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Variation of dust endotoxin concentrations by location and time within homes of young children

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Abstract

Endotoxin may affect the development of allergic disease in childhood but little is known about endotoxin variation within homes. We sought to determine endotoxin concentration agreement within homes when five locations were each sampled twice 5 months apart. Endotoxin was measured using the recombinant *Limulus* factor C assay in dust samples from 585 homes of children enrolled in a prospective study and again in 335 homes 5 months later. The five locations sampled in each home were the child's bedroom floor, child's bed, mother's bedroom floor, mother's bed and living room floor. Concentrations of 4 allergens (Can f 1, Fel d 1, Der f 1 and Bla g 2) were also measured from the child's bedroom floor. In pair-wise comparisons, endotoxin concentrations in all locations within each home were significantly different from all other locations ($p < 0.001$) except for the child's and mother's bedroom floors ($p = 0.272$). Spearman correlations between endotoxin concentrations from the different locations were all statistically significant ($p < 0.05$) but of modest magnitude ($r = 0.24–0.54$). Similarly, correlations at each site over the 5 month observation interval were statistically significant but modest ($r = 0.17–0.44$). Pets and season of the year did not affect correlations, although correlations were lower if the floor was not carpeted. Endotoxin concentrations at all locations were minimally correlated with allergen concentrations in both negative and positive directions ($r = −0.12$ to 0.12). We conclude that a single measurement of endotoxin from a home dust sample provides an imprecise estimate of dust endotoxin concentrations in other locations within the home and over a relatively short observation interval.

Keywords

endotoxin; house dust; location; cats; dogs; carpets; seasons

Recently there has been intense interest in the hygiene hypothesis related to the increasing prevalence of allergic disease among children (1–3). Studies have used home endotoxin concentrations as a proxy of hygiene in the home. Some of these studies have shown that

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exposure to higher levels of endotoxin protects against allergic disease (4–8) while other studies have suggested that higher levels of endotoxin are associated with earlier and more frequent wheezing (9–11). The apparent dichotomy between beneficial and harmful effects of home endotoxin is seen in the study by Braun-Fahrlander et al. which showed a declining risk of allergic asthma but an increasing risk of non-allergic asthma in children associated with increasing bed endotoxin concentrations (8).

The majority of published studies have used a single measurement of endotoxin as an independent variable. Even when investigators have obtained endotoxin measurements from several locations in homes, their analyses are typically based on a single concentration from a single location at one time point (9, 12–15). The locations sampled have been empirically selected based on assumptions about where children are most likely to be exposed to endotoxin and few studies have examined allergic outcomes in relationship to endotoxin concentrations from multiple locations in homes (16, 17).

The goal of this study was to determine how well house dust endotoxin concentrations from multiple locations within a home agreed with each other. We also examined how well samples obtained from each location 5 months later agreed with the original sample. After finding modest agreement between endotoxin concentrations between locations and over time, we explored the possibility that other variables such as the presence of cats or dogs, carpeting on floors or the season of sampling modified the agreement between endotoxin concentrations within homes.

Methods

This study uses data collected as part of the Wayne County Health, Environment, Allergy, and Asthma Longitudinal Study (WHEALS). All components of this study have been approved by the institutional review boards (IRB) of Henry Ford Health System (HFHS) in Detroit and the Medical College of Georgia in Augusta. After giving informed consent, pregnant women were recruited prior to delivery from an area of western Detroit and adjacent suburbs defined by contiguous ZIP codes. Recruitment began in August, 2003 and is complete. To be eligible, pregnant women in their 2nd or 3rd trimester had to be at least 21 yr of age; live in the defined recruitment area; and attend one of five selected HFHS clinics for their prenatal care. Women were required to speak English well enough to provide written informed consent.

Collection of dust

At 1- and 6 months post-partum, research staff visited the homes of participating families and collected dust samples from five locations in the home. The locations sampled included the surface of the child's mattress and the surface of the mother's mattress after removing bedding, the floor next to the mother's bed, the floor next to the child's bed, and the floor of the room the mother reported the child would spend the most time other than their bedroom. This room was primarily the living or family room and for simplicity this location will be consistently referred to as the living room. A standardized sampling procedure was used to collect dust. A new dust collecting fine-weave cloth sock was placed into the vacuum hose and held in place with a clean nozzle. A measured string loop was laid out in the shape of a

square covering an area of one square meter and a timer was set for 2 min. Samples were obtained by vacuuming the 1 square meter area for 2 min. After collection, the sample sock was removed from the vacuum and placed into a pre-labeled plastic zip-lock bag and the bag sealed tightly, frozen at −80° C and shipped to the laboratory for assay on dry ice. In the laboratory, samples were processed and assayed without any additional freeze thaw cycles.

Dust sample preparation and endotoxin assay

In preparation for assay, dust samples were thawed, removed from the socks and sieved through a 292 micrometer mesh filter (Spectra Mesh Polyethylene, Spectrum, Laguna Hills, CA, USA) on an orbital shaker for 2 h. An aliquot of the fine dust passing through the sieve was then extracted in PBS containing 0.05% Tween 20 at room temperature (21–24° C) for a minimum of 2 h at a ratio of 50 micrograms of dust to 1 ml of PBS with constant agitation. Our preliminary experiments established that this concentration of Tween 20 did not alter the performance of the endotoxin assay used. These dust extracts were used for both measurement of endotoxin and the extract from the child's bedroom floor was also assayed for allergen content. After extraction the dust particles were removed from the extracting fluid using a serum filter system (Fisher Scientific, Pittsburgh, PA, USA) followed by centrifugation at 25,000 × *g* for 20 min.

Endotoxin assay

The endotoxin concentration in each dust sample was measured using a fluorescent microplate assay based on recombinant *Limulus* factor C (Pyrogene Recombinant Factor C (rFC), Cambrex Bio-Science, Walkersville, MD, USA) and endotoxin standards from Cambrex. Reference standard endotoxin (US Pharmacopoeia, Inc., Rockville, MD, USA) has been used to validate the endotoxin assay standard. Care was used to avoid endotoxin contamination by using endotoxin-free microtiter plates and pipette tips. For the initial assay of each sample, 100 μl of a 1:10,000 dilution of dust extract was placed into four wells of 96 well, flat-bottomed microplates. A known quantity of endotoxin was added to two of the four sample wells as an internal control to determine whether the sample contained substances capable of inhibiting the assay. The assay was started by addition of 100 μl of rFC and the fluorescent reagent. The plate was then placed into a 37° C incubating plate reader for an initial reading of fluorescence using excitation/emission wave lengths of 380/440 nm. A second reading was obtained 1 h later. The difference between the initial and 1 h reading was used to construct a standard curve on each plate and to calculate results of unknowns per manufacturer's recommendations. Following US Pharmacopoeia guidelines, the recovery of the internal control had to be between 50% and 200% for every sample. If the recovery was outside of these limits, the sample was reassayed at a greater dilution to try to reduce the influence of the suspected inhibitor. Results are reported as endotoxin units (EU) based on the US Pharmacopoeia, Inc. standard. If a value fell below the lower limit of the assay at the dilution allowing satisfactory recovery of the internal control, a value of 50% of the lower limit of the assay was assigned to the sample. The average inter-assay coefficient of variation was 18.9%.

Levels of allergens in dust

Because of funding limitations only samples from the floor of the child's bedroom at 1 month were assayed for 4 common allergens including: dog (Can f 1), cat (Fel d 1), dust mite (Der f 1) and cockroach (Bla g 2) using commercial monoclonal antibody assays (Indoor Biotechnologies, Ltd., Charlottesville, VA, USA) per manufacturer's protocol. The units for allergen dust measurements were nanograms of allergen per milligram of fine dust (ng/mg). The values determined to be the limit of detection for the assays were set at 0.1 ng/mg for Can f 1, 0.16 ng/mg for Fel d 1, 0.5 for ng/mg Der f 1 and 0.4 ng/mg for Bla g 2. For analyses samples with values below the detection limit were assigned a value of one-half the limit.

Statistical methods

Initial examination of the data showed that in nearly one half of the homes, the locations identified as the floor of the mother's bedroom and the floor of the child's bedroom were the same floor. When the mother and child were sleeping in the same room or in the same bed only one sample was collected from this floor or bed and analyzed. For analyses this common bed or bedroom floor was considered the mother's value. When comparisons and correlations were calculated only results from physically separate locations were analyzed (i.e., all comparisons of the child's and mother's bedroom floors and beds are based on samples from separate rooms and beds resulting in reduced sample sizes).

The results of the endotoxin concentrations were highly skewed so geometric means were used in calculations and in the results presented. Also because of the highly skewed nature of the data, non-parametric statistics were used for comparative analysis. For comparisons between locations and for the same locations over time the Wilcoxon matched-pairs signed-rank statistic was used. Significant differences with this test indicate that the relationships between the endotoxin concentrations are not consistent by location or over time within each home. The Kruskal–Wallis statistic was used for comparisons whenever multiple groups are compared. All correlations were computed with the Spearman rank order statistic. p-values of <0.05 were considered statistically significant. No adjustments were made for multiple comparisons because of the *a priori* nature of the hypotheses to be tested.

Results

The geometric means of the measured endotoxin concentrations in the five locations obtained when the child was 1 and 6 months of age are shown in Table 1. The concentrations ranged from 2.55 EU/mg to 20.36 EU/mg at 1 month and from 3.20 to 17.00 EU/mg at 6 months. At both sampling times, the lowest concentrations were from the beds and the highest from the living room floor.

When all possible pair-wise comparisons of the 1 month endotoxin concentrations were performed between the five locations, the differences by location were all statistically significant, with the exception of the mother's and child's bedroom floors, as shown in Table 2. Table 3 shows the non-parametric correlations between the endotoxin concentrations from each of the five locations sampled. All of the correlations are statistically significant but of

modest magnitude. The results of similar analyses at 6 months gave essentially the same results (data not shown).

Comparing the endotoxin concentrations at each location over 5 months showed a statistically significant change only in the child's bed where the geometric mean concentration increased from 2.36 EU/mg to 3.74 EU/mg of dust as shown in Table 4. Decreases in concentrations in the living room and mother's bedroom floors over the 5 months approached but did not reach statistical significance. Similarly, Table 5 shows the correlations between the 1 and 6 month samples for each location. All correlations are again statistically significant; however, the strongest correlation for a location between 1 and 6 months was only 0.44 for the child's bedroom floor. Consistent with the significant increase over 5 months, the weakest correlation between 1 and 6 months was for the child's bed.

We questioned whether factors that might influence dust endotoxin concentrations such as the presence of cats or dogs or carpets in the home might modify the degree of correlation between locations and that seasonal differences might alter the same location over time. Cats and dogs have been associated with endotoxin in other reports and such pets may prefer or be limited to certain rooms in the home. Stratifying the homes into those without and with cats or dogs in residence for analysis did not change any correlations to a statistically significant extent suggesting that between-home differences in cat or dog keeping were not a major factor reducing the correlations between locations (online Table S1). Similar analysis stratifying by the presence or absence of carpets or large area rugs showed a significant change in the correlation between the child's bedroom floor and the living room floor in homes without carpets and a borderline difference between the child's and mother's floors. Both correlations were lower for floors without carpets, Table S2. In the analyses of the effects of carpets, we only compared homes in which the child's bedroom and the living room were concordant for the presence or absence of carpets.

Our sampling scheme of separating home visits by 5 months meant that the 1 and 6 month samples were always taken in different seasons in a temperate climate. To examine the question of whether the change of seasons between the samples systematically affected the correlations, we calculated the correlations after stratifying by the season in which the 1 month sample was collected. Two of the 40 correlations significantly changed (Table S3). The two correlations that changed were between the living room floor and the mother's bed when the 1 month sample was obtained in either the fall or winter. When the initial sample was taken in the fall the correlation between the living room floor and the mother's bed improved but if taken in the winter, it was markedly reduced. Inspection of the changes in the other correlations does not suggest a consistent pattern of change related to season of sampling.

Discussion

Because of interest in the hygiene hypothesis many studies have evaluated endotoxin in house dust samples in relationship to allergic outcomes (1–3). An important factor in epidemiological studies is the relationship between actual exposure and the measure used to estimate the exposure (18, 19). When we compared endotoxin concentrations from five

different locations in each of 585 homes, the concentrations at all locations significantly differed from all other locations in the same home with the exception of the child's and mother's bedroom floors. Even though they were significantly different, concentrations between rooms were significantly but modestly correlated. The highest correlation coefficient, between the floors of the child's and mother's bedrooms, of 0.54 means that measuring endotoxin from the child's floor only explains 29.2% (0.54^2) of the variation of the endotoxin measured from the mother's floor.

While a number of studies have evaluated home endotoxin concentrations in relationship to allergic outcomes in children, information on endotoxin variation with time and by location is very modest in these studies as shown in Table S4 in the on-line supplement. The studies in Table S4 were selected by querying the Ovid database from 1996 to February, 2008 using the search terms of `endotoxin' combined with `immediate hypersensitivity' limited to etiology. Only primary research studies published in English were included. In the 20 of 117 articles meeting inclusion criteria, the study results were based on endotoxin measured at one location in 15 (75%), two or three locations in four (20%) and five locations in only one (5%) study. Only four studies examined the question of whether the health outcome of interest was more closely related to the endotoxin concentration in one vs. other locations(7, 16, 20, 21). In three of these studies the endotoxin concentration at only one location was significantly related to outcome (7, 20, 21). When adults were considered in the fourth study by Thorne et. al., only the bedroom floor was significant for all six asthma and wheezing outcomes in adults (≥18 yr) compared with bedding only being significant for one outcome and the family room floor being significant for two outcomes. However, in the same study there were no significant associations between endotoxin concentrations and any health outcome for children (< 18 yr) (16).

Gereda et al. commented that there was no change in endotoxin concentrations over a 6-month interval in 11 homes(4). Park et al. reported examined bed, bedroom floor and kitchen floor dust in 20 homes monthly for 1 yr. They found a seasonal influence on kitchen floor dust endotoxin levels but no seasonal influence on bed or bedroom floor levels. A subsequent study by the same group, reported no significant changes in endotoxin concentrations within homes over approximately 6 months after adjusting for season of the year (17). However, only 21 (4%) of the 470 homes in this study had complete data for all three sampling locations at both sampling times. A study by Heinrich et al. examined two samples of living room floor dust obtained approximately 7 months apart and found a crude correlation of 0.59 for endotoxin (22). This correlation is substantially greater than the correlation of 0.36 we found in living room floors over a similar interval. Unfortunately it is not possible to tell if the range of housing conditions in the study of Heinrich et al. is similar to the broad range in our study. After adjusting for season, Abraham et al. concluded that within-home variation was less than between-home variation in each of the three rooms they studied (17).

The study by Park et al. also reported modest correlations between bed and bedroom floor endotoxin of 0.45 ($p > 0.05$) and between bedroom floor and kitchen floor of 0.46 ($p < 0.05$) (23). Thorne et al. reported correlations between bedroom floors, beds, family room floors,

sofas and kitchen floors ranging from 0.12–0.44 in 831 homes (16). The correlations found by Thorne et al. are similar to those we found of 0.24 to 0.54 in 585 homes.

Based on the manufacture's claims that there were smaller lot-to-lot variations in reagents and that the rFC assay was more resistant to external influences we chose to use the rFC assay in this study rather than the more commonly used Limulus amebocyte lysate (LAL) assay for endotoxin. In a direct comparison of the two assays, Alwis and Milton, found that the LAL tended to give higher values but the results were highly correlated ($r = 0.86$, $p < 0.001$), leading them to conclude that either assay could be used for studies of house dust samples (24). Consistent with the results of the comparison study, the concentrations of endotoxin we measured are similar to those from other studies. For example, the geometric mean endotoxin concentration of the mother's bed in the LISA cohort (20) was 2.91 EU/mg compared with 3.54 EU/mg in our study. The geometric mean living room floor and child's bedroom floor concentrations in Manchester, UK (25) were 36.11 and 17.21 EU/mg respectively, compared with 20.36 and 12.78 EU/mg respectively, in our study. The concentrations in the survey by Thorne et al. (16) tended to be higher than we found but this may be due to a wide variety of possible differences besides the type of assay.

We did not find evidence that the modest correlations between locations in homes were related to differences in the presence of cats or dogs in the homes nor did we find consistent patterns in correlations over the 5 month sampling interval related to the seasons of the year when samples were taken (22). Correlations tended to be lower from non-carpeted floors.

Strengths of our study include the large number of homes selected through a population based cohort and sampled at five different locations in each home. The homes we sampled included a wide range of housing from inner-city apartments to suburban single-family homes. We also sampled homes with variable numbers of cats and dogs and homes with and without wall-to-wall carpeting.

Limitations of our study are the fact that we only evaluated dust and not airborne concentrations of endotoxin. Another limitation is that we only studied homes from one metropolitan area. Some of the variation we found over time in the homes may have been because of changes in the household related to the addition of a new child. While we had a broad range of housing types, our range of endotoxin concentrations did not reach that reported from rural homes in Europe but none of the homes in our study would be considered a farm home. Another limitation is that we do not have both 1 and 6 month samples for all of the homes which modestly reduces the precision of our estimates of correlations over time. We also did not sample any locations within a home multiple times on the same visit or over a short time interval to determine the reproducibility of our sampling methods for endotoxin. However the reproducibility of our endotoxin assay was relatively good with an inter-assay CV of 18.9%.

In conclusion endotoxin concentrations in settled dust vary significantly between different locations in homes and in the same locations over a few months. These results could be interpreted in at least two ways. The first interpretation would be that obtaining samples from several locations within each home at multiple intervals during a study would be

necessary to precisely estimate the relationship between endotoxin and the risk of allergic disease. An alternative interpretation is that even though estimates of endotoxin exposure are imprecise they are significantly associated with the risk of allergic disease suggesting that the relationship of endotoxin exposure to allergic disease is very strong. For allergists the question remains as to whether this line of research will lead to trials designed to prevent allergic disease.

Supporting Information

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Table 1

Median, minimum, maximum, 25% and 75% percentiles, values* of endotoxin concentrations from each of the five locations sampled in each home when the infant was 1 and 6 months of age

Time (month)	Location	n†	Minimum	25%	Median (GM‡)	75%	Maximum
1	Child's bed	515	0.05	1.10	2.80 (2.55)	6.20	692.10
	Child's bedroom floor	268	0.05	5.75	13.65 (12.78)	31.85	500.60
	Living room floor	585	0.05	9.80	22.40 (20.36)	46.00	997.00
	Mother's bed	585	0.05	1.50	4.10 (3.54)	9.70	873.30
	Mother's bedroom floor	585	0.05	6.10	16.00 (14.48)	36.00	886.20
6	Child's bed	286	0.05	1.40	3.65 (3.62)	9.20	538.90
	Child's bedroom floor	216	0.20	4.60	12.80 (11.38)	29.15	312.90
	Living room floor	335	0.10	8.70	18.20 (17.00)	38.50	1195.50
	Mother's bed	335	0.05	1.30	3.50 (3.20)	8.30	143.20
	Mother's bedroom floor	335	0.05	5.40	13.30 (12.66)	30.50	1109.80

*AII endotoxin values are given as EU/g of fine dust.

†The number of homes with samples at each location. The numbers vary because of missing or inadequate samples and numbers for the child's bedroom floor are substantially smaller because there were 317 homes at one month and 119 at 6 months where the child was sleeping in the same room as the mother. The results for the floors when the child and mother are sleeping in the same room are grouped as the mother's bedroom floor.

‡For easier comparison with some previous studies the geometric mean (GM) is given in addition to the median.

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Table 2

Pair-wise comparisons of endotoxin concentrations at 1 month between the five home locations studied

Location	n*	GM	Ln mean	Ln SD	Location	n*	GM	Ln mean	Ln SD	p-Value†
Child's bed	261	2.41	0.88	1.68	Child's bedroom floor	261	12.94	2.56	1.40	0.001
	515	2.53	0.93	1.50	Living room floor	515	20.09	3.00	1.28	0.001
	515	2.53	0.93	1.50	Mother's bed	515	3.78	1.33	1.49	0.001
	515	2.53	0.93	1.50	Mother's bedroom floor	515	14.73	2.69	1.43	0.001
Child's bedroom floor	268	12.81	2.55	1.39	Living room floor	268	19.30	2.96	1.20	0.001
	268	12.81	2.55	1.39	Mother's bed	268	3.32	1.20	1.62	0.001
	268	12.81	2.55	1.39	Mother's bedroom floor	268	14.01	2.64	1.44	0.272
Living room floor	585	20.29	3.01	1.28	Mother's bed	585	3.53	1.26	1.55	0.001
	585	20.29	3.01	1.28	Mother's bedroom floor	585	14.44	2.67	1.43	0.001
Mother's bed	585	3.53	1.26	1.55	Mother's bedroom floor	585	14.44	2.67	1.43	0.001

GM, geometric mean in endotoxin units (EU)/mg; Ln mean, natural logarithm of the mean concentrations of endotoxin expressed as endotoxin units (EU)/mg of dust; Ln SD, standard deviation of the log mean.

*Sample size for each comparison varies primarily because of homes in which the child was sleeping in the same room as the mother at the time the samples were obtained and the floor values from these rooms are considered as the maternal bedroom floor. There are also minor variations because of missing samples.

†Wilcoxon matched-pairs signed-rank test.

Table 3

Correlations* between endotoxin concentrations from each of the five locations studied at 1 month

	Child's bedroom floor	Living room floor	Mother's bed	Mother's bedroom floor
Child's bed	0.32	0.24	0.41	0.28
Child's bedroom floor		0.49	0.38	0.54
Living room floor			0.29	0.48
Mother's bed				0.39

* Based on samples from 261 to 515 homes as indicated in Table 2, Spearman Rank Order Correlations, all correlations are statistically significant, $p < 0.01$.

Table 4

Pair-wise comparisons of endotoxin concentrations at 1 and 6 months at each of the five home locations studied[*]

	1-month values				6-month values				
Location	**n[*]**	**GM[**]**	**Ln mean[**]**	**Ln SD**	**n[*]**	**GM[**]**	**Ln mean[**]**	**Ln SD**	**p-Value[†]**
Child's bed	262	2.36	0.86	1.41	262	3.74	1.32	1.45	0.001
Child's bedroom floor	143	12.18	2.50	1.42	143	10.59	2.36	1.45	0.268
Living room floor	335	19.69	2.98	1.29	335	16.95	2.83	1.32	0.079
Mother's bed	335	3.74	1.32	1.52	335	3.19	1.16	1.46	0.130
Mother's bedroom floor	335	14.88	2.70	1.34	335	12.68	2.54	1.45	0.072

[*] Sample size is limited to homes with valid sample values at both 1 and 6 months for each location. The numbers for the child's floor exclude homes in which the child slept (or was sleeping) in the same room as the mother at either 1 or 6 months.

[**] GM, geometric mean in endotoxin units (EU)/mg; Ln mean, natural logarithm of the mean concentrations of endotoxin expressed as EU/mg of dust; Ln SD, standard deviation of the log mean.

[†] Wilcoxon matched-pairs signed-rank test.

Table 5

Correlations between endotoxin concentrations from each of the five locations studied at 1 and 6 months

Comparison	Correlation*
Child's bed	0.17
Child's bedroom floor	0.44
Living room floor	0.36
Mother's bed	0.24
Mother's bedroom floor	0.39

* Based on results of samples from 262 to 335 homes with samples from all five locations at both 1 and 6 months as shown in Table 3, Spearman Rank Order Correlations, all correlations are significant, $p < 0.01$.