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Association of gas cooking with children's respiratory health: results from GINIplus and LISAplus birth cohort studies

Abstract Previous studies have found inconsistent results on the association between asthma in children and gas cooking emissions. We aimed to assess the effects of the long-term exposure to gas cooking on the onset of asthma and respiratory symptoms, focusing on wheezing, in children from two German birth cohorts: LISAplus and GINIplus. A total of 5078 children were followed until the age of 10 years. Asthma, wheezing, gas cooking, and exposure to other indoor factors were assessed through parental reported questionnaires administered periodically. Logistic and multinomial regressions adjusting for potential confounders were performed. The prevalence of asthma and persistent wheezing was higher among children exposed to gas cooking but the results were not statistically significant. Exposure to gas cooking was positively associated (P-value < 0.05) with exposure to other indoor factors (dampness, environmental tobacco smoke, and pets). Our results did not show a statistically significant association between the exposure to gas cooking and children's respiratory health.

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Practical Implications

These analyses are consistent with the assumption of no effect of the exposure to low doses of nitrogen dioxide. The strong positive associations found between gas cooking and other indoor factors highlight the importance of considering other indoor factors when assessing health effects of gas cooking. Low-dose exposure to indoor nitrogen dioxide through gas cooking might not contribute to increase the risk of asthma and respiratory symptoms in children.

Background and aims

Gas for cooking is commonly used across Europe with rates of use varying between and within countries. The European Community Respiratory Health Survey reported that gas-cooking-use percentages in Europe ranged from $<10\%$ in Sweden to more than 90% in the Netherlands. Across German cities, gas-cooking-use percentages varied between 12% and 49% (Jarvis et al., 1998). Gas cooking, together with gas heating, is a main source of indoor nitrogen dioxide (NO_2) (Koistinen et al., 2008). For this reason and because of the difficulty in obtaining indoor measurements of NO_2 (Cyrys et al., 2000; Topp et al., 2004), most epidemiological studies use gas cooking as a surrogate of indoor exposure to this pollutant.

Several experimental studies that investigated the effects of NO_2 on respiratory health in animals and humans found an adverse effect on respiratory health in acute exposure to doses of this pollutant that are higher than average indoor NO_2 concentrations in homes. These results are summarized in a WHO report (WHO, 2005). A systematic review on outdoor NO_2 and PM_{10} exposures and respiratory symptoms in asthmatic children could not draw a firm conclusion on the associations observed between the exposure to NO_2 and respiratory symptoms in these children (Weinmayr et al., 2010). Regarding indoor exposure to NO_2, population-based studies in children and adults have found inconsistent results on the association between asthma and emissions from gas cooking or heating, assessed by the presence of gas appliances (Dekker et al., 1991; Willers et al., 2006; Wong et al., 2004). In addition, studies examining the effects of these exposures on other respiratory outcomes, such as lower respiratory tract symptoms, general respiratory illnesses, or objective markers of lung function, did not show a more consistent picture (Belanger et al., 2006; Berkey et al., 1986; Corbo et al., 2001; De Bilderling et al., 2005; Dekker et al., 1991; Moshammer et al., 2010; Ponsonby et al., 2001; Willers et al., 2006). Nevertheless, the US Environmental Protection Agency recently took steps to strengthen the US National Ambient Air Quality Standard for NO_2 (US EPA, 2011).

To our knowledge, most studies assessing the effects of NO_2 or gas appliances' exposure on respiratory health are cross-sectional studies. In this study, we aimed to assess the effects of long-term exposure to gas cooking on the onset of asthma and respiratory symptoms, focusing on wheezing, in children from birth up to the age of 10 years participating in two birth cohorts in Germany.

Methods

Study design and participants

The study population consisted of two ongoing population-based birth cohorts conducted in Germany with the common focus on assessing the effects of environmental exposure and genetics on the development of the immune system and allergies: the influence of life-style factors on the development of the immune system and allergies in East and West Germany plus the influence of traffic emissions and genetics study (LISAplus), and the German Infant Study on the influence of Nutrition Intervention plus environmental and genetic influences on allergy development study (GINIplus). Children in both studies were recruited at birth, at similar time periods (1995–1999), in the same German regions (Munich and Wesel, LISAplus also included children from Leipzig and Bad Honnef), and following the same inclusion and exclusion criteria. Neonates displaying at least one of the following criteria were excluded from the study: preterm newborns (<37 gestational weeks), low birth weight (<2500 g), congenital malformation, symptomatic neonatal infection, antibiotic medication, hospitalization, or intensive medical care during neonatal period, as well as newborns from women with immune-related diseases, on long-term medication, or who suffered from drugs and/or alcohol abuse, and newborns from parents with nationalities other than German or who were not born in Germany. Written informed consent was obtained from all participants, and the study was approved by the local ethics committees in each participating region.

Briefly, for LISAplus, the parents of neonates admitted to maternity hospitals in Munich, Leipzig, Wesel, and Bad Honnef, Germany, were contacted. A total of 3097 healthy full-term neonates were recruited in the study between December 1997 and January 1999. Screening, recruitment, and exclusion criteria were described elsewhere (Heinrich et al., 2002; Zutavern et al., 2006).

In the GINIplus study, a total of 5997 full-term neonates born in Munich and Wesel were recruited between September 1995 and June 1998. At recruitment, after administering the first questionnaire, children with a family history of allergy were invited to participate in a nutritional intervention trial. Finally, 2252 children with a family history of allergy agreed to participate in the trial. A total of 3739 children without a family history of allergy or whose parents declined to participate in the trial were part of the observational group. The nutrition intervention consisted of the administration of three hydrolyzed formulas, the standard formula, and dietary recommendations. The intervention study was performed only during the first 12 months of life; after this period, no further intervention was applied to this group. A description of the study design has been published previously (Chen et al., 2009; Filipiak et al., 2007).

A total of 5078 (56% of the recruited participants) children were followed until the age of 10 years, 1761 in LISAplus and 3317 in GINIplus birth cohorts.

Follow-up in both studies was performed following the same protocols and questionnaires from birth to the age of 10 years. Children included in the study were not different from those not included regarding most characteristics, although participants had a higher parental education in both birth cohorts ($P < 0.05$).

Asthma and wheezing before the age of 10 years

Asthma and wheezing during the first 10 years of life were assessed using parental reported questionnaires. Doctor-diagnosed asthma was recorded yearly from the first to the 10th year of life. Ever having asthma was defined as a parental report of a doctor diagnosis of asthma in any of the administered questionnaires. A child was included in the never-asthma group, if a doctor-diagnosed asthma was never reported in any of the administered questionnaires and no information was missing for any of the years of study. According to this categorization, the number of children with information on ever asthma in the first 10 years of life was 4313.

Wheezing episodes were recorded yearly during the first 2 years of life and then at years 4, 6, and 10. Children were classified into three groups: never wheezing, transient or late onset of wheezing, and persistent wheezing. The 'never-wheezing' group included children for whom no wheezing episode in any of the surveys was reported; children included in the group of transient or late onset of wheezing were those who reported at least one wheezing episode before 3 years or between 3 and 10 years of age, but not during both periods. When parents reported wheezing episodes at both periods (before 3 years of age and between 3 and 10 years of age), children were included in the persistent wheezing group. According to this categorization, the number of children with information on wheezing episodes in the first 10 years of life was 4490.

Gas cooking and other respiratory health–related indoor factors

Information on the indoor factors assessed in the present study was taken from questionnaires administered to the parents from birth until the child was 10 years old. Assessed indoor factors were gas cooking, dampness, visible mold, pets at home, and environmental tobacco smoke (ETS).

Exposure to gas cooking was assessed at years 0, 1, 2, 4, 6, and 10 in LISAplus and at years 1, 2, and 10 in GINIplus. We summarized exposure during the whole study period in two categories: never exposed or ever exposed to gas cooking. Children included in the never-exposed group were those whose mothers reported not having gas stoves at home in all the surveys. Children classified as ever-exposed were those of mothers who answered they used gas for cooking in at least one of the surveys. Children were excluded from the analysis if their parents never reported using gas for cooking but information on this was missing in at least one questionnaire ($N = 244$).

Dampness and visible mold information was obtained at years 0, 1, 4, 6, and 10 in LISAplus and at years 1, 6, and 10 in GINIplus, and information on pet ownership in the home was obtained from each year from birth to 10 years old in both cohorts. The same procedure was followed to create dichotomous variables (never vs. ever exposed) for these variables. ETS exposure was evaluated as a four-category variable with data on maternal smoking during pregnancy and anybody smoking in the home during the first 10 years of life. Exposure was categorized as follows: never exposed, only pre-natal exposure, only post-natal exposure, and both pre- and post-natal exposure.

Potential confounders

In addition to indoor factors, other potential confounders were considered in the present study. Information on the sex of the child, study region, parental education, parental history of asthma and/or atopy, and having older siblings was obtained through a general questionnaire administered to the mothers at birth. Information on exclusive breast feeding during the first 4 months of life was obtained at the child's age of 6 months, and day care attendance was asked at the first- and second-year questionnaires.

Statistical analysis

Descriptive statistics of the study population and the exposure to gas for cooking and other indoor factors included frequencies and percentages. Crude associations were evaluated using chi-square test. Crude odds ratios (OR) and their 95% confidence intervals (CI) were obtained by logistic and multinomial regressions.

Multivariable logistic and multinomial regression models were developed for asthma and wheezing, respectively. All potential confounders with a P-value below 0.2 were included in the final logistic regression models. Exposure to gas cooking was included in all models, and the exposure to other indoor factors was added to the final models. Statistical analyses were conducted using STATA SE 10.0 statistical software (Stata Corporation, College Station, TX, USA).

Results

The population characteristics are briefly described in Table 1. The percentage of asthmatic children and children with parental report of persistent wheezing at the age of 10 years in the study population was 8.7% and 11.1%, respectively. Regarding the use of gas for cooking, 12.3% of the children had ever lived in a home with gas stoves in the kitchen.

Table 1 Description of the study population of the LISAplus and GINIplus cohorts who completed the 10-year follow-up (N = 5078)

	N (%)
Sex	
Male	2590 (51.0)
Female	2488 (49.0)
Missing	0 (0.0)
Parental education	
Low	303 (6.0)
Medium	1314 (25.9)
High	3236 (63.7)
Missing	225 (4.4)
Region	
München	2670 (52.6)
Leipzig	435 (8.6)
Bad Honnef	207 (4.1)
Wesel	1766 (34.8)
Missing	0 (0.0)
Exclusive breast feeding during the first 4 months	
No	2141 (42.2)
Yes	2779 (54.7)
Missing	158 (3.1)
Day care center attendance in the first 2 years	
No	4372 (86.1)
Yes	205 (4.0)
Missing	501 (9.9)
Older siblings	
No	2712 (53.4)
Yes	2355 (46.4)
Missing	11 (0.2)
Parental asthma and/or atopy	
Never	1423 (28.0)
Ever	3457 (68.0)
Missing	198 (3.9)
Asthma	
Never	3940 (77.6)
Ever	373 (7.4)
Missing	765 (15.1)
Wheezing	
Never	2759 (54.3)
Transient or late onset	1234 (24.3)
Persistent wheezing	497 (9.8)
Missing	588 (11.6)
Gas cooking	
Never	4241 (83.5)
Ever	593 (11.7)
Missing	244 (4.8)

The associations between gas cooking exposure and the other assessed indoor factors are shown in Table 2. Basically adjusted OR for cohort and region showed statistically significant associations (P-values < 0.05) between gas cooking and each of the studied indoor factors. This association was higher for ETS exposure, especially for post-natal and pre- and post-natal exposures (OR: 1.69, 95% CI: 1.37–2.08; and 1.53, 95% CI: 1.14–2.05, respectively).

Table 3 shows the prevalence of doctor-diagnosed asthma and reported wheezing during the first 10 years of life according to gas cooking exposure and the other studied indoor factors. The unadjusted prevalence of asthma and persistent wheezing was higher among children exposed to gas cooking, but crude OR were not statistically significant. The prevalence of asthma and wheezing in the children exposed to dampness, mold, and ETS was also higher but only statistically significant for ETS exposed. Among children who ever had a pet in the house, prevalence of doctor-diagnosed asthma was significantly lower (P-value < 0.05).

Table 2 Associations [odds ratios (OR) and 95% confidence intervals (CI)] between gas cooking and other indoor factors

	Gas cooking		
	Never, N (%)	Ever, N (%)	OR (95% CI)[a]
Dampness			
Never	3711 (90.7)	479 (86.5)	1
Ever	382 (9.3)	75 (13.5)	1.40 (1.07–1.83)
Visible mold			
Never	2008 (50.8)	219 (40.6)	1
Ever	1942 (49.2)	320 (59.4)	1.27 (1.05–1.53)
Pets			
Never	1888 (47.6)	234 (43.7)	1
Ever	2075 (52.4)	302 (56.3)	1.21 (1.00–1.45)
Environmental tobacco smoke			
Never	2260 (56.2)	268 (49.3)	1
Only pre-natal	108 (2.7)	15 (2.8)	1.07 (0.61–1.88)
Only post-natal	1222 (30.4)	194 (35.7)	1.69 (1.37–2.08)
Pre- and post-natal	434 (10.8)	67 (12.3)	1.53 (1.14–2.05)

[a]Adjusted for cohort and region.

Associations between asthma and gas cooking as well as between wheezing and gas cooking at three different levels of adjustment (I, II, and III) are shown in Table 4. Adjustment I was a basic adjustment for cohort, region, sex, and parental education; adjustment II included parental atopy, breast feeding, and day care attendance; and adjustment III also included the other indoor factors (dampness, visible mold, pets at home, and ETS). The detailed adjustments (adjustments II and III) did not substantially change either the crude or the basically adjusted (adjustment I) effect estimates for gas cooking and both outcomes.

Table 5 shows adjusted OR for gas cooking and doctor-diagnosed asthma and persistent wheezing stratified by dampness, mold, ETS, and pets. Adjusted OR among those exposed to dampness and mold were higher than among those non-exposed, while in groups exposed to ETS and pets, these OR were lower than in the non-exposed groups, although differences were not statistically significant. Additional analyses indicated that there was no statistically significant interaction between gas cooking and each of the other indoor factors investigated.

Discussion

In the present study, we did not observe a statistically significant effect of exposure to gas cooking during the first 10 years of life on the development of asthma and persistent wheezing during that period. However, we found a strong significant association between the use of gas for cooking and other indoor factors (dampness,

Table 3 Description (number and percentage) of doctor-diagnosed asthma and wheezing before the age of 10 years, according to gas cooking and other indoor factors' exposures. Crude associations [odds ratios (OR) and 95% confidence intervals (CI)] between health outcomes and indoor factors

	Asthma ever			Wheezing				
		Ever			Transient early or late onset		Persistent	
	Never, *N* (%)	*N* (%)	OR (95% CI)	Never, *N* (%)	*N* (%)	OR (95% CI)	*N* (%)	OR (95% CI)
Gas cooking								
Never	3353 (91.61)	307 (8.39)	1	2400 (62.32)	1041 (27.03)	1	410 (10.65)	1
Ever	438 (90.87)	44 (9.13)	1.10 (0.80–1.50)	306 (58.96)	152 (29.29)	1.15 (0.93–1.41)	61 (11.75)	1.17 (0.87–1.57)
Dampness								
Never	3420 (91.74)	308 (8.26)	1	2445 (62.42)	1060 (27.06)	1	412 (10.52)	1
Ever	357 (89.70)	41 (10.30)	1.30 (0.90–1.80)	239 (56.10)	138 (32.39)	1.33 (1.07–1.66)	49 (11.50)	1.22 (0.88–1.68)
Visible mold								
Never	1800 (91.84)	160 (8.16)	1	1343 (65.35)	497 (24.18)	1	215 (10.46)	1
Ever	1844 (91.2)	178 (8.80)	1.09 (0.87–1.36)	1226 (57.94)	649 (30.67)	1.43 (1.24–1.65)	241 (11.39)	1.23 (1.01–1.5)
Environmental tobacco smoke								
Never	2200 (92.83)	170 (7.17)	1	1580 (63.58)	644 (25.92)	1	261 (10.50)	1
Only pre-natal	105 (92.11)	9 (7.89)	1.11 (0.55–2.23)	74 (61.67)	27 (22.50)	0.90 (0.57–1.40)	19 (15.83)	1.55 (0.92–2.62)
Only post-natal	1109 (89.58)	129 (10.42)	1.51 (1.18–1.91)	787 (61.10)	370 (28.73)	1.15 (0.99–1.35)	131 (10.17)	1.01 (0.80–1.26)
Pre- and post-natal	380 (90.91)	38 (9.09)	1.29 (0.90–1.87)	248 (56.24)	140 (31.75)	1.38 (1.10–1.74)	53 (12.02)	1.29 (0.94–1.79)
Pets at home								
Never	1811 (90.64)	187 (9.36)	1	1320 (63.22)	539 (25.81)	1	229 (10.97)	1
Ever	1933 (92.98)	146 (7.02)	0.73 (0.58–0.92)	1315 (59.99)	639 (29.15)	1.19 (1.04–1.37)	238 (10.86)	1.04 (0.86–1.27)

Table 4 Adjusted odds ratios (OR) and 95% confidence intervals (CI) for asthma and persistent wheezing in relation to the indoor factors

	Asthma			Persistent wheezing		
	Adj I *N* = 4079 OR (95% CI)	Adj II *N* = 3645 OR (95% CI)	Adj III *N* = 3222 OR (95% CI)	Adj I *N* = 4299 OR (95% CI)	Adj II *N* = 3845 OR (95% CI)	Adj III *N* = 3387 OR (95% CI)
Gas cooking						
Never	1	1	1	1	1	1
Ever	1.23 (0.87–1.74)	1.33 (0.92–1.93)	1.33 (0.88–2.00)	1.12 (0.83–1.53)	1.22 (0.88–1.68)	1.09 (0.76–1.57)
Dampness						
Never			1			1
Ever			1.23 (0.79–1.90)			1.30 (0.89–1.9)
Mold						
Never			1			1
Ever			1.16 (0.87–1.53)			1.11 (0.87–1.43)
Environmental tobacco smoke						
Never			1			1
Only pre-natal			1.23 (0.55–2.75)			1.11 (0.57–2.16)
Only post-natal			1.19 (0.87–1.61)			0.92 (0.69–1.22)
Pre- and post-natal			0.85 (0.51–1.41)			1.26 (0.84–1.87)
Pets at home						
Never			1			1
Ever			0.69 (0.52–0.91)			1.05 (0.83–1.33)

Adj I: adjusted for cohort, region, sex, and parental education; Adj II: adjusted for the same variables in Adj I plus parental atopy, exclusive breast feeding during the first 4 months of life, and day care center attendance in the first 2 years; Adj III: adjusted for the same variables in Adj II plus dampness, mold, maternal smoking, and pets.

visible mold, ETS, and pets) that highlights the importance of considering other indoor factors when assessing the effects of gas cooking on health outcomes.

Studies assessing the association between the use of gas stoves for cooking or the indoor measures of NO_2 and children's respiratory health have not found clear significant associations between respiratory outcomes and this exposure (Heinrich, 2011). A Canadian study found a statistically significant association between the use of gas for cooking and asthma (Dekker et al., 1991), and a study performed in the UK also found an association between gas cooking and wheezing (De Bilderling et al., 2005). On the other hand, a study in the PIAMA birth cohort (in the Netherlands) could not find any significant association between exposure and both outcomes (Willers et al., 2006). Studies conducted in the United States and Australia observed an increased risk of asthma symptoms in asthmatic children (Hansel et al., 2008; Nitschke et al., 2006), and others found an increased risk of asthma symptoms and reductions in lung function also in asthmatic population, only when stratifying by either housing characteristics,

Table 5 Gas cooking exposure adjusted odds ratios (OR) and 95% confidence intervals (CI) for asthma and persistent wheezing stratified by other indoor factors

		Asthma ever		Persistent wheezing	
	Total (*N*)	*N*	OR (95% CI)[a]	*N*	OR (95% CI)[a]
Dampness					
Never	4263	308	1.33 (0.88–2.02)	412	1.07 (0.74–1.55)
Ever	478	41	1.94 (0.69–5.47)	49	1.77 (0.70–4.48)
Visible mold					
Never	2252	160	1.25 (0.67–2.31)	215	1.11 (0.66–1.85)
Ever	2360	178	1.40 (0.85–2.31)	241	1.25 (0.80–1.95)
Dampness and/or mold					
Never	2203	155	1.29 (0.69–2.39)	208	1.07 (0.63–1.82)
Dampness or mold	1829	139	1.49 (0.83–2.68)	185	1.06 (0.62–1.80)
Dampness and mold	436	35	2.36 (0.81–6.88)	41	1.61 (0.60–4.30)
Environmental tobacco smoke					
Never	2571	170	1.63 (1.00–2.67)	261	1.37 (0.89–2.11)
Ever	2128	176	1.07 (0.60–1.92)	203	0.87 (0.51–1.48)
Pets at home					
Never	2172	187	1.47 (0.90–2.42)	229	1.28 (0.79–2.07)
Ever	2503	146	1.07 (0.57–2.03)	238	1.07 (0.66–1.72)

[a]Adjusted for cohort, region, sex and parental education, parental atopy, exclusive breast feeding during the first 4 months of life, day care center attendance in the first 2 years.

sex, or atopy (Belanger et al., 2006; Chapman et al., 2003; Kattan et al., 2007). Small reductions in lung function parameters have also been described in non-asthmatic specific population subgroups, such as sensitized or female children (Corbo et al., 2001; Moshammer et al., 2010; Ponsonby et al., 2001).

Most of these referenced studies had a cross-sectional design that might lead to a misclassification bias in the results. Moreover, not all the studies adjusted for ETS and furry pets at home, and very few considered dampness and/or mold as potential confounders. To our knowledge, only one longitudinal study assessed the respiratory effects of the co-exposure to NO_2 and dog allergens on incident asthma from 1 to 7 years of age, but this study was based on a high-risk birth cohort (Carlsten et al., 2011).

In our longitudinal study that included two birth cohorts with participants from four different regions in Germany, data were collected following similar protocols in both cohorts. Exposure and health data included in the present study were collected longitudinally in several surveys. Thus, we have repeated information on the occurrence of each of the studied health outcomes during the 10 years of follow-up, and a good estimate of the presence or absence of gas for cooking and other indoor factors in the home over time. This longitudinal design allows us to have a very well-defined group of children who were never exposed to gas cooking. Unfortunately, although the study protocols in both cohorts were similar, information on the use of gas for cooking at ages 4 and 6 years, and on the other indoor factors at the age of 4 years, was not collected in the GINIplus study. Therefore, LISAplus had additional information on the exposure variables. Analyses excluding the LISAplus information that was not available for the GINIplus subjects showed increased risk of asthma and wheezing.

In addition, information on certain kitchen characteristics that can determine the intensity of the exposure, such as the use of a fan for air extraction or the effectiveness of kitchen ventilation (Dennekamp et al., 2001; Hölscher et al., 2000), information on other sources of cooking-related indoor pollution, and information on the amount of time the child spent in the kitchen were not obtained. As a result, we cannot exclude that some individuals with very low exposure to gas cooking because of high effective ventilation and/or short time spent in the kitchen were included in the same exposure category as the individuals without effective kitchen ventilation and/or large amount of time spent in the kitchen while cooking. Also, all individuals in both groups, exposed or non-exposed to gas cooking, may have been exposed to other sources of indoor pollutants related to cooking, such as particulate matter from food. However, we had information on potential sources of outdoor air pollution such as self-reported proximity to roadway that could act as a confounder in the assessed associations; adding this variable to the adjusted models did not change the effect estimates for gas cooking (data not shown).

Owing to the small number of exposed individuals, we could not define a stricter group of those exposed including only children with reported exposure to gas cooking in all surveys. Also, it was not possible to define a more time-specific exposure; therefore, we cannot guarantee that the symptoms actually occur after the exposure. The small number of exposed individuals, together with the small number of children with doctor-diagnosed asthma and reported persistent wheezing, decreased the power of our study and might be affecting the statistical significance of the associations.

Nevertheless, the number of those exposed to gas cooking was not a limitation to assess the association between this indoor pollutant factor and other indoor factors known to increase the risk of respiratory illnesses and symptoms. We observed that exposure to other common indoor factors like dampness, mold, pets, and ETS was strongly and statistically significantly associated with exposure to gas cooking, suggesting that when studying a specific indoor exposure, one should also take into account other potential indoor factors. Additional analyses stratifying by each indoor factor showed changes in the association estimates of gas cooking and respiratory health outcomes (data not shown), although because of the small number of subjects, the obtained OR were not statistically significant.

In conclusion, results in our study did not show a statistically significant association between the exposure to gas cooking and children respiratory health. Nevertheless, we observed a significant association between the use of gas cooking and other reported indoor factors that could have an effect on the associations between gas cooking and respiratory health. More longitudinal studies considering other indoor factors associated with respiratory health are needed to confirm these results.

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