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A population-based study on the association between type 2 diabetes and periodontal disease in 12,123 middle-aged Taiwanese (KCIS No. 21)

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Abstract

Aim: We investigated the association between type 2 diabetes mellitus (T2DM) and periodontal disease (PD) in the context of the current periodontal aetiology model.

Material and Methods: In total, 14,747 community residents aged 35–44 years were invited to a community-based PD survey between 2003 and 2006 using the community periodontal index. Significant factors modifying the association between T2DM and PD were ascertained. We further assessed the association between T2DM and the risk for PD, within strata of significant effect modifiers, after controlling for other putative factors.

Results: The prevalence rate was 10% higher in subjects with T2DM than in those without. After controlling for significant factors, T2DM was positively associated with the risk for PD (adjusted odds ratio = 1.34,95% confidence interval: 1.07–1.74). The results of interaction assessment showed that only the waist was identified as a statistically significant effect modifier for such a positive association.

Conclusions: The association between T2DM and the risk for PD among young adult was demonstrated. This finding, together with other aetiological factors, fit with the current hypothesized model of the aetiology of periodontitis. However, the effect of T2DM modified by waist measurement should be verified in future studies.

Key words: community periodontal index; community-based integrated sreening; periodontal disease; risk factor; type 2 diabetes

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An association between diabetes and periodontal disease (PD) has been well documented in previous studies (Grossi 2001, Taylor 2001, Jansson et al. 2006, Lalla 2007). Some studies support type 2 diabetes mellitus (T2DM) or hyperglycaemia as being a risk factor for PD (Katz 2001, Soskolne & Klinger 2001, Mattout et al. 2006, Lim et al. 2007, Shimazaki et al. 2007). Moreover, poor glycaemic control may lead to increased severity of periodontitis (Salvi et al. 2008). Despite these findings, the relationship between T2DM and PD has been barely addressed in the context of a theoretically sound model of periodontitis aetiology, which conceptualizes life-style factors, physical activity, diet, comorbidity (e.g. T2DM), and genetic factors as contributory causes responsible for alterations in the host immune response, which in turn encourage microorganisms, calculus, and plaque, leading to periodontitis. In the context of the periodontitis aetiology model, the interactive effect between T2DM and other aetiological factors on PD, that is, ''effect modification'' (Hyman 2006, Ylöstalo & Knuuttila 2006), can be investigated and possible confounding

Conflict of interest and source of funding statement

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factors derived from the literatures can also be controlled.

To examine any association between early age onset of T2DM or hyperglycaemia and PD, conducting a population-based study with the enrolment of middle-aged (35–44 years) adults would be useful. Thus, by collecting and analysing data from a community-based PD assessment among participants aged 35–44 years, the current study examined the prevalence of PD in the presence of T2DM to assess whether any extraneous variable(s) can modify the association between T2DM and PD. Moreover, the study further estimated the magnitude of the association, after adjusting for possible confounding factors within strata of effect modifiers.

Materials and Methods

Study subjects

The study population included participants who were aged 35–44 years and attended the Keelung Community-based Integrated Screening (KCIS) programme between 2003 and 2006, an outreaching, integrated screening service that included breast cancer, colorectal cancer, cervical cancer, oral cancer, liver cancer, and three chronic diseases: hypertension, T2DM, and hyperlipidaemia. Details of the study design and early findings of KCIS have been described elsewhere (Chen et al. 2004, Chiu et al. 2006a). Those patients with severe systemic diseases, such as cancer, rheumatic heart disease, heart valve prolapse with regurgitation, and bleeding tendencies that contraindicated periodontal probing, were excluded from the periodontal assessment. Before beginning screening activities, all participants were given relevant information regarding the questionnaire, blood biochemical tests, and periodontal assessments, and they then provided written informed consent to take part in the study (Chiu et al. 2006a).

Periodontal pocket depth measurement

The methodology and descriptive results of this periodontal survey were published previously (Lai et al. 2007). In brief, to assess the periodontal status, we measured the pocket depth as in most of the previous studies with community periodontal index (CPI) by using sextant as a unit with the following classification: 0 for a healthy periodontium, 1 for gingival bleeding, 2 for calculus, 3 for a 4–5 mm periodontal pocket, or 4 for a 6 mm or deeper periodontal pocket. The CPI, as an indicator of periodontal status, is used to assess gingival bleeding, calculus, and periodontal pockets using a specially designed lightweight probe with a 0.5 mm ball tip, black bands at 3.5–5.5 mm, and rings at 8.5 and 11.5 mm from the ball tip. The full mouth was divided into six sextants (teeth numbers: 18–14, 13–23, 24–28, 38–34, 33–43, and 44–48) and was examined only when two or more teeth present were in a sextant (WHO 1997). The CPI score was taken if each sextant had at least two teeth. In our study, the number of completed sextant measurements were 93.46%, 3.85%, 1.44%, 0.68%, 0.32%, and 0.24% for 6, 5, 4, 3, 2, and 1 sextants, respectively. The highest score among the six sextants was adopted to represent the CPI status for each individual. Using the highest CPI each individual, the mean and median of CPI were 2.04 and 2 in this study population. PD in this study is therefore defined by the measurement of periodontal pockets with CPI score >3 (periodontal pocket $\geqslant 4$ mm). Those with destructive PD were included in the category of CPI score >4 (periodontal pocket $\geqslant 6$ mm). All dentists involved in this periodontal assessment were trained to carry out calibration to attain the consistency across examiners before the community-based survey. The detailed procedure has been described elsewhere (Lai et al. 2007).

Definition of T2DM and other chronic diseases

T2DM was defined by the criteria of the American Diabetes Association (ADA 2005); those with a previous history of T2DM or fasting plasma glucose (FPG) $\geqslant 126$ mg/dl were considered to be T2DM cases. Those with no history of T2DM, but with FPG between 110 and 125 mg/dl, were defined as pre-diabetes cases (Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus 1997, WHO 1999). Hypertension was defined according to the JNC 7 guidelines (Chiu et al. 2006b). Body mass index (BMI) $\geqslant 25\,kg/m^2$ was defined as obesity. Males with waist measurements $\geqslant 90$ cm and females with $\geqslant 80$ cm were considered to have central obesity. Both criteria were adjusted to the WHO Asian obesity definition (WHO 2000). Three biomarker indicators, total cholesterol, triglycerides (TG), and high-density lipoprotein (HDL), were measured for blood lipids. Abnormal cholesterol was defined at total cholesterol $\geqslant 200$ mg/dl. HDL <40 mg/dl was considered abnormal. These biomarker definitions have been described in previous studies (Chiu et al. 2006b, 2007, Yen et al. 2006).

Measurement of anthropometric and biochemical variables

For biochemical variables, 12 h fasting blood samples for serum and blood cell account biomarkers were drawn at recruitment. Anthropometric measurements were made by trained staff. Height, weight, and waist circumference were measured with a standard ruler, standardized scales (to 0.1 kg), and a measuring tape (to 0.1 cm), respectively. Waist size was measured midway between the inferior margin of the ribcage and the iliac crest horizontally and hip circumference, measured as the maximum horizontal circumference around the buttocks. Serum biomarkers included FPG, TG, total cholesterol (TC), and low-density (LDL) and high-density lipoprotein (HDL) cholesterol.

Questionnaire data on dietary and lifestyle factors

Questionnaire data on demographic features, life style (betel-quid chewing, cigarette smoking, and alcohol consumption), frequency of teeth brushing, and dietary habits were obtained by face-to-face interviews by a group of well-trained public health nurses. The details of measurements of these factors have been described elsewhere (Chiu et al. 2006b, 2007, Yen et al. 2006). In brief, regarding betel-quid chewing, cigarette smoking, and alcohol, the consumption of quantity and frequency per time or per day were recorded. We classified those exposed to alcohol consumption, cigarette smoking, and betel nut chewing into low and high intake using the median value of each exposure. The frequency of teeth brushing was classified into three categories, none, once, or two or more times per day, to represent an oral hygiene index. Dietary patterns over the previous 6 months were also collected by the same questionnaire, including consumption of meat, seafood, fish, egg products, beans, milk, coffee, vegetables, and

fruits. Food moulds and standard containers were shown for quantitative reference records. The frequency of consumption was categorized into five levels: never or seldom, 1–2, 3–4, 5–6, and >7 times/week. We classified >5 times/week as ''frequent'' and otherwise as ''infrequent'' (Chiu et al. 2006b).

Statistical analyses

The prevalence of PD is presented as a percentage, by gender and T2DM status. A univariate logistic regression model was used to estimate crude odds ratios (ORs) and the 95% confidence intervals (95% CIs) for the association between T2DM and the risk for PD and other possible effect modifiers and confounding factors, based on the epidemiological method (Rothman & Greenland 1998) as to how confounding and effect modification can affect results regarding an association between periodontitis and systemic disease (Hyman 2006, Ylöstalo & Knuuttila 2006).

To assess whether the effects of diabetes on PD were modified by extraneous factors, we compared the model including interaction terms between diabetes and each possible effect modifier, not including the corresponding interaction terms, using the likelihood ratio test (LRT). The LRT considers the effect modifier as statistically significant if the difference in the -2 log likelihood value between the two models (see below) is larger than the χ^2 value, given the degrees of freedom. Such a model comparison was illustrated by considering the one effect modifier identified in our study: abnormal waist measurement.

Model I (without interaction): logit $P = \alpha + \beta_1 x_1 + \beta_2 x_2$.
Model II (with interaction): logit $P = \alpha + \beta_1 x_1 + \beta_2 x_2 + r x_1 x_2$.
x_1 = presence of type 2 diabetes (yes = 1, no = 0).
x_2 = a binary variable representing the presence of a larger waist (larger = 1, normal = 0).
α, β, and γ denote respective regression coefficients.

In this example, the interaction's statistical significance was assessed by determining whether the χ^2 value of $x^2_{(df=1)}$ was larger than 3.84, equivalent to a p-value <0.05. Similar models were also constructed to test other effect modifiers. In each stratum of the effect modifier, a multivariable logistic regression model was further used to assess the effect of T2DM on the risk for PD by extending the model II with the incorporation of all possible confounding factors. The stratum-specific adjusted ORs and 95% CIs were computed. The statistical significance level was set at p-values <0.05 or a 95% CI not including 1. All statistical analyses were conducted using SAS software (version 9.1; SAS Institute Inc. 2007).

Results

In total, 14,747 subjects aged 35–44 years were invited to participate in the PD assessment between 2003 and 2006. Of them, 12,123 completed the PD assessment. The mean age (± standard deviation, SD) was 39.46 (± 2.91 SD) years; Table 1 shows that the overall attendance rate was 82.2% (83.4% for men and 81.6% for women). The attendance rate was higher in participants aged 41–44 years (88–89%) than in those aged 35–40 years (75–82%). Only a small difference in uptake of the PD examination was observed between the less educated (81.6%) and the well educated (82.2%).

Prevalence of PD by T2DM status

The prevalence of T2DM in those aged 35–44 years was 2.61%. Men had a higher rate than women by about 1.5-fold (3.49% *versus* 2.12%). Of 316 patients with T2DM, 155 (49.05%) were previously diagnosed and 161 (50.95%) were newly diagnosed with T2DM. The mean duration for the previously diagnosed cases was 4.12 years. Using the periodontal pocket ⩾4 mm definition of PD, the prevalence rate was 28.6%. Men had a higher prevalence than women by about 10%. The prevalence of PD increased with age, particularly from 40 years for men and 43 years for women.

Table 2 shows that the prevalence rate of PD was 10% higher in subjects with T2DM (38.9%) than those without T2DM (28.3%). These findings were similar for men and women, although the prevalence rate for men was consistently higher than that for women. Similar findings were also noted for the prevalence rates of PD in subjects with hyperglycaemia (FPG ⩾ 110 mg/dl).

Risk factors associated with PD

Table 3 shows the crude OR for each possible risk factor in association with the risk for PD. T2DM, including previously diagnosed and newly diagnosed cases, was associated with the risk for PD (OR = 1.62, 95% CI: 1.29–2.03). The associated risks of T2DM for PD were statistically significant for newly diagnosed (OR = 1.56, 95% CI: 1.12–2.16) and previously diagnosed (OR = 1.67, 95% CI: 1.22–2.30) *versus* those without T2DM. Because the association between the presence of T2DM and PD did not vary substantially with either previously diagnosed or newly diagnosed cases, we combined both in one group in the following interaction and confounding assessment. The association between pre-diabetes and PD was not statistically significant (OR = 1.19, 95% CI: 0.91–1.55).

Table 3 also presents other factors related to PD. For demographic vari-

Table 1. Attendance rate for the periodontal assessment

Variable	Classification	No. invited	Attendees	Attendance rate (%)
Gender	Male	5186	4325	83.4
	Female	9561	7798	81.6
Screening year	2003	5038	3969	78.8
	2004	3946	3193	80.9
	2005	2193	1856	84.6
	2006	3570	3105	87.0
Age group (years)	35–36	3208	2532	78.9
	37–38	2964	2426	81.8
	39–40	3131	2332	74.5
	41–42	2688	2390	88.9
	43–44	2756	2443	88.6
Education (years)	<6	711	580	81.6
	6–12	9333	7623	81.7
	⩾12	4703	3920	83.4
	Overall	14,747	12,123	82.2

ables, an increase of 1 year in age led to an elevation in the risk for PD by 5% (95% CI: 4–7%). Men had a 1.64-fold higher risk for PD than women. By classifying years of education into three levels, the risk values for PD were 1.51 and 1.26-fold for <6 and 6–12 years, respectively, compared with the baseline group of 12 years or more. The unemployed, housewives, manual workers, and tradesmen/commercial service workers were at an increased risk of developing PD compared with white collar occupations. Other significant risk factors included betel-quid chewing, cigarette smoking, alcohol consumption, obesity, abnormal waist measurement, high TG, and low HDL. Those with higher consumptions of alcohol, cigarettes, and betel nuts were at an increased risk for PD compared with those with less exposure. The frequency of brushing teeth at least twice a day was positively associated with the risk for PD (OR = 0.59, 95% CI: 0.46–0.75). A negative association between a frequency of fruit intake at least once a week and the risk for PD risk was noted (OR = 0.90, 95% CI: 0.81–0.99). The risk for PD was inversely associated with a duration of physical activity per week for fewer than 100 min. (OR = 0.91, 95% CI: 0.84–1.00) and more than 100 min. (OR = 0.89, 95% CI: 0.79–1.00), as compared with infrequent physical activity.

Interaction assessment and stratification analysis

Table 4 shows the estimated stratum-specific ORs by all putative factors presented in Table 3 and the assessment of effect modification for each factor using the LRT test. The relationship between T2DM and the risk for PD did not vary with the putative factors, as can be seen in Table 4, except waist measurement, which was statistically significant ($p = 0.020$) based on the LRT test. The stratum-specific OR for the effect of T2DM on the risk for PD was 2.05 (95% CI: 1.46–2.88) for normal waist and 1.15 (95% CI: 0.83–1.58) for abnormal waist measurement.

Table 5 shows the adjusted odds ratios (aORs) of the multivariable logistic regression for the overall group and subgroup analysis by two levels of waist measurement. In each level, other confounding factors were also controlled and presented. After controlling for

Table 2. Gender-specific prevalence of periodontal disease by the presence of T2DM

Group	Age (years)	FPG < 126			FPG ⩾ 126			Overall		
		periodontal status			periodontal status			periodontal status		
		normal	CPI ⩾ 3*	%	normal	CPI ⩾ 3*	%	normal	CPI ⩾ 3*	%
Female	35–36	1249	332	21.0	10	5	33.3	1259	337	21.1
	37–38	1178	355	23.2	16	8	33.3	1194	363	23.3
	39–40	1085	355	24.7	30	10	25.0	1115	365	24.7
	41–42	1135	374	24.8	22	16	42.1	1157	390	25.2
	43–44	1103	467	29.8	31	17	35.4	1134	484	29.9
	Total	5750	1883	24.7	109	56	33.9	5859	1939	24.9
Male	35–36	621	291	31.9	19	5	20.8	640	296	31.6
	37–38	579	269	31.7	12	9	42.9	591	278	32.0
	39–40	518	298	36.5	20	16	44.4	538	314	36.9
	41–42	529	283	34.9	14	17	54.8	543	300	35.6
	43–44	471	315	40.1	19	20	51.3	490	335	40.6
	Total	2718	1456	34.9	84	67	44.4	2802	1523	35.2
Overall	35–36	1870	623	25.0	29	10	25.6	1899	633	25.0
	37–38	1757	624	26.2	28	17	37.8	1785	641	26.4
	39–40	1603	653	29.0	50	26	34.2	1653	679	29.1
	41–42	1664	657	28.3	36	33	47.8	1700	690	28.9
	43–44	1574	782	33.2	50	37	42.5	1624	819	33.5
	Total	8468	3339	28.3	193	123	38.9	8661	3462	28.6

*CPI index ⩾3 is equal to CPI pocket depth ⩾4 mm.
CPI, community periodontal index; FPG, fasting plasma glucose; T2DM, type 2 diabetes mellitus.

Table 3. Risk factors associated with periodontal disease (CPI ⩾ 3)

Variable	Level	PD cases	Normal cases	Crude OR (95% CI)
Type 2 diabetes	Normal	3339	8468	1.00
	New cases (1)	59	96	1.56 (1.12–2.16)*
	Previous history (2)	64	97	1.67 (1.22–2.30)*
	(1)+(2)	123	193	1.62 (1.29–2.03)+
Pre-diabetes	Normal	3256	8290	1.00
	Pre-diabetes (FPG110–125 and no history)	83	178	1.19 (0.91–1.55)
	DM (new+previous)	123	193	1.62 (1.29–2.03)+
Age (years)		3462	8661	1.05 (1.04–1.07)+
Gender	Female	1939	5859	1.00
	Male	1523	2802	1.64 (1.52–1.78)+
Education (years)	⩾12	772	1555	1.00
	6–12	1722	4154	1.26 (1.15–1.39)+
	<6	968	2952	1.51 (1.35–1.70)+
Occupation	Teacher, office holder, military	619	1795	1.00
	Unemployed	289	503	1.67 (1.40–1.98)+
	Housewife	583	1606	1.05 (0.92–1.20)
	Manual labourer	363	683	1.54 (1.32–1.80)+
	Professional, government staff	146	463	0.91 (0.74–1.13)
	Tradesmen/commercial service	1462	3611	1.17 (1.05–1.31)*
Tooth brushing	No	114	169	1.00
	Yes (⩾2 times/day)	3237	8142	0.59 (0.46–0.75)+
Alcohol consumption	None	2339	6488	1.00
	<3 glass/week	372	823	1.25 (1.10–1.43)*
	⩾3 glass/week	419	729	1.59 (1.40–1.81)+
	Drinking but no quantity stated	332	621	1.48 (1.29–1.71)+
Cigarette smoking	None	2164	6290	1.00
	<12/day	511	1128	1.32 (1.17–1.48)+
	⩾12/day	686	1023	1.95 (1.75–2.17)+
	Smoking but no quantity stated	101	220	1.33 (1.05–1.70)*

Table 3. (Contd.)

Variable	Level	PD cases	Normal cases	Crude OR (95% CI)
Betel-quid Chewing	None	2967	7898	1.00
	<5 nuts/day	193	300	1.69 (1.34–2.12)$^{+}$
	⩾5 nuts/day	182	238	1.95 (1.65–2.31)$^{+}$
	Chewing no quantity stated	120	225	1.42 (1.13–1.78)*
Hypertension	No	2707	7078	1.00
	Yes	755	1583	1.25 (1.13–1.38)$^{+}$
BMI (kg/m^2)	<25	2194	5934	1.00
	⩾25	1262	2686	1.27 (1.17–1.38)$^{+}$
Waist (cm)	F<80, M<90	2610	6862	1.00
	F⩾80, M⩾90	1723	841	1.28 (1.17–1.41)$^{+}$
Total cholesterol (mg/dl)	<200	2260	5755	1.00
	⩾200	1201	2904	1.05 (0.97–1.14)
Triglycerides (mg/dl)	<200	2845	7568	1.00
	⩾200	616	1091	1.50 (1.35–1.67)$^{+}$
HDL (mg/dl)	⩾40	2816	7519	1.00
	<40	645	1139	1.51 (1.36–1.68)$^{+}$
Meat	Infrequent	200	503	1.00
	Frequent	3137	7770	1.02 (0.86–1.20)
Fish	Infrequent	2220	5381	1.00
	Frequent	1102	2876	0.93 (0.85–1.01)
Beans	Infrequent	204	489	1.00
	Frequent	3121	7785	0.96 (0.81–1.14)
Coffee	Infrequent	2057	5160	1.00
	Frequent	1262	3103	1.02 (0.94–1.11)
Fruit	Infrequent	668	1523	1.00
	Frequent	2648	6740	0.90 (0.81–0.99)*
Physical activity	Never or seldom	1442	3409	1.00
	<100 min./week	1332	3436	0.91 (0.84–1.00)
	⩾100 min./week	547	1455	0.89 (0.79–1.00)

*$0.0001 \leqslant p < 0.05$. $^{+}p < 0.0001$.

BMI, body mass index; CI, confidence interval; CPI, community periodontal index; FPG, fasting plasma glucose; HDL; high-density lipoprotein; OR, odds ratio.

other putative factors, T2DM was positively associated with the risk for PD (adjusted OR = 1.34,95% CI: 1.07–1.74). Positive associations were also found for education level, occupation status, cigarette smoking, and HDL status, whereas factors responsible for the inverse associations included tooth brushing and intake of fruit. By the stratification of waist measurement, in those with normal waist measurements, the association between T2DM and PD (aOR = 1.67, 95% CI: 1.17–2.38) was found but there was no statistical significance for the association with a larger waist (aOR = 1.15, 95% CI: 0.82–1.61) after adjusting for other significant factors.

Discussion

Using a community- and population-based study to assess PD status in adults aged 35–44 years, we found that the prevalence rate of PD in subjects with T2DM was 10% higher than in those without T2DM in both men and women. However, as pointed out earlier, PD is a systemic disease and may also be affected by other aetiological factors. The independent roles of obesity, dyslipidaemia, oral hygiene habits, and smoking may also support the current knowledge of periodontal aetiology from the literatures (Merchant &and Pitiphat 2007). These factors, together with T2DM, in turn, alter the host immune response. In our study, obesity and low HDL may also be regarded as comorbidities, smoking as a lifestyle factor, and poor oral hygiene habits as a promoter that enables microorganisms to increase the risk for PD.

Comparisons of different aetiological factors with previous studies can be made. Our findings were consistent with previous findings from Western countries that showed a two- to fourfold increased risk for PD in patients with diabetes compared with non-diabetics. However, the temporal relationship between T2DM and PD is still unclear because poor periodontal status also reportedly leads to increased glycaemic levels (Taylor et al. 1996). Based on the current model of periodontitis aetiology, the role of T2DM has been thought of as a comorbidity, affecting the host immune response via the generation of advanced glycation end-products (AGEs) that are deposited in the periodontium; this triggers an inflammatory process that induces oxidative stress and leads to PD. Obesity and low HDL levels may be regarded as having the same effects because both are major components of metabolic syndrome, which is believed to be related to elevated inflammatory markers such as TFN-α, leading to an altered immune response (Pischon et al. 2007). Associations between aspects of the metabolic syndrome and PD using CPI have been reported in several previous studies, including that of low HDL (Shimazaki et al. 2007), obesity as defined by a high waist measurement (Shimazaki et al. 2007) or BMI (Al-Zahrani et al. 2003), LDL (Losche et al. 2000, Katz 2001), and TG (Losche et al. 2000). The results regarding waist measurements and HDL were similar to our study, but our result on hyperglycaemia was not significant after adjusting for other factors. Several possibilities may account for this difference. The previous study did not take into account education level, which was a factor associated with the metabolic syndrome in Taiwan (Chiu et al. 2007). The age range (40–79 years) in the previous study was also different from ours (35–44 years), as was the definition of PD.

Regarding lifestyle factors and demographic variables, our findings were similar to previous studies on smoking (Teng et al. 2003, Susin et al. 2004, Nishida et al. 2005, Okamoto et al. 2006, Wang et al. 2007), education level (Teng et al. 2003, Wang et al. 2007),

Table 4. Stratum-specific odds ratio and assessment of effect modification for the association between T2DM and PD (CPI index) using the likelihood ratio test (LRT)

Variable	Level	FPG ⩾ 126/ FPG < 126	*p*-value for LRT interaction test†
Age (years)	35–39	1.30 (0.89–1.89)	0.3552
	40–44	1.79 (1.33–2.39)+	
Gender	Female	1.57 (1.13–2.18)*	0.8936
	Male	1.49 (1.07–2.07)*	
Education (years)	⩾12	1.42 (0.94–2.16)	0.5645
	6–12	1.46 (1.04–2.06)*	
	<6	1.98 (1.24–3.15)*	
Tooth brushing	No	1.12 (0.38–3.31)	0.6928
	Yes (⩾2 times/day)	1.66 (1.31–2.10)	
Alcohol consumption	None	1.65 (1.24–2.20)*	0.4223
	<3 glass/week	1.46 (0.75–2.83)	
	⩾3 glass/week	2.07 (1.11–3.87)*	
	Drinking no quantity stated	0.84 (0.38–1.86)	
Cigarette smoking	None	1.60 (1.19–2.17)*	0.5124
	<12/day	1.34 (0.74–2.42)	
	⩾12/day	1.52 (0.93–2.47)	
	Smoking but no quantity stated	1.47 (0.41–5.33)	
Betel-quid chewing	None	1.66 (1.28–2.14)*	0.8028
	<5 nuts/day	1.31 (0.56–3.10)	
	⩾5 nuts/day	1.13 (0.51–2.51)	
	Chewing but no quantity stated	0.83 (0.25–2.75)	
Hypertension	No	1.84 (1.36–2.49)+	0.1977
	Yes	1.21 (0.84–1.72)	
BMI (kg/m^2)	<25	2.12 (1.43–3.14)*	0.1241
	⩾25	1.28 (0.96–1.70)	
Waist (cm)	F<80, M <90	2.05 (1.46–2.88)+	0.0197
	F⩾80, M ⩾90	1.15 (0.83–1.58)	
Total cholesterol (mg/dl)	<200	1.73 (1.26–2.38)*	0.7851
	⩾200	1.49 (1.06–2.09)*	
Triglycerides (mg/dl)	<200	1.62 (1.20–2.19)*	0.5546
	⩾200	1.25 (0.86–1.81)	
HDL (mg/dl)	⩾40	1.76 (1.33–2.32)+	0.1203
	<40	1.09 (0.72–1.67)	
Meat	Infrequent	1.79 (0.67–4.77)	0.9185
	Frequent	1.66 (1.30–2.11)+	
Fish	Infrequent	1.70 (1.27–2.27)*	0.7898
	Frequent	1.58 (1.06–2.35)*	
Beans	Infrequent	1.39 (0.57–3.35)	0.6828
	Frequent	1.67 (1.31–2.13)+	
Coffee	Infrequent	1.56 (1.16–2.10)*	0.1876
	Frequent	1.81 (1.24–2.65)*	
Fruit	Infrequent	1.42 (0.87–2.31)	0.7600
	Frequent	1.72 (1.32–2.24)+	
Physical activity	Never or seldom	1.62 (1.13–2.31)*	0.9422
	<100 min./week	1.61 (1.12–2.32)*	
	⩾100 min./week	1.71 (0.95–3.06)	

†Interaction test for DM and PD (CPI index) using the log-likelihood ratio test.
*$0.0001 \leqslant p < 0.05$.
+$p < 0.0001$.
BMI, body mass index; CPI, community periodontal index; HDL, high-density lipoprotein; PD, periodontal disease; T2DM, type 2 diabetes mellitus.

alcohol consumption (Okamoto et al. 2006), betel-quid chewing (Teng et al. 2003), teeth brushing (Teng et al. 2003), and intake of fruit and vitamins (Diplock 1991, Nishida et al. 2000).

Our study has several limitations. First, only the frequency of brushing teeth was collected in our questionnaire to represent oral hygiene and no information on dental floss use was obtained. Second, information on regular dental checkups was lacking. In housewives and other groups with higher risks of PD, this may have been due to infrequent dental checkups compared with those in white collar occupations. Such information should be collected to examine this issue. Third, genetic influences on PD have been mentioned in previous studies. Studies of familial aggregation, linkage, genome research, and/or genetic susceptibility across patients and potential pathogens (Rylev & Kilian 2008) should be conducted to investigate and identify those more likely to suffer from PD. Finally, regarding any causal relationship between T2DM and PD, our study cannot fully account for the temporal relationship between T2DM and PD. However, the role of T2DM associated with the risk for PD can be suggested for two reasons. Such a positive association between T2DM and PD was modified by waist level in the effect modification assessment. The effect of T2DM was remarkable in subjects with normal waists, whereas the corresponding effect was not significant in those with abnormal waist measurements after controlling for other potential risk factors. From a biological viewpoint, that the effect of T2DM on PD was largely manifested in those with normal waists suggests that T2DM constitutes an independent pathway to PD. Moreover, established factors accounting for the risk for PD are well controlled, and so the likelihood of an independent role for T2DM is high. More importantly, as our study subjects were all middle-aged adults, confounding effects within each stratum of waist measurement as a result of other environmental or acquired risk factors are less likely. However, as our study was not longitudinal, these issues need to be further clarified. Regarding the role of effect modification related to waist measurement, the possibility of multiple comparisons after a series of interaction assessments cannot be ruled out in the analysis of data regarding the association between systemic diseases and PD suggested by a recent article (Hyman 2006, Ylöstalo & Knuuttila 2006). The significant finding stemming from such a subgroup analysis should be verified in a future study.

In conclusion, a large population-based and community-oriented programme showed that middle-aged patients (35–44 years) with T2DM had about a 10% higher risk for PD than those without T2DM. The impact of T2DM, together with other independent aetiological factors, on the risk for PD is consistent with the current model of

Table 5. Multivariable logistic regression model for the association between T2DM and PD, controlling other variables and stratified by waist level (effect modifier)

Variable	Classification	Overall	Categories of waist	
			normal waist	larger waist
1. Main variable				
T2DM	T2DM/no T2DM	1.34 (1.07–1.74)*	1.67 (1.17–2.38)*	1.15 (0.82–1.61)
2. Confounding factors				
Age (years)		1.05 (1.03–1.06)+	1.04 (1.02–1.06)+	1.07 (1.04–1.10)+
Gender	Male/female	1.43 (1.27–1.62)+	1.41 (1.22–1.62)+	1.47 (1.14–1.90)*
Education (years)	6–12/⩾12	1.21 (1.09–1.33)*	1.20 (1.07–1.35)*	1.23 (0.98–1.54)
	<6/⩾12	1.38 (1.21–1.58)+	1.35 (1.16–1.57)+	1.48 (1.13–1.96)*
Occupation†	Unemployed	1.04 (0.86–1.27)	1.11 (0.88–1.39)	0.86 (0.58–1.29)
	Housewife	1.18 (1.01–1.37)*	1.25 (1.05–1.49)*	0.95 (0.67–1.35)
	Manual labourer	1.13 (0.95–1.34)	1.10 (0.90–1.34)	1.20 (0.86–1.68)
	Professional	0.98 (0.80–1.21)	1.03 (0.81–1.31)	0.85 (0.55–1.32)
	Tradesmen/commercial service	1.10 (0.97–1.23)	1.17 (1.03–1.34)*	0.85 (0.66–1.10)
Tooth brushing	⩾2 times per day/no	0.74 (0.58–0.94)*	0.68 (0.51–0.90)*	0.98 (0.60–1.59)
Alcohol consumption	<3 glass per week/none	0.95 (0.81–1.11)	0.96 (0.81–1.16)	0.89 (0.64–1.23)
	⩾3 glass per week/none	1.05 (0.91–1.22)	0.97 (0.82–1.15)	1.26 (0.96–1.64)
	Drinking but no quantity stated/none	1.13 (0.96–1.34)	0.96 (0.79–1.17)	1.84 (1.32–2.58)*
Cigarette smoking	<12 per day/none	1.08 (0.95–1.24)	1.16 (1.00–1.36)*	0.87 (0.66–1.15)
	⩾12 per day/none	1.36 (1.18–1.57)+	1.49 (1.26–1.78)+	1.07 (0.82–1.40)
	Smoking but no quantity stated/none	1.29 (0.96–1.73)	1.32 (0.93–1.86)	1.11 (0.60–2.05)
HDL (mg/dl)	<40/⩾40	1.19 (1.05–1.34)*	1.10 (0.94–1.28)	1.31 (1.07–1.61)*
Triglycerides (mg/dl)	⩾200/<200	1.09 (0.96–1.23)	1.15 (0.98–1.36)	0.93 (0.76–1.14)
Fruit	Frequent/infrequent	0.90 (0.81–0.99)*	0.86 (0.77–0.97)*	0.99 (0.79–1.23)

†All compared with teacher, office holder (white collar occupations) as baseline.
*$0.0001 \leqslant p < 0.05$.
+$p < 0.0001$.
PD, periodontal disease; T2DM, type 2 diabetes mellitus.

periodontitis aetiology. However, the finding of a positive association varying with waist measurement should be corroborated in future studies.

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Clinical Relevance

Scientific rationale for the study: Although the association between PD and T2DM is well documented, the corresponding association in middle-aged adults, particularly effect modification by extraneous variable(s), has scarcely been addressed using a large population-based PD survey in the context of a theoretically sound periodontal aetiology model.

Principal findings: The prevalence of PD using the CPI index was about 10% higher in subjects with T2DM than in those without T2DM. The association between T2DM and PD using CPI as the outcome varied with the waist measurement.

Practical implications: Our results found a positive association between T2DM and PD in subjects aged 35–44 years. Intervention programmes for PD and T2DM may be considered starting from the young adult. Findings on subgroup analysis such as waist measurement should be verified before the subgroup intervention programme is designed.