

Biologic pollution in infant bedding in New Zealand: High allergen exposure during a vulnerable period

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Background: High exposure to house dust mite and cat allergens in early life predisposes to allergic sensitization and to the development and persistence of asthma. Prevalence and severity of asthma are high in New Zealand.

Objective: The objective of this study was to determine the concentrations of Der p 1 and Fel d 1 in infant bedding in Wellington, New Zealand.

Methods: Infants were visited at home at a mean age of 11 weeks and again at 15 months. The concentration ($\mu\text{g/g}$ fine dust) and content ($\mu\text{g/m}^2$) of Der p 1 (154 infants) and Fel d 1 (75 infants) were measured in dust samples taken from each infant's bed.

Results: At 11 weeks, geometric mean (95% confidence intervals) Der p 1 levels were 18.3 $\mu\text{g/g}$ (13.8 to 24.1) and 3.51 $\mu\text{g/m}^2$ (2.4 to 5.2). By 15 months, Der p 1 had risen significantly to 44.0 $\mu\text{g/g}$ (35.0 to 55.3) and 49.0 $\mu\text{g/m}^2$ (36.0 to 66.8). Bedding that included a sheepskin was used by a third of the infants and contained higher concentrations ($\mu\text{g/g}$) and content ($\mu\text{g/m}^2$) of Der p 1 than beds without sheepskins. Cat ownership was the major determinant of Fel d 1 levels, with 48% of infants living with cats. At the first visit, the mean concentration of Fel d 1 in bedding was 44.6 $\mu\text{g/g}$ (23.5 to 84.9) for houses with cats and 3.0 $\mu\text{g/g}$ (2.1 to 4.3) for those without cats, remaining essentially unchanged at the second visit. When expressed as micrograms per square meter, there was a significant increase between visits, from 8.1 (3.9 to 16.6) to 39.6 (19.9 to 78.5) in the cat-inclusive households.

Conclusions: Extremely high levels of house dust mite allergen have been found in these infants' environments, which, together

with the high levels of cat allergen in almost half who kept cats, are likely to be a major determinant of asthma prevalence and severity in New Zealand. (*J Allergy Clin Immunol* 1998;102:765-70.)

Key words: Der p 1, Fel d 1, house dust mite, cat, allergens, bedding, infants, New Zealand

An increasing body of evidence suggests that the human infant and possibly the fetus are at risk from exposure to high concentrations of environmental allergens, with a heightened likelihood of the development of allergen-specific IgE.^{1,2} Relevant work in animal models has demonstrated that the normal development of immunologic tolerance or nonresponsiveness that follows inhalation of antigen is disturbed in the neonatal period; tolerance does not develop, and the normally transient production of specific IgE persists.^{3,4}

Exposure to concentrations of the major house dust mite (HDM) allergen Der p 1 of more than 2 $\mu\text{g/g}$ of dust has been associated with an increased prevalence of specific sensitization to HDM by the age of 5 years.^{1,5,6} For Fel d 1, 8 $\mu\text{g/g}$ was initially proposed as a threshold level.^{7,8} However, recent data have suggested that exposures as low as 0.5 to 1.0 $\mu\text{g/g}$ may result in sensitization⁹ and that the concept of fixed threshold levels for sensitization may be inappropriate.¹⁰ Increasing frequency of sensitization has been associated with increasing exposure to HDM and cat allergens,¹¹ whereas continuing exposure to high allergen concentrations has resulted in the persistence of the IgE response.¹²

Studies from several countries, including New Zealand, have determined that the development of specific IgE sensitization to common perennial allergens, including HDM, cat, or *Aspergillus fumigatus*, confers a substantially increased risk of subsequent development of asthma.¹³⁻¹⁵ An almost 5-fold increased risk of development of asthma by the age of 11 years was identified in English infants exposed to Der p 1 levels of more than 10 $\mu\text{g/g}$ in the first year of life.¹⁶

Sensitization to HDM and exposure to Der p 1 concentrations of more than 10 $\mu\text{g/g}$ have also been associated both in adults^{7,8} and in children,¹⁷ with an increased risk of attacks of severe asthma. In asthmatic adults, sig-

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Abbreviation used

HDM: house dust mite

nificant correlations between bed Der p 1 and the severity of asthma and airway hyperresponsiveness have been demonstrated in both longitudinal¹⁸ and cross-sectional studies.^{19,20}

Studies in New Zealand have consistently demonstrated a high prevalence of asthma, recently emphasized in large international studies of asthma prevalence in children²¹ and in adults.²² We have recently confirmed high levels of Der p 1 in the homes and bedding of 8- to 10-year-old children²³ in Wellington. The geometric mean bedding concentration was 46.6 $\mu\text{g/g}$, whereas those on bedroom and living room floors were 26.4 $\mu\text{g/g}$ and 25.5 $\mu\text{g/g}$, respectively. In this study, we measured the levels of Der p 1 and Fel d 1 in the bedding of a group of New Zealand infants.

METHODS**Infant study group**

Over a 12-month period from November 1993 to November 1994, all Wellington-based mothers of healthy infants born at Wellington Women's Hospital were approached on the postnatal ward during a 3-week period each 3 months, thereby ensuring that sampling took place during each season.

Mothers and infants were visited in their homes, and dust samples were obtained from the bedding, bedroom, and living room floors of 154 infants at age 5 and 16 weeks (mean 11 weeks) and again at age 12 to 19 months (mean 15 months). The caregiver provided questionnaire details regarding the house, the number of occupants, and the infant's bed and bedding.

Dust sampling

Bedding dust samples were collected with a 1-kW (Hitachi) vacuum cleaner into a nylon sock attachment inserted into the head of the cleaner²³ for 2 minutes over an area 0.25 m² (0.75 $\times$ 0.33 m). The sample was collected from whatever article lay directly under the infant in the bed, other than the undersheet. Floor dust samples were collected by vacuum sampling 1 m² for 1 minute on carpeted floor and 2 minutes on uncarpeted floor. The dust was sieved, and the resulting fine dust was weighed, extracted (100 mg in PBS for 30 minutes at room temperature), and analyzed for Der p 1 by ELISA with well-established 2-site mAb methodology.²⁴ The interassay coefficient of variation was 10%. The laboratory is part of an international quality control program for Der p 1 analysis.²⁵ Results are presented as both Der p 1 concentration ($\mu\text{g/g}$) and content ($\mu\text{g/m}^2$). Fel d 1 analysis was carried out by ELISA²⁶ on the samples from 75 of the infants' beds and floors, randomly selected from the whole sample of 154. The interassay coefficient of variation for the Fel d 1 assay was consistently less than 10%.

Data analysis

As allergen concentrations were found to be log-normally distributed, geometric mean levels were calculated, together with 95% confidence intervals, and analysis used log-transformed data. Comparisons of allergen levels between visits used

a paired *t* test; those between bedding with and without sheepskin used a simple 2-tailed *t* test. A *P* value less than .05 was considered significant. The Central Regional Ethics Committee approved this study.

RESULTS

All infants (49% female) lived with their birth mother. Ninety-two (59.7%) of the infants were the firstborn children in the family, 34 (22.1%) had 1 sibling, and 28 (18.2%) had 2 or more siblings. At the first visit (mean age 11 weeks), 78 (50.6%) slept in a cot (crib); 70 (45.5%) in a bassinet, cradle, or basket; and 2 (1.3%) in the parental bed. At the second visit, 129 (84.3%) were in a cot, 13 (8.5%) in their own bed, and 10 (6.5%) in the parental bed. The majority of beds included an underblanket, most commonly woollen. Only 16% (11 weeks) or 23% (15 months) did not include sheepskin and/or woollen underbedding.

Der p 1 levels

At the first visit, geometric mean (95% confidence intervals) Der p 1 concentration in bedding was 18.3 $\mu\text{g/g}$ (13.9 to 24.1), and the content was 3.5 $\mu\text{g/m}^2$ (2.4 to 5.2). Levels had risen significantly to 44.0 $\mu\text{g/g}$ (35.0 to 55.3) and 49.0 $\mu\text{g/m}^2$ (36.0 to 66.8) at the second sampling (Fig 1, A and B).

To determine whether increased allergen content resulted from higher allergen concentration or from higher dust weight, these two measures were analyzed for the beds with allergen contents of less than 2, 2 to less than 10, and 10 or more $\mu\text{g/m}^2$. At the first visit, the Der p 1 concentrations were 5.1, 21.8, and 36.6 $\mu\text{g/g}$, respectively, and the dust weights were 0.018, 0.05, and 0.137 g (*n* = 58, 45, and 51, respectively). Equivalent figures at the second visit, with houses divided according to allergen levels at that visit, were 1.3, 16.5, and 64.8 $\mu\text{g/g}$ and 0.081, 0.074, and 0.377 g (*n* = 8, 21, and 125, respectively). Both higher Der p 1 concentrations in dust and higher dust weights therefore contributed to higher Der p 1 content.

At the first visit, 116 (75%) of the 154 bedding samples contained more than 10 $\mu\text{g/g}$ Der p 1, with 148 (96%) containing more than 2 $\mu\text{g/g}$. At this second visit, 142 (92%) bedding samples contained more than 10 $\mu\text{g/g}$ Der p 1, with 149 (97%) more than 2 $\mu\text{g/g}$. No significant differences in Der p 1 levels in different types of bed were detected at either visit.

The child's bedroom floor showed mean Der p 1 concentrations of 21.4 $\mu\text{g/g}$ (17.3 to 26.3) and 26.4 $\mu\text{g/g}$ (20.9 to 33.4) at the first and second visits, with Der p 1 contents of 15.8 $\mu\text{g/m}^2$ (12.1 to 20.7) and 18.5 $\mu\text{g/m}^2$ (14.2 to 24.1), respectively. The living room concentrations were 18.9 $\mu\text{g/g}$ (15.4 to 23.2) and 30.5 $\mu\text{g/g}$ (24.2 to 38.3), and contents were 16.0 $\mu\text{g/m}^2$ (12.3 to 20.9) and 30.9 $\mu\text{g/m}^2$ (22.9 to 41.6) for first and second visits, respectively. The increases seen in the living room levels between visits were significant (*P* < .05).

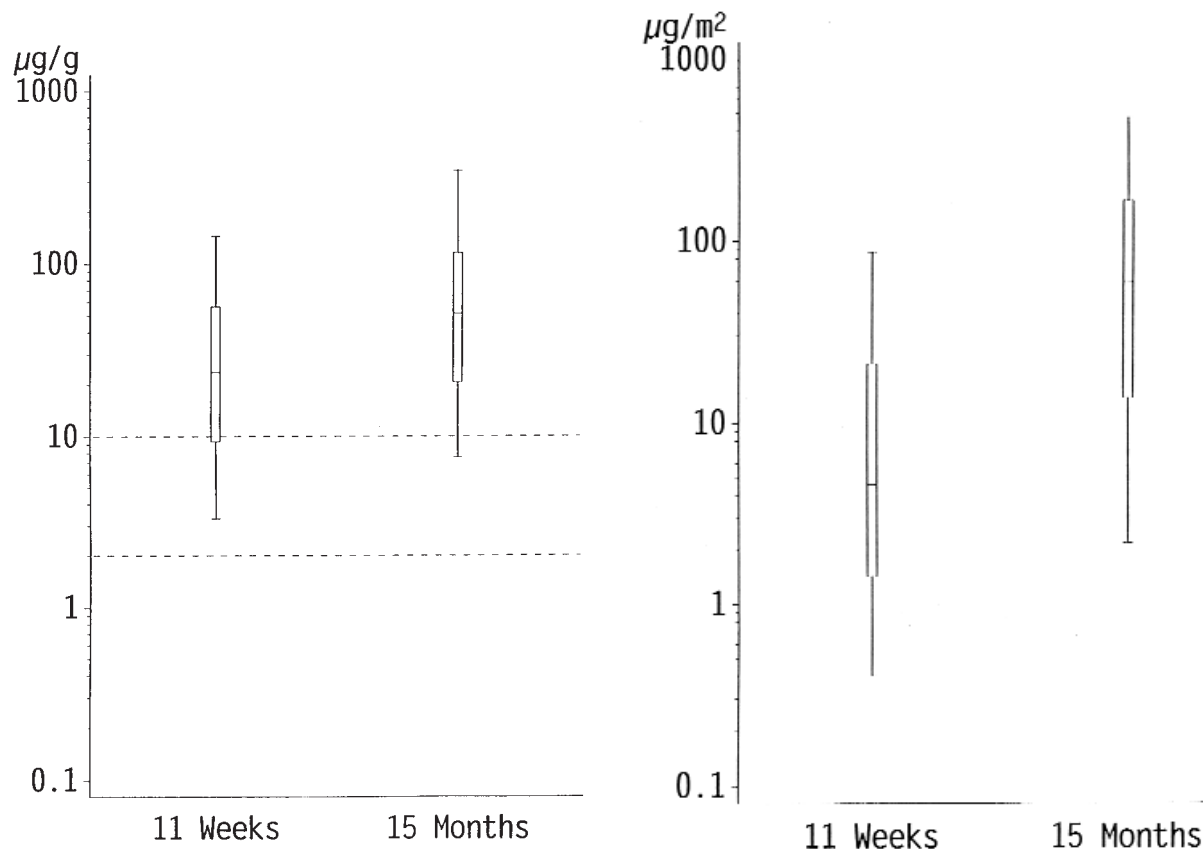


FIG 1. Der p 1 concentration ($\mu\text{g/g}$) (**A**) and content ($\mu\text{g/m}^2$) (**B**) are shown as box-and-whisker plots with upper and lower margins of box being 75th and 25th percentiles of data and upper and lower extents of whiskers being 95th and 5th percentiles, respectively. Median value is shown as cross-bar within box. **A** also shows suggested level of risk for sensitization ($2 \mu\text{g/g}$) and for symptom exacerbation ($10 \mu\text{g/g}$).

Fel d 1

Thirty-six (48%) of the 75 infants in this study lived in homes with 1 or more cats at the first visit, a rate that changed minimally between the 2 visits to 35 (47%) of 75. Other types of pets were much less common among this urban population, with 14% of children living in a home with a dog and only 1% in a home with birds. No association of pet ownership with socioeconomic status or ethnicity was detected in this study.

Fel d 1 levels for the 2 visits are presented in Table I. There were major differences between homes with and those without cats, but differences in mean allergen levels in milligrams per gram between the 2 visits were not statistically significant. However, for Der p 1, lower bedding dust weights at the first visit resulted in lower Fel d 1 $\mu\text{g/m}^2$ at the first visit ($P < .05$).

For those with cats, the mean Fel d 1 level on the living room floor was higher than those on the child's bedroom floor (paired t test: visit 1, $P < .01$; visit 2, $P < .05$) and in the child's bedding (paired t test: visit 1, $P < .01$; visit 2, $P < .001$). Although bedroom floors

had higher levels of Fel d 1 than in the bedding, this difference did not reach significance ($P = .06$).

At the first visit, 20 (56%) of the 36 households with cats permitted the cat to enter the infant's room, whereas at the second visit, 24 (69%) of 35 did. At both visits, Fel d 1 levels were lower in those bedrooms from which cats were excluded, although these differences failed to reach significance. For bedding at visit 1, cat excluded was $29.1 \mu\text{g/g}$ (10.3 to 82.6), and cat permitted was $62.7 \mu\text{g/g}$ (26.6 to 147.8); at visit 2, cat excluded was $24.8 \mu\text{g/g}$ (6.8 to 62.8), and cat permitted was $46.8 \mu\text{g/g}$ (20.9 to 91.1).

In homes without cats there were no significant differences between Fel d 1 levels at different sites.

Sheepskin use in bedding

The use of sheepskins in the bed was common (33% at the first visit, 38% at the second). Bedding with sheepskins contained higher concentrations of Der p 1 than beds without, the difference reaching significance at the second visit. Der p 1 content was also significantly higher in beds with sheepskins compared with those without

TABLE I. Fel d 1 levels in infant environment: Geometric means (95% confidence intervals)

	Visit 1 (11 weeks)			Visit 2 (15 months)	
	Fel d 1			Fel d 1	
	µg/g	µg/m ²		µg/g	µg/m ²
Bedding					
Total n = 75	11.0 (6.9-17.6)	2.5 (1.5-4.3)	Total n = 75	10.2 (6.7-15.6)	10.3 (6.2-16.9)
Cat n = 36	44.6 (23.5-84.9)	8.1 (3.9-16.6)	Cat n = 35	38.6 (21.6-68.2)	39.6 (19.9-78.5)
No cat n = 39	3.0 (2.1-4.3)	0.9 (0.5-1.5)	No cat n = 40	3.2 (2.3-4.5)	3.2 (1.9-5.2)
Bedroom floor					
Total n = 75	14.4 (8.8-23.6)	10.9 (6.5-18.4)	Total n = 75	11.0 (6.8-17.7)	8.2 (5.1-13.3)
Cat n = 36	70.7 (42.3-118)	57.2 (31.5-103.8)	Cat n = 35	54.0 (30.5-95.4)	36.6 (19.9-67.2)
No cat n = 39	3.3 (2.1-5.3)	2.4 (1.5-3.8)	No cat n = 40	2.7 (1.8-4.1)	2.2 (1.5-3.5)
Living room floor					
Total n = 75	18.4 (10.4-32.4)	14.9 (8.2-27.3)	Total n = 75	13.4 (8.0-22.7)	13.5 (7.9-23.0)
Cat n = 36	123.7 (67.6-226)	91.3 (43.0-193.8)	Cat n = 35	85.2 (46.6-155.7)	71.8 (36.1-143.1)
No cat n = 39	3.2 (1.9-5.2)	2.8 (1.6-4.9)	No cat n = 40	2.7 (1.8-3.9)	3.1 (2.0-4.9)

TABLE II. Der p 1 concentration ($\mu\text{g/g}$) and content ($\mu\text{g/m}^2$) in bedding

Bedding type	Concentration $\mu\text{g/g}$ (95% confidence interval) range (n)		Content $\mu\text{g/m}^2$ (95% confidence interval) range (n)	
	11 weeks	15 months	11 weeks	15 months
Sheepskin bedding	25.7 (15.1-43.8) 0-310.7 (n = 50)	67.8 (46.0-99.8) [†] 0-533.1 (n = 59)	7.0 (3.2-14.9)* 0-477.2 (n = 50)	83.1 (51.7-133.8) [‡] 0.1-2811.6 (n = 59)
Other bedding	15.5 (11.3-21.4) 0-205.4 (n = 104)	33.6 (25.5-44.2) [†] 0.1-473.2 (n = 95)	2.5 (1.6-3.9)* 0-136.1 (n = 104)	35.3 (23.7-52.5) [‡] 0-1548.3 (n = 95)

For differences between sheepskin and other bedding types, * $P < .05$, [†] $P < .01$, [‡] $P < .01$.

sheepskins at both visits (Table II). In the homes of cat owners, bedding Fel d 1 concentrations were higher in sheepskin-containing beds (71.0 $\mu\text{g/g}$ [21.9 to 230.4]) than in those without sheepskins (37.4 $\mu\text{g/g}$ [13.5 to 115.1]), but this difference was not significant.

DISCUSSION

The reservoir concentrations of Der p 1 in bedding identified in this study are approximately 10-fold higher than those previously reported for infant mattresses from the Netherlands.²⁷ Given the increased risks of sensitization and the development of asthma associated with levels considerably lower than those found in our study, it is perhaps not surprising that asthma in New Zealand is common, severe, and dominated by allergy to HDM. The great majority (96%) of the study infants were exposed to a Der p 1 concentration of more than 2 $\mu\text{g/g}$ within weeks of birth, with exposures continuing and increasing during the early months of life. These early life exposures have been clearly demonstrated to enhance the likelihood of development^{1,5,6,11} and persistence¹² of IgE sensitization to HDM and the subsequent development of clinical asthma.¹²⁻¹⁵ Furthermore, by a mean age of 15 months, 92% were exposed to levels of HDM allergen associated with major symptomatic exacerbations of asthma.^{7,8} Long-term follow-up of the infants in this

study is planned, with a focus on development of allergic sensitization and atopic disease.

The reasons for such mite proliferation cannot yet be clearly defined. However, a number of factors are likely to encourage mite growth: a temperate climate with high year-round humidity (mean relative humidity at 9 AM, 79% to 86%), wooden houses with suspended wooden floors, and almost universal wall-to-wall fitted carpets, which have been progressively introduced over the last 50 years. Central heating is uncommon, and dampness is frequently reported in both bedrooms and living areas.²³ By 15 months, the mean bedding Der p 1 concentration, 44.0 $\mu\text{g/g}$, was very similar to the 46.6 $\mu\text{g/g}$ previously found in bedding of 8- to 10-year-old children in Wellington. It seems likely, therefore, that the Der p 1 concentrations found at 15 months are maintained throughout childhood and may be an appropriate measure of likely childhood exposure levels in the absence of mite avoidance measures. The increase seen in floor Der p 1 levels at the second visit is consistent with previous data showing higher floor levels associated with a higher number of children in the household.²³

It is unlikely that season contributed to the changes seen in Der p 1 levels between visits because visits were spread throughout the year and infants were revisited in

the same season, 12 months later. In addition, although an autumn seasonal increase in Der p 1 levels on floors has been described, no such variation was found for bed levels in New Zealand.²³

Sheepskins have been commonly used as infant bedding since the late 1960s in New Zealand and Australia. Studies in the 1970s suggested that sheepskins may contain considerably greater numbers of mites than other forms of infant bedding, although considerable variability between individual sheepskins has been noted.²⁸⁻³¹ However, their use was not found to be a risk factor for asthma in one retrospective study in New Zealand.³² Our results, on the other hand, suggest that the use of sheepskins as infant bedding in atopic families is inappropriate because these lead to significantly greater exposures to HDM allergens. Similarly, the presence of a woollen underlay in older children's beds was associated with significantly higher Der p 1 concentrations.²³

Infant bedding has not previously been identified as a reservoir of cat allergen, although the high level of cat ownership in New Zealand has been previously noted.²¹ Levels of cat allergen depend primarily on the presence of a cat in the household. The Fel d 1 levels found on floors in our study are comparable with those reported from North American studies³³ but much higher than those reported from Sweden.³⁴

Given the compelling evidence for the risks of sensitization and allergic disease associated with these allergens, the levels of exposure documented in this study are of considerable concern. They suggest that at least in New Zealand, the concept of the infant bed as a low allergen environment is incorrect and that for infants at risk of atopic disease, early allergen avoidance measures will need to include a focus on the bed and bedding.

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