

Clinical atopy and associated factors in primary-school pupils

Kuehr J, Frischer T, Karmaus W, Meinert R, Barth R, Urbanek R.
Clinical atopy and associated factors in primary-school pupils.
Allergy 1992; 47: 650–655. © Munksgaard 1992

To investigate potential risk factors for clinical atopy in childhood, we obtained cross-sectional data from a cohort of 1376 8-year-old pupils. Parental atopy (hay fever, asthma, eczema), gestational age, maternal smoking habits, and the child's history of asthma, hay fever, and eczema were ascertained by questionnaire. Combining the history and the result of a skin prick test using seven aeroallergens, we defined the child's atopic diseases.

Of the population evaluated, 25.4% were categorized as atopic (10.2% allergic asthma, 17.3% eczema, 6.9% hay fever). As compared with the clear nonatopics (40.2%), parental atopic diseases were more prevalent in each of the atopic groups. Significant associations of the parents' and child's disease were obvious for eczema and hay fever. Low gestational age (LGA) was more frequent in children with any atopy or with an allergic asthma (odds ratio (OR) 1.7; 95% confidence interval (CI) 1.02–2.97; OR 2.8; 95% CI 1.5–5.4). Hay fever and allergic asthma occurred less frequently in girls (OR 0.5; 95% confidence interval 0.3–0.8; OR 0.6; 95% CI 0.4–0.9).

In conclusion, our data underline the importance of parental atopy for the clinical outcome in the offspring. In addition, LGA appears to be a risk factor for allergic asthma and for general atopy in later life.

Clinical aspects

Atopic diseases are frequent conditions in a cohort of 1376 primary-school pupils. Starting from any atopy (25.4%), seven disjunctive subgroups exist: eczema, allergic asthma, and hay fever alone were found in 12.5%, 4.1%, and 1.7%, respectively; combinations of eczema with allergic asthma or hay fever were obvious in 2.0% and 1.0% respectively; and allergic asthma together with hay fever occurred in 2.3%; all of the three atopic diseases were present in 1.8%. On the basis of associations found by us, we conclude that not only children of atopic parents but also subjects born with low gestational age have to be regarded as populations at risk for the development of clinical atopy.

A wide spectrum of tests can be used to define atopic diseases in epidemiologic studies. Standardized questionnaires or interviews are instruments to ascertain experienced symptoms of atopy in the subject's history (8). Furthermore, a previous doctor's diagnosis might identify those with a history of illness (8). Specific IgE antibodies and reactions to skin testing with allergens are biologic markers; however, even when positive, both may occur without clinical significance (15). The aims of this study were the description of clinical phenotypes based on a history of illness combined with sensitization in skin

prick test (SPT), and the analysis of risk factors for clinical atopy at school age.

Material and methods

Population

The data were collected in a first cross section of a longitudinal study designed to assess risk factors for atopy in childhood. In order to obtain a population-based sample of schoolchildren 6–8 years of age, we distributed questionnaires to all parents whose children had started primary school in three urban areas

J. Kuehr¹, T. Frischer¹, W. Karmaus²,
R. Meinert¹, R. Barth¹, R. Urbanek³

¹ University Children's Hospital, Freiburg, Germany; ² NORDIG Institute for Health Research and Prevention, Hamburg, Germany; ³ University Children's Hospital, Vienna, Austria

Key words: asthma; atopy; eczema; hay fever; low gestational age; parental atopy; skin prick test.

Joachim Kuehr
University Children's Hospital
Mathildenstr. 1, W-7800 Freiburg
Germany

Accepted for publication 3 August 1992

Table 1. Definition and prevalence of different indicators of atopy

Indicator	Definition	Prevalence (%)		
		Atopic group (n=350)	Unspecif. group (n=473)	Total group (n=1376)
1) Cough after exercise	Does your child frequently cough after exercise or when exposed to cold air or fog?	23.6	23.1	13.9
2) Wheeze	Did you ever hear a whistling or wheezing noise during your child's breathing?	47.6	46.6	28.2
3) Shortness of breath/cough in the night/morning	Does your child have a cough or shortness of breath at night or in early morning?	15.1	4.5	5.4
4) Attacks of shortness of breath	Did your child ever have attacks of shortness of breath or breathlessness?	24.1	16.5	11.7
5) Doctor's diagnosed asthma	For your child, has bronchial asthma ever been diagnosed by a doctor?	9.7	4.4	4.0
"allergic asthma" "eczema"	One of 1) to 5) and 9)	40.0	0.0	10.2
	Has your child ever had itchy skin reaction in the bends of the elbows and knees and on the cheeks (also called neurodermatitis or eczema)?	68.0	0.0	17.3
6) Conjunctivitis	Did your child frequently have inflamed or itching eyes last spring/summer without having a cold?	27.4	8.5	10.1
7) Rhinitis	Did your child have a running, sniffy, or itching nose last spring/summer without having a cold?	29.0	23.6	15.7
8) Doctor's diagnosed hay fever	For your child, has hay fever ever been diagnosed by a doctor?	18.6	5.1	6.5
9) Sensitization in SPT "Hay fever"	Wheal ≥ 2 mm in reaction to at least one of the seven aeroallergens	56.3	21.8	21.8
	One of 6) to 8) and 9) to pollens	27.1	0.0	6.9

in southwestern Germany (Freiburg, Kehl, and Lörrach/Weil). The questionnaires were returned by 1812 families (70%). Written consent was obtained for participation in the SPT, which took place from March to June in the same year. Of the 1812 children, 1470 participated in the SPT. The study was approved by the ethics committee of the University of Freiburg and also by local school authorities.

Combination of history gathered by questionnaire and sensitization in SPT

The clinical rationale concentrated on three atopic diseases: asthma (i.e. allergic asthma), hay fever, and eczema. Allergic asthma was defined by two conditions: individual symptoms or a doctor's diagnosis of asthma and a reaction (wheal ≥ 2 mm) to at least one of seven aeroallergens tested by SPT (Table 1). Hay fever was based on rhinitis/conjunctivitis in spring/summer or a prior diagnosis of hay fever combined with at least one positive SPT reaction to either grass, birch, or hazel pollen (Table 1). The SPT result was not used to define eczema (Table 1). The overlap between the atopic groups can be seen in Table 2. For the nonatopic group it was required that none of the indicators of Table 1 occur. The group with an unspecific combination of signs (Table 2) was classified according to three criteria: 1) diagnosis of wheezy bronchitis or bronchial asthma, or a history of asthma-related symptoms, both without sensitization in SPT; 2) hay fever or rhinitis/conjunctivitis in spring/summer, both, however without an SPT reaction to either grass, birch, or hazel pollen; 3) a positive SPT to one of the seven aeroallergens but without clinical signs. These three

Table 2. Frequencies of the different features in the atopic and nonatopic subpopulation and in the group with unspecific signs

Subpopulation		Signs of atopy		
Atopic (n=350)	Eczema (n=172) Hay fever (n=24) Asthma* (n=56)	Eczema+hay fever (n=14) Eczema+asthma* (n=27) Hay fever+asthma* (n=32)	Eczema+hay fever+asthma* (n=25)	
Nonatopic (n=553)				
Unspecific (n=473)	Asthmatic symptoms+no sensitization (n=238)	Rhinitis/conjunctivitis+no sensitization to pollen (n=80)	Doctor's diagnosis of asthma or hay fever+no sensitization (n=65)	Sensitization +no other (n=90)

* Asthma = "allergic asthma".

criteria define a subpopulation disjunctive to the other groups (Table 2).

Hypothetical risk factors for clinical atopy

The hypothetical risk factors were as follows:

- parental atopy (PA) – maternal or paternal history of eczema, hay fever, or asthma
- low gestational age (LGA) – child born at least 3 weeks preterm
- maternal history of cigarette smoking during pregnancy, the child's first year of life, and the year preceding the cross-sectional investigation
- duration of breast-feeding (in months)

- e) history of whooping cough in the child's first year of life
- f) prior and current exposure to pet animals at home
- g) educational level (highest level mother or father achieved at school)
- h) gender of the child.

SPT procedure

Seven purified and immunochemically characterized aeroallergens (ALK, Denmark) were applied to each child's forearm with an uncoated Phazet needle. Extracts from grass pollens (mixture of the six most common species), birch and hazel pollens, *Dermatophagoides pteronyssinus* and *D. farinae*, and cat and dog dander were used (concentration: 10 histamine equivalent in prick testing). As control solutions, histamine hydrochloride (10 mg/ml) and sodium chloride (9 g/l) were applied. The reactions were recorded after 15 min (3). The largest and the orthogonal diameters of a wheal were measured and the arithmetic mean was calculated. Diameters greater than or equal to 2 mm were classified as a positive reaction (7). A reaction to the negative control was subtracted from the allergen value beforehand. The tests were carried out by trained personnel under supervision of a physician.

Statistical analysis

Relative proportions of risk factors for the atopic groups are provided. The estimation of the relative risks is based on logistic regression (12, 16) and provides odds ratios (OR) and their 95% confidence intervals (95% CI). In the assessment we followed the backward procedure of Greenberg et al., which starts with a model including potential confounders, which here are the different levels of education (9). Reducing the model backwardly, we eliminated the confounding variables if no change in the ORs of the variables of interest occurred ($\leq 10\%$ in change). Analyses were performed with the statistical analysis system (SAS) (16).

Results

Ninety-four (6.4%) of the 1470 schoolchildren with SPT were excluded because of missing data for the questionnaire. Of the 1376 remaining children, 25.4% ($n = 350$) were part of at least one of the three atopic categories (Table 2). Eczema was reported in 17.3% of the population. Allergic asthma was found in 10.2% of the 1376 children. Hay fever occurred in 6.9%. The proportion of combined occurrences of the atopic diseases is shown in Table 2. Of 1376 children, 40.2% ($n = 553$) did not have any of these indicators and were classified as definite nonatopic.

The group of unspecific signs (Table 2) was made up by 473 children (34.4%).

To analyze risk factors, we used the group showing none of the indicators of atopy ($n = 553$) as a reference. The frequency of potential risk factors in the different groups is shown in Table 3. In each of the atopic groups the proportions of parental atopic diseases were higher than in the nonatopic group (Table 3). With the exception of paternal eczema, each parental disease was most prevalent in the group of children with the corresponding disease. In each of the atopic groups LGA, breast-feeding for 4 months or more, prior exposure to pet animals, and high educational level were found more frequently than in the nonatopic children. Maternal smoking in pregnancy was reported as 7.4–11.3% in the different atopic groups, as compared with 14.4% in the nonatopic group. The proportion of girls was lower in the hay fever and the asthma group (below 40%), as compared with a predominance in the eczema group (58%). The statistical importance of childhood risk factors (OR and 95% CI) was estimated by means of logistic regression under mutual control of all other predictors (Table 4). Parental atopic diseases, LGA, prior exposure to pets, and gender gained statistical significance in one or more models.

For hay fever and eczema, specific associations with the corresponding maternal as well as paternal history were obvious (e.g. maternal hay fever with hay fever of the offspring: OR 3.95; Table 4). For eczema, an association with paternal asthma and hay fever could also be seen. A significant relationship between parental and children's asthma could not be demonstrated. However, for this group, maternal hay fever and paternal eczema gained significance.

LGA was significant for allergic asthma as well as any atopic disease. Prior exposure to pets gained statistical importance for eczema and any atopy. Hay fever and allergic asthma were less frequent in girls than in boys.

Discussion

In the present study, atopic phenotypes were defined, taking the children's history and their current sensitization to aeroallergens into account. Since a retrospective ascertainment derived from questionnaires alone might be affected by information biases, it seemed preferable to combine the clinical history with an objective measurement of sensitization (19). Although sensitization to aeroallergens is not inevitable in eczema (10), 35.7% ($n = 85$) of these 238 children were reactive in the SPT. Several discrepancies between clinical history and sensitization were obvious. For example, children with asthmatic symptoms but without sensitization made up a relatively

Table 3. Risk factors for clinical atopy (percentages are based on numbers after "missings" have been deleted)

Factor	Manifestation					
	No disease n=553 %	Any atopic disease n=350 %	Eczema n=238 %	Hay fever n=95 %	Allergic asthma n=140 %	Unspecific signs n=473 %
Maternal atopy						
Hay fever	8.7	18.0	15.1	29.5	19.3	12.7
Eczema	6.3	14.3	16.4	9.5	13.6	8.9
Asthma	3.1	6.0	6.3	5.3	7.1	3.6
Paternal atopy						
Hay fever	8.9	17.7	17.2	20.0	14.3	9.1
Eczema	4.2	12.6	13.9	14.7	11.4	5.3
Asthma	2.0	6.3	6.7	7.4	6.4	4.9
Low gestational age (LGA)	6.4	10.7	8.1	9.5	15.1	6.0
Breast-feeding						
≥ 4 months	36.8	43.6	43.0	43.2	46.0	36.5
≥ 1 month < 4 months	36.4	34.9	36.6	30.5	29.9	37.0
< 1 month	26.8	21.5	20.4	26.3	24.1	26.5
Maternal smoking						
In pregnancy	14.4	11.2	11.3	9.9	7.4	18.0
First year of life	21.8	20.0	21.3	16.1	17.5	25.3
Previous year	26.7	25.0	26.8	18.3	21.3	34.5
Whooping cough in first year of life	4.1	6.3	5.8	6.6	5.3	7.7
Prior exposure to pets	26.0	32.0	33.2	30.5	33.6	28.5
Educational level						
Low	36.8	30.3	31.3	28.6	30.2	30.5
Medium	26.4	26.5	26.2	30.8	28.8	32.2
High	36.8	43.2	42.5	40.7	41.0	37.3
Gender (female)	52.4	49.7	58.0	34.7	38.6	50.7

Table 4. Odds ratios/95% confidence intervals of risk factors for clinical atopy under mutual adjustment. Outcome variables include any atopic disease (n=315), eczema (n=213), hay fever (n=87), allergic asthma (n=126), and unspecific signs (n=430), each in relationship to no atopy (n=513). Children with missing values for any of the predictors were excluded

Factor	Manifestation				
	Any atopic disease	Eczema	Hay fever	Allergic asthma	Unspecific signs
Maternal atopy					
Asthma	1.56/0.74–3.30	1.75/0.75–4.05	1.12/0.33–3.84	2.01/0.80–5.06	1.18/0.56–2.46
Hay fever	1.94/1.25–3.02	1.47/0.87–2.48	3.95/2.16–7.24	1.92/1.07–3.47	1.37/0.89–2.10
Eczema	2.04/1.23–3.38	2.63/1.53–4.52	1.05/0.45–2.46	1.79/0.91–3.54	1.37/0.83–2.27
Paternal atopy					
Asthma	2.25/1.02–4.99	2.39/1.01–5.64	1.90/0.56–6.41	2.18/0.77–6.19	2.46/1.16–5.23
Hay fever	1.88/1.21–2.91	1.91/1.17–3.13	2.08/1.06–4.09	1.14/0.59–2.18	0.80/0.50–1.28
Eczema	2.70/1.55–4.72	3.09/1.69–5.66	2.88/1.30–6.41	2.25/1.06–4.78	1.08/0.59–1.98
Low gestational age	1.74/1.02–2.97	1.29/0.67–2.49	1.41/0.59–3.36	2.82/1.48–5.37	1.05/0.61–1.82
Prior exposure to pets	1.38/1.00–1.91	1.58/1.10–2.27	1.35/0.78–2.33	1.33/0.84–2.08	1.05/0.78–1.41
Gender (female)	0.91/0.67–1.22	1.32/0.94–1.86	0.50/0.30–0.82	0.59/0.39–0.90	0.94/0.72–1.23

large group (Table 2; $n = 238$). Despite the clinical history, it did not seem to be justified to classify these children as atopics, since the origin of their complaints (e.g. infections) remains unclear. Never-

theless, in a few cases asthma at early school age can occur without allergic sensitization. By our definition, seasonal rhinitis (hay fever) required sensitization to pollen. A separate analysis of children with

perennial rhinitis without colds was added ($n = 144$). Of those children, 14.2% ($n = 17$) were sensitized to nonseasonal allergens, of whom seven had at least a unilateral parental history of atopy.

The prevalence of eczema (17.3%) in our sample of schoolchildren 6–8 years of age is comparable with previous findings in the UK (11–15%), Sweden (21.1%), and Germany (19.7%) (6, 14, 22). The lifetime prevalence of an “allergic asthma” (10.2%) is higher than that of “doctor’s diagnosed asthma” (4.0%). Although the doctor’s diagnosis is commonly used for epidemiologic purposes, it depends on the practitioner’s training and might be a relatively insensitive indicator of the disease (20). Indeed, it has been estimated that about 11% of children suffer episodes of wheezing in the UK (13).

Hay fever was found in 6.9% of the 1376 children (Table 1). In Sweden the prevalence of hay fever was 14.4% in 11-year-olds, whereas in Italy 7.4% of 1691 primary-school pupils were reported to be suffering from allergic rhinitis or conjunctivitis (6, 2). This discrepancy might indicate decreasing prevalences from northern to southern Europe. This assumption is supported by the European prevalences of childhood atopy in total. In Sweden as well as in Germany, about 30% of the whole sample were found to be atopic (6, 14). In contrast, the prevalence of atopy in Italy was found to be 14% (2). Taking the dependence on age and region into account, we found the prevalences of atopic manifestations to be at the expected level for central Europe.

Parental atopy plays a predominant role as a risk factor for atopy in the offspring. In each of the atopic groups the parental affections were more frequent than in the reference group (Table 3). The importance of the parents’ atopy for the child’s outcome in general is further indicated in the multivariate analysis (Table 4). For each relationship between a child’s disease and parental history, the ORs exceeded 1. In addition, each parental disease, with the exception of maternal asthma, was significant for “any atopic disease”. Specific associations between the parental and the child’s disease regarding the same entity gained significance for hay fever and eczema. We found the risk of parental atopy history to be similar to the results reported by others (range of ORs: 1.5–5.5 (2, 6)). To our knowledge, data about the association of specific diseases regarding parents and their offspring are available only for asthma (17). The association of parents’ and children’s asthma did not depend on parental atopy (17). It has been suggested that the asthmatic and atopic dispositions are inherited independently (18). Furthermore, a coincidence of atopic diseases within families might also result from the same environmental conditions, as is plausible, for example, in exposure to pollen, house-dust mites, and pet dan-

der. This could explain an occurrence of the same disease on the basis of a general atopic disposition.

LGA was significantly associated with clinical atopy in general (Table 4; OR 1.74). Only weak associations were found between LGA and hay fever as well as eczema (ORs 1.41 and 1.29, respectively). A significant relationship could be seen between LGA and allergic asthma (OR 2.8). The last-named finding is in accordance with other results which suggest that in neonates with low birth weight, lower respiratory symptoms at the age of 7 years might depend on the level of neonatal respiratory treatment (4).

An increased risk from prior exposure to pets for a history of eczema might reflect the importance of allergen exposure for this atopic disorder. A gender predominance of males is obvious in the groups with hay fever and allergic asthma (Table 3). For bronchial asthma and asthmatic symptoms, higher prevalences in boys than in girls have been reported from several studies (5, 11, 21). In two of these studies the male predominance diminished with increasing age, in part indicating a higher incidence in girls and a higher rate of remission in boys (5, 21). In accordance with this, similar prevalences but significantly different cumulative incidences among boys and girls (higher in boys) have been found in a large Swedish population aged 7–14 years (1).

Children with clinical atopy were more frequently categorized to “breast-feeding for 1 month or more” than to “breast-feeding less than 1 month” (Table 3). However, a similar relation was obvious for the group with no disease. Thus, breast-feeding was not of importance in the multivariate models and could be eliminated in each of them. The same applied to “high education level” as well as “maternal smoking in pregnancy” (Table 3).

On the basis of cross-sectional data for the analysis of risk factors, no doubt should exist in respect to the timing of cause and effect. Therefore, the evaluations are restricted to influences which do not change (i.e. genetic disposition) or might be referred to early life (i.e. immaturity at birth). The analyses could be performed only for a subpopulation ($n = 1376$) of the total sample. The frequency of relevant variables (educational status, smoking habits, parental atopy) did not differ substantially between the total ($n = 1812$) and the analyzed study population.

In conclusion, our data confirm the role of parental atopy as a general risk for atopy in the offspring. In addition, specific relationships are indicated by an association of the parents’ and the child’s disease for eczema and hay fever. Furthermore, LGA is shown to be a risk for asthma and for atopy in general. For boys, as compared with girls, an increased prevalence of hay fever and allergic asthma was found,

presumably caused by a higher inborn susceptibility or by an increased exposure or response to environmental agents among males.

Acknowledgments

We thank Mrs Monika Mortensen, Mrs Sabine Schraub, and Mrs Sabine Linser-Haar for their excellent assistance in the field study, in handling the questionnaires and conducting the SPT. The study was supported by the German Federal Ministry for Research and Technology (Grant no. 07015655).

References

1. ABERG N, ENGSTRÖM I, LINDBERG U. Allergic diseases in Swedish schoolchildren. *Acta Paediatr Scand* 1989; 78: 246–51.
2. ANGIONI AM, FANCIULLI G, CORCHIA C. Frequency of and risk factors for allergy in primary schoolchildren: results of a population survey. *Paediatr Perinat Epidemiol* 1989; 3: 248–55.
3. BELIN L, DREBORG S, EINARSSON R, et al. PhazetTM—a new type of skin prick test calibration and stability. *Allergy* 1985; 40 Suppl: 60–3.
4. CHAN KN, ELLIMAN A, BRYAN E, SILVERMAN M. Respiratory symptoms in children of low birth weight. *Arch Dis Child* 1989; 64: 1294–1304.
5. CLIFFORD RD, RADFORD M, HOWELL JB, HOLGATE ST. Prevalence of respiratory symptoms among 7- and 11-year-old schoolchildren and association with asthma. *Arch Dis Child* 1989; 64: 1118–25.
6. CRONER S, KJELLMAN NIM. Development of atopic disease in relation to family history and cord blood IgE levels. Eleven-year follow-up in 1654 children. *Pediatr Allergy Immunol* 1990; 1: 14–20.
7. DREBORG S. Skin tests used in type I allergy testing. 6. Skin tests used for epidemiological studies. *Allergy* 1989; 44 Suppl: 52–9.
8. FERRIS BJ Jr. Epidemiology standardization project. *Am Rev Respir Dis* 1978; 118: 36–47.
9. GREENBERG S, KLEINBAUM DG. Mathematical modelling strategies for the analysis of epidemiologic research. *Am Rev Public Health* 1985; 6: 223–45.
10. HAAHELA TMK. The prevalence of allergic conditions and immediate skin test reactions among Finnish adolescents. *Clin Allergy* 1979; 9: 53–60.
11. KAPLAN BA, MASCHIE-TAYLOR CG. Asthma and wheezy bronchitis in a British national sample. *J Asthma* 1987; 24: 289–96.
12. KLEINBAUM DG, KUPPER LL, MORGENTHAU H. *Epidemiologic research. Principle and quantitative methods*. New York: Van Nostrand, Reinhold, 1982.
13. LEE DA, WINSLOW NR, SPEIGHT AN, HEY EN. Prevalence and spectrum of asthma in childhood. *BMJ [Clin Res]* 1983; 286: 1256–8.
14. MUTIUS EV, DOLD S, WIJST M, et al. Münchener Asthma- und Allergiestudie. Prävalenzen atopischer und asthmatischer Erkrankungen im Kindesalter in Bayern. *Munch Med Wschr* 1991; 133: 675–9.
15. ROWNTREE S, COGSWELL JJ, PLATTS-MILLS TAE, MITCHELL EB. Development of IgE and IgG antibodies to food and inhalant allergens in children at risk of allergic disease. *Arch Dis Child* 1985; 60: 727–35.
16. SAS Institute, Inc. SAS Technical Report P-200. SAS/STAT Software: CALIS and LOGISTIC Procedures. Release 6.04. Cary, NC: SAS, 1990.
17. SHERMAN CB, TOSTESON TD, TAGER IB, SPEIZER FE, WEISS ST. Early childhood predictors of asthma. *Am J Epidemiol* 1990; 132: 83–95.
18. SIBBALD B, TURNER-WARWICK M. Factors influencing the prevalence of asthma among first degree relatives of extrinsic and intrinsic asthmatics. *Thorax* 1979; 34: 332–7.
19. SMITH JM. Epidemiology and natural history of asthma, allergic rhinitis, and atopic dermatitis (eczema). In: MIDDLETON E Jr, REED CE, et al., eds. *Allergy principles and practice*. St Louis, MO: Mosby, 1988: 893.
20. SPEIGHT ANP, LEE DA, HEY EN. Underdiagnosis and undertreatment of asthma in childhood. *BMJ* 1983; 286: 1253–6.
21. STRACHAN DP. Wheezing presenting in general practice. *Arch Dis Child* 1985; 60: 457–60.
22. TAYLOR B, WADSWORTH J, WADSWORTH M, PECKHAM C. Changes in the reported prevalence of childhood eczema since the 1939–45 war. *Lancet* 1984; 1255–7.

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.