Indoor Air Pollution Sources and Respiratory Symptoms in Bronchopulmonary Dysplasia

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Objective To evaluate the impact of exposure to indoor air pollution on respiratory health outcomes (healthcare utilization, symptoms, medication use) in infants and children with bronchopulmonary dysplasia (BPD).

Study design A total of 244 subjects were included from the Johns Hopkins Bronchopulmonary Dysplasia registry. Parents completed an environmental exposure questionnaire including secondhand smoke and indoor combustion (gas/propane heat, gas or wood stove, gas/wood burning fireplace) exposures in the home. Respiratory symptoms, both acute (healthcare utilization, steroid/antibiotic use) and chronic (cough/wheeze, nocturnal cough, use of beta-agonists, tolerance of physical activity), were also collected.

Results Three-quarters of the infants were exposed to at least 1 combustible source of air pollution in the home, and this exposure was associated with an increased risk of hospitalization in infants and children on home respiratory support. Only 14% of the study population reported secondhand smoke exposure, but we found that this was associated with chronic respiratory symptoms, including activity limitation and nocturnal cough. Infants on respiratory support also had increased daytime cough and wheezing. Approximately one-third reported having an air purifier in the home, and its presence attenuated the effect of secondhand smoke exposure on reported activity limitation.

Conclusions Exposure to combustible sources of indoor air pollution was associated with increased respiratory morbidity in a group of high risk of infants with BPD. Our results support that indoor air pollution is a modifiable risk factor for respiratory health in infants with BPD. *(J Pediatr 2020;222:85-90)*.

Premature birth affects approximately 10% of infants born in the US and is the leading cause of infant mortality.[1] Bronchopulmonary dysplasia (BPD), characterized by impaired alveolar growth and lung injury, is one of the most common and serious complications of preterm birth, occurring in 40% of infants born between 22 and 28 weeks of gestation.[2,3] Although children with BPD can exhibit alveolar catch-up growth during childhood,[4] early-life exposures, such as to tobacco smoke, can adversely impact long-term trajectories in lung function.[5]

The US Environmental Protection Agency has concluded that fine particle pollution ($PM_{2.5}$) is a serious health threat based on its association with multiple adverse health effects.[6] For infants and children, exposure to indoor or outdoor sources of $PM_{2.5}$ has been linked to respiratory-related infant mortality,[7] development of asthma,[8] slowed lung function growth,[9] and increased asthma morbidity.[10] Exposure to nitrogen dioxide (NO_2), a gaseous air pollutant that results from combustion, also has harmful effects on the lungs, including increased inflammation, cough, and asthma exacerbations.[11]

Although secondhand smoke exposure (a major source of indoor air pollution when present) has been associated with increased hospitalizations for respiratory illnesses in infants and children with BPD,[12] little is known about the health effects of air pollution exposure specifically in this vulnerable population. Because management of these infants focuses on minimizing further lung injury, identifying and minimizing modifiable risk factors, such as environmental exposures, is of critical importance. In this study, we hypothesized that parent-reported exposures to indoor air pollution at home would be associated with increased healthcare utilization, symptoms, and medication use.

Methods

The subjects (n = 244) in this study were recruited from the Johns Hopkins Bronchopulmonary Dysplasia Clinic during routine follow-up visits between April 2016 and October 2018. The BPD Patient registry is a cohort of approximately 900 infants and children that actively collects respiratory

BPD	Bronchopulmonary dysplasia
NICU	Neonatal intensive care unit
$PM_{2.5}$	Fine particulate matter
NO_2	Nitrogen dioxide

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health data at each pulmonary follow up visit, thus providing an opportunity to investigate the influence of air pollution exposures on clinical outcomes. Patients seen in clinic were referred from at least 13 neonatal intensive care units (NICUs) across the state of Maryland. Inclusion criteria included being born preterm (<32 weeks of gestation) and being diagnosed with BPD according to National Institute of Child Health and Human Development criteria.[13] This study was approved by the Johns Hopkins Institutional Review Board (protocol #NA_051884). All parents and caregivers provided oral consent as required by the Institutional Review Board.

Clinical and demographic data were obtained through chart review. Race/ethnicity was self-reported; for the purpose of our analysis, subjects reporting any nonwhite ancestry were coded as nonwhite. Median household income was derived from 2010 US Census data based on residential ZIP code (median household income: $50 502 in the US; $70 004 in Maryland). Health insurance information was obtained from billing records. Birth weight percentiles reflect birth weight corrected for gestational age.[14]

The environmental exposure questionnaire is an unvalidated set of questions adapted from the Johns Hopkins Center for Childhood Asthma in the Urban Environment. Subjects were classified as having indoor smoking exposure if caregivers reported that smoking occurred inside the home or inside a shared building (eg, apartment building) within the past 3 months. Subjects were classified as having exposure to indoor combustion if caregivers reported the presence of a gas or propane heater, gas or wood stove, and/or gas or wood fireplace within the home. These exposures and the use of air purifiers were only determined to be present or absent; use patterns were not captured. Only 1 environmental questionnaire per subject was completed.

Acute respiratory outcomes (eg, emergency department visits and hospitalizations for respiratory symptoms, steroid courses, antibiotic courses for respiratory illnesses over the preceding 2 months) and chronic respiratory symptoms (eg coughing/wheezing, nighttime cough, use of short-acting beta-agonists at home, shortness of breath with play or activities over the past week) were collected via an unvalidated questionnaire at routine follow-up clinic visits. Currently, validated instruments for outpatients with BPD are not available. A total of 865 outcome questionnaires were completed for 244 subjects, with 2.1% of all questions left blank by caregivers (mean number of questionnaires per subject, 3.5 ± 3.6; range, 1-25).

Statistical Analyses

Differences for demographic and clinical factors between subjects exposed to indoor smoking and/or combustion compared with those who were not were assessed using the appropriate parametric tests (χ^2 and t tests). The relationships between respiratory morbidities (dependent variable) and self-reported environmental exposures (independent variable) were assessed using multivariable logistic regression to generate ORs adjusted for the age of the subject at the time of environmental and clinical questionnaire completion, and clinical/demographic factors that differed by self-reported exposures (specifically race/ethnicity and insurance status for indoor smoking and median household income for indoor combustion). Given that caregivers may have completed outcomes questionnaires at several clinic visits, the logistic regressions accounted for the possibility of more than 1 questionnaire per subject using generalized estimating equations methodology (clustered by subject).[15]

To assess the effects of the presence of an air purifier in the home, logistic regressions were rerun only for outcomes associated with exposures stratified by the presence of an air purifier. Kaplan-Meier methodology was used to analyze the time at which subjects on home supplemental oxygen via nasal cannula were weaned off oxygen, stratified by exposures and unadjusted for other factors. All statistical analyses were conducted using Stata IC 15.0 (StataCorp, College Station, Texas). P values <.05 were considered statistically significant.

Results

A total of 244 subjects completed environmental exposure questionnaires, at a mean age of 2.3 years at form completion (**Table I**). The cohort was 44.3% female, born at a mean gestational age of 26.8 weeks, and discharged to home for the first time at a mean age of 4.9 months. A total of 14.3% of caregivers reported indoor smoking exposure within the past 3 months, including 3.7% within the living space and 12.1% within a shared building (**Table II**). A total of 75.8% reported at least 1 combustion source indoors, with the most common sources being gas stoves (50.2%) and gas heaters (43.2%). Those exposed to indoor smoking were more likely to be nonwhite (80.0% vs 60.8%; P = .03) and covered by public insurance (71.4% vs 50.2%; P = .02) compared with those not exposed (**Table I**). Those exposed to indoor combustion had a higher median household income ($67 587 vs $60 438; P = .031) (**Table III**). There were no other significant differences in clinical or demographic factors between those exposed to smoking/combustion and those not exposed.

Respiratory Outcomes

Clustered logistic regressions adjusted for age at the time of form completion and other confounders were used to test for association between indoor smoking/combustion and acute/chronic respiratory outcomes (**Table IV**). Reported indoor smoking was found to be associated with several chronic respiratory symptoms, including a 2.0-fold increase in activity limitations (P = .05) and a 2.4-fold increase in nighttime symptoms (P = .03). These findings were more severe in subjects requiring respiratory support in the home setting (defined as oxygen, ventilator, and/or tracheostomy dependence at initial NICU discharge), with a 2.9-fold increase in coughing/wheezing (P = .01), a 4.3-fold increase in activity limitations (P = .004), and a 6.5-fold increase in nighttime symptoms (P = .001). Acute care use and rescue beta-agonist use were not found to be

Table I. Patient demographic data and clinical information by reported indoor smoking status

Characteristics	Entire population (n = 244)	Reported indoor smoking (n = 35)	No reported indoor smoking (n = 209)	*P* value
Demographics				
Age at form completion, y, mean ± SD (range)	2.3 ± 2.6 (0.2-16.4)	1.7 ± 1.7 (0.3-7.2)	2.4 ± 2.7 (0.2-16.4)	.12
Female sex, %	44.3	45.7	44.0	.85
Nonwhite race/ethnicity, %	63.5	80.0	60.8	**.029**
Gestation, wk, mean ± SD (range)	26.8 ± 2.4 (22.9-32)	26.2 ± 2.4 (23-32)	26.9 ± 2.3 (22.9-32)	.10
Birth weight, g, mean ± SD (range)	921 ± 354 (380-2310) (n = 243)	875 ± 310 (480-2188)	929 ± 361 (380-2310) (n = 208)	.41
Birth weight percentile, mean ± SD (range)	41.6 ± 25.8 (1-94) (n = 243)	48.9 ± 28.7 (2-94)	40.4 ± 25.1 (1-92) (n = 208)	.07
Median household income, $ 000s, mean ± SD (range)	65.9 ± 22.2 (21.9-132.7)	63.1 ± 19.7 (28.9-103.6)	66.3 ± 22.6 (21.9-132.7)	.42
Public insurance, %	53.3	71.4	50.2	**.020**
Clinical Data				
Age at NICU discharge, mo, mean ± SD (range)	4.9 ± 3.5 (0.5-26.5) (n = 243)	4.4 ± 2.5 (0.7-15.0)	4.8 ± 3.5 (0.5-26.5) (n = 208)	.49
Home supplemental oxygen, %	45.5	45.7	45.5	.98
Home ventilator, %	6.2	8.6	5.7	.52
Gastrostomy tube, %	32.0	34.3	31.6	.75
Ventricular shunt, %	10.3	8.6	10.5	.72
Inhaled corticosteroid use before age 2 y, %	81.6	82.9	81.3	.83
Air purifier in home, %	33.3 (n = 243)	31.4	33.7 (n = 208)	.80

Significant *P* values are in bold type.

associated with reported indoor smoking. Reported indoor combustion tended to be associated with an increased risk of inpatient hospitalization after initial NICU discharge (aOR, 2.2; $P = .06$), and this risk was again more pronounced in subjects requiring respiratory support at home (aOR, 6.0; $P = .04$) (Table IV).

Indoor Pollution Mitigation

To test whether the presence of an air purifier in the home affected outcomes, logistic regressions that were significant for associations between indoor air pollution and respiratory outcomes as observed above were rerun stratified by the presence of an air purifier. Within the study population, 33.3% of caregivers reported having an air purifier in the home. The previously observed association between indoor smoking and activity limitations/nighttime symptoms was not present when an air purifier was reported to be present (Table V; available at www.jpeds.com). Specifically, there was a 2.6-fold increased risk of activity limitations ($P = .01$) and a 2.3-fold increased risk of nighttime symptoms ($P = .06$) with indoor smoking when an air purifier was not present vs nonsignificant aORs for activity limitations ($P = .98$) and for nighttime symptoms ($P = .18$) when an air purifier was present. Likewise, similar findings were seen when examining only subjects on home respiratory support, with the presence of an air purifier appearing to mitigate associations (Table VI; available at www.jpeds.com). Specifically, there was a 2.3-fold increased risk of cough/wheeze ($P = .08$) and a 3.7-fold increased risk of activity limitations ($P = .02$) with indoor smoking when an air purifier was not present vs nonsignificant ORs for cough/wheeze ($P = .18$) and activity limitations ($P = .11$) when an air purifier was present. Reported indoor smoking remained associated with a >6-fold risk of nighttime symptoms in subjects on home respiratory support irrespective of the presence ($P = .05$) or absence ($P = .005$) of an air purifier. Because all subjects on respiratory support reporting indoor combustion and the presence of an air purifier also reported hospitalization, the regressions could not be performed for this outcome and exposure. Although questionnaires did ascertain whether a stove exhaust fan was present (as a mitigating factor for indoor combustion from gas stoves), of the 121 caregivers who reported the presence of a gas stove, the majority (88.4%) also reported the presence of a stove exhaust fan; thus, any such analyses likely were underpowered.

Table II. Indoor environmental exposures (N = 244)

Exposures	Value, %
Smoking exposures	
Smoking inside living space within the past 3 mo	3.7
Smoking inside shared building within the past 3 mo (n = 224)	12.1
Any reported smoking inside	14.3
Combustion exposures	
Gas stove (n = 243)	50.2
Wood stove (n = 243)	0.4
Gas heater (n = 220)	43.2
Propane heater (n = 220)	2.7
Gas fireplace (n = 243)	11.5
Wood fireplace	17.6
Any of the above combustion sources	75.8
Air purifier present in the home (n = 243)	33.3

Oxygen Weaning

To assess whether sources of indoor air pollution were associated with duration of home supplemental oxygen use, Kaplan-Meier methodology was used. Of the 98 subjects discharged to home on supplemental oxygen via nasal cannula, 85 were weaned off of supplemental oxygen during follow-

Table III. Patient demographic data and clinical information by reported indoor combustion sources

Characteristics	Entire population (n = 244)	Reported indoor combustion (n = 185)	No reported indoor combustion (n = 59)	*P* value
Demographics				
Age at form completion, y, mean ± SD (range)	2.3 ± 2.6 (0.2-16.4)	2.5 ± 2.7 (0.2-16.4)	1.8 ± 2.1 (0.2-9.2)	.10
Female sex, %	44.3	41.6	52.5	.14
Nonwhite race/ethnicity, %	63.5	62.7	66.1	.64
Gestation, wk, mean ± SD (range)	26.8 ± 2.4 (22.9-32)	26.8 ± 2.4 (22.9-32)	26.8 ± 2.2 (23-31.3)	.88
Birth weight, g, mean ± SD (range)	921 ± 354 (380-2310) (n = 243)	935 ± 364 (380-2310) (n = 184)	879 ± 317 (380-1800)	.29
Birth weight percentile, mean ± SD (range)	41.6 ± 25.8 (1-94) (n = 243)	42.3 ± 25.5 (1-94); (n = 184)	39.3 ± 26.7 (2-89)	.44
Median household income, $ 000s, mean ± SD (range)	65.9 ± 22.2 (21.9-132.7)	67.6 ± 22.3 (25.2-132.7)	60.4 ± 21.2 (21.9-108.1)	**.031**
Public insurance, %	53.3	54.1	50.9	.67
Clinical Data				
Age at NICU discharge, mo, mean ± SD (range)	4.9 ± 3.5 (0.5-26.5) (n = 243)	4.9 ± 3.5 (0.5-26.5) (n = 184)	4.4 ± 2.7 (0.9-14.3)	.30
Home supplemental oxygen, %	45.5	46.0	44.1	.80
Home ventilator, %	6.2	6.0	6.8	.82
Gastrostomy tube, %	32.0	34.6	23.7	.12
Ventricular shunt, %	10.3	11.9	5.1	.13
Inhaled corticosteroid use before age 2 y, %	81.6	81.6	81.4	.96
Air purifier in home, %	33.3 (n = 243)	34.2 (n = 184)	30.5	.60

Significant *P* values are in bold type.

up; censored data were used for the remaining 13. The median time from NICU discharge to weaning from oxygen was 5.8 months (IQR, 3.0-12.4 months). No differences in length of time to oxygen weaning were observed with either indoor smoking ($P = .53$, log-rank test) or indoor combustion ($P = .76$, log-rank test) (**Figures 1** and **2**; available at www.jpeds.com).

Discussion

Our study found that most of the infants (76%) were exposed to at least 1 combustible source of air pollution in the home (primarily gas stoves and heaters). Exposure to any combustible source of indoor air pollution was associated with increased risk of hospitalization in infants and children on home respiratory support. Although only 14% of the study population reported a smoker residing in the home, we found that secondhand smoke exposure was associated with chronic respiratory symptoms, including activity limitations and nocturnal symptoms. Infants on respiratory support also had increased daytime cough and wheezing. There was no association between air pollution exposure in the home and the duration of supplemental oxygen use at home.

These findings are significant because there are currently no targeted long-term therapies for treating BPD, and thus

Table IV. Reported indoor smoking as a predictor of selected respiratory outcomes

	All subjects			Subjects on home respiratory support*		
Outcomes	OR† (95% CI)	Number	*P* value	OR (95% CI)	Number	*P* value
Emergency department visit	1.40 (0.67-2.92)	244 (860 forms)	.37	1.54 (0.66-3.60)	114 (449 forms)	.32
Inpatient hospitalization	0.97 (0.43-2.21)	244 (860 forms)	.95	1.19 (0.46-3.04)	114 (449 forms)	.72
Systemic steroid use	1.23 (0.56-2.72)	244 (857 forms)	.61	1.37 (0.51-3.65)	114 (446 forms)	.54
Antibiotic use	1.64 (0.72-3.75)	244 (852 forms)	.24	1.70 (0.47-6.18)	114 (443 forms)	.42
Cough or wheeze	1.45 (0.77-2.74)	243 (852 forms)	.25	2.89 (1.24-6.74)	114 (446 forms)	**.014**
Rescue β-agonist use	1.78 (0.88-3.61)	244 (829 forms)	.11	2.31 (0.85-6.24)	114 (434 forms)	.10
Activity limitations	1.98 (1.00-3.91)	244 (817 forms)	**.050**	4.26 (1.59-11.41)	114 (427 forms)	**.004**
Nighttime symptoms	2.43 (1.12-5.29)	244 (850 forms)	**.025**	6.45 (2.05-20.26)	114 (444 forms)	**.001**
Reported indoor combustion sources as a predictor of selected respiratory outcomes						
Emergency department visit	1.60 (0.84-3.06)	244 (860 forms)	.16	2.42 (0.92-6.37)	114 (449 forms)	.07
Inpatient hospitalization	2.20 (0.96-5.05)	244 (860 forms)	.06	5.95 (1.08-32.76)	114 (449 forms)	**.040**
Systemic steroid use	1.34 (0.86-2.07)	244 (857 forms)	.20	1.44 (0.71-2.95)	114 (446 forms)	.31
Antibiotic use	0.84 (0.48-1.50)	244 (852 forms)	.57	1.78 (0.77-4.15)	114 (443 forms)	.18
Cough or wheeze	1.21 (0.76-1.92)	243 (852 forms)	.42	1.39 (0.66-2.94)	114 (446 forms)	.39
Rescue β-agonist use	1.02 (0.65-1.61)	244 (829 forms)	.92	0.85 (0.43-1.69)	114 (434 forms)	.64
Activity limitations	1.18 (0.74-1.88)	244 (817 forms)	.50	0.97 (0.44-2.11)	114 (427 forms)	.94
Nighttime symptoms	1.64 (0.93-2.89)	244 (850 forms)	.09	2.24 (0.60-8.45)	114 (444 forms)	.23

Significant *P* values are in bold type.

*Of the 244 subjects in this study, 98 were on supplemental oxygen via nasal cannula, 15 were on home ventilators, and 1 had a tracheostomy without ventilator or oxygen use. ORs were calculated in a similar manner to the all-subjects regressions.

†ORs for respiratory outcomes (dependent variable) given reported indoor smoking (independent variable) were generated through logistic regression clustered by subject and adjusted for potential confounders, including race/ethnicity, insurance status, age at the time of environmental assessment, and age at the time of respiratory outcomes questionnaire completion.

management is focused on supportive respiratory care, reducing further lung injury, and optimizing nutrition to encourage lung growth.[16] Identification of modifiable risk factors is important, because intervention during this critical time of growth and development may lead to improved outcomes in respiratory health and lung function. Our results suggest that the impact may be even greater for the highest-risk infants on respiratory support, because as reducing air pollution exposure in the home may reduce expensive hospitalizations.

The limited data available regarding the effects of secondhand smoke and other sources of indoor air pollution exposure in premature infants are mixed. Several authors, including a previous study in this cohort, have found that secondhand smoke exposure is associated with respiratory illnesses necessitating acute care, recurrent wheezing, and activity limitations,[12,17] but others have not corroborated these results. Martinez et al found that despite high exposure to secondhand smoke in a population of infants with BPD living in France, exposure was not associated with a diagnosis of asthma, the need for asthma medication, supplemental oxygen use, or hospitalization for respiratory illness in the first 2 years of life. Because smoke exposure was based on parental report, this might have underestimated the number of children truly exposed.[18] In a 2014 sample of our registry population, we found that respiratory outcomes were not associated with secondhand smoke exposure. The survey used in that study might have underestimated the number of exposed participants because parents were asked, "is there a smoker living in your home?" In our updated questionnaire, we asked whether smoking occurred inside the home or inside a shared building (eg, apartment building) within the past 3 months, because children not living with smokers in multiunit housing have higher nicotine metabolite levels than those living in detached homes.[19] The 2014 study also noted that infants who were exposed to secondhand smoke were more likely to be on inhaled corticosteroids, which might have mitigated their symptoms.[20]

In a study that evaluated combustible sources of indoor air pollution on the respiratory health in very low birth weight infants, authors reported that living with a smoker and exposure to pests was associated with increased acute care visits, but having a wood stove was not.[21] Other infant cohorts evaluating the association of indoor NO_2 exposure and early-life respiratory symptoms also found negative results.[22,23] In a 2013 meta-analysis, the authors concluded that indoor NO_2 exposure was associated with current wheeze, and gas cooking increased the risk of asthma. Only 4 of the 41 included studies were completed in infants; the remainder were in school-aged children with and without asthma.[24] There are fewer studies measuring indoor $PM_{2.5}$ in infant populations, but Hunt et al demonstrated that in a full-term infant cohort at risk for asthma, a $PM_{2.5}$ level >15 $\mu m/m^3$ was associated with increased risk of wheezing during the first year of life.[25]

In terms of potential mitigation of indoor air pollution, approximately one-third of the study population reported having an air purifier in the home. In a previous study investigating household activities associated with particulate matter concentrations in the homes of 300 children with asthma in Baltimore, only 1% of the study population reported using an air purifier.[26] In our study, the presence of an air purifier attenuated the effect of secondhand smoke exposure on reported activity limitation. The use of a high-efficiency particulate air indoor air purifier has been shown to be effective in reducing indoor air pollution, resulting in decreased asthma symptoms,[27] and this intervention may merit further study in homes of high-risk infants with chronic lung disease.

Our study is limited by the fact that parents may underreport their child's exposure to secondhand smoke and that our unvalidated home characteristics questionnaire might not accurately predict levels of indoor particulate matter, because it does not quantify gas stove, fireplace, exhaust fan, or air purifier use. In this study, only 14% of caretakers reported smoking in the home. A previous study in this registry population found that 20%-28% of parents reported smoking in the home but in a follow-up study, hair nicotine analysis found that almost 50% of participants may have been misclassified when relying on parent questionnaire.[12,20] It also may be necessary in future survey design to include a question about a child's known exposure to secondhand smoke outside the home.[28] We also did not collect any data on in utero smoking exposures or distinguish between secondhand and thirdhand exposures, which might impact outcomes.

In summary, we have shown that combustible sources of indoor air pollution (eg, gas stoves, fireplaces, and tobacco smoke exposure) were associated with increased respiratory morbidity in a group of high-risk infants born prematurely with BPD. Our data suggest that the use of an air purifier may attenuate some of the adverse effects of secondhand smoke exposure. Our results support that indoor air pollution is a somewhat modifiable risk factor for respiratory health in infants with BPD. Future research should consider measuring indoor air pollution and secondhand smoke exposure directly, given that parent-reported exposures may lead to misclassification bias. The use of air purifiers may be a method of reducing indoor air pollution short of eliminating exposure sources, but clinical trials are needed to evaluate the clinical impact of this intervention. ■

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Table V. Presence of air purifiers as a modifier of selected respiratory outcomes

	No air purifier			Air purifier		
Outcomes	**OR* (95% CI)**	**Number**	***P* value**	**OR (95% CI)**	**Number**	***P* value**
Indoor smoking						
Activity limitations	2.56 (1.21-5.40)	162 (539 forms)	**.014**	1.03 (0.18-5.88)	81 (274 forms)	.98
Nighttime symptoms	2.27 (0.96-5.38)	162 (563 forms)	.06	2.83 (0.62-12.85)	81 (283 forms)	.18

Of the 244 subjects in this study, 162 reported no air purifier in the home, 81 reported having one, and 1 subject was missing this data. Significant *P* values are in bold type.
*ORs for respiratory outcomes (dependent variable) given reported indoor smoking (independent variable) were generated through logistic regression clustered by subject and adjusted for potential confounders, including race/ethnicity, insurance status, age at the time of environmental assessment, and age at the time of respiratory outcomes questionnaire completion.

Table VI. Presence of air purifiers as a modifier of selected respiratory outcomes for subjects on respiratory support

	No air purifier			Air purifier		
Outcomes	**OR* (95% CI)**	**Number**	***P* value**	**OR (95% CI)**	**Number**	***P* value**
Indoor smoking						
Cough or wheeze	2.35 (0.91- 6.07)	82 (307 forms)	.08	2.98 (0.61-14.62)	32 (139 forms)	.18
Activity limitations	3.67 (1.29-10.41)	82 (292 forms)	**.015**	5.06 (0.69-36.87)	32 (135 forms)	.11
Nighttime symptoms	6.85 (1.79-26.17)	82 (303 forms)	**.005**	6.10 (1.02-36.50)	32 (141 forms)	**.047**
Indoor combustion						
Inpatient hospitalization	3.18 (0.61-16.46)	82 (5305 forms)	.17	All subjects reporting combustion were hospitalized		

Of the 244 subjects in this study, 162 reported no air purifier in the home, 81 reported having one, and 1 subject was missing this data. Of the 244 subjects in this study, 98 were on supplemental oxygen via nasal cannula, 15 were on home ventilators, and 1 had a tracheostomy without ventilator or oxygen use. ORs were calculated in a similar manner to the all-subjects regressions. Significant *P* values are in bold type.
*ORs for respiratory outcomes (dependent variable) given reported indoor smoking (independent variable) were generated through logistic regression clustered by subject and adjusted for potential confounders, including race/ethnicity, insurance status, age at the time of environmental assessment, and age at the time of respiratory outcomes questionnaire completion. ORs for respiratory outcomes (dependent variable) given reported indoor combustion (independent variable) were generated through logistic regression clustered by subject and adjusted for potential confounders, including log of median household income, age at the time of environmental assessment, and age at the time of respiratory outcomes questionnaire completion.

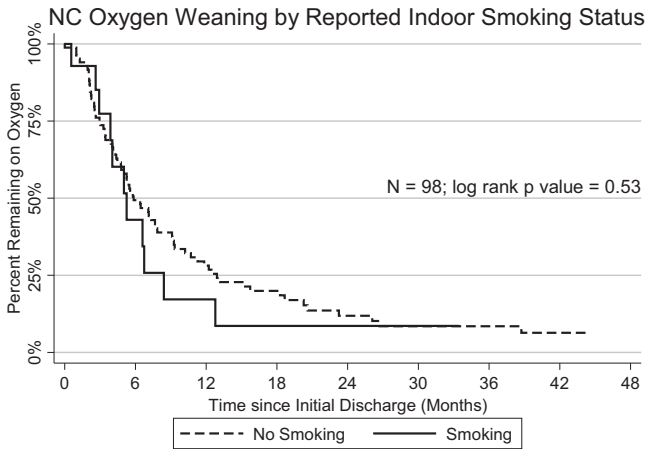

Figure 1. Kaplan-Meier plot depicting age at weaning off nasal cannula oxygen by reported indoor smoking status within the previous 3 months.

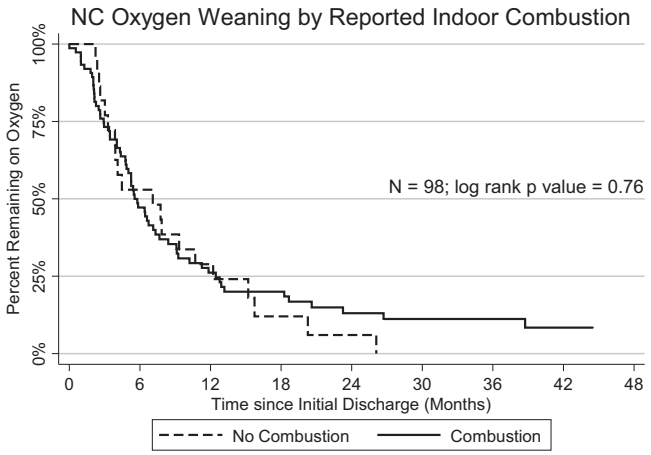

Figure 2. Kaplan-Meier plot depicting age at weaning off nasal cannula oxygen by reported indoor combustion exposure status.