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# Tooth loss and periodontitis by socio-economic status and inflammation in a longitudinal population-based study

**Sabine Buchwald[1], Thomas Kocher[1], Reiner Biffar[2], Ali Harb[3], Birte Holtfreter[1] and Peter Meisel[1]**

[1]Unit of Periodontology, Centre of Oral Health, University of Greifswald, Greifswald, Germany; [2]Unit of Prosthodontics, Gerodontology and Biomaterials, Centre of Oral Health, University of Greifswald, Greifswald, Germany; [3]Institute of Community Medicine, University of Greifswald, Greifswald, Germany

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***Abstract***

**Aim:** To examine the associations between factors of socio-economic status (SES), systemic inflammation and the progression of periodontitis and incidence of tooth loss.

**Material and Methods:** Data of 2566 participants from the Study of Health in Pomerania (SHIP) with a 5-year follow-up were analysed. We evaluated attachment loss and tooth loss with regard to social variables including education, income, marital status and related risks such as smoking and obesity.

**Results:** Socio-economic factors were associated with the progression of attachment level and tooth loss during the follow-up period. Low education and low income were associated with tooth loss (incidence risk ratio IRR 1.63, $p < 0.001$ and 1.25, $p < 0.001$ respectively) and also progression of mean clinical attachment level ($p < 0.010$ and $p = 0.046$ respectively). SES as well as smoking and obesity were also associated with increased levels of C-reactive protein (CRP) with effect modifications between SES factors and CRP, also between gender and marital status. Tooth loss was associated with disadvantageous SES, particularly under conditions of high CRP levels ($p < 0.001$).

**Conclusions:** The progression of periodontal disease is influenced by socio-economic factors. Effect modifications of socio-behavioural factors by CRP indicate that under conditions of systemic inflammation adverse SES effects may be aggravated.

Key words: C-reactive protein; follow-up study; periodontitis; socioeconomic status; tooth loss

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Periodontitis is an inflammatory chronic disease of the gingival tissue caused by bacteria forming the subgingival biofilm on the tooth root surface. The inflammatory reaction elicited by the biofilm leads to gingival bleeding, pocket formation, attachment and bone loss and eventually to tooth loss as final outcome. It has been shown that periodontitis is a multifactorial disease modified by numerous risk factors. Some of these risk factors are strongly influenced by socio-economic factors especially with respect to health behaviour and access to health services.

Current knowledge of socio-economic risk factors influencing periodontal health is mainly based on cross-sectional data. In such studies, when adjusted for diabetes or smoking,

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socio-economic factors lost their significance in multivariate analyses (Locker & Leake 1993, Borrell & Papapanou 2005). There is still demand for long-term follow-up studies of large population-representative samples to identify socio-economic status (SES) as a possible independent risk factor (Locker & Leake 1993, Beck 1998, Heitz-Mayfield 2005). A special problem will be the fact that SES factors are reflected in smoking habits, diets, diabetes and use of medical services, which are important factors influencing periodontal diseases (Pischon et al. 2007, Gorman et al. 2012). Even though various studies have documented differences in periodontal health by socio-economic indicators or were adjusted on them, these indicators have rarely been investigated as independent variables of main interest.

Socio-economic factors are also associated with systemic markers of inflammation such as C-reactive protein and congeners. These markers increase in response to or concomitant with different subclinical and pathological states such as obesity, diabetes and systemic inflammatory diseases. Although periodontal diseases are local inflammations within the oral cavity, they are associated with systemic reactions, many of them represented by markers of inflammation such as C-reactive protein (CRP), interleukin-6 (IL-6) or fibrinogen (Paraskevas et al. 2008). Accordingly, increased concentrations of these markers are also associated with periodontitis and tooth loss (Noack et al. 2001, Moutsopoulos & Madianos 2006, Paraskevas et al. 2008). Periodontal diseases share these and other factors with numerous systemic diseases with inflammatory background whose causal relationships with each other are widely disputed.

Conventional wisdom postulates a pathogenetic sequence with socio-economic factors as initial step leading to health characteristics following a social gradient. The higher the social position, the better the health (Marmot & Wilkinson 2006). Periodontal diseases are more frequent and severe among individuals of low socio-economic status (Borrell et al. 2006). Greater attachment and bone loss were found in individuals with financial strain and inadequate coping skills. Occupational status was strongly associated with disease progression (Moutsopoulos & Madianos 2006). Also, SES factors such as income or level of education are associated with markers of peripheral inflammation such as CRP, IL-6 or fibrinogen (Friedman & Herd 2010).

By postulating a direction in the pathogenesis of the type SES – risky behaviour – poor health (e.g. periodontitis) – systemic markers of inflammation, two links would be missing, i.e.

(i) It is quite unclear whether in periodontitis the oral inflammation induces systemic effects or whether systemic factors induce increased susceptibility to periodontitis or even whether, in some cases, periodontitis exists merely *with* systemic pathologies.
(ii) No doubt, systemic markers of inflammation are increased by many conditions, which are completely independent from periodontitis. With respect to socio-economic factors, then the question arises if and how such systemic risk factors have an influence on the impact of SES on periodontitis.

In this longitudinal study, we examined social factors, especially education and income to evaluate their effects on progression of mean attachment loss and incidence of tooth loss in relationship to systemic factors during a 5-year period. Database was the cohort Study of Health in Pomerania (SHIP).

## Materials and Methods

### Study design and sample

The Study of Health in Pomerania is a longitudinal population-based medical–dental health survey of a 20- to 79-year-old population in the north-east of Germany. Approved by the local Ethics Committee, SHIP-0 is based on a representative, age-stratified cluster sample that was examined from 1997 to 2001 in West Pomerania. 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 of Caucasian ethnicity and German citizenship were included. A total of 6262 adults aged 20–80 years were invited to participate in SHIP, 4308 of whom were examined, corresponding to a response rate of 68.8% (John et al. 2001). Data collection and instruments comprised four parts: oral health examination, medical examination, health-related interview and a health- and risk factor-related questionnaire. A total of 4290 subjects received an oral examination. Of the subjects, 515 were edentulous, 11 subjects refused periodontal examination. Periodontal measurements were not recordable in 21 subjects due to medical reasons. In 186 subjects, attachment level could not be determined, mainly due to crowns, leaving 3557 subjects for analysis in SHIP-0 (Holtfreter et al. 2009). From October 2002 until 2006, subjects from SHIP-0 were again invited to participate in SHIP 1 as a 5-year follow-up. A total of 3300 subjects were examined with a follow-up response rate of 76.9%. Of these subjects, 312 were edentulous at baseline and 32 did not have an oral examination. Furthermore, in 150 subjects, attachment level was not recordable at baseline, leaving 2806 subjects for analysis, 1346 of them men and 1460 women.

### Dental examination

Dental examinations were conducted in rotation by five trained, calibrated and licensed dentists. Calibration exercises were performed on persons not connected to the study every 6 months, yielding an intra-class correlation of 0.82–0.91 per examiner and an inter-class correlation of 0.84 for attachment loss (Hensel et al. 2003). Assessment of number of teeth included all teeth except the third molars (28 teeth maximum). Measurements of clinical attachment loss (CAL) were taken at distobuccal, midbuccal, mesiobuccal and midlingual sites according to the half-mouth method, alternating on the left or right side (maximum at 14 teeth). At follow-up, for each subject, the same quadrants were re-examined as at baseline using the same recording protocol. If recession was present at the examined site, attachment loss was directly measured as the distance between the cemento–enamel junction (CEJ) and the pocket base rounded to whole millimetres.

#### Independent variables

The SES indicators and socio-demographic variables were taken from the health-related interview (education, occupation) and the personal questionnaire (risk factors and resources for health in living and working conditions like marital status, household income). Level of education was divided according to the final school grade (<10 years, 10 years, >10 years), monthly household income was divided by the number of persons living in the household and categorized into quartiles: low with less than 600 euros per month, 600–960, >960 euros (3rd and 4th quartiles) respectively. Body mass index (BMI) was categorized into quartiles; obesity was defined according to the WHO criteria as BMI $\geq$ 30 kg/m$^2$.

High-sensitive CRP was determined using particle-enhanced immuno-nephelometry (hsCRP kit, Dade Behring Inc.) with a test sensitivity of 0.2 mg/l. CRP concentrations were categorized with cut-off points <1 mg/l, 1–3 mg/l and >3 mg/l (Centers for Disease Control & Prevention & the American Heart Association 2003).

#### Data analysis

The Kruskal–Wallis test was used to compare continuous variables. A $p$-value $< 0.05$ was considered as statistically significant, for interaction it was set at $< 0.10$.

The influence of social determinants and health behaviour was determined by multiple regression analyses for mean attachment loss difference (difference between baseline attachment loss and attachment loss after 5-years in mm). Poisson regression analyses were applied for tooth loss (difference between number of teeth in SHIP 0 and SHIP 1). All subjects edentulous at baseline and those with missing CAL data were excluded. Follow-up time varied between 4.4 and 8.5 years, mean 5.26 (95% confidence interval 5.24–5.28). In all regression analyses, the log-transformed follow-up time was included as an offset variable. Analyses were performed using software STATA 10.0 (Stata Corp LP, College Station, USA).

### Results

In Table 1, the baseline characteristics of the dentate subjects are collected. The variables of interest were most distinctive between male and female participants, both influenced by age. Women showed less attachment loss, but higher CRP levels than men. Significant differences were also noticed in smoking variables, marital status, education level and dental hygiene.

During the follow-up time, 436 men (35.4%) and 408 women (30.5%) lost one or more teeth. A substantial progression of attachment loss mean CAL $\leq -1.0$ mm (baseline minus follow-up figures) was registered in 222 men (18.0%) and 192 women (14.4%). In Table 2, the variables of interest are collected, that is, the crude (unadjusted) figures of baseline CAL and the progression of mean clinical attachment loss, baseline number of teeth and mean changes in number of teeth during the follow-up period and the baseline CRP concentrations. Consistently, the outcome was the better, the less risky the SES factors were or the health-related behaviour was. For most of the factors shown, a dose–response relationship was observed. Both the baseline values and the incidence of tooth loss and attachment loss increased with lower education, lower income or with being unmarried. Similarly, dose-dependent trends were noticed with smoking or being obese. Bearing in mind that periodontitis is an inflammatory disease, systemic concentrations of baseline C-reactive protein (CRP) show analogous gradients along the factors displayed in Table 2.

The summing up of binary defined risk factors irrespective of the particular risk resulted in the relationships

*Table 1.* Demographic and diagnostic characteristics of the 3300 participants of the Ship-1 study (follow-up) at baseline

| Age group | 20–40 | | >40–60 | | >60 | |
|---|---|---|---|---|---|---|
| Sex (male/female) | m | f | m | f | m | f |
| No. of subjects in follow-up $n$ = 3300 | 457 | 561 | 629 | 700 | 503 | 450 |
| No of edentulous subjects | 1 | 0 | 15 | 27 | 137 | 132 |
| No of subjects in study analysis‡ $n$ = 2806 | 453 | 546 | 579 | 645 | 314 | 269 |
| Age, median | 32.2 | 31.4 | 50.0 | 50.1 | 66.0 | 65.9 |
| Number of teeth, mean¶ | 25.2 ± 3.1 | 25.1 ± 3.5 | 21.0 ± 5.9 | 20.6 ± 5.6 | 15.2 ± 7.6 | 14.6 ± 7.2 |
| % CAL $\geq$ 4 mm | 6.3 ± 12.3 | 5.4 ± 12.1 | 36.0 ± 30.1 | 26.7 ± 27.8* | 59.0 ± 32.0 | 48.3 ± 31.3* |
| Mean CAL, mm | 1.3 ± 1.0 | 1.2 ± 0.9 | 3.2 ± 1.7 | 2.6 ± 1.5* | 4.5 ± 2.0 | 3.7 ± 1.5* |
| C-reactive protein, CRP mg/l | 1.6 ± 2.8 | 2.6 ± 3.9* | 2.3 ± 4.9 | 2.8 ± 4.3 | 2.9 ± 4.8 | 3.3 ± 4.7 |
| CRP > 3 mg/l, % | 20.5 | 27.7† | 22.3 | 32.1† | 28.0 | 32.3† |
| Body mass index | 26.1 ± 3.7 | 24.2 ± 4.5* | 28.3 ± 3.9 | 27.4 ± 5.2* | 28.5 ± 3.8 | 28.5 ± 4.7 |
| Obesity (BMI $\geq$ 30), % | 14.6 | 11.9 | 29.7 | 27.8 | 29.3 | 32.0 |
| Smokers at present, % | 39.7 | 32.4 | 31.1 | 19.5† | 10.8 | 5.2 |
| Pack-years, mean | 6.4 ± 9.0 | 3.5 ± 5.9* | 14.9 ± 18.0 | 4.4 ± 9.0* | 15.0 ± 20.8 | 2.2 ± 6.7* |
| Married, % | 41.5 | 54.6† | 82.6 | 79.2 | 90.4 | 56.9† |
| Widowed or divorced, % | 4.4 | 6.0 | 10.2 | 16.0† | 8.3 | 35.7† |
| Income < 960 4.6 €/month, % | 60.7 | 64.8 | 49.4 | 50.7 | 42.4 | 60.6† |
| Education less than 10th grade, % | 10.8 | 4.4† | 32.5 | 29.5 | 65.3 | 72.9 |
| Tooth brushing < twice/day, % | 23.0 | 6.4† | 24.0 | 5.1† | 30.9 | 12.3† |
| Last dental visit > 12 months back, % | 14.8 | 6.4† | 11.6 | 7.8 | 8.9 | 5.6 |

Differences between sexes in each age stratum: †$p < 0.001$ (chi-square), *$p < 0.001$ (Mann–Whitney).
‡Remaining subjects after exclusion of the edentulous and those with indefinable cemento–enamel junction.
¶Excluding the third molars.

*Table 2.* Characteristics of the study population according to socio-economic status. Baseline mean CAL figures and CAL progression, baseline number of teeth and tooth loss during the 5-year follow-up, C-reactive protein (CRP) at baseline, mean ± SD

| | | Baseline CAL mm | Δ CAL, mm*(− loss, + gain) | Baseline No. of teeth | No. of teeth lost* | Baseline CRP, mg/l |
|---|---|---|---|---|---|---|
| Education | <10th grade | 3.7 ± 1.9‡ | −0.10 ± 1.15‡ | 16.9 ± 7.3‡ | 1.5 ± 2.5‡ | 2.9 ± 4.8‡ |
| | 10th grade | 2.1 ± 1.6 | 0.04 ± 1.01 | 22.8 ± 5.39 | 0.7 ± 1.7 | 2.5 ± 4.4 |
| | >10th grade | 2.0 ± 1.5 | 0.18 ± 0.92 | 23.5 ± 5.2 | 0.6 ± 1.5 | 1.9 ± 2.9 |
| Marital status | Divorced/widowed | 3.2 ± 1.8‡ | −0.08 ± 1.05‡ | 18.3 ± 7.2‡ | 1.4 ± 2.5‡ | 2.7 ± 3.5‡ |
| | Married | 2.8 ± 1.8 | 0.08 ± 1.03 | 20.6 ± 6.5 | 0.9 ± 1.9 | 2.6 ± 4.5 |
| | Single | 1.4 ± 1.3 | −0.10 ± 1.03 | 24.6 ± 5.0 | 0.6 ± 1.7 | 2.2 ± 4.0 |
| Monthly income | < 600 | 2.4 ± 1.8 † | −0.09 ± 1.02‡ | 20.8 ± 7.0 | 1.0 ± 2.0† | 2.5 ± 3.9 |
| | 600–959 | 2.6 ± 1.9 | −0.02 ± 1.12 | 20.8 ± 6.8 | 1.1 ± 2.2 | 2.8 ± 5.3 |
| | ≥960 EURO | 2.6 ± 1.7 | 0.12 ± 0.98 | 21.4 ± 6.3 | 0.8 ± 1.8 | 2.3 ± 3.7 |
| Smoking | Current | 2.7 ± 1.9‡ | −0.10 ± 1.05‡ | 21.3 ± 6.5‡ | 1.2 ± 2.4‡ | 2.4 ± 3.1 |
| | Quit | 3.0 ± 1.9 | 0.15 ± 1.09 | 20.0 ± 6.9 | 1.0 ± 2.0 | 2.6 ± 5.2 |
| | Never | 2.4 ± 1.7 | 0.04 ± 1.00 | 21.4 ± 6.5 | 0.8 ± 1.7 | 2.6 ± 4.4 |
| Body mass index | Obese | 3.1 ± 1.9‡ | −0.04 ± 1.11 | 19.5 ± 7.0‡ | 1.2 ± 2.2‡ | 3.8 ± 5.7‡ |
| | Overweight | 2.8 ± 1.8 | 0.08 ± 1.04 | 20.5 ± 6.8 | 1.0 ± 2.1 | 2.4 ± 3.5 |
| | Normal weight | 2.0 ± 1.6 | 0.02 ± 1.00 | 22.8 ± 5.7 | 0.6 ± 1.5 | 1.8 ± 3.9 |
| Age groups at baseline | 20.22–35.00 years | 1.1 ± 0.8‡ | −0.03 ± 1.00† | 25.9 ± 2.5‡ | 0.3 ± 1.0‡ | 2.0 ± 3.3‡ |
| | 35.01–50.00 years | 2.3 ± 1.4 | 0.04 ± 1.00 | 22.3 ± 5.1 | 0.8 ± 2.0 | 2.4 ± 3.7 |
| | 50.01–65.00 years | 3.4 ± 1.7 | 0.10 ± 1.10 | 19.0 ± 6.5 | 1.3 ± 2.3 | 2.7 ± 5.0 |
| | > 65 years | 4.3 ± 1.9 | −0.09 ± 1.16 | 13.5 ± 7.3 | 1.5 ± 2.2 | 3.4 ± 5.3 |

Kruskal–Wallis test for all dose-related differences within the categorical scales of risk factors †$p < 0.050$, ‡$p < 0.001$.
*Figures represent differences (baseline minus follow-up values).

illustrated in Fig. 1. The higher the baseline CRP levels, the more risk factors were combined in an individual. Likewise, even after the short follow-up time of 5 years, tooth loss and change in attachment level show an outcome pattern comparable to that of CRP when related to the accumulated number of risk factors. This illustrates the multifactorial character of the disease with multiple combinations of modifying factors.

In multivariate analyses, we assessed the impact of the education, income and marital status variables on incident tooth loss (Table 3) and CAL progression (Table 4) during the follow-up. Education and income were both significant predictors of tooth loss or CAL progression. This influence was maintained after adjustment for marital status with significant sex interaction (model 1). Neither health behaviour-related variables such as smoking (model 2) nor even the inclusion of CRP and obesity as independent variables (model 3) attenuated the coefficients of education or income variables. Nevertheless, all included variables contributed significantly to the progress of the disease. For tooth loss, (Table 3) the incidence risk ratio IRR associated with smoking was 1.18 (95% CI 1.07–1.32, $p = 0.002$) and 1.79 (1.62–1.97, $p < 0.001$) for former smokers or current smokers respectively (model 2). BMI threshold at BMI = 30 (obesity, approx. the 4th BMI quartile) resulted in an IRR of 1.32 (1.16–1.50, $p = 0.001$). CRP cut-offs 1–3 mg/l and > 3 mg/l contributed with IRR 1.07 (0.97–1.14, $p = 0.088$) and 1.25 (1.13–1.34, $p < 0.001$) respectively (model 3). Even though the coefficients were less significant, similar results were observed with the attachment level progression, that is, loss or gain of mean CAL change in mm (Table 4). Combined SES factors education and income factors were attenuated by each other, but remained unchanged by marital status. Interaction between gender and marital status revealed that being married is protective in men more than in women (Tables 3 and 4). Despite this effect modification, women still lost fewer teeth than men, irrespective of being married or not. Men suffered preferably from severe attachment loss progression if unmarried.

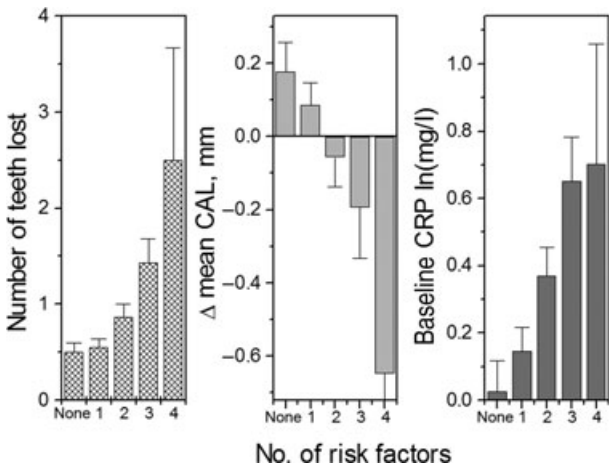


*Fig. 1.* Incidence of tooth loss (left panel) and CAL progression (middle) and CRP concentration (right) by number of risk factors present: categories used: school education < 10th grade, income < 960 €, current smoking, BMI ≥30, in any combination (error bars 95% confidence intervals).

Effect modifications were observed between income and education, also

*Table 3*. Incidence of tooth loss (IRR and 95% CI) regressed on SES factors and adjusted for age and sex

| | | Education | Income | Marital | Model 1 | Model 2 | Model 3 |
|---|---|---|---|---|---|---|---|
| Age (ref. 20–35 years) | Age 35.01–50.00 years | 2.90‡ | 3.18‡ | 3.58‡ | 3.46‡ | 3.35 (2.85–3.94)‡ | 3.17 (2.69–3.73)‡ |
| | Age 50.01–65.00 years | 3.94‡ | 5.14‡ | 5.83‡ | 4.91‡ | 5.28 (4.47–6.24)‡ | 4.90 (4.14–5.80)‡ |
| | Age > 65 years | 4.19‡ | 5.94‡ | 6.58‡ | 5.27‡ | 6.24 (5.18–7.52)‡ | 5.64 (4.67–6.82)‡ |
| Education (ref. >10th grade) | 10th grade | 1.35‡ | | | 1.28‡ | 1.22 (1.07–1.40)† | 1.21 (1.06–1.38)† |
| | Less than 10th grade | 2.00‡ | | | 1.79‡ | 1.69 (1.48–1.93)‡ | 1.63 (1.43–1.86)‡ |
| Income/person (ref > 960€) | 600 –959/person | | 1.44‡ | | 1.27‡ | 1.24 (1.13–1.36)‡ | 1.25 (1.14–1.38)‡ |
| | < 600/person | | 1.54‡ | | 1.27‡ | 1.22 (1.09–1.36)‡ | 1.21 (1.09–1.35)‡ |
| Marital status | Unmarried male (ref.) | | | 1.00 | 1.00 | 1.00 | 1.00 |
| | Unmarried female | | | 0.63‡ | 0.64‡ | 0.69 (0.60–0.79)‡ | 0.70 (0.61–0.81)‡ |
| | Married male | | | 0.62‡ | 0.68‡ | 0.69 (0.61–0.78)‡ | 0.69 (0.61–0.79)‡ |
| | Married female | | | 0.80‡ | 0.85† | 0.90 (0.80–1.02) | 0.90 (0.79–1.02) |
| Smoking (ref. never smoked) | Smoking quitted | | | | | 1.18 (1.07–1.32)† | 1.18 (1.06–1.31)† |
| | Smoking current | | | | | 1.79 (1.62–1.97)‡ | 1.82 (1.65–2.00)‡ |
| BMI (ref. 1st quartile) | BMI 2nd quartile | | | | | | 1.11 (0.98–1.27) |
| | BMI 3rd quartile | | | | | | 1.24 (1.10–1.41)† |
| | BMI 4th quartile | | | | | | 1.32 (1.16–1.50)‡ |
| CRP (ref. < 1 mg/L) | CRP 1 to < 3 mg/l | | | | | | 1.07 (0.97–1.18) |
| | CRP ≥ 3 mg/l | | | | | | 1.25 (1.13–1.38)‡ |

†$p < 0.050$, ‡$p < 0.001$.
The first three columns show the SES factors separated. Model 1 combines the particular SES factors. Model 2 is the same as model 1 additionally adjusted for smoking (never, quit, current), model 3 as model 2 additionally adjusted for obesity (BMI quartiles, 16.79–23.79, 23.80–26.82, 26.83–30.02, 30.03–58.43) and for CRP categories (< 1, 1 to < 3, ≥3 mg/l).

*Table 4*. Progression of mean attachment loss in mm as difference baseline minus follow-up CAL (ß-coefficients and 95% CI) regressed on SES factors

| | | Education | Income | Marital | Model 1 | Model 2 | Model 3 |
|---|---|---|---|---|---|---|---|
| Age (ref. 20–35 years) | Age 35.01–50.00 years | 0.10 | 0.05 | −0.00 | 0.00 | −0.00 (−0.12; 0.11) | −0.01 (−0.12; 0.11) |
| | Age 50.01–65.00 years | 0.27† | 0.14† | 0.08 | 0.16 | 0.13 (0.00; 0.26)† | 0.12 (−0.01; 0.25) |
| | Age > 65 years | 0.13 | −0.065 | −0.11 | 0.00 | −0.05 (−0.23; 0.12) | −0.05 (−0.23; 0.12) |
| Education (ref. > 10th grade) | 10th grade | −0.14† | | | −0.12† | −0.11 (−0.21; 0.00) | −0.11 (−0.21; 0.00) |
| | Less than 10th grade | −0.35‡ | | | −0.30‡ | −0.28 (−0.41; −0.15)‡ | −0.27 (−0.40; −0.14)‡ |
| Income/person (ref > 960 €) | 600 –959/person | | −0.15† | | −0.09 | −0.08 (−0.18; 0.02) | −0.08 (−0.18; 0.01) |
| | < 600/person | | −0.23‡ | | −0.13† | −0.12 (−0.23; −0.01)† | −0.11 (−0.22; −0.00)† |
| Marital status | Unmarried male (ref.) | | | 0.00 | 0.00 | 0.00 | 0.00 |
| | Unmarried female | | | 0.20† | 0.19† | 0.19 (0.05; 0.35)† | 0.23 (0.08; 0.37)† |
| | Married male | | | 0.30‡ | 0.25‡ | 0.23 (0.09; 0.36)† | 0.22 (0.08; 0.36)† |
| | Married female | | | 0.09 | 0.06 | 0.05 (−0.07; 0.18) | 0.04 (−0.06; 0.17) |
| Smoking (ref. never smoked) | Smoking quitted | | | | | 0.11 (−0.00; 0.21) | 0.11 (−0.00; 0.21) |
| | Smoking current | | | | | −0.13 (−0.23; −0.03)† | −0.12 (−0.22; −0.02)† |
| BMI (ref. 1st quartile) | BMI 2nd quartile | | | | | | 0.14 (0.03; 0.26)† |
| | BMI 3rd quartile | | | | | | 0.09 (−0.04; 0.21) |
| | BMI 4th quartile | | | | | | 0.03 (−0.10; 0.16) |
| CRP (ref. < 1 mg/l) | CRP 1 to <3 mg/l | | | | | | −0.02 (−0.11; 0.08) |
| | CRP ≥ 3 mg/l | | | | | | −0.20 (−0.30; −0.10)‡ |

†$p < 0.050$, ‡$p < 0.001$.
The first three columns show the SES factors separated. Model 1 combines the particular SES factors. Model 2 is the same as model 1 additionally adjusted for smoking (never, quit, current), model 3 as model 2 additionally adjusted for obesity (BMI quartiles, 16.79–23.79, 23.80–26.82, 26.83–30.02, 30.03–58.43) and for CRP categories (< 1, 1 to < 3, ≥3 mg/l).

between sex and obesity or income and CRP thresholds. Significant interactions were also revealed between the systemic marker CRP with educational status less than 10th grade ($p = 0.075$) or with an income below the official poverty threshold ($p = 0.023$ and $p < 0.001$, for CRP 1–3 mg/l and CRP >3 mg/l respectively). In contrast, interaction terms with respect to the CAL progression were without significance ($p > 0.100$), except for marital status. As indicated for all subjects in Table 5 (left column), incidence of tooth loss was associated with all the SES factors included and also with those factors related to inflammation as obesity. Effect modifications by the sex of the participants are shown with respect to marital status and obesity. IRR of tooth loss is reduced in married men, but increased in obese men as compared with their female counterparts. These interactions were demonstrated for tooth loss and CAL progression as well. Stratifying the regression

analysis by CRP level threshold (Table 5) gives an impression of the various interactions or effect modifications. Within the stratum with high CRP, the impact of lower level of education is stronger than in the low-CRP group. Likewise, pronounced differences were observed with respect to income and smoking being aggravated at high CRP levels.

Neglected preventive manoeuvres such as sloppy tooth care or delayed dental appointments were associated with low income. The impact of smoking or marital status seemed to be independent of income status (data not shown). Again, with respect to the CAL progression, these interaction terms were without significance ($p > 0.100$).

Clinical attachment loss and tooth loss are strongly related to age. Interaction with age was significant with the educational levels ($p < 0.001$). Because tooth loss in younger age groups may be due to causes other than periodontal disease, we stratified the analyses by age. Low level of education was associated with the highest incident risk ratio for tooth loss in the youngest (20–35-years old), still significant in the middle age groups (between 35 and 65 years) and negligible in the oldest subjects >65 years (data not shown).

## Discussion

In this follow-up study, we have shown that level of education, income and marital status are associated with the progression of periodontitis and tooth loss. Accumulation of SES factors and/or behaviour-related factors such as smoking and obesity augmented the disease progression and were associated with increased baseline levels of CRP. Level of education and income remained important determinants of periodontal destruction even after adjustment to other risk factors or to CRP. Nevertheless, neither smoking nor obesity nor CRP attenuated the relation between the SES factors and disease progression.

The association between SES factors, especially education and income, and markers of inflammation such as CRP, IL-6 or fibrinogen was considered as one of the mechanisms by which SES "gets under the skin", that is, may have an influence on health (Friedman & Herd 2010). Periodontal studies have consistently proved that extent and severity of periodontal diseases is associated with increased concentrations of these markers within the circulation (Moutsopoulos & Madianos 2006, Paraskevas et al. 2008). Smoking is one of the most important risk factors of periodontitis. Also, smoking was suggested to be a "link" between SES and the disease. People with lower education are more often smokers compared with those of higher education and they are often burdened by more pack-years.

Thus, if CRP or smoking would mediate the associations between education or income and periodontal progression or tooth loss, the following relations must be fulfilled:

(a) The SES factors must be associated with both the outcome (CAL or tooth loss) and the intervening variable (CRP or smoking). This was shown in our data (Table 2, Fig. 1).
(b) CRP or smoking must also be associated with the outcome – true as well.
(c) Inclusion of CRP or smoking in the outcome models should attenuate the association between

*Table 5.* Incidence of tooth loss regressed on age, gender and the SES factors education, income, marital status including smoking and obesity stratified by CRP level categories. Incident risk ratios (95% CI) are given

| Independents* | All subjects | *p* | CRP ≤ 3 mg/l | *p* | CRP > 3 mg/l | *p* |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| Age 35.01–50.00 years | 3.271 (2.78–3.85) | <0.001 | 3.41 (2.77–4.22) | <0.001 | 2.98 (2.30–3.86) | <0.001 |
| Age 50.01–65.00 years | 5.17 (4.37–6.10) | <0.001 | 6.12 (4.96–7.55) | <0.001 | 3.75 (2.85–4.95) | <0.001 |
| Age > 65 years | 6.09 (5.06–7.34) | <0.001 | 7.59 (6.99–9.59) | <0.001 | 4.05 (2.96-5.54) | <0.001 |
| Education | | | | | | |
| Education 10th grade | 1.22 (1.07–1.39) | 0.003 | 1.13 (0.97–1.32) | 0.109 | 1.47 (1.12–1.92) | 0.006 |
| Less than 10th grade | 1.65 (1.45–1.89) | <0.001 | 1.46 (1.25–1.71) | <0.001 | 2.13 (1.62–2.79) | <0.001 |
| Income per month | | | | | | |
| > 960 (reference) | 1 | – | 1 | – | 1 | – |
| ≤960 € | 1.23 (1.13–1.34 | <0.001 | 1.13 (1.02–1.25) | 0.017 | 1.50 (1.29–1.76) | <0.001 |
| Marital status | | | | | | |
| Unmarried male (reference) | 1.00 | – | 1.00 | – | 1.00 | – |
| Unmarried female | 0.78 (0.67–0.92) | 0.002 | 0.84 (0.69–1.02) | 0.074 | 0.61 (0.47–0.80) | <0.001 |
| Married male | 0.71 (0.63–0.81) | <0.001 | 0.76 (0.65–0.84) | 0.001 | 0.61 (0.49–0.75) | <0.001 |
| Married female | 0.91 (0.80–1.03) | 0.119 | 0.93 (0.80–1.09) | 0.387 | 0.90 (0.73–1.11) | 0.333 |
| Smoking | | | | | | |
| Former smoking | 1.16 (1.04–1.29) | 0.007 | 1.06 (0.94–1.21) | 0.343 | 1.42 (1.17–1.73) | <0.001 |
| Current smoking | 1.78 (1.62–1.97) | <0.001 | 1.53 (1.35–1.72) | <0.001 | 2.47 (2.07−2.94) | <0.001 |
| Health consciousness | | | | | | |
| Tooth brushing less than twice/day | 1.07 (0.97–1.18) | 0.191 | 1.07 (0.95–1.21) | 0.285 | 1.04 (0.86-1.24) | 0.704 |
| Last dental visit > 12 months before | 1.17 (1.04–1.32) | 0.011 | 1.42 (1.23–1.64) | <0.001 | 0.75 (0.60-0.95) | 0.015 |
| Obesity | | | | | | |
| BMI ≥ 30 male (reference) | 1.00 | – | 1.00 | – | 1.00 | – |
| BMI ≥ 30 female | 0.78 (0.67–0.92) | 0.002 | 0.84 (0.69–1.02) | 0.074 | 0.61 (0.47–0.80) | <0.001 |
| Obesity, BMI ≥ 30 male | 1.35 (1.21–1.51) | <0.001 | 1.41 (1.23-1.62) | <0.001 | 1.07 (0.88–1.30) | 0.514 |
| Obesity, BMI ≥ 30 female | 0.99 (0.87–1.13) | 0.901 | 0.81 (0.67–0.99) | 0.035 | 1.15 (0.94–1.40) | 0.168 |

*Reference categories unless otherwise stated: age 20–35 years, education better than 10th grade, smoking never.

the SES factors and the outcome. This condition was not observed in our data (Tables 3 and 4).

Thus, presumably neither CRP nor smoking can be considered as mediators between SES factors and CAL progression or tooth loss. On the other hand, unfavourable social conditions were associated with an increased impact of high CRP or high BMI on tooth loss. Smoking, however, exhibits an increased incident risk ratio under conditions of high CRP levels (Table 5). Others suggested that smoking may lie on the pathway between SES and periodontal outcome (Zini et al. 2011) despite the fact that SES factors were found to be significant also in never smokers (Paulander et al. 2004), an observation also made in other studies and obvious in our data. Also with regard to CRP, it was suggested and seems to be reasonable that it could lie on the path between SES and inflammatory diseases in general (Friedman & Herd 2010). The results suggest that low SES status may be associated with increased susceptibility to inflammation or, conversely, inflammatory challenges (noticeably obesity and CRP) are most adverse in subjects of low SES characteristics.

Socio-economic status gradients are also important in the development of inflammatory processes in various diseases such as cardiovascular diseases or diabetes (Gruenewald et al. 2009), diseases which are themselves associated with periodontitis. In cardiovascular diseases, inflammation was considered as the link between SES factors and the disease (Gemes et al. 2008, Sethi et al. 2008). From diabetes studies, however, it was concluded that inflammation cannot be the "missing link" between SES and the disease (Rathmann et al. 2006). With respect to the role of CRP and other inflammatory markers in such diseases, there is an ongoing discussion about whether an individual susceptibility to inflammatory diseases is a lifelong burden associated with genetic imprinting, intra-uterine conditions and social inequalities of parents and in childhood. Also, these associations are still partially explained by the influence on well-known disease risk factors such as smoking or adipositas among others (Pollitt et al. 2007, Loucks et al. 2010). Changes in socio-economic disparities determine differing levels of inflammation and cardiovascular events (Packard et al. 2011) and of oral health in adulthood as well (Thomson et al. 2004). Frequently, obesity is associated with a mild or low-grade inflammatory state (Bastard et al. 2006).

Of the SES factors, education usually peaks early in life and precedes the onset of periodontitis, thus allowing to separate cause from effect (Boillot et al. 2011). Many studies revealed the strong influence of educational level on periodontal health (Klinge & Norlund 2005, Sabbah et al. 2009). Low level of education increases the risk of tooth loss, but is especially disadvantageous when combined with low income. Although inter-related, in our study, education does not outsource income and *vice versa*, there was only a slight attenuation of the corresponding risk ratios. Similarly, this was shown in other studies too (Benabé & Marcenes 2011). Education and income are of great importance for tooth maintenance when reflecting on socio-economic factors and dental health.

A number of epidemiological studies have shown that there are gender differences in appearance as well as social and psychological impact on oral health (McGrath & Bedi 2003). Gender differences are determinants in the variations of the social impact on oral health. To that extent, results of a tooth loss evaluation were confirmed (Mundt et al. 2011). In this SHIP study, more women than men were found in higher educational grades, but still earn lesser than their male counterparts. Women take more care of their health (Åstrøm & Rise 2001). They smoke less, pay more attention to their diet and perform preventive oral health measures more actively than men. In our results, being married was protective in men with respect to the outcome as compared to women. Nevertheless, women lost fewer teeth and showed better periodontal outcome after 5 years, irrespective of their marital status. SES interactions with peripheral CRP may contribute to gender differences, as it was consistently shown in several studies that women exhibit higher CRP levels than men (Khera et al. 2005, Zhang et al. 2008). This corresponds to reports showing that staying married affords men robust protection against elevated levels of CRP, a relationship remaining significant after accounting for different variables of health behaviour and putative psychological stress (Sbarra 2009).

Even though the relationships between SES parameters and dental health are comprehensible, the causal pathways are still speculative. The analytical methods are insufficient to elucidate sequentially caused associations (Newton & Bower 2005). One may speculate that various effects act in concert, especially that SES influences periodontal health and that early-life health has a certain impact on later SES. The sequence of the development of periodontal disease goes back to life histories in each individual long before this follow-up study. Tooth loss and periodontal health might also be influenced by individual prosthodontic decisions regarding questionable or strategic teeth before baseline examinations in SHIP; replacement of missing teeth is related to SES too (Mundt et al. 2009). Small intervention studies have shown that an effective treatment of periodontal inflammation may contribute to a profound decrease in markers of inflammation (Shimada et al. 2010, El Fadl et al. 2011). It could be an interesting task to elucidate if the outcome of such interventions varies according to the SES background. Similarly, as it was suggested for coronary heart disease risk estimation (Franks et al. 2010), incorporating SES factors in treatment decision making may improve the outcome in periodontal settings.

A 5-year follow-up period may be too short to reveal the combined influence of social, psychosocial and behaviour determinants on periodontal disease progression. This time was too short to reach decisive results in CAL measures similar to those regarding the incidence of tooth loss. Long-time follow-up studies support the results shown here (Gorman et al. 2012). Another problem was the tendency that mainly socially disadvantaged and the very old avoided re-participation in the follow-up examination possibly underestimating the social gradient.

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Address:
*Peter Meisel*
*Unit of Periodontology, Centre of Oral Health*
*University of Greifswald*
*Rotgerber Str. 8, D-17475, Greifswald*
*Germany*
E-mail: meiselp@uni-greifswald.de

**Clinical Relevance**

*Scientific rationale for the study*: Socio-economic factors have been associated with periodontal diseases. There are no population-based longitudinal studies evaluating the interaction of these factors with markers of inflammation or the established risk factors of periodontitis.

*Principal findings*: Attachment loss progression and tooth loss over 5 years were associated with level of education, income, marital status as well as with smoking, obesity and gender. Disadvantageous socio-economic status aggravated the impact of systemic inflammation and obesity on incident tooth loss.

*Practical implications*: These findings underscore the need to include the socio-economic situation of patients into prognostic scores of tooth loss. Interaction of social or economic positions with systemic inflammation supports the view of periodontitis as a problem of the organism as a whole.