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Are Cats and Dogs the Major Source of Endotoxin in Homes?

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Abstract

Previous studies have suggested that exposure to cats and dogs during early childhood reduces the risk of allergic disease, possibly by increasing home endotoxin exposure. This study asked the question of whether cats and dogs are the dominant influence on dust endotoxin concentrations in homes after considering other variables reportedly associated with endotoxin. The presence of cats or dogs in homes, household and home characteristics, and dust endotoxin concentrations from 5 locations were assessed in 966 urban and suburban homes. Whether considered together as pets or as cats and dogs separately, the presence of cats and dogs significantly contributed to living room and bedroom floor endotoxin concentrations but not to bed endotoxin concentrations. However, the two variables consistently related to endotoxin in all home sites were the home occupant density (occupants/room) and cleanliness of the home. Our data suggests that reducing occupant density and improving home cleanliness would reduce home endotoxin concentrations more than removing pet cats or dogs from the home.

Keywords

Allergy; asthma; beds; cats; children; cleanliness; condition of home; dogs; endotoxin; floors; homes; occupant density; pets; prevention

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The hygiene hypothesis has been proposed to explain the increased prevalence of allergic disease in the past few decades. One line of investigation supporting this hypothesis has been studies demonstrating that living on farms or the presence of pets (cats or dogs) in homes lowers the risk of allergic disease in children (Langan et al., 2007; Lodge et al., 2012; Takkouche et al., 2008; Tse and Horner, 2008). Many investigators have suggested that the biological mechanism through which pet and other animal exposure lowers the risk of allergic disease is by increasing exposure to endotoxin (Abraham et al., 2005; Bolte et al.,

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2003; Celedon et al., 2007). Braun-Fahrlander and colleagues demonstrated a strong inverse relationship between endotoxin concentrations in a child's bed and allergic asthma (Braun-Fahrlander et al., 2002). Other studies have also shown that higher endotoxin concentrations are associated with reduced risk of allergic disease in children (Bottcher et al., 2003; Gehring et al., 2008) but some studies have suggested increased asthma with higher endotoxin concentrations (Thorne et al., 2005).

As endotoxin exposure may alter the risk of developing allergic disease and has many known effects on health it is valuable to understand what factors contribute to endotoxin levels in the home. If endotoxin concentrations are important factors in the sequence of events linking environmental exposures to the risks of allergy and asthma, knowledge of the characteristics associated with home endotoxin are important for designing potential for intervention (Douwes et al., 2004; Liu, 2003).

Many studies have examined relationships between household characteristics and endotoxin concentrations producing variable results. Unfortunately, none of these studies have specifically addressed the question of whether indoor cats and dogs are the dominant influences on endotoxin. The existing studies have other limitations such as examining endotoxin from only one location in the home, usually the living room floor, or relatively small sample sizes compared to the variability in endotoxin concentrations. An exception to these limitations is the study of a representative sample of 831 homes in the United States by Thorne and colleagues (Thorne et al., 2009). Five sites were sampled in each home. They found that house dust endotoxin concentrations were principally associated with the location sampled in the home, census division (geographic location in the United States), educational attainment of adults in the home, presence of children, dog but not cat ownership, evidence of cockroaches and smoking. A major strength and limitation of this study is the nationally representative nature of the sample. The variation in endotoxin found across the U.S. limited the power of the study to characteristics of the homes beyond geographic location that strongly influenced endotoxin concentrations. Another report from this same study demonstrated significant relationships between home endotoxin concentrations and asthma, asthma symptoms, use of asthma medications and wheezing. A study of 1065 German, Dutch and Swedish homes with pre-school children found relatively small differences in mean home endotoxin concentrations but keeping pets and having more than four persons residing in the home were associated with increased concentrations of endotoxin in both mattress and floor dust (Giovannangelo, M et al., 2007a). This study also demonstrated large variations in the variables related to endotoxin by country leading to the conclusion that cats and dogs were related to mattress endotoxin concentrations in Germany but not in Sweden or the Netherlands.

Considering the large number of studies suggesting a reduced risk of childhood allergic disease and asthma associated with indoor cats and dogs but conflicting findings concerning the relationship of home endotoxin to asthma, we felt it was important to address the hypothesis that cats and dogs are the major sources of endotoxin in mid-west U.S. homes, adjusting for other variables. Additionally we examined how consistently other home variables mentioned in the literature were significantly related to endotoxin concentrations in multiple locations within homes.

METHODS

Study population

The creation of our cohort has been previously described (Aichbhaumik et al., 2008). Briefly, all expectant mothers meeting enrollment criteria in metropolitan Detroit, Michigan, who were visiting prenatal clinics of Henry Ford Health System, were invited to participate in the geographically based Wayne County Health, Environment, Allergy and Asthma Longitudinal Study (WHEALS) regardless of family allergic history and insurance status. To be eligible, pregnant women had to be at least 21 years of age; live in an area of urban and suburban Detroit defined by contiguous ZIP codes, and expect to remain in the area for at least 2 years. Enrollment in this cohort began in August, 2003 and was completed in 2007. This study was approved by the Institutional Review Boards of Henry Ford Health System and Georgia Health Sciences University.

Data collection

Staff administered detailed questionnaires to participating mothers prior to delivery, as well as at 1month following birth. Questions included socio-demographic information, allergy and asthma history, and details about various environmental exposures (e.g., pets). More specific information on cats and dogs and other variable such as the location of the home were based on maternal report during the 1 month home interview. Women were asked about the numbers of cats or dogs and the amount of time each animal spent indoors on a daily basis. If the mother stated that the pet was outdoors 24 hours per day, the home was not considered as a home with a pet in these analyses of endotoxin concentrations. Mothers were also asked if they smoked and about the numbers of children (<18 years) and adults (≥18 years) living in their home. Occupant density was defined as the number of persons living in the home divided by the number of rooms in the home excluding bathrooms and basements.

At 1 month post-delivery, staff visited homes of participants, administered questionnaires, collected dust samples and assessed the condition of general cleanliness of the home using a standardized data collection form. The scale used to rate the cleanliness of the home was: 1. extremely poor housekeeping (no evidence of recent cleaning, lack of organization, greasy cooking area, clutter throughout); 2. not as bad as class 1 (but unless some attention is given to housekeeping, could become a 1); 3. average level of housekeeping,(periodic cleaning evident); 4. above average (very clean, little clutter); 5. good housekeeping award (neatly organized, nothing out of place, clean throughout). Visual evidence of vermin (rats, mice, cockroaches) or their remains was assessed and recorded as present or not present.

The methods of dust collection and analysis for endotoxin and allergens has been previously described (Williams et al., 2006). The five sites from which dust samples were collected included the mother's bedroom, infant's bedroom and living room floors and the mother's and infant's mattresses. At each location dust samples were collected from an area of 1 m^2 by vacuuming for 2 minutes. Sieved dust samples were extracted in PBS containing 0.05% Tween 20 at room temperature (21–24° C) for a minimum of 2 hours at a ratio of 50 micrograms of dust to 1 ml of PBS.

Endotoxin concentrations in dust samples from all 5 home locations were measured as previously described (Ownby et al., 2009) using the recombinant *Limulus* factor C assay (PyroGene Recombinant Factor C, Cambrex Bio Science, Walkersville, Maryland). Results were reported as endotoxin units (EU) per milligram of dust (EU/mg). Results falling below the lower limit of the assay were assigned a value of 50% of the assay's lower limit if there was satisfactory recovery of the internal control in the sample.

Concentrations of four major allergens (dog [Can f 1], cat [Fel d 1], dust mite [Der f 1] and German cockroach [Bla g 2]) were measured in the dust samples only from the child's bedroom floor using commercially available monoclonal antibody assays as previously reported (Nicholas et al., 2008; Nicholas et al., 2010). Dust samples from other areas could not be assayed for allergens because of limited resources.

Statistical Analysis

Initial analyses focused on endotoxin in the living room floor because many previous papers have focused on this site. The mother's and child's bedroom floors were also sampled but if the mother and child slept in the same room, the floor dust sample from the room was only considered as the mother's bedroom floor. Similarly, if the mother and child slept in the same bed, the dust sample from the bed was considered to be from the mother's bed. These limitations were necessary to avoid using the same value twice in calculations.

Since endotoxin concentrations were not normally distributed they were transformed to $\log_e$ equivalents for analysis resulting in a distribution that appeared normal on visual inspection of a frequency histogram. Multivariable linear regression based on the transformed data was performed to describe the relationship between endotoxin concentrations (dependent variable) and other independent variables *a priori* thought to be related to the presence of endotoxin in homes. Thus, coefficients of regression equations are in relationship to a 1 natural log change in endotoxin concentration. The variables included in models were selected based on the previous literature and the results of univariate analyses: number of pets, cats or dogs; whether the home was located in the City of Detroit (urban) or outside the city (suburban); occupant density; cleanliness of the home; maternal smoking; and evidence of vermin. In these analyses the comparator group was always homes with no cats or dogs. The variables for one or two or more pets were always kept in the multivariate models as all other variables were added to each model. A p-value ≤ 0.05 was considered statistically significant. All analyses were performed using SAS for Windows version 9.1; SAS Institute, Cary, NC.

Results

A total of 1258 women enrolled in the study during pregnancy and of these 966 (76.8%) remained in the study, provided complete information on pets and allowed collection of dust samples for endotoxin analysis from the living room floor during a home visit when the child was 1 month old. The characteristics of this study population and a comparison to those excluded from analyses are shown in Table 1. The significant differences were that excluded homes were more likely to have at least one pet, to be located in the urban area, and have a slightly larger average family size. Among homes included, at least one cat or dog was present at least part of most days in 33.1% (320/966) of the homes.

Households with indoor dogs (n=228, 23.6%) outnumbered those with indoor cats (n=139, 14.4%). A slight majority (56.6%) of the homes was located within the City of Detroit (urban) and all others were located in Detroit suburbs. Only 10.1% of mothers stated that they smoked at the 1 month visit. Endotoxin concentrations in dust were higher on floors than in beds with living room floors having the highest average concentration.

As shown in Table 2 all selected independent variables were significantly related to endotoxin concentrations from the living room floor. Having at least one cat or dog was always related to the endotoxin concentration, although the differences between having 1 or ≥2 pets in the home were not consistently significant. The rated cleanliness of the home shows a negative coefficient meaning that the more cleanly the home (the better the house keeping appeared) the lower the concentration of endotoxin.

The results of considering all of the selected variables except for the allergens, Can f 1 and Fel d 1, together are shown as three models in Table 3. The results are relatively consistent in all models, the density of people in the home and the cleanliness of the home are significant in all models as is a variable for pets, dogs or cats. Two or more cats did not significantly contribute more endotoxin than one cat. The location of the home, urban or suburban, was not significant nor was evidence of vermin. Maternal smoking approached significnace.

Twelve other variables were considered by adding each variable individually to the models in Table 3. The 12 variables evaluated were: use of air conditioning, allergen levels from the infant's bedroom floor dust (Can f 1, Der f 1, Fel d 1, Bla g 2), the presence of a rug in the living room, mother's educational attainment, father's educational attainment, number of persons in the family, number of children (<18 years) in the family, family income, and season of the year. Descriptions of these variables are provided in supplemental on-line Tables 1 A & B. When these variables were considered the only significant variables were the concentrations of Can f 1 and Fel d 1 as shown in Table 2 and on-line Table 2. The addition of Can f 1 and Fel d 1 to models resulted in minimal changes in the coefficients of the other variables already in the models but Fel d 1 and not Can f 1 was significant (p =0.003) in the model considering pets. Similarly, in the model considering cats, Fel d 1 was significant (coefficient 0.31, p = 0.009) again with minimal changes in the coefficients or p-values of the other variables. Can f 1 was not significant in the model considering dogs, (coefficient 0.16, p=0.143) but the variable for 1 dog lost significance with the inclusion of Can f 1 concentrations while the variable for ≥2 dogs was minimally changed.

The relationships of the same variables and a variable for 1 or ≥2 pets to endotoxin in the four other sampled home sites (the mother's and child's beds, and the mother's and child's bedroom floors) were analyzed and the individual models for each location are shown in Table 4. Models 1 and 2 in predict endotoxin in the child's and mother's bedroom floors, respectively, and are similar to the results of the living room floor shown in Model 1 of Table 3. The major difference found for the bedroom floors compared to the living room

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floor is that a single pet is no longer significant. When endotoxin concentrations in the child's and mother's beds were considered in Models 3 and 4, the pet variables were no longer significant. The cleanliness of the home remained significant while the variable for occupant density was significant in all models except for model 3 of the child's bed were is was of borderline significance (p=0.053).

Finally we examined the question of whether there were important differences in the amount of endotoxin contributed by cats compared to dogs. The results of this model in which only pets were considered is shown in the on-line Table 3. The equation coefficients indicate the relative contribution of each variable to the living room endotoxin concentration above the baseline condition of no pets. The presence of cats contributed slightly more (0.35 $\log_e$ units of endotoxin, p=0.018) than the presence of dogs (0.23 $\log_e$ units of endotoxin, p=0.031) and the combination of both cats and dogs contributed more (0.55 $\log_e$ units of endotoxin, p=0.005) than either cats or dogs alone.

Discussion

We found that the two variables most consistently associated with endotoxin concentrations from the floors and beds sampled within each home were the occupant density in the home and the cleanliness of the home. The presence of cats or dogs in the home was associated with significantly higher endotoxin concentrations on the living and bedroom floors but not in the child's or mother's beds. These findings are not consistent with the initial hypothesis that cats and dogs are the major source of endotoxin throughout homes.

Our findings are generally consistent with those of others. Our geometric endotoxin concentrations in five locations are similar to those reported by Thorne et al. (Thorne et al., 2009). The slightly higher concentrations reported by Thorne et al. are likely due to the differences between the amebocyte lysate used by Thorne et al. and recombinant factor C assays which we used. Many studies have also found that home endotoxin concentrations are related to dogs, cats, home cleaning practices, and the number of inhabitants or inhabitants per room or floor area (Campo et al., 2006; Dassonville et al., 2008; Giovannangelo, M et al., 2007a; Heinrich et al., 2001; Park et al., 2001). For example, Heinrich et al. in Germany found that dogs, cats and vermin all contributed significantly to the amount of endotoxin in dust from living room floors, but they did not attempt to determine the relative contributions of different home characteristics (Heinrich et al., 2001). Gereda et al. examined the effects of multiple variables and found that the only variables related to endotoxin were the presence of pets and central air conditioning (Gereda et al., 2001). Based on their paper we evaluated air conditioning but it was not significantly related to endotoxin in our homes.

In contrast to our findings, Platts-Mills et al, examined 71 homes in Virginia and found that the presence of dogs contributed more to airborne endotoxin than did the presence of cats (Platts-Mills et al., 2005). But, consistent with our findings, they found that the combination of cats and dogs was associated with higher endotoxin that either a cats or dogs alone. The differences between measuring airborne versus settled dust and perhaps geographic distance may explain these differences. Singh et al. have shown only modest correlations between airborne and dust endotoxin concentrations and they did not find significant associations between dogs or cats and either airborne of dust concentrations of endotoxin (Singh et al., 2011). The lack of association in this study is presumably from the small number of homes examined (n=184) since a previous report from the same parent study demonstrated an association between dogs and endotoxin (Campo et al., 2006).

Other possible sources of variation between studies of home endotoxin concentrations include sampling and assay methods. Many studies used the older *Limulus* amebocyte lysate assay to measure endotoxin in contrast to the newer recombinant *Limulus* factor C assay. A study concluded that both assays are adequate for assaying house dust samples even though the newer assay appears more resistant to the influence of other substances in dust (Alwis and Milton, 2006). Dust sampling methods also varied between studies but these variations would be more likely to affect measures of total endotoxin rather than endotoxin concentrations. The sites chosen for sampling in the home is important. Both Thorne et al. and we have shown significant variation in endotoxin between sites in the same home (Ownby et al., 2009; Thorne et al., 2009). There are also variations in endotoxin within homes over time and by season of the year (Ownby et al., 2009; Park et al., 2000).

Differences between studies related to the association of cats and dogs to endotoxin concentrations are more difficult to explain. Among the many potential reasons for these differences are: the way pets are treated in homes (e.g. are they allowed in all rooms, allowed on furniture, frequency of washing), the relative abundance of keeping different breeds (size) of cats or dogs, and the frequency with which the pet goes outdoors and whether they are free outdoors to do things like roll on the ground.

Strengths of our study included the large number of homes sampled and obtaining samples from five sites in nearly all homes. We also sampled homes ranging from small urban apartments to large suburban homes. Extensive information was collected about each home, home occupants, and pets. Homes were also visually surveyed during home visits.

Limitations of our study included only studying a single geographic area of the United States. Because we were establishing a birth cohort all homes had at least one child in the home at the time of sampling so the association of children with endotoxin could not be evaluated. It would have been interesting to sample all homes repeatedly over a year but this would have been a burden on participants and beyond our budget. However, we did consider a variable for seasons in our analyses and it was not significant. Some previous studies have evaluated the question of the best method for evaluating endotoxin exposure of children in homes: are measures in bedding better than floor samples and are calculations of concentration different from calculations of surface load (endotoxin per m^2). Douwes et al found that measures of floor endotoxin were more closely related to allergic outcomes than measure of bedding endotoxin (Douwes et al., 2006). Others have shown that within home variation between floor and bedding endotoxin is less than between homes (Giovannangelo, M et al., 2007b). We evaluated five locations in each home but did not compare endotoxin concentration to endotoxin load for each location.

The two variables most consistently related to endotoxin from all five home sites in this study were the home occupant density and the cleanliness of the home. These variables appeared to capture much of the relevant information of other previously studied variables such as parental education and income, number of persons or children in the home, and air conditioning. The strong relationship between endotoxin and the density of habitation in the home is not surprising. The roles of humans as a source of bacteria in the home was well demonstrated by Täuble et al. whose data showed that 69–88% of the bacteria in beds are of human origin and from 45–55% of the bacteria found on floors were from humans (Täubel et al., 2009). However, variables for cats and dogs provided information in models about endotoxin beyond that provided by habitation density and cleanliness.

Our findings suggest that the most effective means of minimizing exposure to endotoxin in all areas of homes would be to reduce occupant density and to keep the home clean. Additional methods to reduce endotoxin in floor dust would be to avoid indoor cats and dogs and to avoid smoking in the home. No large prospective studies have shown whether efforts to minimizing home endotoxin concentrations would alter the risk of allergic disease or wheezing in home occupants.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Abbreviations

WHEALS	Wayne County Health, Environment, Allergy and Asthma Longitudinal Study
CI	95% confidence interval
SD	standard deviation of the mean

References Cited

Abraham JH, Finn PW, Milton DK, Ryan LM, Perkins DL, Gold DR. Infant home endotoxin is associated with reduced allergen-stimulated lymphocyte proliferation and IL-13 production in childhood. Journal of Allergy and Clinical Immunology. 2005; 116:431–437. [PubMed: 16083802]

Aichbhaumik N, Zoratti EM, Strickler R, Wegienka G, Ownby DR, Havstad S, Johnson CC. Prenatal exposure to household pets influences fetal immunoglobulin E production. Clin Exp Allergy. 2008

Alwis KU, Milton DK. Recombinant factor C assay for measuring endotoxin in house dust: Comparison with LAL, (1→3)-β-D-glucans. American Journal of Industrial Medicine. 2006; 49:296–300. [PubMed: 16550568]

Bolte G, Bischof W, Borte M, Lehmann I, Wichmann H-E, Heinrich J. Early endotoxin exposure and atopy development in infants: results of a birth cohort study. Clin and Experimental Allergy. 2003; 33:770–776.

Bottcher MF, Bjorksten B, Gustafson S, Voor T, Jenmalm MC. Endotoxin levels in Estonian and Swedish house dust and atopy in infancy. Clin Exp Allergy. 2003; 33:295–300. [PubMed: 12614441]

Braun-Fahrlander C, Riedler J, Herz U, Eder W, Waser M, Waser M, Grize L, Maisch S, Carr D, Gerlach F, Bufe A, Lauener RP, Schierl R, Renz H, Nowak D. Environmental exposure to endotoxin and its relation to asthma in school-age children. The New England Journal of Medicine. 2002; 347:869–877. [PubMed: 12239255]

Campo P, Kalra HK, Levin L, Reponen T, Olds R, Lummus ZL, Cho S-H, Khurana Hershey GK, Lockey J, Villareal M, Stanforth S, LeMasters G, Bernstein DI. Influence of dog ownership and high endotoxin on wheezing and atopy during infancy. Journal of Allergy and Clinical Immunology. 2006; 118:1271–1278. [PubMed: 17157656]

Celedon JC, Milton DK, Ramsey CD, Litonjua AA, Ryan L, Platts-Mills TAE, Gold DR. Exposure to dust mite allergen and endotoxin in early life and asthma and atopy in childhood. Journal of Allergy and Clinical Immunology. 2007; 120:144–149. [PubMed: 17507083]

Dassonville C, Demattei C, Vacquier B, Bex-Capelle V, Seta N, Momas I. Indoor airborne endotoxin assessment in homes of Paris newborn babies. Indoor. Air. 2008; 18:480–487. [PubMed: 19120498]

Douwes J, Le Gros G, Gibson P, Pearce N. Can bacterial endotoxin exposure reverse atopy and atopic disease? Journal of Allergy and Clinical Immunology. 2004; 114:1051–1054. [PubMed: 15536409]

Douwes J, van Strien R, Doekes G, Smit J, Kerkhof M, Gerritsen J, Postma D, de Jongste J, Travier N, Brunekreef B. Does early indoor microbial exposure reduce the risk of asthma? The prevention and incidence of asthma and mite allergy birth cohort study. Journal of Allergy and Clinical Immunology. 2006; 117:1067–1073. [PubMed: 16675334]

Gehring U, Strikwold M, Schram-Bijkerk D, Weinmayr G, Genuneit J, Nagel G, Wickens K, Siebers R, Crane J, Doekes G, Di Domenicantonio R, Nilsson L, Priftanji A, Sandin A, El-Sharif N, Strachan D, van Hage M, Von Mutius E, Brunekreef B. ISAAC Phase Two Study Group. Asthma and allergic symptoms in relation to house dust endotoxin: Phase Two of the International Study on Asthma and Allergies in Childhood (ISAAC II). Clin Exp Allergy. 2008; 38:1911–1920. [PubMed: 18771486]

Gereda JE, Klinnert MD, Price MR, Leung DYM, Liu AH. Metropolitan home living conditions associated with indoor endotoxin levels. Journal of Allergy and Clinical Immunology. 2001; 107:790–796. [PubMed: 11344344]

Giovannangelo M, Gehring U, Nordling E, Oldenwening M, Terpstra G, Bellander T, Hoek G, Heinrich J, Brunekreef B. Determinants of house dust endotoxin in three European countries - the AIRALLERG study. Indoor Air. 2007a; 17:70–79. [PubMed: 17257154]

Giovannangelo M, Nordling E, Gehring U, Oldenwening M, Bellander T, Heinrich J, Hoek G, Brunekreef B. Variation of biocontaminant levels within and between homes--the AIRALLERG study. J Expo. Sci Environ Epidemiol. 2007b; 17:134–140. [PubMed: 16598184]

Heinrich J, Gehring U, Douwes J, Koch A, Fahlbusch B, Bischof W, Wichmann H-E. Pets and vermin are associated with high endotoxin levels in house dust. Clin Exp Allergy. 2001; 31:1839–1845. [PubMed: 11737034]

Langan SM, Flohr C, Williams HC. The role of furry pets in eczema: a systematic review. Arch Dermatol. 2007; 143:1570–1577. [PubMed: 18087010]

Liu AH. Endotoxin exposure in allergy and asthma: reconciling a paradox. Journal of Allergy and Clinical Immunology. 2003; 109:379–392. [PubMed: 11897980]

Lodge CJ, Allen KJ, Lowe AJ, Hill DJ, Hosking CS, Abramson MJ, Dharmage SC. Perinatal cat and dog exposure and the risk of asthma and allergy in the urban environment: a systematic review of longitudinal studies. Clin Dev.Immunol. 2012; 2012:176484. [PubMed: 22235226]

Nicholas C, Wegienka G, Havstad S, Ownby D, Johnson CC. Influence of cat characteristics on Fel d 1 levels in the home. Ann Allergy Asthma Immunol. 2008; 101:47–50. [PubMed: 18681084]

Nicholas C, Wegienka G, Havstad S, Zoratti E, Ownby D, Johnson CC. Dog characteristics and allergen levels in the home. Ann Allergy Asthma Immunol. 2010; 105:228–233. [PubMed: 20800790]

Ownby DR, Peterson EL, Williams LK, Zoratti EM, Wegienka GR, Woodcroft KJ, Joseph CLM, Johnson CC. Variation of dust endotoxin concentrations by location and time within homes of young children. Pediatirc Allergy and Immunology. 2009 in press.

Park J-H, Spiegelman DL, Burge HA, Gold DR, Chew JL, Milton DK. Longitudinal study of dust and airborne endotoxin in the home. Environmental Health Perspective. 2000; 108:1023–1028.

Park J-H, Spiegelman DL, Gold DR, Burge HA, Milton DK. Predictors of airborne endotoxin in the home. Environmental Health Perspective. 2001; 109:859–864.

Platts-Mills JA, Custis NJ, Woodfolk JA, Platts-Mills TAE. Airborne endotoxin in homes with domestic animals: Implications for cat-specific tolerance. Journal of Allergy and Clinical Immunology. 2005; 116:384–389. [PubMed: 16083794]

Singh U, Levin L, Grinshpun SA, Schaffer C, Adhikari A, Reponen T. Influence of home characteristics on airborne and dustborne endotoxin and beta-D-glucan. J Environ.Monit. 2011; 13:3246–3253. [PubMed: 22012201]

Takkouche B, Gonzáles-Barcala F-J, Etminan M, FitzGerald M. Exposure to furry pets and the risk of asthma and allergic rhinitis: a meta-analysis. Allergy. 2008; 63:857–864. [PubMed: 18588551]

Täubel M, Rintala H, Pitkaranta M, Paulin L, Laitinen S, Pekkanen J, Hyvarinen A, Nevalainen A. The occupant as a source of house dust bacteria. Journal of Allergy and Clinical Immunology. 2009; 124:834–840. [PubMed: 19767077]

Thorne PS, Cohn RD, Mav D, Arbes SJ, Zeldin DC. Predictors of endotoxin levels in U.S. housing. Environ Health Perspect. 2009; 117:763–771. [PubMed: 19479019]

Thorne PS, Kulhankova K, Yin M, Cohn R, Arbes SJ, Zeldin DC. Endotoxin exposure is a risk factor for asthma: The National Survey of Endotoxin in the United States. American Journal of Respiratory and Critical Care Medicine. 2005; 172:1371–1377. [PubMed: 16141442]

Tse K, Horner AA. Defining a role for ambient TLR ligand exposures in the genesis and prevention of allergic disease. Semin Immunopathol. 2008; 30:53–62. [PubMed: 17989979]

Williams LK, McPhee RA, Ownby DR, Peterson EL, James M, Zoratti EM, Johnson CC. Gene-environment interactions with CD14 C-260T and their relationship to total serum IgE levels in adults. Journal of Allergy and Clinical Immunology. 2006; 118:851–857. [PubMed: 17030237]

Practical Implications

Many studies have shown that early childhood exposure to indoor cats or dogs is associated with a reduced risk of later allergic disease and asthma. An important question is whether alteration in allergic risk associated with cat and dog exposure results from increased endotoxin exposure or from some other associated exposure. Our findings show that cats and dogs are not the dominant source of endotoxin in homes, rather the density of human occupation and poor cleaning contribute more consistently to higher home endotoxin concentrations especially in the beds.

Table 1

Comparison of Variables for Subjects Included and Excluded from Analyses

Variable	Excluded*	Included*	P-value
	% or mean ± SD or (95% CI)	% or mean ± SD or (95% CI)	
Pets:			
no dogs and no cats	14.3	66.9	0.001
1 dog and no cats	45.2	14.3	
no dog and 1 cat	7.1	5.4	
1 dog and 1 cat	2.4	2.3	
≥ 2 dogs and no cat	28.6	4.5	
no dog and ≥ 2 cats	0	4.1	
≥ 2 dogs and 1 cat	0	0.7	
1 dog and ≥ 2 cats	2.4	1.6	
≥ 2 dogs and ≥ 2 cats	0	0.3	
Cleanliness:			
very dirty	1.9	4.4	0.770
dirty	13.5	14.3	
average	55.8	47.5	
clean	21.2	24.8	
very clean	7.7	9.1	
Urban	75.3	56.6	0.001
Mother smokes	15.1	10.1	0.251
Evidence of vermin	0	1.5	0.377
Family size	4.71 ± 1.51	4.47 ± 1.43	0.006
Number of rooms	5.81 ± 1.68	5.81 ± 1.75	0.590
Occupant density	0.89 ± 0.39	0.83 ± 0.37	0.392
Endotoxin concentration:**			
Living room floor	25.8 (1.84, 361)	18.7 (1.36, 257)	0.136
Mother's bedroom floor	16.1 (1.08, 240)	13.1 (0.75, 228)	0.277
Child's bedroom floor	10.9 (0.60, 198)	12.3 (0,76, 198)	0.217
Mother's bed	4.13 (0.22, 75.2)	3.46 (0.21, 56.8)	0.909
Child's bed	2.46 (0.09, 69.4)	2.63 (0.19, 37.0)	0.979

* A total of 292 homes were excluded and 966 homes were included in the analyses presented. The sample sizes for excluded and included for each comparison varied slightly due to randomly missing data. Specifically, the numbers excluded and included were: for pets 42 and 966; for cleanliness 52 and 883; urban location 292 and 963, mother's smoking 53 and 966; vermin 52 and 947; family size 285 and 925; number of rooms 53 and 966; occupant density 53 and 925; and endotoxin on the living room floor 43 and 921; mother's bedroom floor 46 and 897; child's bedroom floor (separate from mother's) 21 and 368; mother's bed 46 and 906; and child's bed (separate from mother's) 38 and 803.

** The numbers in the table are the geometric mean and 95% confidence interval of the endotoxin concentration in endotoxin units (EU). The means of the logs (S.D.) for the endotoxin concentrations included in the analyses are: living room floor 2.93±1.31, mother's bedroom floor 2.57±1.43, child's bedroom floor 2.51±1.39, mother's bed 1.24±1.40 and child's bed 0.97±1.32.

Table 2

Univariate models predicting endotoxin concentration from the living room floor

Model	Variable	Coefficient (95% CI)	P-value
1*	1 pet in home	0.21 (−0.00, 0.43)	0.055
	≥ 2 pets in home	0.44 (0.19, 0.69)	0.001
2*	1 dog in home	0.18 (−0.04, 0.41)	0.105
	≥ 2 dogs in home	0.61 (0.24, 0.99)	0.001
3*	1 cat in home	0.57 (0.27, 0.87)	0.001
	≥ 2 cats in home	0.18 (−0.17, 0.53)	0.318
4	Urban home	0.27 (0.10, 0.44)	0.002
5	Occupant density	0.44 (0.19, 0.69)	0.001
6	Cleanliness of home	−0.34 (−0.43, −0.25)	0.001
7	Mother smokes in home	0.45 (0.18, 0.73)	0.001
8	Evidence of vermin in home	0.92 (0.23, 1.61)	0.009
9	Ln (Can f 1)	0.06 (0.02, 0.09)	0.003
10	Ln (Fel d 1)	0.07 (0.03, 0.10)	0.001

*The comparison group for pets, cats or dogs is all homes with no pets in the home. There were 35 homes (33 with dogs and 4 with cats) where the pet was kept outdoors at all times and these homes were excluded from analysis.

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Table 3

Multivariable models predicting the endotoxin concentration in dust from the living room floor when variables are added to the pet variables

	Model 1 - Pets		Model 2 - Dogs		Model 3 - Cats	
Variables	**Coefficient (95% CI)**	**P-value**	**Coefficient (95% CI)**	**P-value**	**Coefficient (95% CI)**	**P-value**
1 pet in home *	0.26 (0.04, 0.49)	0.022	0.27 (0.05, 0.50)	0.018	0.50 (0.18, 0.83)	0.002
≥ 2 pets in home *	0.48 (0.22,0.74)	0.001	0.57 (0.19, 0.95)	0.004	0.26 (−0.11, 0.62)	0.169
Urban home	0.12 (−0.07, 03.30)	0.211	0.11 (−0.08, 0.31)	0.248	0.10 (0.11, 0.62)	0.346
Occupant density	0.39 (0.13, 0.65)	0.003	0.34 (0.08, 0.61)	0.011	0.37 (0.08, 0.66)	0.011
Cleanliness of home	−0.26 (−0.36, −0.16)	0.001	−0.29 (0.40, −0.19)	0.001	−0.25 (−0.36, −0.14)	0.001
Mother smokes	0.27 (−0.02, 0.57)	0.069	0.30 (−0.03, 0.62)	0.073	0.31 (−0.01, 0.64)	0.058
Evidence of vermin in home	0.41 (−0.34, 1.16)	0.286	0.28 (−0.55, 1.11)	0.507	0.57 (−0.26, 1.40)	0.178

* The reference group for pets in each model are those homes without any pets in the homes. For a pet variable considering ≥1 pet in model 1 the p-value = 0.001; for ≥1 dogs in model 2 the p-value = 0.002; and for ≥1cats in model 3 the p-value = 0.006.

Table 4

The multivariable models predicting the concentrations of endotoxin in dust from the mother's and child's bedroom floors and beds

	Model 1 - Child's Bedroom Floor		Model 2 - Mother's Bedroom Floor	
Variable	coefficient (95% CI)	P-value	coefficient (95% CI)	p-value
1 pet in home	0.21 (−0.15, 0.57)	0.261	0.12 (−0.13, 0.37)	0.339
≥ 2 pets in home	0.60 (0.20, 0.99)	0.001	0.35 (0.07, 0.63)	0.016
Urban home	0.30 (−0.00, 0.61)	0.052	0.13 (−0.07, 0.34)	0.210
Occupant density	0.58 (0.19, 0.96)	0.003	0.47 (0.16, 0.77)	0.003
Cleanliness of home	−0.33 (−0.49, −0.16)	0.001	−0.37 (−0.48, −0.26)	0.001
Mother smokes	0.16 (−0.34, 0.66)	0.526	0.20 (−0.15, 0.54)	0.263
Evidence of vermin	0.63 (−1.21, 2.48)	0.502	0.07 (−0.89, 1.03)	0.887
	Model 3 - Child's Bed		Model 4 - Mother's Bed	
Variable	coefficient (95% CI)	p-value	coefficient (95% CI)	p-value
1 pet in home	−0.06 (−0.31, 0.19)	0.642	0.08 (−0.17, 0.33)	0.522
≥ 2 pets in home	−0.01 (−0.29, 0.26)	0.918	0.21 (−0.07, 0.50)	0.135
Urban home	0.18 (−0.02, 0.39)	0.083	0.08 (−0.13, 0.28)	0.452
Occupant density	0.27 (−0.00, 0.55)	0.053	0.52 (0.24, 0.80)	0.001
Cleanliness of home	−0.13 (−0.25, −0.02)	0.023	−0.23 (−0.34, −0.12)	0.001
Mother smokes	0.40 (0.05, 0.74)	0.024	0.27 (−0.07, 0.62)	0.117
Evidence of vermin	0.24 (−0.82, 1.30)	0.659	1.02 (0.05, 1.98)	0.089

Model 1 is predicting endotoxin on the child's bedroom floor, the p-value for pets = 0.014; model 2 is predicting endotoxin on the mother's bedroom floor, the p-value for pets = 0.050; model 3 is predicting endotoxin on the child's bed, the p-value for pets = 0.897; model 4 is predicting endotoxin on the mother's bed, the p-value for pets = 0.314.