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Short-term Effect of $PM_{2.5}$ on Childhood Pneumonia Admissions in Ouagadougou Burkina Faso

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Aims: There is evidence that atmospheric $PM_{2.5}$ concentration is higher in Ouagadougou than the World Health Organization recommended threshold; however, its impact on childhood pneumonia admissions is unknown.
Study Design: This was a partial ecological study.
Place and Duration of Study: The study was conducted in Ouagadougou, Burkina Faso at the Centre Hospitalier Universitaire Pédiatrique Charles de Gaulle (CHUP-CDG) and the Centre Hospitalier Universitaire Yalgado Ouédraogo (CHU-YO), from July 2019 to June 2020.
Methodology: The study involved 2012 (boys: 54.6%) children under 15 years who were admitted for respiratory diseases and had complete records. Of the 2012 children, 776 (38.6%) were diagnosed with pneumonia. The daily pneumonia counts and sociodemographic and clinical data were retrieved from the hospital's records. Daily atmospheric $PM_{2.5}$, temperature, and relative humidity were recorded in the same period. Autoregressive integrated moving average (ARIMA) modeling was used to forecast daily childhood pneumonia admissions.
Results: The median (IQR) of daily pneumonia admissions (count) and $PM_{2.5}$ ($\mu g/m^3$) were 5.0(3.0-8.0) and 19.67(6.05-154.50) respectively. While the likelihood of being hospitalized for pneumonia in children ≥ 5 years and those < 1 year were similar [OR: 1.35(0.98-1.85)], the odds of the former being hospitalized for childhood pneumonia was greater than the latter after controlling $PM_{2.5}$ [AOR: 1.38(1.00-1.90)]. The odds increased marginally after the addition of temperature [AOR: 1.39(1.01-1.92)] but not humidity [AOR: 1.39(1.01-1.92). In addition, the ARIMA (0, 1, 1) model was more suitable for forecasting childhood pneumonia admissions.
Conclusion: While no direct association between PM2.5 levels and overall childhood pneumonia admissions was found, the data suggest that PM2.5 may influence the risk of pneumonia in children aged 5 years and older. Further studies covering a longer exposure period are however recommended to validate these findings.

Keywords: Particulate matter; PM 2.5; childhood pneumonia; age; admissions; Burkina Faso.

1. INTRODUCTION

Pneumonia is a public health challenge contributing to the global burden of morbidity and mortality. It is estimated in the developed world that pneumonia contributes to the disease burden of about 10-15 cases per 1000 children and about 1-4 per 1000 hospital admissions annually [1]. According to the global burden of disease study, about 1.5 million deaths were due to pneumonia in 2015 alone and about 99% of these deaths occurred in low-and-middle-income countries (LMIC) [2,3]. Similarly, air pollution is responsible for about 7 million premature global deaths annually [4].

Air pollution comprises a complex mixture of materials and may include particulate matter (PM). According to epidemiological studies, long-term exposure to PM may lead to cardiovascular events, metabolic syndrome, hypertension, and autism spectrum disorder while short-term exposure may be associated with pneumonia [4,5]. While pneumonia may affect every age group, children may be unequally affected due to their immature immune systems [1,2]. Moreover, subgroup analysis in previous studies has demonstrated age-specific variabilities on the effect of air PM on childhood pneumonia hospitalizations. While some showed significant findings in less than 5-year-olds, others were seen in older children [2,6,7]. Even though there are several risk factors for childhood pneumonia including pathogenic agents, sickle cell disease, HIV infection, heart disease and family history of asthma, their impact on childhood pneumonia may be moderated by exposure to atmospheric PM [8].

Atmospheric particulate matter is usually, but not always, comprised of dust, haze, sulfate, primary organic aerosols, nitrate, ammonium, and chloride. Atmospheric particulate matter occurs in different sizes such as PM_1, $PM_{2.5}$ and PM_{10} corresponding to aerodynamic diameters of <1.0, <2.5 and <10 microns (μm) respectively [6]. The sources of PM include but are not limited to, cooking emissions, industrial emissions, traffic emissions, and coal combustion [6]. The mechanisms through which PM influences pneumonia development are complex. Toxicological studies have indicated that nitrate-laden acidic aerosols tend to lower the airways' pH through the deposition of hydrogen ions, which may trigger adverse reactions [9]. Also, there could be interactions between $PM_{2.5}$-

associated metal complexes and *Mycoplasma pneumonia* via mechanisms including oxidative stress induction and modulation of the host immune system [10]. Moreover, PM may simultaneously act as vectors for pathogens, increase the viability of air-borne viral particles as well as reduce human resistance to pathogens [11]. Atmospheric $PM_{2.5}$ and PM_{10} exposure have been shown to increase the adhesion of *Streptococcus pneumoniae* strain D39 to human primary bronchial epithelial cells [12]. According to Liu, Zhao [13], ambient $PM_{2.5}$ may induce apoptosis in lung epithelial cells, marking the onset of pulmonary diseases. While all atmospheric particulate matter may be hazardous to human health, the size of PM may modify its associated effect on health [6]. From the literature, $PM_{2.5}$ is the most studied, robust, and consistent indicator of PM air pollution [2].

There are regional and seasonal variabilities in the impact of PM on health and childhood pneumonia [5]. Documented evidence from 15 sites in 11 cities (including Ouagadougou) across 8 African countries has reported atmospheric $PM_{2.5}$ in the range of 10 to 116 $\mu g/m^3$ which is more than the levels recommended by the World Health Organization (WHO) guidelines on air quality i.e. < 15 $\mu g/m^3$ [14]. Similar findings have been reported in cities of other African countries [15-17]. This may be due to the effect of industrialization, population growth, and windblown dust from the Saharan desert [4]. Burkina Faso may be unequally affected by dust-associated PM due to its proximity to the Saharan desert [15]. According to a previous study, the mean (SD) and median (IQR) of atmospheric PM2.5 in Ouagadougou were 46 (±54) and 10 (3–27) respectively in 2020 [4]. Despite the high atmospheric $PM_{2.5}$ levels in Ouagadougou, its impact on childhood pneumonia admissions is least explored. Also, the pathophysiology of pneumonia may be impacted by genetic variabilities and the differences in the chemical composition of particulate matter between populations which makes it necessary for population-specific studies [18-20]. This study aimed to evaluate the impact of short-term $PM_{2.5}$ exposure on childhood pneumonia admissions in Ouagadougou, Burkina Faso.

2. MATERIALS AND METHODS

2.1 Study Design and Settings

This was a partial ecological study from July 2019 to June 2020. Ecological studies, among others, investigate potential associations between two variables using group-level or aggregate-level data. The study was conducted at the pediatric units of two teaching hospitals in Ouagadougou, Burkina Faso: The Centre Hospitalier Universitaire Pédiatrique Charles de Gaulle (CHUP-CDG) and the Centre Hospitalier Universitaire Yalgado Ouédraogo (CHU-YO). The population of Ouagadougou was estimated at 2,453,496 inhabitants in 2019, representing about 45.4% of the country's urban population [21]. Ouagadougou is located in the middle zone of the country, characterized by a Sudano-Sahelian climate. The annual average temperature of Ouagadougou is 28°C, annual cumulative rainfall is between 600 and 900 mm, and the average relative humidity is about 75% during the rainy season [22]. There are two climatic seasons in a year, comprising a dry season (October to April) and a rainy season (May to September). The harmattan occurs during the dry season, particularly between November and March, which is characterized by dry and dusty winds.

2.2 Study Population

The study involved 2012 children including 1098 boys (54.6%) and 914 girls (45.4%). The children were aged between 0 and 180 months or ≤15 years. Of the 2012 children, 776 (38.6%) were admitted for pneumonia. The study includes all children admitted between July 2019 and June 2020, regardless of whether they were diagnosed with pneumonia. Children whose final diagnosis was unclear or who had missing sociodemographic characteristics were excluded. Since the study was retrospective, pneumonia was defined by hospital code (ICD 10) which was arrived at after all clinical and imaging procedures were performed following both local and international guidelines.

2.3 Study Variables

The primary outcome was the daily childhood pneumonia admission count. The predictor variables included sociodemographic variables (age and sex), pollutant and meteorological variables ($PM_{2.5}$, temperature and humidity), climatic variables (wet and dry season) and clinical variables (type of hospital service, admission type, sickle cell disease status, HIV disease status, history of heart disease and family history of asthma).

2.4 Data Collection

2.4.1 Daily hospital admissions data

Daily hospital admissions records from July 2019 to June 2020 were obtained from the patient records of the two teaching hospitals. The records included the final diagnosis of a disease which was coded according to the International Classification of Diseases (ICD-10) [7]. For each selected case, the age, sex as well and risk factors of pneumonia such as sickle cell disease, HIV status, heart disease and family history of asthma were also collected. The data were subsequently aggregated by sex (boys and girls), age (<1, 1-4, and ≥5 years), season (wet and dry), type of admission, and risk factors. The pneumonia cases alone were further categorized by sex, age, sickle cell disease, HIV, and heart disease.

2.4.2 Daily pollutant and meteorological data

Ouagadougou was one of the 15 sites and 11 cities that participated in the study that measured $PM_{2.5}$ across 8 countries in Africa [4]. In that study, daily $PM_{2.5}$ and other atmospheric variables were monitored at one station in Balkuy, Ouagadougou. Within the study period, air $PM_{2.5}$ and other atmospheric variables were monitored and were then matched with daily hospital admissions. Daily atmospheric $PM_{2.5}$ was measured using PurpleAir II SD® sensors (PurpleAir, UT, USA) at Balkuy, Ouagadougou (GPS coordinates: 12.308310, -1.469981) and which can be seen at www.purpleAir.com. Also, daily values of relative humidity and temperature were collected**.** At the end of each day, the daily average of $PM_{2.5}$, temperature, and relative humidity were recorded and this was repeated for the entire study period.

2.5 Statistical Analysis

The data were analyzed using SPSS (v26) and Graph Pad Prism (v8). The daily cases of childhood pneumonia admissions, as well as the air pollutant and meteorological variables ($PM_{2.5}$, Temperature and humidity), were presented as either frequency (%) for categorical variables or mean (SD) and/or median (IQR) for continuous variables. The correlation between daily childhood pneumonia admission and atmospheric factors was performed using Spearman rank correlation. The differences in the data distribution in atmospheric variables between children with and without pneumonia were determined using the Mann-Whitney U test. The association between childhood pneumonia and independent variables was determined using univariable and multivariable logistic regression analysis, adjusting for daily $PM_{2.5}$ and meteorological variables. Due to the acute and delayed effect of $PM_{2.5}$ exposure on childhood pneumonia admissions, $PM_{2.5}$ was transformed into single-day lags. The $PM_{2.5}$ on the same day of admission was taken as lag0, the day before admission was lag1, two days before as lag2, and so on until lag6. Similarly, lagged (Lag0-Lag6) variables were created for daily mean temperature and humidity for consistency. A test of overdispersion in the daily pneumonia count variable was performed by comparing the mean and the variance. It was observed that the variance was markedly higher than the mean of daily pneumonia count (overdispersion). Negative binomial regression was therefore chosen to create single pollutant models with daily pneumonia count as the dependent variable and the $PM_{2.5}$ lags as the predictor variables [23]. The models were adjusted for atmospheric covariates (temperature and humidity) and the results were reported as adjusted odds ratios with 95% confidence intervals (95% CI). Subgroup analyses were also performed for sex and age since $PM_{2.5}$ may exhibit age and sex-specific differential effects on pneumonia admissions. Forest plots were then created to check for differences in adjusted odds ratios between daily lag intervals in the total and subgroups. Time series plots with and without first-order differencing were plotted for $PM_{2.5}$ and meteorological variables to check for stationarity [24]. Auto- and partial autocorrelation analyses with first-order differencing were performed. The residuals of the model were examined to check whether discernable patterns and autocorrelation, utilizing residual plots for autocorrelation function (ACF) and partial autocorrelation function (PACF). Three traditional autoregressive integrated moving average [ARIMA (p, d, q)] models [A:(0, 1, 0), B:(0, 1, 1) and C:(1, 1, 1)] with value fitting, no transformation, were then created to forecast daily childhood pneumonia admissions, with a constant. The p, d and q in the ARIMA model indicate the autoregressive order, differencing order and moving average respectively. The models were adjusted for covariates with first-order differencing ($PM_{2.5}$, temperature and humidity). To determine the best model to forecast childhood pneumonia admissions, the Bayesian Information Criterion (BIC) and parameter estimates of the models were compared. Also, the predicted values of the

models were saved and then compared with the observed values for reliability using the Bland-Altmann method. All the statistical analyses were 2-tailed at a P-value <0.050, considered statistically significant.

3. RESULTS

3.1 Study Population and Selection

The total number of available paediatric patient records was 14,209. Out of these number of records, 10,695 had complete information and were usable. Of these, 8,490 cases (79.38%) were non-respiratory diseases. From the 2,205 records with a respiratory disease diagnosis, information on the dependent variable and the covariates were available for 2,012 children (91.25%).

3.2 General Characteristics of the Study Variables

The sociodemographic, anthropological and clinical characteristics of the study population are summarized in Table 1. The result showed that the majority of the children who were hospitalized were not found to have childhood pneumonia (61.4%). The sex distribution of the children was fairly similar, however, boys formed the majority with a proportion of 54.6%. regarding the age distribution, children who were less than 1 year were similar in number to those aged between 1

Table 1. The general characteristics of the study population

Characteristics	Frequency	Per cent (%)
Pneumonia		
No	1236	61.4
Yes	776	38.6
Sex		
Girls	914	45.4
Boys	1098	54.6
Age (years)		
<1	909	45.2
1-4	916	45.5
5-15	187	9.3
Service		
Emergency	698	34.7
Hospitalized	558	27.7
Consultation	756	37.6
Season		
Wet	898	44.6
Dry	1114	55.4
Admission		
Direct	1492	74.2
Referred	511	25.4
Transferred	9	0.4
Sickle cell disease		
No	1986	98.7
Yes	26	1.3
HIV disease		
No	1996	99.2
Yes	16	0.8
Heart disease		
No	1972	98
Yes	40	2.0
Asthma (Family history)		
No	1994	99.1
Yes	18	0.9

The results are summarized as frequency and percentages

Table 2. Descriptive statistics of daily atmospheric variables and childhood pneumonia hospitalizations

Variable	Centiles							
	Mean	SD	Min	25	50	75	Max	Range
Pneumonia (n)	5.78	3.74	1.00	3.00	5.00	8.00	22.00	21.00
Meteorological data								
Temperature (°C)	33.87	2.85	25.00	32.00	33.40	35.38	41.00	16.00
Humidity (%)	29.88	17.52	5.00	58.67	13.00	26.00	48.67	3.67
Particulate matter								
$PM_{2.5}$ (µg/m^3)	30.27	28.64	0.00	6.05	19.67	51.50	154.50	154.50

PM=particulate matter, SD=standard deviation, Min=minimum, Max=maximum

Table 3. Correlation between daily childhood pneumonia hospitalizations and atmospheric variables

Variable	Pneumonia	Temperature	Humidity	$PM_{2.5}$
Pneumonia (count)	1			
Temperature (°C)	0.015	1		
Humidity (%)	0.085	0.098	1	
$PM_{2.5}$ (µg/m^3)	0.024	-0.183*	-0.797*	1

*Spearman rank correlation analysis. *P<0.010*

and 5 years with those older than 5 years forming the minority (9.3%). It was observed that the majority of the hospitalization of children during the study period occurred in the dry season where there was no rainfall (55.4%). In addition, 74.2% of the children, the majority, were hospitalized directly relative to those who were referred or transferred. Regarding the occurrence of other medical conditions or comorbidity, only a few children were found to have sickle cell disease (1.3%), were HIV-positive (0.8%), had a history of heart disease (2.0%) or had a family history of asthma (0.9%).

3.3 Descriptive Statistics of Daily Atmospheric Variables and Pneumonia Count

The descriptive statistics of the daily atmospheric variables and daily pneumonia count within the study period are summarized in Table 2. The daily pneumonia count ranged between 1.00 and 22.00 with a mean and median daily count of 5.78 and 5.00 respectively. Regarding the meteorological data, the mean and median atmospheric temperatures within the study period were 33.87^O C and 33.40^O C respectively. For the humidity records during the study period, the mean and median recorded daily humidity were 29.88% and 13.00% respectively with a range of 3.67%. The daily atmospheric particulate matter ($PM_{2.5}$) within the study period averaged 30.27 µg/m^3 with a minimum value of 0.00 µg/m^3 and a maximum of 154 µg/m^3.

3.4 Correlation between Daily Childhood Pneumonia Count and Atmospheric Variables

The relationship between the daily pneumonia count and atmospheric variables was determined (Table 3). It was observed that there was a significant but weak and inverse relationship between temperature and $PM_{2.5}$ (r= -0.183, P<0.050). Similarly, the daily $PM_{2.5}$ showed a strong but inverse relationship with the daily humidity (r= -0.797, P<0.050).

3.5 Atmospheric $PM_{2.5}$ Concentrations in Children with and without Pneumonia

The children were stratified by their pneumonia status and daily $PM_{2.5}$ concentrations were then compared to determine whether there were differences (Table 4). Unexpectedly, results indicated that the median daily $PM_{2.5}$ concentration was lower on days that children between 1-4 years were hospitalized for pneumonia relative to those without pneumonia [14.0(6.0-44.0) vs 21.0(12.0-58.3), P=0.011]. However, direct admission of children with pneumonia at the hospital occurred on days that the daily $PM_{2.5}$ concentration was relatively higher when compared to children who were hospitalized without pneumonia.

3.6 The Impact of PM2.5 on Childhood Pneumonia Hospitalization by Single-day Lag Intervals

Single pollutant models to determine the impact of PM $_{2.5}$ on childhood pneumonia hospitalization by single-day lag intervals were created to determine the impact of daily PM $_{2.5}$ concentration on childhood pneumonia by sex and age (Table 5). Analyses showed that the impact of daily PM $_{2.5}$ concentration on childhood pneumonia hospitalization did not differ by either sex or age (Fig. 1).

3.7 Sociodemographic, Clinical and Seasonal Variability of Childhood Pneumonia Hospitalizations

The association between childhood pneumonia count or hospitalization and sociodemographic, clinical and seasonal variables were determined using univariable and multivariable logistic regression analysis (Table 6). The crude odds ratios indicated that children who received services in the form of hospitalization were referred or transferred, had sickle cell disease or had heart disease were more likely to be hospitalized due to pneumonia while those who received consultation services or had a family history of asthma were less likely to be hospitalized for pneumonia. These observations did not markedly change after controlling for atmospheric variables and particulate matter. However, while the likelihood of being hospitalized for pneumonia of children ≥ 5 years and those < 1 year was similar [OR: 1.35(0.98-1.85)], the odds of the former being hospitalized for childhood pneumonia were greater than the latter after controlling $PM_{2.5}$ [AOR: 1.38(1.00-1.90)]. The odds increased marginally after

Table 4. Atmospheric $PM_{2.5}$ concentrations in children with and without pneumonia

Variable	Pneumonia		P-value
	No	Yes	
Sex			
Girls	14.0(6.0-48.0)	12.0(6.0-41.0)	0.423
Boys	17.0(6.0-50.0)	14.0(6.0-47.0)	0.687
Age (years)			
<1	11.0(4.0-42.0)	12.0(5.0-38.0)	0.912
1-4	21.0(12.0-58.3)	14.0(6.0-44.0)	0.011
≥5	25.0(8.0-43.3)	38.0(9.0-63.5)	0.052
Service			
Emergency	18.0(6.0-44.0)	16.0(6.0-49.8)	0.981
Hospitalized	11.0(5.0-44.0)	12.5(6.0-41.0)	0.381
Consultation	16.0(6.0-52.8)	11.5(6.0-43.8)	0.386
Season			
Wet	5.0(1.0-8.0)	6.0(1.0-9.0)	0.085
Dry	42.0(25.0-69.0)	41.0(24.8-67.3)	0.363
Admission			
Direct	7.0(6.0-8.0)	16.0(6.0-49.0)	<0.001
Referred	11.0(5.0-38.0)	12.0(6.0-37.0)	0.945
Transferred	33.0(18.5-52.5)	19.5(9.3-53.8)	0.413
Sickle cell disease			
No	16.0(6.0-49.0)	13.0(6.0-44.0)	0.345
Yes	19.0(9.8-41.0)	22.5(13.3-47.5)	0.614
HIV			
No	16.0(6.0-49.0)	13.5(6.0-44.0)	0.350
Yes	39.0(5.3-63.0)	23.0(9.5-82.8)	0.953
Heart disease			
No	17.0(6.0-49.0)	14.0(6.0-46.0)	0.570
Yes	9.5(2.5-16.5)	10.0(5.3-20.0)	0.784
Asthma (family history)			
No	16.0(6.0-49.0)	14.0(6.0-44.0)	0.431
Yes	26.0(8.0-52.0)	16.0(8.0-38.0)	0.678

Differences in the median values were determined using the Mann-Whitney U test

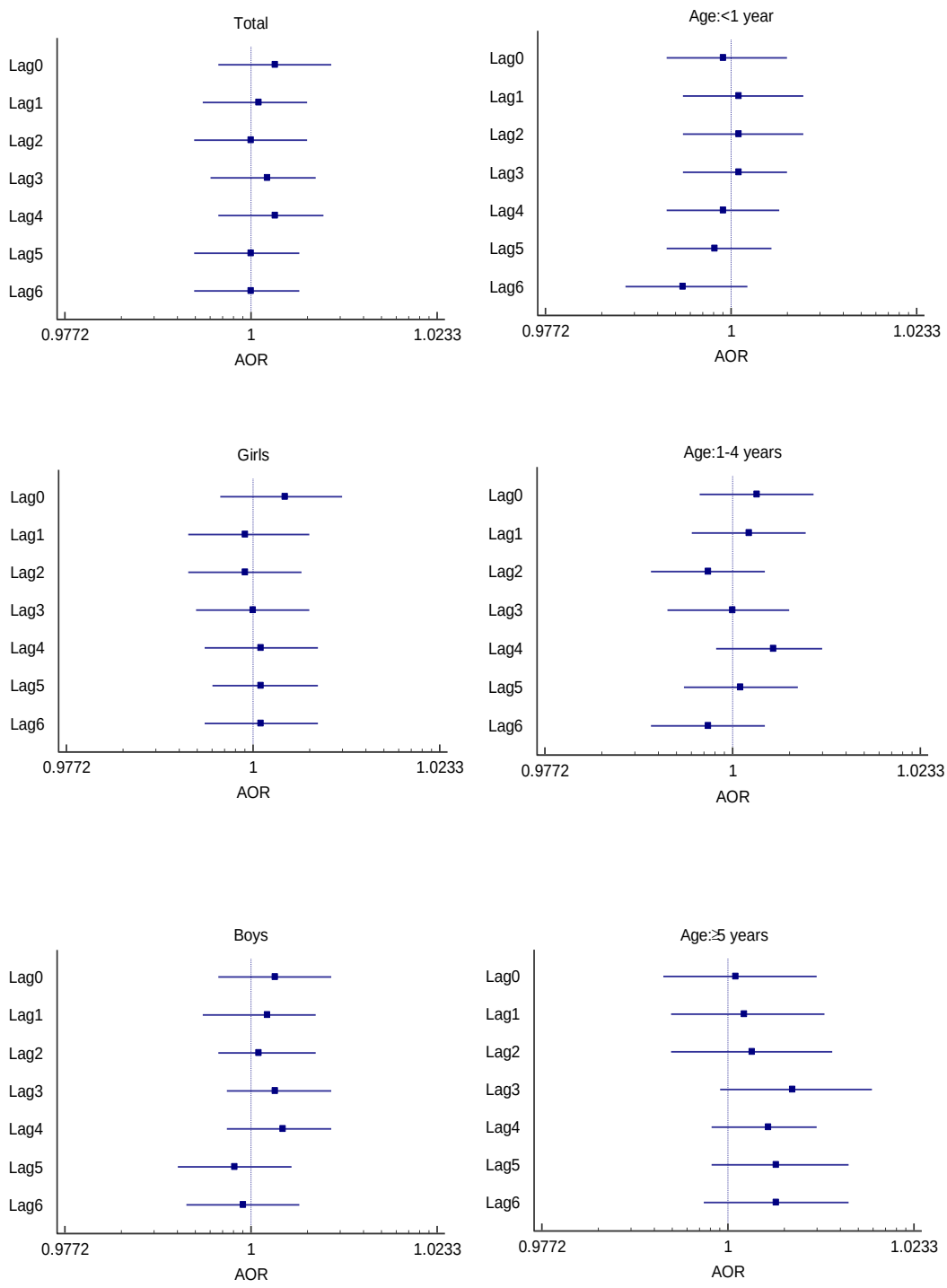

Fig. 1. Forest plots to compare the adjusted odds ratios (95%CI) of the single-day lag intervals of daily PM2.5 exposure and childhood pneumonia admissions

Table 5. Single pollutant models to determine the impact of PM2.5 on childhood pneumonia hospitalization by single-day lag intervals

Pm2.5 (µg/m3)	Total	Sex		Age(years)		
		Girls	Boys	<1	1-4	≥5
Lag0	1.003(0.996-1.010)	1.004(0.996-1.011)	1.003(0.996-1.010)	0.999(0.992-1.007)	1.003(0.996-1.010)	1.001(0.992-1.011)
Lag1	1.001(0.994-1.007)	0.999(0.992-1.007)	1.002(0.995-1.008)	1.001(0.994-1.009)	1.002(0.995-1.009)	1.002(0.993-1.012)
Lag2	1.000(0.993-1.007)	0.999(0.992-1.006)	1.001(0.994-1.008)	1.001(0.994-1.009)	0.997(0.990-1.004)	1.003(0.993-1.013)
Lag3	1.002(0.995-1.008)	1.000(0.993-1.007)	1.003(0.996-1.010)	1.001(0.994-1.007)	1.000(0.992-1.007)	1.008(0.999-1.018)
Lag4	1.003(0.996-1.009)	1.001(0.994-1.008)	1.004(0.997-1.010)	0.999(0.992-1.006)	1.005(0.998-1.011)	1.005(0.998-1.011)
Lag5	1.000(0.993-1.006)	1.001(0.995-1.008)	0.998(0.991-1.005)	0.998(0.992-1.005)	1.001(0.994-1.008)	1.006(0.998-1.015)
Lag6	1.000(0.993-1.006)	1.001(0.994-1.008)	0.999(0.992-1.006)	0.994(0.987-1.002)	0.997(0.990-1.004)	1.006(0.997-1.015)

Negative binomial regression analysis. The models were adjusted for Temperature and humidity. Lag represents the days before the hospitalization. Lag0 represents the day of the hospitalization; Lag1 represents one day before hospitalization etc.

Table 6. The impact of atmospheric variables on childhood pneumonia stratified by sociodemographic, clinical and seasonal variables

Variable	Pneumonia		OR (95%CI)			
	No (n=1236)	Yes (n=776)	M1	M2 (M1+$PM_{2.5}$)	M3 (M2+Temperature)	M4 (M3+Humidity)
Sex						
Female	565 (45.7)	349(45.0)	1	1	1	1
Male	671 (54.3)	427(55.0)	1.03(0.86-1.23)	1.03(0.86-1.24)	1.04(0.87-1.24)	1.03(0.87-1.24)
Age (years)						
<1yr	580(46.9)	329(42.4)	1	1	1	1
1-4yr	550(44.5)	366(47.2)	1.17(0.97-1.42)	1.19(0.99-1.44)	1.19(0.98-1.44)	1.19(0.98-1.44)
≥5	106(8.6)	81(10.4)	1.35(0.98-1.85)	1.38(1.00-1.90)*	1.39(1.01-1.92)*	1.39(1.01-1.92)*
Service						
Emergency	450(36.4)	248(32.0)	1	1	1	1
Hospitalized	170(13.8)	388(50.0)	4.14(3.27-5.25)***	4.14(3.26-5.25)***	4.10(3.23-5.20)***	4.08(3.22-5.18)***
Consultation	616(49.8)	140(18.0)	0.41(0.32-0.52)***	0.41(0.33-0.53)***	0.41(0.32-0.52)***	0.41(0.32-0.52)***
Season						
Wet	540(43.7)	358(46.1)	1	1	1	1
Dry	696(56.7)	418(53.9)	0.91(0.76-1.09)	1.00(0.79-1.27)	0.97(0.77-1.23)	0.86(0.56-1.32)
Admission						
Direct	1045(84.5)	447(57.6)	1	1	1	1
Refer	186(15.0)	325(41.9)	4.09(3.31-5.05)***	4.06(3.29-5.03)***	4.04(3.27-5.00)***	4.05(3.27-5.00)***
Transfer	5	4(0.5)	1.87(0.50-7.00)	1.87(0.50-7.00)	1.90(0.51-7.11)	1.91(0.51-7.18)

Variable	Pneumonia		OR (95%CI)			
	No (n=1236)	Yes (n=776)	M1	M2 (M1+$PM_{2.5}$)	M3 (M2+Temperature)	M4 (M3+Humidity)
Sickle Cell						
No	1226(99.2)	760(97.9)	1	1	1	1
Yes	10(0.8)	16(2.1)	2.58(1.17-5.72)*	2.60(1.17-5.75)*	2.61(1.18-5.79)*	2.62(1.18-5.80)*
HIV						
No	1232(99.7)	764(98.5)	1	1	1	1
Yes	4(0.3)	12(1.5)	4.84(1.56-15.05)**	5.09(1.63-15.91)**	5.00(1.60-15.63)**	5.00(1.600-15.65)**
Heart disease						
No	1228(99.4)	744(95.9)	1	1	1	1
Yes	8(0.6)	32(4.1)	6.60(3.03-14.40)***	6.44(2.95-14.06)***	6.42(2.94-14.02)***	6.42(2.94-14.04)***
Asthma (family history)						
No	1221(98.8)	773(99.6)	1	1	1	1
Yes	15(1.2)	3(0.4)	0.32(0.09-1.10)	0.32(0.09-1.10)	0.32(0.09-1.11)	0.32(0.09-1.11)

*Univariable and multivariable logistic regression analysis. *P<0.050 **P<0.010, ***P<0*

Table 7. Comparison of the predictive ability of three different ARIMA models for daily childhood pneumonia admissions

ARIMA (p, d, q)	Stationary R^2	Normalized BIC	DF	P-value	Parameter Estimate	t	P-value
A (0, 1, 0)	0.010	2.894	18	<0.001	-0.015	-0.067	0.947
B (0, 1, 1)	0.412	2.393	17	<0.001	-0.002	-0.087	0.930
C (1, 1, 1)	0.412	2.412	16	<0.001	-0.002	-0.089	0.926

BIC=Bayesian information criterion,

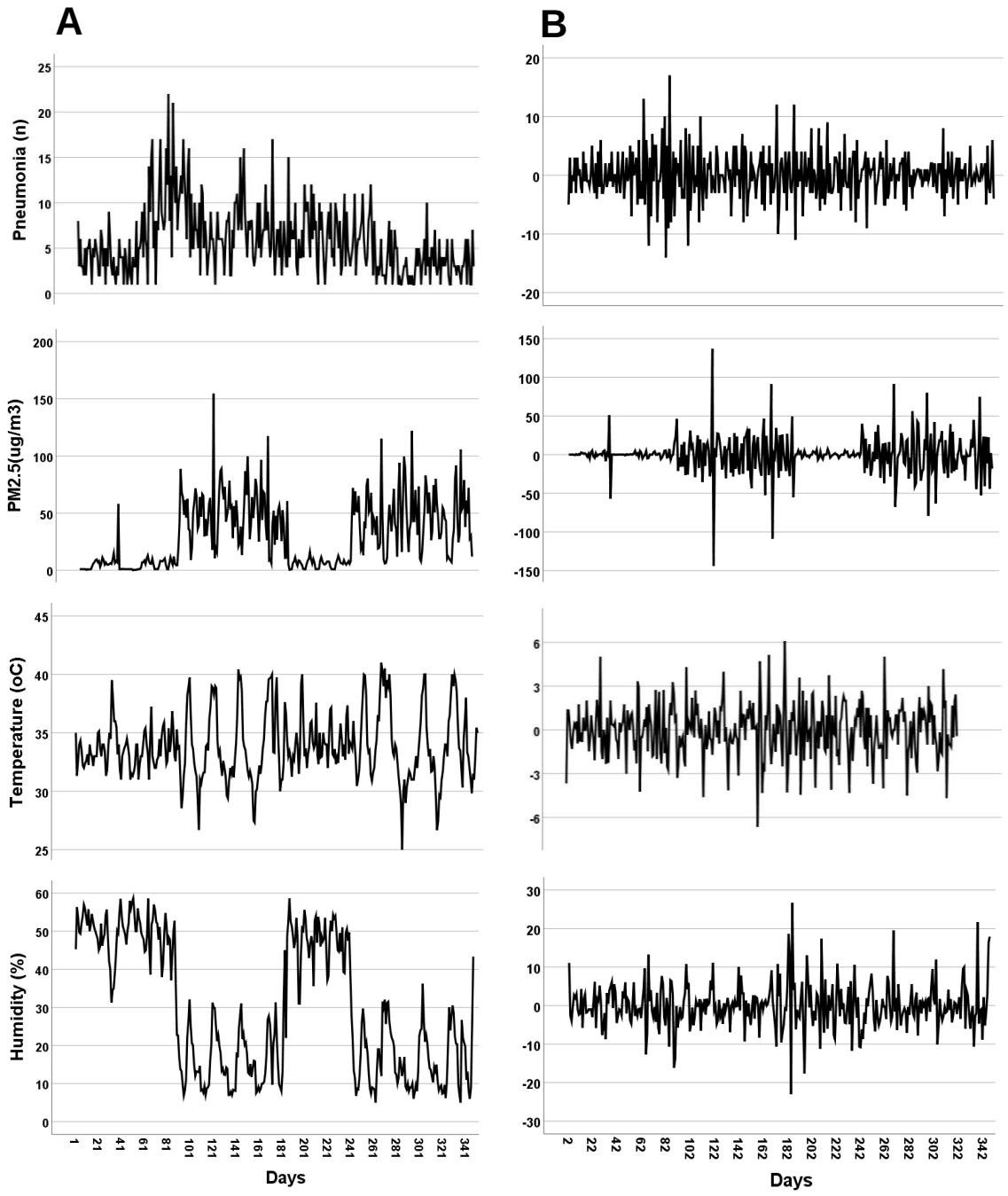

Fig. 2. Time series graph showing the daily variation of hospitalizations for childhood pneumonia and concentrations of particulate matter with aerodynamic diameter <2.5 µm (PM2.5), temperature and humidity without transformation (A) and with transformation at first level 1 differencing (B)

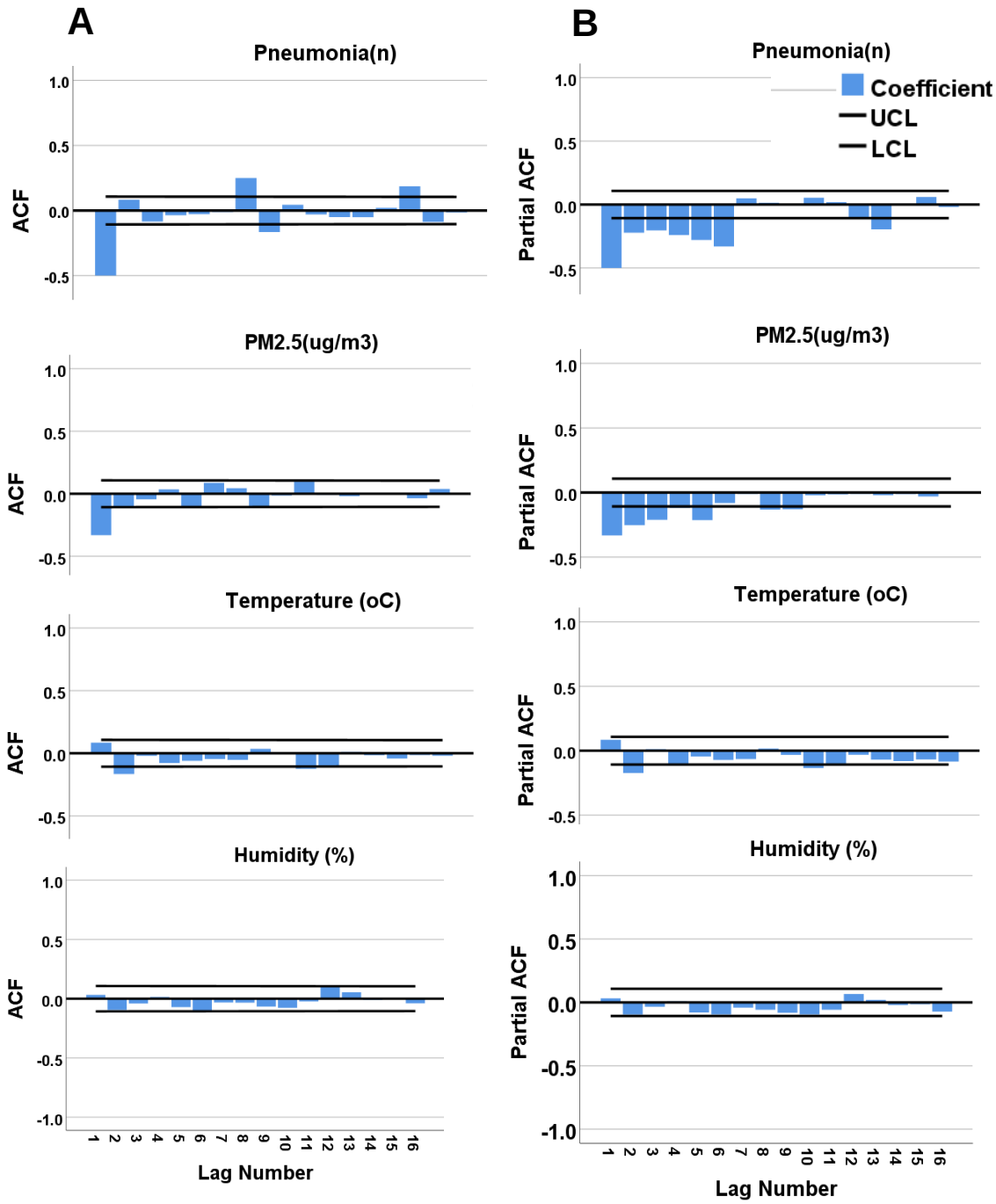

Fig. 3. Residual plots of the auto- (A=ACF) and partial autocorrelation functions (B=PACF) of the time series plots of daily childhood pneumonia admissions, PM2.5, temperature and relative humidity at first-order differencing

UCL=upper confidence limit, LCL=low confidence limit

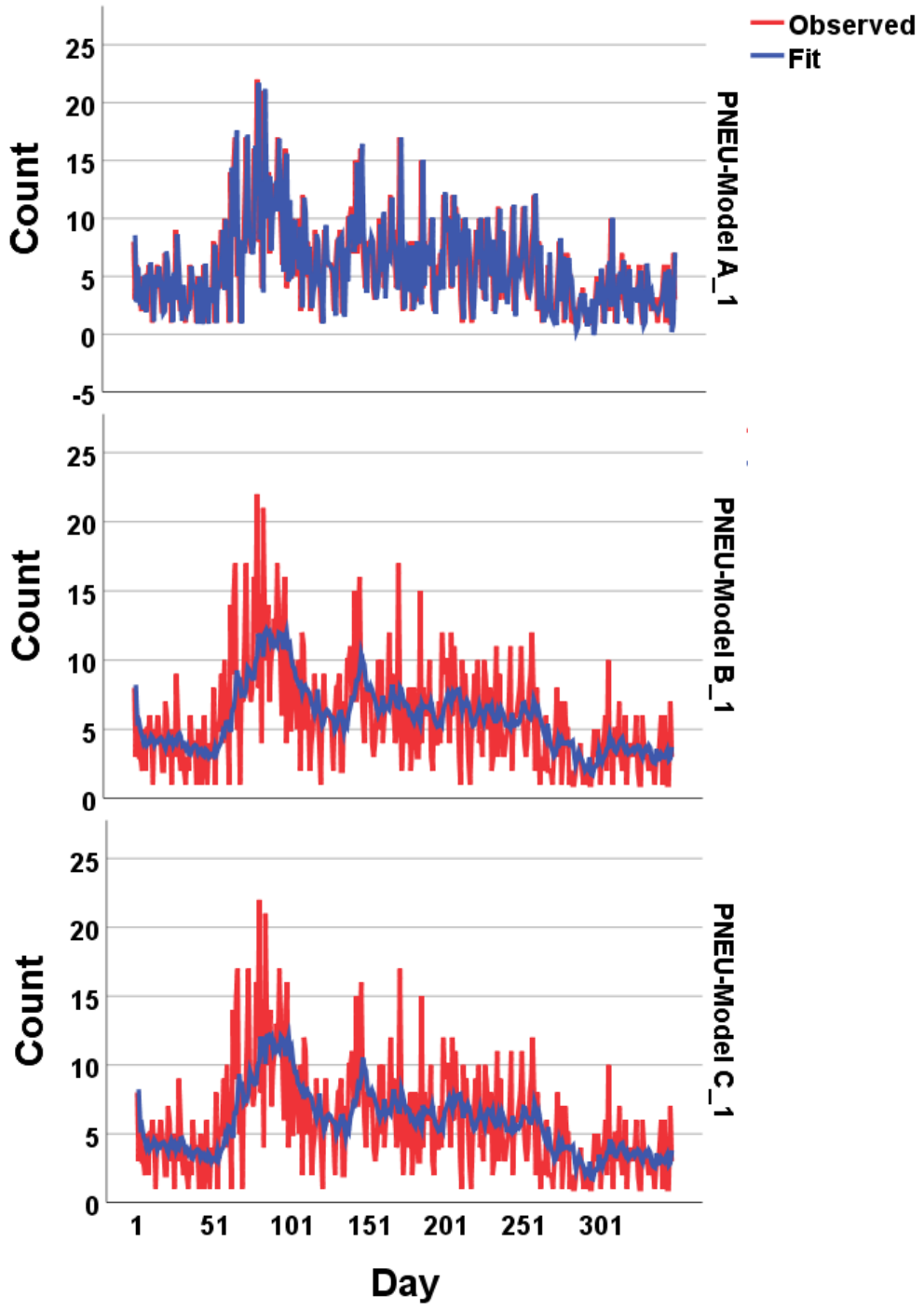

Fig. 4. ARIMA models for the prediction of daily childhood pneumonia admission counts
A=ARIMA (0, 1, 0), B=ARIMA (0, 1, 1) and C=ARIMA (1, 1, 1)

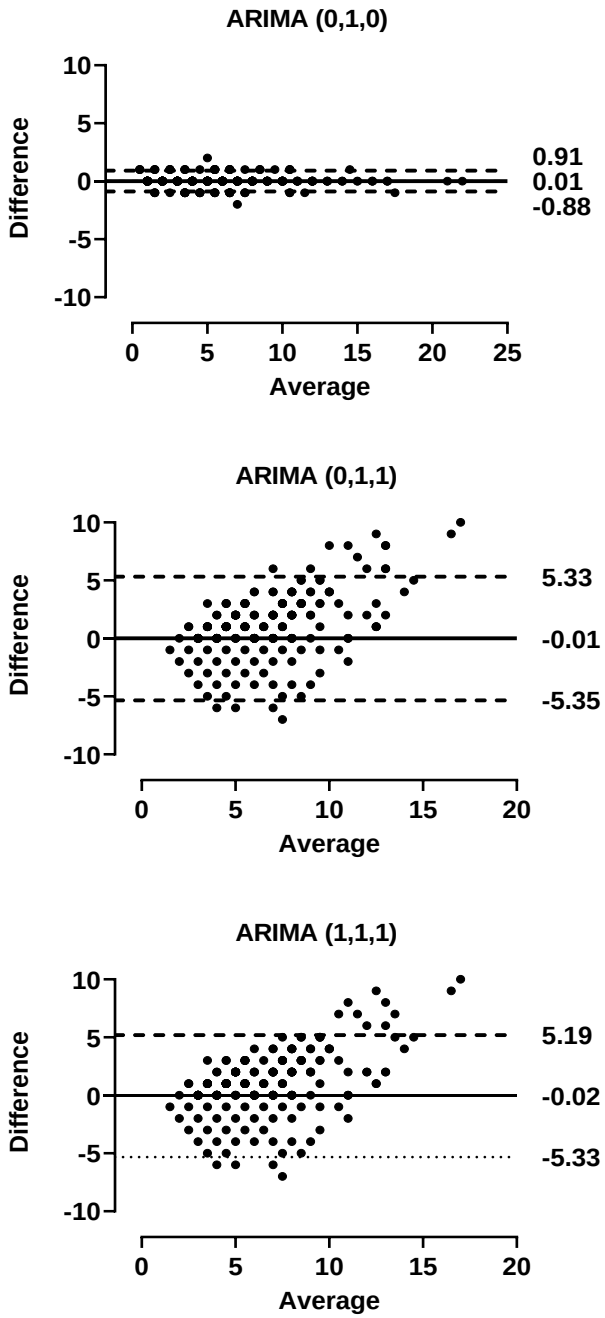

Fig. 5. Bland-Altman plots comparing the observed and the predicted daily childhood pneumonia admissions using three different ARIMA models

adjusting for temperature [AOR: 1.39(1.01-1.92)] but not after adjusting for humidity [AOR: 1.39(1.01-1.92)].

3.8 Forecasting

The times series charts for daily pneumonia admissions showed stationarity at first-order differencing (Fig. 2). The auto- and partial autocorrelation analyses at first-order differencing of the daily childhood pneumonia admissions showed a prominent ACF at lag1 which then began to decompose. In the PACF, there were six prominent lags from lag-lag6 before decomposition, (Fig. 3). From Table 7, and Fig. 3, the Ljun-Box (Q') statistic showed there was

model fitness in all three models (P>0.050). Both ARIMA (0, 1, 1) and (1, 1, 1) had a higher stationary R^2 than ARIMA (0, 1, 0). However, ARIMA (0, 1, 1) had the smallest normalized Bayesian information criterion (BIC). When the observed and the predicted daily childhood pneumonia admission counts were compared, ARIMA (0, 1, 0) had the narrowest limits of agreement although the bias did not differ from another model (Fig. 5).

4. DISCUSSION

The study sought to determine the effect of short-term $PM_{2.5}$ exposure on childhood pneumonia admissions. While there was no direct association between childhood pneumonia hospitalization and daily atmospheric $PM_{2.5}$ concentration, the association between childhood pneumonia hospitalization and age may be moderated by atmospheric $PM_{2.5}$ exposure and temperature. Also, sickle cell disease, HIV and heart disease were predictors of childhood pneumonia, independent of atmospheric $PM_{2.5,}$ temperature and humidity.

There is no direct association between childhood pneumonia hospitalizations and atmospheric $PM_{2.5}$ concentrations. However, PM2.5 may modify the relationship between pneumonia hospitalizations and the age of the children. Atmospheric $PM_{2.5}$ concentration markedly impacted the association between childhood pneumonia admissions and the age of children 5 years or older. Consistent with a previous meta-analysis, there was no evidence of an association between the physical measurement of atmospheric PM 2.5 and childhood pneumonia in low and middle-income countries (LMICs) [25]. In a previous meta-analysis that involved about 573,950 children in LMICs such as Burkina Faso, it was observed that the effect of ambient $PM_{2.5}$ on acute respiratory infection (ARI) in children was modified by sex, age and place of residence. However, contrary to the current study, evidence of $PM_{2.5}$ – associated ARI was observed in boys and children younger than 5 years [26]. Similarly, a previous study a Kenya indicated that long-term $PM_{2.5}$ exposure was significantly associated with symptoms of ARI in younger children, independent of age and sex [27]. It is, however, worth to note that while the current study focused on childhood pneumonia, the meta-analysis and the Kenyan study considered a broader group of acute respiratory infections. This could have accounted for the observed disparities between the studies, particularly in the age-specific effect of atmospheric $PM_{2.5}$.

The differential impact of PM on childhood pneumonia by age group may be due to different behaviours. Older children in most cultures are the most active and are usually allowed to play outdoors, thereby increasing their exposure to outdoor PM. Infants or children <5 years, on the other hand, are mostly kept indoors [6]. Studies have shown that breastfeeding modifies the impact of air pollutants on respiratory effects in children. Breastfeeding has a long-lasting impact on the immunological and physiological environment for respiratory diseases. Breastfeeding may affect immunological memory against inhalant allergens and thereby protect against inflammation and lung tissue damage in early childhood [28]. Breastfed children not only have reduced odds of childhood respiratory symptoms but are also impacted less by the effect of environmental toxicants [29]. Children who are breastfeeding may therefore be protected from the impact of air pollution due to improved immune function [28]. Even though not assessed in this study, children < 5 years are more likely to be breastfeeding compared to those above 5 years in Burkina Faso and other Low- and Middle-Income Countries (LMIC) [30,31]. Notwithstanding, previous studies have indicated that children <5 years were more susceptible to $PM_{2.5}$-associated pneumonia given to their immature lungs or immune system and high breathing rates which may increase their exposure per kilogram of body weight [2,32]. These observed variabilities may be due to differences in genetic and environmental factors and/or their interactions [33].

History of sickle cell disease, HIV infection and heart disease were found to be predictors of childhood pneumonia, independent of atmospheric $PM_{2.5,}$ temperature and humidity. These findings have also been reported by other authors [34]. Sickle cell disease provides an opportunity for invasive pathogens that may be associated with childhood pneumonia. A systematic review in Sub-Saharan Africa (SSA) revealed that the pooled odds of *Streptococcus pneumoniae* and *Haemophilus influenzae* type b infection were 36- and 13 times greater respectively among children with sickle cell disease [35]. It has been explained that immune function is compromised in children with sickle-cell disease due to a deficiency in serum opsonin activity, abnormalities in neutrophil kinetics and loss of splenic function given to repeated sickling

in the spleen [36]. According to a study, the odds of hospital admission for all-cause pneumonia were 6.5 times greater in HIV-infected children relative to uninfected children. Similarly, the risk of death in HIV-pneumonia comorbidity was 5.9 times greater than in pneumonia mono-infection [37]. HIV infection in children may increase their risk of pneumonia-associated pathogens such as *Streptococcus pneumonia* and *Haemophilus influenza* type b. HIV also leads to reduced immunity, paving the way for opportunistic pathogens that can cause pneumonia including *Pneumocystis jirovecii* [38]. Heart conditions such as congenital heart disease (GHD) have been associated with childhood pneumonia [39,40]. The presence of GHD affects the normal function of the circulatory system, and consequently, the respiratory system [39].

The best ARIMA model for predicting daily childhood admissions for pneumonia was the model with autocorrelation at lag0, first-order differencing and moving average 1 (0, 1, 1). While the Bland-Altman plot showed that ARIMA (0, 1, 0) was more precise [41], the model was less fit as compared to ARIMA (0, 1, 1) and ARIMA (1, 1, 1) because of its smaller stationary R^2 and larger BIC [42]. If a smaller BIC indicates a better model, then ARIMA (0, 1, 1) should be the preferred model for forecasting childhood pneumonia admissions in this study.

The current study has some strengths: (1) While particulate matter pollution is widely documented in Africa, there is a paucity of information on the impact of $PM_{2.5}$ on childhood pneumonia admissions, (2) Aside from the total study sample, subgroup analyses were also performed to determine the group-specific impact of $PM_{2.5}$ on childhood pneumonia admissions and (3) models were created and compared to determine the best model for predicting childhood pneumonia admissions. However, the study has some limitations: firstly, while breastfeeding may modify the impact of PM_2 on childhood pneumonia, this was not directly determined in the current study. Secondly, the composition of PM_2 has an impact on pneumonia but this was not determined. Thirdly, while this study involved epidemiological individual data, the associations measured here may not be true at the individual level, given the risk of ecological fallacy. In addition, the generalizability of the findings beyond the study's location and the potential impact of unmeasured confounding variables may be challenged. Finally, the study was retrospective and may be subject to sampling bias [43].

5. CONCLUSION

The air pollutant, $PM_{2.5}$ has an impact on childhood pneumonia admissions but only among children 5 years and above. The impact of $PM_{2.5}$ on childhood pneumonia admissions may be moderated by age. It is recommended that children ≤5 years should be protected from exposure to $PM_{2.5}$ in Ouagadougou, Burkina Faso. It is also preferable to use the ARIMA (0, 1, 1) model to predict future childhood admissions for pneumonia in Burkina Faso.

CONSENT AND ETHICAL APPROVAL

Written informed consent was obtained from all subjects or their legal guardians. The study followed the guidelines for human subject studies as contained in the 1964 Declaration of Helsinki and its later amendments. The study was approved by the Ethics Committee for Health Research (Comité d'Ethique pour la Recherche en Santé, CERS; N#: 2020-8-166) in Burkina Faso. All record codes and patient identifiers were removed to ensure anonymity.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of this manuscript.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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