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The Relationship Between PM2.5 and Chronic Respiratory Disease in Senegal

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THE RELATIONSHIP BETWEEN $PM_{2.5}$ AND CHRONIC RESPIRATORY DISEASE IN SENEGAL

A Thesis Presented

by

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DEDICATION

To Susan Foster and John Baker,

Inspiring scientists, dedicated professors, and invaluable mentors.

Gone but never forgotten.

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ABSTRACT

THE RELATIONSHIP BETWEEN $PM_{2.5}$ AND CHRONIC RESPIRATORY DISEASE IN SENEGAL

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Chronic respiratory diseases such as asthma and chronic bronchitis have significantly increased in prevalence in Africa over the past 10 years. Recent studies have demonstrated that exposure to air pollution may be associated with an increased risk of chronic respiratory diseases. However, such studies have predominantly been conducted in western societies or often used urbanicity as a proxy for exposure to air pollution. Therefore, we evaluated the association between $PM_{2.5}$ exposure and asthma/chronic bronchitis in Senegal. A cross-sectional study was conducted for the time period of 3 October 2010 to 28 April 2011 using annual concentrations of $PM_{2.5}$ measured via multiple satellite instruments, and asthma/chronic bronchitis, which was self-reported at baseline via a health survey questionnaire. We used mixed model logistic regression to evaluate the relationship between $PM_{2.5}$ exposure and asthma/chronic bronchitis risk while adjusting for lifestyle factors, location, and other air pollutants. Sex was evaluated as an effect modifier. The adjusted association between $PM_{2.5}$ and asthma/chronic bronchitis was 1.03 (95%CI: 0.99 – 1.06). In males the adjusted odds ratio was 1.09 (95%CI: 1.03-1.15), compared to females (aOR 1.01 (95%CI: 0.97 – 1.05). Our results suggest that increasing levels of exposure to $PM_{2.5}$ puts individuals at a higher risk for

chronic respiratory diseases, especially men. These findings have significant policy implications and should be built upon in future research.

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CHAPTER 1

INTRODUCTION

1.1 Public Health Impact

Asthma is a common chronic respiratory disease worldwide.[1] In 2019, there were 262 million prevalent cases, 37 million incident cases and 461,000 deaths attributed to asthma.[2] While estimates for Senegal are not readily available due to lack of research, there is evidence that the prevalence of asthma in Africa increased from roughly 74.4 million in 1990 to 119.5 million cases in 2010.[3]

While the exact cause of asthma remains unknown, asthma attacks are often the result of an immune response to a substance or allergen that entered the lungs.[4] If left untreated, lung scarring can occur as well as severe and sometimes fatal asthma attacks that prevent sufficient oxygen intake leading to death through suffocation.[5] There are multiple risk factors for asthma, some of which are modifiable while others are not. Unmodifiable risk factors of asthma include family history and allergies.[6] Modifiable risk factors include prior respiratory infections, occupational exposure to dust, chemical fumes, and mold, smoking, obesity, and air pollution.[6] While air pollution such as ozone or living in urban areas has been identified as a risk factor in western societies, the impact of the dose of such exposures in Africa are less clear. Therefore, air pollution, specifically $PM_{2.5}$ will be the exposure of interest for this study.

Chronic Obstructive Pulmonary Disease (COPD) is an overarching medical term that refers mainly to chronic bronchitis and emphysema. Due to the absence of statistics that solely refer to chronic bronchitis, COPD statistics will be reported. While estimates vary, COPD has a high prevalence worldwide at about 10% of the adult population.[7] In

addition, in 2019, COPD caused over 3.2 million deaths[8], and was the third leading cause of death worldwide, with 90% of COPD deaths occurring in low and middle income countries.[9] While studies on COPD are sparse in Senegal, as well as Africa, a systematic review of COPD prevalence in Africa estimates the COPD prevalence in 2010 to be 26.3 million, a 31.5% increase since 2000.[10]

Chronic bronchitis refers to long term inflammation of the bronchi.[10] Chronic bronchitis is progressive and becomes worse over time, allowing less and less oxygen into the lungs, leading to difficulty breathing and potential suffocation which can result in death.[11] The main risk factor for COPD is smoking, but other risk factors include exposure to air pollution, secondhand smoke, exposure to chemical fumes, alpha-1 deficiency (a genetic condition) and history of childhood respiratory infections.[12]

1.2 Physiology of Relationship

$PM_{2.5}$ refers to particulate matter that has a diameter less than 2.5 micrometers and is known to be a measure of fine particle air pollution. $PM_{2.5}$ when inhaled can get deep into the lungs, irritating and corroding the alveolar wall and therefore inhibiting lung function, leading to chronic respiratory diseases.[13] There are three proposed mechanisms in which this can occur: **(1)** injury from free radical peroxidation where $PM_{2.5}$ can induce free radical production to oxidize lung cells which can cause lung damage and inhibit lung function.[13] **(2)** Imbalanced intracellular calcium homeostasis: $PM_{2.5}$ can increase calcium concentration in cells which activates an inflammatory reaction causing limited lung function and chronic respiratory diseases over time.[13] **(3)** Inflammatory injury: $PM_{2.5}$ stimulates overexpression of inflammatory cytokines in the

lungs, leading to inflammation that inhibits lung function.[13]All three mechanisms lead to cellular damage and or inflammation. With continuous exposure and therefore continued inflammation in the respiratory system, chronic respiratory diseases such as asthma or chronic bronchitis can develop.

1.3 Epidemiology of the Relationship

One study evaluated $PM_{2.5}$ and asthma and chronic bronchitis in Senegal, however this study was observational and did not conduct statistical analyses to determine that relationship between the two variables.[14] Looking more broadly, in Sub-Saharan Africa only one study has evaluated the association between PM 2.5 and asthma,[15] while two studies evaluated the association between PM 2.5 and lung function parameters,[16,17] and seven studies evaluated the association between urbanity and asthma and or chronic bronchitis prevalence.[18–24]

In the only study which evaluated the association between PM 2.5 and asthma, Mustapha et al. conducted a cross-sectional study in Nigeria in 2011.[15] This study created exposure indexes that involved various components, ranging from $PM_{2.5}$ to truck traffic.[15] The authors did not find a statistically significant relationship between $PM_{2.5}$ and asthma when adjusting for sex, age, presence of pets in the house, overcrowding, traffic disturbance at home, type of home cooking fuel (wood or coal; kerosene), and presence of smokers in the house (OR=1.05 95% CI 0.77-1.44).[15]

A total of two epidemiologic studies evaluated the relationship between $PM_{2.5}$ exposure and lung function parameters.[16,17] Both studies were cohort studies, one looking at male e-waste workers in Ghana[16] and the other schoolchildren in South Africa.[17] Both

studies did not find a significant association between $PM_{2.5}$ and any lung function parameters (OR=0.86 95% CI 0.32-2.30).[16,17]

Urbanicity can be considered a proxy for air pollution. A total of seven epidemiologic studies have evaluated the relationship between urbanicity and asthma and or chronic bronchitis prevalence in SSA.[18–24] Six of the studies were cross-sectional[18–22,24]and one study was a case control.[23] The majority of studies focused on children[18–20,23,24] while one study looked at individuals over 12[21] and one looked at adults 35 and older.[21] Most studies measured urbanicity by selecting participants from predetermined rural and urban areas,[20–24] while others used hospital records or questionnaires to obtain addresses which they would later use to categorize urban versus rural based on national criteria.[18,19] These studies found that there were more asthma hospital admissions of individuals from urban areas than from rural areas,[18] that urbanicity appears to negatively impact asthma control,[19] and that there was a higher prevalence of asthma among those living in urban areas compared to rural areas[21–24] with one finding a two-fold increased risk (OR 2.01 95%CI: 1.23-3.27),[22] but others not observing statistically significant results.[24]

1.4 Aims and Hypotheses

*Specific Aim 1***:** To evaluate the association between $PM_{2.5}$ exposure and asthma/chronic bronchitis in Senegal.

Hypothesis 1a: There will be a positive association between $PM_{2.5}$ exposure and asthma/chronic bronchitis.

*Specific Aim 2***:** To evaluate effect modification by sex on the association between $PM_{2.5}$ exposure and asthma/chronic bronchitis in Senegal.

Hypothesis 2a: Sex will be a statistically significant effect modifier of the association between $PM_{2.5}$ exposure and $PM_{2.5}$ and asthma/chronic bronchitis in Senegal.

1.5 Significance and Innovation

This study is innovative in being the first, to our knowledge, to study the relationship between $PM_{2.5}$, and asthma/chronic bronchitis in Senegal. It is innovative in the fact that it measures $PM_{2.5}$ instead of using urbanity as a proxy and that it measures asthma/chronic bronchitis instead of using lung function parameters as a proxy. The significance of the study lies in the fact that chronic respiratory diseases are associated with high levels of morbidity and mortality worldwide and are increasing in prevalence in Africa. $PM_{2.5}$ is a component of air pollution, an identified modifiable risk factor of chronic respiratory diseases. If $PM_{2.5}$ is associated with an increased risk of asthma/chronic bronchitis, then the findings of this study can inform future policy makers and their policies.

CHAPTER 2

METHODS

2.1 Study Design

A cross-sectional design was used to assess the relationship between exposure to $PM_{2.5}$ and asthma/chronic bronchitis among residents of Senegal from October 2010 to April 2011 who participated in the Senegal Demographic and Health Survey (DHS). The DHS is designed to collect data on household characteristics, marriage, fertility, infant and child mortality, maternal health, child health, nutrition, malaria, HIV, adult mortality, and characteristics of survey respondents such as occupation, chronic disease, and education level.[25] The DHS conducted sampling using a two-stage stratified randomly drawn national sample based on census district and urban vs. rural.[25] A sample of 8,232 households in Senegal was drawn with the expectation that 15,044 women and 4,429 men would be surveyed.[25] In total the DHS surveyed 15,688 women 15-49 years old and 5,668 men 15-59 years old.[25] From this dataset our study used asthma/chronic bronchitis prevalence, as well as some of the demographics data to use as covariates in our model. For $PM_{2.5}$ exposure we used the Surface $PM_{2.5}$ dataset from the Atmospheric Composition Analysis Group at the University of Washington in Saint Louis for the year 2011. This dataset provides annual global surface concentrations of $PM_{2.5}$ measured using multiple satellite instruments.[26]

2.2 Study Population

The study population was participants from the DHS, which included 15,688 women aged 15-49 years and 5,668 men aged 15-59 years.[25] To be surveyed the following criteria needed to be met: 1) women aged 15-49 years, men aged 15-59 years

and 2) a household member or someone who slept in the selected household the night before the survey, and 3) provided consent.[25] Therefore, individuals were excluded if they did not fall within the selected age range, were not a member of the selected household in Senegal or did not provide consent to participate in the study.[25] Our analyses included all individuals who were surveyed from October 2010 to April 2011 with the exception of those who were deemed visitors, as opposed to residents, and those for those who had missing $PM_{2.5}$ data.

2.3 Exposure Assessment

For $PM_{2.5}$ exposure we used the Surface $PM_{2.5}$ dataset created by the Atmospheric Composition Analysis Group at Washington University in Saint Louis for the year 2011. Annual global concentrations of $PM_{2.5}$ were measured using aerosol optical depth (AOD) retrievals from multiple satellite instruments as well as models.[26] The satellite instruments used include the NASA Moderate Resolution Imaging Spectroradiometer (MODIS), the Multi-angle Imaging SpectroRadiometer (MISR), and the Sea-Viewing Wide Field-of-View Sensor (SeaWiFS).[26] In addition, the GEOS-Chem chemical transport model was used to calculate the near surface $PM_{2.5}$ based on the total measure of aerosol and the Geographically Weighted Regression (GWR) was used to predict and adjust for residual bias.[26] The dataset is in raster form and was provided at a fine resolution scale of 0.01-degree grids.[26] For our study we evaluated the annual $PM_{2.5}$ concentration at the location of the selected household where the survey participants resided for the year the DHS started, 2010. R was utilized to overlay the $PM_{2.5}$ raster data with the participants' household location to obtain the specific annual $PM_{2.5}$ concentration for the household location.

2.4 Validity of Exposure

The methods that the Surface $PM_{2.5}$ dataset used to calculate the annual $PM_{2.5}$ concentrations were found to be highly consistent with out-of-sample cross validated $PM_{2.5}$ concentrations from monitors (R^2=0.81).[26] While ground-based monitors can provide more insight when measuring $PM_{2.5}$, the lack of ground-based monitors globally, particularly in developing areas, makes it difficult to conduct studies relying only on ground-based monitors.[26] Using satellite data in addition to available monitors and models, this form of $PM_{2.5}$ concentration assessment limits sources of bias associated with satellite data, in addition to globally characterizing $PM_{2.5}$, especially in regions where ground-based monitors are scarce.[26]

2.5 Outcome Assessment

Prevalence of asthma or chronic bronchitis was assessed through self-report in which participants were required to report if they had asthma or chronic bronchitis and if so if it was diagnosed by a medical provider.[25] If a participant responded "yes" to having either asthma or chronic bronchitis they will be categorized as having a chronic respiratory disease, participants who responded "no" to having asthma and chronic bronchitis will be categorized as not having a chronic respiratory disease.

2.6 Validity of Outcome

In this study self-reports of asthma/chronic bronchitis are not validated or confirmed with medical records. To our knowledge few or no validity studies have been conducted in Africa. However, a validity study among Finnish public sector workers on the accuracy of self-reported prevalent asthma found that sensitivity for self-reported asthma was 91% with specificities ranging from 96%-99%.[27] In addition, a validation

study on chronic diseases in Iran found a kappa agreements of 82.33 for diabetes and 86.76 for ischemic heart disease.[28] These validation studies indicate that the form of assessment used to collect our outcome data should be valid even though it was self-reported. It should also be noted that asthma and chronic bronchitis are measured together and not differentiated. Asthma and chronic bronchitis can have similar symptoms and can be difficult to differentiate, combining these measures allows us to assess chronic non-communicable respiratory disease and its relationship with $PM_{2.5}$ exposure.

2.7 Covariate Assessment

We investigated established risk factors for asthma/chronic bronchitis as possible confounders. Covariates considered include location, age, sex, wealth quintile, type of fuel used for cooking, such as solid vs. nonsolid, place of cooking, smoking status, education level, urbanicity, and NO_2.[29] Information on all covariates except NO_2 were collected from the DHS.

2.8 Data Analysis

First, we present an unweighted distribution of sample characteristics. The following analyses considered the clustered sampling design as well as the sample weights provided by the DHS. Sample characteristics were then presented by the outcome, asthma/chronic bronchitis status. Bivariate analyses for the exposure of interest, $PM_{2.5}$, as well as all potential confounders that were previously identified. For multivariate analysis, a mixed effects logistic regression model was used to model the relation between $PM_{2.5}$ exposure and asthma/chronic bronchitis while accounting for cluster level effects and adjusting for all potential confounders. Interaction by sex was

also tested using the same technique. Results were presented as adjusted odds ratios (aORs). All analyses were performed using R Statistical Software (v4.1.3).

CHAPTER 3

RESULTS

3.1 Unweighted Distribution of Sample Characteristics

The unweighted distribution of sample characteristics is presented in Table 1. A total of 19,843 individuals were included in the study. Of the total sample, 23.8% were males ages 15-59 and 76.2% were females ages 15-49, with an average age of 28.3 years. Of the 19,843 individuals, 59.3% lived in a rural area, and 57.5% had no formal education. The majority of individuals used solid fuel (82.2%) and were non-smokers (94.9%). Of all individuals 45.7% were from poor households (poorest and poor together) and 66% were from a household with an indoor kitchen. The overall prevalence of asthma/chronic bronchitis in the sample was 2.99%. The mean of $PM_{2.5}$ exposure was 40.6 µg/m^3 and ranged from 31.8 µg/m^3 to 51.3 µg/m3. The spatial distribution of annual $PM_{2.5}$ concentrations is presented in Figure 1. The mean NO_2 exposure was 0.24ppb.

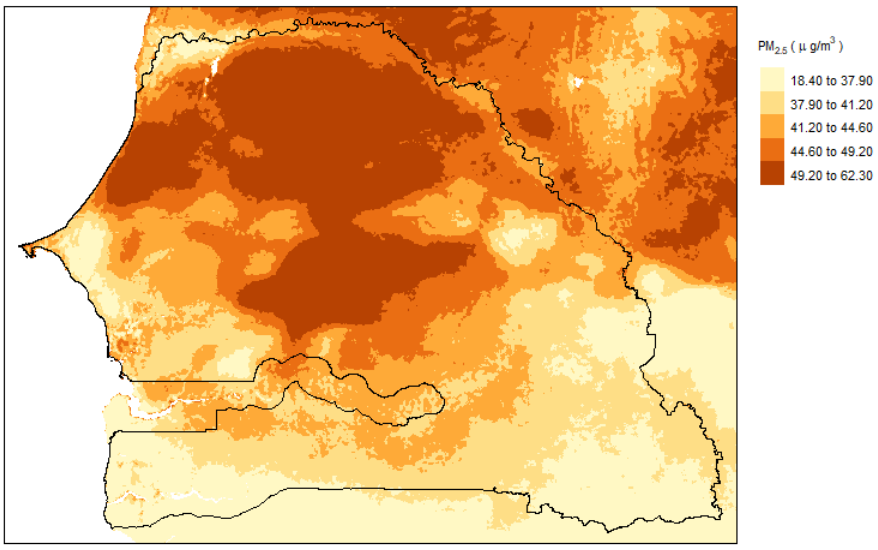

Figure 1: 2010 Annual $PM_{2.5}$ Concentrations (µg/m^3) in Senegal

3.2 Weighted Sample Characteristics by Asthma/Chronic Bronchitis

The bivariate models showed that significant predictors of asthma/chronic bronchitis included cooking fuel type, urbanicity, socioeconomic status, smoking status, and education level (Table 2). Asthma/chronic bronchitis prevalence was higher among individuals who were male (4%) compared to female (3%), used nonsolid fuel for cooking (5%) compared to solid (3%), resided in urban areas (5%) compared to rural areas (2%), smoked (5%) compared to non-smokers (3%), and had a kitchen in their house (4%) compared to a separate building (3%) or outdoors (3%). Asthma prevalence also appeared to increase with increasing socioeconomic status and education level. The mean level of exposure to $PM_{2.5}$ was the same (40.59 µg/m3) between those with and without asthma/chronic bronchitis.

3.3 Multivariable Associations between $PM_{2.5}$ Exposure and Asthma/Chronic Bronchitis

We then conducted a mixed effects logistic regression to estimate the association between $PM_{2.5}$ exposure and asthma/chronic bronchitis, while taking into account sampling design. The unadjusted odds ratio for the association the association between $PM_{2.5}$ exposure and asthma/chronic bronchitis was 0.97 (95%CI: 0.95 - 0.99) (Table 3). After adjusting for age, sex, cooking fuel type, smoking status, socioeconomic status, urbanicity, education level, location of the kitchen and $NO_{2,}$ this association was attenuated and no longer statistically significant 1.03 (95%CI: 0.99 – 1.06) (Table 3). In other words, for every one $\mu g/m^3$ increase in $PM_{2.5}$ exposure there is a 3% increased risk of asthma/chronic bronchitis. In this model significant confounders of the relationship

between $PM_{2.5}$ exposure and asthma/chronic bronchitis included urbanicity, smoking status, and education level.

We then evaluated whether the association between $PM_{2.5}$ exposure and asthma/chronic bronchitis differed within strata of sex. We found that the association was stronger among males with an adjusted odds ratio of 1.09 (95%CI: 1.03-1.15), compared to the association among females (aOR 1.01 (95%CI: 0.97 – 1.05) (p value for interaction term = 0.008) (Table 3).

CHAPTER 4

DISCUSSION

In this nationally representative sample provided by the DHS, we found that exposure to $PM_{2.5}$ is positively associated with asthma/chronic bronchitis, with males facing a greater risk than females. The main results of our study, which agree with our first hypothesis, suggest that for every ten unit $\mu g/m^3$ increase of $PM_{2.5}$ exposure there is a 30% increased risk of asthma/chronic bronchitis (aOR: 1.03 95%CI 0.99-1.06) when adjusting for age, sex, cooking fuel type, smoking status, socioeconomic status, urbanicity, education level, location of the kitchen and NO_2.Although prior studies suggest air pollution is positively associated with asthma, very few studies attempted to evaluate $PM_{2.5}$ as a risk factor for asthma in African countries and can identify the magnitude of the association between $PM_{2.5}$ and chronic respiratory diseases as we have here. To our knowledge only one other study evaluated $PM_{2.5}$ and asthma/chronic bronchitis in Senegal. This 2015-2016 observational study collected $PM_{2.5}$ exposure data and health data using similar method to our study, and similarly found unhealthy levels of $PM_{2.5}$.[14] Unfortunately, the authors did not conduct statistical analyses to evaluate the relationship between $PM_{2.5}$ and asthma/chronic bronchitis.[14] Instead, the authors observed the spatial temporal distribution of asthma and bronchitis and found the highest prevalence to be in the capital city, Dakar and suggested that urban sources contribute to poor air quality.[14]

Within Africa we found only one study that evaluated the relationship between $PM_{2.5}$ and asthma. This cross-sectional study conducted in Nigeria in 2011 did not find a statistically significant relationship between $PM_{2.5}$ and asthma when adjusting for sex,

age, presence of pets in the house, overcrowding, traffic disturbance at home, type of home cooking fuel (wood or coal; kerosene), and presence of smokers in the house (OR=1.05 95% CI 0.77-1.44).[15] Statistical significance aside, $PM_{2.5}$ was found to be a risk factor for asthma, a result very similar to that of our study. Therefore, the findings of our study on the association between $PM_{2.5}$ and asthma/chronic bronchitis are consistent with the current literature by suggesting $PM_{2.5}$ is a risk factor for chronic respiratory diseases such as asthma and chronic bronchitis.

When evaluating the relationship between $PM_{2.5}$ and asthma/chronic bronchitis, sex was found to be a statistically significantly effect modifier (p-value=0.008). Males faced a 9% increased risk (aOR 1.09 95%CI 1.03-1.15) for every unit increase in $PM_{2.5}$ exposure compared to females at 1% (aOR 1.01 95%CI 0.97-1.05). We did not expect males to have a greater risk than females, as a prior study on sex differences in air pollution-related acute circulatory and respiratory mortality and hospitalization in Canadian cities, found women to be more vulnerable and at increased risk for disease compared to males.[30] We found opposing results, that men were more vulnerable compared to women. Upon speculation this could possibly be explained by the gender roles observed in Senegal. Our study looked at outdoor $PM_{2.5}$ exposure, and in Senegal males are more likely than females to spend time outside due to their occupations. More time outside could lead to longer exposures to the outdoor $PM_{2.5}$ present in their area, which could explain why they are at higher risk for asthma/chronic bronchitis than females. To truly understand the mechanism leading to an increased risk in males further research is suggested and encouraged. While previous studies have adjusted for sex as a

confounder, our study is unique in that it evaluated sex as an effect modifier for the relationship between $PM_{2.5}$ and asthma/chronic bronchitis.

The prevalence of asthma/chronic bronchitis in our sample was about three percent. This is lower than expected as the estimated prevalence of asthma in Africa was 12.8% in 2010.[31] It is possible that asthma/chronic bronchitis was under reported in our study because individuals were unaware of their disease status. Lack of visiting medical professionals, and therefore lack of diagnosis for medical problems could explain possible under reporting. In the future study subjects could be evaluated by a doctor, opposed to using self-reporting via questionnaires, to ensure a more accurate assessment of the outcome of interest.

The average annual concentration of $PM_{2.5}$ for the sample was 40.6 $\mu g/m^3$. This level of $PM_{2.5}$ is much greater than the EPA standard of at or below 12 $\mu g/m^3$ and is considered particularly unhealthy for sensitive groups, which include but not limited to asthmatics, children, and the elderly.[32] However, this result is not uncommon as the World Bank reported that the annual $PM_{2.5}$ concentration in Senegal in 2010 was about 35 $\mu g/m^3$.[33] In addition, surrounding countries such as Mali and Mauritania had 2010 annual $PM_{2.5}$ concentrations that fell into the same air quality index category; unhealthy for sensitive groups.[33]

This study has some key strengths. The DHS had a large sample size, of which we were able to include almost 20,000 individuals in our study, after excluding those who were visitors and were missing data for the exposure or outcome of interest. This large sample size gave us greater power to detect differences between groups and allows us to more closely approximate the population. In addition, we used the sample weights in our

models to ensure that our sample was as nationally representative as possible, mitigating any impact of selection bias. Lastly our study used a direct measure of $PM_{2.5}$, opposed to a proxy for air pollution such as urbanicity. This allowed us to add new information to the literature, which is important because our level of specificity could impact policy level changes for acceptable PM2.5 limits.

Our study has a number of limitations. First, since this is a cross-sectional study, we need to take temporal bias into consideration. Temporal bias occurs when we assume the wrong sequence of events of causality. We need to be careful because it is possible that individuals had asthma before they were exposed to high levels of $PM_{2.5}$. We cannot assume $PM_{2.5}$ causes asthma/chronic bronchitis because the temporality of the relationship cannot be defined using a cross-sectional study.

Second, we are aware of the potential non-differential misclassification of exposure. $PM_{2.5}$ exposure was measured using global annual concentrations of $PM_{2.5}$ that were obtained using aerosol optical depth (AOD) retrievals from multiple satellite instruments as well as models.[26] While this method was found to be highly consistent with out-of-sample cross validated $PM_{2.5}$ concentrations from ground monitors, the ground monitors used were likely to have only been found in the capital city of Dakar, meaning that we do not know if the model used to calculate $PM_{2.5}$ exposure was calibrated correctly for Senegal, especially in rural areas. In addition, the locations used for our samples were only available at the cluster level in order to deidentify health data, meaning the coordinates used are only estimates not exact locations of where the subjects live. Lastly $PM_{2.5}$ exposure is based on environmental estimates, not actual human inhalation. Since we did not use personal monitors for each individual, we could have

incorrectly assessed their personal level of exposure to $PM_{2.5}$. Incorrect categorizing of $PM_{2.5}$ exposure will cause the reported OR for asthma/chronic bronchitis to be an underestimation of the true OR, which then causes a bias towards the null value.

Lastly, it is possible that there are confounding factors impacting our results that were not accounted for in our study. For example, common air pollutants in addition to $PM_{2.5}$ include CO, NO2, NO, PM10, SO2 and ozone. Only NO2 was adjusted for in our analysis indicating that all other pollutants have the potential to be confounding the relationship between $PM_{2.5}$ and asthma/chronic bronchitis in our model.

Overall, the present study found that exposure to $PM_{2.5}$ is positively associated with asthma/chronic bronchitis, with males facing a greater risk than females. This study suggests that specific air pollution measures, such as $PM_{2.5}$ can be associated with poor health outcomes such as chronic respiratory diseases and can differ according to sex. It should be noted that this study is likely only generalizable to individuals living within Sub-Saharan Africa, as $PM_{2.5}$ levels in this area are very high comparative to other parts of the world. Nonetheless, understanding the negative health impacts at certain levels of air pollution is critical in informing policy. If policy makers are aware of the relationships between air pollution and disease, they can create policies that help to mitigate air pollution and in turn hopefully reduce burden of disease. Further research examining the associations between air pollutants and chronic respiratory disease is warranted, especially in Africa where there appears to be a paucity of research on this topic.

THE TABLES

Table 1: Unweighted Distribution of Sample Characteristics

Categorical Variables	**N (%)** N=19843
Asthma/CB:	
Yes	593 (2.99%)
No	19250 (97.0%)
Sex:	
Female	15112 (76.2%)
Male	4731 (23.8%)
Cooking fuel type:	
Solid	16305 (82.2%)
Nonsolid	3538 (17.8%)
Smoker:	
Yes	1010 (5.09%)
No	18833 (94.9%)
Wealth Index:	
Poorest	4582 (23.1%)
Poor	4487 (22.6%)
Middle	4600 (23.2%)
Richer	3497 (17.6%)
Richest	2677 (13.5%)
Urbanicity:	
Urban	8069 (40.7%)
Rural	11774 (59.3%)
Education Level:	
None	11412 (57.5%)
Primary	4251 (21.4%)
Secondary	3928 (19.8%)
Higher	252 (1.27%)
Kitchen Location:	
In house	13106 (66.0%)
Separate building	4506 (22.7%)
Outdoors	2183 (11.0%)
Other	48 (0.24%)
Continuous Variables	**Mean (SD)**
Age	28.3 (10.2)
$PM_{2.5}$	40.6 (3.95)
NO2	0.24 (0.09)

*Note these sample characteristics are unweighted and therefore do not account for sampling weights.

Table 2: Weighted Distribution of Sample Characteristics by Asthma/Chronic Bronchitis and Bivariate Analysis (N=19843)

Categorical Variables	Asthma/CB % (95%CI)	No Asthma/CB % (95%CI)	OR (95%CI)
Sex:			
Female	0.03 (0.03, 0.04)	0.97 (0.96, 0.97)	-
Male	0.04 (0.03, 0.04)	0.96 (0.96, 0.97)	1.10 (0.86, 1.41)
Cooking fuel type:			
Solid	0.03 (0.02, 0.03)	0.97 (0.97, 0.98)	-
Nonsolid	0.05 (0.04, 0.06)	0.95 (0.94, 0.96)	1.73 (1.38, 2.10)
Smoker:			
Yes	0.05 (0.03, 0.07)	0.95 (0.93, 0.97)	-
No	0.03 (0.03, 0.04)	0.97 (0.96, 0.97)	0.66 (0.45, 0.94)
Wealth Index:			
Poorest	0.02 (0.02, 0.03)	0.98 (0.97, 0.98)	-
Poor	0.02 (0.02, 0.02)	0.98 (0.98, 0.98)	0.88 (0.64, 1.21)
Middle	0.03 (0.02, 0.04)	0.97 (0.96, 0.98)	1.4 (1.04, 1.88)
Richer	0.04 (0.03, 0.05)	0.96 (0.95, 0.97)	1.93 (1.41, 2.64)
Richest	0.05 (0.04, 0.06)	0.95 (0.94, 0.96)	2.36 (1.73, 3.23)
Urbanicity:			
Urban	0.05 (0.04, 0.05)	0.95 (0.95, 0.96)	-
Rural	0.02 (0.02, 0.03)	0.98 (0.97, 0.98)	0.47 (0.39, 0.58)
Education Level:			
None	0.02 (0.02, 0.03)	0.98 (0.97, 0.98)	-
Primary	0.04 (0.03, 0.05)	0.96 (0.95, 0.97)	1.69 (1.29, 2.21)
Secondary	0.06 (0.05, 0.07)	0.94 (0.93, 0.95)	2.59 (2.01, 3.34)
Higher	0.05 (0.02, 0.09)	0.95 (0.91, 0.98)	2.38 (1.11, 5.07)
Kitchen Location:			
In house	0.04 (0.03, 0.04)	0.96 (0.96, 0.97)	-
Separate building	0.03 (0.03, 0.04)	0.97 (0.96, 0.97)	0.92 (0.71, 1.18)
Outdoors	0.03 (0.02, 0.04)	0.97 (0.96, 0.98)	0.82 (0.59, 1.13)
Other	0.00 (0.00, 0.00)	1.00 (1.00, 1.00)	0.00 (0.00, 0.00)
Continuous Variables	**Mean (SD)**	**Mean (SD)**	**OR (95%CI)**
Age	28.65 (10.38)	28.29 (10.14)	1 (0.98, 1.01)
$PM_{2.5}$	40.59 (4.02)	40.59 (3.95)	0.97 (0.95, 0.99)
NO2	0.24 (0.10)	0.24 (0.09)	1 (0.98, 1.01)

*Note the statistics presented above are weighted using the provided sample weights. The bivariate models were adjusted for the survey design to account for cluster level effects.

Table 3: Odds ratios for the association between $PM_{2.5}$ and asthma/chronic bronchitis

	Odds Ratio	95% Confidence Interval
Unadjusted	0.97	(0.95, 0.99)
Adjusted	1.03	(0.99, 1.06)
Male Adjusted	1.09	(1.03, 1.15)
Female Adjusted	1.01	(0.97, 1.05)

*Note: Adjusted for age, sex, wealth quintile, fuel used for cooking, place of cooking, smoking status, education level, urbanicity and NO_2

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