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Risk determinants of periodontal disease – an analysis of the Study of Health in Pomerania (SHIP 0)

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Abstract

Background: In this study, risk determinants were determined for periodontal disease in the representative population sample (n = 3146) of the Study of Health in Pomerania.

Methods: After examining the net random sample (response 69%) and exclusion of edentulous cases and those with missing values, 2595 subjects remained. Using a multivariate, fully adjusted logistic regression, different definitions of "periodontally diseased/healthy" were examined as the dependent variable (extent of attachment loss (AL \ge 4 mm, combined AL and tooth loss). The independent variables used were sociodemographic factors (age, gender, income, education), medical factors (systemic diseases, drugs), behavioral factors (regular dental checkup, smoking), and oral factors (presence of supragingival calculus and plaque).

Results: The following risk determinants were found for AL: male gender, presence of supragingival plaque and calculus, smoking, low educational level. For the combination of AL and tooth loss, risk determinants were female gender, supragingival plaque, smoking, and low educational level. Consumption of antiallergic medications and regular dental checkups proved to be protective. Smoking was the most influential risk determinant. These parameters explained approximately 43–55% of the variation.

Conclusion: These results concur with those of the literature. In order to explain disease status further, host-response and microbiological factors must also be examined.

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The observation that not all individuals of a population exhibit the same susceptibility to periodontal disease indicates that certain factors modulate the onset and progression of the disease. Thus, the scientific focus is currently on risk factors influencing the susceptibility for onset and progression of destructive periodontal disease (Genco 1996). It is incontestable that the presence of bacteria is necessary for periodontitis to arise; however, bacteria alone are incapable of initiating the disease (Page & Kornman 1997). Genetic disposition (Kornman et al. 1997), lifestyle, and environmental parameters interact with

the bacteria to cause disease (Grossi et al. 1994, 1995, Kocher et al. 2002, Meisel et al. 2003). Education, dental awareness, income, age, and male gender, in addition to smoking, have been shown to be risk factors for periodontal disease (Phillips et al. 1991, Micheelis & Reich 1999, Treasure et al. 2001).

Periodontal disease, caries or endodontic lesions are seldom so advanced that extraction is the only treatment option. The decision to carry out an extraction for periodontal reasons is strongly influenced by treatment decisions of the dentist and his interaction with the patient (Kay & Nuttall 1997). Spontaneous exfoliation of teeth because of severe periodontal breakdown is an uncommon event in subjects who regularly visit a dentist. In a recent study, Splieth et al. (2002) showed that in East Germany, a threshold seems to exist for the decision to extract periodontally diseased teeth with an attachment loss (AL) of as little as 30%; about 20% of the teeth were extracted at this level. Subjects with that level of periodontitis would certainly not have lost these teeth if they had not been treated by dentists. Thus, the transition between "periodontal" and "iatrogenic"

extraction reasons is fluid. Many epidemiological surveys on oral health contain information on mean values and distribution of missing teeth, but no information on the reasons for extractions (Miller et al. 1987, Micheelis & Reich 1999). Over the whole population, the estimates of the proportion of teeth extracted because of periodontitis may be as high as 38% (for a review, see Baelum 1998). Thus, in older adults with missing teeth, a considerable proportion may have been extracted because of periodontal disease. To account for this presumed periodontaldisease effect, we included a statistical analysis with combined variables of missing teeth and periodontal measurements. Four different logistic regression models were examined. In two models, the periodontal case definition refers to AL, and in the other two models to a combination of AL and number of remaining teeth. We assumed that risk determinants for periodontal disease should also be found in the combination models, because in this population, subjects older than 55 years exhibit a substantial proportion of missing teeth.

In the present study, the relative importance of risk determinants for periodontal disease/health status was examined in multivariate models.

Material and Methods

The Study of Health in Pomerania (SHIP 0) is a medical-dental health survey of a 20-79-year-old population, approved by the local Institutional Review board. Details of sampling and methods have been described elsewhere (John et al. 2001). SHIP 0 is based on a representative, age-stratified random sample that was examined from 1997 to 2001 in Vorpommern (the northeastern-most state of Germany, formerly GDR, with 212,157 inhabitants). First, three cities, 12 towns, and then the villages were selected; second, from each of the selected communities, subjects were drawn at random from official resident data files proportional to the population size of each community, and stratified by age and gender. Only individuals with German citizenship were included. The present evaluation is based on a net random sample of 3146 data sets (response 69%, John et al. 2001, Hensel et al. 2003). After examining the raw data (n = 3146), 20 flawed data sets, 387 edentulous subjects, 127 subjects without AL records, and 17 subjects with incomplete data were excluded, resulting in 2595 subjects for analyses.

The data collection and instruments comprised four parts: oral health examination, medical examination, healthrelated interview, and risk-related questionnaire (John et al. 2001, Hensel et al. 2003). The dental examination was conducted in rotation by five trained dentists. Every six months to a year, calibration exercises were performed on a subset of persons not connected with the study, yielding an intraclass correlation of 0.82–0.91 per examiner and an inter-rater correlation of 0.84.

The following parameters were used:

Number of teeth: wisdom teeth were excluded.

Probing depth/AL: The probing depth and AL were determined in the halfmouth using a periodontal probe (PCP 11, Hu Friedy, Chicago, IL, USA) on teeth 11–17 and 41–47 or 21–27, and 31–37 at four sites (distobuccal, midbuccal, mesiobuccal, and midlingual) on the right or left side in alternate subjects. Where determination of the cemento-enamel junction was indistinct (wedge-shaped defects, fillings, crown margins), the attachment level was not recorded.

Data analysis

Odds ratios (ORs) for periodontal disease as the dependant variable were calculated with a logistic stepwise regression model (significance level: 0.05) adjusted for multiple independent variables (SPSS 10.0 Inc. Chicago, IL, USA). Four different models of periodontal disease experience were tested. In models A and \overline{C} , the healthy subjects and in models B and D the diseased subjects constituted the dependent variables. In model A, subjects had no AL \geq 4 mm, and in model B, they had severe disease with AL $\ge 4 \text{ mm}$ on \geq 51% of all sites examined; this health or disease status was defined as belonging to the lower or upper quartile of the distribution of sites with AL \geq 4 mm, respectively. In models C and D, the dependent variable consisted of the combination of the median AL extent $\geq 4 \text{ mm}$ and number of teeth. To belong to the healthy group in model C. subjects had to have <16% sites with AL $\geq 4 \text{ mm}$ and ≥ 23 teeth; in model D they exhibited $\geq 16\%$ sites with AL $\ge 4 \text{ mm}$ and had < 23teeth (Fig. 1). Age and gender were included independently in the *p*-value. A variable was included in all four models if it was significant in one model. Problems of multicollinearity were documented.



Fig. 1. Scatter plot for number of teeth and attachment loss $(AL) \ge 4$ mm. Subjects are grouped according to the number of teeth and AL. Subjects in section 4 constitute the "healthy" subjects (model C) and those in section 1 are the "diseased" subjects (model D).

Plaque, calculus: the occurrence of supragingival plaque and supragingival calculus was dichotomously evaluated on the first molar, the canine, and the central incisor at the four sites mentioned above. If that tooth was missing, the next distal tooth was used instead. The presence of plaque was determined by scratching with the periodontal probe; the presence of calculus was determined visually. Calculus and plaque in percent of sites was categorized according to a quartile distribution (number of sites with calculus: 0-10%, 11-27%, 28-45\%, \geq 45%, plaque: 0-16%, 11–33%, 34–53%, ≥54%).

Age was stratified into six decades from 20 to 79 years, reference: 20–29 years

Education was grouped into 8, 10, and 12 years of schooling

Monthly household income was categorized into quartiles.

Dental checkup: regular checkup was defined as a visit to the dentist within the last 6 months if the reason for the visit was ascribed to prevention. The subjects were asked why they visited their dentist.

Smoking was categorized into "non-, former, and current smoker". We discerned between smokers with "<10 cigarettes/day", "10–19 cigarettes/ day" and " \geq 20 cigarettes/day". A current smoker was defined as someone who usually smokes more than one cigarette/ day. A former smoker was defined as someone who used to smoke regularly. We did not include packyears since this variable did not provide further information.

Medication: defined as consumption of any medication within the past week prior to SHIP examination.

History (yes/no) of cardiovascular disease, diabetes, arthritis, hypertension, osteoporosis, allergy (without and with drugs), asthma, and medication for asthma, hypertension and allergy. The health status was defined according to self-reported information.

Results Description of sample

Our sample consisted of 52% females and 48% males, distributed proportionally among the six age categories, except in the youngest and the oldest decade. At the time of German unification (1990), 98% of the investigated subjects lived in eastern Germany and 1.6% came from the western part of Germany. Nineteen percent attended school for 12 years, 49% for 10 years, and 32% for 8 or fewer years. Thirty-six percent were non-smokers and 32% current smokers. More men than women smoked (current/former smoker: 35%/ 44% (male) versus 29%/22% (female)). The most prevalent self-reported disease was allergy (25%), and 8% of all subjects took anti-allergy medications. Six percent had diabetes mellitus. AL increased nearly linearly for thresholds of mean AL \geq 4 and 6 mm. On average, subjects in the second decade had no AL on about 70% of their sites and AL $\geq 2 \text{ mm}$ on 30% of their sites, while the oldest subjects had AL on over 90% of their sites: on 30% of their sites they had AL $\geq 6 \text{ mm}$, on 30% AL of 4-6 mm, and on 30% of sites AL of 2-4 mm (Fig. 2). With increasing age, the mean number of teeth decreased linearly from 26.1 to 5.5 (Fig. 3).

For the four models, problems of multicollinearity were observed for income with age. Only age appears for this reason. Furthermore, the estimators for plaque and calculus are correlated.

Model A (chance for periodontal health)

In model A, the periodontally healthy subjects (no AL ≥ 4 mm) constituted



Fig. 2. Attachment loss as median percentage ≥ 2 , ≥ 4 , ≥ 6 mm across the population (dentate subjects).



Fig. 3. Tooth loss (median and mean) (dentate subjects).

the dependent variable; in this model we searched for determinants that favored periodontal health. An OR < 1 implies that a risk determinant exerted a negative influence on AL and OR > 1is protective against AL. The age of periodontally healthy subjects (A healthy) and the periodontally diseased group (A diseased) lay at 30 and 52 years, respectively. The number of teeth in A diseased was 21 and in A healthy 26. In A healthy, no AL $\geq 4 \text{ mm}$ was present, and in A diseased 28% of sites exhibited AL ≥ 4 mm. The A healthy group had better oral hygiene conditions than the *A diseased* group (plaque: 20%) versus 37%, calculus 16% versus 31%). A much smaller proportion of men (39%) belonged to the A healthy group than to the A diseased group (51%). Smoking was somewhat more prevalent in the A diseased group than in A healthy group (Table 1).

Regression analysis. Men have a 0.7fold lower chance than females of belonging to the healthy group. The more plaque or calculus present, the lower the chance is of belonging to the healthy group; for both variables, there is a significant dose-response relationship. The more the subjects currently smoked, the lower the chance they had of belonging to the healthy group. Subjects smoking ≥ 20 cigarettes a day had a 0.57 lower chance of belonging to the healthy group. Education, former smoking, allergy or dental checkup did not significantly influence the chance of belonging to the healthy group (Table 2).

Model B (risk for periodontal disease)

In model B, the periodontally diseased subjects (AL $\ge 4 \text{ mm on } \ge 51\%$ of all sites examined) constituted the dependent variable. In this model, an OR < 1implies that a protective determinant exerts a positive influence on periodontal health and an OR >1 denotes a risk determinant for AL. The B diseased group had a much higher age (61 years) than the *B* healthy group (42 years). *B* diseased had 13 teeth and B healthy 24 teeth. In *B* diseased, AL $\ge 4 \text{ mm}$ was found in 80% of all sites, and in only 6% of sites in B healthy. The oral hygiene conditions were better in Bhealthy, with 29% plaque and 25% calculus, than in *B* diseased with 50% plaque and 41% calculus. Education

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Model]	Model A	Model B		
	healthy A (first quartile)	diseased A (second, third, fourth quartile)	diseased B (fourth quartile)	healthy B(first, second, third quartile)	
Number of teeth	26.0 ± 5.0	21.0 ± 10.0	13.0 ± 12.2	24.0 ± 7.0	
$\% \text{ AL} \ge 4 \text{ mm}$	0 ± 0	28.0 ± 53.0	79.5 ± 28.2	6.0 ± 21.0	
Ν	617	1978	630	1965	
Age	30.1 ± 14.0	52.1 ± 21.2	61.4 ± 16.8	41.5 ± 23.0	
% Male	39.4	50.9	58.3	44.9	
Plaque	20.0 ± 28.0	37.0 ± 34.0	50.0 ± 44.0	29.0 ± 30.0	
Calculus	16.0 ± 29.0	31.0 ± 38.0	41.0 ± 45.0	25.0 ± 31.5	
Bleeding	20.0 ± 27.0	37.0 ± 46.0	55.0 ± 49.2	25.0 ± 33.0	
Regular dental check up	42.8	38.2	30.7	42.1	
Education (years)					
8	11.5	39.0	60.6	23.5	
10	62.2	44.6	30.3	54.8	
12	26.3	16.3	9.0	21.8	
Monthly income	1250 ± 950	1250 ± 875	1750 ± 500	1250 ± 950	
Current smoker (cigarettes/day	y)				
<10	16.0	7.6	6.5	10.6	
10–19	13.6	11.6	9.5	12.9	
≥20	7.8	9.8	13.0	8.1	
Former smoker (cigarettes/day	7)				
<10	14.9	15.9	17.1	15.2	
10–19	4.5	8.4	10.6	6.5	
≥20	6.5	10.9	12.9	8.9	
Diabetes mellitus	1.0	7.7	13.0	3.9	

The reference group for model A is composed of subjects from the second, third and fourth quartiles. The reference group for model B is composed of first, second, third quartiles. AL, attachment loss.

4.2

8.1

4.6

15.3

6.7

2.7

67.5

0.8

5.2

1.5

21.1

13.9

2.3

60.9

7.0

9.2

6.7

10.8

4.9

3.5

74.0

AL, attachment loss.

Cardiovascular disease

Allergy without drug

Arthritis

Osteoporosis

Allergy drug

Asthma drug

Any drug

was much better in *B healthy* than *B diseased*. In *B diseased*, less than 10% subjects attended school more than 10 years, where as in *B healthy*, 22% of all subjects went to school for 12 years. In the *B diseased* group, more subjects were or had been moderate or heavy smokers, 13% were heavy current and former smokers, while in *B healthy*, only 8% were heavy current and 9% heavy former smokers (Table 1).

Regression analysis. Both parameters of oral hygiene had an overall significant destructive influence on periodontal conditions. Only very high plaque accumulation had a significant OR of 2.3, where for calculus a significant dose–response relationship was found (OR, 1.3–2.8). Current smoking exerted a detrimental effect on periodontal health in relation to the smoking dose (OR, 1.9–6.8). Former smoking affected the periodontal status irrespective of dose (OR, about 1.6). A high level of schooling had a protective effect on periodontal health (OR, 0.4) (Table 2).

Model C (chance for periodontal health and many teeth remaining)

To belong to the healthy group in model C, subjects had to have <16% sites with AL $\geq 4 \text{ mm}$ and ≥ 23 teeth (Fig. 1). In model C, we searched for determinants, which favored retention of periodontally healthy teeth. An OR <1 denotes a risk determinant and OR > 1 a chance to retain healthy teeth. The age of the periodontally healthier subjects (C healthy) were 35 years and of more diseased subjects (C diseased) 55 years, respectively. C healthy had 26 teeth and C diseased 19 teeth. In C healthy, AL of $\geq 4 \text{ mm}$ was found in only 1% of all sites, and in C diseased in 39% of sites. C healthy subjects had better oral hygiene conditions than the Cdiseased group (plaque: 25% versus 40%, calculus 20% versus 33%). Among C *healthy* subjects, 28% had 12 years of schooling in comparison with 13% of the *C* diseased subjects. Heavy regular and former smoking was more prevalent in the *C* diseased than in the *C* healthy group (prevalence of heavy regular and heavy former smokers: about 12% versus 7%). Twenty-one percent of the *C* healthy and 14% of *C* diseased had an allergy, 13% of the *C* healthy and 7% *C* diseased took antiallergic drugs (Table 3). Forty-six percent of *C* healthy versus 35% of the *C* diseased subjects visited their dentists regularly.

2.3

6.8

3.0

18.5

9.6

2.3

63.4

Regression analysis. The more plaque present, the lower was the probability of retaining more teeth in a periodontal healthy condition (OR, 0.6-0.3). The higher the education, the higher was the chance to belong to the *C healthy* group (OR, 1.8-3.2). The higher the daily smoking dose of current smokers, the lower was the chance of retaining

	AL			AL and teeth				
	Model A Little AL		Model B High AL		Model C Little AL and many teeth		Model D High AL and few teeth	
	р	OR (95%-CI)*	р	OR (95%-CI)*	р	OR (95%-CI)*	р	OR (95%-CI)*
Sex (ref: female)	0.006	0.70 (0.55-0.90)	0.468	1.10 (0.85–1.42)	0.064	1.25 (0.99–1.58)	0.013	0.73 (0.58-0.94)
Plaque (ref: little)	< 0.001		< 0.001		< 0.001		< 0.001	
minor	< 0.041	0.72 (0.53-0.99)	0.272	0.81 (0.56-1.18)	0.005	0.64 (0.47-0.87)	0.009	1.56 (1.12-2.17)
moderate	0.001	0.54 (0.38-0.77)	0.409	1.16 (0.81-1.67)	< 0.001	0.46 (0.33-0.65)	< 0.001	2.07 (1.46-2.92)
much	< 0.001	0.34 (0.21-0.55)	< 0.001	2.28 (1.60-3.25)	< 0.001	0.28 (0.18-0.44)	< 0.001	2.87 (1.99-4.16)
Calculus (ref: little)	0.002		< 0.001		0.327		0.08	
minor	0.020	0.69 (0.51-0.94)	0.112	1.33 (0.94-1.88)	0.789	0.96 (0.70-1.31)	0.607	0.92 (0.66-1.27)
moderate	0.023	0.66 (0.46-0.94)	0.005	1.68 (1.17-2.41)	0.862	0.97 (0.68-1.37)	0.391	0.86 (0.61-1.22)
much	< 0.001	0.41 (0.26-0.66)	< 0.001	2.83 (2.00-4.00)	0.117	0.72 (0.48-1.09)	0.059	1.42 (0.99-2.04)
Education (ref: 8 years)	0.552		< 0.001		< 0.001		< 0.001	
10	0.550	1.12 (0.78-1.60)	0.110	0.81 (0.62-1.05)	< 0.001	1.82 (1.33-2.48)	< 0.001	0.62 (0.48-0.80)
12	0.287	1.25 (0.83-1.87)	< 0.001	0.44 (0.31-0.63)	< 0.001	3.23 (2.27-4.59)	< 0.001	0.34 (0.25-0.48)
Smoking (ref: no.)	0.016		< 0.001		< 0.001		< 0.001	
Current (cigarettes/day)								
<10	0.573	1.12 (0.76–1.63)	0.009	1.86 (1.17-2.96)	0.016	0.62 (0.42-0.91)	< 0.001	2.17 (1.41-3.32)
10-19	0.009	0.61 (0.42-0.88)	< 0.001	2.46 (1.62-3.76)	< 0.001	0.45 (0.31-0.65)	< 0.001	2.52 (1.70-3.71)
≥20	0.013	0.57 (0.36-0.89)	< 0.001	6.82 (4.34–10.71)	< 0.001	0.28 (0.19-0.43)	< 0.001	5.07 (3.30-7.79)
Former (cigarettes/day)								
<10	0.312	0.83 (0.58–1.19)	0.003	1.67 (1.19–2.35)	0.080	0.74 (0.53-1.04)	0.143	1.28 (0.92–1.79)
10–19	0.333	0.76 (0.45-1.32)	0.052	1.51 (1.00-2.28)	0.658	0.90 (0.58-1.42)	0.007	1.78 (1.17-2-70)
≥20	0.468	1.19 (0.75–1.88)	0.035	1.54 (1.03-2.29)	0.044	0.65 (0.43-0.99)	< 0.001	2.02 (1.37-2.98)
Allergy (ref: no.)	0.076		0.484		0.004		0.019	
without drug	0.744	1.05 (0.78–1.42)	0.310	0.84 (0.60-1.17)	0.084	1.28 (0.97–1.71)	0.273	0.84 (0.62–1.14)
drug	0.023	1.54 (1.06-2.23)	0.455	0.83 (0.51-1.35)	0.002	1.84 (1.26-2.70)	0.007	0.54 (0.34-0.84)
Dental check (ref: no.)	0.453	0.91 (0.72–1.16)	0.082	0.82 (0.65-1.03)	0.003	1.40 (1.12–1.75)	0.032	0.79 (0.63-0.98)
Intercept	< 0.001	7.06	< 0.001	0.001	< 0.001	9.32	< 0.001	0.006
Nagelkerke R^2		0.47		0.43		0.55		0.52

Table 2. ORs and CIs for models A, B, C and D

*Adjusted by age (ref: 20-29 years, 10 years age groups).

ORs, odds ratios; CI, confidence interval; AL, attachment loss; ref, reference.

healthy teeth (OR, 0.6–0.3). Within the former smoker category, only the heavy smokers had a lower chance of belonging to the healthy group. Antiallergic drug intake (OR, 1.8) had a protective effect on retaining periodontally healthy teeth. Subjects who regularly visited their dentist for preventive measures had a 1.4 greater chance of retaining more teeth healthy (Table 2).

Model D (risk for high AL and few teeth remaining)

To belong to the diseased group in model D, subjects had to have $\geq 16\%$ sites with AL ≥ 4 mm and < 23 teeth (Fig. 1). In model D, we searched for determinants, which are associated with high AL and few retained teeth. In this model, an OR < 1 denotes a low risk of just having a few teeth remaining and OR > 1 a risk determinant of having only a few teeth remaining and these exhibit high AL.

The mean age of the *D* diseased subjects and of the *D* healthy subjects was 60 and 39 years, respectively, *D*

diseased had 15 teeth and D healthy 25 teeth. In D diseased subjects, AL \geq 4 mm comprised 61% of all sites and in D healthy 3% of sites. The D diseased group had worse oral hygiene conditions than the D healthy group (plaque: 45% versus 29%, calculus 37% versus 25%). The D healthy subjects were much better educated than the Ddiseased subjects (24% versus 10% with 12 years of schooling, respectively). Among the D diseased subjects the "former" smoking category was more prevalent than among D healthy subjects (38% versus 29%). Seventeen percent of the D diseased group and 30% of the D healthy group reported having an allergy or took anti-allergy medications. Thirty-three percent of the D diseased and 43% of D healthy subjects regularly visited their dentists (Table 3).

Regression analysis. Males had a 0.7 lower risk of belonging to the *D* diseased group than females. Plaque accumulation exhibited a significant,

detrimental dose-response effect on periodontal health (OR, 1.6-2.9). A higher educational level lowered the risk of belonging to D diseased (10 years of schooling, OR = 0.6; 12 years of schooling, OR = 0.3). Current smokers had a risk of up to 5.1-times higher than non-smokers belonging to the Ddiseased group. A similiar trend could be detected in the former smoking category: heavy former smokers had a twofold higher risk than non-smokers belonging to *D* diseased. Being allergic (OR = 0.5) or taking allergy medications (OR = 0.8) lowered the risk of being among the D diseased subjects. Regular dental checkups also lowered the risk of having only a few teeth and these in bad periodontal condition (Table 2).

Discussion

General consideration

The validity and reliability of variables are important issues for each study. The \pm 1-mm agreement of replicate AL mea-

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Model	М	odel C	Model D		
	Diseased (High AL or few teeth)	Healthy (Little AL and many teeth)	Healthy (Little AL or many teeth)	Diseased (High AL and few teeth)	
Number of teeth	19.0 ± 10.0	26.0 ± 3.0	25.0 ± 4.0	15.0 ± 10.0	
$\% \text{ AL} \ge 4 \text{ mm}$	39.0 ± 53.0	1.0 ± 5.0	3.0 ± 12.0	61.0 ± 48.8	
Ν	1615	980	1655	940	
Age	55.3 ± 20.0	34.9 ± 15.4	38.8 ± 20.2	60.0 ± 17.1	
% male	50.2	44.7	45.9	52.0	
Plaque	40.0 ± 31.0	25.0 ± 25.0	29.0 ± 29.0	45.0 ± 41.0	
Calculus	33.0 ± 39.0	20.0 ± 25.0	25.0 ± 33.0	37.0 ± 40.8	
Bleeding	41.0 ± 45.0	20.0 ± 29.0	25.0 ± 35.0	45.0 ± 50.0	
Regular dental check up	35.3	45.9	43.0	32.9	
Education (years)					
8	46.1	10.0	18.1	57.9	
10	40.9	61.9	58.3	32.1	
12	13.0	28.1	23.6	10.0	
Monthly income	1250 ± 875	1250 ± 950	1250 ± 950	1750 ± 500	
Current smoker (cigarettes/	/day)				
<10	7.6	13.1	11.2	6.9	
10–19	11.0	13.9	13.5	9.6	
≥20	10.3	7.6	8.6	10.4	
Former smoker (cigarettes/	'day)				
<10	15.8	15.5	15.7	15.6	
10–19	8.4	6.0	6.0	10.1	
≥20	11.7	6.7	8.0	13.1	
Diabetes mellitus	8.9	1.5	2.7	12.0	
Cardiovascular disease	4.9	1.0	1.6	6.6	
Arthritis	8.8	5.1	6.4	9.1	
Osteoporosis	5.3	1.4	2.2	6.7	
Allergy without drug	13.9	21.1	19.1	12.3	
Allergy drug	5.8	12.9	10.6	4.6	
Asthma drug	2.7	2.4	2.1	3.4	
Any drug	70.5	58.5	61.2	74.3	

Table 3. Characteristics of risk determinants of model C and D as a median (\pm inter quartile range) or as a proportion

The reference group of model C is composed of subjects from sections 1-3 in Fig. 1. The reference group for the model D is composed of subjects from sections 2-4.

AL, attachment loss.

surement differences varied between 83% and 95%, and the difference of the standard deviation between 0.9 and 1.2 mm. The intra-individual intra-class correlation coefficients were between 0.8 and 0.92 (Hensel et al. 2003). The ICC over all examiners was 0.85. These data are comparable with those of Osborn et al. (1990, 1992) and Badersten et al. (1984). Information on smoking, dental-appointment attendance, and socioeconomic factors was obtained with a questionnaire and an interview. The proportion of smokers (32%) is comparable with data from the Social Ministry of the Federal State of Mecklenburg-Vorpommern (29%, Sozialministerium Mecklenburg-Vorpommern 2001). Although it is conceivable that subjects who provide answers in the context of a dental/general health survey may tend to exaggerate, their dental attendance level or frequency of daily tooth brushing were similar to another representative dental health study in Germany (IDZ 1996).

Periodontal treatment has not been widely performed in this part of Germany. Up to the German unification in 1990, periodontal treatment and effective maintenance was hampered by the lack of adequate professional and oral home-care supplies. It is evident that this population is periodontally nearly untreated. For this geographical region, Splieth et al. (2002) have shown that a considerable proportion of teeth had been extracted before periodontitis made it necessary.

Data analysis

Establishment of a definition of a periodontal case is arbitrary (Baelum & Lopez 2003). Various cut-off points to define a periodontal case based on AL, bone loss, or pocket depth had been suggested, but none are universally agreed upon. To circumvent this problem, a statistical definition of disease was chosen and quartiles were used for

thresholding (Fletcher et al. 1996). Furthermore, investigators usually look for factors that make people ill; in this study, we also included the reverse question of what keeps subjects periodontally healthy. For all models, we found nearly identical results, i.e., the ORs were reversed. Thus, there exist no variables that are only connected with health or with disease. The variable "gender" was the only one that appeared contradictory in the reverse analyses: Females had a higher chance of belonging to the periodontally healthier group, and at the same time, they had a higher risk of having fewer teeth than males. For further discussion of this point, see below.

The comparison of the results from the analysis with the combined variables (tooth loss plus periodontal disease status (models C and D)) versus periodontal disease status (models A and B) showed that the independent variables education and plaque had higher ORs for models C and D than for models A and B. Furthermore, the variables calculus and dental checkup were not significant in periodontal disease models (A and B), but were significant in combination models (C and D). This may be interpreted to mean that the dentist-patient interaction which is strongly influenced by socioeconomic factors - has a greater influence on preserving or extracting a tooth than these factors exert on the actual periodontal disease status. For example, in subjects with less education and bad oral hygiene (much plaque), dentists may be more willing to extract teeth than in better-educated subjects with better oral hygiene habits. Another interpretation of these data may be that less-educated subjects are not as interested in retaining teeth, or their financial resources are not as great as those of more highly educated subjects. The variable dental checkup also seems to reflect the dentist-patient interaction rather than treatment needs. Although regular dental checkup does not prevent the initation or progression of periodontal disease, regular dental attendance strongly influences the dentist's treatment decision to extract or not to extract a tooth. A regular attender has a better chance of retaining his or her teeth than an erratic attender to a German dental office. With a different analytical approach, Weihrauch (2003) searched for predictors of tooth loss using the same data (subgroup < 55years old). She found the same predictors (education, income, smoking, plaque control) and the interactions gender/marital status and gender/education/income.

Risk factors

Beck (1998) classified putative risk factors for periodontal disease into six classes: sociodemographic factors (age, gender, income, education, etc.), psychological or environmental factors (dental awareness, social support, etc.), physical and medical factors (systemic diseases, drugs, etc.), behavioral factors (smoking, tooth brushing, etc.), oral factors (subgingival microbiota, saliva, etc.), and host defense factors. Except for oral and host factors, some of the other putative risk factors were incorporated into our multivariate models. In our models, we included 19 variables, but only eight turned out to be statiscally significant.

Plaque and calculus

Our models (A and B) confirm plaque and calculus as risk determinants for periodontitis. This is in accordance with a number of reports (Ismail et al. 1986, Grossi et al. 1995, Corbet et al. 2001). It is established that mass of supragingival plaque is causative for gingivitis; however, its association with periodontitis is controversial. Baelum et al. (1988, 1996) showed a widespread variation of periodontal disease in Tanzanian and Chinese poulations, irrespective of supragingival plaque accumulation. However, the long-term intervention study by Axelsson et al. (1991) showed that patients with little supragingival plaque had very little detectable periodontal disease after 15 years of continuous supportive treatment. Supragingival plaque may provide a favorable environment for colonization with specific subgingival pathogens (Ximenez-Fvvie et al. 2000). Calculus and its relationship to periodontitis is complex. Since they coexist, it is difficult to prove that calculus per se is a risk determinant for periodontitis. It is probably a deposit that forms during the initiation of periodontal disease (Beck 1988).

Education

Oliver et al. (1991) showed that the strongest difference among socioeconomic variables was education. This observation agrees with our results. A high educational level is protective against periodontitis and tooth extraction in models B-D; it decreases the risk of suffering from periodontitis or from periodontitis and tooth loss by about 300%. The relationship between a low level of education and an increased risk of periodontitis has also been reported by other authors (Beck et al. 1990, Locker & Leake 1993, Dolan et al. 1997, Treasure et al. 2001). The more highly educated subjects are usually more interested in oral hygiene, have better access to medical care, and are able to afford dental care. Although in Germany, dental treatment has been covered to a large extent by medical insurance companies, the present health policy did not succeed in evening out these socioeconomic differences. Even more astonishing is the fact that the former East German state, which negated the influence of socioeconomic status and which wanted to eradicate social imbalance, did not succeed in

providing the same access to care for everyone, irrespective of education. Nearly all SHIP subjects were brought up in East Germany, and their oral health status reflects the history of dental care of the former East German state to a certain degree.

Smoking

We confirmed that at the present time, smoking is the major risk determinant for periodontal disease. Former heavy smokers (models B and D: OR, 1.6 and 2.0) had a lower risk of disease than current heavy smokers had (OR, 6.8/ 5.1). We found a direct and linear doseresponse between smoking quantity and destructive periodontal disease, as already described by Grossi et al. (1995) and Tomar & Asma (2000). Longitudinal studies have confirmed that smokers exhibited greater disease progression than did non-smokers, as well as more tooth loss (Holm 1994, Machtei et al. 1999, König et al. 2002). Based on NHANES III data. Tomar & Asma (2000) found ca. 75% of the identified cases of periodontitis to be in smokers. The mechanisms by which cigarette smoking affects the periodontium are quite diverse. There may be an interaction between genetic background factors and smoking (Meisel et al. 2002, 2003). Furthermore, direct effects may cause a constriction of the gingival blood vessels and have deleterious effects on leukocyte and IgG2 function (Zambon et al. 1996).

Gender

The gender findings are not easy to understand: males have a 0.7 times lower chance of being among the periodontally healthy subjects than do females (model A). This agrees with many other reports (Miller et al. 1987, Horning et al. 1992, Douglass et al. 1993. Grossi et al. 1994. 1995. Dolan et al. 1997). At the same time (model D). males had a 0.7 times lower risk than females of belonging to group D with fewer teeth. Further analysis of our data confirmed that males had more periodontal disease but retained more teeth than females (analysis not shown here). To reconcile these contradictory observations, the following explanations are offered: a biological explanation may be that males (an observation which is valid across most mammal taxa) are more susceptible to infection by parasites and microbes (Moore & Wilson 2002). Another reason for the beneficial effect bestowed by belonging to the female gender may be a protective estrogen level (Owens 2002). A third reason may be that more men have risky lifestyle behaviors than females (smoking, worse oral hygiene, lower health awareness). That males have more teeth than females may be based on socio-economic factors or on dentist–patient interrelation. Further detailed examinations of our data on this topic still have to be performed.

Allergy

In models C and D, allergy medications are protective against periodontitis (OR, 1.8/0.5). The protective influence of suffering from allergy/taking allergy medications was first described by Grossi et al. (1994). They suspected that allergy medications modulate the immune response of the host to periodontal pathogens.

Conclusion

The present study explored the relative importance of risk determinants for the periodontal disease or health status in four multivariate models to provide the best prediction of risk. Irrespective of the chosen model, we ascertained age, education, supragingival plaque, and smoking as risk determinants. Smoking was the most important risk determinant. About 43-55% of the variation could be explained with our models. Our findings are in agreement with the conclusion of Baelum (1998), who showed in an extensive review that for the factors age, low income, smoking, Porphyromonas gingivalis and Prevotella intermedia, that circumstantial evidence for a causal role is present. To develop better prediction models, it will be necessary to include more microbiological, host-response, and genetic data in future studies.

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References

- Axelsson, P., Lindhe, J. & Nyström, B. (1991) On the prevention of caries and periodontal disease. Results of a 15-year longitudinal study in adults. *Journal of Clinical Periodontology* 18, 182–189.
- Badersten, A., Nilveus, R. & Egelberg, J. (1984) Reproducibility of probing attachment level measurements. *Journal of Clinical Periodontology* 11, 475–485.
- Baelum, V. (1998) The epidemiology of destructive periodontal disease. PhD thesis, Aarhus.
- Baelum, V., Chen, X., Manji, F., Luan, W. M. & Fejerskov, O. (1996) Profiles of destructive periodontal disease in different populations. *Journal of Periodontal Research* 31, 17–22.
- Baelum, V., Fejerskov, O. & Manji, F. (1988) Diseases in adult Kenyans. *Journal of Clinical Periodontology* 15, 445–452.
- Baelum, V. & Lopez, R. (2003) Defining and classifying periodontitis: need for a paradigm shift? *European Journal of Oral Science* 111, 2–6.
- Beck, J. D. (1988) Epidemiologic changes in in older adult periodontal disease. *Gerodontol*ogy 7, 103–107.
- Beck, J. D. (1998) Risk revisited. Community Dentistry 26, 220–225.
- Beck, J. D., Koch, G. G., Rozier, R. G. & Tudor, G. E. (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.
- Corbet, E. F., Wong, M. C. M. & Lin, H. C. (2001) Periodontal conditions in adult southern Chinese. J 80, 1480–1485.
- Dolan, T. A., Gilbert, G. H., Ringelberg, M. L., Legler, D. W., Antonson, D. E., Foerster, U. & Heft, M. W. (1997) Behavioral risk indicators of attachment loss in adult Floridians. *Journal of Clinical Periodontology* 24, 223–232.
- Douglass, C.W, Jette, A. M., Fox, C. H., Tennstedt, S. L., Joshi, A. & Feldman, H. A. (1993) Oral health status of the elderly in New England. *Journal of Gerontology* 48, M39–M46.
- Fletcher, R. H., Fletcher, S. W. & Wagner, E. H. (1996) *Clinical Epidemiology*, 3rd edition, pp. 19–42. Philadelphia: Williams & Wilkins.
- Genco, R. J. (1996) Current view of risk factors for periodontal disease. *Journal of Periodontology* 67, 1041–1049.

- Grossi, S. G., Genco, R. J., Machtei, E. E., Ho, A. W., Koch, G., Dunford, R., Zambon, J. J. & Hausmann, E. (1995) Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. *Journal of Periodontology* 66, 23–29.
- Grossi, S. G., Zambon, J. J., Ho, A. W., Koch, G., Dunford, R. G., Machtei, E. E., Norderyd, O. M. & Genco, R. J. (1994) Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *Journal of Periodontology* 65, 260–267.
- Hensel, E., Gesch, D., Biffar, R., Bernhardt, O., Kocher, T., Splieth, C., Born, G. & John, U. (2003) Study of health in pomerania (SHIP): a health survey in an East German region – objectives and design on the oral health section. *Quintessence International* 34, 370–378.
- Holm, G. (1994) Smoking as an additional risk for tooth loss. *Journal of Periodontology* 65, 996–1001.
- Horning, G. M., Hatch, C. L. & Cohen, M. E. (1992) Risk indicators for periodontitis in a military treatment population. *Periodontology* **63**, 297–302.
- IDZ, Institut der Deutschen Zahnärzte (ed.) (1996) Risikogruppenprofile bei Karies und Parodontitis. Statistische Vertiefungsanalysen der Mundgesundheits-studien des IDZ von 1989 und 1992. Köln: Deutscher Ärzte-Verlag.
- Ismail, A. I., Eklund, S. A., Burt, B. A. & Calderone, J. J. (1986) Prevalence of deep periodontal pockets in New Mexico adults aged 27 to 74 years. *Journal of Public Health Dentistry* 46, 199–206.
- John, U., Greiner, B., Hensel, E., Lüdemann, J., Pieck, M., Sauer, S., Adam, C., Born, G., Alte, D., Greiser, E., Härtel, U., Hense, H. W., Haerting, J., Willich, S. & Kessler, C. (2001) Study of health in pomerania (SHIP): a health examination survey in an East German region. Objectives and design. Soz 46, 186–194.
- Kay, E. & Nuttall, N. (1997) Clinicial Decision Making. An Art or a Science. London: British Dental Association.
- Kocher, T., Sawaf, H., Fanghänel, J., Timm, R. & Meisel, P. (2002) Association between bone loss in periodontal disease and polymorphism of *N*-acetyltransferase (NAT2). *Journal of Clinical Periodontology* 29, 21–27.
- König, J., Plagmann, H. C., Rühling, A. & Kocher T. (2002) Tooth loss and pocket probing depths in compliant periodontally treated patients: a retrospective analysis. *Journal of Clinical Periodontology* 29, 1092–1100.
- Kornman, K. S., Crane, A., Wang, H. Y., di Giovine, F. S., Newman, M. G., Pirk, F. W., Wilson, T. G. Jr., Higginbottom, F. L. & Duff, G.W (1997) The interleukin-1 genotype as a severity factor in adult periodontal disease. *Journal of Clinical Periodontology* 24, 72–77.
- Locker, D. & Leake, J. L. (1993) Risk indicators and risk markers for periodontal disease experience in older adults living

independently in Ontario, Canada. *Journal of Dental Research* 72, 9–17.

- Machtei, E. E., Hausmann, E., Dunford, R., Grossi, S., Ho, A., Davis, G., Chandler, J., Zambon, J. & Genco, R. J. (1999) Longitudinal study of predictive factors for periodontal disease and tooth loss. *Journal* of Clinical Periodontology 26, 374–380.
- Meisel, P., Siegemund, A., Dombrowa, S., Sawaf, H., Fanghänel, J. & Kocher, T. (2002) Smoking and polymorphisms of the interleukin-1 gene cluster (IL-1alpha, IL-1beta, and IL-1RN) in patients with periodontal disease. *Journal of Periodontology* 73, 27–32.
- Meisel, P., Siegemund, A., Gimm, R., Herrmann, F. H., John, U., Schwahn, C. & Kocher, T. (2003) The interleukin-1 polymorphism, smoking, and the risk of periodontal disease in the population-based SHIP study. *Journal of Dental Research* 82, 189–193.
- Micheelis, W. & Reich, E., (eds.) (1999) Dritte deutsche Mundgesundheitsstudie. Köln: Deutscher Ärzteverlag.
- Miller, A. J., Brunelle, J. A., Carlos, J. P., Brown, L. J. & Löe, H. (1987) Oral Health of United States Adults: National Findings. Bethesda, MD: National Institute of Dental Research, NIH Publication No87-2868.
- Moore, S. L. & Wilson, K. (2002) Parasites as viability cost of sexual selection in natural population of mammals. *Science* 297, 2015–2018.
- Page, R. C. & Kornman, K. S. (1997) The pathogenesis of human periodontitis: an introduction. *Periodontology 2000* 14, 9–11.

- Phillips, K. R., Reifel, N. & Bothwell, E. (1991) The oral health status, treatment needs, and dental utilization patterns of native American elders. *Journal of Public Health and Dentistry* **51**, 228–233.
- Oliver, R., Brown, L. & Löe, H. (1991) Variations in the prevalence of and extent of periodontitis. *Journal of the American Dental Association* **122**, 43–48.
- Osborn, J. B., Stoltenberg, J. L., Huso, B. A., Aeppli, D. M. & Pihlstrom, B. L. (1990) Comparison of measurement variability using a standard and constant force periodontal probe. *Journal of Periodontology* 61, 497–503.
- Osborn, J. B., Stoltenberg, J. L., Huso, B. A., Aeppli, D. M. & Pihlstrom, B. L. (1992) Comparison of measurement variability in subjects with moderate periodontitis using a conventional and constant force periodontal probe. *Journal of Periodontology* 63, 283–289.
- Owens, I. P. (2002) Ecology and evolution. Sex differences in mortality rate. *Science* 297, 2008–2009.
- Sozialministerium Mecklenburg-Vorpommern (ed.) (2001) *Gesundheitsbericht 1999*. Schwerin.
- Splieth, C., Giesenberg, J., Fanghanel, J., Bernhardt, O. & Kocher, T. (2002) Periodontal attachment level of extractions presumably performed for periodontal reasons. *Journal of Clinical Periodontology* 29, 514–518.
- Tomar, S. L. & Asma, S. (2000) Smokingattributable periodontitis in the United States:

findings from NHANES III. Journal of Periodontology **71**, 743–751.

- Treasure, E., Kelly, M., Nuttall, N., Bradnock, G. & White, D. (2001) Factors associated with oral health: a multivariate analysis of results from the 1998 adult health survey. *British Dental Journal* 190, 60–68.
- Weihrauch, D. (2003) Entwicklung eines epidemiologischen Modells zur Identifizierung von Faktoren für Zahnverlust- Ergebnisse der Study of Health in Pomerania. Medical thesis, Greifswald.
- Ximenez-Fyvie, L. A., Haffajee, A. D. & Socransky, S. S. (2000) Microbial composition of supra- and subgingival plaque in subjects with adult periodontitis. *Journal of Clinical Periodontology* 27, 722–732.
- Zambon, J. J., Grossi, S. G., Machtei, E. E., Ho, A. W., Dunford, R. & Genco, R. J. (1996) cigarette smoking increases the risk for subgingival infection with periodontal pathogens. *Journal of Periodontology* 67, 1050–1054.

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