ORIGINAL ARTICLE

Long-term effects of ambient particulate matter on hypertension among royal Thai army officers: a retrospective cohort study

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ABSTRACT

Recent research on the link between exposure to ambient particulate matter with an aerodynamic diameter up to 2.5 microns ($PM_{2.5}$) and hypertension risk has primarily concentrated on spatial variation (or between-individual comparisons). Research into temporal variations (or within-individual comparisons over time) has largely been neglected. This study sought to examine the spatial–temporal effects of long-term exposure to $PM_{2.5}$ on hypertension risk among military personnel in Thailand. A retrospective cohort study was conducted, encompassing 40,984 Royal Thai Army officers from 400 army units stationed across 51 provinces in Thailand. Medical check-up data from 2018 to 2021 were analyzed alongside ambient $PM_{2.5}$ data from 2015 to 2017, sourced from the Geo-Informatics and Space Technology Development Agency. Two parameters, $PM_{2.5}$-baseline and $PM_{2.5}$-change, were introduced to assess the impact of spatial and temporal $PM_{2.5}$ variations on hypertension incidence. Cox proportional hazard regression was used, with hazard ratios (HR) with 95% confidence intervals (95% CI) serving as the measure of association. The association between $PM_{2.5}$-baseline and hypertension incidence yielded hazard ratios in Quartiles 2 to 4 compared to Quartile 1 of: Q2 HR: 1.19, 95% CI: 1.10–1.28; Q3 HR: 1.10, 95% CI: 1.02–1.20; Q4 HR: 1.13, 95% CI: 1.05–1.22. Additionally, the $PM_{2.5}$-change showed a J-shaped association with hypertension incidence. Our findings underscore the role of both temporal and spatial factors in hypertension development and highlight the necessity for comprehensive investigations into the causal relationship between $PM_{2.5}$ exposure and hypertension risk. They also provide valuable insights for devising effective strategies to mitigate the adverse health impacts of $PM_{2.5}$ pollution.

Key words:
ambient particulate matter; hypertension; cohort study

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INTRODUCTION

Air pollution is a global health concern caused by urbanization and industrialization increasing emissions of harmful substances into the air. A major air pollutant is particulate matter with an aerodynamic diameter of less than or equal to 2.5 micrometers $PM_{2.5}$.[1] Studies have demonstrated a significant association between $PM_{2.5}$ and hypertension.[2] Hypertension is a major global health issue due to its impact on individuals and healthcare systems.[3] The hypothesis that $PM_{2.5}$ can elevate the risk of hypertension is plausible. The likely underlying processes involve systemic inflammation and oxidative stress in the blood vessels after PM inhalation deep into the alveoli[4] and absorption into the blood stream.[5] These mechanisms collectively contribute to blood vessel narrowing and subsequent hypertension.[6]

Extensive research has been conducted to investigate the association between $PM_{2.5}$ and hypertension.[7-12] Most studies primarily concentrate on developed countries.[13-14] These findings have contributed to raising awareness and enabled several developed countries to consistently reduce $PM_{2.5}$ emissions, in contrast to developing countries where there is a trend toward increasing $PM_{2.5}$ levels.[15] In Thailand, a few studies have linked ambient $PM_{2.5}$ exposure to adverse health effects such as hospital admission for respiratory and cardiovascular diseases[16] and an increased incidence of diabetes.[17] However, only one cross-sectional study exists for hypertension.[18] All the studies have focused on spatial relations (calculating the average $PM_{2.5}$ concentration for each geographical area and comparing them) as the primary approach in assessing the health impact of air pollution. There has been limited emphasis on temporal relationships, that is, calculating the annual deviation in $PM_{2.5}$ concentrations from the area averages for each unit of time and comparing within individuals during follow-up).[19]

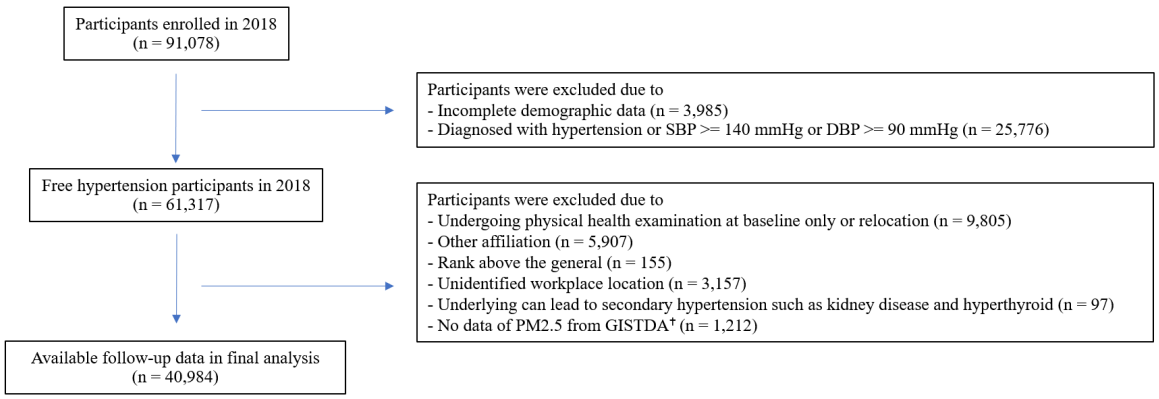
Differentiating between the spatial and temporal components of air pollution exposure and their association with health effects can have practical implications for environmental and medical science. First, differentiating between the impact of spatial versus temporal variations in $PM_{2.5}$ exposure may provide deeper insights into the principal sources (stationary versus mobile) of ambient $PM_{2.5}$ and the factors influencing hypertension risk (stable, area-specific contextual factors versus more labile meteorological factors), thereby providing information for more relevant $PM_{2.5}$ control strategies.[19] Second, as spatial and temporal variation tend to capture the effects of longer- and shorter-term exposure respectively,[20] understanding their relative impacts may direct detailed investigations into the pathophysiological mechanism of $PM_{2.5}$ exposure and subsequent hypertension.

This study, therefore, sought to examine the spatial–temporal effects of long-term exposure to $PM_{2.5}$ on hypertension risk among military personnel in Thailand. Specifically: (a) to determine the association of the spatial variation in $PM_{2.5}$ exposure with subsequent hypertension incidence by comparing baseline $PM_{2.5}$ exposure between individuals working in different army units/subdistricts from 2018 to 2021; and (b) to determine the association of temporal variation in $PM_{2.5}$ exposure with hypertension, i.e., by comparing the deviation of $PM_{2.5}$ exposure level from the baseline with hypertension incidence within individuals working in a particular army unit/subdistrict, over each follow-up year from the same time period mentioned in (a).

METHODS

Study design

This retrospective cohort study used data from the medical examinations database of the Royal Thai Army spanning from 2018 to 2021. The study included officers stationed in 51 of the 77 provinces in Thailand. In 2018, a total of 91,078 participants underwent health examinations, and 25,776 were diagnosed with hypertension.[21] Individuals with incomplete demographic data records (3,985), those from other affiliations (5,907), and individuals holding a military rank of general or higher (155), were excluded. We also excluded individuals with underlying conditions related to secondary hypertension (97), unclear workplace information (3,157), missing $PM_{2.5}$ data (1,212), and those who only underwent baseline physical examinations or relocated (9,805). Ultimately, the study included 40,984 individuals who met the participation criteria. (Figure 1)



†GISTDA: Geo-Informatics and Space Technology Development Agency

Figure 1. Flow chart of the study population selection

We determined the required sample size for this study using the sample size formula for hypothesis tests comparing two incidence rates in a follow-up study,[22] with Type I error = 0.05, statistical power = 80%, follow-up time = 3 years, and the presumptive hypertension incidence rates of 47.8 and 33.3 per 1000 person-years for individuals in Quartile I and IV $PM_{2.5}$ exposure levels respectively.[23] The estimated number of participants needed was 5,500; therefore, our sample size was adequate to ensure robust statistical power for addressing the study research questions.

The follow-up duration was reported in person-years, calculated from the date of study enrollment to the date of the last interview, death, or incidence of hypertension. The diagnosis of hypertension was considered as the outcome event, while other conditions were treated as censored data. The follow-up rates after 1, 2, and 3 years were 22.43%, 50.70%, and 26.87%, respectively.

Ethics approval

Ethical approval was granted by the institutional review boards of the Faculty of Medicine, Chulalongkorn University (COA No. 0170/2023) and the Royal Thai Army Medical Department (IRBRTA.No. 0513/2023).

Assessment of hypertension

As part of the Royal Thai Army health examinations, blood pressure measurements were taken by a physician with a sphygmomanometer. Two measurements were made with a rest period of at least five minutes between them and the average measurement was used for analysis. Hypertension was defined according to guidelines, as a systolic blood

pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg, or self-reported previous diagnosis of hypertension made by a physician.[21]

Assessment of Air pollution exposure

The method has been described in our previous study.[17] We used $PM_{2.5}$ concentration data from the Geo-Informatics and Space Technology Development Agency,[24] obtained through Moderate Resolution Imaging and Multi-angle Imaging Spectro-Radiometers. We then created a geographically weighted regression model for assessing ground-level $PM_{2.5}$ concentrations at a 3 × 3 km resolution, which was extrapolated to subdistrict levels. These estimates were then linked to the geographic locations of military units nationwide.

We constructed two parameters to portray long-term $PM_{2.5}$ exposure. **First**, '$PM_{2.5}$-baseline'was defined as the baseline exposure using the average of $PM_{2.5}$ concentration from 2015 to 2017, representing the three years preceding the survey. This parameter was used to examine the impact of the spatial difference in baseline $PM_{2.5}$ exposure level on hypertension incidence. **Second**, '$PM_{2.5}$-change' was calculated by measuring the disparity between the lag 1–3-year average $PM_{2.5}$ level before the follow-up visit and the '$PM_{2.5}$-baseline.' This parameter was used to assess the impact of the temporal change in $PM_{2.5}$ exposure level on hypertension incidence.

The data for provincial wind speed, temperature, relative humidity and rainfall were obtained from the Air4Thai system.[25] Furthermore, the Gross Provincial Product data were obtained from the Office of the National Economic and Social Development Council.[26]

Covariates

Covariates, including age, gender, height, weight, smoking status, alcohol consumption, and physical activity[27] were collected from the Royal Thai Army medical examination data. The category "not specified" was used for missing survey data. Obesity was defined as having a body mass index (BMI) ≥25 kg/m^2 according to the Asia-Pacific guidelines.[28]

Statistical analysis

Descriptive analysis was performed to summarize the variables. Continuous variables are presented as mean (± standard deviation; SD) or median (interquartile range), while categorical variables are reported as numbers and percentages. The high blood pressure incidence rate was calculated by dividing the number of new hypertension cases by the person-years of follow-up. Hazard ratio analysis was performed using Cox proportional hazard regression. Individual factors included age, gender, BMI, blood pressure, smoking status, alcohol consumption, current exercise status, diabetes, and dyslipidemia. Area factors included $PM_{2.5}$ levels, relative humidity, wind speed, temperature, and rainfall.

We assessed the effects of spatial variation using '$PM_{2.5}$-baseline' as the exposure variable, considering potential variables and calculating the 95% confidence intervals (CIs). We examined the impact of temporal variation using the '$PM_{2.5}$-change' as the second exposure variable. $PM_{2.5}$-baseline was divided into quartiles, with Quartile 1 as the reference category. As $PM_{2.5}$-change includes reductions and increases, a temporal change of 0 μg/m^3 was set as the reference, representing a no change scenario. Then, we considered it in two separate directions to facilitate interpretation. The potential for non-linearity of the exposure-outcome association was addressed by incorporating the fractional polynomials into the Cox

proportional hazard regression[29]. The crude model included only the '$PM_{2.5}$-baseline' variable. **Adjusted Model 1** incorporated individual variables, such as age, gender, BMI, blood pressure, smoking status, alcohol consumption, current exercise status, diabetes, and dyslipidemia. **Adjusted Model 2** additionally incorporated the area variables, including relative humidity, wind speed, temperature, and rainfall. **Adjusted Model 3** further included the '$PM_{2.5}$-change' variable. Before including the variables in the equations, potential collinearity was solved by excluding any correlation among the independent variables exceeding 70%.[30] Consequently, temperature and Gross Provincial Product were removed from the model.

The analyses were performed using STATA Statistical Software, version 17.0 (StataCorp LLC, College Station, TX). A two-sided *p*-value of less than 0.05 was considered statistically significant.

RESULTS

$PM_{2.5}$ exposure

The average $PM_{2.5}$ levels during the baseline period 2015–2017, referred to as '$PM_{2.5}$-baseline' exposure, ranged from 14.64 to 25.15 μg/m^3. The mean (SD) of $PM_{2.5}$-baseline was 20.79 (2.73) μg/m^3. The $PM_{2.5}$-baseline was categorized into four quartiles. Quartiles 1, 2, 3 and 4 comprised $PM_{2.5}$-baseline values of less than 19.09, between 19.09 and 21.20, between 21.21 and 22.57, and over 22.57, respectively. During the follow-up period, the mean (SD) of the '$PM_{2.5}$-change' was −0.49 (0.88) μg/m^3 and maximum and minimum values were 4.59 μg/m^3 and −3.80 μg/m^3, respectively.

Participant characteristics

The average age of the participants was 37.09 years, with 88.86% male. The average BMI at baseline was 24.22 kg/m^2. The participants were divided into quartiles according to the '$PM_{2.5}$-baseline.' Significant group differences were observed for age, gender, BMI, smoking status, alcohol consumption, and exercise levels (Table 1). These differences were considered in the analysis of $PM_{2.5}$ exposure and hypertension association.

Table 1. Demographic characteristics of the participants at baseline (n = 40,984)

Characteristics	$PM_{2.5}$-baseline† (μg/m^3)			
	Quartile 1 (<19.09)	**Quartile 2** (19.09–21.20)	**Quartile 3** (21.21–22.57)	**Quartile 4** (>22.57)
Age				
Early working age (18–29 yrs)	4121 (38.77)	3374 (32.16) [a]	3360 (32.60) [a,b]	2136 (22.35) [a,b,c]
Middle working age (30–44 yrs)	4432 (41.70)	4205 (40.08)	4258 (41.31)	3771 (39.46)
Late working age (45–60 yrs)	2076 (19.53)	2913 (27.76)	2689 (26.09)	3649 (38.19)
Mean ± SD	37.09 ± 10.96			
Gender				
Male	10122 (95.23)	9570 (91.21) [a]	9301 (90.24) [a,b]	7427 (77.72) [a,b,c]
Female	507 (4.77)	922 (8.79)	1006 (9.76)	2129 (22.28)
BMI				
Non-obese	6975 (65.62)	6706 (63.92) [a]	6656 (64.58)	5897 (61.71) [a,b,c]

Characteristics	$PM_{2.5}$-baseline† (μg/m³)			
	Quartile 1 (<19.09)	**Quartile 2** (19.09–21.20)	**Quartile 3** (21.21–22.57)	**Quartile 4** (>22.57)
Obese	3654 (34.38)	3786 (36.08)	3651 (35.42)	3659 (38.29)
Mean ± SD	24.22 ± 3.54			
Smoking				
Non-current smoker	6912 (65.03)	7558 (72.04) [a]	6934 (67.27) [a,b]	6975 (72.99) [a,b,c]
Current smoker	3686 (34.68)	2835 (27.02)	2736 (26.55)	2517 (26.34)
Not specified	31 (0.29)	99 (0.94)	637 (6.18)	64 (0.67)
Alcohol				
Non-current drinker	4492 (42.26)	3482 (33.19) [a]	3013 (29.23) [a,b]	3485 (36.47) [a,b,c]
Current drinker	6095 (57.34)	6910 (65.86)	6658 (64.60)	6040 (63.21)
Not specified	42 (0.40)	100 (0.95)	636 (6.17)	31 (0.32)
Exercise				
Insufficient exercise	4360 (41.02)	5100 (48.61) [a]	3060 (29.69) [a,b]	3062 (32.04) [a,b,c]
Sufficient exercise	6175 (58.10)	5180 (49.37)	6605 (64.08)	5820 (60.91)
Not specified	94 (0.88)	212 (2.02)	642 (6.23)	674 (7.05)
Diabetes mellitus				
Yes	474 (4.46)	957 (9.12) [a]	771 (7.48) [a,b]	715 (7.48) [a,b]
No	10155 (95.54)	9535 (90.88)	9536 (92.52)	8841 (92.52)
Dyslipidemia				
Yes	3426 (32.23)	3874 (36.92) [a]	3586 (34.79) [a,b]	4478 (46.86) [a,b,c]
No	7208 (67.77)	6616 (63.08)	6720 (65.21)	5076 (53.14)

Note: † Average concentration of $PM_{2.5}$ from 2015 to 2017, a: significant difference from Q1 at p-value <0.05, b: significant difference from Q2 at p-value <0.05 and c: significant difference from Q3 at p-value <0.05.

Table 2. The incidence rate of hypertension according to $PM_{2.5}$-baseline† quartiles

$PM_{2.5}$-baseline†	Person - years	New case	Incidence rate (per 1000)
Quartile 1 (<19.09μg/m³)	35059	3427	97.74
Quartile 2 (19.09–21.20μg/m³)	34162	3513	102.83
Quartile 3 (21.21–22.57μg/m³)	33703	3059	90.76
Quartile 4 (>22.57μg/m³)	31611	3008	95.15
Total	**134535**	**13007**	**96.68**

Note: † Average concentration of $PM_{2.5}$ from 2015 to 2017.

Table 3. Hazard ratios for the association between long-term exposure to $PM_{2.5}$ and hypertension incidence

	Crude model			Adjusted Model 1			Adjusted Model 2			Adjusted Model 3		
$PM_{2.5}$ level ($\mu g/m^3$)	HR	95 % CI	*p*-value	HR	95 % CI	*p*-value	HR	95 % CI	*p*-value	HR	95 % CI	*p*-value
The spatial relation using $PM_{2.5}$-baseline												
Quartile 1	Reference			Reference			Reference			Reference		
Quartile 2	1.23	1.16, 1.31	<0.001	1.23	1.16, 1.30	<0.001	1.19	1.10, 1.28	<0.001	1.89	1.68, 2.13	<0.001
Quartile 3	1.15	1.08, 1.23	<0.001	1.16	1.08, 1.24	<0.001	1.10	1.02, 1.20	<0.001	1.28	1.13, 1.46	<0.001
Quartile 4	1.23	1.15, 1.32	<0.001	1.24	1.15, 1.33	<0.001	1.13	1.05, 1.22	<0.001	1.41	1.25, 1.59	<0.001

Note: HR = Hazard ratio, CI = Confidence interval, Crude model: Cox proportional hazard regression for the sampling sites and only included $PM_{2.5}$-baseline, Adjusted Model 1: Cox proportional hazard regression for the sampling sites further adjusted for Crude Model, age, gender, BMI, blood pressure, smoking status, alcohol consumption, exercising at the present, diabetes, and dyslipidemia, Adjusted Model 2: Cox proportional hazard regression for the sampling sites further adjusted for Adjusted Model 1, relative humidity, wind speed, rainfall and Adjusted Model 3: Cox proportional hazard regression for the sampling sites further adjusted for Adjusted Model 2 and $PM_{2.5}$-changed

$PM_{2.5}$ exposure and hypertension association

The hypertension incidence in Quartiles 1,2,3, and 4 were 97.74, 102.83, 90.76, and 95.15 per 1000 person-years, respectively, as presented in Table 2. The association between $PM_{2.5}$ exposure and hypertension was assessed using Cox proportional hazard regression (Table 3). For the inter-individual level comparison using $PM_{2.5}$ -baseline as the exposure variable (Adjusted Model 2), there was a statistically significant increase in the hazard ratio in Quartiles 2 to 4 compared to Quartile 1 (Q2 HR: 1.19, 95% CI: 1.10–1.28; Q3 HR: 1.10, 95% CI: 1.02–1.20; Q4 HR: 1.13, 95% CI: 1.05–1.22, respectively).

When the variable '$PM_{2.5}$-change,' was added (Adjusted Model 3), a significant increase in the hazard ratio of $PM_{2.5}$-baseline was observed in Quartiles 2 to 4 compared to Quartile 1 (Q2 HR: 1.89, 95% CI: 1.68–2.13; Q3 HR: 1.28, 95% CI: 1.13–1.46; Q4 HR: 1.41, 95% CI: 1.25–1.59, respectively). Further analysis based on the continuous term of $PM_{2.5}$-change revealed its J-shaped association with hypertension incidence when analyzed using Adjusted Model 3. (Figure 2)

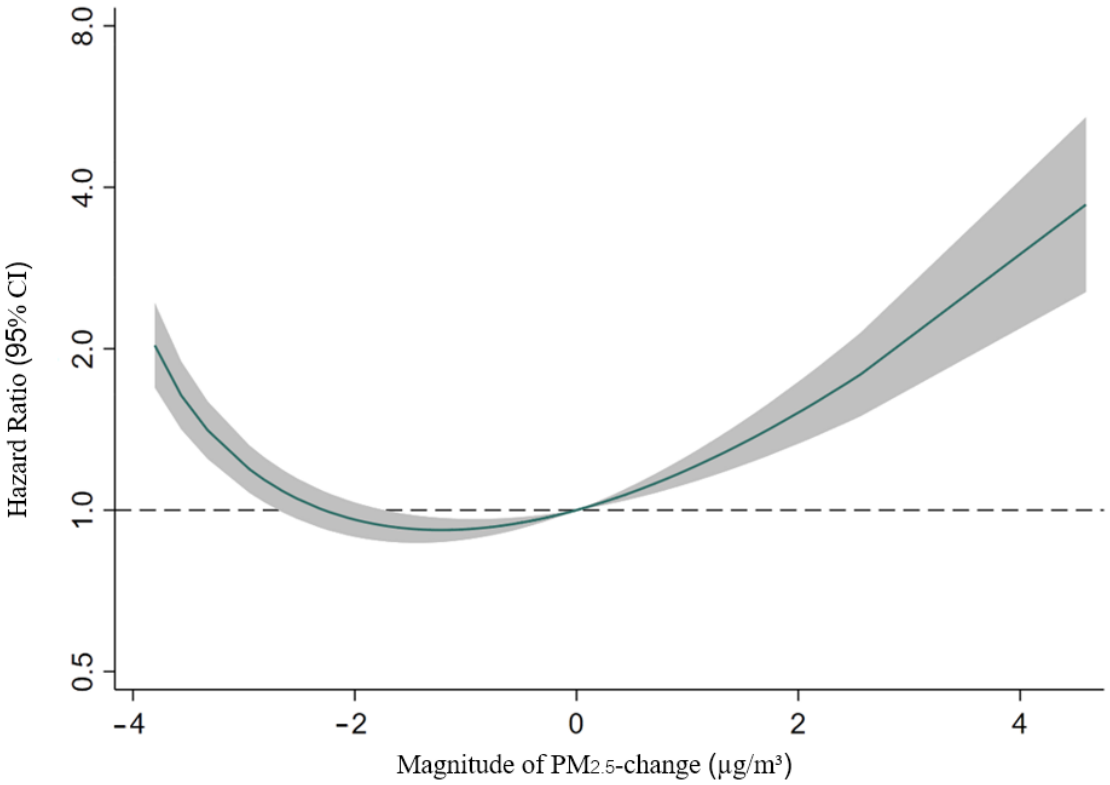


Figure 2. Hazard ratios for the association between $PM_{2.5}$-change and hypertension incidence The middle category ($PM_{2.5}$-change = 0.00 µg/m^3) was designated as the reference.

In the subgroup analyses of potential confounding factors using Adjusted Model 3, the associations of $PM_{2.5}$-baseline with hypertension were stronger among men (HR: 1.34, 95% CI: 1.17–1.53), individuals of early working age (HR: 1.63, 95% CI: 1.26–2.09), individuals doing insufficient exercise (HR: 1.86, 95% CI: 1.48–2.32), and non-diabetic (HR: 1.29, 95% CI: 1.13–1.48) and non-dyslipidemic participants (HR: 1.42, 95% CI: 1.20–1.68), compared with their respective counterparts. (Figure 3)

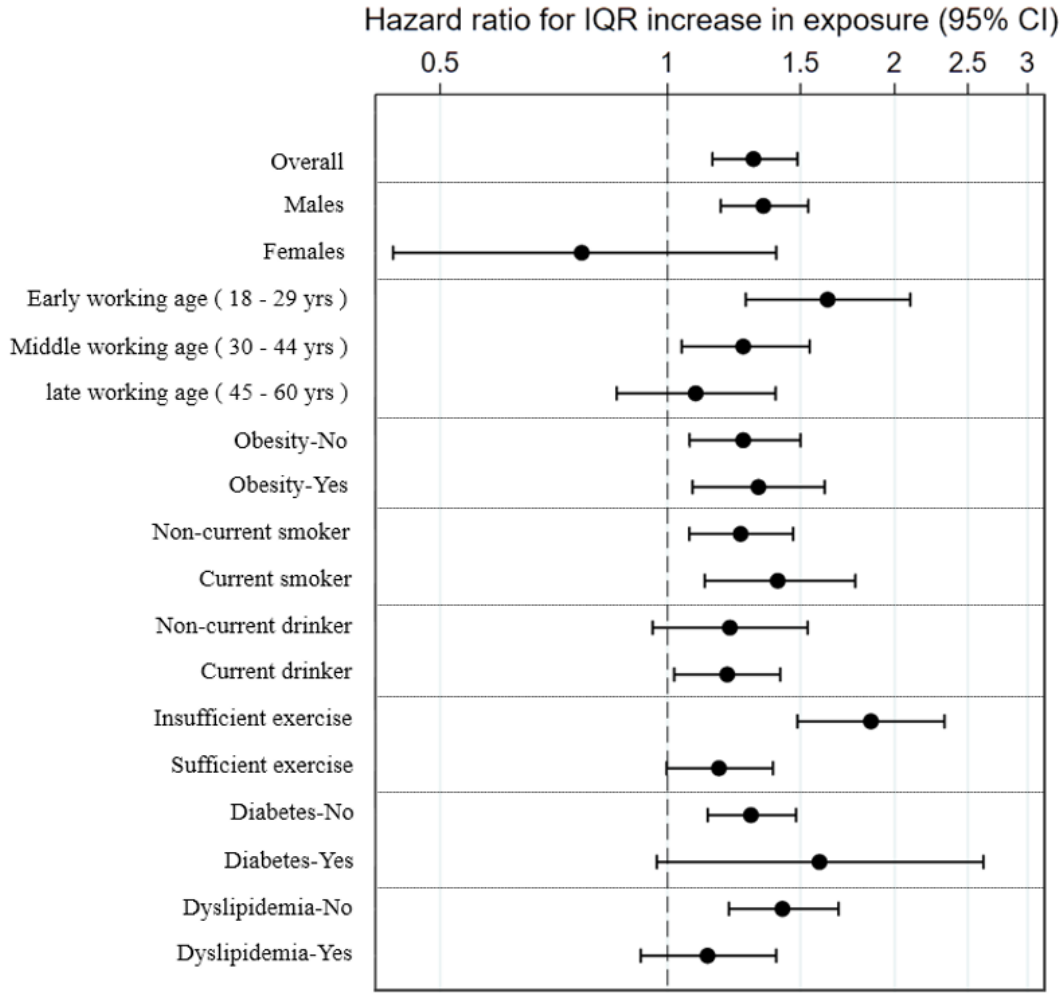


Figure 3. Subgroup analysis of the association of long-term exposure to $PM_{2.5}$ with hypertension risk. Hazard ratio is shown by closed circles, with whiskers representing the 95% confidence interval.

DISCUSSION

This 3-year retrospective cohort study aimed to investigate the association between long-term exposure to $PM_{2.5}$ and the incidence of hypertension among Thai military officers. This is the first study to concurrently evaluate the extended impact of ambient $PM_{2.5}$ on hypertension in Thailand. Our findings reveal a positive correlation between ambient $PM_{2.5}$ exposure and risk of hypertension when comparing each quartile with the reference quartile.

The positive associations closely align with other investigations, such as the study conducted by Viyey et al. (HR = 1.48, 95% CI: 1.09–2.00),[31] the study by Zhang et al. (HR = 1.04, 95% CI: 1.00–1.07),[12] and Prabhakaran et al. (HR = 1.16, 95% CI: 0.95–1.43); this also showed a similar non-linear pattern in hazard ratios within Quartiles 2–4 when utilizing a 2-year average $PM_{2.5}$ measurement.[32]

It is important to acknowledge the challenges associated with accurately identifying hypertension. Variations in clinical practice between study sites can contribute to these challenges. Moreover, even small increases in blood pressure, which might not lead to overt hypertension can be clinically significant. Such minor elevations could impact individuals with

baseline blood pressure levels close to abnormal values, putting them at risk of developing hypertension.[33] Song et al.[2] observed a concentration-response relationship between long-term exposure to $PM_{2.5}$ particulate matter and hypertension prevalence. The study reported ORs of 1.02 (95% CI: 1.01–1.04), 1.01 (95% CI: 1.00–1.02), and 1.00 (95% CI: 1.00–1.00) for $PM_{2.5}$ levels of 15, 25, and 35 $\mu g/m^3$, respectively. Notably, the ORs increased more steeply when $PM_{2.5}$ concentrations exceeded 50 $\mu g/m^3$. It is important to note that the $PM_{2.5}$ levels in our study ranged from 14.64 to 25.15 $\mu g/m^3$. However, further research is necessary to confirm these results.

We also found that the temporal change in the $PM_{2.5}$-change exposure was significantly associated with hypertension incidence. The strong impact of temporal change in $PM_{2.5}$ exposure on hypertension suggests that the effect may be more prominent with shorter-term exposure than with cumulative extended exposure. This was supported by a study conducted by Prabhakaran et al., which focused on urban adults in Delhi, India. The average exposure over a two-year period before blood pressure measurements did not show statistically significant results (HA = 1.16, 95% CI: 0.95–1.43). However, when compared with exposure periods of one year (HA = 1.53, 95% CI: 1.19–1.96) and 1.5 years (HA = 1.59, 95% CI: 1.31–1.92), statistically significant associations with hypertension were observed.[32] Additionally, Zhou's study demonstrated that the further the average $PM_{2.5}$ levels were from the year of the event or last interview, the lower the hazard ratio.[34] However, these two studies did not clearly disentangle the impact of temporal changes in $PM_{2.5}$ exposure from overall $PM_{2.5}$ exposure.

Our findings further support the impact of temporal variation in $PM_{2.5}$ exposure on hypertension risk (Figure 2). In addition, the decrease in hypertension risk with the temporal decrease in $PM_{2.5}$ exposure implies that reducing exposure to $PM_{2.5}$ over an extended period may reduce hypertension risk. Focusing on the temporal rather than the spatial variation in $PM_{2.5}$ exposure may provide better insights into its etiological mechanism and identify effective interventions to mitigate its harmful health effects. More studies are needed to confirm this finding.

The subgroup analysis showed that the association between $PM_{2.5}$ exposure and hypertension was stronger among men, consistent with findings from other studies.[2,31] This discrepancy could be attributed to the differing duties of male and female officers. Female personnel often hold administrative or medical positions with less outdoor exposure and operational activity than their male counterparts. Furthermore, individuals in the early working age group faced the highest risk, followed by the middle and late working age groups. This trend aligns with the findings of the study by Viyey et al., especially among individuals under 60 years of age.[31] This may be because younger, lower ranking military personnel often participate in more demanding operational and field tasks, with irregular shifts and duties in critical locations. This study also highlighted that insufficient exercise was associated with an elevated risk of hypertension, consistent with Guo et al's findings.[35] This can be attributed to the well-known benefits of regular exercise in mitigating the risk of hypertension by reducing cardiac output, plasma norepinephrine levels, and sympathetic nervous system activity.[36]

However, interestingly, the present study found non-diabetic or dyslipidemic individuals had a higher risk of developing hypertension, which contradicts the findings of Jiali et al.[2] This may be

attributed to the frequency of medical attention mandated by military regulations,[37] which require yearly medical check-ups. Individuals with health risks or illnesses, have scheduled physician appointments and are monitored as needed, resulting in better health awareness. This aligns with the findings by Suka et al[38]., who showed that individuals with ample health information from various sources are less prone to engage in risky behaviors such as smoking, drinking, and inadequate exercise. However, further investigation is necessary to better understand this relationship.

This study had some limitations. First, the estimation of the participants' $PM_{2.5}$ exposure levels was limited to officers' workplace locations where they typically spend only 33% of their daily time.[39] Second, the weather data used for our analysis was only available at the provincial level, whereas the $PM_{2.5}$ data required was at subdistrict level, which may have greater relevance to the participants in our study. However, in other studies, weather data was not extensively addressed and should be explored in future research. Furthermore, the outbreak of COVID-19 in 2019 resulted in a significant decrease in annual medical check-up participation. Medical check-up data could not be retrieved beyond the year 2018 due to an ongoing transition to a centralized survey-based system to facilitate data collection and storage. However, the sample size had more than sufficient statistical power to meet the study objectives.

In conclusion, our study demonstrated a significant impact of both spatial and temporal variation in $PM_{2.5}$ exposure on hypertension incidence. This observation offers a more nuanced understanding for future investigation of the relationship between $PM_{2.5}$ and hypertension and the formulation of more targeted interventions to mitigate the adverse health effects of $PM_{2.5}$. Nevertheless, further research is required to validate these findings.

RECOMMENDATIONS

Future research should aim to enhance $PM_{2.5}$ exposure assessment by incorporating diverse settings and finer-grained weather data. Longitudinal health monitoring will distinguish dynamic health effects from external factors like the COVID-19 pandemic. A more diverse sample representation, multivariate confounder analysis, and cross-comparisons of spatial variation and temporal variation exposure effects would deepen understanding. The significant effects of temporal variation can serve as an important indicator of time-varying factors when designing interventions to enhance public health.

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DISCLOSURE

Approval of the research protocol: This study was approved by the institutional review board of the Faculty of Medicine, Chulalongkorn University (COA No. 0170/2023) and the institutional review board, Royal Thai Army Medical Department (IRBRTA.No. 0513/2023).

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