

House-dust mite content in mattresses in relation to residential characteristics and symptoms in atopic and nonatopic children living in northern Norway

Dotterud LK, Korsgaard J, Falk ES. House-dust mite content in mattresses in relation to residential characteristics and symptoms in atopic and nonatopic children living in northern Norway. Allergy 1995; 50: 788–793. © Munksgaard 1995.

The occurrence of house-dust mites (HDMs) was investigated in the mattresses of 19 children previously found to be skin prick test (SPT) positive to HDM and in 19 nonatopic children derived from an extensive survey of 424 schoolchildren, all living in northern Norway. Domestic mites were counted and identified microscopically. Mite counts ranging from 10 to 1800 mites per gram mattress dust were found in 10 of the 19 HDM-sensitized children compared to none in the control group, corresponding to an odds ratio of more than 20. Of the 540 domestic mites found, 70 were identified by species. *Dermatophagoides pteronyssinus* (*Dpt*) was the only HDM species identified (64 mites), while five were storage mites and one was a *Tarsonemus* species. Positive radioallergosorbent tests (RAST) to *Dpt* were demonstrated in 9/10 children with and in 5/9 without mite infestation compared to none in the control group. Elevated IgE levels were also found more frequently in children with mite-infested mattresses than in those without. IgE levels were within normal levels in all 19 children in the control group. Latent atopy was found in four children, three with and one without mite infestation. There was no correlation between the concentration of mites and the degree of sensitization. Poor ventilation, increased humidity, and water leak(s) were associated with the presence of domestic mites in mattresses. As HDM growth is highly dependent on humidity and microhabitat, it should be possible to avoid HDM exposure and allergy in this region.

L. K. Dotterud¹, J. Korsgaard²,
E. S. Falk¹

¹ Department of Dermatology, University of Tromsø, Norway; ² Department of Chest Diseases, Silkeborg Municipal Hospital, Denmark

Key words: atopy; building characteristics; domestic mites; mattress infestation; schoolchildren.

Dr L. K. Dotterud
Department of Dermatology
University of Tromsø
Tromsø
Norway

Accepted for publication 1 February 1995

Although inherited constitution is one of the main background factors of atopic diseases (2, 7–9), environmental factors seem of great importance in atopic manifestations, not only of asthma and allergic rhinoconjunctivitis (AR) (24, 29), but also of atopic dermatitis (AD) (2). The increase in prevalence of allergic diseases in recent years cannot be due to changes in heredity (7–9) and must therefore be explained by a change in environmental factors (4, 22, 24–26, 29).

Recent studies have indicated a high frequency of allergic diseases among children in a rural community in northern Norway close to the Russian border, and a relationship with industrial pollution from Russian smelting plants is suspected (8). Epidemiologic studies suggest a relationship between risk of

sensitization and increased exposure to both indoor and outdoor airborne pollutants (4, 24, 27, 29).

House-dust mite (HDM) antigen is known to be a major inhalation antigen in asthma and AR (17, 27, 28), whereas its etiologic role in AD remains questionable (11, 26). AD patients have shown high exposure rates to HDM, and sensitization to HDM is frequent in atopic subjects (3, 6, 15). Moreover, patch testing with HDM antigen applied for 48 h to superficially abraded skin can elicit dermatitis lesions (6, 11, 23).

The geographic distribution of HDM varies because of differences in climate. Domestic mites are less common in areas with a dry inland climate than in the more humid coastal areas (18, 27, 29), and in this respect northern Norway has been regarded as

an area of low-grade HDM infestation. In recent decades, building techniques have changed everywhere, as well as in northern Norway, and well-insulated, energy-efficient houses have been constructed. In addition to accumulation of indoor pollutants, there is a risk of increased indoor air humidity, which promotes the growth of molds and HDM (5, 17, 18, 25–27, 29).

Therefore, the purpose of this study was to investigate the occurrence of domestic mites in homes of HDM-sensitized children and controls in an area of northern Norway situated above 70° latitude north, particularly in relation to clinical symptoms, and to estimate the strength of association between exposure and sensitization. Secondly, mite exposure was investigated in relation to building characteristics and indoor climate conditions.

Material and methods

Patient sampling

A total of 424 schoolchildren aged 7–12 years from nine schools in the community of Sør-Varanger, an area located at 70° latitude north, close to the Russian border, were investigated for allergic diseases, and 20 had a positive skin prick test to extract of HDM. One child with HDM sensitivity moved from Sør-Varanger before this study had been completed. The remaining 19 children, together with a control group of 19 nonatopic children from the same group of schoolchildren matched for age, sex, and residential area, who acted as a control group, were selected for the trial. The study design and clinical examinations have previously been described (8, 9). AD was defined according to the criteria of Hanifin & Rajka (14). A diagnosis of asthma and/or AR was established on the basis of the children's history in response to known or strongly suspected allergens (L.-K. Dotterud et al., personal communication). Children without a clinical history of atopic disorders but with positive skin prick test(s) to at least one allergen were defined as having latent atopy (13).

Skin prick tests

Skin prick tests had been performed on 420 of the 424 children during the clinical examinations (February 1992 to March 1993) using 11 commercially available allergen extracts, including *Dermatophagoides pteronyssinus* (*Der p*), dog, cat, *Cladosporium herbarum*, birch, timothy, mugwort, white flour, cow's milk, cod, and egg white, obtained from ALK (Allergologisk Laboratorium, Hørsholm, Denmark). Test procedures were performed according to Aas & Belin (1) and evaluated as previously described; i.e., positive when the reaction was 2+ or more, except

for cow's milk and white flour, for which a reaction of 3+ was required to be considered positive.

Serum IgE

The IgE concentrations in serum were determined using the Pharmacia CAP system, IgE FEIA (Pharmacia A/B, Uppsala, Sweden). The upper limit for normal IgE is in this study estimated to be 180 u/ml (9).

Radioallergosorbent test (RAST)

The Pharmacia CAP system (Pharmacia AB, Uppsala, Sweden) was used to quantify circulating IgE antibodies to *D. pteronyssinus* (*Der p*), *C. herbarum*, *Aspergillus fumigatus*, *Alternaria alternata*, *Penicillium notatum*, *Mucor racemosus*, birch, timothy, dog, cat, horse, mugwort, and some food allergens (pediatric panel = F × 5, Pharmacia CAP system, Pharmacia AB, Uppsala, Sweden). The RAST results were expressed in RAST classes 0–6, according to the manufacturer, with RAST class 2 or higher being considered a positive RAST result.

Dust collection

During the period January to March 1994, dust samples were collected in the homes of the children by vacuum cleaning half of the upward facing surface and vertical side of the child's mattress and the inside of the bed for 1.5 min. The parents had been instructed not to vacuum the mattresses for at least 1 week prior to sampling. The winter climate in this arctic region at 70° latitude north is cold with an average temperature in these 3 months of between –4.5°C and –13.5°C, and a medium relative humidity of 85–90%, corresponding to a mean absolute outdoor humidity of 1.6–2.8 g/kg (Asta Gutormsdottir, personal communication, Norwegian Meteorological Institute). Dust was sampled on Whatman qualitative filter paper (Whatman International Ltd, Maidstone, UK), cat. no. 1001070 with a diameter of 7.0 cm (38.5 cm²) (pore size retaining 97% of particles 0.5–1.0 µm in size, according to the manufacturer). The nozzle (ALK, Hørsholm, Denmark) was connected to the end of the suction tube of a household Nilfisk GM 90 vacuum cleaner (Fisker and Nielsen A/S, Copenhagen, Denmark). The same equipment was used for all samples. The filter paper was changed between each sampling, and the equipment was disassembled and cleaned. Each dust sample was sealed in a plastic bag and stored at –18°C until preparation. Domestic mites were counted, identified microscopically, and recorded as number of mites per gram dust, as previously described (19).

Temperature and relative humidity

The calibrated termohygrometer, Therm 1250-1 no. 887944 (Ahlborn Meß- und Regelungstechnik, Holzkirchen, Germany), and standard swing psychrometer (S. Brannan and Son Ltd, Cleator Moor, Cumbria, UK) were used to measure the indoor temperature and relative humidity, which were converted to absolute indoor humidity (AIH) with a Mollier diagram. The outdoor temperature and relative humidity were measured at Kirkenes Airport (local station, Norwegian Meteorological Institute).

Questionnaires

Questionnaires were delivered to the families on the day of the investigation, requesting information on items such as type of house and its location, number of family members, pets, family smoking habits, carpeted area, insulation, renovation, cleaning habits, and the children's home environment before the age of 2 years. Special attention was given to the ventilation system and any indication of dampness such as damp patches, condensation on windows, and previous water leak(s).

Statistics

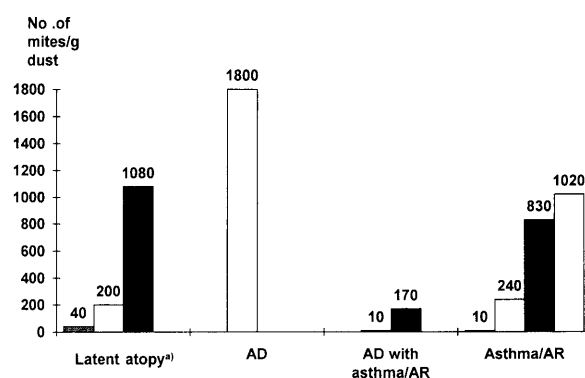
The data were manually computed. The differences between the number of individuals in the various groups were assessed with the chi-square test and the Fisher exact test. The odds ratio (OR) and a confidence interval (CI) of 95% were also indicated, and if the 95% CI did not include 1.0, the results were regarded as significant at $P < 0.05$. The OR was defined in terms of the odds of disease/sensitivity in exposed children relative to the odds of disease/sensitivity in the unexposed. The study was approved by the ethics committee of Tromsø University.

Results

HDM were demonstrated in the mattresses of 10 children (seven boys and three girls) SPT positive to *Der p* compared to none in the control group. This corresponds to an OR of more than 20 (Table 1). A total of 540 domestic mites were found, of which 70 were identified by species. The only HDM species identified was *D. pteronyssinus* (64 mites), while four mites were identified as *Lepidoglyphus destructor*, one as *Acarus siro*, and one as *Tarsonemus* species. The highest and lowest mite counts per gram were 1800 and 10, respectively (Fig. 1). Mite exposure indicated a high degree of sensitization since 9/10 children with mite-infested mattresses had positive RAST to *Der p*, compared to 5/9 without mite infestation (Table 2). Moreover, 6/10 children with

Table 1. Occurrence of house-dust mites (HDM) in mattresses of 19 HDM-sensitized and 19 nonatopic schoolchildren (control group). Odds ratio (OR) and confidence interval (CI) of 95% are also indicated

		HDM sensitization		OR	95% CI
		+	-		
Mite infestation	+	10	0	≥ 20	2.0-487.7
	-	9	19		



^{a)}All three children with latent atopy were both SPT and RAST positive to house-dust mite. No mite infestation was seen in the control group.

Fig. 1. Number of house-dust mites in mattresses in relation to atopic diseases.

Table 2. Occurrence of domestic mites in mattresses in relation to specific IgE antibodies to house-dust mite (HDM), serum IgE, and atopy in 19 HDM-sensitized children from northern Norway

	(n)	RAST		Serum IgE		Atopy	Latent atopy
		Positive	Negative	Elevated	Normal		
Mite-infested mattresses	10	9	1	6	4	7	3
Mattresses without mite infestation	9	5	4	3	6	8	1

mite infestation had elevated serum IgE compared to 3/9 among children without mite infestation (Table 2). None of the children in the control group had either positive RAST to *Der p* or elevated serum IgE.

There was a good correlation between the strength of SPT reactions to *Der p* and RAST classes; i.e., strong positive SPTs ($\geq 3+$) paralleled high RAST classes (≥ 3). Furthermore, both strong SPT ($\geq 3+$) and RAST classes (≥ 3) were associated with domestic mite infestation in mattresses ($P < 0.05$). Atopic symptoms were reported in 15 children, seven with mite-infested and eight without mite-infested mattresses, whereas the remaining four children had latent atopy (Table 2). Of the children with mite-infested mattresses, four had symptoms of asthma

Table 3. Relationship between exposure to domestic mites and atopic symptoms in 19 house-dust mite sensitized and 19 nonatopic children (control group) from northern Norway. Odds ratio (OR) and confidence interval (CI) of 95% are also indicated

	Atopic symptoms		OR	95% CI
	+	-		
Mite exposure				
+	7	3 ^a	5.8	1.0-39.0
-	8	20 ^b		

^a Latent atopy.

^b Latent atopy (1) plus control group (19).

and/or AR, two of asthma and/or AR combined with AD, and one of AD only, whereas three had no symptoms at all (latent atopy) (Fig. 1). Of those without mite-infested mattresses, one had asthma and AR, four asthma and/or AR combined with AD, and three AD only, whereas one was without symptoms (latent atopy). In relating mite exposure to atopic symptoms, an OR of 5.8 was found (Table 3). In Table 4, the relation between mite infestation and factors which favor mite existence is indicated. Homes with structural defects, poor ventilation, and dampness/increased indoor humidity had a higher occurrence of aerospores and mite infestation. Moreover, mite infestation was found more frequently in the homes of children with eye irritation and respiratory problems. No differences in mite infestation of mattress dust were found between bedrooms with carpeted floors and bedrooms with smooth floors, or between new and old houses. Furthermore, no differences in mite infestation were found between children with first-floor (7/26 = 27%) and second-floor (3/11 = 27%) bedrooms. Six of the

nine HDM-sensitized children without mite-infested mattresses had moved to a different home before the age of 2 years, but only one of these six was born and had spent the first 2 years of life in southern Norway. All but one of the 38 children investigated had poly-ether foam mattresses. In only two of the 19 families with HDM-sensitized children had the parents tried some allergen-avoidance measures, such as a change of mattress and/or improved cleaning.

Discussion

The present results demonstrate a very strong relationship between sensitization and the occurrence of HDM in mattress dust with an OR of more than 20. As compared with previous sensitization studies, our sensitization rates are in accordance with those of Wood et al. (30) but are considerably higher than those found by several other studies (21, 28, 29). In addition, in our study group, sensitization occurred in children with very low mite counts (10-40 mites/g) as opposed to previous suggestions of at least 100 mites per gram (17, 24, 27). The low mite counts found in the children may be due to seasonal influence; i.e., our dust sampling was carried out during the coldest part of the year (25-27, 29). It appears likely that live mites survive within mattresses to produce allergen that comes to the surface largely in the form of fecal pellets or secretions (16, 25). Thus, sensitization without identifiable mites on the surface of the mattresses could have occurred. It is well known that mites are dried out in winter; however, a period of higher humidity during the summer is too short to permit their reestablishment (18, 25, 29). A further explanation of the disparity between sensitization and exposure to live mites may be that some of the HDM-sensitized children without mite-infested mattresses had moved to different homes in early childhood. On the other hand, it is suggested that daily low exposure to mites may elicit sensitization and possibly later atopic symptoms (24). No mite infestation was found in mattresses of children in the control group. This might be due to the generally low mite counts found in this arctic region with subarctic climate. However, if children in the control group with a genetic predisposition to mite allergen had been exposed, sensitization could have occurred.

As expected, *Dpt* was the mite most frequently found in house dust, representing 99% of all mites. This has previously been shown by others (15, 26, 29) and also that *Dermatophagoides* species cross-react (10, 26, 27) and that cross-antigenicity between different species of dust mites occurs (12, 26). Until recently, it has been suggested that HDM are of no importance in sensitization in arctic regions, but both Wickman (29) and R. Bolle (personal communica-

Table 4. Mite infestation in relation to residential characteristics in 38 schoolchildren in community of Sør-Varanger

	Children with positive skin prick tests to <i>Der p</i>		Control group (skin prick test negative to <i>Der p</i>)
	Mite-infested mattresses <i>n</i> =10	Mattresses without mite infestation <i>n</i> =9	Mattresses without mite infestation <i>n</i> =19
AIH ≥ 7.0 (g/kg)	7	2	8
Aerspores ≥ 42 (cfu/m ³) ^c	4	3	6
No balanced ventilation system	10	6	17
Water leakage(s) and/or damp patches	7 ^{ab}	1 ^a	2 ^b
Subjective symptoms ^d	7 ^b	3	3 ^b

^a $P < 0.05$.

^b $P < 0.01$.

^c Mean colony-forming units (cfu) of aerospores per m³ in bedrooms=42.

^d Symptoms of dry indoor air, eye, and airways irritation.

AIH=absolute indoor humidity in g/kg.

tion) have shown that domestic mites occur and may be a risk factor for sensitization to HDM. This is also confirmed by the correlation between positive RASTs and skin prick tests, IgE, and mite infestation in the present investigation.

There was, in fact, a clear correlation between mite exposure and atopic symptoms in the children; i.e., no one in the control group was exposed to mites or had ever had atopic symptoms, whereas 7/10 children exposed to mites had atopic symptoms (OR = 5.8). This is in accordance with previous studies (17, 25, 28). On the other hand, four children had no symptoms of atopy despite mite exposure and/or positive RASTs and skin prick tests to *Der p*, but these children may still be at risk of developing symptoms later.

Three children with AD had mite-infested mattresses and two of them had high mite counts and complained of pruritus and morning exacerbation of dermatitis, in accordance with other reports (3, 11, 15). It is suggested that mite allergens may penetrate eczematous and scratched skin (6, 11, 23). Six of the nine HDM-sensitized children without mite-infested mattresses had moved to different homes, but only one had spent the first 2 years of life in southern Norway.

Exposure to environmental risk factors in early childhood is considered to be of importance for the development of atopic diseases (29). The children in our study were exposed to additional risk factors such as high occurrence of smoking, furred pets, and outdoor air pollution, factors which may explain why almost 50% of them had or had had symptoms indicating atopy or atopy-like disease (8, 9). Mucous irritation in the upper respiratory tract usually gives the impression of dry air; investigations, however, have shown that there is no relationship between this symptom and the relative humidity measured. In fact, electrically heated homes designed for maximum energy efficiency are especially airtight, and relative humidity in these homes can reach levels in excess of 75% during even subzero outdoor temperatures (5).

It is possible that modern building techniques, i.e., efficient insulation applied in cold climates in order to save energy, may cause increased humidity and replication of mites and molds even in a subarctic area (29). Increased humidity and/or water leak(s) and bad ventilation systems increase the risk of mite infestation and not only of sensitization to HDM, but also of atopic sensitization per se. In our study, in fact, all HDM-sensitized children were found in homes with elevated AIH and poor ventilation, whereas all houses with a balanced mechanical ventilation system were free of mite infestation and dampness, and this is in line with a previous study (20). Surprisingly, we could not confirm previous

findings of higher mite counts on the first than on the second floor (29).

In a geographic region such as northern Norway with an outdoor climate unsuitable for mite growth, it should be possible to prevent exposure and sensitization to domestic mites. It is important to provide a dry environment, e.g., by improving ventilation and thereby reducing AIH, or by leaving mattresses outside in the arctic cold overnight and then vacuuming and beating them.

References

1. AAS K, BELIN L. Standardization of diagnostic works in allergy. *Acta Allergol* 1972; 27: 439–68.
2. ATHERTON DJ. Allergy and atopic eczema. *Clin Exp Dermatol* 1981; 6: 317–25.
3. BECK HI, KORSGAARD J. Atopic dermatitis and house-dust mites. *Br J Dermatol* 1989; 120: 245–51.
4. BRABÄCK L, KÄLVESTEN L. Urban living as a risk factor for atopic sensitization in Swedish schoolchildren. *Pediatr Allergy Immunol* 1991; 2: 14–19.
5. BURGE H. Bioaerosols: prevalence and health effects in the indoor environment. *J Allergy Clin Immunol* 1990; 86: 687–701.
6. CARSWELL F, THOMPSON S. Does natural sensitisation in eczema occur through the skin? *Lancet* 1986; *i*: 13–15.
7. COOKSON WOCM, YOUNG RP, SANDFORD AJ, et al. Maternal inheritance of atopic IgE responsiveness on chromosome 11q. *Lancet* 1992; 340: 381–4.
8. DOTTERUD LK, KVAMMEN B, BOLLE R, FALK ES. A survey of atopic diseases among schoolchildren in Sør-Varanger Community. Possible effects of subarctic climate and industrial pollution from Russia. *Acta Derm Venereol (Stockh)* 1994; 74: 124–8.
9. DOTTERUD LK, KVAMMEN B, LUND E, FALK ES. Prevalence and some clinical aspects of atopic dermatitis in the community of Sør-Varanger. *Acta Derm Venereol (Stockh)* 1995; 75: 50–3.
10. FALK ES, BOLLE R. IgE antibodies to house dust mite in patients with scabies. *Br J Dermatol* 1980; 102: 283–8.
11. GONDO A, SEAKI N, TOKUDA Y. Challenge reactions in atopic dermatitis after percutaneous entry of mite antigen. *Br J Dermatol* 1986; 115: 485–93.
12. GRIFFIN P, FORD AW, ALTERMAN L, et al. Allergenic and antigenic relationship between three species of storage mites and the house-dust mite, *Dermatophagoides pteronyssinus*. *J Allergy Clin Immunol* 1989; 84: 108–17.
13. HAAHTELA T. Skin tests used for epidemiologic studies. In: DREBORG S, FREW A, eds. *Allergen standardization and skin tests*. *Allergy* 1993; 48 (Suppl. 14): 76–80.
14. HANIFIN JH, RAJKA G. Diagnostic features of atopic dermatitis. *Acta Derm Venereol (Stockh)* 1980; Suppl. 92: 44–7.
15. HARVING H, KORSGAARD J, DAHL R, BECK HI, BJERRING P. House-dust mites and atopic dermatitis. A case-control study on the significance of house-dust mites as etiologic allergens in atopic dermatitis. *Ann Allergy* 1990; 65: 25–31.
16. HEWITT M, BARROW GI, MILLER DC, TURK F, TURK S. Mites in the personal environment and their role in skin disorders. *Br J Dermatol* 1973; 89: 401–9.
17. KORSGAARD J. Mite asthma and residence: a case-control study on the impact of exposure to house-dust mites in dwellings. *Am Rev Respir Dis* 1983; 128: 231–5.
18. KORSGAARD J. House-dust mites and absolute indoor air humidity. *Allergy* 1983; 38: 85–92.

19. KORSGAARD J, HALLAS TE. Tarsonemid mites in Danish house-dust. *Allergy* 1979; 34: 225-32.
20. KORSGAARD J. Mechanical ventilation and house-dust mites: a controlled investigation. In: *Dust mite allergens and asthma: report of the Second International Workshop*, Minster Lovell, Oxfordshire, England, 1991: 87-9.
21. LAU S, FALKENHORST G, WEBER A, et al. High mite-allergen exposure increases the risk of sensitization in atopic children and young adults. *J Allergy Clin Immunol* 1989; 84: 718-25.
22. LØWENSTEIN H, GRAVESEN S, LARSEN L, LIND P, SCHWARTZ B. Indoor allergens. *J Allergy Clin Immunol* 1986; 78: 1035-9.
23. MITCHELL EB, CROW J, CHAMPAN MD, JOUHAL SS, POPE FM, PLATTS-MILLS TAE. Basophils in allergen-induced patch test sites in atopic dermatitis. *Lancet* 1982; *i*: 127-30.
24. PLATTS-MILLS TAE, WARD GW Jr, SPORIK R, GELBER LE, CHAMPAN MD, HEYMANN PW. Epidemiology of the relationship between exposure to indoor allergens and asthma. *Int Arch Allergy Appl Immunol* 1991; 94: 339-45.
25. PLATTS-MILLS TAE, HAYDEN ML, CHAPMAN MD, WILKINS SR. Seasonal variation in dust mite and grass-pollen allergens in dust from the houses of patients with asthma. *J Allergy Clin Immunol* 1987; 79: 781-91.
26. PLATTS-MILLS TAE, THOMAS WR, AALBERSE RC, VERVLOET D, CHAPMAN MD. Dust mite allergens and asthma: summary report. In: *Dust mite allergens and asthma: report of the Second International Workshop*, Minster Lovell, Oxfordshire, England, 1991.
27. PLATTS-MILLS TAE, DE WECK AL. Dust mite allergens and asthma - a worldwide problem. *J Allergy Clin Immunol* 1989; 83: 416-27.
28. SPORIK R, HOLGATE ST, PLATTS-MILLS TAE, COGSWELL JJ. Exposure to house-dust mite allergen (*Der p 1*) and the development of asthma in childhood. *N Engl J Med* 1990; 323: 502-7.
29. WICKMAN M. Residential characteristics and allergic sensitization in children especially to mites. Thesis, Karolinska Hospital, Stockholm, Sweden, 1993.
30. WOOD RA, EGGLESTON PA, MUDD KE, ADKINSON NF. Indoor allergen levels as a risk factor for allergic sensitization. *J Allergy Clin Immunol* 1989; 83: 199.