I, Stephenロック, hereby submit this original work as part of the requirements for the degree of Master of Science in Epidemiology (Environmental Health).

It is entitled:
The Association Between Childhood Traffic Exhaust Exposure and Asthma Differs Between Normal and Overweight Children

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This work and its defense approved by:

Committee chair: Linda Levin, PhD
Committee member: David Bernstein, MD
Committee member: Patrick Ryan, PhD
The Association Between Childhood Traffic Exhaust Exposure and Asthma Differs Between Normal and Overweight Children

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Graduate School
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Master of Science

in Epidemiology,
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Committee Chair: Linda Levin, Ph.D.
ABSTRACT

Objective: Examine the association between diesel exhaust particles and asthma development in normal and overweight children.

Study Design: Newborn children (n = 592) were monitored for exposure to diesel exhaust particles from birth to age seven. Clinical examinations were performed at age seven to determine the presence of asthma based on the American Thoracic Society diagnostic criteria. Weight assessments were performed at age seven and children were categorized as either normal or overweight based on their body mass index (BMI) and waist circumference.

Results: There was a significant association between average lifetime diesel exhaust particle exposure and asthma diagnosis at seven for the entire cohort (aOR 1.82, 95% CI 1.02 – 3.40). When the cohort was stratified by their BMI status, exposure to DEP was significantly associated with asthma in children who were normal (aOR 2.16, 95% CI 1.05 – 4.45) but not overweight (aOR 1.87, 95% CI 0.62 – 5.69). Exposure to diesel exhaust particles was not significant for either normal or overweight children when classified by waist circumference.

Conclusion: The results of this study support the link between diesel exhaust particle exposure and childhood asthma. The strength of this association at age seven appears to depend on childhood weight status.
ACKNOWLEDGMENTS

I would like to recognize the substantial effort of my mentors and thesis committee Linda Levin, PhD, Patrick Ryan, PhD, David Bernstein, MD, and Grace LeMasters, PhD, for their unwavering support throughout this process.
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INTRODUCTION

Childhood asthma is a major public health problem in the United States and the world due to its chronic and often debilitating effects on quality of life. Health deficits associated with asthma include persistent wheezing, coughing, dyspnea, and chest tightness.\(^1\) The World Health Organization reported the prevalence of childhood asthma to be between 3% and 20%, affecting a disproportionate number of socioeconomically disadvantaged patients.\(^2\) Additionally, the recent decline in the death rates due to asthma was significant in every demographic group except children.\(^3\)

The Centers for Disease Control and Prevention reported the prevalence of asthma reached as high as 8.2% in 2009, including 7.1 million children in the general population.\(^4\) The impact of asthma on development and quality of life in children is substantial. The National Health Statistics Reports stated childhood asthma accounted for 10.5 million missed school days in 2008 and 6.7 million visits to private physician offices in 2007.\(^4\) Coping with this disease has also placed substantial economic burdens on individual patients and society; the estimated annual direct cost for asthma was $1,004.60 for children in 2004.\(^5\) Another study estimated the incremental cost of asthma to society to be $56 billion, including nearly $6 billion in productivity losses associated with asthma morbidity and mortality.\(^6\) Some studies have reported long-term respiratory deficits during adolescence and adulthood are associated with childhood asthma.\(^7,8\) Additionally, several longitudinal studies of adults concluded that asthma is associated with accelerated loss of lung function over time.\(^9,10\) Prevention of asthma during early childhood is important because of this evidence suggesting increased morbidity over the course of life.

There are many known environmental risk factors associated with asthma development including exposure to house dust mites, cockroaches, respiratory syncytial virus (RSV), and environmental tobacco smoke.\(^11\) Exposure to traffic pollution has received considerable attention in recent years as a risk factor for childhood asthma incidence. Particulate matter (PM) is a key component of traffic pollution, and is classified into coarse, fine, or ultrafine particles.\(^12,13\) Recent studies have reported a significant association between exposure to PM\(_{10}\) (fine) and PM\(_{2.5}\) (coarse) and asthma incidence.\(^14,15\) Diesel exhaust particles (DEP) make up as substantial portion of airborne PM from vehicles.\(^16,17\) Several studies have demonstrated the association between DEP and increased inflammatory cells and airway resistance, as well as enhanced production of IgE and Th2 cytokines (IL-4, IL-5, IL-10, and IL-13).\(^16,18,19,20,21,22,23\)

Childhood obesity has also been linked to incident asthma.\(^24,25\) From 2000 to 2008, the prevalence of obesity had increased form 14.8 to 19.3% in boys and 14.8% to 16.8% in girls.\(^26\)
One potential cause of obesity is food insecurity, which is characterized by families who have experienced an inability to acquire enough food due to insufficient resources. Food insecurity affects a disproportionate number of low-income and minority groups, and has been associated with risk of obesity. Unfortunately, traffic and road density are also much higher in minority and poor communities. Both obesity and traffic exposures result in increased inflammation, though few studies have examined both of these.

The purpose of this study is to determine if the relationship between exposure to traffic pollution and development of asthma varies between normal and overweight/obese children. To our knowledge, this is the first study to examine differences in the association of asthma incidence and exposure to traffic related particles between normal and overweight children. The central hypothesis of this study is that exposure to traffic related air pollution, specifically DEP, during early childhood is significantly associated with asthma at age seven, and that this association is modified by weight.

**METHODS**

**Study Population**

The Cincinnati Childhood Allergy and Air Pollution Study (CCAAPS) is an at-risk birth cohort recruited from the greater Cincinnati metropolitan area. Newborns were identified from 2001-2003 from birth certificate records. Eligibility requirements included residential proximity to major roads (defined as > 1000 trucks daily), subsequent parental responses to allergy symptom questionnaires (ASQ), and skin prick test (SPT) results of parents to 15 aeroallergens. Participants were identified by geocoding residential addresses, and those with residences less than 400 m or greater than 1500 m from a major road were further considered for inclusion. The ASQ was used to identify parents more likely to be clinically allergic, and infants were enrolled if at least one parent was determined to be atopic following a positive SPT. Clinical examinations of the children were completed at ages one, two, three, four, and seven. An at home visit was completed at one year of age for an indoor environmental assessment including mold and allergen exposure. Children who completed a clinical visit at age seven were available for this analysis (n=592).

**Asthma Outcome**

The clinical criteria for asthma diagnosis at seven years of age for children enrolled in the CCAAPS cohort were previously described. Spirometry testing by trained technicians was performed according to the American Thoracic Society criteria. Children with previous asthma...
symptoms, exhaled nitric oxide concentration > 10 ppb, or a predicted FEV₁ less than 90% and/or an FEV₁ to FVC ratio less than the lower limit of normal were further tested. Children with 12% increase in FEV₁ following treatment with 2.5 mg of levalbuterol underwent methacholine challenge during a later visit. A child was classified as asthmatic if he/she met both of the following criteria. The child’s caregiver had to report at least one of the following asthma symptoms: a tight or clogged chest or throat in the past 12 months, difficulty breathing or wheezy after exercise, wheezing or whistling in the chest in the previous 12 months, or a previous physician diagnosis of asthma. The second criterion was demonstration of airway reversibility (defined as ≥12% increase in FEV₁ after bronchodilatiton) or a positive methacholine challenge test result (defined as ≥20% decrease from saline control baseline FEV₁ at an inhaled methacholine concentration of ≤4 mg/mL).

**DEP Exposure Assessment**

A land-use regression (LUR) model was used to estimate DEP exposure in the CCAAPS cohort ($r^2 = 0.73$), which was previously described by Ryan and colleagues. The personal elemental carbon attributable to traffic (ECAT) was used to estimate individual DEP exposure for each child. The average daily ECAT was derived by sampling ambient air PM$_{2.5}$ at 27 different sites from December 2001 through December 2006 in the Cincinnati metropolitan area. The ECAT values at each of the monitoring stations was determined using multivariable UNMIX and chemical mass balance models. A land-use regression equation was developed to estimate ECAT for each location a child spent more than eight hours/week for each individual year of the study. The final model used to estimate ECAT for each child in this study contained the following independent variables: elevation of residence, truck traffic intensity within 400 m, and length of bus routes within 100 m. The average DEP exposure over seven years was equivalent to the average of all annual ECAT values for each child at ages one, two, three, four, and seven.

**Weight Assessment**

There is debate in the literature on the best obesity anthropometric measure of obesity to predict incident asthma. Body mass index (BMI) is the most common measure used to assess weight status. However, Musaad and colleagues found that measures of central obesity (waist circumference, conicity index, waist-to-height ratio) were the best predictors of childhood asthma. Therefore, both BMI and waist circumference were used in this study in conjunction with exposure to DEP. Children enrolled in the study were evaluated during the seven year
clinical visit. The height (in), weight (lb) and waist circumference (in) was taken for each child. Each measure was converted to the International System of Units of meters (m), kilograms (kg), and centimeters (cm), respectively. The BMI was calculated for each child according to the CDC recommended formula of weight (kg)/height\(^2\) (m\(^2\)). Each child was assigned to be either normal or overweight after stratifying by gender and age. Children at or above the 85\(^{th}\) percentile after stratification were classified as overweight. This process was repeated for waist circumference with cutoffs derived from curves published by Musaad et al.\(^{39}\)

**Statistical Analysis**

Two sets of obese/normal weight children (four weight groups) were identified from cut-points determined separately by BMI and waist circumference measurements. Stratified logistic regression analyses were performed for each weight group, in which average DEP exposures measured across ages one through seven, were assessed with respect to asthma prediction. Exposure to DEP was dichotomized into high and low (defined as > 0.42 ug/m\(^3\) and < 0.42 ug/m\(^3\), respectively) due to the skewedness of the distribution of exposure in the study population. Demographic characteristics, and covariates that were previously identified to be possible modifiers of asthma development in children, were evaluated for inclusion in multiple logistic regression models by backwards elimination. Possible covariates included: gender, race, maternal education (categorized as non high school graduate, high school graduate, some college/college graduate) parental asthma (yes/no), dogs in the home during prenatal period (yes/no), cats in the home during prenatal period (yes/no), breastfeeding at 12 months (yes/no), secondhand smoke exposure (yes/no), visible mold in the home (yes/no), daycare attendance prior to 12 months old (yes/no). The DEP exposure was constrained to be included in the regression models at each stage of reduction. Variable were retained in the model at a significance level of 0.1.

**RESULTS**

**Prevalence of Asthma**

There were 762 children originally enrolled in the study and 592 children who completed the year seven clinical visit (Table 1). The differences between this population and the original CCAAPS cohort were previously described (Table 2).\(^{40}\) As shown in Table 1, there were 95
children (16.1%) who received a positive asthma diagnosis. Of all asthmatics, 32 (33.7%) were exposed to high levels of DEP compared to 63 asthmatics (66.3%) that were exposed to less than 0.42 µg/m². According to the BMI measure, there were an elevated percentage of children with asthma in the overweight (27.3%) relative to the normal weight stratum (12.6%). This difference was determined to be statistically significant (p < 0.0001). There was not a statistically significant difference in the number of asthmatics between the waist circumference strata. Although the number of asthmatics was similar in both overweight strata, there were more children considered to be overweight by the waist circumference definition (n = 258) compared to BMI (n = 139).

Of the 592 children in the cohort, 132 (22.3%) were exposed to greater than 0.42 µg/m² DEP, classifying them as high exposure (Table 1). There were 86 and 83 children who were exposed to high DEP in the normal weight stratum according to BMI and waist circumference, respectively. Conversely, of children who were stratified as overweight according to BMI and waist circumference, there were 36 and 59 children who were exposed to high DEP, respectively. According to the BMI measure, there were eleven (11.6%) asthmatics classified as overweight and simultaneously exposed to high DEP. Based on the waist circumference measure, there were 12 (12.6%) children with asthma who were considered overweight and exposed to high DEP. Alternatively, there were 37 (38.9%) asthmatics who were considered normal BMI and low DEP. A total of 33 (34.7%) asthmatics were considered to have normal waist circumference and exposed to low DEP.

There were notable differences in the classification of weight status between the two measurements. Of the 453 children categorized as having normal BMI, 160 (35.3%) were considered overweight according to their waist circumference. Additionally, 41 (29.5%) children were considered to be normal weight based on their waist circumference, but were overweight in their BMI measurement. There were 293 children categorized as normal and 98 as overweight according to both measurements of weight status.

**Univariate Evaluation of Primary Exposure and Asthma at Age Seven**

High exposure to DEP was a significant predictor of asthma at age seven univariately (uaOR 1.68 95% CI 1.04 – 2.72) in the total cohort. Overall, BMI was a significant predictor of asthma in the total cohort (uaOR = 2.61, 95% CI 1.64 – 4.16), but waist circumference was not (uaOR 1.09, 95% CI 0.70 - 1.69). However, this association varied by weight status. Exposure to high DEP in the normal weight stratum for BMI (uaOR 1.95 95% CI 1.08 – 3.53) and waist circumference (uaOR 1.96 95% CI 1.05 – 3.68) was a significant predictor of asthma. High DEP
exposure was not a significant predictor of asthma in the overweight strata defined by BMI (uaOR 1.24 95% CI 0.54 – 2.85) and waist circumference (uaOR 1.38 95% CI 0.66 – 2.90).

Multivariate Evaluation of Predictors and Asthma at Age Seven

The results of the multivariate analysis are shown in Table 3. In order to obtain the best model for predicting asthma, the following variables were included for the logistic regression: gender, race, visible mold in home at one year, cats in the home during the prenatal period, breastfeeding prior to one year of age, secondhand smoke exposure, parental asthma, maternal education, dogs in the home during the prenatal period, daycare attendance prior to one year of age, and DEP exposure. Inclusion in the initial model required a variable to be univariately significant in predicting asthma in this study or significant in previous models with the CCAAPS cohort to predict childhood wheeze.

As shown in Table 3, gender, race, visible mold in the home at one year, cats during the prenatal period, breastfeeding at age one, and secondhand smoke exposure (ETS) were not significantly associated with asthma at age seven. Several covariates were significantly associated with asthma. Parental asthma (aOR 2.22, 95% CI 1.29 – 3.90) and daycare attendance at age one (aOR 2.52, 95% CI 1.37 – 4.66) were strongly predictive of the asthma outcome. Additionally, both maternal education (aOR 0.41, 95% CI 0.26 – 0.61) and dog ownership during the prenatal period (aOR 0.51, 95% CI 0.28 – 0.93) were significantly protective against development of the disease.

The primary exposure of DEP for this study was determined to be significantly associated with asthma at age seven in the total cohort (aOR 1.82, 95% CI 1.02 – 3.40).

Multivariate Results for BMI Strata

As shown in Table 3, all variables determined to be nonsignificant for the total cohort were also removed from the model for both BMI groups. Gender, race, visible mold in the home at one year, cats during the prenatal period, breastfeeding at age one, and secondhand smoke exposure (ETS) were not significantly associated with asthma in either the overweight or normal BMI strata. Surprisingly, parental asthma was not associated with childhood asthma in the normal weight group, but was significantly associated with asthma in the overweight group (aOR 3.87, 95% CI 1.48 – 10.10). Maternal education was significantly protective for both overweight and normal children, however dog ownership was only protective in the normal stratum (aOR 0.52, 95% CI 0.25 – 1.07). Daycare attendance at age one was significantly predictive of
asthma in children who were normal weight (aOR 2.37, 95% CI 1.13 – 4.97), but then removed from the model in the overweight group.

The association between average DEP exposure and asthma at age seven was stronger in the normal weight group (aOR 2.16, 95% CI 1.05 – 4.45) than the total cohort. Contrastingly, DEP exposure was associated with asthma in the overweight stratum (aOR 1.87, 95% CI 0.62 – 5.69), but was not statistically significant.

**Multivariate Results for Waist Circumference Strata**

The results for the multivariate analysis for the waist circumference groups are also shown in Table 3. Gender, visible mold in the home at one year, cats during the prenatal period, and breastfeeding at age one were not significantly associated with asthma in either the normal or overweight groups. African American race (aOR 2.93, 95% CI 1.22 – 7.07) and secondhand smoke exposure (aOR 2.64, 95% CI 1.17 – 5.97) were determined to be significantly associated with asthma in children who were overweight based on their waist circumference. This was the only stratum (including the entire cohort) for which these covariates were not removed from the model. Parental asthma was determined to be significantly associated with asthma in both normal and overweight children. Similar to the results reported above, maternal education (aOR 0.38, 95% CI 0.20 – 0.70) and dogs in the home during the prenatal period (aOR 0.48, 95% CI 0.22 – 1.09) were significantly protective against asthma in the normal but not the overweight stratum. Daycare attendance at age one was significantly predictive of asthma only in the normal weight group.

Average exposure to DEP was associated with asthma in both the weight groups, but neither result was statistically significant.

**DISCUSSION**

To our knowledge, this is the first study to measure the association between diesel exhaust exposure and asthma incidence in normal versus overweight children. There were significant associations between asthma at age seven and average DEP exposure over the course of life. However, the significance of this association varied between weight divisions.
This is the first report of stronger association between incident asthma and average DEP exposure in children who are normal weight. This result supports the hypothesis of this study that there would be a difference in the association between DEP and asthma depending on weight division.

In studies of O₃ exposure, animal models have indicated that greater changes in airway responsiveness, O₃-induced injury, and inflammation occur in obese mice compared to lean controls. Although this study did not measure exposure to environmental O₃, which is a secondary product of traffic pollution, the results of this study do not indicate a protective effect of weight status on DEP exposure. Rather, the diminished impact of pollution on asthma in overweight children is likely due to the association between weight status and asthma. The oxidative stress caused by traffic pollutants induces production of cytokines, which have pro-inflammatory effects on pulmonary tissue. Obesity related asthma also causes cytokine production, including the secretion of TNF-α, Interleukin-6 and other pro-inflammatory adipokines. Weight status provides a more constant level of exposure compared to DEP, which is dependent on a series of variables including location, climate, etc. Therefore, the pro-inflammatory mechanisms induced by weight status may be active for greater periods of time than DEP related inflammation. Obesity related inflammation of pulmonary tissue may also be more severe relative to effects caused by DEP. In conjunction, the presence of inflammation due to increased serum concentrations of adipokines and the more constant pro-inflammatory process in overweight children may explain the diminished association between DEP and incident asthma in this study.

There are several notable secondary findings in this study. Maternal education and dogs in the home during prenatal period were significantly protective against asthma diagnosis at seven. Maternal education was significant for each stratum except for overweight based on waist circumference, and dogs were borderline significant in both normal weight strata. One study demonstrated that asthma was more common in children with mothers with less than a high school education, which is consistent with the results in this study (Table 3). The diminished significance of dog ownership is likely due to loss of power after the cohort was stratified in the normal weight groups, however there appeared to be no protective benefit from dogs in the overweight strata. The protective effect of dog ownership in the total cohort is consistent with previous reports of decreased risk of eczema at age four. The decreased allergen sensitization associated with dog ownership may be protective against asthma development later in life in the CCAAPS cohort.
Parental asthma and daycare attendance prior to one year of age were both significant risk factors for childhood asthma in the total cohort. Parental asthma was also significantly associated with childhood asthma in the overweight BMI and normal weight waist circumference strata. These results are consistent with previous studies, but the reason for the variation between strata is less clear. Daycare attendance during early childhood has been reported as both protective and associated with increased risk of childhood asthma. The results of this study suggest day care attendance, perhaps a surrogate for respiratory infections, prior to one year increase risk of asthma development at age seven. However, this association was not seen in children who were overweight by both definitions. A nearly equal proportion of all strata attended daycare prior to one year of age, but children who are normal weight appear to be at greater risk for asthma if they attended daycare compared to those who are overweight. This is likely due to the same dominance of obesity related pro-inflammatory mechanisms that diminished the association of asthma with DEP exposure.

Several variables were not significant for asthma in the whole cohort and most of the subsequent strata. Although female gender has been linked to more severe respiratory deficits associated with allergic sensitization relative to males during adulthood, the opposite is typically the case in children. Gender was not a significant covariate in any model used in this study. Breastfeeding prior to one year of age was not significantly protective against asthma in this study. This result was consistent with a previous case-control analysis of persistent and transient cases of childhood asthma, which did not find a significant association with breastfeeding. African American race and secondhand smoke exposure are known risk factors for childhood asthma.

Environmental moldiness has been linked to risk of developing asthma in several studies. However, one study defined asthma risk using an Asthma Predictive Index, rather than strict American Thoracic Society criteria listed above. The other found significant risk of asthma (aOR 2.6, 95% CI 1.10 – 6.26) using the same criteria for asthma diagnosis, but also included a more quantitative assessment of mold using the Environmental Relative Moldiness Index. For this study, we categorized a subject as having mold exposure if there was visible mold in the residence during the one year visit. This may explain the lack of significance of visible mold in this study. Finally, cat ownership during early childhood was demonstrated to increase risk of allergic sensitization. Although the inflammatory mechanisms associated with allergic sensitization increase the risk of asthma development, there was not a direct association between cat ownership and asthma development in this study.
The significant association between overweight status according to BMI and asthma in this study was consistent with the results of a meta-analysis of seven prospective studies.\textsuperscript{51} Waist circumference was the only significant unadjusted anthropometric predictor of asthma reported by Musaad \textit{et al}.\textsuperscript{39} However, using overweight cutoffs derived from the waist circumference curves did not yield the same results in this study. This discrepancy may be due to the stricter definition of the asthma outcome in this study, including demonstration of airway reversibility or positive methacholine challenge test. Additionally, this study assigned overweight cutoffs based on the curves provided by Musaad \textit{et al},\textsuperscript{39} which may not be as reliable of a measure for children in this cohort.

There was a significant association between average lifetime DEP exposure and asthma at age seven for the overall cohort. This is consistent with results previously reported by McConnell \textit{et al}\textsuperscript{14} and Gehring \textit{et al},\textsuperscript{15} who reported associations of 1.51 and 1.28, respectively. However, the association between asthma and DEP exposure was greater in this study, which may be due to different methods of measuring pollution exposure. McConnell and colleagues did not use a land-use regression model to estimate pollution exposure. Instead, the group used a line source dispersion model, and included distance to roadways, number of vehicles, NO\textsubscript{x} emission, wind direction and speed, and elevation of mixing layer in their exposure estimates. The variables included in their model are notably different than the ones reported in this study. Gehring and colleagues also used a land-use regression model to estimate exposure of each subject, however their primary exposure was PM\textsubscript{2.5} rather than total elemental carbon attributable to traffic. Additionally, there are substantial differences in the definition of asthma between McConnell \textit{et al} and this study. McConnell and colleagues categorized children as asthmatics if they responded to having physician diagnosed asthma on a questionnaire. The strict clinical criteria performed on each subject enrolled in both this study and the work performed by Gehring and colleagues is likely a more reliable measure of asthma, and may account for some of the variation.

Finally, a recent study reported a significant association between traffic density exposure and attained BMI.\textsuperscript{52} There was no association between average DEP exposure and overweight BMI or waist circumference at age seven. Additionally, neither of the interactions between DEP exposure and the overweight strata was significant. The lack of significant association between DEP and overweight status in this study compared to the results reported by Jerrett \textit{et al} \textsuperscript{51} could be due to major differences in age of the two cohorts. Jerrett and colleagues reported an attained BMI at age 18. In addition to a longer period of exposure in their cohort, BMI status in a post-adolescent child is not necessarily comparable to our measures at age seven, which
further emphasizes the need to determine the most generalizable anthropometric measure of weight status.

The clinical examinations performed by trained professionals also contributed to the study’s reliability. The asthma outcome assigned to each member of this study did not rely on questionnaires, but rather clinical data gathered by study collaborators, which provided additional reliability for the outcome measure.

The limitations of this study include our estimates of DEP exposure over the course of each subject’s life and limited power after stratification. Although the land-use regression model has a reported $R^2 = 0.73$, the estimates are subject to reporter bias because parental report of weekly hours spent inside and away from the residence. Additionally, the number of children characterized as overweight by both measures is significantly smaller than the normal weight groups likely reducing the power of the study.

In conclusion, the results of this study support the link between DEP exposure and childhood asthma. The association between traffic pollution and asthma is diminished in children who are overweight, which may be due to the inflammatory process caused by obesity related cytokines. When considered concurrently with DEP exposure, BMI appears to be the best predictor of childhood asthma when compared to waist circumference.
Table 1. Demographics table of primary outcome (asthma) and exposure (diesel exhaust particles) variable of cohort and respective strata.

<table>
<thead>
<tr>
<th></th>
<th>BMI</th>
<th>Waist circumference</th>
<th></th>
<th></th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>Overweight</td>
<td>Normal</td>
<td>Overweight</td>
<td>Total</td>
</tr>
<tr>
<td>Asthmatics n (%)</td>
<td>57 (12.6)</td>
<td>38 (27.3)</td>
<td>52 (15.6)</td>
<td>43 (16.7)</td>
<td>95 (16.1)</td>
</tr>
<tr>
<td>DEP &gt; 0.423 µg/m² n (%)</td>
<td>106 (23.4)</td>
<td>36 (25.9)</td>
<td>83 (24.9)</td>
<td>59 (22.9)</td>
<td>132 (22.3)</td>
</tr>
</tbody>
</table>
Table 2. Description of total study population and individual strata

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Total</th>
<th>Normal Weight</th>
<th>Overweight</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 592</td>
<td>N = 453</td>
<td>N = 139</td>
</tr>
<tr>
<td>BMI</td>
<td>N = 139</td>
<td>N = 139</td>
<td>N = 139</td>
</tr>
<tr>
<td>Waist Circumference</td>
<td>N = 258</td>
<td>N = 258</td>
<td>N = 258</td>
</tr>
<tr>
<td>Gender n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>female</td>
<td>266 (44.9)</td>
<td>213 (47.0)</td>
<td>125 (37.4)</td>
</tr>
<tr>
<td>male</td>
<td>326 (55.1)</td>
<td>240 (53.0)</td>
<td>209 (62.6)</td>
</tr>
<tr>
<td>Race n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>466 (78.7)</td>
<td>368 (81.2)</td>
<td>260 (77.8)</td>
</tr>
<tr>
<td>African American</td>
<td>126 (21.3)</td>
<td>85 (18.8)</td>
<td>74 (22.2)</td>
</tr>
<tr>
<td>Parental Asthma n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>240 (40.5)</td>
<td>179 (39.5)</td>
<td>125 (37.4)</td>
</tr>
<tr>
<td>No</td>
<td>352 (59.5)</td>
<td>274 (60.5)</td>
<td>209 (62.6)</td>
</tr>
<tr>
<td>ETS n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>175 (30.4)</td>
<td>114 (26.1)</td>
<td>89 (26.7)</td>
</tr>
<tr>
<td>Absent</td>
<td>400 (69.6)</td>
<td>322 (73.9)</td>
<td>245 (73.3)</td>
</tr>
<tr>
<td>Breastfed prior to 12 months n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>378 (63.9)</td>
<td>307 (67.8)</td>
<td>217 (65.0)</td>
</tr>
<tr>
<td>No</td>
<td>214 (36.1)</td>
<td>146 (32.2)</td>
<td>117 (35.0)</td>
</tr>
<tr>
<td>Daycare at 12 months n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>102 (18.3)</td>
<td>79 (18.3)</td>
<td>65 (20.4)</td>
</tr>
<tr>
<td>No</td>
<td>457 (81.7)</td>
<td>352 (81.7)</td>
<td>254 (79.6)</td>
</tr>
<tr>
<td>Visible Mold in Home n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>215 (40.5)</td>
<td>239 (58.6)</td>
<td>118 (39.2)</td>
</tr>
<tr>
<td>No</td>
<td>317 (59.5)</td>
<td>169 (41.4)</td>
<td>183 (60.8)</td>
</tr>
<tr>
<td>Maternal Education n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non High School</td>
<td>31 (5.4)</td>
<td>19 (4.4)</td>
<td>17 (5.3)</td>
</tr>
<tr>
<td>High School Grad</td>
<td>98 (17.1)</td>
<td>60 (13.7)</td>
<td>61 (18.9)</td>
</tr>
<tr>
<td>Some college/grad</td>
<td>443 (77.5)</td>
<td>358 (81.9)</td>
<td>244 (75.8)</td>
</tr>
<tr>
<td>Prenatal Dog(s) n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>220 (40.2)</td>
<td>174 (41.4)</td>
<td>125 (40.1)</td>
</tr>
<tr>
<td>No</td>
<td>328 (59.8)</td>
<td>246 (58.6)</td>
<td>187 (59.9)</td>
</tr>
<tr>
<td>Prenatal Cat(s) n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>148 (27.2)</td>
<td>118 (28.4)</td>
<td>81 (26.5)</td>
</tr>
<tr>
<td>No</td>
<td>397 (72.8)</td>
<td>297 (71.6)</td>
<td>225 (73.5)</td>
</tr>
<tr>
<td>Mean Height (s.d.)</td>
<td>124.0 (7.17)</td>
<td>123.5 (7.40)</td>
<td>122.6 (5.59)</td>
</tr>
</tbody>
</table>
Table 3. Adjusted odds ratios and 95% confidence intervals for predictor variables in total cohort and individual strata.

<table>
<thead>
<tr>
<th>Variable</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td>--</td>
</tr>
<tr>
<td>African American Race</td>
<td>--</td>
</tr>
<tr>
<td>Visible Mold in Home</td>
<td>--</td>
</tr>
<tr>
<td>Cats in Home Prenatal</td>
<td>--</td>
</tr>
<tr>
<td>Breastfed at one year</td>
<td>--</td>
</tr>
<tr>
<td>ETS</td>
<td>--</td>
</tr>
<tr>
<td>Parental Asthma</td>
<td>2.22 (1.29 - 3.90)*</td>
</tr>
<tr>
<td>Maternal Education</td>
<td>0.41 (0.26 - 0.64)*</td>
</tr>
<tr>
<td>Dogs in Home Prenatal</td>
<td>0.51 (0.28 - 0.93)*</td>
</tr>
<tr>
<td>Daycare</td>
<td>2.52 (1.37 - 4.66)*</td>
</tr>
<tr>
<td>DEP high vs. low</td>
<td>1.82 (1.02 - 3.40)*</td>
</tr>
</tbody>
</table>

“--” indicates the variable was not a significant predictor of asthma and removed from the model.

* Variable were retained in the model at a significance level of 0.1
REFERENCES


