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Children's respiratory hospitalizations linked to short-term air pollution exposure in Peninsular Malaysia's urban areas

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Urban air pollution is a major global health issue for children. While air pollution is linked to respiratory hospitalizations, the relationship between ambient air pollutants and schoolchildren's health remains unclear. This study examined the short-term impact of air pollution on respiratory hospitalizations among schoolchildren (aged 7–12) in urbanized areas of Peninsular Malaysia (2018–2020). A Quasi-Poisson Generalized Linear Model (GLM) combined with Distributed Lag Non-linear Model (DLNM) was applied to daily hospital admission data and ambient air quality data (SO_2, NO_2, O_3, CO, PM_{10}, $PM_{2.5}$, benzene, toluene, ethylbenzene, and xylene) from the Department of Environment Malaysia at five air monitoring stations (ST1: Seberang Jaya; ST2: Cheras; ST3: Shah Alam; ST4: Malacca City; ST5: Kuala Terengganu). At ST1, a 10 µg/m^3 increase in pollutant levels was significantly associated ($p < 0.05$) with children's hospitalizations, with the highest relative risks (RR) observed at lag 3 for benzene (RR = 1.091), lag 0 for PM_{10} (RR = 1.134), and lag 6 for SO_2 (RR = 1.331). At ST3, toluene (RR = 1.256), $PM_{2.5}$ (RR = 1.121), and PM_{10} (RR = 1.092) showed significant immediate effects at lag 0. At ST4, NO_2 was significantly associated at lag 6 (RR = 1.124). For cumulative–lag effect, benzene (RR = 1.834) and PM_{10} (RR = 1.883) at ST1 were significant at lag 06; while toluene (RR = 1.291) at lag 01 for ST4. The findings indicate both immediate and delayed respiratory effects of specific pollutants in children. Urban policymakers must implement targeted actions including low-emission school zones, stringent industrial discharge controls, and enhanced haze protocols to protect children's respiratory health in urban settings.

Keywords Ambient air pollution, Respiratory illness, Schoolchildren, Hospital admissions, Distributed lag non-linear model

Urban air pollution has emerged as a critical global health concern, with profound implications for both environmental sustainability and human health. Ambient air quality refers to the concentration of pollutants such as particulate matter (PM), nitrogen dioxide (NO_2), sulphur dioxide (SO_2), ozone (O_3), carbon monoxide (CO), and volatile organic compounds (VOCs)[1]. These pollutants are primarily generated by anthropogenic activities, including industries, traffics, and biomass burning, which are prevalent in rapidly urbanizing regions worldwide[2–7]. Despite regulatory efforts to control pollutant levels, studies consistently show that exposure to even moderately elevated concentrations of these pollutants can result in significant adverse health effects, particularly for vulnerable population, including children[8–10].

Epidemiological evidence has firmly established associations between urban air pollution and a wide range of health issues, notably respiratory diseases and cardiovascular diseases[11–16]. Hospital admissions for these conditions often rise in tandem with elevated levels of air pollutants, reflecting the direct impact of urban air quality on public health. Studies in Malaysia on ambient air pollution exposure on hospitalizations have been

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reported widely, particularly on the criteria pollutants including SO_2, NO_2, CO, O_3, and PM_{10}, even where the current air quality standards were being met[17–20].

Schoolchildren are particularly vulnerable to the adverse effects of air pollution, as they spend a significant portion of their daily lives in school environments with school days lasting between 5 and 8 h[21]. This makes them an at-risk group for exposure to ambient pollutants[22–24]. Given their developing respiratory and immune systems, children are more susceptible to the harmful effects of air pollution compared to adults[25]. Furthermore, schools are often located in areas with high traffic and industrial activity, exacerbating exposure levels[7,26–28].

Despite a growing body of research globally on the exposure to ambient air pollutants and their associations with hospital admissions, there is a lack of studies examining the specific relationship between ambient benzene, toluene, ethylbenzene, and xylene, or also known as BTEX compounds, on exposure and respiratory hospitalizations. Numerous studies have reported the adverse effects of inhaling these organic compounds in urban and industrial areas worldwide[2, 29,30], yet their impact on hospital admissions remains underexplored, particularly in terms of exposure-specific health outcomes in schoolchildren. This study aims to fill in the knowledge gap on the impacts of exposure to ambient air pollutants, including SO_2, NO_2, O_3, CO, PM_{10}, $PM_{2.5}$, and BTEX, on the respiratory admissions among schoolchildren, using the robust quasi-Poisson Generalized Linear Models (GLM) with Distributed Lag Non-Linear Model (GLM-DLNM) statistical approach which accounts for the lagged and non-linear effects of air pollution[31]. This combined approach allows for a comprehensive analysis of both immediate and delayed health effects, providing crucial insights into the timing and magnitude of exposure-related risks.

Materials and methods

Study area

Malaysia is situated between 2°30'N, 112°30'E, within 150 km of the Equator, in central Southeast Asia on the South China Sea. The country is comprised of Peninsular Malaysia (West Malaysia) and states of Sabah and Sarawak (East Malaysia) on the island of Borneo with approximately 330,000 square kilometers in area[32]. Malaysia has two monsoon seasons, known as the Northeast monsoon (NEM) and the Southwest monsoon (SWM), which occur from November to March and from May to September. Occasionally, the north-east monsoon brings heavy rain while the south-west monsoon causes a less of cloud formation, resulting in dry and drought season during this period. Other than the monsoon seasons, Malaysia is safe from natural disasters such as volcanic eruptions and typhoons[32–34].

For this study, monitoring stations were selected from four key regions in Peninsular Malaysia: Northern, Central, Eastern, and Southern regions of Peninsular Malaysia, as presented in Table 1; Fig. 1. These regions are rapidly developing, with growing populations, increasing numbers of vehicles, rising traffic density, and expanding industrial zones[35–38].

Exposure assessment

For the exposure assessment of ambient air pollutants, daily air quality data spanning a three-year period (April 2018–December 2020) from five continuous air quality monitoring stations were retrieved from the Air Quality Division of the Department of Environment (DOE), Malaysia based on the availability of data. These areas were chosen not only for data availability but also due to their frequent reports of high Air Pollutant Index (API) levels. Contributing factors include heavy traffic, industrial and development activities, and the impact of transboundary haze[1].

This study selected 10 pollutants, which included SO_2, NO_2, O_3, CO, $PM_{2.5}$, PM_{10}, and BTEX compounds, and two meteorological parameters (temperature and relative humidity). Except for O_3 and CO which were computed using a maximum of 8-hour moving average concentration, other pollutants were calculated by

Station	Name of hospital	Distance from CAQMS	Coordinate (Lat, Long)	Area characteristics
Seberang Jaya, Penang (ST1)	Hospital A	3.6 km	5.39, 100.40	Located in the residential and commercial areas such as Seberang Jaya town. This is a popular area and located in the centre of the Penang mainland. The population is among the highest because having road networks that connect of the North, South, East, and Penang Island[39].
	Hospital B	11.2 km	5.36, 100.46	
Cheras, Kuala Lumpur (ST2)	Hospital C Hospital D	12.6 km 9.1 km	3.17, 101.70 3.12, 101.76	ST2 and ST3 located in the Central region of Peninsular Malaysia in the Klang Valley, Malaysia, which comprises Kuala Lumpur and its suburbs and adjoining cities and towns in the state of Selangor[35]. Cheras station includes both residential and industrial areas. There are several schools that are located here and it is nearby a major highway[37].
Shah Alam, Selangor (ST3)	Hospital E	14.3 km	3.07, 101.48	Located in a residential area and surrounded by an airport and it is also near to the major roads and a few industries operate here, thus the traffic is significantly higher during peak hours, which are in the morning and late afternoon[35].
	Hospital F	20.0 km	3.02, 101.43	
Bandaraya Melaka, Melaka (ST4)	Hospital G	4.5 km	2.21, 102.26	Located in the urban centre of Melaka state faces the Malacca Straits to the west in a residential area and is near to tourist attractions, commercial areas, Malacca Port and Melaka Airport. This area is expected to be congested with heavy traffic due to its nearby attractions[38].
Kuala Terengganu, Terengganu (ST5)	Hospital H	7.9 km	5.33, 103.15	Located in the urban East Coast of Peninsular Malaysia, is less than 2 km from the shoreline, with an estimated population of over one million (Mohd Tahir et al., 2009). Besides situated closely to the sea, this area experiences two main monsoons annually and two inter-monsoons in between and the weather characteristics varies greatly here[36].

Table 1. Locations and area characteristics of the air monitoring stations and selected hospitals. CAQMS=Continuous air quality monitoring station; Lat=Latitude; Long=Longitude; ST= station.

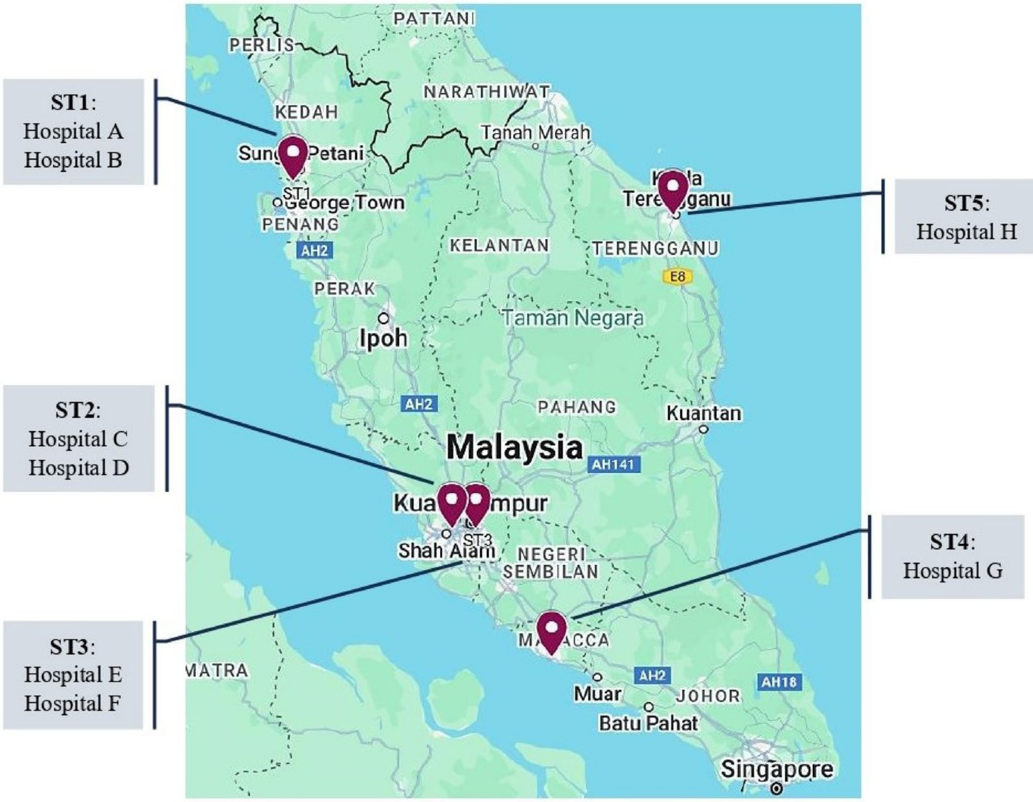

Fig. 1. Locations of the studied areas on the map of Malaysia.

averaging the hourly concentrations and all the air quality data were presented in daily means. The daily values of air quality data were determined using the rule of 75% completeness (18-hour observation) and the values were considered as "missing" if do not meet the rule[18]. The missing values accounted in this study were 13% (ST1), 14% (ST2), 14% (ST3), 12% (ST4), and 15% (ST5), respectively, of all observations across the study period. The missing values for the air quality data were imputed using the Multivariate Imputation by Chained Equations (MICE) algorithm from the MICE package in R Studio. This approach was chosen due to the extensive used in epidemiological air pollution studies and has demonstrated better performance than other imputation methods[40–43].

Outcome assessment

Daily respiratory hospital admission records for primary schoolchildren were retrieved from eight selected government-owned public hospitals from the database of Ministry of the Health Malaysia. These hospitals are located within 20 km of the air monitoring stations[44] (Table 1). These records cover the period from the start of air pollutant data collection until its completion. A hospital stay of at least one night is characterized as a hospital admission[45]. Daily counts of hospital admissions from respiratory health diseases (J00-J99) were selected in accordance with the International Classification of Diseases, 10th Revision (ICD10). The hospital admission records note the variables that are required in this study which includes; date of admission, principal diagnosis of discharge, and date of discharge. The selection of the patients from the medical records was based on the inclusion criteria, included male and female primary schoolchildren aged 7 to 12 years old; living near to the air monitoring stations (within 20 km); do not have any history of chronic respiratory health diseases; and being admitted to ward for at least one night during study period. As this was done anonymously, informed consent to the patients is not required.

Data analysis

An analytical method of time-series was adapted in this study from the common Public Health and Air Pollution in Asia (PAPA) protocol[46]. This protocol includes specifications for the selection of monitoring stations, quality assurance and quality control (QA/QC) procedures for data collection and data analysis, which has been adopted in past studies[15,18,19].

Descriptive analysis was conducted by presenting the quantitative data, including hospital admissions, air pollutant concentrations, and meteorological parameters, in terms of mean, standard deviation, and percentiles. Time series plots of air pollutants and meteorological parameters were created to observe potential trends throughout the study period. Spearman's correlation test was then used to assess the relationships between ten air pollutants and two meteorological variables.

Generalized linear model (GLM) and distributed lag non-linear model (DLNM)
A significant number of previous studies utilized the generalized additive model (GAM), generalized linear model (GLM), and distributed lag model (DLM) to estimate the short-term effects of air pollution on health outcomes[15,47–50]. However, according to Gasparrini[31], those methods are limited in capturing non-linear exposure-response relationships. Thus, in this present study, a distributed lag non-linear model (DLNM) was applied, adjusting for various covariates to describe the non-linear relationship between air pollution and incidence of respiratory hospital admissions among schoolchildren. The function captures the total effect of hospitalizations on the same day from the accumulated effects of air pollutant concentration over that day and up to a 6-day lag[31].

At the beginning of the procedure, the core model of quasi-Poisson GLM was established by incorporating adjustments for long-term trends, seasonality and other potential confounding variables such as temperature, relative humidity, holiday indicators, and days of the week (DOW). The DOW variable was coded sequentially from Sunday (1) to Saturday (7), while the public holiday indicator was coded as 1 for a holiday and 0 for a non-holiday. In this quasi-Poisson model, a natural spline smoothing function of time with 8 degrees of freedom (*df*) per year was applied in the regression model to adjust for seasonality patterns in respiratory cases and other time-varying covariates that might confound the association between air pollution and respiratory hospitalizations among children. The quasi-Poisson Akaike Information Criterion (Q-AIC) was used as the basis to determine the model fits by selecting the degree of freedom (*df*) for seasonal and long-term trends of respiratory admissions, air pollutants and their lags, and a natural spline with 3 *df* was used to adjust for the effects of temperature and relative humidity in this study, as applied by the previous studies[15,18,51].

After the establishment of the core model, cross-basis matrices for all pollutants were then constructed and included in the formula. As the main element of DLNM is the construction of a cross-basis function, this will simultaneously express the effects in the space of exposure and its lag dimension. In this study, natural cubic splines were carried out for both exposure and lag-response in this cross-basis function with a maximum lag of six days, given that the correlation between children's respiratory diseases and short-term exposure to air pollutants may last for seven days[45,52]. The value of *df* natural cubic spline that generated the lowest Q-AIC (Appendix A) value was selected in both exposure-response and lag-response relationships[53]. The centering or reference value for each pollutants is assumed to be the risk exposure level that minimizes respiratory diseases at the population level, which was defined as the 1st percentile of observed concentration[18]. Formula in the Eq. (1) was used to present the main model of this study.

$$\begin{aligned}\log\left(E\left(Yt\right)\right) = \ & \alpha\ + cb\left(xt\right) + ns\left(Time,\ df = 8/year\right) + \ ns\left(Temp,\ df\ =\ 3\right) \\ & +\ ns\left(RH,\ df = 3\right) + DOW + Holiday\end{aligned} \tag{1}$$

where, E(Yt) = the expected number of respiratory hospitalizations on day *t*; α = the intercept; *cb* = the cross-basis function; x_t=the daily mean level of each pollutant; ns = the natural spline function; Time = to model the trend and seasonality; *df* = the degree of freedom; Temp=daily temperature; RH=relative humidity; DOW = day of week; Holiday=public holiday.

The relative risk (RR) and 95% confidence interval (CI) were used to represent the findings of the analysis for the daily respiratory hospital admissions for the per 10 μg/m^3 increment of BTEX, $PM_{2.5}$, PM_{10}, SO_2, NO_2, O_3 and 1 mg/m^3 in CO at single-lag effect (from lag 0 to lag 6) and cumulative-lag effects of lag 01 to 06. RRs were reported per 10 μg/m³ increase to allow direct comparison with similar local time-series studies using the same increment (e.g. Ibrahim et al.[18], Tajudin et al.[16], Rahim et al.[54].

Sensitivity analysis
Sensitivity analysis was performed to measure the consistency and robustness of the estimates and impact of the model choices by altering various levels of dfs with 6, 8, and 10 *df* per year for the long-time trend and *df* 3–5 for the meteorological factors in the core model. The models were considered as stable if there was no apparent change in the risk estimates of hospital admissions after the adjustments[19]. All statistical analyses and visualizations were performed by R Studio Software Ver 4.4.1 with the "dlnm" and "splines" packages to run the regression analyses.

Results and discussion
Descriptive analysis
The descriptive statistics of hospital admissions for respiratory diseases and the distribution of air pollution and atmospheric parameters are presented in Table 2. Based on the analysis, from 1st April 2018 to 31st December 2020, there were 6,454 cases of hospital admissions among schoolchildren aged 7 to 12 years old for respiratory diseases in those selected areas. The highest daily average concentrations of the air pollutants and meteorological parameters from year 2018 to 2020 were recorded as follow: benzene and xylene at ST1 (3.94 μg/m^3, 12.13 μg/m^3); toluene, ethylbenzene and O_3 at ST2 (9.34 μg/m^3, 2.55 μg/m^3, 44.37 μg/m^3); $PM_{2.5}$, PM_{10}, CO and NO_2 at ST3 (25.11 μg/m^3, 34.06 μg/m^3, 1.10 mg/m^3, 16.10 μg/m^3); SO_2 and temperature at ST4 (1.79 μg/m^3, 28.29 °C); while relative humidity at ST5 (83.24%), respectively.

Appendix B shows the trends of daily air pollutant levels, temperature and relative humidity across stations throughout the study period. Air quality in all studied stations were relatively acceptable and below the recommended limits (below permissible level for SO_2, O_3, and CO) suggested by the Standard 2020 of Malaysia Ambient Air Quality Standard (MAAQS)[55], excepts for $PM_{2.5}$ and PM_{10} at all stations (24-h $PM_{2.5}$ = 35 μg/m^3; 24-h PM_{10} = 100 μg/m^3). The yearly visible peaks of particulate matter concentrations in year 2019 were probably caused by local and transboundary haze events, which are prevalent in Southeast Asia countries[56].

Traditional agricultural practices, including "slash and burn" deforestation and the cultivation of oil palm on peatland, particularly in Indonesia, have been identified as major contributors to severe transboundary haze episodes in Malaysia[4,57]. Additionally, due to no recommended standard of BTEX introduce in Malaysia, and WHO does not advocate any safe level of exposure to these compounds, thus these pollutants were compared with guideline established by other country. The Massachusetts Department of Environmental Protection in the United States of America (MassDEP)[58] implemented 24–hour safe benzene, toluene, ethylbenzene, and xylene exposure at 0.6 $\mu g/m^3$, 80 $\mu g/m^3$, 300 $\mu g/m^3$, and 11.8 $\mu g/m^3$, accordingly. Based on daily trends of BTEX, benzene and xylene across stations had exceeded this guideline. These cities have significant vehicular traffic and industrial zones, which are major sources of volatile organic compounds emissions from the incomplete combustion of petrol and diesel and industrial activities[59,60]. Northern, Central, and Southern regions had the greatest number of pollutants with the highest readings. Located in the most developed and urbanized areas of Malaysia, these stations are expected to have poor ambient air quality compared to others[61–63].

Moreover, high level of temperature at ST4 may be due to the location of the station in the urban centre of Melaka closed to Malacca Port and Malacca Airport which had encounter high temperature in most of the days over the year, without much fluctuation[64,65]. ST5 had the highest reading of relative humidity which may related to the presence of sea breeze from the coastal line that has been proven as one of a very important cooling effect factors to seaside cities[66]. Besides, Terengganu experiences Northeast monsoon every year with heavy rainfall for few months, thus might influenced the level of relative humidity in this area[67].

Correlation between air pollutants and meteorological variables

The results of the Spearman's correlation test showed strongest positive correlation between $PM_{2.5}$ and PM_{10} ($r > 0.90$, $p < 0.01$) across all stations, which indicates that as the level of one variable increases, the level of the other one is also increasing significantly[68]. The relation between $PM_{2.5}$ and PM_{10} was correlated with each other as they share common sources and physical processes that generate and transport them, which include vehicle emissions, industrial and construction activities, and burning of fossil fuel[3, 69–71]. These pollutants also have been associated with the transboundary haze event from the biomass burning that often impact the air quality in Malaysia[4, 72]. Result of this present study also was consistent with a previous study conducted by Li et al.[73] which reported on the strong positive correlation between PM_{10} and $PM_{2.5}$ with $r = 0.92$.

In addition, strong positive relationships ($r > 0.60$ to 0.79, $p < 0.01$) were found between the BTEX species across the stations. Positive correlation coefficient between inter-species explained that gasoline vehicular exhaust could be the major source of BTEX in the studied areas. Previous studies had reported on the most widespread source of anthropogenic BTEX in ambient air particularly in urban areas was which through the burning of gasoline and diesel fuels in motor vehicles, stationary engines, and other equipment[2,74–76].

Association between air pollutants and children's respiratory hospitalizations

Relative risk (RR) is often used to express the likelihood of an event happening in an exposed group, compares to the non–exposed group. This value helps measures the strength of the association between exposure and the health outcome[77]. Based on the analysis, significant associations were observed between ambient air pollutants with the children's hospital admissions at ST1 (Table 3), ST3 (Table 4) and ST4 (Table 5). Thus, this section highlights and discuss the findings from these three stations only.

Single-lag exposure examines the effects of exposure at a specific time point (lag) separately on the outcome, meanwhile cumulative-lag exposure combines the effects over a range of lags to provide the overall or total influence of exposure across multiple time points[31]. Based on the results in Table 3 for ST1, benzene was observed for single-lag of lag 3 with RR 1.091 (95% CI 1.008–1.180, $p = 0.031$) and cumulative-lag of 06 with RR 1.834 (95% CI 1.057–3.183, $p = 0.031$); PM_{10} at lag 0–4 where the highest was at immediate exposure of lag 0 with RR 1.134 (95% CI 1.002–1.286, p = 0.047) for single-lag and lag 06 with RR 1.883 (95% CI 1.270–2.792, $p = 0.002$) for cumulative-lag; and SO_2 at lag 4–6 where the highest was at lag 6 with RR 1.331 (95% CI 1.051–1.686, p = 0.018). These indicated that every 10 $\mu g/m^3$ increase in benzene, PM_{10} and SO_2 levels, corresponded to the following RR increased in children's respiratory hospitalizations at ST1. Based on the findings at ST1, these suggest that the health outcomes related to benzene exposure are not immediate but delayed, where repeated or sustained exposure over several days amplifies health risks. This implies that prolonged or sustained exposure may be more harmful than shorter, isolated exposure events[31]. This delayed effect is consistent with benzene's biological mechanism, where exposure can lead to systemic inflammation, oxidative stress, and immunosuppressive effects that develop over time rather than triggering immediate respiratory symptoms[78,79]. As for PM_{10}, this reveals that PM_{10} exposure has both immediate and delayed health impacts, with a particularly notable effect occurring nearly a week after exposure. The lag structure from day 01 to day 06 indicates a cumulative inflammatory response or delayed onset of respiratory conditions, which is consistent with the pathophysiological effects of particulate matter[80,81]. Meanwhile, health effects of SO_2 exposure on respiratory admissions are not immediate but occur after several days, which is aligns with the idea that SO_2 can trigger inflammation and respiratory stress that manifest with a time lag[82]. The elevated levels of benzene, PM_{10}, and SO_2 observed at the Seberang Jaya monitoring station are likely attributable to a combination of vehicular traffic (located near to the North-South Expressway)[61,39], industrial activity (notably from the nearby Perai Industrial Zone)[83], shipping emissions (from nearby Penang Port or Butterworth)[84] and possibly transboundary haze or local biomass burning[85].

As for ST3, the findings were presented in Table 4. Significant associations for single-lag were found for toluene at lag 0 with RR 1.256 (95% CI 1.084–1.461, $p = 0.002$) and at lag 01 with RR 1.291 (95% CI 1.080–1.544, $p = 0.004$) for cumulative-lag; $PM_{2.5}$ at lag 0 with RR 1.121 (95% CI 1.036–1.213, $p = 0.003$); and PM_{10} at lag 0 with RR 1.092 (95% CI 1.019–1.170, $p = 0.009$). The findings suggest that immediate exposure to toluene and particulate matter contributes to acute respiratory events in children living near the ST3. Toluene, commonly emitted from traffic exhaust and industrial processes[59], is known to irritate the respiratory tract and affect lung

Variable	Respiratory admissions	Benzene (μg/m^3)	Toluene (μg/m^3)	Ethylbenzene (μg/m^3)	Xylene (μg/m^3)	$PM_{2.5}$ (μg/m^3)	PM_{10} (μg/m^3)	*O_3 (μg/m^3)	*CO (mg/m^3)	SO_2 (μg/m^3)	NO_2 (μg/m^3)	Temperature (°C)	Relative humidity (%)
ST1													
Total	837												
Min	1	0.15	0.24	0.06	0.37	6.07	8.81	2.93	0.46	0.25	2.6	24.3	58.73
Q1	1	3.19	4.59	1.38	8.8	13.95	20.03	24.8	0.89	0.84	11.34	26.92	74.23
Median	1	3.84	9.22	2.15	12.27	18.11	25.59	33.5	1.07	1	13.82	27.82	77.9
Mean	1	3.94	8.58	2.07	12.13	20.1	27.45	33.96	1.07	1.06	13.83	27.74	77.74
Q3	2	4.62	12.24	2.74	15.61	23.84	32.52	43.19	1.22	1.24	16.16	28.63	81.98
Max	5	8.1	21.94	5.22	25.26	126.22	130.93	77.35	2.17	3.51	28.47	30.58	92.59
ST2													
Total	1668												
Min	1	0.13	0.1	0.19	1.18	6.93	10.62	8.29	0.37	0.91	3.13	24.71	54.07
Q1	1	1.31	5.67	1.77	5.2	14.7	21.35	34.66	0.81	0.6	12.23	27.27	72.43
Median	2	2.06	9.51	2.55	7.33	19.18	27.33	43.31	0.99	0.79	15.64	28.14	76.86
Mean	2	2.23	9.34	2.55	7.98	22.34	30.44	44.37	0.99	0.9	15.47	28.1	76.63
Q3	3	3.03	13.13	3.31	10.33	25.62	35.55	53.26	1.16	1.06	19.05	28.92	81.3
Max	10	6.7	24.25	6.16	23.47	135.53	151.47	84.88	2.25	4.11	31.55	30.97	93.23
ST3													
Total	1966												
Min	1	0	0	0	0	5.97	8.43	3.31	0.34	0.23	2.47	23.65	56.35
Q1	1	2.04	4.21	1.22	5.93	17.12	24.38	34.75	0.85	0.92	12.31	26.61	74.84
Median	2	2.82	7.85	2.19	9.16	22.03	30.81	42.73	1.06	1.17	16.08	27.61	78.93
Mean	2	2.94	9.25	2.42	10.14	25.11	34.06	44.24	1.1	1.26	16.1	27.56	78.7
Q3	3	3.69	12.43	3.4	13.58	29.54	39.75	52.81	1.3	1.5	19.98	28.49	83.18
Max	11	7.73	41.14	10.61	47.48	144.92	156.55	99.15	2.38	4.69	33.69	30.67	96.63
ST4													
Total	943												
Min	1	0	0	0	0	3.2	7.57	10.62	0.19	0.1	0.92	23.3	61.19
Q1	1	1.15	1.33	0.48	1.57	11.01	16.57	31.54	0.48	1.18	4.91	27.68	75.57
Median	1	1.76	3.28	0.85	2.57	15.2	21.57	38.19	0.6	1.69	6.79	28.4	78.51
Mean	2	1.76	3.93	1.1	2.85	18.46	24.9	39.93	0.62	1.79	6.81	28.29	78.3
Q3	2	2.33	6.03	1.59	3.85	21.87	28.51	47.01	0.72	2.28	8.45	28.94	81.42
Max	6	4.26	26.44	4.52	10.38	151.1	173.9	99.17	2.26	6.15	15.84	30.67	94.2
ST5													
Total	1040												
Min	1	0	0	0	0	3.01	5.97	5.75	0.19	0.1	0.95	22.66	71.2
Q1	1	0.38	0.2	0.41	1.48	11.16	18.84	20.33	0.56	0.58	4.07	26.26	80.2
Median	1	0.74	0.46	0.74	2.87	15.45	24.32	25.04	0.66	0.81	5.12	26.98	82.43
Mean	2	0.97	1.24	0.79	3.68	16.99	25.51	26.01	0.67	0.86	5.07	26.94	83.24
Continued													

Variable	Respiratory admissions	Benzene (μg/m^3)	Toluene (μg/m^3)	Ethylbenzene (μg/m^3)	Xylene (μg/m^3)	$PM_{2.5}$ (μg/m^3)	PM_{10} (μg/m^3)	*O_3 (μg/m^3)	*CO (mg/m^3)	SO_2 (μg/m^3)	NO_2 (μg/m^3)	Temperature (°C)	Relative humidity (%)
Q3	2	1.33	1.36	1.05	4.69	20.7	29.74	30.72	0.76	1.03	6.08	27.72	85.71
Max	6	4.39	9.33	3.63	21.3	103.26	118.19	55.78	1.44	2.69	14.37	30.3	99.01

Table 2. Summary statistics of daily hospital admissions for children's respiratory hospitalizations and air quality across stations from 2018 to 2020. min=minimum; Q1 = first quartile; Q3 = third quartile; max=maximum; ST=station; *8-h moving average concentration.

	Benzene	Toluene	Ethylbenzene	Xylene	$PM_{2.5}$	PM_{10}	O_3	*CO	SO_2	NO_2
Lag	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
0	1.063 (0.869–1.299)	0.948 (0.845–1.064)	1.044 (0.880–1.239)	0.954 (0.834–1.091)	1.019 (0.969–1.071)	**1.134 (1.002–1.286)**	1.066 (0.984–1.156)	0.994 (0.816–1.211)	0.880 (0.703–1.101)	1.008 (0.839–1.210)
1	1.072 (0.926–1.240)	0.966 (0.889–1.049)	1.039 (0.913–1.183)	0.969 (0.879–1.068)	1.018 (0.980–1.058)	**1.121 (1.020–1.232)**	1.015 (0.971–1.061)	1.020 (0.876–1.187)	0.942 (0.801–1.109)	1.030 (0.898–1.182)
2	1.081 (0.979–1.194)	0.985 (0.932–1.041)	1.035 (0.941–1.138)	0.985 (0.923–1.051)	1.018 (0.989–1.047)	**1.108 (1.034–1.187)**	0.977 (0.940–1.016)	1.046 (0.932–1.174)	1.010 (0.903–1.129)	1.052 (0.952–1.163)
3	**1.091 (1.008–1.180)**	1.004 (0.963–1.047)	1.031 (0.952–1.115)	1.001 (0.953–1.051)	1.017 (0.994–1.041)	**1.095 (1.035–1.158)**	0.960 (0.916–1.007)	1.073 (0.973–1.184)	1.082 (0.988–1.185)	1.076 (0.989–1.169)
4	1.100 (0.995–1.216)	1.023 (0.969–1.081)	1.026 (0.936–1.124)	1.017 (0.955–1.083)	1.017 (0.991–1.047)	**1.082 (1.014–1.154)**	0.971 (0.933–1.011)	1.101 (0.986–1.229)	**1.159 (1.029–1.306)**	1.100 (0.995–1.214)
5	1.110 (0.957–1.286)	1.043 (0.961–1.132)	1.021 (0.901–1.157)	1.033 (0.940–1.136)	1.016 (0.981–1.052)	1.069 (0.979–1.167)	1.004 (0.963–1.046)	1.129 (0.978–1.305)	**1.242 (1.044–1.477)**	1.123 (0.980–1.287)
6	1.119 (0.914–1.370)	1.063 (0.948–1.192)	1.016 (0.861–1.201)	1.050 (0.921–1.198)	1.016 (0.970–1.064)	1.056 (0.939–1.189)	1.048 (0.973–1.128)	1.159 (0.959–1.399)	**1.331 (1.051–1.686)**	1.148 (0.957–1.377)
01	1.139 (0.806–1.610)	0.916 (0.752–1.116)	1.086 (0.805–1.465)	0.924 (0.733–1.165)	1.037 (0.950–1.133)	**1.272 (1.023–1.581)**	1.083 (0.960–1.222)	1.015 (0.717–1.437)	0.830 (0.564–1.219)	1.038 (0.754–1.428)
02	1.231 (0.794–1.909)	0.902 (0.704–1.157)	1.124 (0.763–1.656)	0.910 (0.680–1.219)	1.055 (0.941–1.184)	**1.409 (1.063–1.868)**	1.058 (0.923–1.212)	1.062 (0.673–1.676)	0.837 (0.514–1.363)	1.092 (0.723–1.650)
03	1.343 (0.827–2.180)	0.906 (0.690–1.190)	1.159 (0.745–1.802)	0.911 (0.661–1.256)	1.073 (0.941–1.224)	**1.542 (1.118–2.127)**	1.018 (0.878–1.175)	1.141 (0.673–1.934)	0.906 (0.528–1.552)	1.175 (0.735–1.877)
04	1.477 (0.897–2.432)	0.927 (0.703–1.222)	1.189 (0.741–1.908)	0.926 (0.669–1.283)	1.091 (0.947–1.258)	**1.669 (1.183–2.352)**	0.987 (0.844–1.154)	1.257 (0.706–2.237)	1.050 (0.602–1.831)	1.291 (0.783–2.130)
05	1.639 (0.988–2.718)	0.967 (0.735–1.271)	1.215 (0.737–2.003)	0.957 (0.693–1.322)	1.109 (0.955–1.288)	**1.783 (1.242–2.559)**	0.990 (0.842–1.165)	1.420 (0.763–2.643)	1.304 (0.736–2.316)	1.450 (0.854–2.462)
06	**1.834 (1.057–3.183)**	1.028 (0.767–1.378)	1.235 (0.712–2.143)	1.005 (0.713–1.417)	1.127 (0.957–1.326)	**1.883 (1.270–2.792)**	1.038 (0.867–1.241)	1.646 (0.826–3.276)	1.735 (0.917–3.283)	1.665 (0.927–2.991)

Table 3. Relative risk with 95% CI of children's respiratory hospitalizations associated with 10 µg/m^3/1 mg/m^3 increase of air pollutants at ST1. Data from ST1, Hospital A and Hospital B. * per 1 mg/m^3 increase in daily concentration. Significant associations were highlighted in bold at $p < 0.05$. CI: Confidence interval.

function, especially in sensitive populations such as children[79,86]. The lagged association observed cumulative-lag may reflect the time required for toluene-induced respiratory symptoms, such as wheezing, coughing, or bronchial inflammation, to develop to a level requiring medical attention[87,88]. The significant associations observed for toluene, $PM_{2.5}$, and PM_{10} in Shah Alam are consistent with emissions from urban transportation, industrial solvent use, and construction-related activities[89,90]. Shah Alam's proximity to major highways and industrial estates likely intensifies pollutant exposure, particularly from vehicular VOCs and particulate matter from combustion and dust sources[91].

On the other hand, NO_2 exposure at ST4 was observed for single-lag effect at lag 4–6 with the highest RR at lag 6 of 1.124 (95% CI 1.006–1.256, $p = 0.031$), as presented in Table 5. This indicated that the delayed impact of NO_2 on respiratory health may manifest several days after exposure. NO_2 is a direct result of fuel combustion in cars, buses, and motorcycles especially diesel engines, which are more common in public transport and trucks[92,93]. This city is also a major tourist destination, increasing traffic load significantly during peak seasons[94]. Melaka has manufacturing industries[95], and shipping routes near Straits of Malacca that contribute to NO_2[84]. Urban characteristics such as high vehicular density, industrial activity, and climatic conditions in Melaka may exacerbate the health impacts of NO_2[96,97].

According to Boothe and Baldauf[98], motor vehicles release thousands of primary pollutants, particularly particulate matter, and volatile organic compounds including benzene and toluene, through fuel combustion, evaporation, brake and tire wear, as well as the degradation of engine and exhaust compounds[99,100]. Meanwhile, children living or studying near industrial areas are exposed to emissions from manufacturing processes, chemical plants, and power generation facilities, which release substantial quantities of SO_2, NOx, particulate matter, and hazardous air pollutants including benzene and other volatile organic compounds[101,102]. Industrial emissions often result in persistent ambient pollution levels due to continuous operations, contributing to long-term exposure risks[103]. Children, from infancy up to the age of 14, are especially vulnerable to health effects caused by traffic and industrial emissions due to their rapidly developing immune, neurological, and respiratory systems, and high ventilation rates relative to body mass[104,105].

Lag	Benzene	Toluene	Ethylbenzene	Xylene	$PM_{2.5}$	PM_{10}	O_3	*CO	SO_2	NO_2
	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
0	1.113 (0.816–1.517)	**1.256 (1.084–1.461)**	1.411 (0.648–3.074)	0.999 (0.921–1.084)	**1.121 (1.036–1.213)**	**1.092 (1.019–1.170)**	0.921 (0.836–1.014)	1.078 (0.896–1.297)	0.873 (0.692–1.101)	1.034 (0.883–1.211)
1	1.116 (0.889–1.402)	1.026 (0.948–1.111)	0.487 (0.256–0.925)	1.003 (0.946–1.062)	0.943 (0.889–1.001)	0.959 (0.910–1.010)	0.938 (0.869–1.012)	1.076 (0.933–1.241)	0.897 (0.749–1.073)	1.005 (0.919–1.099)
2	1.120 (0.954–1.315)	0.910 (0.811–1.020)	0.569 (0.322–1.003)	1.006 (0.970–1.044)	0.949 (0.904–0.995)	0.962 (0.922–1.004)	0.955 (0.899–1.014)	1.075 (0.963–1.198)	0.921 (0.802–1.057)	0.987 (0.902–1.080)
3	1.124 (0.984–1.284)	0.921 (0.848–1.000)	0.970 (0.441–2.130)	1.010 (0.983–1.037)	1.052 (0.986–1.122)	1.039 (0.981–1.101)	0.972 (0.923–1.025)	1.073 (0.976–1.179)	0.946 (0.840–1.065)	0.989 (0.889–1.100)
4	1.128 (0.955–1.333)	0.970 (0.864–1.087)	0.367 (0.167–0.808)	1.013 (0.976–1.051)	1.007 (0.960–1.057)	1.009 (0.967–1.052)	0.990 (0.935–1.049)	1.071 (0.962–1.193)	0.972 (0.852–1.108)	1.018 (0.931–1.113)
5	1.132 (0.894–1.434)	0.960 (0.884–1.040)	0.231 (0.094–0.569)	1.017 (0.959–1.077)	0.948 (0.895–1.004)	0.964 (0.916–1.014)	1.009 (0.939–1.083)	1.070 (0.929–1.230)	0.999 (0.843–1.182)	1.069 (0.978–1.168)
6	1.136 (0.825–1.564)	0.907 (0.778–1.058)	0.564 (0.230–1.378)	1.020 (0.940–1.106)	0.951 (0.883–1.024)	0.961 (0.901–1.025)	1.027 (0.938–1.125)	1.067 (0.890–1.281)	1.025 (0.823–1.278)	1.133 (0.967–1.328)
01	1.242 (0.727–2.123)	**1.291 (1.080–1.544)**	0.687 (0.246–1.920)	1.002 (0.872–1.151)	1.058 (0.968–1.156)	1.047 (0.968–1.133)	0.863 (0.727–1.024)	1.160 (0.837–1.608)	0.782 (0.519–1.180)	1.040 (0.821–1.317)
02	1.392 (0.702–2.757)	1.175 (0.965–1.430)	0.391 (0.108–1.409)	1.008 (0.848–1.199)	1.004 (0.909–1.108)	1.008 (0.923–1.101)	0.824 (0.656–1.035)	1.247 (0.813–1.912)	0.720 (0.420–1.236)	1.026 (0.782–1.348)
03	1.564 (0.728–3.362)	1.082 (0.880–1.330)	0.379 (0.081–1.776)	1.018 (0.844–1.228)	1.056 (0.950–1.173)	1.048 (0.954–1.150)	0.801 (0.612–1.049)	1.338 (0.814–2.198)	0.681 (0.363–1.278)	1.015 (0.745–1.382)
04	1.765 (0.791–3.939)	1.048 (0.847–1.298)	0.139 (0.022–0.884)	1.031 (0.857–1.241)	1.063 (0.951–1.189)	1.057 (0.957–1.167)	0.794 (0.588–1.072)	1.432 (0.830–2.472)	0.662 (0.332–1.320)	1.033 (0.729–1.464)
05	1.999 (0.865–4.617)	1.006 (0.802–1.260)	0.032 (0.003–0.315)	1.048 (0.877–1.252)	1.008 (0.890–1.142)	1.019 (0.912–1.138)	0.801 (0.575–1.114)	1.531 (0.847–2.770)	0.660 (0.313–1.395)	1.104 (0.760–1.605)
06	2.270 (0.894–5.762)	0.912 (0.724–1.150)	0.018 (0.002–0.218)	1.069 (0.887–1.289)	0.958 (0.843–1.090)	0.979 (0.874–1.097)	0.822 (0.569–1.190)	1.635 (0.842–3.175)	0.677 (0.296–1.551)	1.251 (0.827–1.892)

Table 4. Relative risk with 95% CI of children's respiratory hospitalizations associated with 10 $\mu g/m^3$/1 mg/m^3 increase of air pollutants at ST3. Data from ST3 with Hospital E and Hospital F. * per 1 mg/m^3 increase in daily concentration. Significant associations were highlighted in bold at $p < 0.05$. CI: Confidence interval.

As the present study is the first that conducted BTEX associations with children's hospital admissions due to respiratory diseases in Malaysia, thus the findings for benzene and toluene were discussed with previous researches performed by other countries. An early study evaluated by Oftedal et al.[106] estimated the associations between seven ambient air pollutants related to traffic emissions, including benzene and toluene, with acute hospital admissions for respiratory diseases in Norway, and the results showed that benzene had the strongest association with respiratory diseases with RR 1.095 (95% CI 1.031–1.163), followed by toluene with RR 1.044 (95% CI 1.000–1.090). This study suggested that the main sources of these pollutants in ambient air of Norway are combustion of fuel by vehicles and burning of wood for heating. Another early study in Australia and the US also reported on higher incidence risks of asthma associated with per 10-unit increase in benzene[107]. Other studies also suggested a correlation between short-term exposure to benzene and toluene on cardiorespiratory hospital admissions, particularly among high-risk population[108–110]. Many studies have demonstrated various health impacts on benzene and toluene exposures, both acute and chronic[87, 111–113]. Therefore, given the reported toxic and carcinogenic properties of these aromatic compounds, early evaluation of exposure-related risks may help reduce potential health implications that could be correlated with broader social and healthcare consequences.

For PM_{10}, this pollutant is often found to be correlated with the event of respiratory hospitalizations, primarily in the urban areas, indicated high-risk of exposure to this particle. Zahari et al.[20] conducted an assessment to determine the risks of cardiorespiratory admissions associated with ambient air pollutants in Klang Valley and result was significant for PM_{10} where 10 $\mu g/m^3$ increment in the concentration of this pollutant had linked to the 1% increase in the risk of respiratory admissions in urban settings. Another study explored the relationship between ambient air pollutants and hospital admissions due to acute respiratory infection (ARI) among children in urban region of Klang Valley indicated that PM_{10} contributed to high risk of acute bronchiolitis (odd ratio (OR): 1.115, 95% CI: 1.093–1.138), acute upper respiratory infection of multiple and unspecified sites (OR: 1.065, 95% CI: 1.034–1.096), and unspecified acute lower respiratory infection (OR: 1.055, 95% CI: 1.051–1.059) and concluded that children were mainly exposed to air pollution in urban area and susceptible to experience ARI. Added to that, Ibrahim et al.[49] reported on the association of hospital admissions due to the upper respiratory

	Benzene	Toluene	Ethylbenzene	Xylene	$PM_{2.5}$	PM_{10}	O_3	*CO	SO_2	NO_2
Lag	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
0	0.871 (0.739–1.025)	0.995 (0.882–1.121)	0.965 (0.801–1.161)	0.949 (0.782–1.152)	0.956 (0.912–1.002)	0.965 (0.928–1.004)	0.985 (0.882–1.100)	0.941 (0.649–1.364)	1.368 (0.992–1.887)	0.956 (0.855–1.068)
1	0.888 (0.786–1.003)	0.994 (0.910–1.084)	0.963 (0.838–1.106)	0.946 (0.818–1.095)	0.963 (0.930–0.998)	0.972 (0.943–1.001)	1.002 (0.921–1.090)	1.033 (0.705–1.514)	1.248 (0.986–1.579)	0.982 (0.905–1.066)
2	0.905 (0.829–0.989)	0.993 (0.935–1.054)	0.961 (0.870–1.061)	0.943 (0.847–1.049)	0.971 (0.947–0.996)	0.978 (0.957–0.999)	1.019 (0.956–1.085)	1.026 (0.758–1.389)	1.139 (0.963–1.347)	1.009 (0.952–1.069)
3	0.923 (0.858–0.993)	0.992 (0.946–1.040)	0.959 (0.886–1.038)	0.940 (0.860–1.027)	0.979 (0.959–0.999)	0.984 (0.967–1.001)	1.036 (0.981–1.093)	1.013 (0.777–1.321)	1.039 (0.896–1.205)	1.036 (0.989–1.086)
4	0.941 (0.862–1.027)	0.991 (0.933–1.052)	0.957 (0.873–1.049)	0.936 (0.844–1.039)	0.987 (0.964–1.011)	0.990 (0.970–1.010)	1.053 (0.990–1.121)	0.926 (0.678–1.265)	0.948 (0.783–1.148)	**1.065 (1.005–1.128)**
5	0.960 (0.850–1.083)	0.990 (0.908–1.080)	0.955 (0.840–1.086)	0.933 (0.810–1.076)	0.995 (0.963–1.028)	0.996 (0.969–1.025)	1.071 (0.986–1.163)	0.857 (0.591–1.241)	0.865 (0.661–1.132)	**1.094 (1.008–1.187)**
6	0.978 (0.831–1.151)	0.989 (0.878–1.114)	0.953 (0.801–1.134)	0.930 (0.769–1.124)	1.003 (0.960–1.049)	1.003 (0.965–1.041)	1.089 (0.977–1.214)	1.069 (0.741–1.541)	0.789 (0.551–1.130)	**1.124 (1.006–1.256)**
01	0.773 (0.582–1.027)	0.988 (0.804–1.210)	0.929 (0.673–1.283)	0.899 (0.641–1.261)	0.921 (0.849–1.001)	0.938 (0.876–1.006)	0.988 (0.813–1.199)	0.973 (0.654–1.447)	1.708 (0.981–2.973)	0.938 (0.774–1.138)
02	0.701 (0.485–1.008)	0.981 (0.755–1.275)	0.893 (0.590–1.353)	0.848 (0.547–1.313)	0.895 (0.805–0.995)	0.918 (0.840–1.003)	1.007 (0.782–1.296)	0.999 (0.657–1.517)	1.946 (0.961–3.942)	0.947 (0.740–1.211)
03	0.645 (0.427–0.976)	0.973 (0.727–1.301)	0.857 (0.536–1.370)	0.797 (0.484–1.312)	0.877 (0.778–0.988)	0.904 (0.817–0.999)	1.043 (0.780–1.396)	1.012 (0.659–1.553)	2.023 (0.916–4.465)	0.981 (0.745–1.293)
04	0.607 (0.391–0.942)	0.964 (0.714–1.301)	0.820 (0.500–1.346)	0.747 (0.438–1.273)	0.866 (0.763–0.983)	0.895 (0.805–0.996)	1.099 (0.801–1.509)	0.938 (0.592–1.485)	1.919 (0.828–4.444)	1.045 (0.783–1.395)
05	0.582 (0.367–0.925)	0.954 (0.703–1.295)	0.784 (0.470–1.308)	0.697 (0.396–1.226)	0.862 (0.755–0.983)	0.892 (0.799–0.997)	1.178 (0.838–1.655)	0.804 (0.495–1.304)	1.661 (0.675–4.082)	1.143 (0.848–1.542)
06	0.570 (0.342–0.950)	0.943 (0.677–1.314)	0.748 (0.431–1.299)	0.649 (0.348–1.210)	0.865 (0.750–0.998)	0.895 (0.793–1.009)	1.284 (0.879–1.875)	0.859 (0.544–1.357)	1.311 (0.465–3.699)	1.285 (0.926–1.784)

Table 5. Relative risk with 95% CI of children's respiratory hospitalizations associated with 10 µg/m^3/1 mg/m^3 increase of air pollutants at ST4. Data from ST4 and Hospital G. * per 1 mg/m^3 increase in daily concentration. Significant associations were highlighted in bold at $p < 0.05$. CI: Confident interval.

tract infection (URTI) and revealed that short-term exposure to ambient PM_{10} was significantly associated with the URTI hospitalizations among children at 1.42% (95% CI 0.00–2.86) at lag 0–4, thus proved that short-term exposure to air pollution increased the hospital admissions among vulnerable population. Meanwhile, a study carried out by Vahedian et al.[114] on the relationship between ambient air pollutants and respiratory hospital admissions in the urban population of Arak, Iran, where the results indicated that every 10 µg/m^3 increase in PM_{10} corresponded to RR = 1.01 (95% CI, 1.004–1.017) increased in respiratory disease hospitalizations. This study also found that males and the elderly were more susceptible than females and other age groups to air pollutants in regard to respiratory disease admissions. Adebayo-Ojo et al.[47] investigated the short-term association between respiratory admissions with ambient air pollution in Cape Town, Africa and PM_{10} was significantly associated at RR value of 1.9% (95% CI 0.5–3.2%) and the association was strongest among children and warm season for respiratory diseases.

Meanwhile, for $PM_{2.5}$, there are limited numbers of studies that reported on the association of this pollutant with childhood hospitalizations in Malaysia as routine and widespread monitoring of $PM_{2.5}$ in this country only became more established in recent years[115]. Ibrahim et al.[18] found a significant association of hospital admissions for respiratory disease among children aged 0–4 years with $PM_{2.5}$ at lag 0–2 where the highest RR at lag 0 with 1.040 (95% CI 1.001–1.080). The short interval of $PM_{2.5}$ interfering with the respiratory system was in line with other studies in other Asian countries[116–118]. Due to the smaller particle size, this enables the particle to penetrate deeper into the lower respiratory tract and induce lower respiratory diseases[119]. A study in Vietnam conducted by Ho et al.[120] revealed that each 10 µg/m^3 increase in $PM_{2.5}$ at lag 2 and lag 3 were associated with the excess risk of total asthma hospital admission at 2.71% and 2.63%, and children aged from two to five years had more significant effects. Another recent study reported in Thailand by Varopichetsan et al.[121] indicated that $PM_{2.5}$ concentrations > 50 µg/m^3 were significantly associated with asthma admissions at multiple time lags (lag 0 and lag 2) with odds ratios ranging from 1.41 to 1.64. Additionally, children aged 6–11 years and 12–17 years exhibited greater susceptibility to asthma hospital admissions, particularly at $PM_{2.5}$ concentrations of 15–25 µg/m^3.

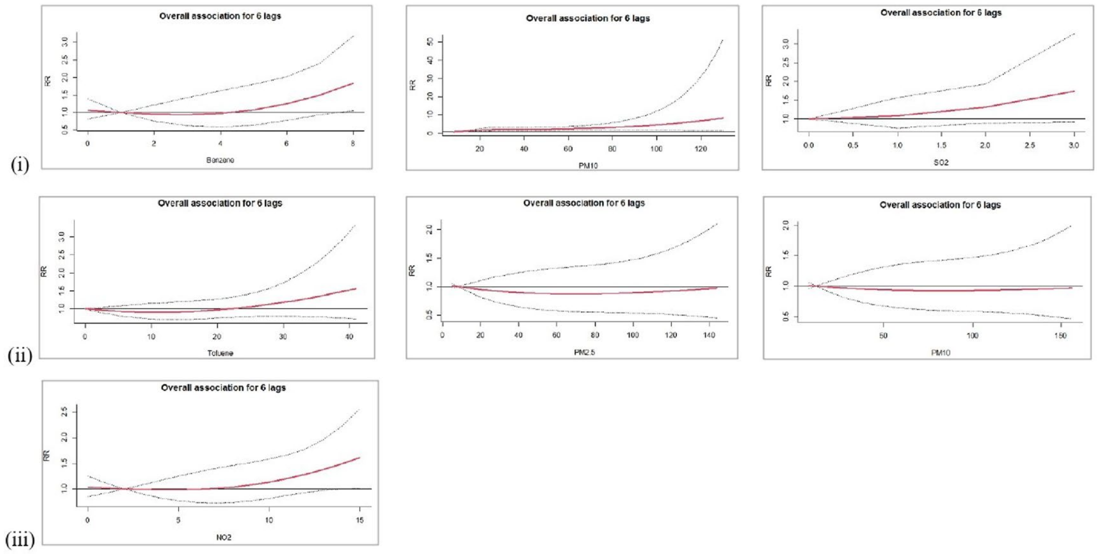

Fig. 2. Overall relative risk with 95% CI of children's respiratory hospitalizations respect to pollutants' concentration at ST1, ST3 and ST4.

Moreover, in line with the finding from the present study, Ibrahim et al.[49] also reported on the significant relationship between SO_2 exposure with the URTI hospitalizations among children in Klang Valley at lag 2 days with excess risk of 3.22% (95% CI 0.01–6.53). Another study carried out by Ibrahim et al.[18] recorded a significant single-lag effect of SO_2 for the total childhood hospital admissions due to respiratory diseases in a natural gas industrial area in Bintulu, Sarawak and the highest effect was observed at lag 4 with RR 1.029 (95% CI 1.004–1.054). This study also found that female children are susceptible to the exposure of SO_2 at lag 2 with RR value of 1.042 (95% CI 1.005–1.081), especially due to the exposure to industrial emissions. A study by Tajudin et al.[15] observed an association for SO_2 with the respiratory admissions in Bandar Tun Razak, Kuala Lumpur and the result was obtained at RR 1.025, (95% CI 1.005–1.046) at lag 4. In addition, Nascimento et al.[122] reported on a greater immediate risk of hospital care and admissions due to acute respiratory diseases of SO_2 exposure among children up to 12 years of age living in Brazilian urban area at RR value of 1.28 (95% CI 1.22–1.34). According to McAllister et al.[123], sulfur-containing fuels, particularly those used in diesel engines and power plants, are a major source of SO_2 emissions, which can lead to a decline in lung function among exposed population.

The present study also recorded a significant association between short-term exposure to NO_2 and respiratory hospitalizations among schoolchildren. This finding is consistent with existing evidence that NO_2, a key traffic-related pollutant, can induce airway inflammation, reduce lung function, and trigger respiratory symptoms, especially in children[124, 125]. Ibrahim et al.[49] reported on the excess risk of girls hospitalized for URTI that associated with NO_2 with the highest ER for every 10 μg/m^3 increase of the pollutant was 3.92% (CI 95% 1.06–6.87) at lag 0–5 days. A recent study carried out by Rahim et al.[54] revealed a significant positive association between short-term NO_2 exposure and cardiorespiratory admissions, particularly at lags of 0 and 1 days. For cardiovascular diseases, each 10 μg/m^3 increase in NO_2 was associated with a 1.07% to 9.17% increase in admissions at lag 0. For respiratory diseases, a 10 μg/m^3 increase corresponded to a 0.60% to 12.64% increase in admissions at lag 0. Another previous study recorded in urbanized area of Kuala Lumpur by Tajudin et al.[15] where delayed effects on cardiovascular and respiratory hospitalizations were found with SO_2 and NO_2. The highest RR value was observed at lag 4 for respiratory admissions with SO_2 with RR 1.123 (95% CI 1.045–1.207), followed by NO_2 at lag 5 for cardiovascular admissions with RR 1.025 (95% CI 1.005–1.046). The existing evidences together with present finding suggests that public health strategies should account for both the immediate and delayed impacts of NO_2 on vulnerable populations.

Exposure-response curves for children's respiratory hospitalizations

The findings on the fitted overall associations from this analysis are presented in Fig. 2. The relative risk (RR) is presented on the y-axis and concentration of pollutants is presented on the x-axis. The solid red line represents the estimated RR and the dotted lines above and below are the confidence intervals, indicating the range of uncertainty around the estimate.

Based on the findings, all the exposure-response curves demonstrated a non-linear trend for all stations. Referring to Fig. 2(i), the plot for benzene showed that as the concentrations of benzene increase, the relative risk (RR) of respiratory admissions rises gradually at first and then accelerates. The RR line remains close to 1 at lower concentrations, suggesting that there is little to no effect of benzene exposure on hospital admissions at these lower levels. However, as benzene levels increase beyond 4.0 μg/m^3, the RR begins to rise steadily, indicating a positive relationship between higher benzene concentrations and an increased risk of hospital admissions, demonstrating a non-linear dose-response relationship. As ST1 recorded the highest maximum value of benzene at 8.10 μg/m^3 (mean = 3.94 μg/m^3), this finding is aligns with the potential harmful health effects of benzene exposure, particularly at elevated levels, among children living in urban area[79,104].

Meanwhile, the graph for the overall association between PM_{10} concentration and hospital admissions over 6-lags displayed an initial flat region at lower PM_{10} levels, suggesting there is little observable effect of PM_{10} on hospital admissions. The graph then showed a gradual increase from the concentration of 60 μg/m^3 to 100 μg/m^3, increased the RR thus indicating the risk of hospital admissions rises slowly with increasing of PM_{10} concentrations. Next, after the concentrations exceeded 100 μg/m^3, the RR increased sharply, with a steep slope appearing in the graph. This revealed that high levels of PM_{10} are strongly associated with a dramatic increase in hospital admissions and posed health risks among children. Based on this, populations at these stations were in high-risk where the maximum concentrations of PM_{10} were recorded above 100 μg/m^3, thus pointing to the severe health risks among schoolchildren when exposed to high PM_{10} levels[126–128].

Moreover, the plot for the overall association between SO_2 concentrations and respiratory hospitalizations from Fig. 2(i), proposed that at lower concentrations, SO_2 exposure has minimal observable effect on hospital admissions due to the initial flat region observed. The RR then showed a gradual upward trend between 0.5 μg/m^3 to 2.0 μg/m^3, highlights a stronger association between SO_2 exposure and admissions at higher levels, and the RR values continued to rise sharply when SO_2 levels exceeded 2.0 μg/m^3. This indicates a positive, approximately linear exposure–response relationship, where higher cumulative SO_2 exposure over six days is associated with progressively higher risks of respiratory hospital admissions in children, highlighting SO_2 as a significant contributor to health impacts at elevated concentrations. Previous studies had reported on the high impacts of high SO_2 levels on the health outcomes, which supporting the findings from this present study[13,122, 129].

Meanwhile, as for the exposure-response relationships of pollutants at ST3 presented in Fig. 2(ii), the graph for toluene illustrates that at lower concentrations, the RR stays close to 1.0, suggesting no significant increase in risk. The RR begins to rise gradually at approximately 10 to 15 μg/m^3 and the pattern becomes clearer from around 25 μg/m^3 onwards, with RR reaching above 1.5 at around 40 μg/m^3. The gradual curve shape of the graph implies the health risk becomes more pronounced at moderate to high toluene levels, aligning with its known respiratory irritant properties, which can substantially increase the risk of respiratory hospital admissions in children[86,130,131].

On the contrary, the exposure–response curves for $PM_{2.5}$ and PM_{10} in this study show relatively flat patterns at low to moderate concentration ranges, with the RR values hovering around or slightly below 1.0. This suggests that within these lower concentration ranges, there is no strong or consistent evidence of an increased risk of respiratory hospitalizations among children. In fact, the slight dip below 1.0 may reflect a neutral or even reduced risk, which could be attributed to factors such as children's adaptive physiological responses to background pollutant levels or the influence of confounding variables not fully captured in the model. However, it is important to note that at higher concentration levels, the curves begin to show an upward trend at concentration of 140 μg/m^3 ($PM_{2.5}$) and 150 μg/m^3 (PM_{10}), indicating increased risk. This pattern aligns with previous findings where health effects associated with PM exposure are more pronounced beyond specific threshold concentrations, emphasizing the non-linear nature of the exposure–response relationship for $PM_{2.5}$ and PM_{10}[132–134].

In addition, the overall association between NO_2 exposure and respiratory hospitalizations among schoolchildren at ST4 presented in Fig. 2(iii) also demonstrates a non-linear positive association. Based on the graph, the relative risk (RR) remains close to 1.0 at lower NO_2 concentrations (approximately 0–5 μg/m^3), indicating a minimal or neutral effect at background exposure levels. However, beyond 5 μg/m^3, the RR curve begins to gradually rise, and after approximately 10 μg/m^3, the increase becomes more pronounced, suggesting a stronger association between NO_2 exposure and hospitalization risk. This rising trend reflects the cumulative and potentially delayed impact of NO_2 exposure on respiratory health. NO_2 is a known TRAP and respiratory irritant that can cause airway inflammation, oxidative stress, and increased susceptibility to respiratory infections, especially in children with developing lungs[25,125,135,136]. These findings are consistent with studies showing delayed onset of respiratory symptoms and increased hospital admissions following short-term exposure to elevated NO_2 levels[13,116,137,138].

Sensitivity analysis

For sensitivity analysis, the model was tested with varying *df* at 6, 8 and 10 *df*/year for time, and 3, 4 and 5 *df* for meteorological parameters. The results of the sensitivity analysis revealed that the use of different temperature and humidity lags did not significantly impact the associations between air pollutant levels and hospital admissions for respiratory diseases. However, these associations were affected by changes in the *df* when accounting for long-term trends for ST1 for benzene and PM_{10} (Appendix C). This revealed how sensitive the results are to different model specifications. Essentially, it suggests that the choice of *df* when modeling long-term trends can affect the observed relationships between variables. This method for conducting sensitivity analysis has been proven reliable by previous studies[12,18,,139].

Conclusions

This study provides robust evidence that short-term exposure to ambient air pollutants significantly increases respiratory hospitalizations among schoolchildren in urbanized areas of Peninsular Malaysia. By applying a time-series modeling approach, the study not only confirms the health risks of criteria pollutants (SO_2, NO_2, PM_{10}, $PM_{2.5}$) but also introQ4duces novel insights into the impacts of BTEX compounds, particularly benzene and toluene, which remain underrepresented in air quality research and policy frameworks. These findings highlight a critical gap in existing air quality standards and call for regulatory updates that explicitly consider BTEX exposure as part of child health protection strategies.

Importantly, this research extends beyond the well-studied Klang Valley to highlight significant risks in other urban centres such as Penang and Malacca, emphasizing the need for geographically inclusive monitoring and interventions. The observed variations across regions reflect the interplay of emission sources, meteorology, and population vulnerability, reinforcing the importance of adaptive, region-specific policies and stronger inter-agency collaboration to manage urban air quality.

A key limitation is the use of fixed-site ambient air monitoring data as a proxy for population exposure, which may not fully capture spatial variability or individual-level exposures within hospital catchment areas. Although this approach is widely used in time-series studies, some exposure measurement error is unavoidable. In addition, hospital admission data may not reflect less severe respiratory outcomes. Nevertheless, the study provides critical empirical evidence on pollutant-specific risks in children, offering direct implications for public health policy. Future research should integrate refined exposure assessments, such as spatial modeling or personal monitoring, to strengthen exposure characterization and interpretation of health associations. Collectively, this study advances the evidence base for child-centred air quality management and emphasizes the urgency of emission reduction policies to safeguard the health of future generations.

Data availability

The datasets generated during and/or analysed during the current study are not publicly available due to privacy and third-party restrictions.

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Declarations

Competing interests
The authors declare no competing interests.

Ethics approval
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