

# Indoor Risk Factors for Asthma and Wheezing Among Seattle School Children

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Indoor risk factors for physician-diagnosed asthma and wheezing in the past 12 months without previous asthma diagnosis were assessed in a survey of parents of 5–9-year-old Seattle primary school students. Among the 925 respondents, 106 (11%) reported a physician diagnosis of asthma, 66 (7%) had wheezing without diagnosis, and 753 (82%) were asymptomatic. After adjusting for age, sex, gender, ethnicity, medical history, socioeconomic status (SES) and parental asthma status, an increased risk of physician diagnosis of asthma was associated with household water damage, the presence of one or more household tobacco smokers, and at least occasional environmental tobacco smoke (ETS) exposure. Similarly, an increased risk of wheezing in the past 12 months among children without diagnosed asthma was associated with household water damage, presence of one or more household tobacco smokers, and occasional or more frequent ETS exposure. No increased risk of either condition was associated with gas, wood, or kerosene stove use, household mold, basement water, or wall/window dampness. Similarities in the indoor risk factors patterns between diagnosed asthma and wheezing without diagnosis suggested a similar etiology of these two conditions. The slightly higher association between ETS and asthma may indicate that parents of diagnosed asthmatics were more conscious of ETS, and were more likely to prohibit household smoking by resident smokers. Future research is needed to quantify which aspects of household water damage are related to respiratory illness. *Key words:* asthma, childhood, environmental tobacco smoke, gas stove, household dampness, wheezing. *Environ Health Perspect* 105:208–214 (1997)

Childhood asthma prevalence has increased in the United States (1,2), Europe, and Australia (3–7) since the late 1970s. Although the exact causes of this increase are unknown, current theories suggest that increases in asthma risk factors may be responsible for a substantial amount of this increase (8). Population-based studies have described an increased risk of childhood asthma associated with male sex, race, socioeconomic status (SES), geographic region of residence, low birth weight, parental asthma, previous viral infection, hay fever, atopy, infant lung disease, and environmental factors (9–13). Recent interest has focused on the influence of the indoor environment because of a decline between 1981 and 1990 in the U.S. outdoor concentration of several airborne pollutants (14), and a possible increase in indoor air pollution resulting from modern building methods that reduce outside air exchange. One possible mechanism describes increased production of asthma symptoms as the result of localized airway inflammation produced by increases in lung tissue toxins or airborne allergens (15). Experimental studies have demonstrated that household environmental tobacco smoke (ETS) and stove exhaust are sources of air pollutants detrimental to human lung function, such as nitrous oxide (16), carbon monoxide, and respirable particles (17,18). Household dampness can contribute to the

growth of mold (19) and house dust mites (20), both of which appear to produce an IgE-mediated allergic response (21,22). Epidemiologic studies have described an increased risk of childhood asthma due to household tobacco smoke exposure (23–26), dampness (25,27–30), and gas stove use (25,31). However, the association between the indoor environment and any unrecognized childhood asthma remains largely unknown. Brunekreef et al. (28) described a larger effect of household dampness on respiratory illness among wheezing, nonasthmatic 6–12-year-old children than on asthmatic children, and Dekker et al. (25) has described an increased risk of wheezing among 5–8-year-old Canadian children without a diagnosis of asthma associated with household dampness and tobacco smoking. The ability to characterize the etiology of this unrecognized disease has been limited by the lack of a standardized method to assess asthma and respiratory symptom prevalence. In response to this problem, the International Study of Asthma and Allergy in Childhood (ISAAC) was developed to assess respiratory and allergy symptoms with a validated questionnaire according to a standard protocol. Although studies using this protocol have been conducted in Europe (32,33), Australia (34), and New Zealand (35), no studies of U.S. children have previously been published.

In this analysis, we present information using the ISAAC protocol to describe the effect of the indoor environment on the risk of asthma and undiagnosed asthmalike illness among U.S. school children. We assessed the risk of physician-diagnosed asthma and wheezing in the past 12 months without previously diagnosed asthma, associated with parent-reported household exposure to ETS, stove or fireplace use, and dampness. We also assessed the influence of subject age, gender, ethnicity, socioeconomic status, allergy history, parental asthma, and season of survey as potential confounders in these associations.

## Methods

The information for this analysis was collected as part of an addition to a year-long prevalence study of asthma and asthmalike illness in Seattle school children (W.C. Maier et al., unpublished data). Subjects in this analysis were first- and second-grade students (ages 5–9 years) enrolled at participating Seattle public primary schools sampled between February and June 1994. All subject information, except for SES, was collected using a single mailing of a parent-completed ISAAC questionnaire (36), with additional questions on household environment. These questions were added to the end of the existing ISAAC questionnaire to maintain the standardized format and content as used in other studies. SES was determined by assigning each subject a value describing the proportion of total first- and second-grade Seattle school students receiving a reduced-price school lunch (family income <\$27,500/year) in their school and ethnicity subcategory. All school lunch information was provided by the Seattle public school district.

Children were classified into three mutually exclusive groups based on their response

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to the respiratory symptom questionnaire: physician-diagnosed asthma, wheezing in the past 12 months without diagnosed asthma (current wheezing), and neither condition (normal). Unadjusted relative prevalence odds ratios and 95% CI were calculated to describe the increased prevalence of either diagnosed asthma or current wheezing relative to the normal population, associated with the reported household presence in the past 12 months of the following potential sources of air pollution: presence of one or more tobacco smokers, use of a gas-, wood-, or kerosene-burning stove or wood-burning fireplace, and presence of mold/mildew growth, water damage, basement water, or routine condensation of water on the windows or walls. Multivariate logistic regression was used to describe the effect of any stove or fireplace use, the presence of one or more household tobacco smoker, occasional or more frequent ETS exposure, household water damage and any household dampness except household water damage, on either asthma or current wheezing risk, using odds ratios adjusted for young age (5–7, 8–9 year referent), gender (male, female referent), ethnicity (black, other nonwhite, white referent), lower SES (defined as being from a school with more than 14% participation in the school lunch program), history of allergies, chronic sore throat, chronic earache, parental asthma, and winter season of survey. Variables describing age, chronic sore throat, chronic earache, and winter season of survey were iteratively removed from the final model, because these factors were neither significant in the model ( $p < 0.2$ ) nor resulted in a 10% or larger change in the estimate of the effect of any environmental factor.

The effect of response bias in our sample was also assessed by calculating weighted asthma and wheezing prevalence estimates that assigned a larger weight to schools and ethnic groups with lower survey response (see Appendix A for methodology). The magnitude of the difference between the weighted and unweighted estimates indicated the potential for bias because of increased response by parents of children with known respiratory illness.

## Results

Our analysis was based on 925 subjects, or approximately 31% of the target group sampled between February and June 1994. The analysis population was equally divided by gender, and the majority were 5–7 years old (69%). Subjects were predominantly white (66%) and had a lower rate of participation in the reduced price school lunch program [mean = 23%, standard deviation (SD) = 22, median = 10] relative to the total first- and second-grade Seattle school population,

which was 45% white and had a mean across schools of 39% (SD = 25, median = 33) participating in the school lunch program. Response to this section of our questionnaire was also associated with ethnic and socioeconomic differences. Schools with a response rate over 33% were predominantly white (74 vs. 55% among low response schools,  $p < 0.001$ ) and had more children in the upper SES group (79 vs. 29% among low response schools,  $p < 0.001$ ). However, the distribution of the three disease categories was similar in both response groups ( $p = 0.407$ ), and school response rate was not correlated with the prevalence of asthma or wheezing. The similarity of the unweighted and weighted estimates for both diagnosed asthma (11.4 and 12.5%) and current wheezing (7.1 and 6.9%) also indicated that

the magnitude of any response bias in our sample was small. Comparisons across all three disease states found no difference in age, SES, or season of survey (Table 1). The three disease states were, however, significantly different with respect to gender, ethnicity, allergy history, and parental asthma. Diagnosed asthmatics had the highest percentages of males, and the current wheezing group had the next highest. A similar trend was also present for ethnicity, allergy history, and parental asthma.

Household dampness was the most commonly reported household exposure (Table 2). More than 66% of each disease group reported one or more marker of household dampness and more than 50% reported household mold or mildew growth. The three disease states were significantly

**Table 1.** Demographics

	Diagnosed Asthma ( <i>n</i> = 106) (%)	Current wheezing ( <i>n</i> = 66) (%)	Normal ( <i>n</i> = 753) (%)	<i>p</i> -value
Age				
5–7 yr.	72 (68)	48 (73)	520 (69)	0.789
8–9 yr.	34 (32)	18 (27)	233 (31)	—
Gender				
Male	65 (61)	38 (58)	359 (48)	0.014
Female	41 (39)	28 (42)	394 (52)	—
Ethnicity				
Blacks	21 (20)	10 (15)	69 (9)	0.013
Whites	65 (61)	43 (65)	507 (67)	—
Other nonwhites	20 (19)	13 (20)	177 (24)	—
Lower SES	52 (49)	26 (39)	313 (42)	0.305
Allergies	59 (56)	15 (23)	129 (17)	<0.001
Chronic ear ache	27 (25)	10 (15)	139 (18)	0.161
Chronic sore throat	7 (7)	5 (8)	28 (4)	0.158
Parental asthma	38 (36)	14 (22)	122 (16)	<0.001
Season of survey				
Winter	52 (49)	33 (50)	379 (50)	0.970
Spring	54 (51)	33 (50)	374 (50)	—

*p*-value from chi-square test, degrees of freedom = 2 for nonordered data.

**Table 2.** Distribution of indoor exposures in each disease group

	Diagnosed asthma, ( <i>n</i> = 106) (%)	Current wheezing, ( <i>n</i> = 66) (%)	Normal ( <i>n</i> = 753) (%)	PR <sup>a</sup>	PR <sup>b</sup>	<i>p</i> <sup>c</sup>
Household smokers	28	30	21	1.4 (0.9, 2.1)	1.6 (1.0, 2.6)	.051
Tobacco Smoke Exposure						
Never	69	74	83	1.0	1.0	0.005
Occasionally	24	18	12	2.1 (1.4, 3.2)	1.6 (0.9, 2.9)	—
Several hr/day	8	8	5	1.8 (0.9, 3.5)	1.8 (0.8, 4.3)	—
Gas stove use	18	18	21	0.9 (0.6, 1.4)	0.9 (0.5, 1.6)	0.707
Kerosene stove use	2	3	2	1.0 (0.3, 3.7)	1.5 (0.4, 5.6)	0.843
Wood stove use	9	11	10	0.9 (0.5, 1.7)	1.0 (0.5, 2.1)	0.944
Fireplace use	35	40	35	1.0 (0.7, 1.4)	1.2 (0.7, 1.9)	0.803
Any use of the above	51	53	56	0.9 (0.6, 1.3)	0.9 (0.6, 1.4)	0.646
Mold	59	58	53	1.3 (0.9, 1.9)	1.2 (0.7, 1.9)	0.369
Water damage	27	27	18	1.6 (1.1, 2.4)	1.7 (1.0, 2.8)	0.015
Basement water	20	23	22	0.9 (0.6, 1.4)	1.0 (0.6, 1.7)	0.859
Water condensation	39	39	33	1.2 (0.8, 1.7)	1.3 (0.8, 2.1)	0.374
Any of the above	74	68	67	1.4 (0.9, 2.1)	1.1 (0.7, 1.8)	0.292

PR, prevalence ratio.

<sup>a</sup>Unadjusted PR (95% CI) of diagnosed asthma compared to normal population.

<sup>b</sup>Unadjusted PR (95% CI) of current wheezing compared to normal population.

<sup>c</sup>*p*-value from chi-square test of no association across all three disease groups.

different only for reported household water damage. Use of a fireplace or stove was reported by over 50% of each disease group. Fireplaces were used by approximately 35% of each group, and gas stoves by approximately 20% of each group. However, no type of fireplace or stove use was significantly different across the three disease groups. Tobacco smoke exposure was common among diagnosed asthmatics and was significantly different across each of the three disease groups. Over 25% of both asthmatic and current wheezing groups reported their child either living in a house with a tobacco smoker or having at least occasional exposure to tobacco smoke. The risk factors for current wheezing were similar to those for diagnosed asthma. Stove and fireplace use was not either individually or collectively associated with an increase in the risk of either condition. The presence of a household tobacco smoker was associated with moderate increase in the risk of both diagnosed asthma and current wheezing. A report of at least one form of household dampness was associated with a small increase in the risk of diagnosed asthma, but not current wheezing. However, an increase in the risk of both conditions was associated with a report of household water damage.

Several subgroups of the population experienced a higher risk of disease associated with factors in the indoor environment. Household water damage had a strong effect on current wheezing among both blacks [unadjusted prevalence ratio (PR) = 3.2, 95% CI = 1.0, 9.9] and other nonwhites (PR = 2.5, 95% CI = 0.7, 8.3). The risk of diagnosed asthma associated with household water damage was also high among other nonwhites (PR = 3.9, 95% CI = 1.6, 8.8), but similar to the crude estimate among blacks. The effect of household water damage on the prevalence of diagnosed asthma (PR = 2.4, 95% CI = 1.4, 4.0) and current wheezing (PR = 3.4, 95% CI = 1.6, 7.1) was also larger among children from the lower SES group. Household smoking had a larger effect on the risk of diagnosed asthma among those with a history of chronic sore throat (PR = 4.2, 95% CI = 0.9, 18.6), and a larger effect on the risk of current wheezing among those with lower SES (PR = 2.3, 95% CI = 1.1, 4.8), winter season of survey (PR = 2.1, 95% CI = 1.1, 4.0), allergies (PR = 3.3, 95% CI = 1.3, 8.4), a history of chronic earaches (PR = 4.1, 95% CI = 1.3, 13.2), household water damage (PR = 2.5, 95% CI = 1.1, 5.9), and household dampness, except water damage, (PR = 2.1, 95% CI = 1.2, 3.8). Stove or fireplace use among children with a history of chronic sore throat was associated with a consistent nonsignificant increase in the risk of both diagnosed asthma (PR = 2.3,

**Table 3.** Crude and adjusted OR with 95% CI to estimate the relative risk of diagnosed asthma and current wheezing associated with the indoor environment

	Diagnosed crude OR	Adjusted OR <sup>a</sup>	Current wheezing crude OR	Adjusted OR
Household tobacco smoker	1.5 (1.0, 2.4)	1.6 (0.9, 2.7)	1.7 (1.0, 2.9)	1.8 (1.0, 3.2)
Tobacco smoke exposure—occasional or more	2.2 (1.4, 3.5)	2.5 (1.5, 4.3)	1.7 (1.0, 3.1)	1.8 (1.0, 3.2)
Any gas, wood, or kerosene stove or fireplace use	0.8 (0.5, 1.6)	0.9 (0.6, 1.4)	0.9 (0.5, 1.5)	0.9 (0.5, 1.5)
WD	1.8 (1.1, 2.8)	1.7 (1.0, 2.9)	1.7 (1.0, 3.1)	1.7 (0.9, 3.0)
Any wetness/no WD	1.4 (0.9, 2.3)	1.2 (0.7, 2.0)	1.1 (0.6, 1.8)	1.0 (0.6, 1.8)

Abbreviations: OR, odds ratio; WD, water damage.

<sup>a</sup>OR of either diagnosed asthma or current wheezing without diagnosis relative to the unaffected population adjusted for gender, ethnicity, allergy history, lower socioeconomic status, and parental asthma.

95% CI = 0.6, 8.7) and current wheezing (PR = 2.6, 95% CI = 0.5, 13.4).

Confounding due to age, gender, ethnicity, allergy history, SES, and parental asthma was minimal because most odds ratio estimates did not change by more than 10% after adjustment for these factors (Table 3). The exceptions were a small decline in the risk of diagnosed asthma associated with a report of any type of household dampness and an increase in the risk of asthma associated with ETS exposure. The adjusted odds ratio (OR) estimates were also constant in multivariate models containing variables describing all environmental factors, and all the previously mentioned potential confounders considered in Table 4. Since both tobacco smoke exposure variables were collinear, only the variable describing presence of a household tobacco smoker was included in these models. Use of the ETS variable instead of this variable produced a similar model (not shown), and the adjusted associations for ETS with diagnosed asthma (adjusted OR = 2.4, 95% CI = 1.4, 4.1) and current wheezing (adjusted OR = 1.7, 95% CI = 0.9, 3.1) were unchanged from the estimates presented in Table 4. The magnitude of the point estimates for diagnosed asthma and current wheezing were slightly different between the two models. Fireplace or stove use, water damage, other wetness, ethnicity, and SES had similar point estimates in both models. Male gender, allergy history, and parental asthma had a larger effect on the risk of asthma than the risk of current wheezing.

## Discussion

To our knowledge, this was the first study to describe the association between current wheezing (wheezing in the past 12 months in the absence of physician-diagnosed asthma) and the indoor environment among a group of U.S. school children aged 5–9 years. Household dampness and stove or fireplace use were reported by over 50% of

**Table 4.** Adjusted odds ratio of the risk of diagnosed asthma and current wheezing associated with each environmental exposure, demography, and medical history factor

	Diagnosed asthma	Current wheezing
Household tobacco smoker	1.6 (0.9, 2.7)	1.7 (0.9, 3.1)
Any fireplace or stove use	0.9 (0.6, 1.4)	1.0 (0.6, 1.6)
WD	1.7 (1.0, 2.8)	1.7 (0.9, 3.2)
Other wetness/no WD	1.1 (0.6, 1.8)	0.9 (0.5, 1.6)
Male	1.7 (1.1, 2.8)	1.5 (0.9, 2.6)
Black	1.8 (0.8, 3.9)	2.0 (0.8, 5.2)
Other nonwhite	1.0 (0.5, 1.9)	1.1 (0.5, 2.6)
Low SES	1.1 (0.6, 2.0)	0.6 (0.3, 1.4)
Allergies	5.9 (3.8, 9.2)	1.4 (0.7, 2.5)
Parental asthma	2.9 (1.8, 4.7)	1.4 (0.8, 2.7)

Abbreviations: WD, water damage; SES, socioeconomic status.

Estimates are adjusted for all factors listed.

parents, and household tobacco smoking was reported by over 20%. Neither stove nor fireplace use was associated with an increase in the risk of either current wheezing or diagnosed asthma. Household tobacco smoking increased the risk of both current wheezing and diagnosed asthma. The effect of household smoking on current wheezing was higher among children with a lower SES, winter season of survey, allergies, and a history of chronic earaches. Household mold/mildew growth, basement water, and water condensation on walls or windows were not associated with an increase in the risk of either diagnosed asthma or current wheezing. However, a report of household water damage was associated with an increased risk of both conditions, and the effect of household water damage on the risk of both current wheezing and diagnosed asthma was larger among blacks than whites. Also, the effect of both tobacco smoke exposure and household dampness remained constant in a model containing both variables in addition to age, gender, season of survey, ethnicity, SES, allergy history, medical history, or parental asthma.

Information used in this analysis was obtained from a series of questions added after the beginning of a year-long asthma

**Table 5.** Prevalence of asthma and wheezing using the ISAAC questionnaire among 5–9-year-old children in the United States, Canada, Norway, Italy, United Kingdom, and South Africa

	Seattle (United States) (n = 925)	Hamilton (52) (Canada) (n = 3117)	Oslo (53) (Norway) (n = 2700)	Verona (54) (Italy) (n = 2075)	United Kingdom (55) (n = 1573)	Cape Town (56) (South Africa) (n = 1955)
Asthma	11.4%	17.2%	9.5%	11.7%	12.8%	10.8%
Wheezing	14.5%	19.7%	12.9%	7.3%	16.7%	26.8%
Response	31%	76%	90%	90%	*	90%
Age (yrs)	5–9	6–7	6–7	6–7	5–7	7–8

Wheezing pertains to children wheezing in the past 12 months either with or without a diagnosis of asthma.  
\*Representative national sample of the United Kingdom.

prevalence study. Consequently, our information was only reflective of the household environment between February and June 1994. Because of confidentiality concerns by the Seattle School district, we were not able to contact parents directly. As a result, we were not able to either directly characterize nonrespondents or validate parental reports of child asthma and respiratory symptoms using medical records. However, the predictive validity of the ISAAC questionnaire has been determined using bronchial hyperresponsiveness (BHR) after histamine challenge in adolescents and adults (37–39). Although it appears to be an inadequate gold standard for asthma, BHR still remains an important indicator of asthma status. Two previous studies by Shaw and colleagues (38,39) have demonstrated high specificity (0.70–0.96) and moderate sensitivity (0.4–0.7) in the ability of the symptom questionnaire to predict BHR. Other validation studies of the ISAAC questionnaire have demonstrated that it was also consistently sensitive (49–80%) and specific (74–90%) to both an increase in BHR after challenge by hypertonic saline or exercise (40), and an independent physician diagnosis of childhood asthma (41).

The response rate in this sample was relatively low (925/3,000), and the responding population was composed of a larger percentage of whites and upper SES individuals than was present in the total first- and second-grade population of the Seattle public school system. However, the asthma diagnosis and wheezing rates obtained in our study were comparable to other ISAAC studies with high response rates (Table 5). Our asthma and wheezing prevalence estimates were lower than those found in Canada, similar to those in Italy, South Africa, and the United Kingdom, and higher than those from Norway. It was also possible, because African-American children and those from lower SES backgrounds are at an increased risk for asthma, that our study may have slightly underestimated the prevalence of asthma and wheezing in Seattle. Additionally, we were unable to detect any effect of school response rate

on the prevalence of either asthma or wheezing measured in our study. Given all these conditions, we feel that our estimates of indoor effects were not significantly biased due to elevated response among parents of children with asthma or wheezing.

The environmental questions used in our paper were novel to our study and were not, to our knowledge, measured by other ISAAC study sites elsewhere in the world. School district confidentiality concerns prohibited us from using school address information to contact individual parents to directly validate the exposures measured. The lack of additional information on the indoor environment limited our ability to determine the measurement characteristics of our environmental questions. However, previous studies have demonstrated that parent-completed questionnaire-based assessments of ETS exposure in their children were well correlated with the presence of a tobacco smoke metabolite, cotinine, in the urine (47) and hair (48). Our household dampness questions were modeled on those used in previous studies which linked presence of mold or mildew growth, water damage, basement water, or presence of damp spots to increased childhood asthma (28–30). Subjective questionnaire-based assessment of household dampness has been associated with an increase in household fungi spore counts (27). Our questions on stove or fireplace use were based on previous studies of the respiratory effects of gas stove, kerosene heaters, and wood-burning fireplaces and stoves (31,49). Although our questions have not been previously validated, Neas et al. (31) did demonstrate a higher concentration of nitrogen dioxide in houses with either a kerosene heater or gas stove. Examples of the questions asked are provided in Appendix B.

The lack of an association between stove or fireplace use on the risk of either current wheezing or diagnosed asthma reported in our study was similar to previous studies in this area. Unvented gas or kerosene stoves can result in high levels of nitrogen dioxide (16), which is a known and demonstrated lung toxin (50). Wood stoves and fireplaces that are improperly vented may emit carbon

monoxide and respirable particles (17,18). Household wood-burning in stoves or fireplaces has not been associated with an increased risk of childhood respiratory symptoms or asthma (25,49). Some studies have demonstrated an effect of gas stove or kerosene heater use on the risk of childhood asthma (25) or wheezing (31), while others have not (24,51). In our study, we did not determine the household stove or fireplace location, and we did not collect information on room size, house age, or other variables that might have affected the concentration of airborne toxins from these sources. We did measure an increase in the risk of both diagnosed asthma and current wheezing associated with any stove or fireplace use among children with a history of chronic sore throat. This increase may be attributable to either increased asthma detection among children with chronic sore throats or may be a marker of chronic subclinical inflammation resulting from repeated infection of the lower and upper respiratory tract. Whereas previous research indicates that chronic respiratory tract infection may predispose children to chronic airway inflammation (51), there is reason to believe that children with chronic sore throats may be especially sensitive to emissions from gas stoves and wood-burning fireplaces. However, because the precision of our estimates was low due to small sample size, additional research is needed to confirm the existence of this association.

In contrast to the effects of wood smoke, exposure to tobacco smoke was a consistent risk factor in our study for both diagnosed asthma and current wheezing. Previous U.S. national studies have described an increase in asthma and wheezing risk associated with parental smoking (23,52). The effect of parental smoking and secondhand tobacco smoke exposure has also been consistently described in smaller individual or multicommunity studies. Results from the Harvard Six Cities study of U.S. children aged 6–14 years have demonstrated an increased risk of respiratory symptoms with increased parental smoking that was larger for maternal than paternal smoking (24), and an increased risk in respiratory symptoms associated with parental smoking after adjustment for age, race, city, parental illness, parental education, single child status, single parent status, air-conditioning, and body mass index. Similar findings on the relationship between the effects of parental smoking were reported in the earlier Tecumseh, Michigan, study of children 0–19 years old (53); parental smoking habits were found to be unrelated in the development of asthma in a cohort of 5–9-year-old Boston school children (11). Parental smoking has also

been associated with an increase in childhood asthma exacerbations (47) and bronchial responsiveness (54). Our results were also similar to those found among 5–8-year-old Canadian children by Dekker et al. (25). Although we found a similar risk of diagnosed asthma and current wheezing regardless of the number of household tobacco smokers, our risk estimates for the effect of having one or more household tobacco smoker were similar to those presented by Dekker et al. for the risk of diagnosed asthma (OR = 1.4–1.7) and current wheezing (OR = 1.4–1.6) associated with multiple household tobacco smokers. We were unable to determine the relationship between maternal and paternal smoking in our sample because school district confidentiality concerns prohibited us from determining the identity of the household tobacco smokers reported in our study. The relative similarity of the associations between the two tobacco smoke-exposure measures and the risk of wheezing, and the difference between the effects of having a household tobacco smoker and being at least occasionally exposed to tobacco smoke, suggested that parents of asthmatic children were more likely to notice child tobacco smoke exposure, but less likely to have actual household tobacco smoke exposure.

Our results also indicated that some form of household dampness may be a risk factor for diagnosed asthma and current wheezing. Household dampness may increase the risk of childhood asthma and other respiratory symptoms by promoting the growth of either house dust mites (Pyroglyphidae) or fungi, both of which appear to be relatively ubiquitous in human environments. House dust mites (HDM) flourish in environments at 25°C and at relative humidities of 55–75%; they are found in carpets, mattresses, and dust humid enough to support them (55). Even though HDM predominate in the hot, humid summer months, HDM-sensitive people have more symptoms during cold months (56,57). HDM sensitivity appears to be most severe at night (58,59); increased sensitivity appears to result from elevated exposure during sleep to HDM body parts and feces in the mattress. Mold species thrive at slightly higher relative indoor humidities (75–90%), and the allergic potential of mold varies by species (60). HDM feces and body parts (22) and mold spores (21) appear to induce IgE-mediated allergic responses. Elevated serum IgE to HDM or mold has been associated with childhood residence in homes with either inadequate ventilation (61) or household dampness (62). The risk of either asthma or chronic cough associated with household dampness also appears to be larger among

children with a sensitivity to HDM or mold (62). Several previous studies have also described a similar increase in the risk of both diagnosed asthma and other respiratory symptoms associated with some measure of household dampness (26,28,29,30,62,63). Brunekreef et al. (29) also demonstrated a stronger effect of household dampness among nonasthmatic than asthmatic children. Our estimates were similar for both the diagnosed asthma and current wheezing associated with household dampness after adjusting for gender, ethnicity, SES, parental asthma, and allergy history. Although we did not specifically collect information about the presence of pets in the children's households, our results were similar to those of Verhoeff et al. (62), which demonstrated that the association between household dampness and respiratory symptoms was independent of the presence of hairy or feathered pets in Dutch children aged 6–12 years. Based on this information, we feel our results were probably not substantially affected by uncontrolled confounding due to our lack of information about household pets. Although it is theoretically possible that household water damage may influence the household residence of rodents or the placement of household pets, previous research has not demonstrated any modifying effect of household water damage on the relationship between the presence of household pets or other animals and an increase in either asthma or other respiratory symptoms.

Of the four household dampness components we measured, only reported household water damage was a consistent risk factor for both diagnosed asthma and current wheezing. Mold growth (adjusted OR = 1.2, 95% CI = 0.7, 2.0) and water condensation (adjusted OR = 1.3, 95% CI = 0.9, 2.2) were associated with small, nonsignificant increases in current wheezing; no increase in risk was associated with reported basement water. The reason for the predominance of household water damage was unknown. Household water damage may have been more easily recognized by the participants than other forms of household dampness because it was more likely to either require attention or be present independent of seasonal changes. It is also possible that household water damage may be an indicator of poorer housing quality and a surrogate marker for lower SES. We did find a stronger effect of household water damage on the unadjusted risk of current wheezing among both blacks and those in lower SES categories. Reduced medical care access in the Seattle area has been associated with lack of health insurance resulting from lower income (64); and U.S. children without health insurance have been shown to be

two times more likely than insured children to receive no care for symptoms suggestive of asthma (65). However, because our sample was disproportionately drawn from whites and those from an upper SES group, the effect of limitations in medical care access was likely to be less among our population relative to the total population of Seattle school children. Further research is needed to determine whether or not household water damage is a surrogate for lower SES and to quantify what aspects of household water damage may result in either asthma or other respiratory symptoms.

There was also the possibility that our adjustment for confounding factors influencing the risk of respiratory illness may have produced adjusted estimates that distorted the effect of these household exposures on the risk of diagnosed asthma and current wheezing (66). Our use of an ecological measure of SES may have resulted in misclassification of individual SES. However, SES can be difficult to classify because personal information, such as income or education, may be both sensitive information and not representative of individual access to societal resources to improve or maintain health. In our study, subject recall bias was avoided because the school lunch information used as a surrogate for SES was obtained directly from Seattle school district records. It was also possible that our process of grouping into ecological measures may have controlled for variables not otherwise controlled in the individual analysis (67). Both parental asthma history and childhood allergy history may be affected by the same household factors as childhood asthma. We were unable to determine which parent (mother or father) had a history of asthma due to the Seattle school district's desire to maintain parent confidentiality. Allergy history was collected with a single question as we felt it was the most representative marker of allergic status given that the presence and effect of individual allergens could not be determined because of the sampling methodology and questionnaire-length constraints. Examples of the questions asked are provided in Appendix B. However, all adjusted estimates of disease risk from reduced models without parental asthma history and child allergy history were similar to those from the full models. This result, along with the similarity between the crude and adjusted estimates from the full model, indicated that bias because of over-control was unlikely.

## Conclusions

The findings of our study indicated that the household environment can profoundly increase the risk of both childhood asthma and wheezing. More research is needed to

characterize those aspects of household dampness and water damage that need to be changed to reduce the occurrence of childhood asthma and wheezing. Household tobacco smoking and child tobacco smoke exposure were relatively common. Physicians should improve their efforts to inform the parents of asthmatic patients about the detrimental effects of tobacco smoke exposure. Public health efforts to inform the general public about the need to eliminate childhood tobacco smoke exposure should be also expanded.

## Appendix B. Questions Used in this Analysis

### I. Symptom Questions

1. Did a doctor ever tell you that your child had asthma?  
Yes \_\_\_\_\_ No \_\_\_\_\_
2. Has your child had wheezing or whistling in the chest in the last 12 months?  
Yes \_\_\_\_\_ No \_\_\_\_\_
3. Has a doctor ever told you that your child had any of the following conditions?  
Allergies \_\_\_\_\_ Chronic/frequent earaches \_\_\_\_\_  
Yes \_\_\_\_\_ No \_\_\_\_\_ Yes \_\_\_\_\_ No \_\_\_\_\_  
Chronic cough \_\_\_\_\_ Chronic/frequent sore throat \_\_\_\_\_  
Yes \_\_\_\_\_ No \_\_\_\_\_ Yes \_\_\_\_\_ No \_\_\_\_\_

## Appendix A. Weighted Estimate of Prevalence

Note: This method assumes subjects were gathered into the study using simple random sampling.

$$\text{Weighted estimate of prevalence} = \frac{\sum_j \sum_i c_{ij} \left[ \frac{1}{\binom{p_s}{e_{ij}}} \right]}{\sum_j \sum_i t_{ij} \left[ \frac{1}{\binom{p_s}{e_{ij}}} \right]}$$

### Definitions

#### Constants

1.  $p_s$  = probability of being in a school that participated in this section of the survey = 27 of 42 = 0.6.

#### Variables

1.  $c_{ij}$  = Number of study subjects with symptom of interest in each school ( $j = 1-27$ ) and ethnicity subgroup ( $i = 0, 1$ ) sampled.
2.  $t_{ij}$  = Total number of study subjects in each school and ethnicity sampled.
3.  $e_{ij}$  = Total number of enrolled first- and second-graders in each school and ethnicity sampled.

4. Did a doctor ever tell you, the parent, that you had asthma?  
Yes \_\_\_\_\_ No \_\_\_\_\_
5. Did a doctor ever tell your child's other parent that he or she had asthma?  
Yes \_\_\_\_\_ No \_\_\_\_\_

### II. Environmental Questions

1. How many people living in your household smoke?  
0 (none) \_\_\_\_\_  
1 person \_\_\_\_\_  
2 persons \_\_\_\_\_  
3 persons \_\_\_\_\_  
If more than 3 people, how many? \_\_\_\_\_
2. In your household during the past 12 months how often were the following used? (*Never, <1/month, 1-3/month, About 1/week, Almost every day*)  
Natural-gas kitchen stove \_\_\_\_\_  
Kerosene heater \_\_\_\_\_  
Wood-burning stove \_\_\_\_\_  
Wood-burning fireplace \_\_\_\_\_
3. During the past 12 months, how often have you noticed the following inside your home? (*Never, Sometimes, Often, Always*)  
Mold or mildew on surface \_\_\_\_\_  
Water damage \_\_\_\_\_  
Water in the basement or crawl space \_\_\_\_\_  
Wet patches on the walls or windows \_\_\_\_\_

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