both genes) is involved in asthma genetics on the basis of our current knowledge. Care must be taken when interpreting association results of single genes, and when interpreting association studies with a candidate gene, other genes in the genomic neighbourhood that are possibly in linkage disequilibrium with that gene should be considered, too. This task should become much more feasible once the haplotype pattern and linkage disequilibrium 'islands' of the genome are known (6).

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Increased personal respirable endotoxin exposure with furry pets

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Key words: asthma; endotoxin; personal monitoring; pets.

We recently reported on the utility of using small portable monitors to more precisely measure particulate (1) and endotoxin (2) exposure levels and relate these to asthma control. Utilizing these personal monitors to measure exposure, at the same time monitoring pet contact, we report on the consequences of pet ownership and animal play on endotoxin exposure in children with asthma.

Children who live with pets are exposed to higher endotoxin levels independent of pet play.

Ten schoolchildren aged 6–13 years were monitored over a 1-month period in January–February 2000. Personal monitoring was performed as previously described (1). Ethical and scientific approval for this study was obtained from the National Jewish Center and RTI International's Institutional Review Boards before initiation of recruitment.

Families completed a questionnaire before the study began describing demographic characteristics including pet ownership. During the monitoring period, children also filled out questionnaires describing whether and how long they had played with cats or dogs. Filters were weighed to quantify $PM_{2.5}$ before and after monitoring following the procedure of Lawless and Rodes (3). Endotoxin measurements and statistical analysis was performed as previously described (1, 4).

The median personal endotoxin exposure level was 0.06 EU/m^3 [25th–75th interquartile range (IQR) = 0.10 EU/m^3]. The median personal $PM_{2.5}$ exposure level was 12.54 μg/m^3 (IQR = 18.64 μg/m^3). There was no significant correlation between endotoxin and $PM_{2.5}$ exposures ($r = -0.103$, $P = 0.36$). Four households reported owning and living with furry pets, three with dogs and one with a cat (all indoors). Two children without pets reported playing with furry animals on single days and a third reported animal play on 2 days during the personal monitoring interval. Estimated playtime ranged from 10 to 30 min.

Figure 1 summarizes the relationship between furry animal ownership and/or play and personal endotoxin exposure levels. Children living in a household with dogs or a cat were exposed to significantly higher levels of endotoxin compared with those not owning pets (median 0.13 *vs* 0.04 EU/m^3, $P = 0.02$). In this comparison no change in overall $PM_{2.5}$ exposure was observed. In children not owning pets, there was a trend ($P = 0.08$) towards higher exposure on days that they reported playing with furry animals (median 0.07 *vs* 0.04 EU/m^3) although $PM_{2.5}$ exposure did not change. There was no further increase in endotoxin exposure levels on days when children living with pets reported playing with them compared with days when they did not (median 0.13 *vs* 0.13 EU/m^3, $P = 0.61$).

There was a trend towards higher exposures if nonpet owning children played with a furry animal on a particular day. However, if one lived in a household with dogs or cats, exposures were similar whether or not children reported playing with pets suggesting that pet-containing households carry a near-maximum exposure load. This relationship was not observed with total $PM_{2.5}$, confirming that the association was as a

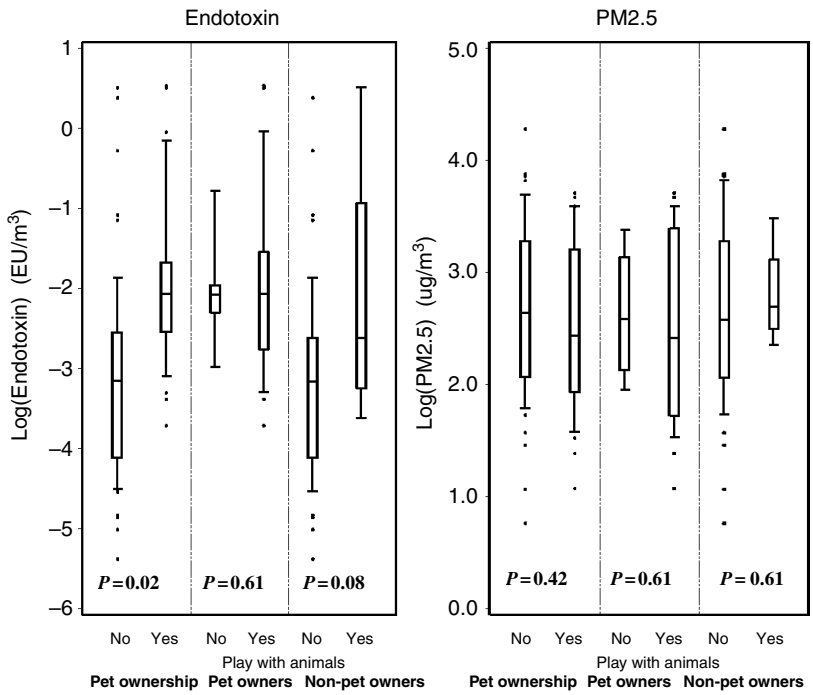

Figure 1. Illustrates personal endotoxin exposures as they relate to pet ownership or play with dogs or cats. The bottom and top edges of the box are located at the sample 25th and 75th percentiles. The center horizontal line is drawn at the 50th percentile (median). The bottom and top end of the whiskers show the 10th and 90th percentiles of the sample.

result of endotoxin concentrations (or a bioactive factor highly correlated with endotoxin) and not to increased overall particulate exposures. The observation of increased exposure among children living with pets independent of playing with them suggests that exposures are related to behaviors that are more likely to increase the endotoxin 'personal cloud', but are not necessarily associated with direct pet contact *per se*. Examples would include the re-suspension of previously settled endotoxin-rich particles into an individual's breathing zone with running on a carpet, or close proximity to surfaces where these particles have settled (1). In this context, environmental interventions to decrease endotoxin exposure by decreasing actual pet contact may not be beneficial without totally removing the pet from the home environment. However, limiting pet contact outside the home and decreasing endotoxin levels by removing pets may benefit children with asthma not only by decreasing allergen levels, but by decreasing endotoxin exposure as well.

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