Being overweight increases susceptibility to indoor pollutants among urban children with asthma

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Abstract

Background—Both being overweight and exposure to indoor pollutants, which have been associated with worse health of asthmatic patients, are common in urban minority populations. Whether being overweight is a risk factor for the effects of indoor pollutant exposure on asthma health is unknown.

Objectives—We sought to examine the effect of weight on the relationship between indoor pollutant exposure and asthma health in urban minority children.

Methods—One hundred forty-eight children (age, 5–17 years) with persistent asthma were followed for 1 year. Asthma symptoms, health care use, lung function, pulmonary inflammation, and indoor pollutants were assessed every 3 months. Weight category was based on body mass index percentile.

Results—Participants were predominantly African American (91%) and had public health insurance (85%). Four percent were underweight, 52% were normal weight, 16% were overweight, and 28% were obese. Overweight or obese participants had more symptoms associated with exposure to fine particulate matter measuring less than 2.5 μm in diameter ($PM_{2.5}$) than normal-weight participants across a range of asthma symptoms. Overweight or obese participants also had more asthma symptoms associated with nitrogen dioxide (NO_2) exposure than normal-weight participants, although this was not observed across all types of asthma symptoms. Weight did not affect the relationship between exposure to coarse particulate matter measuring between 2.5 and 10 μm in diameter and asthma symptoms. Relationships between indoor pollutant exposure and health care use, lung function, or pulmonary inflammation did not differ by weight.

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Conclusion—Being overweight or obese can increase susceptibility to indoor $PM_{2.5}$ and NO_2 in urban children with asthma. Interventions aimed at weight loss might reduce asthma symptom responses to $PM_{2.5}$ and NO_2, and interventions aimed at reducing indoor pollutant levels might be particularly beneficial in overweight children.

Keywords

Asthma; *overweight*; *obesity*; *indoor pollutants childhood asthma*; *inner-city asthma*

The prevalence of both asthma and overweight status has increased dramatically over the past 20 years, suggesting that these 2 conditions might be linked.[1,2] In addition, African Americans, who have greater asthma morbidity than other racial/ethnic groups, also have a higher prevalence of being over-weight or obese.[1,2] Several recent studies suggest that being overweight is a risk factor for both having a diagnosis of asthma and asthma morbidity.[3–7] Although relationships between being overweight and asthma have been examined in adult asthmatic populations, there are less data in children, particularly in high-risk populations, such as urban minority children, who might be most affected by both being overweight and having asthma.

A few studies suggest that the association between being overweight and asthma morbidity might be explained, in part, by resistance to the treatment effects of corticosteroids.[8,9] However, being overweight might confer susceptibility to worse asthma health through mechanisms other than corticosteroid resistance. For example, being overweight is associated with an underlying state of oxidative stress and inflammation, potentially reducing the capacity to defend against oxidative and proinflammatory exposures, such as particulate and gaseous pollutants.[10,11] In addition, fine-particle deposition in the lungs is greater in over-weight children, so that lung exposure to a given airborne particulate matter concentration is greater in overweight children than in normal-weight children.[12]

Because fine particulate matter measuring less than 2.5 μm in diameter ($PM_{2.5}$), coarse particulate matter measuring between 2.5 and 10 μm in diameter ($PM_{2.5–10}$), and nitrogen dioxide (NO_2) are all associated with respiratory symptoms and rescue medication use in asthmatic patients,[13–19] it is plausible that the combination of a high prevalence of overweight status and higher indoor pollutant exposure among urban minority populations contributes to the disproportionate asthma morbidity in this population. We therefore hypothesized that overweight and obese urban children and adolescents with asthma are more susceptible to the effects of indoor pollutants than their normal-weight counterparts. To test our hypothesis, we examined relationships among weight status, asthma health, and indoor pollution in a population of urban, predominantly African American, asthmatic 5- to 17-year-olds.

RESULTS

Study population

The study population was predominantly male, African American, and low income (Table I). At baseline, 4% of the participants were underweight, 52% were normal weight, 16% were overweight, and 28% were obese. The population was highly atopic because 91% had at least 1 positive skin test response and total IgE levels were increased. The population also had a mean of more than 4 days per 2 weeks of rescue medication use, and more than 75% of participants had an emergency department visit for asthma in the previous year (Table II).

BMI and asthma characteristics

Overweight and obese participants had more days of exercise-induced and nocturnal asthma symptoms (over a 2-week period) than normal-weight participants (Fig 1 and Table III). For example, participants who were overweight or obese had approximately 0.7 more days of exercise-related symptoms than normal-weight participants (2.1 ± 3.6 vs 1.4 ± 2.6 [mean ± SD], respectively; $P = .05$). Overweight or obese participants also had approximately 0.6 more nights of wakening caused by asthma symptoms than normal-weight participants (1.7 ± 3.1 vs 1.1±2.2 [mean±SD], respectively; $P=.04$). However, there was little difference among BMI categories in other asthma symptoms, such as cough, wheeze, or chest tightness and short-acting β-agonist use. Overall, there were no differences in asthma-related health care use, lung function, or number of positive skin test responses among BMI categories. However, overweight or obese participants tended to have lower F_{ENO} values and total IgE levels (see Table E1 in this article's Online Repository at www.jacionline.org).

BMI, indoor pollutant exposure, and asthma symptoms

Relationships between indoor pollutants and symptom outcomes were examined across BMI categories, and data were captured from clinical and home assessments that were performed every 3 months for 1 year. Median $PM_{2.5-10}$, $PM_{2.5}$, and NO_2 levels were as follows: 13.2 $\mu g/m^3$ (interquartile range [IQR], 7.9–19.9 $\mu g/m^3$), 20.8 $\mu g/m^3$ (IQR, 12.9–34.3 $\mu g/m^3$), and 20.7 ppb (IQR, 13.7–31.3 ppb), respectively (see Table E2 in this article's Online Repository at www.jacionline.org).

There were positive and statistically significant associations between $PM_{2.5}$ levels and most asthma symptom outcomes among overweight and obese participants, whereas associations among normal-weight participants were either absent or substantially weaker (Fig 2 and Table IV). For example, every 10-fold increase in $PM_{2.5}$ level was associated with a 3- to 5-fold increase in the odds of cough without a cold among overweight and obese participants but only a 2-fold increase among normal-weight participants (interaction $P = .02$). Similarly, there was a 2.5- to 2.9-fold increase in the odds of slowed activity with every 10-fold increase in $PM_{2.5}$ levels among overweight and obese participants compared with a 1.6-fold increase in the odds among normal-weight participants (interaction $P =.08$). There were no differences seen in relationships between $PM_{2.5}$ levels and asthma-related health care use, lung function, or F_{ENO} values among normal-weight, overweight, and obese participants.

For $PM_{2.5-10}$ levels, although associations with some of the symptom outcomes were greater among overweight participants, obese participants, or both than among normal-weight participants, none of the associations between $PM_{2.5-10}$ levels and symptom outcomes within BMI categories were statistically significant, and none of the interactions between $PM_{2.5-10}$ levels and BMI were statistically significant (Table V). There were also no differences seen in relationships between $PM_{2.5-10}$ levels and asthma-related health care use, lung function, or F_{ENO} values among BMI categories.

There were no associations between NO_2 levels and any of the asthma symptom outcomes among normal-weight participants (Table VI). However, NO_2 levels were associated with some asthma symptom outcomes among overweight and obese participants. For example, for every 10-fold increase in indoor NO_2 levels, overweight and obese participants had a 2.6- to 4.5-fold increased odds of nocturnal symptoms, whereas no association was seen among normal-weight participants. However, BMI did not modify the effect of NO_2 exposure on nocturnal symptoms (interaction $P =.29$). Additionally, overweight and obese participants had a 2.9- to 4.3-fold increase in the odds of exercise-related symptoms for each 10-fold increase in indoor NO_2 exposure compared with a 1.5-fold increase in normal-weight participants (interaction $P =.05$). Similarly, overweight and obese participants had a

1.3- to 2.0-fold increase in the odds of short-acting β-agonist use for each 10-fold increase in indoor NO_2 exposure compared with association between NO_2 levels and short-acting β-agonist use in normal-weight participants (interaction $P = .07$). There were no differences seen in relationships between NO_2 levels and the other symptom outcomes, asthma-related health care use, lung function, and F_{ENO} values among BMI categories (data not shown).

DISCUSSION

In this urban, predominantly African American population, the effect of indoor $PM_{2.5}$ exposure on asthma symptoms was greater in overweight and obese children compared with that seen in normal-weight children. We also found a similar pattern of more asthma symptoms associated with indoor NO_2 exposure among overweight or obese children than their normal-weight counterparts. Our results suggest that being overweight might increase susceptibility to the pulmonary effects of indoor $PM_{2.5}$ and NO_2 and that the combination of a high prevalence of overweight status and high indoor pollutant exposure in urban children with asthma could explain some of the disproportionate asthma morbidity seen in this population.

Although exposure to indoor pollutants, including $PM_{2.5}$ and NO_2, is known to be associated with worse asthma health, the effect of being overweight on the relationships between indoor pollutant exposure and asthma health has not previously been examined to our knowledge. In fact, there are biologically plausible mechanisms by which being overweight could confer susceptibility to the pulmonary effects of pollutants in asthmatic patients. First, being overweight is associated with an underlying state of oxidative stress and inflammation, potentially reducing the capacity of overweight persons to defend against oxidative and proinflammatory exposures, such as pollutants. Second, overweight and obese children have greater pulmonary deposition of fine particles than normal-weight children, so that lower airways exposure to a given airborne particulate pollution concentration is greater in overweight than in normal-weight children. Third, it is possible that corticosteroid resistance, which has been reported in overweight or obese children and adults with asthma,[8,9] could account for greater respiratory effects of indoor pollutant exposure in overweight asthmatic participants. Interestingly, BMI did not modify the relationship between $PM_{2.5-10}$ levels and asthma symptoms, as was observed for $PM_{2.5}$ and NO_2 levels. Although the reasons for this finding are not clear, it is possible that overweight children have greater fine-particle deposition in the lungs but that coarse-particle deposition, which occurs in the more proximal airways, is less affected by weight.

In contrast to other studies that found associations between weight and asthma morbidity,[26,27] we found that although overweight and obese urban children and adolescents with asthma appear more likely to have nocturnal and exercise-related symptoms than normal-weight children and adolescents, other measures of asthma morbidity did not vary by weight. However, our findings are more consistent with findings from an urban minority adult population in which there was no association between BMI and asthma control.[28] Because the only asthma symptoms that were increased in the overweight and obese participants were nocturnal and exercise-related symptoms, it is possible that these symptoms are a manifestation of obesity-related comorbidities, such as sleep-disordered breathing and deconditioning, respectively, rather than poor asthma control. We considered whether the overweight and obese children had been given an incorrect diagnosis of asthma; however, the overweight and obese participants had features consistent with asthma, such as decreased FEV_1/forced vital capacity ratios and higher F_{ENO} values. In addition, the degrees of pulmonary obstruction and inflammation observed in the overweight and obese participants were similar to those observed in normal-weight participants and other pediatric asthma populations.[9] The positive association between BMI category and FEV_1 percentage,

although unexpected and not well understood, has been described in at least 1 other pediatric population.[29] Therefore these findings suggest that future studies should determine whether overweight children and adolescents with asthma are at risk for underrecognition and undertreatment of overweight-associated comorbidities, the symptoms of which are incorrectly attributed to asthma.

Because our study population was composed predominantly of African American children and adolescents living in Baltimore City, our findings might not be generalizable to all children and adolescents with asthma. However, our study population is similar to pediatric asthmatic populations in other large urban centers in the Northeastern and Midwestern United States, so that being overweight might confer susceptibility to the effects of indoor pollutants in other urban minority populations. Secondhand smoke exposure is known to be a major contributor to indoor $PM_{2.5}$. Although our results suggest that being overweight might increase susceptibility to the pulmonary effects of indoor $PM_{2.5}$, the contribution of secondhand smoke exposure to this finding is unclear. In addition, although the associations between risk of symptoms and pollutant concentrations were expressed per 10-fold increase in the pollutant levels, we did observe 10-fold differences in pollutant concentrations in our study population. Our findings of the association between weight status and the effect of indoor pollutant exposure on childhood asthma were limited to symptom outcomes. This is not surprising because symptoms are the most robust asthma outcomes in this patient population, particularly with respect to pollutant exposure.[14,15,18,19,23] Although the symptom outcomes are self-reported, symptoms were captured by using questions that have been used successfully for more than a decade in other inner-city populations.[23,30] In addition, lung function outcomes in particular are known to be less sensitive than other measures of disease burden in pediatric populations.[31–33] Larger studies that provide greater statistical power are needed to determine whether weight status has similar effects on these other measures of asthma health.

Even though multiple symptom outcomes were assessed, we did not apply statistical methods to correct for multiple analyses because they are overly conservative, particularly when the multiple outcomes assessed are related to one another.[34] In addition, the threshold of a P value of less than .1 that was used for interactions differs from the P value threshold of .05 typically used for main effects, but it is a commonly used threshold for interactions.[35,36] However, it is notable that although only 1 of the interactions met a more conservative statistical significance threshold ($P < .05$), there were consistent relationships across symptom outcomes, and the consistency in the pattern provides additional evidence for an interaction. Larger studies that provide greater statistical power are needed to confirm the effects we have observed of weight status on relationships between pollutant exposure and asthma symptoms.

In this urban, predominantly African American population, the effects of indoor $PM_{2.5}$ and NO_2 exposure on asthma symptoms are greater in overweight and obese than normal-weight children and adolescents. Children and adolescents with asthma in communities with a high prevalence of overweight status and indoor pollutant exposure might have greater asthma morbidity because of the interaction between these 2 factors. These findings suggest that weight loss might reduce symptom responses to these indoor pollutants and that overweight and obese children and adolescents might benefit from indoor pollutant reduction to a greater extent than normal-weight children and adolescents.

METHODS

Lung function testing

Lung function, including FEV_1 and forced vital capacity, was assessed by using forced expiratory spirometry. Predicted values and percent predicted values were determined by using the standards of Hankinson et al.[E1] All pulmonary function testing maneuvers were overread to determinevalidity of the data.

Questionnaires

A structured questionnaire that included symptom questions that have been successful in inner-city populations for more than a decade was administered.[E2] The asthma symptom outcomes were captured from the following questions:

- "In the last 2 weeks, how many days did [child] have wheezing, coughing or tightness in the chest?"

- "In the last 2 weeks, how many days did [child] have to slow down or stop his/ her activities while at home or playing with other children because of asthma, wheezing or tightness in the chest or cough?"

- "In the last 2 weeks, how many days has [child]'s wheezing ever been so bad that he/she could only speak one or two words at a time between breaths?"

- "In the last 2 weeks, has [child] had wheezing, coughing, tightness in the chest when running or going up stairs?"

- "In the last 2 weeks, has [child] had a cough that wasn't from a cold?' If yes, how many days?"

- "In the last 2 weeks, did [child] wake up at night with cough, wheeze, shortness of breath or tightness in chest?"

Recruitment

Participants were recruited primarily from patients seen for asthma in the Pediatric Emergency Department at Johns Hopkins Hospital. In addition, participants were recruited from subspecialty clinics, including the Pediatric Allergy/Immunology Clinic at Johns Hopkins and the University of Maryland, and an institutional review board-approved database of past study participants who had consented to being contacted for future studies.

Acknowledgments

Abbreviations used

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BMI	Body mass index
F_{ENO}	Fraction of exhaled nitric oxide
IQR	Interquartile range
NO_2	Nitrogen dioxide
$PM_{2.5}$	Fine particulate matter measuring less than 2.5 μm in diameter
$PM_{2.5-10}$	Coarse particulate matter measuring between 2.5 and 10 μm in diameter

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Clinical implications: Being overweight might increase susceptibility to the pulmonary effects of indoor $PM_{2.5}$ and NO_2 exposure in urban children with asthma. Interventions aimed at weight loss might reduce asthma symptom responses to indoor $PM_{2.5}$ and NO_2. Interventions aimed at reducing indoor pollutant levels could be particularly beneficial in overweight children and adolescents.
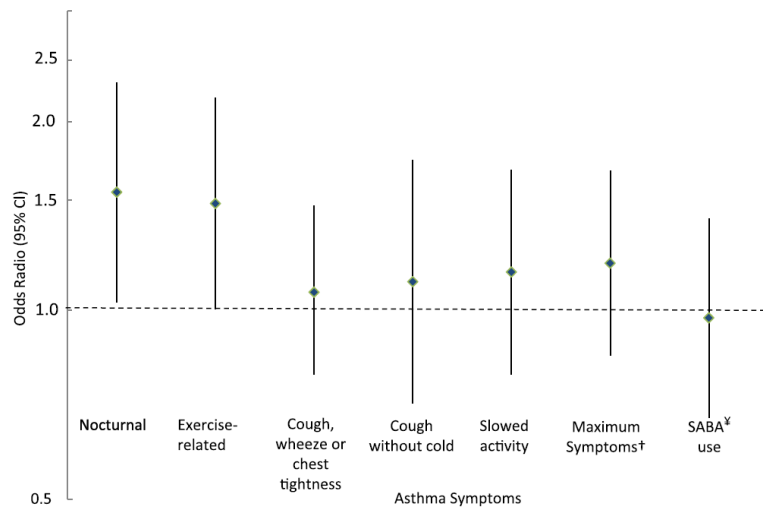
FIG 1.
Relative odds of a day of asthma symptoms for overweight or obese participants versus normal-weight participants. Odds ratios and 95% CIs are generated from binomial models adjusted for age and sex; generalized estimating equations were used to account for repeated outcome measures. †Maximum days of slowed activity or exercise-related or nocturnal symptoms. ¥*SABA*, Short-acting β-agonist.
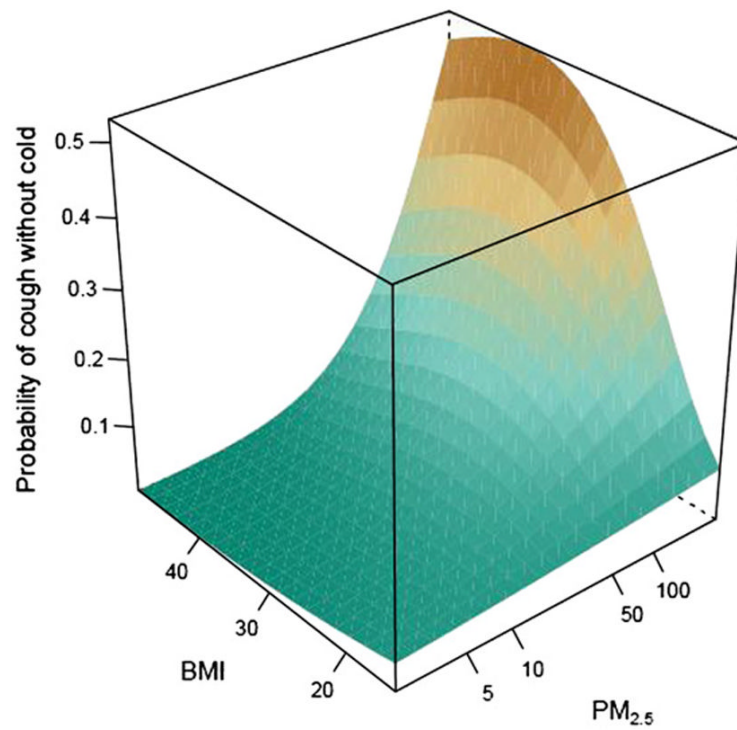
FIG 2.
Three-dimensional representation of the predicted probability of cough without cold by BMI and indoor $PM_{2.5}$ exposure. The figure is derived from a binomial model adjusted for age and sex; generalized estimating equations were used for repeated outcome measures. BMI is expressed in kilograms per square meter, and $PM_{2.5}$ is expressed in micrograms per cubic meter.

TABLE I

Baseline sociodemographic characteristics (n = 148)

Age (y), median (range)	11.2 (5.0–17.8)
Male sex, no. (%)	85 (57)
Black/African American, no. (%)	135 (91)
Household annual income <$30,000 no. (%)[*]	86 (64)
Primary caregiver educational attainment, no. (%)	
Less than high school	42 (28)
High school graduate	51 (34)
Some college or more	55 (37)
Public health insurance, no. (%)	126 (85)
Households with smokers, no. (%)	84 (57)

[*] n = 134.

TABLE II

Baseline clinical characteristics (n= 148)

Allergic characteristics	No. (%)
Atopic (1 positive SPT response)[*]	134 (91)
Skin test sensitivities[*]	
Cat	96 (65)
Cockroach	91 (62)
Dust mite	85 (58)
Mouse	78 (53)
Dog	26 (18)
Total IgE (kU/L), median (IQR)[†]	190 (56–458)
Lung function	Mean ± SD
FVC (% predicted)[‡]	100.5 ± 14.9
FEV_1 (% predicted)[‡]	93.7 ± 17.9
FEV_1/FVC ratio[‡]	80.6 ± 9.6
F_{ENO} (ppb), median (IQR)[§]	33 (16–62)
Asthma-related health care use (12 mo before enrollment)	No. (%)
Hospitalization	29 (20)
Emergency department visit	121 (82)
Unscheduled doctor's office visit	61 (41)
Controller medication use	106 (72)
Days of short-acting β-agonist use/2 wk, mean ± SD	4.2 ± 5.0
BMI category	No. (%)
Underweight (<5th percentile)	6 (4)
Normal weight (5th–<85th percentile)	77 (52)
Overweight (85th–<95th percentile)	23 (16)
Obese (95th percentile)	42 (28)

FVC, Forced vital capacity; *SPT*, skin prick test.

[*] n = 147.

[†] n = 145.

[‡] n = 132.

[§] n = 131.

TABLE III

Days of asthma symptoms by BMI category (n = 147)

Symptoms	Normal weight (82 participants,[*] 376 observations)	Overweight (23 participants,[*] 104 observations)	Obese (42 participants,[*] 172 observations)	Pvalue[†]
Cough, wheeze, or chest tightness	2.6 ± 3.4	2.9 ± 3.8	3.0 ± 3.7	.65
Nocturnal	**1.1 ± 2.2**	**1.5 ± 3.3**	**1.8 ± 2.9**	**.02**
Exercise related	**1.4 ± 2.6**	**2.0 ± 3.6**	**2.2 ± 3.6**	.07
Cough without cold	1.4 ± 3.0	1.6 ± 3.3	1.8 ± 3.7	.48
Slowed activity	1.9 ± 3.3	2.4 ± 3.7	2.1 ± 3.4	.44
Maximum symptom[‡]	2.6 ± 3.7	3.1 ± 4.3	3.3 ± 4.3	.17
SABA use	3.4 ± 4.6	3.2 ± 3.9	3.5 ± 4.4	.96

Values are presented as means ± SDs.

[*] Number of participants in each BMI category reflects who contributed at least once in each categorical analysis because there were participants who crossed into multiple categories.

[†] Results were derived from binomial models adjusted for age and sex; generalized estimating equations were used for repeated outcome measures. *P* values shown represent trends across BMI categories, and statistically significant associations are indicated in boldface.

[‡] Maximum days of slowed activity or exercise-related or nocturnal symptoms.

TABLE IV

Relationships between $PM_{2.5}$ and symptoms stratified by BMI category (n = 141)[*]

Outcome	Normal weight, OR (95% CI)	Overweight, OR (95% CI)	Obese, OR (95% CI)	P value, interaction[*]
Cough, wheeze, or chest tightness	**2.19 (1.18–4.08)**	**2.84 (1.16–6.95)**	2.23 (0.99–5.05)	.05
Nocturnal	1.04 (0.52–2.07)	**5.59 (1.57–19.94)**	3.02 (0.88–10.39)	.24
Exercise related	1.70 (0.74–3.93)	**4.36 (1.14–16.71)**	3.06 (0.87–10.73)	.16
Cough without cold	2.06 (0.79–5.37)	**3.17 (1.33–7.57)**	**5.00 (1.37–18.22)**	.02
Slowed activity	1.58 (0.75–3.33)	**2.88 (1.15–7.23)**	2.45 (0.78–7.65)	.08
Maximum symptom[†]	1.53 (0.79–2.99)	**4.83 (1.49–15.64)**	2.18 (0.71–6.65)	.14
SABA use	**1.97 (1.01–3.84)**	**3.31 (1.37–8.03)**	2.26 (0.84–6.03)	.57

OR, Odds ratio; *SABA*, short-acting β-agonist.

[*] Results are from binomial models adjusted for age and sex and account for repeated assessment of the symptom outcome. The *P* values were generated from models including a nonlinear interaction term for the interaction between BMI and pollutant exposure, and statistically significant associations are indicated in boldface.

[†] Maximum days of slowed activity or exercise-related or nocturnal symptoms.

TABLE V

Relationships between $PM_{2.5-10}$ and symptoms stratified by BMI category (n = 141)[*]

Outcome	Normal weight, OR (95% CI)	Overweight, OR (95% CI)	Obese, OR (95% CI)	P value, interaction
Cough, wheeze, or chest tightness	1.40 (0.83–2.36)	2.00 (0.31–13.01)	2.33 (0.96–5.64)	.33
Nocturnal	1.21 (0.75–1.94)	1.31 (0.10–17.21)	1.32 (0.59–2.96)	.55
Exercise related	1.23 (0.74–2.04)	0.87 (0.09–8.53)	2.17 (0.90–5.23)	.62
Cough without cold	1.71 (0.97–3.04)	0.96 (0.14–6.52)	4.67 (0.93–23.50)	.15
Slowed activity	0.83 (0.49–1.41)	1.07 (0.20–5.73)	1.71 (0.86–3.39)	.14
Maximum symptom[†]	1.06 (0.65–1.74)	1.43 (0.24–8.72)	1.88 (0.88–4.03)	.25
SABA use	0.97 (0.66–1.44)	1.19 (0.44–3.21)	1.83 (0.64–5.27)	.47

OR, Odds ratio; *SABA*, short-acting β-agonist.

[*] Results are from binomial models adjusted for age and sex and account for repeated assessment of the symptom outcome. The *P* values were generated from models including a nonlinear interaction term for the interaction between BMI and pollutant exposure.

[†] Maximum days of slowed activity or exercise-related or nocturnal symptoms.

TABLE VI

Relationships between NO_2 and symptoms stratified by BMI category (n = 142)[*]

Outcome	Normal weight, OR (95% CI)	Overweight, OR (95% CI)	Obese, OR (95% CI)	P value, interaction
Cough, wheeze, or chest tightness	1.34 (0.83–2.14)	1.42 (0.65–3.11)	1.93 (0.77–4.82)	.65
Nocturnal	0.91 (0.49–1.69)	**4.50 (1.31–15.44)**	**2.56 (1.09–5.99)**	.29
Exercise related	1.51 (0.82–2.76)	2.87 (0.87–9.51)	**4.26 (1.24–14.65)**	**.05**
Cough without cold	0.71 (0.40–1.25)	**2.56 (1.05–6.23)**	3.18 (0.57–17.66)	.24
Slowed activity	1.13 (0.65–1.99)	2.21 (0.84–5.83)	1.11 (0.30–4.06)	.41
Maximum symptom[†]	1.08 (0.66–1.76)	2.37 (0.85–6.60)	1.47 (0.42–5.12)	.43
SABA use	0.89 (0.59–1.35)	1.31 (0.74–2.31)	2.05 (0.75–5.61)	**.07**

OR, Odds ratio; SABA, short-acting β-agonist.

[*] Results are from binomial models adjusted for age and sex and account for repeated assessment of the symptom outcome. The P values were generated from models including a nonlinear interaction term for the interaction between BMI and pollutant exposure, and statistically significant associations are indicated in boldface.

[†] Maximum days of slowed activity or exercise-related or nocturnal symptoms.

TABLE E1

Lung function, asthma-related health care use, and inflammatory outcomes by BMI category[*]

Outcome	Normal	Overweight	Obese
Lung function (mean ± SD)			
FEV$_1$ (% predicted)	91.7 ± 15.8	95.5 ± 16.0	97.7 ± 19.5
FEV$_1$/FVC ratio (%)	80.3 ± 9.3	79.7 ± 9.3	81.2 ± 7.7
Asthma-related health care use			
Acute care visits (% ever during the 1-y study)	58	54	54
Inflammatory outcomes, media	(IQR)		
F$_{ENO}$ (ppb)	43 (19–73)	20 (13–39)	24 (13–47)
Total IgE (kU/L)	213 (65–773)	65 (24–223)	170 (64–302)

FVC, Forced vital capacity.

[*] There were no statistically significant differences between any of the pairwise comparisons across BMI categories. Comparisons were made by using binomial models adjusted for age and sex; generalized estimating equations were used for repeated outcome measures.

TABLE E2

Indoor pollutant concentrations

	Median (IQR)	Range
$PM_{2.5}$ ($\mu g/m^3$)	20.8 (12.9–34.3)	1.7–300.3
$PM_{2.5-10}$ ($\mu g/m^3$)	13.2 (7.9–19.9)	0.3–173.3
NO_2 (ppb)	20.7 (13.7–31.3)	0.1–351.3