

Effect of Cat and Dog Ownership on Sensitization and Development of Asthma among Preteenage Children

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An inverse relationship has been proposed between exposure to high quantities of cat allergen at home and both asthma and cat allergy. First- and second-grade children from Luleå, Kiruna, and Piteå, Sweden participated in an asthma questionnaire study ($n = 3,431$) and incidence was evaluated over the next 3 years. Skin testing was performed on the children in Luleå and Kiruna ($n = 2,149$). The strongest risk factor for incident cases of asthma was Type 1 allergy (relative risk [RR], 4.9 [2.9–8.4]), followed by a family history of asthma (RR, 2.83 [1.8–4.5]). Living with a cat was inversely related both to having a positive skin test to cat (RR, 0.62 [0.47–0.83]) and incidence of physician-diagnosed asthma (RR, 0.49 [0.28–0.83]). This effect on incident asthma was most pronounced among the children with a family history of asthma (RR, 0.25 [0.08–0.80]). The evidence also suggests that many of the children exposed to cats at home can develop an immune response that does not include immunoglobulin E. Weaker protective trends were seen with dog ownership. The traditional thinking that not owning cats can provide protection against developing allergy and asthma among those with a family history of allergy needs to be re-evaluated. In a community where cat sensitization was strongly associated with asthma, owning a cat was protective against both prevalent and incident asthma.

Keywords: allergy; asthma; cat; dog; tolerance

An increase in asthma over the past several decades has been documented in many different parts of the world, including countries in Scandinavia (1–4). A strong association between asthma and immediate hypersensitivity to perennial allergens, including those from dust mite, cockroach, and animal dander, has been well established (5–8). Sensitization to animal dander may be particularly important to understanding the increase in asthma that has occurred in dust mite-free areas of northern Sweden (4). Because of the ease with which cat and dog allergens are passively transferred, moderate concentrations of pet allergens are ubiquitous in public buildings (9–13). In fact, it is well recognized that some cat-allergic children have never lived in a house with a cat (14). In contrast to the data for mite and cockroach (15–18), there is little evidence supporting a linear dose-response relationship between cat exposure and the prevalence of sensitization to cat. Reports have suggested that living with a cat may actually

protect against developing an allergic response to this allergen and against asthma (7, 17, 19, 20); however, this observation is not consistent (21, 22). It is also not clear whether the protective effect of having a cat in the home is conferred in the first years of life, independent of continued exposure (23). In a finding that may help explain the mechanism of the protective effect of living with a cat, it has been shown that high exposure to cat allergen can induce both immunoglobulin (Ig) G and IgG4 antibodies specific for *Fel d 1* without IgE antibodies (17).

The aim of the current study was to determine what effect living with a cat or dog has on the development of allergy and asthma. The study design included a longitudinal cohort of children living in three towns in northern Sweden, with asthma prevalence data at ages 7–8 years and 3 years of incidence data to ages 10–11 years. The focus was on the preteenage years since this is an important age in the onset of allergic asthma (24). Because of the cold, dry climate dust mites and cockroaches are rare in this area of Sweden, located at the Arctic Circle, a fact confirmed by our analysis of more than 600 dust samples taken from schools and homes (25). This makes it an ideal location to study the effects of exposure to domestic animal allergens. The prevalence of physician-diagnosed asthma among these children at age 7–8 years was 6%, with a strong correlation between asthma and allergy to cats, dogs, and birch pollen (26). Moderate to high concentrations of pet allergen were found in a majority of the dust samples from the schools of these children and also from homes without pets, confirming that persistent cat and dog allergen exposure occurs in this community even in the absence of an animal in the home (25). The present study was designed to investigate risk factors for incident asthma over a 3-year period.

METHODS

Study Population

In 1996 all of the children from the first- and second-grade classes in Luleå, Kiruna, and Piteå, Sweden (median ages, 7 and 8 years) were invited to participate in an asthma study, and of those 97% were enrolled ($n = 3,431$). The parents of the participants completed a questionnaire modeled on the International Study of Asthma and Allergies in Childhood (ISAAC) format and distributed through the schools (27). The questionnaire used in this study has been described previously along with a validation study conducted by local physicians (7, 26). The three definitions of asthma used, “ever-asthma,” “physician-diagnosed asthma,” and “wheezing in the last 12 months,” were based on questionnaire responses. A family history of asthma was defined as either a parent or sibling having asthma. Any child who lived in a house with a pet at any point during the study period (1996–1999) was considered a “current pet owner,” and before 1996 a “previous pet owner.” “Ever-pet” was a combination of these two groups.

Prevalence and Incidence of Asthma

All of the children whose parents completed a questionnaire in 1996 were used in the analysis of prevalent asthma in 1996 (7, 26). The

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TABLE 1. CUMULATIVE INCIDENT CASES OF ASTHMA AND CONDITIONS RELATED TO ASTHMA OVER 3 YEARS: PERCENTAGE OF POPULATION AT RISK

	Male	Female	All
Ever-asthma % (n)	4.7 (72)	3.4 (53)	4.1 (125)
Physician-diagnosed asthma % (n)	2.5 (39)	2.2 (34)	2.4 (73)
Wheezing in last 12 mo % (n)	9.6 (123)*	7.1 (96)	8.3 (219)
Frequent use of asthma medicines† % (n)	3.4 (54)	2.6 (40)	3.0 (94)

* There were significantly more male than female incident cases of wheezing in the last 12 months ($p = 0.023$, χ^2 test).

† Parents reported that the child used asthma medicines often or every day. Children who had ever taken asthma medicines in 1996 were excluded from the population at risk for incidence.

parents were again asked to complete questionnaires in 1997, 1998, and 1999, and 97% ($n = 3,453$), 97% ($n = 3,446$), and 97% ($n = 3,406$) participated in each year, respectively. Any child who participated in 1996 as well as at least one other year was used in the analysis of incident asthma. Children who were reported in 1996 as ever having asthma, as having a physician diagnosis of asthma, or were identified as asthmatic in the validation study were excluded from the population at risk for incident "ever-asthma" and "physician-diagnosed asthma." Children who reported ever wheezing or wheezing in the last 12 months were excluded from the population at risk for incident "wheezing in the last 12 months."

Assessment of Type 1 Allergy

In 1996 the study children in Luleå and Kiruna ($n = 2,454$) were invited to be skin tested, and of these 88% participated. Children were prick tested with a lancet on the forearm to birch, timothy, mugwort, cat, dog, horse, *Dermatophagoides pteronyssinus*, *Dermatophagoides farinae*, *Cladosporium*, and *Alternaria*, the positive control histamine (10 mg/ml), and the negative control glycerol (ALK, Hørsholm, Denmark). Mean wheal diameters were measured after 15 minutes. Type 1 allergy was defined as a positive response (≥ 3 mm) to at least one allergen.

In 2000 all of the children in Kiruna ($n = 658$) and a randomly selected one-quarter of the children in Luleå ($n = 427$) were invited to give a blood sample and of those 78% participated. IgE antibodies to cat, dog, birch, and timothy were measured by covalent allergen coupling (CAP; reagents generously provided by Pharmacia, Uppsala, Sweden). IgG and IgG4 antibodies to *Fel d 1*, the major cat antigen, were measured by radioimmunoprecipitation assay (method described previously [17, 28]; and see the online data supplement).

Statistical Analysis

Risk ratios (RRs) with 95% confidence intervals were calculated for determinants of incident asthma. The χ^2 test was used to compare sex-based differences in asthma prevalence and incidence as well as the antibody groups. Logistic regression was performed for multivariate analysis of incident cases of asthma, using the eight risk factors that were significantly associated ($p < 0.05$) with at least one classification of asthma by univariate analysis.

RESULTS

In a study over a 3-year period of more than 3,000 children, ranging in age from 7–8 to 10–11 years, the cumulative incidences of ever-asthma, physician-diagnosed asthma, and wheezing in the last 12 months were 4.1, 2.4, and 8.3%, respectively (Table 1). In general, the incidence of asthma was higher among boys than among girls, and the prevalence of physician-diagnosed asthma

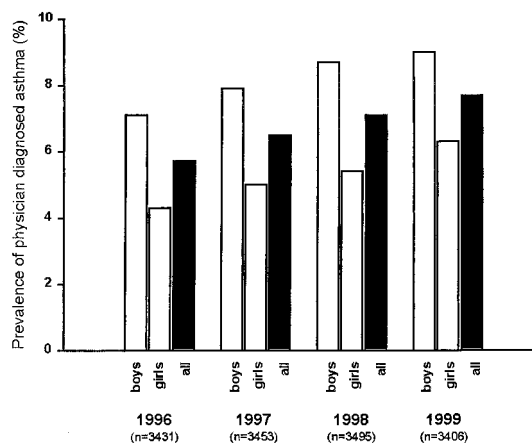


Figure 1. Prevalence of parents responding to the child ever having "physician-diagnosed asthma" reported in each year the questionnaire was distributed. There was a significantly higher proportion of subjects with asthma among males in all of the years ($p < 0.01$, χ^2 test). The prevalence of asthma was significantly higher in 1999 than in 1996 for both boys and girls ($p < 0.001$, χ^2 test).

remained significantly higher for boys over the 3 years (Figure 1). The incidence rate was similar in each of the 3 years of the study, with a slightly declining trend (Figure 2). The prevalence data from 1996 and the associated risk factors have been reported previously (7, 26).

Risk Factors for Incident Cases of Asthma

For each of the three asthma classifications, Type 1 allergy and family history of asthma were the greatest risks for incidence both in univariate (Table 2) and multivariate (Table 3) analyses. Although there is an extensive overlap in cases between the asthma groups, the variations provide subtle differences that both highlight different risk factors as well as stress those risks identified by all three definitions. For "ever-asthma," low birth weight remained an independent risk factor in multivariate analysis, but only 4.2% of the population fell into this category. Cat ownership and dog ownership were inversely related to incident

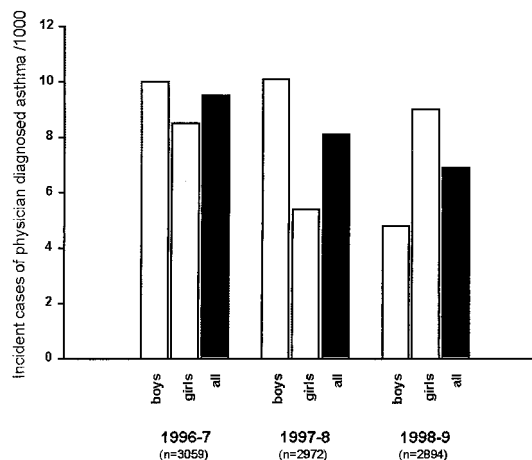


Figure 2. Incidence of "physician-diagnosed asthma" for the 3 years of the study. The differences between the sexes were not significant for any of the years. The incidence of asthma was not significantly different in the third year of the study compared with the first year ($p = 0.20$, χ^2 test).

TABLE 2. RELATIVE RISK RATIOS FOR DETERMINANTS SIGNIFICANTLY ASSOCIATED WITH AT LEAST ONE OF THE ASTHMA 3-YEAR INCIDENT GROUPS*

	Population with Risk (%)	Incident Cases		
		Ever-Asthma	Physician-diagnosed Asthma	Wheeze [†]
Male sex	51.0	1.35 (0.96–1.92)	1.14 (0.73–1.8)	1.34 (1.04–1.73)
Family history asthma	24.2	2.67 (1.89–3.77)	2.83 (1.80–4.46)	1.98 (1.52–2.57)
Birth weight < 2,500 g	4.2	2.13 (1.14–3.96)	1.41 (0.52–3.81)	0.86 (0.39–1.88)
Respiratory infection	56.7	1.98 (1.34–2.93)	1.76 (1.06–2.90)	1.42 (1.09–1.85)
Lived in a damp house	25.7	1.14 (0.77–1.69)	1.16 (0.69–1.94)	1.45 (1.09–1.92)
Cat at home ever	38.6	0.70 (0.48–1.02)	0.49 (0.28–0.83)	0.97 (0.74–1.26)
Dog at home ever	45.0	0.92 (0.65–1.30)	0.60 (0.37–0.98)	1.11 (0.86–1.43)
Type 1 allergy [‡]	20.6	4.67 (2.99–7.27)	4.92 (2.90–8.40)	3.16 (2.33–4.29)
Cat	13.4	5.93 (3.81–9.22)	7.42 (4.35–12.68)	3.50 (2.54–4.81)
Dog	8.7	5.78 (3.60–9.28)	4.04 (2.12–7.69)	3.48 (2.41–5.04)
Birch	7.9	2.01 (1.03–3.95)	3.14 (1.56–6.32)	2.81 (1.90–4.16)

Significant risk ratios are in boldface (95% confidence interval).

* Neither mother smoking nor having been breast fed for 3 months or less was significantly associated with any of the asthma groups.

[†] Incidence of wheeze in the last 12 months.

[‡] Skin test positive (≥ 3 mm) to at least one allergen tested. The risk ratios for allergy are based on the two-thirds of the population that was skin tested.

“physician-diagnosed asthma” in the univariate analysis, but this relationship was not significant in the multivariate model. However, pet ownership and Type 1 allergy are strongly inversely related (*see* the next section), and when Type 1 allergy was removed from the analysis, cat ownership was inversely associated with incidence of physician-diagnosed asthma (odds ratio [OR], 0.56; $p = 0.047$). The association with dog ownership was also negative but remained nonsignificant (OR, 0.79; $p = 0.36$). It is important to note that because only two-thirds of the children were skin tested and thus have the variable “Type 1 allergy,” these two-thirds are the population in the logistic regression analysis. Univariate analysis of the other risk factors as well as

the prevalence of symptoms both including and excluding the one-third not skin tested showed the groups to be similar.

Pet Ownership and Type 1 Allergy

There was a significant inverse relationship between ever living in a house with a cat and having a positive skin test to cat (RR, 0.62 [0.47–0.83]), an association not significant for dog (RR, 0.79 [0.58–1.1]). These inverse associations were stronger among the children with a family history of allergy (Table 4). Having a cat was also associated with a decreased prevalence of having a positive skin test to birch or dog allergens ($p < 0.001$).

TABLE 3. LOGISTIC REGRESSION EQUATIONS FOR DETERMINANTS THAT HAD SIGNIFICANT RISK RATIOS IN THE UNIVARIATE ANALYSIS*

	OR	95% CI	p Value	Without Type 1 Allergy [‡]
Incident cases of “ever-asthma”				
Type 1 allergy [†]	5.85	5.35–6.35	< 0.001	
Family history of asthma	2.61	2.10–3.11	< 0.001	
Low birth weight	3.06	2.14–3.98	0.017	
Respiratory infection	1.81	1.27–2.35	0.033	
Incident cases of physician-diagnosed asthma				
Type 1 allergy	5.52	4.93–6.11	< 0.001	
Family history of asthma	3.30	2.71–3.90	< 0.001	2.67 (1.6–4.4)
Respiratory infection	1.59	0.96–2.22	0.15	1.78 (1.1–3.0)
Ever-cat at home	0.55 [‡]	0.25–1.21	0.14	0.56 (0.31–0.99)
Ever-dog at home	1.2	0.60–1.80	0.56	0.79 (0.47–1.3)
Incident cases of wheeze in last 12 mo				
Type 1 allergy	3.56	3.17–3.94	< 0.001	
Family history of asthma	2.38	1.99–2.76	< 0.001	
Respiratory infection	1.58	1.20–1.95	0.018	
Ever lived in a damp house	1.26	0.87–1.66	0.24	
Male sex	1.27	0.90–1.63	0.20	

Definition of abbreviations: CI = confidence interval; OR = odd ratio.

Significant risk ratios are in boldface (95% CI).

* Only variables that were significantly associated with asthma by univariate analysis (Table 3) are shown, but the equations shown are for models with all eight variables considered in the equation (allergy, family history, low birth weight, respiratory infection, ever cat, ever dog, damp house, and male sex).

[†] Skin test positive (≥ 3 mm) to at least one allergen tested.

[‡] OR are shown for the model when Type 1 allergy is removed from the analysis of risk factors for incident cases of physician-diagnosed asthma.

TABLE 4. PETS AT HOME AS A NEGATIVE RISK FACTOR FOR A POSITIVE SKIN TEST* FOR THOSE WITH AND WITHOUT A PARENT OR SIBLING WITH A HISTORY OF ALLERGY†

Skin Test and Family History of Allergy		
Risk ratio for a positive skin test to cat if there has ever been a cat in the home		
Parent with allergy	Neither parent with allergy	
0.52 (0.32–0.84)	0.77 (0.53–1.1)	
Risk ratio for a positive skin test to dog if there has ever been a dog in the home		
Parent with allergy	Neither parent with allergy	
0.58 (0.37–0.91)	1.2 (0.75–1.8)	

Significant risk ratios are in boldface (95% confidence interval).

* Skin test positive (≥ 3 mm).

† A family history of allergy was defined as a positive answer to questions either about a parent or sibling having allergic symptoms (i.e., runny nose or itchy eyes).

Pet Ownership, Heredity, and Asthma

In 1996 there were significant inverse relationships between ever having a cat at home and the prevalence of “ever-asthma” (RR, 0.50 [0.35–0.70]), “physician-diagnosed asthma” (RR, 0.54 [0.38–0.76]), and “wheezing in the last 12 months” (RR, 0.71 [0.57–0.88]). Similar, although less striking, inverse correlations were found for dog ownership with the corresponding RRs of 0.69 (0.51–0.92), 0.75 (0.55–1.01), and 0.82 (0.67–1.01). As is shown in Table 5, the prevalence of asthma was highest among the children with a family history of asthma. When the children were separated into those with and without a family history of asthma, the inverse relationship with cat ownership was found to be significant only among the children with a family history of asthma. This was true for all three classifications of asthma. Interaction analysis demonstrated that the protective effect of

having a cat was not significantly stronger for the children with a family history of asthma than for children without a family history of asthma.

As shown in Table 2, having a cat or dog at home was a significant protective effect for incident cases of “physician-diagnosed asthma.” For the children with a family history of asthma, owning a cat had a significant protective effect against developing incident cases of “physician-diagnosed asthma” as well as “ever-asthma” (Table 6). The relationship remained significant for “physician-diagnosed asthma” when corrected for other risk factors in multivariate analysis. Having a dog at home indicated a similar protective effect as having a cat, but the relationship was not significant.

Serum IgE and IgG Antibody Responses

The analysis of the serum was focused on comparing the allergic children, that is, those who had made IgE to cat were compared with those who had not made IgE. The latter group was further stratified into those who had made IgG and those who had made no antibody response to cat. Of the 117 children who were living with a cat when a serum sample was collected, 6.8% had measurable IgE antibody to cat (≥ 0.35 IU of IgE per ml), whereas 48.7% had anti-*Fel d 1* IgG (≥ 125 units/ml) without IgE. Among the 586 who had never lived in a house with a cat, significantly more had IgE, 15.4% ($p = 0.015$), whereas only 9.2% had IgG without IgE ($p < 0.001$). The 116 children who had lived with a cat in the past but were not currently living with one fell in between these two groups, with 11.2% of them making IgE to cat and 21.6% making IgG without IgE. The prevalence of antibodies to *Fel d 1* of IgG4 isotype was much higher among the children living with a cat, but was not different

TABLE 5. PREVALENCE OF ASTHMA IN CHILDREN 7 AND 8 YEARS OLD (1996) RELATIVE TO A FAMILY HISTORY OF ASTHMA*: UNIVARIATE AND MULTIVARIATE ANALYSES OF THE EFFECT OF HAVING A CAT AT HOME ON DEVELOPING ASTHMA

	Ever-Asthma	Physician-diagnosed Asthma	Wheeze in Last 12 mo
Univariate			
No family history of asthma			
Cat never at home (n = 1,688)†	4.7%	4.3%	9.3%
Cat ever at home (n = 841)	3.2%	3.0%	7.7%
	RR‡ 0.68 (0.45–1.1)	0.69 (0.44–1.1)	0.83 (0.63–1.1)
Family history of asthma			
Cat never at home (n = 624)	15.9%	13.9%	22.4%
Cat ever at home (n = 180)	6.7%	7.1%	15.1%
	RR 0.42 (0.24–0.75)	0.51 (0.29–0.90)	0.67 (0.47–0.98)
Multivariate			
OR for ever having a cat at home among family history-positive children corrected in a multivariate model§	0.50 (0.28–0.89)	0.58 (0.32–1.0)	0.72 (0.46–1.1)
OR for the “cat never” by “family history” interaction term	1.2 (0.57–2.4)	1.0 (0.49–2.1)	1.1 (0.67–1.9)

Definition of abbreviations: OR = odds ratio; RR = relative risk. Significant risk ratios are in boldface (95% confidence interval).

* A family history of asthma was defined as a parent or sibling with asthma.

† The numbers of children in the exposure groups are slightly different for each asthma classification due to the absences of some responses. For physician-diagnosed asthma there were 1,672, 837, 618, and 182 children, respectively, in the four groups from top to bottom. For wheeze in the last 12 months there were 1,713, 854, 633, and 185 children.

‡ Risk ratios (RR's) calculated for the univariate analysis.

§ The odds ratio for having a cat in the home for only those children with a family history of asthma was corrected for other factors in a logistic regression model including the variables low birth weight, respiratory infection, ever dog, damp house and male gender.

|| The odds ratio for the interaction term “cat never” (the inverse of “cat ever”) by “family history of asthma” was calculated in a logistic regression model including those children with and without a family history of asthma, and the variables low birth weight, respiratory infection, ever dog, damp house, and male sex.

TABLE 6. THREE-YEAR CULULATIVE INCIDENCE OF ASTHMA (1996–1999) RELATIVE TO HAVING A FAMILY HISTORY OF ASTHMA*: UNIVARIATE AND MULTIVARIATE ANALYSES OF THE EFFECT OF HAVING A CAT AT HOME ON DEVELOPING ASTHMA

	Ever-Asthma	Physician-diagnosed Asthma	Wheeze in Last 12 mo
Univariate			
No family history of asthma			
Cat never at home (n = 1,421) [†]	3.0%	1.9%	6.6%
Cat ever at home (n = 984)	3.0%	1.4%	7.3%
	RR [‡] 0.97 (0.61–1.5)	0.75 (0.39–1.4)	1.12 (0.81–1.5)
Family history of asthma			
Cat never at home (n = 470)	9.4%	6.2%	14.3%
Cat ever at home (n = 196)	4.6%	1.5%	12.2%
	RR 0.49 (0.24–0.99)	0.25 (0.08–0.80)	0.86 (0.53–1.4)
Multivariate			
OR for ever having a cat at home among family history-positive children corrected in a multivariate model [§]	0.52 (0.24–1.1)	0.26 (0.08–0.88)	0.33 (0.41–1.3)
OR for the “cat never” by “family history” interaction term	1.75 (0.22–1.7)	2.67 (0.67–10.6)	1.39 (0.71–2.7)

Definition of abbreviations: OR = odds ratio; RR = relative risk.

Significant risk ratios are in boldface (95% confidence interval).

* A family history of asthma was defined as a parent or sibling with asthma.

[†] The numbers of children in the exposure groups are slightly different for those in the “wheeze in last 12 months” group: 1,221, 846, 384, and 163 children, respectively, from top to bottom.

[‡] Risk ratios (RR's) calculated for the univariate analysis.

[§] The odds ratio for having a cat in the home for only those children with a family history of asthma was corrected for other factors in a logistic regression model including the variables low birth weight, respiratory infection, ever dog, damp house and male gender.

^{||} The odds ratio for the interaction term “cat never” (the inverse of “cat ever”) by “family history of asthma” was calculated in a logistic regression model including those children with and without a family history of asthma, and the variables low birth weight, respiratory infection, ever dog, damp house, and male sex.

between those children also making IgE to cat and those children not making IgE to cat (see Table E1 in the online data supplement).

Close to one-quarter of the children with serum IgE to cat reported a physician diagnosis of asthma at some time during the study (i.e., 29 of 119). A significantly smaller proportion of the children who had IgG antibodies without IgE, 8.1% ($p < 0.001$), and children with no antibody response to cat, 6.1% ($p < 0.001$), reported a physician diagnosis of asthma. In a multivariate model including IgE and IgG antibodies, having IgE antibodies to cat was significantly associated with incident cases of asthma (OR, 6.36 [2.5–16.2]), whereas the OR for having IgG to *Fel d 1* was 2.38 (0.94–6.1).

DISCUSSION

In the examination of the global epidemic of asthma, understanding the role of allergy is fundamental, specifically the influence of indoor allergens. The link between asthma and allergy to indoor antigens has been well established (5–8, 15), and avoiding known risk factors is a logical tactic for preventing the onset of any disease. Because of the amount of time spent in the home, especially early in life, this has been the target of most allergen avoidance programs. For both dust mite and cockroach some success has been achieved through avoidance (29–31). Traditional thinking has also presumed that avoidance of sensitization to pet allergens can best be achieved by not keeping one in the house. Cat and dog allergens, however, are now known to behave quite differently than mite and cockroach allergens because of their airborne characteristics and transfer properties. In communities where cats and dogs are commonly kept as pets, moderate allergen exposure is present in the majority of public buildings and in most homes without a pet (9–13).

The prevalence of asthma among these children in northern

Sweden was similar to reports from other areas of Europe, although not as high as from the inner cities of the United States or Australian suburbs (22, 32–34). For both prevalence and 3-year incidence of asthma, Type 1 allergy and family history were the strongest risk factors for all definitions of asthma. In an effort to understand the onset of asthma in this population, two logical steps were to examine the exposure risks for becoming pet allergic and to compare those children with and without a family history of asthma.

Do the children exposed to the largest quantity of allergen have the greatest chance of becoming allergic? Unlike dust mite and cockroach allergens, the relationship between exposure to cat and dog allergens and sensitization is not direct. Cat and dog allergens are ubiquitous in public buildings including schools, where 82 and 97% of desk and chair samples of the children in this study had at least moderate concentrations ($\geq 1 \mu\text{g/g}$) of the major cat antigen, *Fel d 1*, and the major dog allergen, *Can f 1*, respectively (25). This moderate exposure to cat allergen appears to be sufficient for sensitization because 237 of the 287 children in this study with a positive skin test to cat (i.e., 83%) had never lived in a house with a cat (131 of 185 for dog). In fact, ever having lived with a cat significantly reduced a child's chance of being allergic to cat allergens. This effect was strongest for the children with a family history of allergy. There have been some data published in accordance with these findings, suggesting that the protective effect is more common in those with a family history of allergy (14, 35).

Although the mechanisms of the protective effect of cat ownership are not known, it is clear that many of the children living with a cat make serum IgG antibodies to the cat allergen *Fel d 1* without becoming allergic (17, 36). This includes children who are atopic to other allergens or have a family history of allergy. In the present study, 49% of the children living in a house with a cat had significant IgG antibodies to *Fel d 1* without IgE

antibodies. Among children without a cat, having IgE antibodies to cat was significantly more common (16 vs. 7%), but IgG without IgE was much less common. Furthermore, as in previous studies, IgG to *Fel d 1* without IgE antibodies was not a significant risk factor for asthma (17). This immune response to *Fel d 1* without IgE antibodies could be the mechanism of tolerance to cat allergens. However, we would stress that it does not have the features of a helper T cell type 1 (Th1) response and is better seen as a modified Th2 response. Evidence for this view comes from the finding, confirmed here, that the prevalence of IgG4 antibodies for those children with IgG antibodies to *Fel d 1* is equally common among the nonallergic children as among the allergic children (17).

In this cohort, exposure to a dog in the home had a similar protective trend for asthma as having a cat, but this effect was not as strong and was not significant when corrected for cat ownership. The few reports in the literature on the relationships between dog ownership and both sensitization and asthma are conflicting (19, 20, 23). However, among those with a family history of allergy, we found that owning a dog had a significant protective effect against sensitization to dog allergens. The finding that cat ownership was protective against the development of Type 1 allergy in general, merits further investigation into the immunological mechanism of this protective effect of cat ownership.

One possible confounder of the protective pet owner effect is that allergic parents may choose not to own pets either because of their own symptoms or as avoidance against their children developing allergies. Evidence of this trend was seen in this population, where 18% of the children with asthmatic parents lived with a cat compared with 29% without an asthmatic parent. Nevertheless, avoiding having a pet at home did not have a protective effect. In fact, among the children with a family history of asthma, sensitization to cats, prevalent cases of asthma, and incident cases of asthma were all significantly higher among those who had never owned a cat.

If owning a cat decreases the risk of sensitization, and sensitization (rather than exposure) is the primary risk factor for asthma, then we might expect that pet ownership would be too closely related to allergy and therefore not show an independent protective effect against asthma in multivariate analysis. The important finding is that pet ownership was a strong negative risk factor for incident asthma among families with a history of the disease. In this community, avoiding pet ownership does not have any beneficial effect in relation to allergic sensitization or asthma. In a population where asthma has increased and sensitization to cat allergens is the strongest risk factor for incidence of physician-diagnosed asthma (RR, 7.42 [4.4–12.7]), owning a cat is associated with a decreased risk of asthma. The results raise some challenging questions about the reasons for the increase in asthma prevalence in the population. Thus it seems unlikely that the increase in Sweden could be explained by an increase in exposure. Equally, the results do not support the hypothesis that the increase can be explained by a community-based shift from Th1 to Th2, because the IgG antibody response in children exposed to cat has the features of a modified Th2 response.

The central message is clear: avoiding owning a pet does not protect against developing asthma or allergy. For those with a hereditary risk of asthma or allergy this effect was the most pronounced. More important, it is evident that understanding the mechanisms by which children with a family history of asthma or allergy become tolerant to cats could provide important insight into the relationship between allergen exposure and asthma.

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