094.*

KEY WORDS

Birth; low birth weight; periodontal disease; preterm; risk factors.

* Department of Periodontology, Faculty of Dentistry, Baskent University, Ankara, Turkey.
† Department of Biostatistics, Faculty of Medicine, Cukurova University, Adana, Turkey.
‡ Private practice, Ankara, Turkey.
§ Department of Periodontology, Faculty of Dentistry, Ankara University, Ankara, Turkey.

Adequate birth weight is the most important factor for the survival, growth, and development of a newborn.[1] Lifelong health is determined primarily during intrauterine development even if the infant is exposed to environmental factors after birth. Preterm low birth weight (PLBW) is believed to negatively influence the ability to thrive. Low birth weight (LBW) is defined as a birth weight <2,500 g. Preterm or premature birth (PTB) is defined as delivery before 37 weeks' gestation. PTB and LBW pose major social and economic public health problems and are the major causes of neonatal mortality. PLBW infants who survive the neonatal period face a higher risk for several neurodevelopmental disorders, health problems (asthma, upper respiratory infections, and ear infections), and congenital anomalies.[2]

The increasing incidence of LBW and PTB has been attributed to many causes, and risk factors vary among developing populations and industrialized countries. Identified risk factors for LBW and PTB include older or younger maternal age; racial characteristics; low socioeconomic status; inadequate prenatal care; drug, alcohol, or tobacco abuse; hypertension; genitourinary tract infection; diabetes mellitus; multiple pregnancies; environmental, genetic, obstetric, maternal, and fetal factors; infections; and toxic exposure. Periodontal infection is considered the most important preliminary reason

doi: 10.1902/jop.2007.070092

for PTB and LBW,[3,4] although some authors[5] disagree. A low socioeconomic level, difficulty in accessing health services, smoking or alcohol consumption, carbohydrate-rich diets, and inadequate oral hygiene are some of the causes of periodontal disease.[6] In recent years, various periodontal diseases have received attention because of their infectious characteristics and their adverse effects on the outcome of pregnancy.

It has been postulated that a link between chronic infection and premature birth develops when microorganisms or their products and toxins, such as lipopolysaccharides, enter the uterine cavity via an ascending route in genitourinary tract infections or via the circulation in women with a non-genital infection. Those pathogens or their products can stimulate an inflammatory cascade characterized by the increased production of proinflammatory mediators, such as interleukin-1β and tumor necrosis factor-alpha (TNF-α). The production of those mediators eventually leads to an increase in prostaglandin synthesis, which, in turn, causes uterine muscle contraction, cervical dilation, and premature rupture of the membranes.[7]

Increasing evidence suggests that the molecular and cellular inflammatory effector pathways that underlie the pathogenesis of PTB also are involved in growth restriction and developmental problems.[8] Most preterm infants usually have a LBW. It is important to distinguish between PLBW and intrauterine growth retardation, which is difficult to diagnose. Offenbacher et al.[8] suggested that all mouse fetuses of dams challenged by *Campylobacter rectus* were affected by that infection, although in various degrees of severity, regardless of whether they exhibited fetal growth restriction.

The development of periodontal infection, LBW, and PTB are affected by many factors, and large study populations are essential for accurate statistical analysis and results. The aims of our study were to examine the relationship between maternal periodontal disease and LBW and PTB in a Turkish population and to investigate the periodontal status of pregnant women as a marker for future obstetric complications in infants.

MATERIALS AND METHODS

Study Population

The study population consisted of 3,576 Turkish Caucasian women who gave birth within 24 hours of the onset of labor at Ankara Zekai Tahir Burak Women's Public Health Hospital, Ankara, Turkey between January 1999 and December 2001. The study protocol was approved by the Ethics Committee. All subjects were informed about the purpose of the research and their medical diagnosis, and all subjects provided informed consent. The women selected for this study were those who were present and able to examined and interviewed in their hospital bed from Monday to Friday between 10:00 am and 4:00 pm.

Patients with a history of hypertension, diabetes, gestational diabetes, renal failure, placental or uterine abnormalities, other systemic or genetic diseases, or <18 natural teeth were excluded from the study. Only mothers with a singleton gestation were included in the study. Before her periodontal evaluation, each patient was interviewed about her sociocultural background, including age, economic and professional status, and level of education. The patients were also asked about the frequency of their dental and periodontal visits. For each patient, nausea and vomiting during pregnancy, smoking habits (before and during pregnancy), and toothbrushing frequency (before and during pregnancy) were recorded. The study subjects were asked whether they had received routine prenatal care; based on their answers they were divided into two groups: women who had received prenatal care at least two times and women who had not received prenatal care during pregnancy.

Maternal demographic and obstetric data (gestational age, number of previous pregnancies, types of prior deliveries, weights of previously delivered newborns, marital consanguinity, infections of the genitourinary tract or other infections during pregnancy, and health disorders requiring medication) were obtained from the patients' medical records. Estimated gestational age was based on the date of the last menstrual period, ultrasound examination, sequential physical examinations, and postnatal examination. Women for whom the gestational age was absent or questionable in their medical records were excluded. Each subject confirmed the content of her medical records.

Periodontal Evaluation

Periodontal examinations were performed in the maternity ward when the patients were resting after being positioned at the end of the bed to enable reproducible examinations. All periodontal examinations were performed by the same dentist, who was calibrated before each examination. All examinations were performed in sufficient artificial‖ light with the use of a dental mirror and a World Health Organization (WHO) periodontal probe.¶ Periodontal probing depth (PD) was measured on six surfaces (mesio-buccal, buccal, disto-buccal, disto-lingual, lingual, and mesio-lingual) of 10 Ramfjord[9] teeth using a double-ended WHO probe according to the Community Periodontal Index of Treatment Needs (CPITN).[10] The highest value of the six measurements for each tooth was recorded.

‖ J Morita, Osaka, Japan.
¶ J Morita.

Each sextant was assigned a code number reflecting the condition of the most severely affected site according to the following criteria: 0: no signs of disease; 1: gingival bleeding after gentle probing; 2: supra- or subgingival calculus/overhanging restoration margins; 3: PD 4 or 5 mm deep; and 4: PD ≥6 mm. The highest code of the sextant was recorded as the code of the patient. Then the subjects were grouped into treatment need (TN) categories according to the highest code number for any sextant. The definitions used for the TN categories were as follows: TN-0 = no treatment needed (code 0); TN-I = improvement in personal oral hygiene (code 1); TN-II = TN-I + scaling (codes 2 + 3); and TN-III = TN-I + TN-II + complex treatment (code 4).

Variables

Mothers. The subjects were divided into two groups by age (17 to 35 years and <17 years or >35 years). Patients in the latter group were considered to be at greater risk for complications during pregnancy and delivery.

The women were classified into three groups according to national education system categories: uneducated, low level of education, and high level of education.

Smoking habits were evaluated according to frequency and permanence: smoking during pregnancy (heavy smokers, >10 cigarettes per day; light smokers, ≤10 cigarettes per day; and non-smokers) and smoking before pregnancy (heavy smokers, light smokers, and non-smokers). Smoking habits also were evaluated in two main categories: smokers and non-smokers during and before pregnancy.

Toothbrushing habits were evaluated according to frequency before and during pregnancy. The first group (''brushing occasionally'') consisted of women who answered "no" or "sometimes," whereas the second group (''brushing regularly'') consisted of women who answered "yes; one or more times a day.''

Infants. Infants were classified according to the following characteristics.

Gestational week: PTB was defined as delivery <37 weeks' gestation.[11] In-term birth (ITB) was defined as delivery ≥37 weeks' gestation.

Birth weight: Birth weight was classified as LBW (<2,500 g) or NBW (>2,500 g).[11]

Gestational week and birth weight: Babies were classified as preterm with NBW (PNBW); in term with LBW (ILBW); preterm with LBW (PLBW), or in term with NBW (INBW).

Main outcome: Two outcomes were considered: abnormal births (AB = PTB + LBW) or normal births (INBW).

Statistical Analyses

Normality was checked for each continuous variable. Because the data were not distributed normally, an appropriate non-parametric test was chosen. Continuous variables between groups were analyzed with the one-way analysis of variance or the Kruskal-Wallis test. The χ^2 test was used for categorical variable analyses. A trend analysis was performed for CPITN levels. Three stepwise logistic regression models were performed for multiple analyses. In model 1, birth weight (categorized as NBW or LBW) was defined as a dependent variable. In model 2, gestation week (categorized as ITB or PTB) was defined as a dependent variable. In model 3, main outcome (categorized as INBW and total number of ABs) was defined as a dependent variable. Variables, such as age, level of education, type of birth, parity, prenatal care, smoking, toothbrushing habits, and CPITN code, which were found to be risk factors in univariate analyses, were defined as independent risk factors. A P value <0.05 was considered significant. Statistical analyses were performed with a statistical program.#

RESULTS

The demographics and descriptive data of the study population are presented in Table 1. The age of the subjects ranged from 14 to 40 years (mean: 25.7 ± 5.4), and 288 mothers (8.1%) were younger than 17 years of age or older than 35 years of age. The mean number of pregnancies was 2.2 ± 1.4 (range, 1 to 9). The mean number of weeks of gestation was 38.8 ± 2.0 (range, 25 to 44 weeks), and the mean birth weight of 3,576 infants was 3,219.4 ± 532.4 g (range, 625 to 5,000 g).

Table 2 shows the distribution of newborns according to the classifications of birth weight and week of gestation. There were 566 (15.8%) AB outcomes; the PTB with LBW rate was 4.2%; the PTB with NBW rate was 8.3%; the in term birth with LBW or small for gestational age (SGA) rate was 3.3%, and the in term birth with NBW rate was 84.2%. The overall PTB rate was 12.5% (N = 447; 8.3% were preterm infants weighing >2,500 g, and 4.2% were preterm infants with a LBW), and the LBW rate was 7.5% (N = 269).

The unadjusted odds ratios (OR) for prematurity risk factors are defined in Table 3. Only the education level and toothbrushing frequency were significant risk factors for LBW, PTB, and AB. However, cesarean birth may be a risk factor for LBW, PTB, and AB. Similar results were found when demographic and descriptive data of the pregnant women were compared to four classification groups of birth outcome (Table 4).

The demographic and descriptive data of the study subjects according to CPITN scores are presented in Table 5. The frequency of TN-II was higher in the at-risk age group, but the frequency of TN-III was higher in the normal age group (P <0.012). CPITN

Statistical Package for the Social Sciences, version 14.0, SPSS, Chicago, IL.

Table 1.

Demographic and Descriptive Data of the Study Population

		Study Subjects (n [%])
Age group (years)	Normal (17 to 35)	3,288 (91.9)
	Maternal age at risk (<17 or >35)	288 (8.1)
Education	Uneducated	232 (6.5)
	Low level	2,648 (74.0)
	High level	696 (19.5)
Consanguineous marriage	No	2,931 (82.0)
	Yes	645 (18.0)
Pregnancies (parity) (n)	1	1,316 (36.8)
	≥2	2,260 (63.2)
Nausea and vomiting	No	1,439 (40.2)
	Yes	2,137 (59.8)
Type of birth	Normal (vaginal)	3,008 (84.1)
	Cesarean	568 (15.9)
Prenatal care	No	1,588 (44.4)
	Yes	1,988 (55.6)
Smoking during pregnancy	No	2,924 (81.8)
	Light smoker	555 (15.5)
	Heavy smoker	97 (2.7)
Smoking prior to pregnancy	No	2,765 (77.3)
	Light smoker	502 (14.0)
	Heavy smoker	309 (8.7)
Toothbrushing during pregnancy	Occasionally	594 (16.6)
	Regularly	2,982 (83.4)
Toothbrushing prior to pregnancy	Occasionally	373 (10.4)
	Regularly	3,203 (89.6)

Table 2.

Classification of Newborns According to Birth Outcome

Classification	Newborns (n [%])
Weeks of gestation	
ITB	3,129 (87.5)
PTB	447 (12.5)
Birth weight	
NBW	3,307 (92.5)
LBW	269 (7.5)
Weeks of gestation and birth weight	
INBW	3,010 (84.2)
PNBW	297 (8.3)
ILBW	119 (3.3)
PLBW	150 (4.2)
Main outcome	
INBW	3,010 (84.2)
AB	566 (15.8)
Total	3,576 (100.0)

levels increased in uneducated women (P <0.0001) and in women with a low level of education (P <0.05); in mothers who had more than one pregnancy (P <0.0001), had no prenatal care (P <0.01), or brushed their teeth occasionally (P <0.0001); and in women who gave birth to infants with PNBW (P <0.028), ILBW (P <0.0005), or PLBW (P <0.003).

The unadjusted ORs for CPITN codes according to CPITN classifications are shown in Table 6. Although only CPITN code 4 subjects had risk factors for PTB, CPITN codes 2 + 3 and CPITN code 4 subjects had risk factors for LBW and AB. The subjects with CPITN codes 2 + 3 had a 1.8 (95% confidence interval [CI]: 1.3 to 2.6) times greater risk for LBW than did subjects with a CPITN code of 0 or 1. Subjects with CPITN code 4 were at 3.5 (95% CI: 1.7 to 7.0) times greater risk for LBW than were subjects with a CPITN code of 0 or 1. The OR of code 4 for all ABs was 3.2 (95% CI: 1.9 to 5.3).

The adjusted ORs for three multivariate logistic models for all mothers are shown in Table 7, in which the results of this study are summarized. TN-II and TN-III were independent risk factors for LBW, PTB, and AB after adjusting the factors which are related with CPITN and LBW, PTB, AB in univariate analyses such as age, level of education, type of birth, smoking, toothbrushing, number of pregnancies, and prenatal care. CPITN codes 2 + 3 (OR = 1.87; 95% CI: 1.31 to 2.66) and code 4 (OR = 3.56; 95% CI: 1.74 to 7.25) were markers for independent risk factors for LBW. Although CPITN codes 2 + 3 (OR = 1.21; 95% CI: 0.95 to 1.56) were not found to be a significant marker for independent risk factors for PTB, code 4 (OR = 2.96; 95% CI: 1.72 to 5.09) was a significant marker for independent risk factors for PTB. In model 3, CPITN codes 2 + 3 and code 4 were markers of independent risk factors for AB (OR = 1.44; 95% CI: 1.14 to 1.81 and OR = 3.14; 95% CI: 1.86 to 5.29, respectively).

Similar results were found in separate multivariate analyses performed for the mothers who had prenatal care and for those who had no prenatal care (Tables 8 and 9). Only code 4 was not a significant risk factor for PTB in mothers who had not had prenatal care (OR = 2.1; 95% CI: 0.8 to 5.2; P <0.114).

Figure 1 shows the relationship between CPITN score and the birth weight and gestational week. The CPITN scores increased, whereas the mean number of weeks of gestation and the birth weight decreased (P <0.0001 and P <0.0001, respectively).

Table 3.

Unadjusted OR for Prematurity Risk Factors

	Birth Weight (g)		Gestation (weeks)		Main Outcome	
	≥2,500 (n)	<2,500 (n [%])	≥37 (n)	<37 (n [%])	INBW (n)	AB (n [%])
Age group (years)						
Normal (17 to 35)	3,040	248 (7.5)	2,872	416 (12.7)	2,767	521 (15.8)
At risk (<17 or >35)	267	21 (7.3)	257	31 (10.8)	243	45 (15.6)
OR (95% CI)		0.9 (0.6 to 1.5)		0.8 (0.5 to 1.2)		0.9 (0.7 to 1.3)
Education level						
Uneducated	211	21 (9.1)	191	41 (17.7)	181	51 (22.0)
Low level	2,439	209 (7.9)	2,321	327 (12.3)	2,228	420 (15.9)
High level (reference)	657	39 (5.6)	617	79 (11.4)	601	95 (13.6)
OR (95% CI) (for low education)		1.44 (1.0 to 2.9)*		1.1 (1.8 to 1.4)		1.2 (0.9 to 1.5)
OR (95% CI) (for uneducated)		1.7 (0.9 to 3.0)		1.7 (1.1 to 2.6)†		1.8 (1.2 to 2.7)†
Consanguineous marriage						
No	2,718	213 (7.3)	2,567	364 (12.4)	2,474	457 (15.6)
Yes	589	56 (8.7)	562	83 (12.9)	536	109 (16.9)
OR (95% CI)		1.2 (0.8 to 1.6)		1.0 (0.8 to 1.3)		1.1 (0.8 to 1.3)
Pregnancy (parity) (n)						
1	1,192	124 (9.4)	1,144	172 (13.1)	1,089	227 (17.2)
≥2	2,115	145 (6.4)	1,985	275 (12.2)	1,921	339 (15.0)
OR (95% CI)		0.6 (0.5 to 0.8)*		09 (0.7 to 1.1)		0.8 (0.7 to 1.0)
Nausea and vomiting						
No	1,332	107 (7.4)	1,249	190 (13.2)	1,204	235 (16.3)
Yes	1,975	162 (7.6)	1,880	257 (12.0)	1,806	331 (15.5)
OR (95% CI)		1.0 (0.7 to 1.3)		0.9 (0.7 to 1.1)		0.9 (0.7 to 1.1)
Prenatal care						
No	1,467	121 (7.6)	1,387	201 (12.7)	1,330	258 (16.2)
Yes	1,840	148 (7.4)	1,742	246 (12.4)	1,680	308 (15.5)
OR (95% CI)		0.9 (0.7 to 1.2)		0.9 (0.6 to 1.1)		0.9 (0.7 to 1.1)
Type of birth						
Normal	2,833	175 (5.8)	2,670	338 (11.2)	2,583	425 (14.1)
Cesarean	474	94 (16.5)	459	109 (19.2)	427	141 (24.8)
OR (95% CI)		3.2 (2.4 to 4.2)*		1.9 (1.4 to 2.3)*		2.0 (1.6 to 2.4)*
Smoking prior to pregnancy						
No	2,567	198 (7.2)	2,425	340 (12.3)	2,341	424 (15.3)
Yes	740	71 (8.8)	704	107 (13.2)	669	142 (17.5)
OR (95% CI)		1.2 (0.9 to 1.7)		1.1 (0.8 to 1.4)		1.2 (0.9 to 1.4)
Smoking during pregnancy						
No	2,715	209 (7.1)	2,565	359 (12.3)	2,475	449 (15.4)
Yes	592	60 (9.2)	564	88 (13.5)	535	117 (17.9)
OR (95% CI)		1.3 (0.9 to 1.7)		1.1 (0.8 to 1.4)		1.2 (0.9 to 1.5)
Toothbrushing prior to pregnancy						
Regularly	2,978	225 (7.0)	2,823	380 (11.9)	2,719	484 (15.1)
Occasionally	329	44 (11.8)	306	67 (18.0)	291	82 (22.0)
OR (95% CI)		1.7 (1.2 to 2.4)*		1.6 (1.2 to 2.1)*		1.5 (1.2 to 2.0)*
Toothbrushing during pregnancy						
Regularly	2,775	207 (6.9)	2,630	352 (11.8)	2,533	449 (15.1)
Occasionally	532	62 (10.4)	499	95 (16.0)	477	117 (19.7)
OR (95% CI)		1.5 (1.1 to 2.1)*		1.4 (1.1 to 1.8)*		1.3 (1.1 to 1.7)*
Total (N)	3,307	269	3,129	447	3,010	566

* $P < 0.05$.
† $P < 0.01$.

Table 4.

Characteristics of Mothers According to Birth Outcome

		INBW (n)	PNBW (n [%])	ILBW (n [%])	PLBW (n [%])
Age group (years)	Normal (17 to 35)	2,767	273 (8.3)	105 (3.2)	143 (4.3)
	At risk (<17 or >35)	243	24 (8.3)	14 (4.9)	7 (2.4)
Education	Uneducated	181	30 (12.9)*	10 (4.3)	11 (4.7)
	Low level	2,228	211 (8.0)	93 (3.5)	116 (4.4)
	High level	601	56 (8.0)	16 (2.3)	23 (3.3)
Consanguineous marriage	No	2,474	244 (8.3)	93 (3.2)	120 (4.1)
	Yes	536	53 (8.2)	26 (4.0)	30 (4.7)
Pregnancy (parity) (n)	1	1,089	103 (7.8)	55 (4.2)	69 (5.2)
	≥2	1,921	194 (8.6)	64 (2.8)	81 (3.6)
Nausea and vomiting	No	1,204	128 (8.9)	45 (3.1)	62 (4.3)
	Yes	1,806	169 (7.9)	74 (3.5)	88 (4.1)
Type of birth	Normal	2,583	250 (8.3)	87 (2.9)	88 (2.9)
	Cesarean	427	47 (8.3)	32 (5.6)†	62 (10.9)†
Prenatal care	No	1,330	137 (8.6)	57 (3.6)	64 (4.0)
	Yes	1,680	160 (8.0)	62 (3.1)	86 (4.3)
Smoking prior to pregnancy	No	2,341	226 (8.2)	84 (3.0)	114 (4.1)
	Yes	669	71 (8.8)	35 (4.3)	36 (4.4)
Smoking during pregnancy	No	2,475	240 (8.2)	90 (3.1)	119 (4.1)
	Yes	535	57 (8.7)	29 (4.4)	31 (4.8)
Toothbrushing prior to pregnancy	Regularly	2,719	259 (8.1)	104 (3.2)	29 (7.8)†
	Occasionally	291	38 (10.2)	15 (4.0)	121 (3.8)
Toothbrushing during pregnancy	Regularly	2,533	242 (8.1)	97 (3.3)	40 (6.7)†
	Occasionally	477	55 (9.3)	22 (3.7)	110 (3.7)
Total		3,010	297 (8.3)	119 (3.3)	150 (4.2)

* $P < 0.05$.
† $P < 0.01$ compared to INBW (each group compared only to INBW).

DISCUSSION

Our results provided strong evidence that maternal periodontal disease is an independent risk factor for an adverse outcome of pregnancy. In this study, the periodontal status of a large and demographically homogeneous population of Turkish women who recently delivered an infant was evaluated. CPITN codes were used to evaluate subjects during their immediate postpartum hospitalization. The major advantages of CPITN are its simplicity, speed, and international uniformity. Its limitations include partial recording, exclusion of some important signs of past periodontal breakdown (notably attachment loss), and the presence of any marker of disease activity or susceptibility.[10,12] Miyazaki et al.[13] used CPITN in their study, which included 1,565 non-pregnant women and 2,424 pregnant women.

One of the principal goals of this study was to describe the periodontal treatment needs of mothers soon after delivery in a Turkish population. With this information, appropriate oral care services can be planned for populations and individuals. Identification of the needs provides an essential basis for promoting primary health care programs and monitoring their success in improving the periodontal health of this population. The results of this study also underscore the need for periodontal screening before and during pregnancy.

In our study, ages were classified as 17 to 35 years (ages at which uncomplicated delivery is the norm) and <17 or >35 years (ages at which complications during or immediately after pregnancy are more common). The mean age of the mothers studied was 25.7 ± 5.4 years. Yalcin et al.[14] evaluated 61 Turkish women (mean age, 23.62 ± 4.01 years). We also examined the periodontal status of the at-risk mothers to evaluate the effects of maternal age on an adverse outcome of pregnancy. The results of

Table 5.

Characteristics of Pregnant Women by Treatment Needs and CPITN Code

		CPITN (n [%])			
		TN-I (codes 0 + 1)	TN-II (codes 2 + 3)	TN-III (code 4)	*P*
Age (years)	Normal (17 to 35)	817 (24.8)	2,394 (72.8)	77 (2.4)	
	At risk (<17 or >35)	49 (17.0)	234 (81.3)	5 (1.7)	0.012
Education	Uneducated	44 (19.0)	179 (77.1)	9 (3.9)	0.0001
	Low level	628 (23.7)	1,964 (74.2)	56 (2.1)	0.053
	High level	194 (27.9)	485 (69.7)	17 (2.5)	Reference
Consanguineous marriage	No	697 (23.8)	2,163 (73.8)	71 (2.4)	
	Yes	169 (26.2)	465 (72.1)	11 (1.7)	0.125
Pregnancy (parity) (n)	1	381 (29.0)	911 (69.2)	24 (1.8)	
	≥2	485 (21.5)	1,717 (76.0)	58 (2.6)	0.0001
Nausea and vomiting	No	347 (24.1)	1,050 (73.0)	42 (2.9)	
	Yes	519 (24.3)	1,578 (73.8)	40 (1.9)	0.442
Type of birth	Normal	742 (24.7)	2,199 (73.1)	67 (2.2)	
	Cesarean	124 (21.8)	429 (75.5)	15 (2.6)	0.127
Prenatal care	No	347 (21.9)	1,209 (76.1)	32 (2.0)	
	Yes	519 (26.1)	1,419 (71.4)	50 (2.5)	0.016
Smoking during pregnancy	No	729 (24.9)	2,124 (72.6)	71 (2.4)	
	Yes	137 (21.0)	504 (77.3)	11 (1.7)	0.115
Smoking prior to pregnancy	No	696 (25.2)	2,002 (72.4)	67 (2.4)	
	Yes	170 (21.0)	626 (77.2)	15 (1.8)	0.222
Toothbrushing during pregnancy	Occasionally	113 (19.0)	457 (76.9)	24 (4.0)	
	Regularly	753 (25.3)	2,171 (72.8)	58 (1.9)	0.0001
Toothbrushing prior to pregnancy	Occasionally	76 (20.4)	279 (74.8)	18 (4.8)	
	Regularly	790 (24.7)	2,349 (73.3)	64 (2.0)	0.005
Weeks of gestation and birth weight	INBW	762 (25.3)	2,191 (72.8)	57 (1.9)	Reference
	PNBW	64 (21.5)	220 (74.1)	13 (4.4)	0.028
	ILBW	13 (10.9)	103 (86.6)	3 (2.5)	0.0005
	PLBW	27 (18.0)	114 (76.0)	9 (6.0)	0.003
Total		866 (24.2)	2,628 (73.5)	82 (2.3)	

our study demonstrated that age is not a risk factor for adverse outcome of pregnancy in systemically healthy subjects. Ziadeh and Yahaya[15] evaluated outcomes of pregnancy in older mothers and suggested that the rates of preterm delivery, fetal death, LBW, and SGA infants among women aged ≥40 years were similar to those found in younger controls, regardless of parity. These results support the findings of our study.

Intrauterine growth restriction affects the risk for PTB. Impaired growth is associated with maternal conditions (e.g., hypertension) that lead to medically indicated PTB, and growth restriction is itself a common indication for induction before term.[16] Common social and demographic risk factors for PTB differ for SGA PTBs and non-SGA PTBs. Zeitlin et al.[16] suggested that maternal age >35 years, smoking during pregnancy, and low or high maternal body mass index are greater risk factors for SGA PTB than for other types of PTB. Because we were not able to obtain the body mass index of our subjects, we did not evaluate that factor.

Data on whether multiparity is a risk factor for negative fetal outcome are inconsistent. Although some investigators observed a positive relationship between LBW and high parity, others failed to do so.[17,18] Aliyu et al.[19] analyzed data on 22,463,141 mothers who had experienced at least one previous

Table 6.

Unadjusted ORs for CPITN Codes According to Classifications

Classification	TN-I (codes 0 + 1)	TN-II (codes 2 + 3)	TN-III (code 4)
Weeks of gestation			
In-term normal birth (n)	775	2,294	60
PTB (n [%])	91 (10.5)	334 (12.7)	22 (26.8)
OR (95% CI)	Reference	1.2 (0.9 to 1.6)	3.1 (1.8 to 5.3)
P value		0.08	0.0001
Birth weight			
NBW (n)	826	2,411	70
LBW n (%)	40 (4.6)	217 (8.3)	12 (14.6)
OR (95% CI)	Reference	1.8 (1.3 to 2.6)	3.5 (1.7 to 7.0)
P value		0.0001	0.0001
Main outcome			
INBW (n)	762	2,191	57
AB n (%)	104 (12.0)	437 (16.6)	25 (30.5)
OR (95% CI)	Reference	1.4 (1.1 to 1.8)	3.2 (1.9 to 5.3)
P value		0.001	0.0001

singleton live birth. They confirmed other reports that suggest an independent association between parity and a reduction in birth weight. They found increasing parity to be a risk factor for LBW, very LBW, PTB, and very PTB in a dose-dependent pattern.

We analyzed the number of pregnancies (parity) and found that increasing parity was associated more with LBW than with PTB (OR = 1.77; $P<0.000$). In their cross-sectional study, Lunardelli and Peres[20] found no relationship between parity and LBW or PTB. However, many factors (a short interval between pregnancies, outcomes of prior pregnancies, and the age of the mother at first pregnancy) link parity with an adverse outcome of pregnancy;[21,22] our data about mothers and their infants were insufficient to evaluate these relationships.

The periodontal status of the mothers in our study worsened with an increasing number of pregnancies ($P<0.0001$) (Table 5). That finding was compatible with the results of the study by Marin et al.[23] of 152 pregnant Caucasian women ($P<0.01$). Those investigators also suggested that birth weight decreased with worsening maternal periodontal disease ($P = 0.02$).

Although vomiting and nausea seemed to be associated with higher gingival index scores and PD during pregnancy,[24] we found no relationship between periodontal status and nausea or vomiting. Another reason for poor oral hygiene in pregnant women may be that the mothers in a lower socioeconomic echelon had less time or money to devote to their personal oral care and health problems as the number of their children increased.

A significant association was found between increased gingival scores and periodontal probing depth in multiparous women.[24] The reason for increased periodontal diseases in multiparous women may be untreated dental or periodontal disease that developed during their prior pregnancies. As Taani et al.[24] also suggested, low socioeconomic status and a lower level of education might lead to poor oral hygiene in multiparous women.

Studies[4,25] have evaluated the effect of prenatal visits on the outcome of pregnancy. Lopez et al.[4] suggested that having fewer than six prenatal visits is an important risk factor for PLBW. Bosnjak et al.[25] found no relationship between prenatal care and PTB.

We also evaluated the effect of prenatal visits on pregnancy outcome in the following two groups of subjects: women who had at least two prenatal visits (55.6% of the study population) and women who had no prenatal visits. We found no relationship between the frequency of prenatal visits and the outcome of pregnancy. There are many factors that cause an abnormal outcome of pregnancy. The aim of prenatal care is to eliminate the existing risk factors or to prevent the pregnant woman from encountering the risk factors that could cause an adverse outcome of pregnancy. The results of the present study indicate that the prenatal care provided to this population is not sufficient to reduce the rate of adverse pregnancy outcomes. However, periodontal evaluation is not a part of routine prenatal visits at public hospitals in Turkey.

The actual causes that underlie the statistically significant relationship between prenatal care and periodontal health are considered to be confounding factors, such as education level, toothbrushing, and parity. Thus, multivariate analysis established that education level or toothbrushing frequency was significant, whereas prenatal care was not.

A lack of education is one of the main reasons for a low socioeconomic status. We found that mothers with relatively less education had significantly greater periodontal treatment needs than did their better educated counterparts (CPITN code 4, $P<0.0001$). That finding was compatible with the results from several studies.[13,16,23,25,26]

The rates of PTB and LBW and the total number of ABs was statistically significant with respect to cesarean births (OR = 1.84, 3.23, and 1.97, respectively;

Table 7.

Adjusted ORs for Three Multivariate Logistic Models (N = 3,576)

	Dependent Variable								
	Birth Weight (risk for LBW)			Weeks of Gestation (risk for PTB)			Main Outcome (risk for AB)		
	Model 1			Model 2			Model 3		
	OR	95% CI	*P*	OR	95% CI	*P*	OR	95% CI	*P*
Age at risk	0.96	0.59 to 1.56	0.892	0.77	0.52 to 1.15	0.215	0.93	0.66 to 1.32	0.711
High level of education	Ref.			Ref.			Ref.		
Low level of education	1.54	0.85 to 2.77	0.151	1.55	1.01 to 2.39	0.046	1.65	1.11 to 2.47	0.013
Uneducated	1.52	1.05 to 2.19	0.025	1.10	0.84 to 1.44	0.481	1.20	0.94 to 1.54	0.140
Cesarean birth	3.23	2.46 to 4.26	0.000	1.84	1.45 to 2.34	0.000	1.97	1.58 to 2.46	0.000
Pregnancy (parity) (n ≥2)	1.77	1.36 to 2.31	0.000	1.15	0.93 to 1.42	0.188	1.30	1.07 to 1.57	0.007
No prenatal care	1.00	0.77 to 1.30	0.982	1.01	0.81 to 1.23	0.971	0.98	0.81 to 1.18	0.849
Smoking (yes)*	1.40	1.03 to 1.90	0.032	1.14	0.88 to 1.47	0.305	1.24	0.98 to 1.55	0.064
Toothbrushing occasionally*	1.45	1.06 to 1.98	0.019	1.31	1.02 to 1.69	0.036	1.27	1.01 to 1.60	0.044
CPITN									
TN-I (codes 0 + 1)	Ref.			Ref.			Ref.		
TN-II (codes 2 + 3)	1.87	1.31 to 2.66	0.001	1.21	0.95 to 1.56	0.118	1.44	1.14 to 1.81	0.002
TN-III (code 4)	3.56	1.74 to 7.25	0.000	2.96	1.72 to 5.09	0.000	3.14	1.86 to 5.29	0.000

Ref. = reference.
* During pregnancy.

P <0.0001). The rate of cesarean deliveries in our study was 15.9%, a rate similar to that reported by the WHO (15%).[11] We found no relationship between the type of birth (cesarean or vaginal) and periodontal status. Silva et al.[27] suggested that elective cesarean section is a risk factor for LBW. Because we had no data on the reasons for cesarean delivery in our study population, we did not comment on that factor.

LBW is no longer an indicator of poor health and lack of socioeconomic development. In our study, the incidence of LBW was 7.5%. This rate is lower than that in Brazil[27] but higher than that in Finland.[27-29]

Although smoking status is recorded in many studies, it is important to include measures of current and prior smoking exposure. Smoking during pregnancy is an important dose-dependent risk factor for an adverse outcome (especially LBW). Maternal smoking also increases the risk for PTB, although it seems to affect fetal growth more than the duration of gestation.[30] Chiolero et al.[30] evaluated the maternal smoking status in two groups of pregnant women: non-smokers and ex-smokers. They suggested that smoking history was not associated with LBW, SGA, or PTB, but that smoking during pregnancy was associated independently with each of those outcomes. We also found a relationship between smoking habits, adverse outcome of pregnancy, and periodontal treatment needs. When we compared non-smoking mothers to those who smoked during pregnancy, the adjusted OR was 1.3 (95% CI: 0.9 to 1.7). When we compared non-smoking mothers to ex-smokers, the adjusted OR was 1.2 (95% CI: 0.9 to 1.7) with respect to LBW. The adjusted ORs of ex-smokers and smokers during pregnancy in the PTB group were similar, 1.1 (95% CI: 0.8 to 1.4). These results did not support the study of Chiolero et al.[30] Moliterno et al.[31] found an adjusted OR of 1.16 (95% CI: 0.30 to 3.30) for LBW in 151 smoking mothers. Our results showed that smoking history was not associated with the outcome of pregnancy as much as was smoking during pregnancy. We also found that maternal smoking increased the risk for PTB, although it seemed to affect fetal growth more than the number of weeks of gestation.

Smoking is undoubtedly one of the greatest and most prevalent risk factors for chronic periodontitis.[32] Most contemporary studies[32,33] suggest that smoking increases the risk for periodontal diseases from two to six times. In our study, there was no significant

Table 8.

Adjusted ORs for Three Multivariate Logistic Models in Pregnant Women Without Prenatal Care (N = 1,588)

	Dependent Variable								
	Birth Weight (risk for LBW)			Weeks of Gestation (risk for PTB)			Main Outcome (risk for AB)		
	Model 1			Model 2			Model 3		
	OR	95% CI	*P*	OR	95% CI	*P*	OR	95% CI	*P*
Age at risk	0.88	0.43 to 1.82	0.726	0.56	0.30 to 1.05	0.072	0.70	0.42 to 1.20	0.201
Education									
High level	Ref.			Ref.			Ref.		
Low level	1.19	0.46 to 3.04	0.722	1.74		0.109	1.55	0.82 to 2.91	0.175
Uneducated	1.46	0.72 to 2.99	0.296	1.19	0.68 to 2.05	0.542	1.31	0.80 to 2.15	0.292
Cesarean birth	2.52	1.63 to 3.91	0.001	1.84	1.26 to 2.66	0.001	1.72	1.22 to 2.43	0.002
Pregnancy (parity) (n ≥2)	0.65	0.44 to 0.96	0.031	1.15	0.88 to 1.60	0.396	1.04	0.78 to 1.40	0.791
Smoking (yes)*	1.59	1.02 to 2.48	0.04	0.84	0.56 to 1.26	0.407	1.03	0.73 to 1.50	0.863
Toothbrushing occasionally*	1.59	1.04 to 2.44	0.034	1.22	0.85 to 1.74	0.275	1.22	0.89 to 1.70	0.221
CPITN									
TN-I (codes 0 + 1)	Ref.			Ref.			Ref.		
TN-II (codes 2 + 3)	2.00	1.14 to 3.51	0.016	1.14	0.77 to 1.65	0.511	1.47	1.03 to 2.10	0.036
TN-III (code 4)	5.46	1.91 to 15.62	0.002	2.09	0.83 to 5.20	0.114	3.22	1.41 to 7.32	0.005

Ref. = reference.
* During pregnancy.

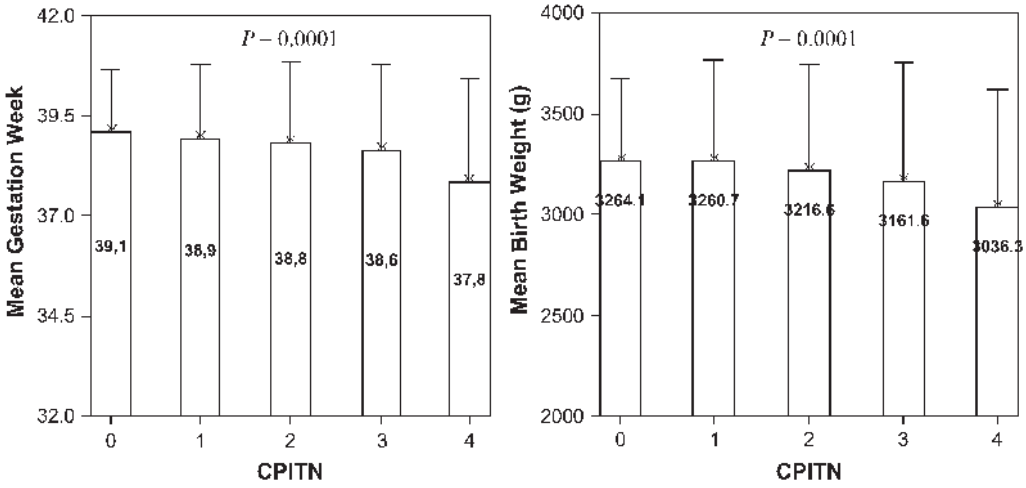


Figure 1.
Distribution of gestation week and birth weight by CPITN code.

difference between smokers and non-smokers in the need for periodontal treatment. It is important to emphasize that our study included few ex-smokers who smoked heavily or mothers who smoked heavily during pregnancy (2.7% of women were heavy smokers during pregnancy, and 8.7% were former heavy smokers.) However, smoking status often is self-reported and may be biased.

The frequency of toothbrushing during and before pregnancy also was evaluated. We investigated the effects of hormonal changes during pregnancy on the accumulation of microbial plaque on gingival tissue and the effects of vomiting and nausea on the frequency of toothbrushing. The results showed that pregnancy did not change the rate of toothbrushing. Several studies[24,26,34] have established a relationship between pregnancy and periodontal conditions. Laine[34] suggested that pregnancy does not cause periodontal disease but may exacerbate a preexisting periodontal condition. Taani et al.[24] showed that periodontal pockets increased as pregnancy progressed. Many

Table 9.

Adjusted ORs for Three Multivariate Logistic Models in Pregnant Women With Prenatal Care (N = 1,988)

	Dependent Variable								
	Birth Weight (risk for LBW)			Weeks of Gestation (risk for PTB)			Main Outcome (risk for AB)		
	Model 1			Model 2			Model 3		
	OR	95% CI	*P*	OR	95% CI	*P*	OR	95% CI	*P*
Age at risk	1.05	0.55 to 2.01	0.883	1.02	0.61 to 1.71	0.931	1.21	0.77 to 1.90	0.420
Education									
High level	Ref.			Ref.			Ref.		
Low level	2.08	0.93 to 4.64	0.075	1.42	0.73 to 2.76	0.290	2.02	1.13 to 3.61	0.018
Uneducated	1.53	1.00 to 2.40	0.052	1.09	0.79 to 1.50	0.579	1.18	0.88 to 1.59	0.259
Cesarean birth	3.91	2.73 to 5.60	0.001	1.80	1.31 to 2.48	0.001	2.15	1.61 to 2.86	0.001
Pregnancy (parity) (n ≥2)	0.51	0.36 to 0.73	0.001	0.69	0.52 to 0.91	0.011	0.61	0.47 to 0.78	0.001
Smoking (yes)*	1.23	0.80 to 1.90	0.353	1.44	1.03 to 2.00	0.030	1.44	1.06 to 1.97	0.019
Toothbrushing occasionally*	1.34	0.85 to 2.12	0.213	1.42	0.99 to 2.05	0.056	1.34	0.96 to 1.89	0.092
CPITN									
TN-I (codes 0 + 1)	Ref.			Ref.			Ref.		
TN-II (codes 2 + 3)	1.79	1.14 to 2.83	0.012	1.28	0.92 to 1.79	0.135	1.44	1.06 to 1.95	0.021
TN-III (code 4)	2.47	1.01 to 6.60	0.049	3.74	1.88 to 7.42	0.001	3.11	1.57 to 6.15	0.001

* During pregnancy.

experts agree that periodontal health declines during pregnancy.[35] In our study, we found a close relationship between toothbrushing frequency and the need for periodontal treatment ($P<0.0001$ and $P<0.005$, respectively). Toothbrushing occasionally during pregnancy is a greater risk factor for LBW than PTB (OR = 1.45; $P<0.019$). These results indicate a possible relationship between PLBW and periodontal diseases.

The most striking result of our study was the significant interaction between the adverse outcome of pregnancy and the need for periodontal treatment (OR = 3.14; 95% CI: 1.86 to 5.29). Women with a preterm or LBW newborn had significantly worse periodontal status than did those who had a newborn weighing >2,500 g or who delivered their infant at term. Three multivariate logistic models showed that the OR for LBW increased from 1.87 to 3.56 as the need for periodontal treatment increased. Moliterno et al.[31] found a relationship between periodontal disease and LBW (OR = 3.48; 95% CI: 1.17 to 10.36). Lopez et al.[4] suggested that periodontal disease is an independent risk factor for PLBW and causes more than a four-fold increase in the risk for PLBW. In our study, the OR for PTB was 1.2 for women classified as TN-II and 2.9 for those classified as TN-III. Periodontal disease was a more significant risk factor for LBW than was PTB.

In our study population, 87% of the women had an INBW newborn, and 12.5% had a preterm newborn. Of those newborns delivered preterm, 8.3% had NBW (PNBW group). To the best of our knowledge, the PNBW group has not been defined in prior studies. Although the risk for morbidity in that group is lower than that in the PTB group, the mechanism that initiates premature delivery is very similar. We found that 92.5% of the mothers had a newborn in the INBW group, and 7.5% had a baby in the LBW group. Of the LBW newborns, 3.3% were ILBW or SGA and 4.2% were PLBW.

Diabetes mellitus is the most frequent metabolic pregnancy complication associated with an increased risk for maternal and neonatal morbidities.[36] The Consensus on Diabetes Classification[37] established type 1 and type 2 diabetes as main groups (type 1: insulin-dependent diabetes; type 2: non-insulin–dependent diabetes), and gestational diabetes was defined as any glucose intolerance detected during pregnancy.[37] Types 1 and 2 pregestational diabetes are associated with early and late complications of pregnancy, including metabolic complications of the newborn.[38] Meanwhile, gestational diabetes is associated with complications in the second half of the pregnancy.[39] Preeclampsia occurs more frequently in diabetic mothers and is the major cause of preterm

delivery. Large for gestational age and macrosomic newborns are the most common perinatal complications of pregestational and gestational diabetes, although they also may occur in association with maternal obesity and excessive weight increase during pregnancy.[40,41] Periodontal disease is one of the major oral health problems encountered in patients with diabetes mellitus. Periodontitis may impair the metabolic control and adequate treatment of the diabetic patient.[42,43] We excluded diabetic patients from the study because of the metabolic and local effects of all types of diabetes. In our study population, 44.4% of women did not receive prenatal care during pregnancy. For this group, the possibility of having gestational diabetes must be considered; 8.3% of ABs were PNBW babies. However, these evaluations are beyond the aims of this study.

The frequency of PTB was higher than LBW; however, periodontal disease is a stronger risk factor for LBW than is PTB. From a clinical standpoint, it seems that the assessment of the periodontal status of pregnant women during an early obstetric evaluation is an important indicator of the risk for future obstetric complications.[44]

Many risk factors for PTB have been identified. Intrauterine growth restriction affects the risk for PTB. Impaired growth is associated with maternal conditions, such as hypertension, which lead to medically indicated PTB, and growth restriction is a common indication for induction before term. However, LBW is one of the results of PTB. In our study, 15% of the births were by cesarean section. There is some evidence[27] for the possible iatrogenic role of elective cesarean section in causing LBW.

One of the most important factors implicated in PTB is acute genitourinary tract infection.[44] The bacteria that cause chronic periodontal inflammation include Gram-negative rods and anaerobes similar to those found in women with bacterial vaginosis. Periodontopathic bacteria are a source of endotoxin and lipopolysaccharides, and inflammatory mediators (including prostaglandin E_2 and cytokines) are increased locally. It was hypothesized that systemic increases in inflammatory mediators may lead to PTB.[1,3,44-47] Some investigators suggested that increases in levels of prostaglandin E_2 and TNF-α seemed to be associated with reduced fetal birth weight.[1,45-48] We found that LBW and PTB were associated closely with periodontal conditions and that the need for periodontal treatment increased as did the risk for PTB and LBW. Because we had no microbiologic data about the oral and vaginal flora of the women studied, these interpretations are based on the results of the recent studies;[45-48] however, there are other factors that affect fetal growth and the duration of gestation. In pregnant women of low socioeconomic status and limited education, other causes of an adverse outcome of pregnancy may include neglected body hygiene, poor nutrition, and emotional stress.

This study has limitations with regard to the data obtained from the subjects' medical records, such as maternal weight and paternal health status. Maternal weight and total weight gain during pregnancy are factors that are important to fetal growth. It was very difficult to obtain data about the health status of the father of each newborn in our study, but paternal periodontal or urinary tract infection may be a risk factor for an adverse outcome of pregnancy because of the possibility of bacterial transmission to the mother.

CONCLUSIONS

In this study, 3,576 women who had just given birth were evaluated for their socioeconomic status and periodontal health. The study population was homogeneous and consisted of a large number of subjects. The results of this clinical study support the relationship between periodontal disease and LBW and PTB. Nonetheless, such a large study population requires the evaluation of laboratory and clinical data from pre- and postperiodontal treatment periods to determine the actual importance and significance of periodontal diseases in pregnancy.

The effect of periodontal pathogens and the mechanism of periodontitis on the outcome of pregnancy also must be evaluated in a population that is well educated and of high socioeconomic status to eliminate the main sources of other risk factors for PTB and LBW.

ACKNOWLEDGMENT

Drs. Toygar, Seydaoglu, Kurklu, Guzeldemir, and Arpak report no conflicts of interest related to this study.

REFERENCES

1. Williams CE, Davenport ES, Sterne JA, Sivapathasundaram V, Fearne JM, Curtis MA. Mechanisms of risk in preterm low-birthweight infants. *Periodontol 2000* 2000;23: 142-150.
2. McGaw T. Periodontal disease and preterm delivery of low-birth-weight infants. *J Can Dent Assoc* 2002;68: 165-169.
3. Offenbacher S, Katz V, Fertik G, et al. Periodontal infection as a possible risk factor for preterm low birth weight. *J Periodontol* 1996;67(10 Suppl.):1103-1113.
4. Lopez NJ, Smith PC, Gutierrez J. Periodontal therapy may reduce the risk of preterm low birth weight in women with periodontal disease: A randomized controlled trial. *J Periodontol* 2002;73:911-924.
5. Davenport ES, Williams CE, Sterne JA, Murad S, Sivapathasundram V, Curtis MA. Maternal periodontal disease and preterm low birthweight: Case-control study. *J Dent Res* 2002;81:313-318.
6. Mumghamba EG, Markkanen HA, Honkala E. Risk factors for periodontal diseases in Ilala, Tanzania. *J Clin Periodontol* 1995;22:347-354.

7. Rose LF, Mealey BL, Genco RJ, Cohen DW. *Periodontics: Medicine, Surgery and Implants*. St. Louis: Elsevier Mosby; 2004:854-855.
8. Offenbacher S, Riche EL, Barros SP, Bobetsis YA, Lin D, Beck JD. Effects of maternal *Campylobacter rectus* infection on murine placenta, fetal and neonatal survival, and brain development. *J Periodontol* 2005;76: 2133-2143.
9. Ramfjord SP. Indices for prevalence and incidence of periodontal disease. *J Periodontol* 1959;30:51-59.
10. Ainamo J, Barmes D, Beagrie G, Cutress T, Martin J, Sardo-Infirri J. Development of the World Health Organization (WHO) community periodontal index of treatment needs (CPITN). *Int Dent J* 1982;32:281-291.
11. World Health Organization. *Oral Health Surveys. Basic Methods*, 3rd ed. Geneva, Switzerland: WHO; 1987: 30-31.
12. Cutress TW, Ainamo J, Sardo-Infirri J. The community periodontal index of treatment needs (CPITN) procedure for population groups and individuals. *Int Dent J* 1987;37:222-233.
13. Miyazaki H, Yamashita Y, Shirahama R, et al. Periodontal condition of pregnant women assessed by CPITN. *J Clin Periodontol* 1991;18:751-754.
14. Yalcin F, Eskinazi E, Soydinc M, et al. The effect of sociocultural status on periodontal conditions in pregnancy. *J Periodontol* 2002;73:178-182.
15. Ziadeh S, Yahaya A. Pregnancy outcome at age 40 and older. *Arch Gynecol Obstet* 2001;265:30-33.
16. Zeitlin JA, Ancel PY, Saurel-Cubizolles MJ, Papiernik E. Are risk factors the same for small for gestational age versus other preterm births? *Am J Obstet Gynecol* 2001;185:208-215.
17. Paulander J, Axelsson P, Lindhe J. Association between level of education and oral health status in 35-, 50-, 65- and 75-year-olds. *J Clin Periodontol* 2003;30: 697-704.
18. Sipila P, von Wendt L, Hartikainen-Sorri AL. The grand multipara–still an obstetrical challenge? *Arch Gynecol Obstet* 1990;247:187-195.
19. Aliyu MH, Salihu HM, Keith LG, Ehiri JE, Islam MA, Jolly PE. High parity and fetal morbidity outcomes. *Obstet Gynecol* 2005;105:1045-1051.
20. Lunardelli AN, Peres MA. Is there an association between periodontal disease, prematurity and low birth weight? A population-based study. *J Clin Periodontol* 2005;32:938-946.
21. Smith GC, Pell JP. Teenage pregnancy and risk of adverse perinatal outcomes associated with first and second births: Population based retrospective cohort study. *BMJ* 2001;323:476.
22. Smith GC, Pell JP, Dobbie R. Interpregnancy interval and risk of preterm birth and neonatal death: Retrospective cohort study. *BMJ* 2003;327:313 (erratum 2003;327: 851).
23. Marin C, Segura-Egea JJ, Martinez-Sahuquillo A, Bullon P. Correlation between infant birth weight and mother's periodontal status. *J Clin Periodontol* 2005; 32:299-304.
24. Taani DQ, Habashneh R, Hammad MM, Batieha A. The periodontal status of pregnant women and its relationship with socio-demographic and clinical variables. *J Oral Rehabil* 2003;30:440-445.
25. Bosnjak A, Relja T, Vucicevic-Boras V, Plasaj H, Plancak D. Pre-term delivery and periodontal disease: A case-control study from Croatia. *J Clin Periodontol* 2006;33:710-716.
26. Machuca G, Khoshfeiz O, Lacalle JR, Machuca C, Bullon P. The influence of general health and sociocultural variables on the periodontal condition of pregnant women. *J Periodontol* 1999;70:779-785.
27. Silva AA, Lamy-Filho F, Alves MT, Coimbra LC, Bettiol H, Barbieri MA. Risk factors for low birthweight in north-east Brazil: The role of cesarean section. *Paediatr Perinat Epidemiol* 2001;15:257-264.
28. Hakala T. Obstetric care, pregnancy risk factors and perinatal outcome in the province of Uusimaa, Finland, in 1980-1981. *Ann Chir Gynaecol Suppl* 1987;203: 1-83.
29. Meurman JH, Furuholm J, Kaaja R, Rintamaki H, Tikkanen U. Oral health in women with pregnancy and delivery complications. *Clin Oral Investig* 2006; 10:96-101.
30. Chiolero A, Bovet P, Paccaud F. Association between maternal smoking and low birth weight in Switzerland: The EDEN study. *Swiss Med Wkly* 2005;135:525-530.
31. Moliterno LF, Monteiro B, Figueredo CM, Fischer RG. Association between periodontitis and low birth weight: A case-control study. *J Clin Periodontol* 2005;32: 886-890.
32. Heasman L, Stacey F, Preshaw PM, McCracken GI, Hepburn S, Heasman PA. The effect of smoking on periodontal treatment response: A review of clinical evidence. *J Clin Periodontol* 2006;33:241-253.
33. Calsina G, Ramon JM, Echeverria JJ. Effects of smoking on periodontal tissues. *J Clin Periodontol* 2002;29: 771-776.
34. Laine MA. Effect of pregnancy on periodontal and dental health. *Acta Odontol Scand* 2002;60:257-264.
35. 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.
36. Metzger BE. Summary and recommendations of the Third International Workshop: Conference on gestational diabetes mellitus. *Diabetes* 1991;40(Suppl. 2):197-201.
37. Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care* 2003;26(Suppl. 1):S5-20.
38. Tyson JE, Hock RA. Gestational and pregestational diabetes: An approach to therapy. *Am J Obstet Gynecol* 1976;125:1009-1027.
39. Kjos SL, Persson B. Gestational diabetes mellitus. *N Engl J Med* 1999;341:1749-1754.
40. Andreasen KR, Andersen ML, Schanz AL. Obesity and pregnancy. *Acta Obstet Gynecol Scand* 2004;83:1022-1029.
41. Forsbach-Sanchez G, Tamez-Perez HE, Vanquez-Lara J. Diabetes and pregnancy. *Arch Med Res* 2005;36: 291-299.
42. Page RC, Beck JD. Risk assessment for periodontal diseases. *Int Dent J* 1997;47:61-87.
43. Rodrigues DC, Taba MJ, Novaes AB, Souza SL, Grisi MF. Effect of non-surgical periodontal therapy on glycemic control in patients with type 2 diabetes mellitus. *J Periodontol* 2003;74:1361-1367.
44. Offenbacher S, Lieff S, Boggess KA, et al. Maternal periodontitis and prematurity. Part I: Obstetric outcome of prematurity and growth restriction. *Ann Periodontol* 2001;6:164-174.

45. Dasanayake AP, Li Y, Wiener H, Ruby JD, Lee MJ. Salivary *Actinomyces naeslundii* genespecies 2 and *Lactobacillus casei* levels predict pregnancy outcomes. *J Periodontol* 2005;76:171-177.
46. Mitchell-Lewis D, Engebretson SP, Chen J, Lamster IB, Papapanou PN. Periodontal infections and preterm birth: Early findings from a cohort of young minority women in New York. *Eur J Oral Sci* 2001;109: 34-39.
47. Offenbacher S. Maternal periodontal infections, prematurity, and growth restriction. *Clin Obstet Gynecol* 2004;47:808-821.
48. Clothier B, Stringer M, Jeffcoat MK. Periodontal disease and pregnancy outcomes: Exposure, risk and intervention. *Best Pract Res Clin Obstet Gynaecol* 2007;21: 451-456.

Correspondence: Dr. Hilal Uslu Toygar, Baskent University, Adana Medical and Research Center, Kazim Karabekir Mah., 59 Sok., No. 91, 01120 Yuregir, Adana, Turkey. Fax: 90-322-322-7979; e-mail: usluhilaladana@yahoo.com.

Submitted February 9, 2007; accepted for publication May 24, 2007.