Original Article

J Prev Med Public Health 2023;56:449-457 • https://doi.org/10.3961/jpmph.23.292

pISSN 1975-8375 eISSN 2233-4521

Journal of Preventive Medicine & Public Health

# The Association Between $PM_{2.5}$ Exposure and Diabetes Mellitus Among Thai Army Personnel

**Apisorn Laorattapong[1,2], Sarun Poobunjirdkul[1,2], Thanapoom Rattananupong[2], Wiroj Jiamjarasrangsi[2]**

*[1]Division of Occupational Medicine, Department of Outpatient Service, Phramongkutklao Hospital, Bangkok, Thailand; [2]Department of Preventive and Social Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand*

**Objectives:** This study investigated the association between baseline exposures to particulate matter with a diameter <2.5 microns ($PM_{2.5}$) and subsequent temporal changes in $PM_{2.5}$ exposure with the incidence of type 2 diabetes among Royal Thai Army personnel.

**Methods:** A retrospective cohort study was conducted using nationwide health check-up data from 21 325 Thai Army personnel between 2018 and 2021. Multilevel mixed-effects parametric survival statistics were utilized to analyze the relationship between baseline (i.e., $PM_{2.5}$-baseline) and subsequent changes (i.e., $PM_{2.5}$-change) in $PM_{2.5}$ exposure and the occurrence of type 2 diabetes. Hazard ratios (HRs) and 95% confidence intervals (CIs) were employed to assess this association while considering covariates.

**Results:** There was a significant association between both $PM_{2.5}$ baseline and $PM_{2.5}$-change and the incidence of type 2 diabetes in a dose-response manner. Compared to quartile 1, the HRs for quartiles 2 to 4 of $PM_{2.5}$-baseline were 1.11 (95% CI, 0.74 to 1.65), 1.51 (95% CI, 1.00 to 2.28), and 1.77 (95% CI, 1.07 to 2.93), respectively. Similarly, the HRs for quartiles 2 to 4 of $PM_{2.5}$-change were 1.41 (95% CI, 1.14 to 1.75), 1.43 (95% CI, 1.13 to 1.81) and 2.40 (95% CI, 1.84 to 3.14), respectively.

**Conclusions:** Our findings contribute to existing evidence regarding the association between short-term and long-term exposure to $PM_{2.5}$ and the incidence of diabetes among personnel in the Royal Thai Army.

**Key words:** Particulate matter, Air pollution, Type 2 diabetes, Military personnel

## INTRODUCTION

Among the many environmental issues that have a negative impact on human health, air pollution is a significant concern. According to the World Health Organization (WHO), air pollution causes more than 7 million deaths per year, with over 90% of these fatalities occurring in low-income to middle-income countries, particularly in East Asia, South Asia, and the Western Pacific region [1]. Among the various air pollutants, particulate matter with a diameter <2.5 microns ($PM_{2.5}$) is considered the most harmful to human health. Evidence from both human and animal studies has suggested that $PM_{2.5}$ is a significant risk factor for type 2 diabetes [2-4], a chronic disease with a significant public health burden worldwide and a rising trend in Thailand [5,6].

Several studies have shown that an increase in $PM_{2.5}$ has a significant impact on the global burden of diabetes. According to the Global Burden of Disease (GBD) 2017 report, 2.94 million deaths were attributed to particulate matter exposure [7]. In 2019, a fifth of the global burden of type 2 diabetes was attributable to $PM_{2.5}$ exposure, with an estimated 3.78 deaths per 100 000 population and 167 disability-adjusted life-years (DALYs) per 100 000 population [8]. However, the exposure-re-

Received: June 27, 2023 Accepted: September 7, 2023

**Corresponding author:** Wiroj Jiamjarasrangsi
Department of Preventive and Social Medicine, Faculty of Medicine, Chulalongkorn University, 1873 Rama 4 Road, Pathumwan, Bangkok 10330, Thailand
E-mail: wjiamja@gmail.com

sponse curve in the GBD does not provide reliable estimates at the upper end of the global ambient air pollution exposure range due to a scarcity of studies in highly polluted areas. In addition, the exposure measurements in these studies were heterogeneous. Specifically, the exposure range over which risks were assessed varied, and the adjustments for potential confounders varied [8].

Most research on the relationship between $PM_{2.5}$ exposure, blood sugar levels, and incident diabetes mellitus has been conducted in developed countries such as Hong Kong [9], Japan [10], Canada [11], Denmark [12], and the United States [13]. Studies from developing countries like India [14], China [15], and Indonesia [16] are relatively scarce, with China contributing most of the available research. Because most of these studies were cross-sectional, results that shed light on causation are limited.

This study investigated the association between ambient $PM_{2.5}$ exposure and type 2 diabetes risk in Thailand. Army personnel were of particular interest due to their outdoor work and regular engagement in combat drills. A longitudinal design was utilized. Both baseline and subsequent $PM_{2.5}$ exposure were assessed, and various potential confounders were considered. Specifically, the study objective was to determine the association of both baseline $PM_{2.5}$ exposures and subsequent temporal changes in $PM_{2.5}$ exposure with the incidence of type 2 diabetes among the Royal Thai Army personnel, considering potential confounders at the individual as well as area level.

## METHODS

### Study Population

This retrospective cohort study was conducted within the Royal Thai Army. The study population consisted of 91 078 individuals who underwent health check-ups in 461 army units across Thailand in 2018 (Figure 1). Participants were excluded who had missing data for workplace locations (n=3157), no data for $PM_{2.5}$ exposure from the Geo-Informatics and Space Technology Development Agency (GISTDA; n=1212), incomplete demographic information (n=4320), and other affiliations (n=5907). Officers with a rank of general and above were also excluded from the study (n=155). The study included individuals who were stationed in active duty positions, were aged 35-60 years, had at least 2 annual health checks between 2018 and 2021, and had not been diagnosed with diabetes before 2018. The study ultimately included 21 325 eligible participants.

The required sample size for this study was determined using the formula for comparing 2 incidence rates [17], with a 5% alpha error and 80% statistical power, a follow-up time of 3 years, and postulated type 2 diabetes incidence rates of 8.1 and 11.4 per 1000 person-years for those in the quintile I and quintile V exposure levels, respectively [13]. The calculated number of participants needed was 18 276. Thus, our actual number of participants was deemed sufficient to achieve adequate statistical power to address the research questions in this study.

The follow-up period between 2018 and 2021 was calculated in person-years from the date of study enrollment (2018) to the date of the participant's last interview, or any date within

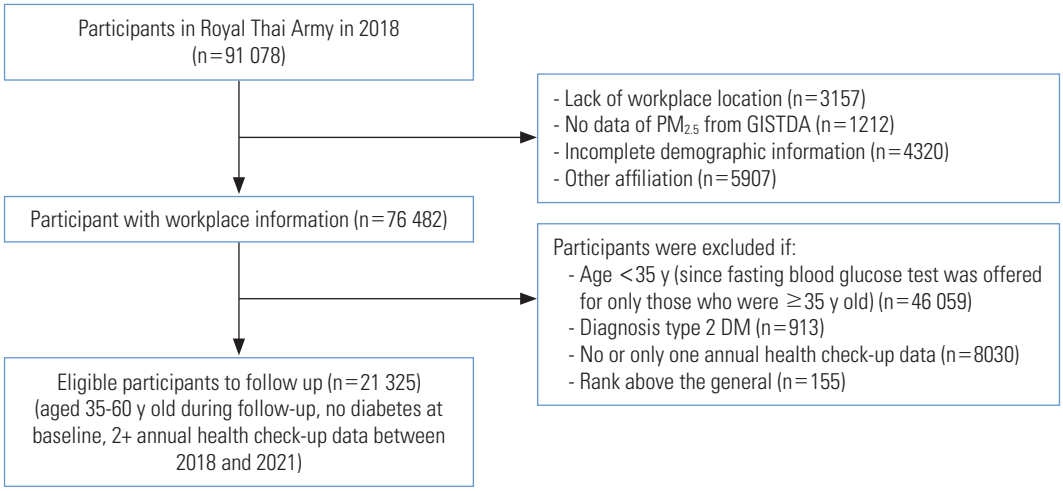


**Figure 1.** The flow chart of study population selection. $PM_{2.5}$, particulate matter with a diameter <2.5 microns; GISTDA, Geo-Informatics and Space Technology Development Agency; DM, diabetes mellitus.

the study period that the participant reached 60 years of age, died, or developed diabetes. The onset of diabetes was considered the outcome event, and the other conditions were considered censored events. The numbers of participants with 1, 2, and 3 follow-up years were 3084 (14.5%); 12 018 (56.4%); and 6223 (29.2%).

## Assessment of Diabetes

The diagnosis of diabetes or treatment for diabetes was based on the diagnostic criteria provided by the American Diabetes Association [18], which included a fasting plasma glucose (FPG) level ≥ 126 mg/dL (7.0 mmol/L) or a medical record of diabetes mellitus.

## Exposure Assessment

The data for $PM_{2.5}$ were obtained from the GISTDA of Thailand. GISTDA uses satellite data to monitor particulate matter concentrations. The agency provides an approximate average of $PM_{2.5}$ coverage across the entire country, using the Aerosol Optical Depth parameter obtained from satellites equipped with the Moderate Resolution Imaging Spectroradiometer. $PM_{2.5}$ levels were forecast and monitored at a resolution of approximately $3 \times 3$ $km^2$, with subsequent averaging at the subdistrict level. Daily $PM_{2.5}$ data were used to calculate the annual average. $PM_{2.5}$ exposure was summarized in 2 parameters: (1) $PM_{2.5}$-baseline, which used the average $PM_{2.5}$ exposure from 2015 to 2017 to assess the effects of long-term exposure, and (2) $PM_{2.5}$-change, which calculated the difference between $PM_{2.5}$-baseline and the $PM_{2.5}$ levels in lag years 1-3 before an outcome event or censorship. These 2 parameters were used respectively to assess the impact of spatial (i.e., inter-cluster) and temporal (i.e., intra-cluster) variations in $PM_{2.5}$ exposure on type 2 diabetes risk. Other weather data, including rainfall, relative humidity, average wind speed, and average temperature were obtained from the Pollution Control Department, Ministry of Natural Resources and Environment. Data on other air pollutants such as sulfur dioxide ($SO_2$), nitrogen dioxide ($NO_2$), ozone ($O_3$), and carbon monoxide (CO) were obtained from the Meteorological Department and the Pollution Control Department's application named Air4Thai [19]. The data used for this analysis covered the period between 2015 and 2017.

## Covariate Data

Covariates included in the analysis were age, sex, height, weight, smoking status (never smoked, ex-smoker, non-regular smoker, regular smoker, not specified), alcohol consumption (never, ex-drinker, non-regular drinker, regular drinker, not specified), and physical activity (never, <150, >150 min/wk, not specified). Overweight was defined as having a body mass index (BMI) between 23.0 $kg/m^2$ and 24.9 $kg/m^2$, and obesity was defined as having a BMI ≥25.0 $kg/m^2$ [20]. These covariates were obtained from the Army Health Examination data. An answer of "not specified" in the questionnaire was considered missing information and the participant was excluded.

## Statistical Analysis

In the descriptive analysis, participants were categorized into 4 groups according to the baseline $PM_{2.5}$ exposure level. Qualitative data, including sex, smoking status, alcohol consumption, and physical activity were presented using frequency and percentage. Quantitative data (e.g., age; BMI; annual average $PM_{2.5}$ exposure; and FPG, $SO_2$, $NO_2$, CO, and $O_3$ levels) were presented using mean and standard deviation (SD) or median and interquartile range, depending on the distribution of data. Differences in demographic characteristics and confounding factors among the participant groups were determined by the chi-square test for qualitative data, and by the one-way analysis of variance or Kruskal-Wallis tests for quantitative data, depending on the distribution of the data. The incidence of diabetes was calculated by dividing the number of new-onset diabetes cases by the total time at risk of diabetes in person-years and presented as an incidence rate per 1000 person-years.

Inferential analysis was conducted using multilevel mixed-effects parametric survival analysis (Weibull distribution) to analyze the relationships of $PM_{2.5}$-baseline and $PM_{2.5}$-change with the incidence of diabetes [21]. The level 1 variables included individual-level factors such as age, sex, BMI, blood sugar level, smoking status, alcohol consumption, and exercise status. The level 2 variables were area-level (or army-unit) factors, including $PM_{2.5}$ exposure level, rainfall amount, relative humidity, average wind speed, and average temperature, as well as $SO_2$, $NO_2$, CO, and $O_3$ levels. Hazard ratios (HRs) and the corresponding 95% confidence intervals (CIs) were calculated and presented as the measure of association.

The exposure-outcome associations were analyzed with 4 models. In the first 3 models, either $PM_{2.5}$- baseline or $PM_{2.5}$-change parameters were analyzed separately as a sole predictor while, in the fourth model, both $PM_{2.5}$-baseline and $PM_{2.5}$-change parameters were simultaneously included as the pre-

dictors. $PM_{2.5}$ was divided into 4 quartiles and the first quartile was used as a reference value. Model I was the crude model where either $PM_{2.5}$-baseline or $PM_{2.5}$-change parameters were included as the sole predictor. In model II, the individual-level covariates, including age, sex, BMI, baseline plasma glucose level, smoking status, alcohol consumption, and physical activity level were added to model I. In model III, area-level covariates such as relative humidity, wind speed, and rainfall were added to model II. Model IV was similar to model III except that both $PM_{2.5}$-baseline and $PM_{2.5}$-change parameters were included as the 2 main predictors. Multicollinearity was solved by including independent variables with a pairwise correlation <70% the statistical models [22].

The statistical analysis was conducted using Stata version 17.0 (StataCorp., College Station, TX, USA), and a 2-tailed *p*-value <0.05 indicated statistical significance.

### Ethics Statement

The study was approved by the Research Ethics Review Committee, Faculty of Medicine, Chulalongkorn University (IRB reference No. 0882/65) and the Institutional Review Board of the Royal Thai Army Medical Department (IRB reference No. 0954/2566).

## RESULTS

### Participant Characteristics

The participants ranged in age from 35 years to 60 years, with a mean±SD age of 46.2±7.3 years. The number of male participants was 17 994 (84.4%). The mean±SD BMI was 24.57±3.44 kg/m$^2$. Most participants never smoked (57.5%), were non-regular drinkers (53.5%), and engaged in physical activity for more than 150 min/wk (59.0%). When categorizing the participants into $PM_{2.5}$-baseline quartiles (Table 1), significant group differences were observed in the composition of age, sex, smoking status, alcohol consumption, and exercise status. However, no significant group differences were observed in BMI.

### Particulate Matter With a Diameter <2.5 Microns Exposure

The average annual $PM_{2.5}$ exposure from 2015 to 2017 ($PM_{2.5}$-baseline) for the 461 army units across Thailand ranged from 14.64 μg/m$^3$ to 25.16 μg/m$^3$, with a median of 21.64 μg/m$^3$ (quartiles 1-3, 19.17-24.38). The temporal change of $PM_{2.5}$ levels in the lag years 1-3 ($PM_{2.5}$-change) from 2018 to 2020 ranged from -5.86 μg/m$^3$ to 6.06 μg/m$^3$, with a mean±SD of 0.22±1.41 μg/m$^3$. The distributions of $PM_{2.5}$-baseline and $PM_{2.5}$-change, according to their quartiles, are shown in Supplemental Material 1A and B, respectively.

### Association Between Particulate Matter With a Diameter <2.5 Microns Exposure and the Incidence of Type 2 Diabetes

The incidence rate of diabetes mellitus tended to fluctuate in relation to increasing $PM_{2.5}$ concentrations: the incidence rates in $PM_{2.5}$ quartiles 1-4 were 21.87, 25.47, 23.07, and 18.13 per 1000 person-years, respectively (Table 2).

This study analyzed the relationship between the spatial and temporal variations in $PM_{2.5}$ exposure (in terms of $PM_{2.5}$-baseline and $PM_{2.5}$-change) with the incidence of type 2 diabetes. A significant association between $PM_{2.5}$-baseline and the increased incidence of type 2 diabetes was observed only in the fully adjusted models (models III and IV). For model IV, the HR (95% CI) for quartiles 2-4 compared to quartile 1 of $PM_{2.5}$-baseline were 1.11 (95% CI, 0.74 to 1.65), 1.51 (95% CI, 1.00 to 2.38), and 1.77 (95% CI, 1.07 to 2.93), respectively. In contrast, $PM_{2.5}$-change was found to be significantly associated with the disease risk with a consistent dose-response pattern all the way from the crude (model I) to the fully adjusted model (model IV), though the magnitudes of association were generally reduced. The HRs (95% CI) for quartiles 2-4 compared to quartile 1 of $PM_{2.5}$-change were 1.36 (95% CI, 1.11 to 1.65), 1.97 (95% CI, 1.61 to 2.42), and 3.67 (95% CI, 2.99 to 4.51) for model I and 1.41 (95% CI, 1.14 to 1.75), 1.43 (95% CI, 1.13 to 1.81), and 2.40 (95% CI, 1.84 to 3.14) for model IV, respectively (Table 3).

Since the $PM_{2.5}$-change category was difficult to interpret, we reclassified it into 7 categories and reanalyzed model IV by designating the middle category (with a temporal change magnitude between -0.5 and 0.5 μg/m$^3$) as the reference category; these results are shown in Figure 2. The increasing categories of $PM_{2.5}$-change were significantly associated with an increased incidence of type 2 diabetes in a progressive manner. For the $PM_{2.5}$-change categories of 0.51 μg/m$^3$ to 1.50 μg/m$^3$, 1.51 μg/m$^3$ to 2.50 μg/m$^3$, and greater than 2.50 μg/m$^3$, the HRs (95% CIs) were: 1.51 (95% CI, 1.24 to 1.84), 3.99 (95% CI, 3.04 to 5.25), and 4.91 (95% CI, 3.33 to 7.23), respectively. In contrast, decreasing categories of $PM_{2.5}$-change were not significantly associated with a decrease in the incidence of type 2 diabetes.

**Table 1.** Comparison of the demographic characteristics of the participants at baseline according to the $PM_{2.5}$ exposure levels (n=21 325)

| Characteristics | $PM_{2.5}$-baseline (μg/m³)[1] | | | |
|---|---|---|---|---|
| | Quartile 1 (14.64-19.17) | Quartile 2 (19.18-21.64) | Quartile 3 (21.65-24.37) | Quartile 4 (24.38-25.16) |
| Age (y) | | | | |
| <45 | 2642 (49.5) | 2531 (45.9)[2] | 2912 (45.3)[2] | 1652 (40.9)[2,3,4] |
| ≥45 | 2691 (50.5) | 2989 (54.1) | 3517 (54.7) | 2391 (59.1) |
| Sex | | | | |
| Male | 5032 (94.4) | 4911 (89.0)[2] | 4984 (77.5)[2,3] | 3067 (75.9)[2,3] |
| Female | 301 (5.6) | 609 (11.0) | 1445 (22.5) | 976 (24.1) |
| BMI (kg/m²) | | | | |
| Underweight | 77 (1.4) | 92 (1.7) | 115 (1.8)[2] | 94 (2.3) |
| Normal | 3034 (56.9) | 3261 (59.1) | 3781 (58.8) | 2300 (56.9) |
| Overweight | 1882 (35.3) | 1812 (32.8) | 2177 (33.9) | 1367 (33.8) |
| Obesity | 340 (6.4) | 355 (6.4) | 356 (5.5) | 282 (7.0) |
| Smoking status | | | | |
| Never | 2788 (52.3) | 3338 (60.5)[2] | 3564 (55.4)[2] | 2574 (63.7)[2,3,4] |
| Ex-Smoker | 873 (16.4) | 873 (15.8) | 1305 (20.3) | 648 (16.0) |
| Non-regular smoker | 566 (10.6) | 561 (10.2) | 785 (12.2) | 399 (9.9) |
| Regular smoker | 1106 (20.7) | 748 (13.5) | 775 (12.1) | 422 (10.4) |
| Alcohol consumption | | | | |
| Never | 1234 (23.1) | 1768 (32.0)[2] | 1774 (27.6)[2,3] | 1337 (33.1)[2,4] |
| Ex-drinker | 851 (16.0) | 514 (9.3) | 734 (11.4) | 377 (9.3) |
| Non-regular drinker | 2438 (45.7) | 3059 (55.5) | 3710 (57.7) | 2208 (54.6) |
| Regular drinker | 810 (15.2) | 179 (3.2) | 211 (3.3) | 121 (3.0) |
| Exercise level (min/wk) | | | | |
| No exercise | 349 (6.5) | 893 (16.2)[2] | 559 (8.7)[3] | 178 (4.4)[2,3,4] |
| ≤150 | 1716 (32.2) | 1911 (34.6) | 1866 (29.0) | 1268 (31.4) |
| >150 | 3268 (61.3) | 2716 (49.2) | 4004 (62.3) | 2597 (64.2) |

$PM_{2.5}$, particulate matter with a diameter <2.5 microns; BMI, body mass index.
[1]Average concentration of $PM_{2.5}$ from 2015 to 2017.
[2]Significant difference from quartile 1 at $p<0.05$.
[3]Significant difference from quartile 2 at $p<0.05$.
[4]Significant difference from quartile 3 at $p<0.05$.

**Table 2.** Incidence rate of type 2 diabetes according to $PM_{2.5}$ exposure at baseline in Thai Army personnel

| $PM_{2.5}$-baseline (μg/m³)[1] | Participants (person) | Person-years | New case | Incidence rate | 95% CI | |
|---|---|---|---|---|---|---|
| | | | | | LL | UL |
| Total | 21 325 | 81 736 | 1834 | 22.44 | 21.43 | 23.49 |
| Quartile 1 (14.64-19.17) | 5333 | 20 534 | 449 | 21.87 | 19.93 | 23.99 |
| Quartile 2 (19.18-21.64) | 5520 | 21 045 | 536 | 25.47 | 23.40 | 27.72 |
| Quartile 3 (21.65-24.37) | 6429 | 24 492 | 565 | 23.07 | 21.24 | 25.05 |
| Quartile 4 (24.38-25.16) | 4043 | 15 665 | 284 | 18.13 | 16.14 | 20.37 |

$PM_{2.5}$, particulate matter with a diameter <2.5 microns; CI, confidence interval; LL, lower limit; UL, upper limit.
[1]Average concentration of $PM_{2.5}$ from 2015 to 2017.

## DISCUSSION

In this study, we investigated the relationship of spatial and temporal variations in $PM_{2.5}$ exposure (based on $PM_{2.5}$-baseline and $PM_{2.5}$-change) with the incidence of type 2 diabetes among Thai Army personnel from 2018 to 2021. While both $PM_{2.5}$ parameters were significantly associated with type 2 diabetes incidence, those for temporal variation in $PM_{2.5}$ exposure were

**Table 3.** Hazard ratios for the association of baseline $PM_{2.5}$ exposure and changes in $PM_{2.5}$ exposure over 3 years with the incidence of type 2 diabetes among Thai Army personnel (n=21 325)[1]

| $PM_{2.5}$ parameter (Min. Max, μg/m³) | Model I | Model II | Model III | Model IV |
|---|---|---|---|---|
| $PM_{2.5}$-baseline[2] | | | | |
| Quartile 1 (14.64, 19.17) | 1.00 (reference) | 1.00 (reference) | 1.00 (reference) | 1.00 (reference) |
| Quartile 2 (19.18, 21.64) | 1.16 (0.86, 1.58) | 1.17 (0.87, 1.56) | 1.16 (0.79, 1.70) | 1.11 (0.74, 1.65) |
| Quartile 3 (21.65, 24.37) | 1.24 (0.89, 1.70) | 1.24 (0.91, 1.69) | 1.37 (0.92, 2.03) | 1.51 (1.00, 2.28)* |
| Quartile 4 (24.38, 25.16) | 0.85 (0.55, 1.19) | 0.85 (0.59, 1.23) | 2.01 (1.23, 3.27)** | 1.77 (1.07, 2.93)* |
| $PM_{2.5}$-change[3] | | | | |
| Quartile 1 (-5.86, -0.65) | 1.00 (reference) | 1.00 (reference) | 1.00 (reference) | 1.00 (reference) |
| Quartile 2 (-0.64, -0.30) | 1.36 (1.11, 1.65)** | 1.30 (1.07, 1.58)** | 1.39 (1.12, 1.71)** | 1.41 (1.14, 1.75)** |
| Quartile 3 (0.31, 1.02) | 1.97 (1.61, 2.42)*** | 1.92 (1.57, 2.36)*** | 1.44 (1.14, 1.81)** | 1.43 (1.13, 1.81)** |
| Quartile 4 (1.03, 6.06) | 3.67 (2.99, 4.51)*** | 3.48 (2.83, 4.29)*** | 2.40 (1.84, 3.13)*** | 2.40 (1.84, 3.14)*** |

Values are presented as hazard ratio (95% confidence interval).
$PM_{2.5}$, particulate matter with a diameter <2.5 microns; Min, minimum; Max, maximum.
[1]Model I: only $PM_{2.5}$-baseline or $PM_{2.5}$-change was included as a predictor; Model II: Model I plus individual-level covariates (such as age, sex, BMI, baseline plasma glucose level, smoking status, alcohol consumption, and physical activity); Model III: Model II plus area-level covariates (such as relative humidity, wind speed, and rainfall); Model IV: both $PM_{2.5}$-baseline and $PM_{2.5}$-change were simultaneously included as the predictors, plus individual-level and area-level covariates.
[2]The average of the annual $PM_{2.5}$ exposure from 2015 to 2017 (baseline).
[3]The difference between the baseline $PM_{2.5}$ and the $PM_{2.5}$ levels in lag years 1-3 before the outcome event or censorship.
*$p$<0.05, **$p$<0.01, ***$p$<0.001.

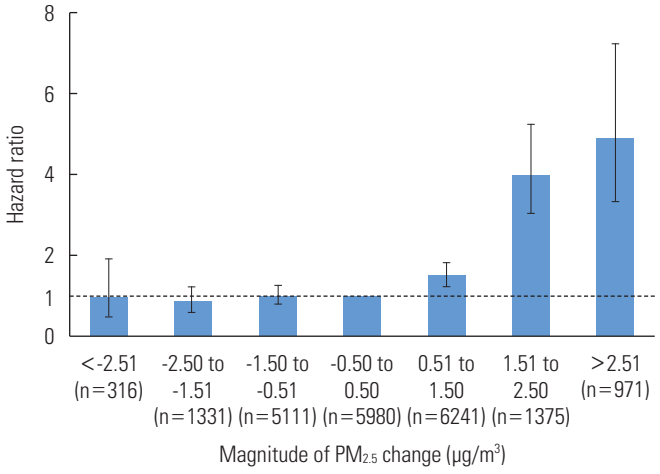


**Figure 2.** Hazard ratios for relationship between the different levels of $PM_{2.5}$ change and risk of type 2 diabetes (the middle category ($PM_{2.5}$ change of -0.50 to 0.50 μg/m³ was designated as the reference). Bar represents the hazard ratio and range represents the corresponding 95% confidence interval. Numbers in parentheses represents the number of participants in each category.

more robust than those for spatial variation. While the temporal increase in $PM_{2.5}$ exposure was significantly and progressively associated with type 2 diabetes risk, a decrease in $PM_{2.5}$ exposure did not decrease disease risk.

We found that higher exposures to $PM_{2.5}$ were associated with an increased risk of diabetes, and the strength of association was proportional to the amount of $PM_{2.5}$. These results are consistent with previous studies, such as those conducted by Liu et al. [15], Li et al. [23] in China and Lee et al. [10] in Japan. However, 2 studies by Curto et al. [14] in India and Coogan et al. [13] in the United States did not find such a relationship. The variation in findings could be attributed to several factors, including the heterogeneity of population characteristics, opportunities for exposure, toxicological properties of $PM_{2.5}$, or other factors related to diabetes such as the eating habits in different study areas.

Furthermore, our finding that the temporal change had a more pronounced impact than the spatial difference of $PM_{2.5}$ exposure on type 2 diabetes risk may reflect the shorter time frame between the increased $PM_{2.5}$ exposure and changes in FPG. The findings of Zhan et al. [24] and Cai et al. [25] indicated that short-term and medium-term exposure to ambient $PM_{2.5}$ was associated with higher FPG levels. These consistent results across studies suggest that short-term and medium-term exposure to $PM_{2.5}$ may contribute to increased FPG levels. However, other alternative explanations are also possible. More studies are therefore needed.

The magnitudes of association between $PM_{2.5}$ exposure and diabetes risk reported in our study were noteworthy and generally stronger than those reported previously. When the HR

for $PM_{2.5}$-change and diabetes incidence in Table 3 was reanalyzed per 1 μg/$m^3$ increase rather than by quartile comparison, the HR (95% CI) was 1.38 (95% CI, 1.27 to 1.50), as compared to the hazard, risk, or odds ratios of 0.95 to 1.25 in previous reports [10,26,27]. This might be because the individuals in our study population had military occupations that primarily involved outdoor duties, thus were exposed to higher levels of $PM_{2.5}$ than the general population. In addition, we observed variations between the $PM_{2.5}$ data collected from satellites (maximum 25.53, minimum 13.59, and average 20.77 μg/$m^3$) and the $PM_{2.5}$ data obtained from field stations (maximum 33.30, minimum 14.52, and average 22.76 μg/$m^3$) (results not shown). Notably, the range of $PM_{2.5}$ values from the field stations was broader than the range observed from satellite data, which suggests that the HR values in this study may be overestimated.

The strengths of our study included the use of a cohort design to investigate the relationship between $PM_{2.5}$ exposure and diabetes. We also considered various potential confounders, both at individual and sub-district levels. Our study also analyzed both the temporal variation and spatial variation associated with $PM_{2.5}$. However, limitations of the study included a lack of individual data on eating behaviors [28] and family history [29] related to diabetes. In addition, we did not conduct hemoglobin A1c tests, which are commonly used for the diagnosis of diabetes. Our participants were soldiers, who may have different eating habits, exercise routines, and exposure levels to $PM_{2.5}$ compared to the general population. Furthermore, the overlap of the coronavirus disease 2019 (COVID-19) pandemic with our study period, with its resultant disruption in social, economic, and physical environments as well as individual daily life behaviors [30,31], might have confounded our study findings. During our study period, we observed that the pandemic had an impact on $PM_{2.5}$ levels from transportation-related sources. However, these sources were approximately 13% of all $PM_{2.5}$ sources [32,33]. Meanwhile, there were other significant sources of $PM_{2.5}$, such as open burning and manufacturing industries that continued their operations by implementing "bubble and seal" control policies [34]. The impact of the pandemic on individuals' daily-life behaviors, such as wearing masks and decreasing outdoor physical activity [35], might have resulted in less exposure to $PM_{2.5}$ than our exposure assessments (based on the ambient $PM_{2.5}$ monitoring data) reflect. However, this exposure measurement error was most likely either non-differential or differential toward the areas and periods with poorer air quality (i.e., air pollution levels relate positively with mask-wearing [36] and inversely with outdoor physical activity levels [37]). This would result in weakening rather than overestimating the magnitude of the exposure-outcome association [38]. Nevertheless, the impact of the COVID-19 pandemic was extensive and could not be considered completely; further studies during a normal period are needed to confirm or refute our findings.

## SUPPLEMENTAL MATERIALS

Supplemental material is available at https://doi.org/10.3961/jpmph.23.292.

## CONFLICT OF INTEREST

The authors have no conflicts of interest associated with the material presented in this paper.

## FUNDING

None.

## ACKNOWLEDGEMENTS

All authors would like to thank: Air4Thai of the Pollution Control Department, the Ministry of Natural Resources and Environment, Thailand; the Geo-Informatics and Space Technology Development Agency (GISTDA), Thailand; and the Royal Thai Army Medical Department for providing the environmental and health data used in this study.

## AUTHOR CONTRIBUTIONS

Conceptualization: Jiamjarasrangsi W. Data curation: Laorattapong A. Formal analysis: Laorattapong A, Poobunjirdkul S, Rattananupong T. Funding acquisition: None. Methodology: Jiamjarasrangsi W, aorattapong A, Rattananupong T. Project administration: Laorattapong A. Visualization: Laorattapong A. Writing – original draft: Laorattapong A, Jiamjarasrangsi W. Writing – review & editing: Laorattapong A, Poobunjirdkul S, Rattananupong T, Jiamjarasrangsi W.

## ORCID

Apisorn Laorattapong https://orcid.org/0009-0000-7454-9934
Sarun Poobunjirdkul https://orcid.org/0009-0001-4274-5663
Thanapoom Rattananupong https://orcid.org/0000-0002-3853-1785
Wiroj Jiamjarasrangsi https://orcid.org/0000-0002-0610-6848

## REFERENCES

1. Climate and Clean Air Coalition. World Health Organization releases new global air pollution data [cited 2022 Jun 11]. Available from: https://www.ccacoalition.org/news/world-health-organization-releases-new-global-air-pollution-data.
2. Balti EV, Echouffo-Tcheugui JB, Yako YY, Kengne AP. Air pollution and risk of type 2 diabetes mellitus: a systematic review and meta-analysis. Diabetes Res Clin Pract 2014;106(2):161-172.
3. Esposito K, Petrizzo M, Maiorino MI, Bellastella G, Giugliano D. Particulate matter pollutants and risk of type 2 diabetes: a time for concern? Endocrine 2016;51(1):32-37.
4. He D, Wu S, Zhao H, Qiu H, Fu Y, Li X, et al. Association between particulate matter 2.5 and diabetes mellitus: a meta-analysis of cohort studies. J Diabetes Investig 2017;8(5):687-696.
5. Guariguata L, Whiting DR, Hambleton I, Beagley J, Linnenkamp U, Shaw JE. Global estimates of diabetes prevalence for 2013 and projections for 2035. Diabetes Res Clin Pract 2014;103(2):137-149.
6. Beagley J, Guariguata L, Weil C, Motala AA. Global estimates of undiagnosed diabetes in adults. Diabetes Res Clin Pract 2014;103(2):150-160.
7. GBD 2017 Risk Factor Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet 2018;392(10159):1923-1994.
8. GBD 2019 Diabetes and Air Pollution Collaborators. Estimates, trends, and drivers of the global burden of type 2 diabetes attributable to PM2·5 air pollution, 1990-2019: an analysis of data from the Global Burden of Disease Study 2019. Lancet Planet Health 2022;6(7):e586-e600.
9. Qiu H, Schooling CM, Sun S, Tsang H, Yang Y, Lee RS, et al. Long-term exposure to fine particulate matter air pollution and type 2 diabetes mellitus in elderly: a cohort study in Hong Kong. Environ Int 2018;113:350-356.
10. Lee M, Ohde S. PM2.5 and diabetes in the Japanese population. Int J Environ Res Public Health 2021;18(12):6653.
11. Chen H, Burnett RT, Kwong JC, Villeneuve PJ, Goldberg MS, Brook RD, et al. Risk of incident diabetes in relation to long-term exposure to fine particulate matter in Ontario, Canada. Environ Health Perspect 2013;121(7):804-810.
12. Hansen AB, Ravnskjær L, Loft S, Andersen KK, Bräuner EV, Baastrup R, et al. Long-term exposure to fine particulate matter and incidence of diabetes in the Danish Nurse Cohort. Environ Int 2016;91:243-250.
13. Coogan PF, White LF, Yu J, Burnett RT, Seto E, Brook RD, et al. PM2.5 and diabetes and hypertension incidence in the Black Women's Health Study. Epidemiology 2016;27(2):202-210.
14. Curto A, Ranzani O, Milà C, Sanchez M, Marshall JD, Kulkarni B, et al. Lack of association between particulate air pollution and blood glucose levels and diabetic status in peri-urban India. Environ Int 2019;131:105033.
15. Liu C, Yang C, Zhao Y, Ma Z, Bi J, Liu Y, et al. Associations between long-term exposure to ambient particulate air pollution and type 2 diabetes prevalence, blood glucose and glycosylated hemoglobin levels in China. Environ Int 2016;92-93:416-421.
16. Suryadhi MA, Suryadhi PA, Abudureyimu K, Ruma IM, Calliope AS, Wirawan DN, et al. Exposure to particulate matter (PM2.5) and prevalence of diabetes mellitus in Indonesia. Environ Int 2020;140:105603.
17. Lwanga SK, Lemeshow S; World Health Organization. Sample size determination in health studies: a practical manual; 1991 [cited 2023 Jun 11]. Available from: https://apps.who.int/iris/handle/10665/40062.
18. American Diabetes Association Professional Practice Committee. 2. Classification and diagnosis of diabetes: standards of medical care in diabetes—2022. Diabetes Care 2022;45(Suppl_1):S17–S38.
19. Air4Thai. Regional air quality and situation reports [cited 2023 Jun 11]. Available from: http://air4thai.pcd.go.th/webV3/#/Home.
20. World Health Organization. The Asia-Pacific perspective: redefining obesity and its treatment; 2000 [cited 2023 Jun 11]. Available from: https://apps.who.int/iris/handle/10665/206936.
21. StataCorp LLC. mestreg — multilevel mixed-effects parametric survival models [cited 2023 Jun 11]. Available from: https://www.stata.com/manuals/memestreg.pdf.
22. Leiby BD, Ahner DK. Multicollinearity applied stepwise stochastic imputation: a large dataset imputation through correlation-

based regression. J Big Data 2023;10(1):1-20.

23. Li CY, Wu CD, Pan WC, Chen YC, Su HJ. Association between long-term exposure to PM2.5 and incidence of type 2 diabetes in Taiwan: a national retrospective cohort study. Epidemiology 2019;30 Suppl 1:S67-S75.
24. Zhan M, Li Z, Li X, Tao B, Zhang Q, Wang J. Effect of short-term ambient PM2.5 exposure on fasting blood glucose levels: a longitudinal study among 47,471 people in eastern China. Environ Pollut 2021;290:117983.
25. Cai L, Wang S, Gao P, Shen X, Jalaludin B, Bloom MS, et al. Effects of ambient particulate matter on fasting blood glucose among primary school children in Guangzhou, China. Environ Res 2019;176:108541.
26. Chilian-Herrera OL, Tamayo-Ortiz M, Texcalac-Sangrador JL, Rothenberg SJ, López-Ridaura R, Romero-Martínez M, et al. PM2.5 exposure as a risk factor for type 2 diabetes mellitus in the Mexico City metropolitan area. BMC Public Health 2021; 21(1):2087.
27. Weinmayr G, Hennig F, Fuks K, Nonnemacher M, Jakobs H, Möhlenkamp S, et al. Long-term exposure to fine particulate matter and incidence of type 2 diabetes mellitus in a cohort study: effects of total and traffic-specific air pollution. Environ Health 2015;14:53.
28. Ueno S, Aung MN, Yuasa M, Ishtiaq A, Khin ET, Latt TS, et al. Association between dietary habits and type 2 diabetes mellitus in Yangon, Myanmar: a case-control study. Int J Environ Res Public Health 2021;18(21):11056.
29. InterAct Consortium; Scott RA, Langenberg C, Sharp SJ, Franks PW, Rolandsson O, et al. The link between family history and risk of type 2 diabetes is not explained by anthropometric, lifestyle or genetic risk factors: the EPIC-InterAct study. Diabetologia 2013;56(1):60-69.
30. Onyeaka H, Anumudu CK, Al-Sharify ZT, Egele-Godswill E, Mbaegbu P. COVID-19 pandemic: a review of the global lockdown and its far-reaching effects. Sci Prog 2021;104(2):36850421101 9854.
31. Miyah Y, Benjelloun M, Lairini S, Lahrichi A. COVID-19 impact on public health, environment, human psychology, global socioeconomy, and education. ScientificWorldJournal 2022; 2022:5578284.
32. Greenpeace. Greenpeace's city rankings for PM2.5 in Thailand [cited 2023 Sep 2]. Available from: https://greenpeace.or.th/s/right-to-clean-air/PM2.5CityRankingsREV.pdf.
33. Nuchdang S, Kingkam W, Tippawan U, Sriwiang W, Fungklin R, Rattanaphra D. Metal composition and source identification of PM2.5 and PM10 at a suburban site in Pathum Thani, Thailand. Atmosphere 2023;14(4):659.
34. Department of Disease Control. Manual for the disease prevention and control in specific area (bubble and seal) for workplace; 2021 [cited 2023 Sep 2]. Available from: https://ddc.moph.go.th/uploads/publish/1183620211001030623.pdf (Thai).
35. Park AH, Zhong S, Yang H, Jeong J, Lee C. Impact of COVID-19 on physical activity: a rapid review. J Glob Health 2022;12: 05003.
36. Yang Q, Wu S. Air pollution in China: health information seeking and protective behaviors. Health Promot Int 2020;35(6): 1495-1506.
37. Tainio M, Jovanovic Andersen Z, Nieuwenhuijsen MJ, Hu L, de Nazelle A, An R, et al. Air pollution, physical activity and health: a mapping review of the evidence. Environ Int 2021;147: 105954.