

Epidemiology of allergic diseases

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The escalation of allergic diseases (hay fever, asthma, atopic eczema) over recent decades has been linked to an increase in environmental pollutants. The prevalence of hay fever is associated with genetic predisposition, and some reports show an association with urban areas, socioeconomic status, and combined high allergen and automobile exhaust exposure. In asthma, there is also some evidence for geographical variations in prevalence; exercise challenge tests prove positive more often in urban areas than in rural areas. Although genetic predisposition is the strongest single risk factor for atopic eczema, air pollutants may aggravate the condition by acting as unspecific irritants and immunomodulators, leading to increased immunoglobulin E expression. In a study of 678 pre-school children, the influence of maternal smoking habits on individual measures of atopy revealed a positive association between smoking during pregnancy/lactation, and a positive history of atopic eczema. An East-West German comparative study examining different types and levels of air pollution, i.e. sulphurous (industrial; East) and oxidising (urban; West), showed that the prevalence of atopic eczema was greatest in East Germany. When various direct and indirect parameters of air pollution exposure were measured, the greatest association with atopic eczema was found with NO_x exposure (indoor use of gas without a cooker hood), and close proximity to roads with heavy traffic. The increased prevalence of atopic eczema cannot be explained by changes in study methodology over time, or conventional risk factors alone; environmental risk factors may be an important contributing factor.

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Introduction

The manifestation of atopic eczema, hay fever, and bronchial asthma, which constitute the classical triad of atopic diseases, largely depends on genetic and environmental factors. Within recent years, increasing interest has been paid to allergic disease by both the scientific community and the general public. The mass media, in particular, has highlighted dramatic increases in all allergic diseases and the causal role of environmental pollutants. With regard to the postulated increase of allergic diseases, several explanations must be taken into account which might suggest a pseudo-increase rather than a true numerical increase. Diagnostic methods have improved thereby making the diagnosis of allergy easier. Furthermore, patients' behaviour and knowledge has also changed – today they are more likely to recognize a potential allergic reaction and seek earlier advice from a doctor. Despite these arguments, longitudinal studies from numerous countries provide good evidence to suggest a significant increase in all major

atopic diseases, but the underlying cause remains unclear. Without doubt, the amount of allergens, and possibly their allergenicity, has increased and therefore the number of allergic reactions may also be enhanced. Research into environmental pollutants as risk factors for allergic disease is relatively new, and while preliminary results show some evidence for an association, further investigation is needed.

Epidemiology: definitions and methodology

Epidemiology is acknowledged as the science of occurrence of disease in the human population (1), although a generally used and accepted definition is not available. In its simplest form, epidemiology can provide measures of frequencies of a disease or symptom for a defined population. Such measures include the prevalence, incidence rate, and cumulative incidence, depending on the type of study and available data. However, the time when epidemiology was regarded as the science of counting

has ended. By using sophisticated techniques of design and data analysis, modern epidemiology is able to identify independent relevant risk factors which can be used to model pathways of exposure, or to discover new, possibly hazardous, influences. Accordingly, epidemiological findings can have a strong impact on public health in terms of knowledge, education, and disease prevention. The advantage of the epidemiological approach is that the results are directly applicable to the population, i.e. no extrapolation from different species and dosages is necessary. Furthermore, all 'real life' influences are already taken into account, in contrast to the scenario in standardized experimental settings.

As well as cross-sectional studies, cohort and case-control studies are the major practical elements of an epidemiological investigation; they allow for different measures of frequency and association. The relationship between exposure and outcome is usually expressed as an odds ratio (OR) in case-control studies or as a relative risk in cohort studies. Stability is provided by the corresponding confidence interval (CI) or p value. Study designs usually allow a 5% probability of committing a type I error, i.e. rejection of the Null hypothesis despite it being true. In order to yield the appropriate answer to the question being addressed, it is necessary to formulate the hypotheses *a priori* and to include the corresponding instruments in the study design. This also implies a power calculation and estimation of the necessary number of cases and controls. With regard to the instruments used, standardized and validated measures should be chosen, which allow for comparisons. Several potential systematic errors (selection bias, misclassification bias, and confounding factors) have to be considered when planning and analyzing a study – even the best statistical methods cannot compensate for flaws in study design or data assessment.

Allergy and atopy

The term 'allergy' was coined by the Austrian paediatrician Clemens von Pirquet (2) at the beginning of this century. The definition has changed over the years and today we see 'allergy' as a specific change of the immune system leading to symptomatic hypersensitivity (3). 'Atopy' was introduced by Coca and Cooke (4) in the 1920s to distinguish different types of hypersensitivity. We understand atopy as familial hypersensitivity of the skin and mucous membranes against environmental substances, associated with increased immunoglobulin E (IgE) synthesis and/or altered non-specific reactivity (5).

Frequency of atopic diseases

Early reports on the frequency of atopic diseases are limited. These early estimates should be interpreted with caution, since the methodology and instruments used are often unclear. Direct comparisons with recent studies, in order to determine changes in frequency, are confounded by such methodological differences and therefore do not result in correct estimates. The assessment of frequency is influenced largely by the instruments used, the structure of the population (sex, age, occupation, other selection criteria), and other external factors (location, time of investigation). There are, however, studies from different countries in which repeated measures have been conducted after periods of 10 years or more, in comparable populations and with comparable instruments. These studies allow a valid estimate of time trends for these atopic diseases.

Hay fever

Frequency estimates for hay fever in different countries are summarized in Table 1. The figures range from 1.6% (20) to 24.0% (14) depending on the population investigated. In our study (21), conducted from 1989–1991 in Germany, 9.2% of 988 pre-school children (5–6 years old) reported symptoms of allergic rhinitis. Overall, 13.2% of their mothers and 10.1% of their fathers had positive reports of allergic rhinitis. As in other atopic diseases, the manifestation of hay fever depends largely on genetic predisposition. In families where one parent suffers from hay fever, the estimated risk of the child developing hay fever is between OR 3.6 (95% CI: 2.9–4.6) (22) and OR 4.4 (95% CI: 2.7–7.0) (21).

Table 1. Frequency of allergic rhinitis

Author	Year	Country	Sample size	Frequency (%)
Hagy and Settupane (6)	1969	USA	1,836	21.1
Kjellman (7)	1977	Sweden	1,325	3.8
Skarpass and Gulsvik (8)	1985	Norway	1,772	5.4
Wüthrich et al. (9)	1986	Switzerland	2,524	9.6
Bakke et al. (10)	1990	Norway	4,992	10.0
Drosner et al. (11)	1991	Germany	511	19.6
Frischer (12)	1991	Germany	2,381	4.4–13.9
Poeyssae et al. (13)	1991	Finland	3,649	6.0
Sibbald et al. (14)	1991	UK	2,969	24.0
Kuehr et al. (15)	1992	Germany	1,376	6.9
von Mutius et al. (16)	1992	Germany	6,081	2.4–8.6
Varjonen et al. (17)	1992	Finland	416	14.0
Saval et al. (18)	1993	Denmark	4,952	10.5
Dotterud et al. (19)	1994	Norway	551	20.6
Leung and Jenkins (20)	1994	Australia	737	1.6
Schäfer and Ring (21)	1995	Germany	988	9.2–13.2

Table 2. Trends in the frequency of allergic rhinitis

Author	Year	Country	Sample size	Prevalence (%)
Eaton (37)	1974	UK	11,065	4.6
	1979		11,042	5.7
Fleming (38)	1970	UK	24,000	10.5
	1981		28,000	19.7
Ninan et al. (39)	1964	UK	2,510	3.2
	1989		3,403	11.9
Varonier et al. (40)	1968	Switzerland	4,781	0.5 (age 4-6)
			2,451	4.4 (age 15)
			3,270	1.1 (age 4-6)
			3,500	6.1 (age 15)
Aberg (41)	1971	Sweden	55,393	4.9
	1981		57,150	8.4

The prevalence of hay fever appears to be low in children and reaches a peak in early adulthood (23). Although there are some variations in the sex ratio, no consistent differences with regard to sex have been reported (24). The question of whether hay fever is more common in urban or rural areas cannot be answered as yet. Some reports suggest a higher prevalence in urban communities (23, 25, 26), but this is not confirmed by others (24). Although socioeconomic status seems to play a role in the process of sensitization and subsequent allergic disease, it is not clear which circumstances contribute to this association. A British survey found the prevalence of hay fever to be higher in children of 'advantaged' households (5.3%) than in those from 'disadvantaged' households (3.1%) (27). This association was confirmed by an American study which took annual income into consideration (23), but was not substantiated by a Danish investigation (28). Environmental contaminants, particularly air pollutants, may contribute to the process of sensitization and therefore enhance the prevalence of hay fever. Experiments have shown an adjuvant effect of diesel exhaust and other particles (29, 30), sulphur dioxide (31), nitrogen dioxide (32), and ozone (33, 34) on IgE synthesis. Furthermore, as well as being carcinogenic, tobacco smoke is known to act as an irritant and an enhancer of IgE synthesis (35). An epidemiological study showed that the prevalence of pollinosis to cedar pollen is significantly greater in areas with high allergen and automobile exhaust exposure compared with those regions with either high allergen or pollutant load, or mountainous areas where allergen and pollutant exposure is minimal (36). At least five studies from Europe indicate that hay fever has increased significantly over recent decades (Table 2).

Asthma

Difficulties in obtaining valid estimates of the frequency of disease are especially prominent when

dealing with asthma. Although well known as a clinical disease, diagnostic criteria need to be further established, particularly for the purpose of epidemiological studies. Frequency assessments may rely on questionnaires or interviews to solicit symptoms, or a doctor's diagnosis. Various lung function parameters may also be considered for the diagnosis of asthma in epidemiological studies. The frequency of asthma or asthma-related symptoms (wheezing) is summarized in Table 3.

As well as differences between various cohorts, it seems reasonable to believe that geographical variation in the prevalence of asthma may exist. Most studies suggest that asthma is more prevalent in boys (45), although this difference seems to disappear in adulthood (46). A few studies have shown that exercise challenge tests are positive more often in urban than in rural areas (47, 48). Again a strong genetic impact is observed, leading to risks between OR 2.0 (95% CI: 0.48-7.3) (22) and 2.6 (95% CI: 1.7-4.0) (21), for children with one affected parent. As for hay fever, there is also good evidence to suggest that asthma has increased in prevalence over recent decades (Table 4).

Atopic eczema

Atopic eczema develops as a result of genetic predisposition combined with environmental influences. It is the most common inflammatory skin disease in childhood, though a lack of generally accepted diagnostic criteria make epidemiological studies difficult. Diagnosis by an experienced dermatologist is the gold standard for frequency estimates. Questionnaires or interviews have to take several types of information into account (location, typical signs, subjective symptoms, duration and course, family history, allergy tests). Prevalence estimates for eczema in the early literature

Table 3. Frequency of asthma/symptoms

Author	Year	Country	Sample size	Frequency (%)
Broder et al. (23)	1974	USA	9,226	6.0-7.2
Taylor et al. (27)	1983	UK	13,135	2.0
Skarpass and Gulsvik (8)	1985	Norway	1,772	1.6-3.1
Hurry et al. (42)	1988	Australia	3,581	10.8-17.3
Angioni et al. (43)	1989	Italy	1,691	3.3
Poeyssae et al. (13)	1991	Finland	3,649	4.3
Barry et al. (44)	1991	New Zealand	873	17.0
		South Wales	965	12.0
Kuehr et al. (15)	1992	Germany	1,376	10.2
von Mutius et al. (16)	1992	Germany	6,081	7.3-9.3
Varjonen et al. (17)	1992	Finland	416	2.5
Dotterud et al. (19)	1994	Norway	551	12.3
Leung et al. (20)	1994	Australia	737	1.9
Schäfer and Ring (21)	1995	Germany	988	1.5-3.8

Table 4. Trends in the frequency of asthma/symptoms

Author	Year	Country	Sample size	Prevalence (%)
Eaton (37)	1974	UK	11,065	3.9
	1979		11,042	4.5
Fleming (38)	1970	UK	24,000	11.6 (men) 8.8 (women)
	1981		28,000	20.5 (men) 15.9 (women)
Ninan and Russell (39)	1964	UK	2,510	4.1
	1989		3,403	10.2
Varonier et al. (40)	1968	Switzerland	4,781	1.7 (age 4-6) 2.451 (age 15)
	1981		3,270	2.0 (age 4-6) 3,500 (age 15)
Aberg (41)	1971	Sweden	55,393	1.9
	1981		57,150	2.8
Burney et al. (49)	1973	UK	29,156	2.0
	1986		29,156	4.1

range from 1.1% to 3.1%. In more recent studies, prevalences as high as 25% for questionnaire-based studies and 12.9% for examination-based investigations have been documented. Table 5 outlines the frequency of eczema in chronological order. As for respiratory atopic diseases, there is good evidence to indicate that atopic eczema has increased

in frequency over time. Relevant information on trends is summarized in Table 6.

Genetic predisposition is the strongest single risk factor for atopic eczema. If one parent also has a positive history, the risk for the offspring is estimated to be between OR 3.4 (95% CI: 2.6-4.4) (22) and 6.2 (95% CI: 3.3-11.5) (21). Eczema usually develops early in life; a study of 1,200 hospital cases (69) suggested that the disease is present in 57% of children before 1 year of age and in 87% before the age of 6 years. Not all patients continue to suffer throughout their lifetime; according to re-examinations after 13-25 years, between 44-83% still had eczema (70-72). Atopic eczema obviously follows an annual course, with 56-92% of patients recognizing season-dependent changes in their skin condition (73-75). In most cases, improvement occurred during the summer months, which may be attributed to the favourable climatic conditions, including increased ultraviolet light, during this period. Some patients, however, experience an exacerbation after light or pollen exposure. Evidence suggests that, at least in childhood, more girls than boys suffer from atopic eczema, with sex ratio values up to 2.6 in favour of girls (51, 55, 57, 76). In our study, gender was also identified

Table 5. Frequency of (atopic) eczema

Author	Year	Country	Sample size	Instrument	Frequency (%)
Service (50)	1939	USA	3,141	Q	2.9
Erisson-Lihr (51)	1955	Finland	48,325	Q	3.0
Walker and Warin (52)	1956	UK	1,024	Q	3.1
Brereton et al. (53)	1959	UK	4,006	Q	1.1
Freemann and Johnson (54)	1964	USA	2,627	Q	1.4
Larsson and Liden (55)	1980	Sweden	8,298	E	3.0
Fergusson et al. (56)	1981	New Zealand	11,435	MR	20.4
Engbak (57)	1982	Denmark	4,400	Q	9.7
Skarpass and Gulsvik (8)	1985	Norway	1,772	Q	8.1
Storm et al. (58)	1986	Denmark	1,210	Q	8.9
Bakke et al. (10)	1990	Norway	4,992	Q	25.0
Drosner et al. (11)	1991	Germany	511	Q/E	1.0
Poeyssae et al. (13)	1991	Finland	3,649	Q	1.7
von Mutius et al. (16)	1992	Germany	6,081	Q	13.9
Kuehr et al. (15)	1992	Germany	1,376	Q	17.3
Varjonen et al. (17)	1992	Finland	416	E	9.7
Buser et al. (59)	1993	Germany	4,651	Q	11.8
Saval et al. (18)	1993	Denmark	4,952	Q	7.0
Okuma (60)	1994	Japan	10,137	Q	9.5
Kay et al. (61)	1994	UK	1,077	Q	20.2
Leung and Jenkins (20)	1994	Australia	737	Q	10.4
Bobak et al. (62)	1995	Czech Republic	2,060	Q	3.9-6.3
Dotterud et al. (63)	1995	Norway	424	Q/E	26.0
Henderson (64)	1995	Tanzania	548	E	0.7
Neame et al. (65)	1995	UK	322	E	14.0
Schäfer and Ring (21)	1995	Germany	988	Q	2.5-8.3
Schäfer et al. (66)	1996	Germany	1,273	E	12.9

Q=Questionnaire; E=Examination; MR=Medical Record.

Table 6. Trends in the frequency of atopic eczema

Author	Year	Country	Instrument	Sample size	Prevalence (%)
Schultz-Larsen et al. (67)	1964–69	Denmark	E	592	3.2
	1970–74				10.2
Taylor et al. (68)	1946	UK	Q/MR	4,624	5.1
	1958			14,498	7.3
	1970			12,982	12.2
Eaton (37)	1974	UK	Q	11,065	9.3
	1979			11,042	11.9
Ninan and Russell (39)	1964	UK	Q	2,510	5.3
	1989			3,403	12.0
Varonier et al. (40)	1968	Switzerland	Q	4,781	2.2 (age 4–6)
				2,451	2.3 (age 15)
	1981			3,270	2.8 (age 4–6)
				3,500	1.5 (age 15)
Aberg (41)	1971	Sweden	Q	55,393	4.9
	1981			57,150	8.4

Q=Questionnaire; E=Examination; MR=Medical Record.

as an independent risk factor (66). Socioeconomic status is also important for the manifestation of this disease. A UK study has shown a positive association between social class and atopic eczema (77), and a similar observation was made when looking at duration of parental school education as proxy for socioeconomic status (66). There is no doubt that food (78, 79), aeroallergens (80, 81), and psychological factors (82) play a role in the expression of the disease. However, the observed increase in atopic eczema cannot be explained by secular changes in methodology or the above-mentioned risk factors alone.

Environmental contaminants, particularly air pollutants, have been suspected as potential aggravating factors in atopic eczema. This is biologically plausible, since most air pollutants act as unspecific irritants as well as immunomodulators, leading to elevated levels of total serum IgE (29, 30, 83). Exposure to air pollutants, particularly tobacco smoke, has been linked to chronic inflammation of the upper respiratory tract, impaired lung function in children, and asthma (84–86). Exposure to maternal environmental tobacco smoke also results in immunological changes in the offspring (87). Children born to mothers who smoked during pregnancy showed elevated levels of IgE, IgA and IgG₃ (88, 89) in their cord blood, as well as other altered immune functions (90). Although biologically plausible, there is still little experimental or epidemiological evidence for an association between exposure to (air) pollutants and atopic eczema.

To address this question we conducted a multi-centre cross-sectional study in different regions of Bavaria/Germany (91). This involved 678 pre-school children (5–6 years old) who were scheduled

for a compulsory school entrance examination. A standardized questionnaire was used to obtain data on personal and family history of atopic diseases as well as other relevant sociodemographic parameters. A skin prick test was performed with common aeroallergens using a multipuncture device.

In total, 12.6% (84/666) of mothers reported that they had smoked during pregnancy (62/84; 73.8%) or lactation (22/84; 26.2%). Risk factors for the manifestation of atopy were analyzed; the outcome variable 'manifestation of atopy' was defined as a positive individual history for atopic eczema, hay fever, or asthma, or a positive skin prick test to grass pollen, house dust mite, or cat fur. Manifestations of atopy were observed in 158/421 (37.5%) children who were included in the final analysis. In multivariate analysis, a significant positive association between maternal smoking during pregnancy and manifestation of atopy was obtained; 24/64 (52.2%) children whose mothers smoked during pregnancy showed manifestations of atopy compared with 134/375 (35.7%) for children of non-smoking mothers ($P<0.044$). The influence of maternal smoking habits on the individual measures of atopy was further analyzed bivariate. Smoking during pregnancy and/or lactation was not associated with the child's skin prick test reactivity or a history of respiratory atopic disease, but was associated with a positive history of atopic eczema. Of the children whose mothers did not smoke, 18.1% (98/541) had a history of atopic eczema, whereas atopic eczema was reported in 33.8% (26/77) of the children whose mothers had smoked during pregnancy and/or lactation (OR 2.30; 95% CI: 1.32–3.12).

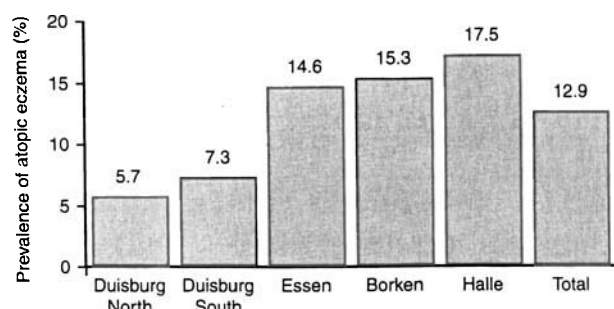


Fig. 1. Prevalence of atopic eczema (examination) in pre-school children in an epidemiological study in East and West Germany ($n=1,086$). From Schäfer et al. (66), with kind permission.

East-West German comparisons

The reunification of Germany in 1989 offered the unique opportunity to compare cohorts of children exposed to different degrees and types of air pollutants over a long period of time. It can be assumed that there is no substantial genetic heterogeneity between subjects in East and West Germany. When discussing air pollutants, a distinction is usually made between two types of pollution (92). The first is characterized as the reducing type, resulting mainly from incomplete combustion of coal (with high sulphur content) and conditions of fog and cool temperatures. Typical components of this air pollutant pattern, also known as the 'London type', are sulphur dioxide, dust, and particles. This air pollutant pattern is most prominent in East Germany.

The second type of air pollution, the so-called oxidizing or photochemical type, is the result of atmospheric reaction products of automobile exhaust fumes and is more prominent in West Germany. It is characterized by components such as oxides of nitrogen (NO_x), hydrocarbons, and photochemical oxidants such as ozone. Development of this type of air pollution is promoted by a combination of high traffic intensity and sunlight, which is common in margin zones of larger cities. Such circumstances are aggravated when the pollutants are trapped by meteorologic inversion layers, which often occurs, for example, in Los Angeles. This distinction also applies for different regions in Germany where type I pollution is characteristic for East Germany and the second type is more prominent in inner parts of West German cities (93).

Since 1991, we have had the opportunity to participate in a study of pre-school children in East and West Germany, investigating the influence of numerous pollutants on different health outcomes. The study was initiated and headed by the Medical Institute for Environmental Hygiene in Düsseldorf,

parts of which have already been published (94–96). In 1991, 1,273 pre-school children (5–7 years old) underwent dermatological examination in East (Halle an der Saale) and West (Duisburg North/South, Essen, Borken) Germany (66). At the time of investigation, 12.9% of the children were diagnosed with atopic eczema. The highest prevalence was found in East Germany (17.5%) (Fig. 1).

Atopic eczema was tested in a logistic regression model for the influence of direct and indirect parameters of air pollutant exposure, including the distance between the home and the nearest road with heavy traffic, exposure to automobile exhaust for >1 h/day, indoor use of gas, exposure to environmental tobacco smoke, and location. Gender, age, genetic predisposition, bedroom sharing, education level of the parents, and the month of investigation were also included as possible confounding factors. An explorative analysis was added to determine the effect of three other possible influencing factors – contact with animals, presence of animal fur in bedrooms, and previous helminthic infection.

As a result of these analyses, girls were found to be more susceptible to atopic eczema than boys (OR 0.63; 95% CI: 0.43–0.92 boys vs girls) (Table 7), with differences being most prominent in Borken (8.9% in boys, 20.2% in girls) and Duisburg-South (3.7% in boys, 11.3% in girls). A positive family history of allergic disease was also associated with atopic eczema (OR 1.52; 95% CI: 1.03–2.25). In all study sites, except Essen, atopic

Table 7. Risk factors for atopic eczema in an epidemiological study in East and West Germany. From Schäfer et al. (66), with kind permission

Parameter	OR	95% CI
Male gender	0.63	0.43–0.92*
Location (reference Borken)		
Duisburg-South	0.52	0.30–0.96*
Duisburg-North	0.42	0.15–1.16
Essen	0.85	0.46–1.54
Halle	1.39	0.77–2.52
Positive family history of atopic disease	1.52	1.03–2.25*
Month of investigation (May vs. April; March vs. February)	0.55	0.37–0.81*
High parental education level	1.83	0.83–4.02 ^a
Contact with rabbits	1.81	0.96–3.41
	2.90	1.36–6.19 ^{a,b}
Animal fur in bedroom	2.17	1.01–4.67*
Indoor use of gas without hood	1.68	1.11–2.56*
Homes close to high traffic road (<50 m)	1.71	1.07–2.73*
Previous helminthic infection	1.61	0.98–2.64

* Adjusted for: age, bedroom sharing, environmental tobacco smoke, exposure to automobile exhaust (>1 h/day).

^a significant in Duisburg-South ($P=0.039$).

^b results for girls.

eczema was more frequent in children whose parents had graduated after 10 years or more at school. This difference was significant for Duisburg-South ($P=0.039$), but not for the total study population (OR 1.83; 95% CI: 0.83–4.02) (Table 7).

As explained earlier, the clinical manifestation of atopic eczema has an annual course. A significantly lower rate of disease was found in the second of two subsequent months during the February–May examination period (OR 0.55; 95% CI: 0.37–0.81 May vs April, and March vs February).

The use of indoor gas without a hood, used as an indirect parameter for NO_x exposure, was found to be associated with atopic eczema (OR 1.68; 95% CI: 1.11–2.56), i.e. around 70% more cases were observed in households with an indoor NO_x source. Higher rates were also found in children whose homes were located closer than 50 m to a heavy traffic road (OR 1.71; 95% CI: 1.07–2.73); this may indicate that automobile exhaust emissions are a risk factor for atopic eczema (Table 7).

After controlling for confounding factors, the prevalence of disease was still markedly lower in Duisburg compared with the control region, Borken. The difference was significant for Duisburg-South (OR 0.52; 95% CI: 0.30–0.96) (Table 7).

With respect to allergen exposure, atopic eczema was found to be associated with animal (rabbit) contact (OR 1.81; 95% CI: 0.96–3.41), which was higher in girls (OR 2.90; 95% CI: 1.36–6.19), and the presence of animal fur in the bedroom (OR 2.17; 95% CI: 1.00–4.67). There was also an association with previous intestinal parasitic infections (OR 1.61; 95% CI: 0.98–2.64) (Table 7). Inclusion of these factors in the basic logistic regression model changed the adjusted OR only marginally.

This first comparative epidemiological study on the prevalence of atopic eczema in East and West Germany, including an actual dermatological examination, yielded a high average prevalence with a great variation between different study sites. The prevalence of disease in East Germany was higher (17.5%) than in all other study regions and also higher than in comparable studies (17, 97).

Anamnestic parameters of exposure to air pollutants, which may lead to an increase in the total IgE level, were associated with the manifestation of atopic eczema (use of gas without hood, living near high traffic roads). This East-West German comparison yielded some unexpected results. As published earlier (94), the frequencies of respiratory allergies and specific IgE levels were not elevated in East Germany, though total IgE levels were

significantly higher compared with West German regions. Similar results were obtained in another East-West German study (16). Atopic eczema seems to follow a different course to respiratory atopic diseases. The complex subject of the influence of air pollutants on allergic sensitization and disease needs further research in experimental, epidemiological and clinical studies.

Conclusion

The increase in all major atopic diseases cannot be explained by secular changes in study methodology or conventional risk factors alone. There is evidence that environmental pollutants can contribute to the manifestation of atopic diseases. Future epidemiological studies must elaborate on the best measure of outcome (actual examination) and exposure (personal sampling). Modern techniques in biostatistics should take known confounding factors into account.

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