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Inverse association between periodontitis and respiratory allergies

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Clinical and
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Summary

Background Periodontitis is an infection with systemic effects and a high prevalence among adults. In the aetiology of allergic diseases the hygiene hypothesis claims that infections in early infancy may protect against allergic diseases.

Objective The aim of the present analyses was to investigate the independent relation between periodontitis and respiratory allergies such as hayfever, house dust mite (HDM) allergy and asthma.

Methods From the Study of Health in Pomerania (SHIP) a total number of 2837 subjects aged 20 to 59 years were included in the analysis. In our study population 326, 111 and 114 subjects were classified as suffering from hayfever, HDM allergy or asthma, respectively. The attachment loss (AL) were measured. Periodontitis was defined according to the percentage of surfaces which exceeded 3 mm AL [healthy: 0–7.7%, mild: 7.8–28.6%, moderate: 28.7–63.9%, severe: > 63.9%]. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated using logistic regression.

Results After adjustment for confounding factors these analyses revealed inverse associations between periodontitis and hayfever as well as periodontitis and HDM allergy. For increasing AL, a trend of decreasing risk could be observed for hayfever (healthy: reference; mild AL: OR 0.87 [95% CI 0.6–1.2]; moderate AL: OR 0.80 [95% CI 0.6–1.2]; severe AL: OR 0.53 [95% CI 0.3–0.9]; $P_{\text{trend}} = 0.01$) and for HDM allergy (healthy: reference; mild AL: OR 0.80 [95% CI 0.5–1.3]; moderate AL: OR 0.64 [95% CI 0.3–1.2]; severe AL: OR 0.39 [95% CI 0.2–0.9]; $P_{\text{trend}} = 0.02$). Furthermore, for asthma were observed a slightly inverse association in the full-adjusted model (healthy: reference; mild AL: OR 1.10 [95% CI 0.6–2.0]; moderate AL: OR 0.96 [95% CI 0.5–1.8]; severe AL: OR 0.48 [95% CI 0.2–1.0]; $P_{\text{trend}} = 0.11$).

Conclusion There is an inverse association between periodontitis and respiratory allergies. Our results might support the hygiene hypothesis.

Keywords asthma, atopy, hayfever, house dust mite allergy, hygiene hypothesis, periodontitis, Study of Health in Pomerania

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Introduction

In recent decades an increase in the prevalence of allergic diseases has been observed in developed countries [1]. In 1989 Strachan [2] found a relation between hayfever and family size and postulated the 'hygiene hypothesis', which suggests that infections in early infancy may have protective effects against the development of allergies. In the 1990s it was expressed in immunological terms [3, 4]. A potential explanation provides the T-helper type 1(Th1)/Th2 paradigm [5, 6]. The two helper subsets Th1 and Th2

exhibit different patterns of cytokine production and different functions. Th2-derived cytokines inhibit the development of Th1 cells and vice versa. Allergic diseases are Th2-mediated and characterized by the release of IgE, whereas bacterial and viral infections are more likely to be Th1 mediated. Thus, the infection-induced Th1 cell-specific cytokine inhibits the development of allergen-specific Th2 cells. Although this biological background explains the association between infections and allergies to some extent, this topic is still controversially discussed. Some studies supported the hygiene hypothesis [7, 8] and

inverse associations between Th1-mediated infections such as tuberculosis, measles and hepatitis A and allergic diseases were demonstrated [9–11]. Likewise, a possible protective effect of infections by helminths against allergies was found [12, 13]. Other studies found no associations between common childhood infections such as mumps, varicella or rubella and allergies [14–16].

Periodontitis is a local oral infection with systemic effects [17, 18] and a high prevalence among adults. The pathway for adult periodontitis is assumed to start with the colonization of the oral cavity with periodontitis inducing Gram-negative, anaerobic microorganisms in childhood [19]. Studies performed in children analysed plaque from the gingival crevice, tooth surface and the dorsum of the tongue and revealed that a sizeable proportion of subjects harboured periopathogenic anaerobes despite the absence of overt gingival inflammation [20, 21]. Thus it seems reasonable to assume that within the framework of the hygiene hypothesis the colonization of these microorganisms elicits a systemic reaction that protects against the development of allergic diseases. We hypothesize that inverse associations of periodontitis with hayfever, house dust mite (HDM) allergy and asthma in adulthood reflect this effect. Therefore, we analysed the associations between periodontitis and these three respiratory allergies.

Material and methods

Study population

The Study of Health in Pomerania (SHIP) is a cross-sectional epidemiological survey of the adult population that was conducted in the northeast of Germany [22]. From the entire population of 212 157 inhabitants living in the area a sample was selected from the population registration offices, where all German inhabitants are registered. A two-stage cluster sampling method was adopted from the WHO MONICA Project Augsburg, Germany [23]. A representative sample of 7008 men and women, aged 20–79 years and 292 individuals of each gender in each of the twelve 5-year age strata, was drawn. Data collection started in October 1997 and was finished in May 2001. A total of 4310 individuals (68.8% of eligible subjects) participated in this study. All participants were investigated in one of two health examination centres established for the purpose of the study. Furthermore, all participants gave written informed consent. The study conformed to the principles of the Declaration of Helsinki as reflected by an *a priori* approval of the Ethics Committee of the University of Greifswald.

All data were weighted to adjust for non-response and to the age-gender distribution of the regional adult population. The regional comparative data were collected from the statistical state office of West Pomerania.

Of the 4310 participants, 1406 individuals who were 60 years and older were excluded from the analyses because of the low number of teeth. Furthermore, 52 edentulous persons and 15 persons who had no oral examination were also excluded. Altogether the final study population comprised 2837 subjects who were available for the present analyses.

Measurements

Trained dentists assessed the dental status. The number of teeth was categorized into three levels (<10 teeth, 10–20 teeth, >20 teeth). Periodontitis was measured by cumulative attachment loss, which represents the distance from the cemento enamel junction to the bottom of the periodontal pocket. The measurements were performed on a maximum of 14 teeth at four surfaces: mesiobuccal, distobuccal, midbuccal and midlingual and made randomly in whole millimetres, either on the left or right quadrant in subjects. For each subject, the percentage of surfaces which exceeded 3 mm attachment loss was determined. Subjects were categorized into four groups according to the quartiles of distribution (healthy periodontal conditions: 0–7.7%, low: 7.8–28.6%, moderate: 28.7–63.9%, severe: >63.9% attachment loss >3 mm).

A computer-aided personal interview was used to gain information on medical history, behavioural and socio-demographic characteristics. Hayfever and HDM allergy were defined according to self-reported answers to the following two questions: 'Do you have any allergy?' and 'Which allergies of the following list, do you have?' Asthma was assessed by the following three key questions: (1) 'Have you had wheezing or whistling in the chest during the past 12 months?', (2) 'Have you had this wheezing or whistling when you didn't have a cold?' and (3) 'Do you take any anti-asthmatic medication at present, e.g. tablets, spray or inhalations?' Asthma was defined as a positive response to either questions (1) and (2) or to question (3) alone. Chronic bronchitis was defined as productive cough at least consecutive 3 months during the past 12 months. Subjects with chronic bronchitis were classified as non-asthmatics. The school education level was categorized into three levels (low (<10 years), medium (10 years), high (>10 years), categories based on the Eastern German three level school system). The smoking status was assessed by information about quality and quantity of smoking in the present and the past, and was categorized into current, former and never-smokers. Two categories for alcohol consumption were defined by using the consumed amount alcohol in gram per day [low (women: <20 g/day, men: <30 g/day), high (women ≥20 g/day, men ≥30 g/day)]. Diabetes mellitus was defined as self-reported physician diagnosis of diabetes or serum haemoglobin A1c (HbA1c) >7%.

Statistical analysis

The study population was divided into four groups with respect to the presence or absence of hayfever, HDM allergy or asthma. Continuous data are expressed as mean and standard error. Nominal data are expressed as percentage. For bivariate analyses, the χ^2 -test (nominal data) or Mann-Whitney-*U*-test (continuous data) was used to compare subjects with respiratory allergies and subjects without allergic diseases. Multivariable logistic regression models were performed to assess the independent associations between periodontitis and respiratory allergies. The models were adjusted for age, gender, school education, smoking, alcohol consumption, family history for allergies or asthma and number of teeth. Odds ratios (OR) with 95% confidence intervals (CIs) were calculated. Further tests for trend in risk with attachment loss were calculated by fitting this variable as continuous covariate in logistic regression. A value of $P \leq 0.05$ was considered statistically significant. Statistical analyses were performed with SAS 9.1 (SAS Institute Inc., Cary, NC, USA).

Results

Overall, respiratory allergies were present in 15.4% ($n = 438$) of the participants. The prevalence proportions for HDM allergy, hayfever and asthma were 3.9%, 11.5%, and 4.0%, respectively. Among the subjects with HDM allergy, 72.0% ($n = 81$) had also hayfever or asthma. Furthermore, 34.3% ($n = 39$) of the asthmatic subjects and 29.5% ($n = 98$) of the subjects with hayfever had at least one of the other diseases. Twelve individuals stated that they had all of the three diseases.

Subjects with hayfever, HDM allergy or asthma were compared with subjects without any of these allergies with respect to general characteristics (Table 1). Individuals with hayfever or HDM allergy were more likely to be younger, higher educated, had fewer siblings and reported to consume fewer cigarettes than individuals without these allergies. Asthmatic persons consumed more cigarettes and alcohol compared with healthy subjects. The frequency of a positive family history of allergies and asthma was higher among subjects with respiratory

Table 1. Selected characteristics among subjects with and without respiratory allergies

Characteristics	Subjects without respiratory allergies	Subjects with		
		Hayfever	House dust mite allergy	Asthma
Number of individuals	2399	326	111	114
Female (%)	48.6	48.6	55.9	44.2
Age (years)	39.6 (0.2)	35.2 (0.6)*	34.5 (1.0)*	37.9 (1.0)
School education (%)				
< 10 years	20.5	13.0	13.9	24.7
10 years	61.4	60.9	60.7	56.0
> 10 years	18.1	26.1*	25.4	19.3
Smoking (%)				
Never-smoker	30.5	38.7	39.1	18.2
Ex-smoker	28.0	32.2	27.1	29.3
Current smoker	41.5	29.2*	33.8	52.5*
Pack-years	8.2 (0.3)	4.3 (0.5)*	3.2 (0.6)*	10.1 (1.2)*
Alcohol consumption (%)				
Low	82.2	79.6	83.7	72.8
High	17.8	20.4	16.3	27.2*
Diabetes mellitus (%)	3.6	2.0	4.2	4.5
Number of siblings (%)				
0	11.9	16.3	15.4	9.9
1	33.9	40.2	33.2	33.4
2	22.9	22.2	30.0	22.4
≥ 3	31.3	21.3*	21.5	34.3
Positive family history for allergies or asthma (%)	13.7	27.0*	32.3*	26.1*
Number of teeth (%)				
> 10	5.9	2.0	2.7	8.3
10–20	16.5	10.9	14.2	13.3
> 20	77.7	87.2*	83.1	78.5
Attachment loss > 3 mm (% of sites)	37.2 (0.7)	24.3 (1.5)*	21.6 (2.6)*	32.7 (3.0)

Percentages and mean are weighted for non-response and were post-stratified. Continuous data are given as mean (standard error); nominal data are given as percentages. χ^2 -test (nominal data) or Mann-Whitney test (interval data).

*Differences between control group and allergy groups are statistical significant ($P \leq 0.05$).

Table 2. Dental care behaviour among subjects with and without respiratory allergies

Characteristics	Subjects without respiratory allergies	Subjects with		
		Hayfever	House dust mite allergy	Asthma
Frequency of brushing one's teeth (%)				
≥ 3 per diem	6.8	7.5	11.3	7.5
Twice daily	76.8	80.9	79.5	71.9
Once daily	14.6	11.0	9.2	17.5
Less frequent	1.8	0.6	0	3.1
Visit to the dentist in the last 12 months (%)				
0	11.8	9.9	10.5	13.0
1	20.7	17.2	17.1	19.5
2	32.8	33.3	30.7	33.2
3	11.7	12.9	13.0	10.3
>3	22.8	26.8	28.8	24.0
Dental care devices other than toothbrush				
Dental floss (%)	19.4	24.9*	27.4*	18.5
Toothpicks (%)	16.6	17.2	17.6	20.5
Interdental brush (%)	5.7	7.8	12.2*	6.1

Percentages are weighted for non-response and were post-stratified.

* χ^2 -test: differences between control group and allergy groups are statistical significant ($P \leq 0.05$)

allergies than among subjects without allergies. With respect to dental factors, subjects with hayfever or HDM allergy had less attachment loss. In asthmatic persons the extent of attachment loss was comparable with subjects having no respiratory allergies. We found no differences with respect to dental care behaviour such as frequency of brushing teeth or utilization of dental services between subjects with and without respiratory allergies, with two exceptions: individuals with hayfever as well as individuals with HDM allergy used more often dental floss or an interdental brush compared with subjects without these allergies (Table 2).

Three multivariable analyses were performed to test the independent associations between attachment loss and each respiratory allergy separately (Table 3). These analyses revealed that the extent of attachment loss was inversely associated with the risk of hayfever as well as HDM allergy. These inverse associations were consistent after adjustment for sex, age, school education, smoking status, alcohol consumption, family history of allergies or asthma and number of teeth. Similar to the crude analyses, individuals with severe periodontal conditions had again the lowest risk of hayfever ($P_{\text{trend}} = 0.01$) or HDM allergy ($P_{\text{trend}} = 0.02$). The results for asthma showed a slightly inverse association in the full-adjusted model, but this association was not consistent over all analyses. The difference between individuals with no periodontitis and severe attachment loss bordered statistical significance ($P = 0.07$).

Table 3. The periodontitis related risk of hay fever, house dust mite (HDM) allergy and asthma

Effect [§]	OR (95% CI) [†]		
	Hayfever	HDM allergy	Asthma
<i>Unadjusted</i>			
Attachment loss > 3 mm (ref: healthy)			
Mild	0.73 (0.5–1.0)*	0.62 (0.4–1.0)*	1.07 (0.6–1.8)
Moderate	0.57 (0.4–0.8)*	0.42 (0.2–0.7)*	0.94 (0.6–1.6)
Severe	0.30 (0.2–0.4)*	0.23 (0.1–0.5)*	0.61 (0.3–1.1)
	$P_{\text{trend}} < 0.0001$	$P_{\text{trend}} < 0.0001$	$P_{\text{trend}} = 0.16$
<i>Adjusted for sex and age</i>			
Attachment loss > 3 mm (ref: healthy)			
Mild	0.82 (0.6–1.1)	0.76 (0.5–1.3)	1.12 (0.7–1.9)
Moderate	0.73 (0.5–1.0)	0.61 (0.3–1.1)	1.04 (0.6–1.9)
Severe	0.44 (0.3–0.7)*	0.39 (0.2–0.9)*	0.73 (0.4–1.5)
	$P_{\text{trend}} = 0.0001$	$P_{\text{trend}} = 0.01$	$P_{\text{trend}} = 0.58$
<i>Adjusted for the full model[‡]</i>			
Attachment loss > 3 mm (ref: healthy)			
Mild	0.87 (0.6–1.2)	0.80 (0.5–1.3)	1.10 (0.6–2.0)
Moderate	0.80 (0.6–1.2)	0.64 (0.3–1.2)	0.96 (0.5–1.8)
Severe	0.53 (0.3–0.9)*	0.39 (0.2–0.9)*	0.48 (0.2–1.0)
	$P_{\text{trend}} = 0.01$	$P_{\text{trend}} = 0.02$	$P_{\text{trend}} = 0.11$

*statistical significant ($P \leq 0.05$)

[†]logistic regression analyses.

[‡]Full model include sex, age, school education, smoking status, alcohol consumption, family history for allergies or asthma and number of teeth.

[§]Periodontal status was formed by dividing participants into four groups according to the attachment loss > 3 mm: low, 0–7.7%; mild, 7.7–28.6%; moderate, 28.6–63.9%; severe, ≥ 63.9% attachment loss > 3 mm.

OR, odds ratio; CI, confidence interval.

Our analyses revealed three other variables, which were independently associated with hayfever, HDM allergy or asthma (Table 4). There was an increased risk of allergies in subjects with a positive family history of allergies or asthma. Moreover, the risk of asthma was increased in current and former smokers and in subjects with alcohol consumption.

Sensitivity analyses were run by using the self-reported physician diagnoses from allergy tests of hayfever or HDM allergy as alternative dependent variables. These analyses did not affect the main results of an inverse association between periodontitis and allergies. Furthermore, an additional analysis was run by using a new combined variable according to the presence of at least one of the three respiratory allergies as an endpoint. Besides this, analyses were run by using different categorizations of attachment loss as well as a combined variable of number of teeth (three categories) and attachment loss > 3 mm (four categories). Moreover, we tested further potential confounding factors like diabetes mellitus, dental hygiene parameters and the number of siblings in the models. All these analyses confirmed the main result of an inverse association between periodontitis and the three

Table 4. Further independent risk factors for hay fever, house dust mite (HDM) allergy and asthma

Effect	OR (95% CI) [†]		
	Hayfever	HDM allergy	Asthma
Sex (ref: men)	1.00 (0.8–1.3)	1.19 (0.8–1.8)	1.01 (0.7–1.6)
Age (ref: 20–29 years)			
30–39	0.96 (0.7–1.3)	0.88 (0.5–1.5)	1.18 (0.7–2.1)
40–49	0.58 (0.4–0.9)*	0.40 (0.2–0.8)*	0.80 (0.4–1.6)
50–59	0.39 (0.2–0.7)*	0.41 (0.2–0.9)*	0.83 (0.4–1.9)
School Education (ref: low)			
Medium	0.91 (0.6–1.3)	0.88 (0.5–1.6)	0.57 (0.3–1.0)*
High	1.14 (0.7–1.8)	1.06 (0.6–2.1)	0.72 (0.4–1.4)
Smoking (ref: never-smokers)			
Former	0.98 (0.7–1.3)	0.87 (0.5–1.4)	1.94 (1.1–3.5)*
Current	0.54 (0.4–0.7)*	0.64 (0.4–1.0)	2.20 (1.2–3.9)*
Alcohol consumption (ref: low)	1.30 (0.9–1.8)	1.14 (0.7–2.0)	1.66 (1.0–2.7)*
Family history for allergy/asthma (ref: negative)	2.07 (1.6–2.8)*	2.59 (1.7–4.0)*	2.24 (1.4–3.5)*
Number of teeth (ref: >20)			
<10	0.78 (0.3–2.0)	1.44 (0.4–6.2)	1.83 (0.6–5.3)
10–20	1.06 (0.7–1.6)	1.69 (0.9–3.3)	1.11 (0.6–2.1)

*statistical significant ($P \leq 0.05$).

[†]logistic regression analyses, adjusted for the full model: sex, age, school education, smoking status, alcohol consumption, family history for allergies or asthma, number of teeth and attachment loss.

OR, odds ratio; CI, confidence interval.

respiratory allergies. Further analyses were run for all subjects aged 20–79 years. Also these analyses confirmed the main results of an inverse association between periodontitis and hayfever ($P_{\text{trend}} = 0.004$), HDM allergy ($P_{\text{trend}} = 0.03$) as well as asthma ($P_{\text{trend}} = 0.04$).

Discussion

Our analysis revealed inverse associations between periodontitis and hayfever as well as periodontitis and HDM allergy in a population-based sample of 2837 subjects. The risk of hayfever or HDM allergy decreased with increased attachment loss. These results persisted after adjustment for confounding factors and over a number of sensitivity analyses. To the best of our knowledge we are the first to investigate such an association.

Periodontitis is an infection caused by Gram-negative, anaerobic bacteria. Periodontopathic bacteria were already detected in saliva and dental plaque of children aged 1–15 years [19]. An early infection with these bacteria may elicit an immune response [24], and thus promotive conditions for the onset of periodontal diseases already emerge in childhood. In the aetiology of periodontal disease the involvement of T cell subsets is equivocal [25]. One aetiology model suggests that periodontitis is

Th2 mediated. The outcome of this is the activation of B cells and the production of IL-1, which probably mediates tissue destruction. A second model suggests that Th1 cells and IFN- γ mediate the infection. IFN- γ stimulates macrophages and monocytes to produce pro-inflammatory cytokines which cause the destruction of tissue [25]. A number of studies with conflicting results have been published. The finding of a decreased IFN- γ and IL-2 mRNA expression in peripheral blood cells in patients with periodontitis [26], an association between IL-4 gene polymorphisms and early-onset periodontitis [27] as well as a decreased number of mast cells in chronic periodontitis tissue [28] implicated a predominance of Th2 cells in periodontitis. On the other hand, other studies reported an increased mRNA expression of INF- γ and IL-2 in adult periodontitis patients [29, 30]. This argues for a major role of Th1 cells in periodontitis. A third model proposes that different phases of the natural history of periodontal disease are mediated by different T cell subsets. Th1 cells are associated with stable lesions, whereas Th2 cells may be the major mediator in advanced and progressive periodontal lesion [31]. All these conflicting results are difficult to interpret and to reconcile because of differences in used material and methods.

The hygiene hypothesis is based on the Th1/Th2 paradigm and the inverse relation between Th1-mediated (infections) and Th2-mediated (allergies) immune responses [5, 6]. While inverse associations between Th1-mediated infections like tuberculosis, measles and hepatitis A and allergic diseases have been demonstrated [9–11], an inverse relation between a Th2-mediated infection by helminths and allergies in children have also been reported [13]. Children with worm antigens had an increased release of IL-10 compared with healthy children. This led to the assumption that IL-10 suppress the effect of IgE by inhibiting the production of further inflammatory cytokines. Another study found that IL-10 releasing inflammatory leucocytes were much more common in periodontitis than pro-inflammatory cytokine-releasing cells [32].

Our study revealed a weak inverse relation between periodontitis and asthma. After adjustment for confounding factors, subjects with severe periodontitis had the lowest risk of asthma. The relation between oral health status and asthma is controversially discussed. Recent studies demonstrated that asthma had no influence on the dental health status particularly on caries and plaque in children [33] and on the gingival health in children and adolescents [33, 34]. On the other hand several studies reported that asthmatic adults had a higher degree of periodontal inflammation and a lower stimulated salivary flow compared with non-asthmatic adults and reasoned that asthmatic subjects had a higher risk of oral diseases [35, 36]. The same was observed in children. Asthmatic children had more plaque deposits, higher levels of

gingivitis as well as more teeth affected by tooth surface loss compared with healthy children [37].

For the pathogenesis of periodontitis the colonization of the oral cavity with periopathogenic microorganisms is necessary. Factors such as smoking, diabetes, male gender or genetic predisposition are also important for the onset of periodontitis [38]. Complex interactions between bacteria and risk factors cause periodontitis and its severity. The present analysis refers to the current diagnosis of periodontitis and we have no information concerning the childhood situation. Therefore, we were not able to investigate which specific germs may have been responsible for the inverse associations between periodontitis and hayfever as well as periodontitis and HDM allergy. Our findings are supported by a cross-sectional study which demonstrated that the subgingival colonization with the causal periodontopathic bacteria *P. gingivalis* and *B. forsythus* was positively associated with alveolar bone loss and attachment loss, whereas allergies were negatively associated with alveolar bone loss and attachment loss [39, 40].

We ascertained that alcohol consumption appears to be a potential risk factor for hayfever. This is in line with studies that have analysed the influence of alcohol consumption on serum IgE levels and found that the consumption of alcohol was associated with increased serum IgE concentrations [41, 42]. Alcohol consumption leads to an imbalance between Th1 and Th2 cytokine production, which might increase IgE production [43]. Moreover, our logistic regression results confirm the known relation between allergies and positive family history for allergies or asthma [44].

The prevalence data of hayfever and asthma from our study are consistent with reported prevalence proportions for adults in Germany [45, 46]. The prevalence of HDM allergy in our study was 3.8% and is rather low when compared with the 11–15% prevalence reported in the MONICA study [47]. However, it should be considered that the present study was performed with in adult East German population and there are considerable regional differences in the prevalence of allergic diseases in Germany, with East Germany having lower proportions of allergies [48].

One of the strengths of the study was the accurate determination of the periodontal status by trained and certified dentists. Moreover, we performed our analyses using data of a large population-based sample. One limitation of the study was the method for defining respiratory allergies, because self-report inherits the risk of recall bias. The interview included no validated questionnaire for the ascertainment of hayfever, HDM allergy and asthma. Furthermore, we had no information about current and past allergic or asthmatic symptoms. However, hayfever, HDM and asthma were related to periodontitis in the same direction. Furthermore, the inverse

associations between periodontitis and hayfever as well as HDM allergy persisted when physician diagnoses of hayfever or HDM allergy were used as alternate exposure variables. These results argue for a good validity of the exposure definition that was used in the present study. A further limitation may exist in our assumption that periodontal disease measured in adults implicates the colonisation of the oral cavity with periodontitis-inducing microorganism in childhood and in youth. Unfortunately, we were not able to obtain additional information on the previous history of periodontal disease in affected persons. While a cross-sectional study like the present is generally not suitable to prove causal relations, the biological plausibility of the association may indicate such a relation. Even though we were careful to adjust for possible confounding factors, further residual confounding cannot be unequivocally excluded. To investigate possible reverse causality, we tested whether allergic diseases influenced oral hygiene. Some differences were only present in descriptive statistics and did not affect the major result of an inverse relation between periodontitis and hayfever, HDM allergy as well as asthma in the multivariable models. Further case-control and cohort studies are needed to confirm the hypothesis of an inverse relation between periodontal and respiratory allergies that was generated by the present study.

In conclusion there is an inverse association between periodontitis and respiratory allergies. The present findings might support the hygiene hypothesis.

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