Eco-Environment & Health 3 (2024) 165–173

Contents lists available at ScienceDirect

Eco-Environment & Health

journal homepage: www.journals.elsevier.com/eco-environment-and-health

Original Research Article

Associations of short-term ambient temperature exposure with lung function in middle-aged and elderly people: A longitudinal study in China

Weihong Qiu [a,b,1], Bin Wang [a,1], Xiaobing Feng [a], Heng He [b], Lieyang Fan [a], Zi Ye [a], Xiuquan Nie [a], Ge Mu [a], Wei Liu [a], Dongming Wang [a], Min Zhou [a,2], Weihong Chen [a,*,2]

[a] Department of Occupational and Environmental Health, School of Public Health, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430030, China
[b] Department of Epidemiology and Health Statistics, School of Public Health, Fujian Medical University, Fuzhou 350122, China

ARTICLE INFO

Keywords:
Ambient temperature
Lung function
Middle-aged and elderly people
Vulnerable groups
Linear mixed-effects model
Generalized additive mixed model

ABSTRACT

The short-term associations of ambient temperature exposure with lung function in middle-aged and elderly Chinese remain obscure. The study included 19,128 participants from the Dongfeng-Tongji cohort's first (2013) and second (2018) follow-ups. The lung function for each subject was determined between April and December 2013 and re-assessed in 2018, with three parameters (forced vital capacity [FVC], forced expiratory volume in 1 s [FEV_1], and peak expiratory flow [PEF]) selected. The China Meteorological Data Sharing Service Center provided temperature data during the study period. In the two follow-ups, a total of 25,511 records (average age: first, 64.57; second, 65.80) were evaluated, including 10,604 males (41.57%). The inversely J-shaped associations between moving average temperatures (lag01–lag07) and FVC, FEV_1, and PEF were observed, and the optimum temperatures at lag04 were 16.5 °C, 18.7 °C, and 16.2 °C, respectively. At lag04, every 1 °C increase in temperature was associated with 14.07 mL, 9.78 mL, and 62.72 mL/s increase in FVC, FEV_1, and PEF in the low-temperature zone (<the optimum temperatures), whereas 5.72 mL, 2.01 mL, and 11.64 mL/s decrease in the high-temperature zone (≥the optimum temperatures), respectively (all $P < 0.05$). We observed significant effect modifications of gender, age, body mass index, body surface area, smoking status, drinking status, and physical activity on the associations (all $P_{\text{modification}} < 0.05$). Non-optimal temperatures may cause lung function decline. Several individual characters and lifestyles have effect modification on the temperature effects.

1. Introduction

Ambient temperature has been a critical environmental and physical factor influencing human survival and health [1,2]. Normally, the body's thermoregulation center can maintain a balance between heat production and dissipation to modulate the body temperature independent of the surrounding temperature. When the ambient temperature is outside the optimal range and exceeds the body's thermoregulation ability, it can elevate the risk of a variety of disorders, including chronic obstructive pulmonary disease (COPD), asthma, hypertension [3,4], and even mortality [5]. It is worth mentioning that in comparison to other age groups, middle-aged and elderly adults are more susceptible to ambient temperature due to their weaker physiological functions and self-regulation capacities [4,5].

The respiratory system, with its extensive surface area (about 70 m^2) and the highest contact frequency with the external environment, is considered one of the most sensitive systems affected by ambient temperature [5]. Ambient temperature can directly stimulate the epithelium, subcutaneous blood vessels, and airway smooth muscles, causing bronchial cavity changes and further interfering with respiratory function [6]. The lung function test is a clinically accepted noninvasive tool for assessing respiratory mechanics and physiology, and lung function reduction is widely regarded as the early phase of lung injury [7,8]. Several studies have assessed the effects of ambient temperature on lung function among the elderly population, but the results are inconclusive, and the majority of previous studies focus on people with chronic respiratory diseases. For example, a cohort study of 5,896 middle-aged and elderly people (average age: 50.4 ± 13.8 years) in the United States observed a decline in lung

---

* Corresponding author.
E-mail address: wchen@mails.tjmu.edu.cn (W. Chen).
[1] Co-first author.
[2] These authors contributed equally to this work.

https://doi.org/10.1016/j.eehl.2024.01.008
Received 29 August 2023; Received in revised form 16 January 2024; Accepted 25 January 2024

function as ambient temperature increased [9]. A study enrolled 4,992 adult asthmatic patients (average age: 45.4 ± 13.6 years) from 25 Chinese cities and found that both low and high temperatures were associated with decreased lung function [10]. However, a study reported no temperature effect on lung function among 69 participants with COPD (average age: 69 ± 8 years) [11], and similar results were also reported in a study of 30 former smokers with COPD (average age: 71.1 ± 8.4 years) from Boston, the United States [12]. Consequently, it is extremely urgent and worthwhile to further elucidate the temperature effect on lung function in a larger middle-aged and elderly population, particularly in light of growing global warming and aging.

Therefore, we performed the current study with a total of 19,128 middle-aged and elderly participants from the first (2013) and second (2018) follow-ups of the Dongfeng–Tongji (DFTJ) cohort to quantify the association between ambient temperature and lung function. The lung function test of each participant was performed from April to December 2013 and repeated in the same period in 2018 [13]. During the study, ambient temperature data were obtained from the nearest meteorological station. In addition, we also investigated the potential influencing factors at the individual level, including individual characteristics (gender, age, body mass index [BMI], body surface area [BSA]), and lifestyles (smoking, drinking, and physical activity).

2. Material and methods

2.1. Study participants

The study participants were from the DFTJ cohort, as previously described [14]. In short, the cohort began in 2008 among retirees of the Dongfeng Motor Corporation in Shiyan, China, and followed-up every five years. In the first (2013) and second (2018) follow-ups, a total of 14,973 and 12,430 participants who completed both lung function tests and questionnaires at Dongfeng General Hospital were recruited in this study, respectively. To avoid the impacts of serious respiratory diseases, participants were excluded if they had any mention of current pulmonary emphysema, tuberculosis, or lung cancer ($n = 1,704$). Furthermore, we excluded participants with missing data on required examinations or questionnaires ($n = 188$). Finally, a total of 19,128 participants providing 25,511 observations (first follow-up: 13,818; second follow-up: 11,693) were enrolled in this study, among whom 6383 provided two observations.

The study protocol was approved by the Ethics and Human Subject Committee of the School of Public Health, Tongji Medical College, Huazhong University of Science and Technology, and Dongfeng General Hospital, Dongfeng Motor Corporation (2010–16). All participants gave written informed consent.

2.2. Covariate data collection

During face-to-face interviews, trained interviewers gathered data on each participant's socio-demographic characteristics, lifestyles (such as smoking, drinking, physical activity), and medical history, etc. In the study, smokers were identified as individuals who had regularly smoked at least one cigarette per day for more than six months, and smoking status was divided into three groups: current, former, and never smoking. The smoking amount (pack-years) was calculated by multiplying cigarette pack(s) (1 pack = 20 cigarettes) per day by years of smoking. Passive smoking status was identified as passive exposure to secondhand smoke for at least 15 min per day for more than one day per week. The passive smoking amount (hour[s]/week·year) was calculated by multiplying passive smoking hours per week by years of passive smoking. Drinkers were identified as participants who had regularly drunk at least one time per week for more than six months and drinking status was classified into three groups: current, former, and never drinking. The drinking amount (time [s]/week·year) was calculated by multiplying drinking times per week by years of drinking. Physical activity was identified as engaging in regular leisure-time exercise for more than 20 min per time. Diseases diagnosed by professional physicians at the community level hospitals or above were reported by the participants themselves, including pulmonary emphysema, pulmonary tuberculosis, lung cancer, asthma, coronary heart disease (CHD), etc. Seasons are classified into four categories: spring (March to May), summer (June to August), autumn (September to November), and winter (December to February).

In addition, physical examinations for all participants were performed by trained medical staff, which covered standing height, body weight, etc. BMI was calculated by dividing the weight (kg) by height in meters squared (m^2) (Eq. 1), and BSA was calculated by the Mosteller formula (Eq. 2).

$$BMI = \frac{Weight}{Height^2} \tag{1}$$

$$BSA = \sqrt{\frac{Weight \times Height}{36}} \tag{2}$$

2.3. Lung function test

The lung function levels of all participants were assessed by trained medical staff using electronic spirometers (CHESTGRAPH HI-101, CHEST Ltd., Tokyo, Japan), following the American Thoracic Society recommendations. The type of equipment stayed the same and was calibrated every day throughout the study period during the two follow-ups. The detailed measuring method has been previously published [13,15]. In short, each participant was advised not to smoke or eat for at least 1 h before the test. During the test, the participants sat with a nose clip after at least 5 min of normal calm breathing. The staff instructed each participant to complete slow vital capacity (SVC) and forced vital capacity (FVC) measurements, respectively. (1) SVC measurement: each participant was instructed to perform three tidal breaths, followed by a slow exhalation to the residual volume level, a slow and forceful inhalation to the total volume level, a slow and forceful exhalation to the residual volume level, and finally a normal breath once. (2) FVC measurement: each participant was instructed to perform one tidal breath, followed by a forceful inhalation to the total volume level, a rapid and as long as possible exhalation to the residual volume level, and finally a normal breath once. Each participant was required to repeat the measurement until achieving three acceptable volume-time curves of lung function parameters, including FVC, forced expiratory volume in 1 s (FEV_1), and peak expiratory flow (PEF).

2.4. Meteorological variables and air pollutants exposure assessment

The China Meteorological Data Sharing Service Center (http://data.cma.cn/) and the National Real-Time Air Quality Monitoring Stations, which were created by the China National Environmental Monitoring Center [16,17], provided daily data on meteorological variables and air pollutants, respectively. The former includes temperature and relative humidity (RH), and the latter includes fine particulate matter ($PM_{2.5}$), inhalable particulate matter (PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), and ozone (O_3).

There was one fixed-site meteorological monitoring station and five fixed-site air quality monitoring stations in Shiyan. The daily exposure levels of meteorological variables and air pollutants during the study period were assessed by using data from the nearest monitoring stations to the Dongfeng General Hospital and calculating their 24-h arithmetic mean values, except for O_3, which was calculated as daily 8-h maximum concentrations.

2.5. Statistical analyses

Spearman's rank correlation analyses were used to evaluate correlations between any two meteorological variables and air pollutants during the study period. The delayed effect of ambient temperature on lung function was assessed by applying lag structures of 1, 2, 4, and 7 days before the day of physical examination (lag01, lag02, lag04, and lag07). For example, lag01 was calculated as the average temperature on the physical examination day and the previous day [18,19].

Among 19,128 participants (25,511 observations), a generalized additive mixed model (GAMM) with penalized splines and a subject-specific random intercept was constructed to evaluate the linearity of each association between ambient temperature and lung function (FVC, FEV_1, and PEF). We further assess the linearity associations among 6,383 participants with two observations based on GAMM. The core statistical model of GAMM was relying on previous studies [9,20]. In brief, we first established a basic model with the smoothed splines for meteorological variables, which provided a flexible modeling tool for exploring nonlinear and non-monotone patterns between ambient temperature and lung function parameters. Next, we included priori covariates known to be associated with ambient temperature and/or lung function [9,21], including age, gender, height, weight, smoking amount, passive smoking amount, drinking amount, current asthma, current CHD, education degree, physical activity, season, and time of follow-up. Air pollutants were also introduced to control their interference with ambient temperature and/or lung function [22]. The core GAMM equation is:

$$\begin{aligned} Y = \alpha + \beta \times s(temperature, df + 1) + s(RH, df + 1) + covariates + AP \\ random = subject \end{aligned} \tag{3}$$

Where Y is the lung function parameters (FVC, FEV_1, and PEF); α is the intercept; β is the regression coefficient; s is the smoother based on penalized splines; *temperature* is the moving average temperature (lag01, lag02, lag04, and lag07); *RH* is previous-day relative humidity [9]. Based on the Akaike information criterion and previous studies [4,9], the three degrees of freedom (*df*) are chosen for *temperature* and *RH*; *AP* is previous-day air pollutants [9]. To avoid collinearity, only air pollutants having an absolute value of less than 0.5 for Spearman's rank correlation coefficient with *temperature* or *RH* were included in the model [23]. *random* = *subject* indicates that the subject was included in the model as a random intercept.

Linear associations of ambient temperature with the lung function parameters (FVC, FEV_1, and PEF) were investigated by linear mixed-effects models (LMEM) with a subject-specific random intercept. Stratified analyses were performed to investigate the associations in different subgroups of individual characteristics, such as gender (male or female), age (<65 years or ≥65 years), BMI (<24.0 kg/m^2 or ≥ 24.0 kg/m^2), BSA (<1.65 m^2 or ≥ 1.65 m^2), and lifestyles like smoking status (yes or no), drinking status (yes or no), and physical activity (yes or no). Then, we further explored the effect modification of the above characteristics on the associations by including an interaction term for ambient temperature multiplied by each character in the LMEM.

Sensitivity analyses were performed by excluding smokers (current and former), drinkers (current and former), and observations with asthma or CHD, and the robustness of the results was also assessed by replacing average RH with minimum RH and including each air pollutant separately in the models.

All statistical analyses were performed in R software (v4.1.1) with the GAMM fitted by the “mgcv” package and the LMEM fitted by the “lmerTest” package. A two-sided P value less than 0.05 was considered statistically significant.

3. Results

3.1. Participants' characteristics

A total of 19,128 participants with 25,511 observations (first follow-up: 13,818 [54.16%]; second follow-up: 11,693 [45.84%]) were incorporated in the analyses. All observations included 10,604 males (41.57%) and 14,907 females (58.43%). Most of the observations were never smokers (19,367 [75.92%]), while the current (3,537 [13.86%]) and former smokers (2,607 [10.22%]) were relative minorities. Similarly, most of the observations were never drinkers (18,513 [72.57%]) and exercisers (23,205 [90.96%]), whereas the current and former drinkers and no-exercisers account for 22.21% (5,666), 5.22% (1,332), and 9.04% (2,306), respectively. The mean (standard deviation, SD) age of participants from the two follow-ups was 64.57 (8.21) years and 65.80 (9.11) years, BMI was 24.33 (3.27) kg/m^2 and 24.91 (3.44) kg/m^2, and BSA was 1.66 (0.16) m^2 and 1.67 (0.16) m^2, respectively. The mean (SD) FVC of participants from the two follow-ups was 2,426.71 (695.37) mL and 2,641.62 (668.86) mL, FEV_1 was 2,090.08 (591.49) mL and 2,059.41 (533.94) mL, and PEF was 4,356.32 (1,892.93) mL/s and 5,263.17 (1,807.68) mL/s, respectively (Table 1).

3.2. Meteorological factors and air pollutants distribution and Spearman’s rank correlation coefficients

As shown in Table 2, the median (interquartile range, IQR) ambient temperature during the study period was 22.7 °C (16.5 °C, 27.5 °C) and the range was from −0.4 °C to 32.7 °C. The median (IQR) value of RH was 73.0% (64.0%, 81.0%), and median concentrations (IQR) of $PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, and O_3 were 39.0 (27.0, 56.0) μg/m^3, 53.0 (40.0, 76.0) μg/m^3, 33.0 (21.0, 44.0) μg/m^3, 16.0 (13.0, 24.0) μg/m^3, 0.7 (0.5, 0.9) mg/m^3, and 72.0 (49.0, 95.0) μg/m^3, respectively.

In the Spearman's rank analyses, we found a high correlation ($|r| \geq 0.7$ and $P < 0.05$) between $PM_{2.5}$ and PM_{10}, and moderate correlations ($0.7 > |r| \geq 0.4$ and $P < 0.05$) between temperature and PM_{10}/NO_2/CO/O_3, RH and O_3, $PM_{2.5}$ and NO_2, PM_{10} and NO_2/CO, and NO_2 and SO_2. Of note, significant negative correlations (from −0.13 to −0.62, all $P < 0.05$) exist among temperature and all other factors (except for O_3) (Fig. S1). Consequently, $PM_{2.5}$, SO_2, and CO were included in the ensuing analyses to control their interference with ambient temperature and/or lung function.

3.3. Associations between short-term exposure to ambient temperature and lung function

The penalized splines between ambient temperature and lung function parameters (FVC, FEV_1, and PEF) at lag04 are displayed in Fig. 1. The results of the splines exhibit inverse J-shaped exposure-response relationships, which are characterized by steeper and more positive associations of ambient temperature with FVC, FEV_1, and PEF in the lower temperature zone, while slightly negative associations are observed in the higher temperature zone. As can be observed from the splines at lag04, the optimum temperatures for FVC, FEV_1, and PEF are 16.5 °C, 18.7 °C, and 16.2 °C, respectively. The other splines (lag01, lag02, and lag07) with the optimum temperatures appeared similar and are illustrated in Fig. S2. The results of 6,383 participants with two observations are shown in Fig. 2. We observed similar inverted J-shaped associations for different lag days, with similar optimum temperatures for FVC, FEV_1, and PEF.

Temperatures below or above and equal to the optimum temperatures were separately defined as low temperature or high temperature. The associations of low and high temperatures at lag04 with lung function are depicted in Fig. 3 and the results at lag01, lag02, and lag07 are shown in Table S1. Each 1 °C increase at lag04 was associated with FVC, FEV_1, and PEF increased by 14.07 mL (95% confidence intervals [CI]: 6.40, 21.74), 9.78 mL (5.75, 13.82), and 62.72 mL/s (36.69, 88.74) in low temperature, whereas decreased by 5.72 mL (−8.47, −2.98), 2.01 mL (−4.65, 0.64), and 11.64 mL/s (−20.30, −2.98) in high temperature, respectively (Fig. 3).

3.4. The estimated alteration of lung function related to short-term ambient temperature exposure at lag04 in different groups

We then investigated the potential modifying effects of individual characteristics (gender, age, BMI, and BSA) and lifestyle factors (smoking, drinking, and physical activity) on the associations between ambient

Table 1
Characteristics of the study participants.

Characteristic	All	First follow-up	Second follow-up
Observations	25,511 (100)	13,818 (54.16)	11,693 (45.84)
Gender			
Males	10,604 (41.57)	5,877 (42.53)	4,727 (40.43)
Females	14,907 (58.43)	7,941 (57.47)	6,966 (59.57)
Age, years	65.13 ± 8.65	64.57 ± 8.21	65.80 ± 9.11
Age groups			
$<$65 years	12,476 (48.90)	7,113 (51.48)	5,363 (45.87)
$\geq$65 years	13,035 (51.10)	6,705 (48.52)	6,330 (54.13)
Height, cm	159.77 ± 7.70	160.00 ± 7.77	159.51 ± 7.62
Weight, kg	62.92 ± 10.43	62.42 ± 10.23	63.51 ± 10.62
BMI, kg/m^2	24.60 ± 3.36	24.33 ± 3.27	24.91 ± 3.44
BMI groups			
$<$24 kgm^2	11,533 (45.21)	6,662 (48.21)	4,871 (41.66)
$\geq$24 kg/m^2	13,978 (54.79)	7,156 (51.79)	6,822 (58.34)
BSA, m^2	1.67 ± 0.16	1.66 ± 0.16	1.67 ± 0.16
BSA groups			
$<$1.65 m^2	12,281 (48.14)	6,787 (49.12)	5,494 (46.99)
$\geq$1.65 m^2	13,230 (51.86)	7,031 (50.88)	6,199 (53.01)
Smoking status			
Current smoking	3,537 (13.86)	2,102 (15.21)	1,435 (12.27)
Former smoking	2,607 (10.22)	1,624 (11.75)	983 (8.41)
Never smoking	19,367 (75.92)	10,092 (73.04)	9,275 (79.32)
Passive smoking status			
Yes	7,400 (29.01)	4,305 (31.16)	3,095 (26.47)
No	18,111 (70.99)	9,513 (68.84)	8,598 (73.53)
Smoking amount, pack-years	7.02 ± 16.44	7.77 ± 16.92	6.13 ± 15.81
Passive smoking amount, hours/week·year	24.37 ± 85.29	29.64 ± 92.88	18.14 ± 74.86
Drinking status			
Current drinking	5,666 (22.21)	3,435 (24.86)	2,231 (19.08)
Former drinking	1,332 (5.22)	785 (5.68)	547 (4.68)
Never drinking	18,513 (72.57)	9,598 (69.46)	8,915 (76.24)
Drinking amount, times/week·year	43.85 ± 102.16	44.58 ± 99.39	43.00 ± 105.33
Physical activity			
Yes	23,205 (90.96)	12,462 (90.19)	10,743 (91.88)
No	2,306 (9.04)	1,356 (9.81)	950 (8.12)
Education degree			
Primary school or illiteracy	4,948 (19.40)	2,157 (20.34)	2,791 (18.72)
Middle school	9,349 (36.65)	3,996 (37.68)	5,353 (35.91)
High school	7,911 (31.01)	2,657 (25.06)	5,254 (35.25)
University or college or higher	3,303 (12.95)	1,794 (16.92)	1,509 (10.12)
Asthma			
Yes	651 (2.55)	355 (2.57)	296 (2.53)
No	24,860 (97.45)	13,463 (97.43)	11,397 (97.47)
CHD			
Yes	4,291 (16.82)	2,158 (15.62)	2,133 (18.24)
No	21,220 (83.18)	11,660 (84.38)	9,560 (81.76)
FVC, mL	2,525.21 ± 691.67	2,426.71 ± 695.37	2,641.62 ± 668.86
FEV$_1$, mL	2,076.02 ± 566.03	2,090.08 ± 591.49	2,059.41 ± 533.94
PEF, mL/s	4,771.98 ± 1,908.57	4,356.32 ± 1,892.93	5,263.17 ± 1,807.68

Results are expressed as mean values ± standard deviation (SD) or number (percentage). BMI, body mass index; BSA, body surface area; CHD, coronary heart disease; FVC, forced vital capacity; FEV1, forced expiratory volume in 1 sone second; PEF, peak expiratory flow.

temperature (lag04) and lung function parameters (FVC, FEV$_1$, and PEF) (Table 3). The results revealed that the low-temperature effect on FVC was stronger in individuals aged $<$65 years (18.09 mL [95% CI: 8.00, 28.18]) compared to observations aged $\geq$65 years (6.98 mL [$-$4.63, 18.59]) ($P_{\text{modification}} < 0.05$). Additionally, the high-temperature effects on lung function were stronger in females, individuals with BMI $<$

Table 2
Summary statistics of meteorological factors and air pollutants.

Variables	Mean	SD	Minimum	P25	Median	P75	Maximum
Temperature (°C)	21.5	7.4	$-$0.4	16.5	22.7	27.5	32.7
RH (%)	73.0	13.2	28.0	64.0	73.0	81.0	99.0
PM$_{2.5}$ (μg/m^3)	45.8	26.5	11.0	27.0	39.0	56.0	207.0
PM$_{10}$ (μg/m^3)	62.0	34.7	16.0	40.0	53.0	76.0	360.0
NO$_2$ (μg/m^3)	34.5	16.6	6.0	21.0	33.0	44.0	93.0
SO$_2$ (μg/m^3)	20.4	13.4	2.0	13.0	16.0	24.0	150.0
CO (mg/m^3)	0.7	0.3	0.3	0.5	0.7	0.9	1.8
O$_3$ (μg/m^3)	74.5	32.5	13.0	49.0	72.0	95.0	169.0

SD, standard deviation; P25, 25th percentile; P75, 75th percentile; RH, relative humidity; PM$_{2.5}$, fine particulate matter; PM$_{10}$, inhalable particulate matter; NO$_2$, nitrogen dioxide; SO$_2$, sulfur dioxide; CO, carbon monoxide; O$_3$, ozone.

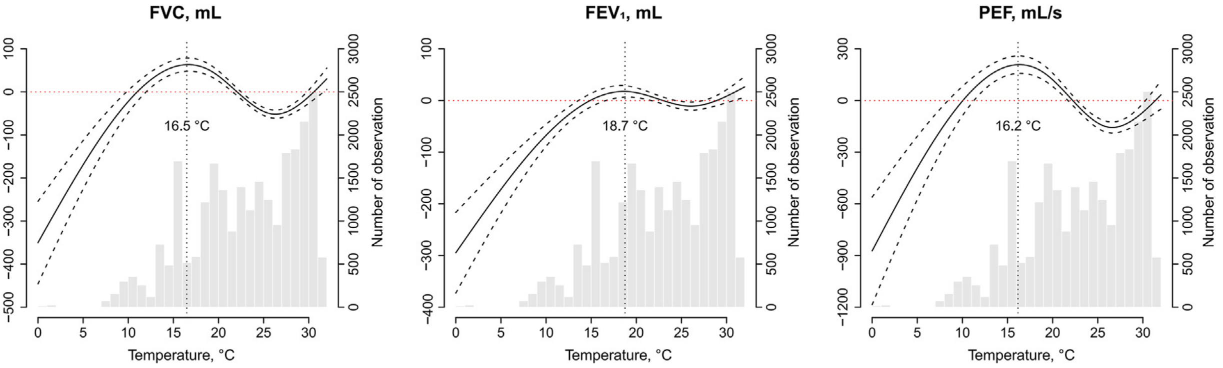

Fig. 1. The association between 4-day average temperature (lag04) and lung function.

24.0 kg/m^2 and BSA < 1.65 m^2, current smokers, drinkers, and non-exercisers (all $P_{\text{modification}} < 0.05$).

3.5. Sensitivity analyses

The sensitivity analyses of the association between ambient temperature and lung function are presented in Table S2. The results remained stable after excluding observations with asthma or CHD and adjusting for daily minimum RH and air pollutants ($PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, and O_3), respectively.

4. Discussion

This study evaluated the short-term temperature effects on lung function in middle-aged and elderly Chinese and observed a significant association between non-optimal temperatures and lung function reduction. Additionally, the effects of non-optimal temperatures on lung function could be modified by gender, age, BMI, BSA, smoking, drinking, and physical activity. Specifically, the low-temperature effects were more pronounced in participants with age < 65 years, and the high-temperature effects were more pronounced in females, individuals with BMI < 24.0 kg/m^2 and BSA < 1.65 m^2, current smokers, drinkers, and non-exercisers.

Although previous studies have recorded the short-term temperature effect on lung function among middle-aged and elderly people, the results were still controversial. A cohort study covering 5,896 middle-aged and elderly people (average age: 50.4 ± 13.8 years) in the United States observed a 5.5 °C increase in the ambient temperature associated with a decrease of 19.8 mL (95% CI: −33.5, −6.1) and 16.2 mL (−31.6, −0.9) in FEV_1 and FVC, respectively [9]. Similarly, a study [24] that enrolled 1,103 American older males (average age: 69.1 ± 7.2 years) found 0.70% (95% CI: −1.24%, −0.20%) decrease in FVC for every 5 °C increase in ambient temperature. However, another American study [25] containing 75 participants with COPD (average age: 69 ± 8 years) discovered that a 10 °F (~5.5 °C) decrease in the ambient temperature was associated with 38 mL (95% CI: −60, −15) and 26 mL (−50, −3) decrease in the morning and evening FEV_1, respectively. In a study including 69 American participants with COPD (average age: 69 ± 8 years), McCormack et al. [11]

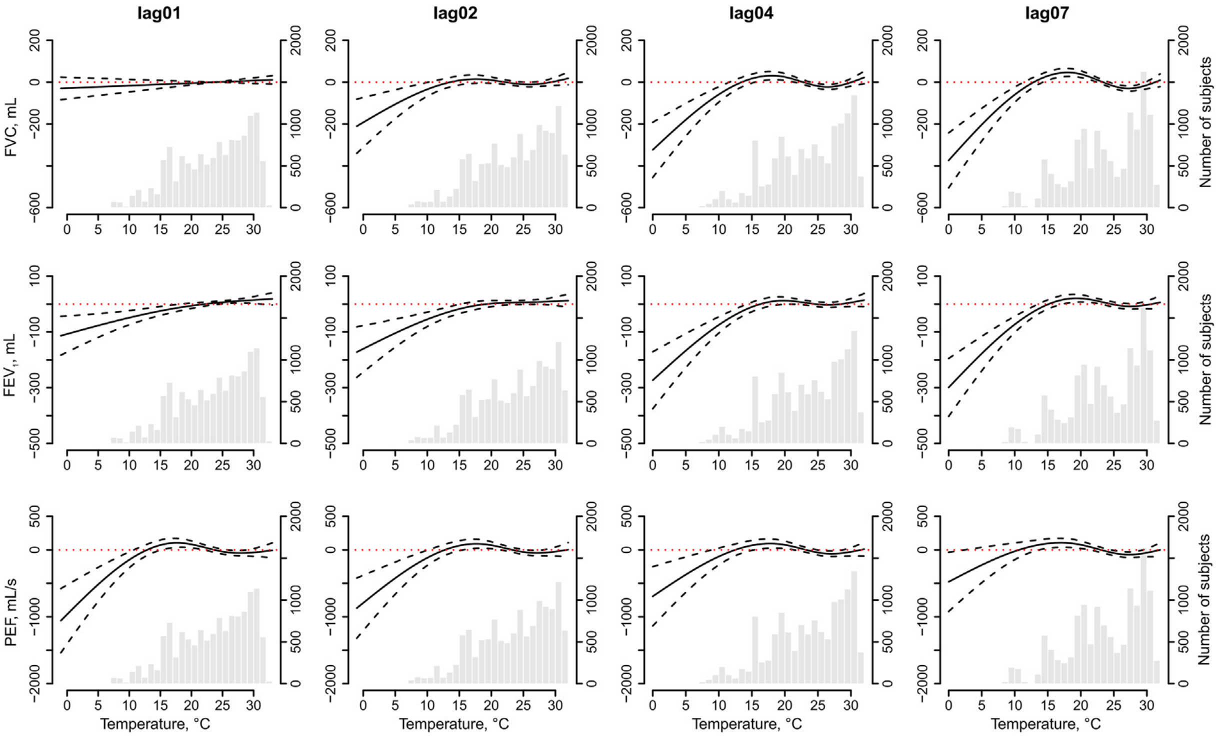

Fig. 2. The associations between ambient temperature and lung function for different lag days among 6,383 subjects with two observations.

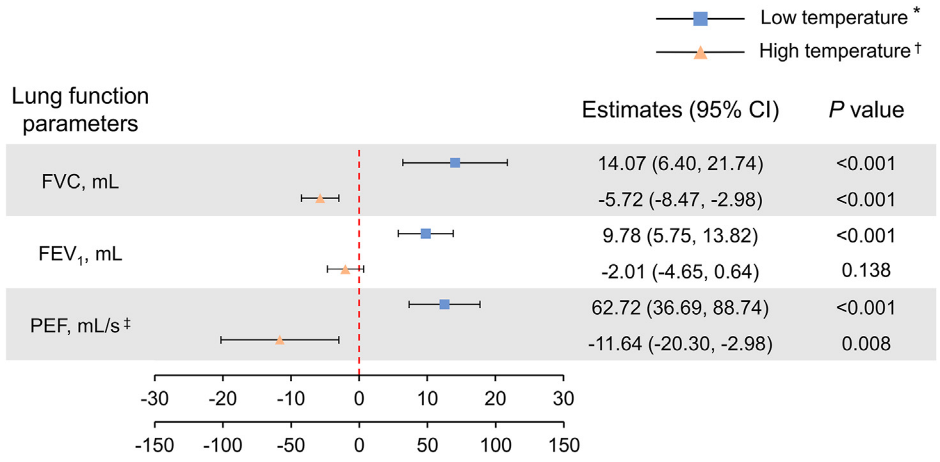

Fig. 3. The associations between low and high temperatures at lag04 and lung function. * Temperatures below the optimum temperatures for FVC (16.5 °C), FEV_1 (18.7 °C), and PEF (16.2 °C) at lag04 were defined as low temperature; † Temperatures above and equal to the optimum temperatures for FVC (16.5 °C), FEV_1 (18.7 °C), and PEF (16.2 °C) at lag04 were defined as high temperature; ‡ The result of the association between low temperature and PEF corresponded to the coordinate axis below. CI, confidence intervals.

did not catch any temperature effect on FEV_1, and similar results were also reported in a study of 30 former smokers with COPD (average age: 71.1 ± 8.4 years) from Boston, the United States [12]. The above inconsistencies may be attributed to the differences in sample size, study design, or health status (healthy or COPD) of the participants. In the present study, we confirmed the mixed results in a relatively larger population (n = 19,128) and found that both low and high temperatures were significantly associated with FVC, FEV_1, and PEF reduction, consistent with the findings of Lei et al [10]. They enrolled 4,992 adult asthmatic patients (average age: 45.4 ± 13.6 years) from China and found inversely J-shaped associations for ambient temperature and FVC, FEV_1, and PEF. We observed similar inverted J-shaped associations among 14,111 participants aged between 35 and 75 from Fuqing cohort (unpublished data), and the optimal temperatures (referent temperature) were all around 16 °C for FVC, FEV_1, and PEF, respectively. Our results also support previous studies [5,26,27] that observed the J-, V-, or U-shaped associations of ambient temperature with respiratory morbidity or mortality, indicating that both low and high temperatures could induce respiratory damage or even death. It is notable, however, that the splines derived from the GAMM analyses suggested that FVC and PEF again increased with increasing temperature after ~26 °C. This was probably because older people might have intentionally reduced outdoor activities to avoid exposure during extremely hot weather, and taken some protective measures like active ventilation, air-conditioning

Table 3
The estimates of lung function per 1 °C increase at lag04 in different groups.

Characters	FVC, mL (95% CI)		FEV_1, mL (95% CI)		PEF, mL (95% CI)	
	Low temperature[a]	High temperature[b]	Low temperature	High temperature	Low temperature	High temperature
Gender						
Males	**20.79 (7.54, 34.04)**	−2.60 (−7.69, 2.50)	14.66 (7.35, 21.97)	−0.23 (−5.26, 4.80)	59.95 (13.71, 106.18)	−11.11 (−27.79, 5.56)
Females	**8.71 (0.02, 17.40)**	**−7.93 (-10.99, -4.86)**	5.88 (1.53, 10.24)	−3.48 (−6.40, −0.56)	65.02 (36.59, 93.46)	−13.34 (−22.54, −4.13)
$P_{\text{modification}}$[c]	0.993	**<0.001**	0.568	**0.002**	0.445	**<0.001**
Age						
<65 years	18.09 (8.00, 28.18)	−8.53 (−12.43, −4.64)	8.63 (3.30, 13.96)	−3.89 (−7.61, −0.17)	64.39 (30.78, 98.01)	−7.35 (−19.20, 4.50)
≥65 years	6.98 (−4.63, 18.59)	−4.36 (−8.33, −0.39)	11.11 (5.00, 17.22)	−0.72 (−4.61, 3.18)	59.03 (19.10, 98.96)	−15.62 (−28.30, −2.95)
$P_{\text{modification}}$	**0.029**	0.767	0.600	0.578	0.428	0.416
BMI						
<24 kg/m^2	14.29 (1.01, 27.58)	−7.52 (−11.75, −3.30)	9.83 (3.08, 16.59)	−3.03 (−6.99, 0.93)	64.17 (21.48, 106.85)	−6.80 (−19.38, 5.79)
≥24 kg/m^2	14.93 (5.56, 24.30)	−5.13 (−8.79, −1.46)	10.11 (5.06, 15.16)	−0.41 (−4.04, 3.22)	61.76 (28.79, 94.74)	−14.42 (−26.44, −2.40)
$P_{\text{modification}}$	0.650	**< 0.001**	0.875	**0.007**	0.543	0.239
BSA						
<1.65 m^2	6.72 (−4.34, 17.79)	−6.41 (−10.02, −2.80)	6.53 (0.99, 12.08)	−2.97 (−6.44, 0.50)	82.27 (46.32, 118.22)	−12.94 (−23.83, −2.05)
≥1.65 m^2	18.84 (8.41, 29.27)	−5.34 (−9.49, −1.18)	11.86 (6.19, 17.53)	−0.96 (−5.00, 3.07)	50.13 (14.00, 86.26)	−11.21 (−24.63, 2.21)
$P_{\text{modification}}$	0.653	**0.002**	0.795	**0.005**	0.175	**0.001**
Smoking status						
Yes	26.00 (8.29, 43.70)	−4.58 (−11.27, 2.10)	13.06 (3.27, 22.85)	−2.45 (−9.02, 4.13)	72.38 (8.78, 135.98)	−11.99 (−33.62, 9.63)
No	11.14 (2.70, 19.58)	−6.20 (−9.18, −3.22)	9.12 (4.77, 13.46)	−2.03 (−4.90, 0.85)	60.85 (33.01, 88.68)	−11.15 (−20.41, −1.89)
$P_{\text{modification}}$	0.161	**0.008**	0.410	**0.014**	0.194	**< 0.001**
Drinking status						
Yes	23.77 (7.79, 39.76)	−5.34 (−11.18, 0.51)	9.65 (0.72, 18.57)	−2.72 (−8.31, 2.87)	73.12 (16.38, 129.86)	−17.32 (−36.20, 1.55)
No	10.26 (1.51, 19.01)	−6.63 (−9.76, −3.49)	9.61 (5.13, 14.09)	−1.70 (−4.76, 1.37)	59.36 (30.17, 88.54)	−8.55 (−18.24, 1.14)
$P_{\text{modification}}$	0.523	**0.013**	0.787	0.287	0.704	**0.025**
Physical activity						
Yes	12.84 (4.80, 20.88)	−5.44 (−8.32, −2.57)	9.84 (5.60, 14.09)	−1.90 (−4.67, 0.88)	60.42 (33.08, 87.76)	−14.12 (−23.18, −5.06)
No	28.08 (1.33, 54.83)	−9.74 (−19.92, 0.45)	10.23 (−2.15, 22.61)	−0.29 (−10.43, 9.86)	69.77 (−18.03, 157.57)	23.21 (−7.58, 54.00)
$P_{\text{modification}}$	0.857	**0.045**	0.614	0.163	0.782	0.479

[a] Temperatures below the optimum temperatures for FVC (16.5 °C), FEV_1 (18.7 °C), and PEF (16.2 °C) at lag04 were defined as low temperature.
[b] Temperatures above and equal to the optimum temperatures for FVC (16.5 °C), FEV_1 (18.7 °C), and PEF (16.2 °C) at lag04 were defined as high temperature.
[c] Effect modification was calculated by including an interaction term of ambient temperature (lag04) multiplied by each character in the model.

cooling, and other temperature reduction approaches [28]. In addition, about 38% of the participants were visited at temperatures above ~26 °C, which might be another possible cause.

The potential mechanisms underlying the effects of low and high temperatures on lung function remain to be fully understood. Published works proposed that inflammation might play one of the major roles in the process [29–32]. For example, non-optimal temperature exposure during early pregnancy was found to elevate the risk of childhood pneumonia, suggesting the role of inflammation [33]. Our previous study enrolled forty-three (20 males, 23 females) healthy non-obese volunteers [34], and found that low temperature exposure could elicit systemic inflammation, which may be involved in low temperature-related lung function reduction. Inflammation is a host-protective response that copes with tissue injury or infection, and a considerable number of inflammatory factors and cells are associated with lung function decline [35,36]. *In vivo* and *in vitro* studies suggested that low temperature could lead to airway inflammation by stimulating monocyte chemoattractant protein-1 (MCP-1), interleukin (IL)-1, IL-6, IL-8, and tumor necrosis factor (TNF)-α, and enhancing the expression of P-selectin and β2 integrin as well as leukocyte infiltration [37–40]. Also, macrophages and nuclear factor (NF)-κB activation and TNF-α, IL-6, and IL-8 overexpression in lung tissue were observed in deep hypothermic circulatory arrest surgery [41]. In addition, high-temperature exposure activates NF-κB and elevates the levels of various inflammatory factors in bronchoalveolar lavage fluid, along with increases in the wet/dry weight ratio, water content, and pulmonary vascular permeability [42,43].

Apart from inflammation, oxidative stress may also play an important part in the respiratory system damage induced by ambient temperature. Our previous study also suggests that oxidative stress may be involved in low temperature-related lung function reduction [34]. Previous toxicological studies indicated that low temperatures could promote the release of reactive oxygen species (ROS) and nitric oxide (NO) from A549 cells, namely human adenocarcinoma alveolar basal epithelial cells [44,45]. Besides, activated ROS and enhancement of autophagy in lung tissue were observed in cold perfusion of lung transplants [30]. Whereas under hot conditions, previous studies [36,46–48] have proven that high temperature could stimulate the activity of inducible NO synthase and further catalyze the synthesis of a large amount of NO, causing increased release of MCP-1 in lung tissue.

This study revealed that the association between ambient temperature and lung function could be modified by several individual factors. We found that the high-temperature effect was more pronounced in females, aligning with findings from previous studies [49,50]. Nevertheless, discrepancies exist among the reports of temperature effects on gender. For example, one study [4] included 184 Chinese cities that reported no differences in the sensitivity of males and females to cold or hot environments. In the present study, the low temperature had a greater impact on lung function among the younger than the older, although previous studies have indicated that the older rather than the younger are more susceptible to the cold environment [5]. Two reasons may contribute to this phenomenon. First, older individuals have lower lung compliance capacity, and their lung adjustment ability is lower than that of younger individuals, making it more difficult to accommodate drastic temperature changes. Second, the immune function of the older is weaker than the younger, and most of them suffer from various basic diseases, such as hypertension or diabetes [51], which may conceal the real effect of ambient temperature on lung function.

BMI is strongly associated with lung function. Our findings suggested that individuals with low BMI instead of high BMI were more vulnerable to high temperature, demonstrating that low BMI might enhance the temperature effect on lung function. A study [52] involving 16,171 Americans pointed out that within a certain range, the lower the BMI, the lower the lung function levels. Our previous study [35] also observed significantly lower levels of FVC and FEV_1 in the low BMI group (<21.63 kg/m^2) compared with the high BMI group (23.76–26.09 kg/m^2). Moreover, we found that the high-temperature effect on lung function was more pronounced in those with low BSA (<1.65 m^2). In the heat dissipation process, the core body temperature reaches the skin via blood circulation and then diffuses into the outer environment by peripheral blood vessel expansion, sweat evaporation, etc., which means that the greater the BSA, the faster the heat dissipation [53]. Our results indicate that people with lower BSA are more susceptible to the adverse effects of high temperatures due to the slower heat dissipation in hot environments, resulting in higher core body temperatures.

Lifestyles are also significant factors that influence the association of ambient temperature with lung function. Our results present that high temperature had a stronger effect on lung function in smokers compared with nonsmokers. Cigarette smoking is a well-recognized risk factor for lung function decline [54]. A range of chemical components in cigarette smoke, such as nicotine, tar, and polycyclic aromatic hydrocarbons, induce lung function decline by stimulating the expression of multiple inflammatory factors and activating peroxidation in lung tissue [55]. We found that the high-temperature effect on lung function was more pronounced in drinkers than nondrinkers, indicating that drinkers are more resilient to the changes in ambient temperature. Multiple previous epidemiological studies [56,57] have indicated that moderate alcohol intake has been linked to improved lung function. And our previous study [13] also found that FEV_1 and FVC separately increased by 70.03 mL (95% CI: 46.72, 93.35) and 74.92 ml (47.47, 102.38) in moderate drinkers compared to non-drinkers. Furthermore, we discovered that the association between high temperatures and lung function was stronger in non-exercisers than in exercisers. Regular physical activity offers a variety of health benefits, including reduced body weight and improved cardiorespiratory fitness [58]. Regular exercisers could adapt to changes in ambient temperature by regulating cardiac output as well as lung capacity [59].

This study has several strengths. First, our study is a prospectively designed and relatively large-sample-sized cohort, which fills part of the research gap in China and provides clues for future research. Second, we observed a rather large temperature range (−0.4 °C to 32.7 °C), and excluded participants with any diseases affecting lung function. Moreover, by controlling potential confounding variables in all analyses, we ensured the results' trustworthiness. However, several potential limitations should be acknowledged. First, this study was limited to middle-aged and elderly retired workers in China, and our estimates should be interpreted with caution when extending to a broader population. Second, we did not adjust the time for indoor activities, indoor temperature, and air pollution. As we know, people spend most of their time indoors, and the usage of interior temperature management equipment, such as fans, heaters, and air conditioners, as well as indoor air pollution, may obscure the true influence of ambient temperature. Nevertheless, we still observed significant temperature effects on lung function, which implies that the health effect of non-optimal temperatures should not be overlooked. Third, because meteorological and air pollutants data are sourced from monitoring stations, the exposure measurement errors should be acknowledged, which might cause the effects of ambient temperature on health to be overestimated or underestimated.

5. Conclusions

Our findings reveal inversely J-shaped associations between ambient temperature and lung function parameters among the middle-aged and elderly population in China. Furthermore, we identified that several individual characteristics (gender, age, BMI, and BSA) and lifestyles (smoking, drinking, and physical activity) have significant effect modifications on these associations. These findings underscore the need to strengthen public awareness of the hazardous effects of non-optimal temperatures and improve public health systems' ability to mitigate the negative effects of non-optimal temperatures on health, especially in light of global climate change.

CRediT authorship contribution statement

W.H.Q.: conceptualization, methodology, formal analysis, investigation, writing–original draft, writing–review & editing, funding acquisition; B.W.: methodology, investigation, writing–review & editing; X.B.F.: methodology, writing–review & editing; H.H., L.Y.F., Z.Y., X.Q.N., G.M., W.L. and D.M.W.: investigation, writing–review & editing; M.Z.: investigation, data curation, writing–review & editing; W.H.C.: conceptualization, investigation, resources, data curation, writing–review & editing, supervision, funding acquisition.

Declaration of competing interests

The authors have no relevant financial or non-financial interests to disclose.

Acknowledgments

The authors thank all the participants from the Dongfeng-Tongji cohort and the staff of the Dongfeng Motor Corporation at Dongfeng General Hospital for their contributions to the study. This work was supported by the National Key Research and Development Program of China (2016YFC1303903); the Major Research Program of the National Natural Science Foundation of China (91843302); and the National Natural Science Foundation of China (82304086). The funders did not play any role in study design; in the collection, analysis, and interpretation of data; in the writing of the report; nor the preparation, review, or approval of the manuscript.

Appendix B. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.eehl.2024.01.008.

References

[1] A.J. Mcmichael, R.E. Woodruff, S. Hales, Climate change and human health: Present and future risks, Lancet 367 (9513) (2006) 859–869, https://doi.org/10.1016/S0140-6736(06)68079-3.

[2] M. Springmann, D. Mason-D'Croz, S. Robinson, T. Garnett, H.C.J. Godfray, D. Gollin, et al., Global and regional health effects of future food production under climate change: A modelling study, Lancet 387 (10031) (2016) 1937–1946, https://doi.org/10.1016/S0140-6736(15)01156-3.

[3] P. Lu, G. Xia, Q. Zhao, R. Xu, S. Li, Y. Guo, Temporal trends of the association between ambient temperature and hospitalisations for cardiovascular diseases in queensland, Australia from 1995 to 2016: A time-stratified case-crossover study, PLoS Med. 17 (7) (2020). https://doi.org/10.1371/journal.pmed.1003176.

[4] Y. Tian, H. Liu, Y. Si, Y. Cao, J. Song, M. Li, et al., Association between temperature variability and daily hospital admissions for cause-specific cardiovascular disease in urban China: a national time-series study, PLoS Med. 16 (1) (2019). https://doi.org/10.1371/journal.pmed.1002738.

[5] R. Chen, P. Yin, L. Wang, C. Liu, Y. Niu, W. Wang, et al., Association between ambient temperature and mortality risk and burden: Time series study in 272 main Chinese cities, BMJ Br. Med. J. (Clin. Res. Ed.) 363 (2018). https://doi.org/10.1136/bmj.k4306.

[6] K. Zhang, L. Guo, Q. Wei, Q. Song, J. Liu, J. Niu, et al., Copd rat model is more susceptible to cold stress and $PM_{2.5}$ exposure and the underlying mechanism, Environ. Pollut. 241 (2018) 26–34. https://doi.org/10.1016/j.envpol.2018.05.034.

[7] G. Mu, M. Zhou, B. Wang, L. Cao, S. Yang, W. Qiu, et al., Personal $PM_{2.5}$ exposure and lung function: potential mediating role of systematic inflammation and oxidative damage in urban adults from the general population, Sci. Total Environ. 755 (Pt 1) (2021). https://doi.org/10.1016/j.scitotenv.2020.142522.

[8] B. Wang, X. Wang, S. Yang, M. Cheng, Y. Zhou, M. Zhou, et al., Acrylamide exposure and pulmonary function reduction in general population: The mediating effect of systemic inflammation, Sci. Total Environ. 778 (2021) 146304, https://doi.org/10.1016/j.scitotenv.2021.146304.

[9] M.B. Rice, W. Li, E.H. Wilker, D.R. Gold, J. Schwartz, A. Zanobetti, et al., Association of outdoor temperature with lung function in a temperate climate, Eur. Respir. J. 53 (1) (2019). https://doi.org/10.1183/13993003.00612-2018.

[10] J. Lei, L. Peng, T. Yang, S. Huang, Y. Zhu, Y. Gao, et al., Non-optimum ambient temperature may decrease pulmonary function: A longitudinal study with intensively repeated measurements among asthmatic adult patients in 25 Chinese cities, Environ. Int. 164 (2022) 107283, https://doi.org/10.1016/j.envint.2022.107283.

[11] M.C. Mccormack, A.J. Belli, D. Waugh, E.C. Matsui, R.D. Peng, D.A.L. Williams, et al., Respiratory effects of indoor heat and the interaction with air pollution in chronic obstructive pulmonary disease, Ann. Am. Thoracic Society 13 (12) (2016) 2125–2131. https://doi.org/10.1513/AnnalsATS.201605-329OC.

[12] C. Scheerens, L. Nurhussien, A. Aglan, A.J. Synn, B.A. Coull, P. Koutrakis, et al., The impact of personal and outdoor temperature exposure during cold and warm seasons on lung function and respiratory symptoms in copd, Erj Open Res 8 (1) (2022) 574. https://doi.org/10.1183/23120541.00574-2021.

[13] D. Wang, L. Cao, M. Zhou, J. Ma, B. Wang, J. Yuan, et al., Alcohol intake, beverage type, and lung function: A multicohort study of Chinese adults, Ann. N. Y. Acad. Sci. 1511 (1) (2022) 164–172. https://doi.org/10.1111/nyas.14744.

[14] F. Wang, J. Zhu, P. Yao, X. Li, M. He, Y. Liu, et al., Cohort profile: The Dongfeng-Tongji cohort study of retired workers, Int. J. Epidemiol. 42 (3) (2013) 731–740. https://doi.org/10.1093/ije/dys053.

[15] Y. Wen, D. Wang, M. Zhou, Y. Zhou, Y. Guo, W. Chen, Potential effects of lung function reduction on health-related quality of life, Int. J. Environ. Res. Publ. Health 16 (2) (2019) 260. https://doi.org/10.3390/ijerph16020260.

[16] H. He, N. Wan, Z. Li, Z. Zhang, Z. Gao, Q. Liu, et al., Short-term effects of exposure to ambient $PM_{2.5}$ and its components on hospital admissions for threatened and spontaneous abortions: a multicity case-crossover study in China, Chemosphere 350 (2024) 141057. https://doi.org/10.1016/j.chemosphere.2023.141057.

[17] W. Qiu, H. He, T. Xu, C. Jia, W. Li, The air quality changes and related mortality benefits during the coronavirus disease 2019 pandemic in China: Results from a nationwide forecasting study, J. Clean. Prod. 308 (2021) 127327, https://doi.org/10.1016/j.jclepro.2021.127327.

[18] W. Qiu, H. He, B. Wang, D. Wang, G. Mu, T. Xu, et al., Short-term impacts of air pollution on the platelet-lymphocyte ratio and neutrophil-lymphocyte ratio among urban adults in China, J. Environ. Sci. 125 (2023) 101–111, https://doi.org/10.1016/j.jes.2021.10.022.

[19] W. Qiu, Y. Zhou, H. He, B. Wang, G. Mu, M. Zhou, et al., Short-term effects of air pollution on liver function among urban adults in China, Atmos. Environ. 245 (2021) 118011, https://doi.org/10.1016/j.atmosenv.2020.118011.

[20] S. Li, P.J. Baker, B.B. Jalaludin, G.B. Marks, L.S. Denison, G.M. Williams, Ambient temperature and lung function in children with asthma in Australia, Eur. Respir. J. 43 (4) (2014) 1059–1066. https://doi.org/10.1183/09031936.00079313.

[21] Y. Zhou, H. Sun, J. Xie, Y. Song, Y. Liu, X. Huang, et al., Urinary polycyclic aromatic hydrocarbon metabolites and altered lung function in wuhan, China, Am. J. Respir. Crit. Care Med. 193 (8) (2016) 835–846. https://doi.org/10.1164/rccm.201412-2279OC.

[22] W. Yang, M.B. Johnson, H. Liao, Z. Liu, X. Zheng, C. Lu, Combined effect of preconceptional and prenatal exposure to air pollution and temperature on childhood pneumonia: A case-control study, Environ. Res. 216 (Pt 4) (2023) 114806, https://doi.org/10.1016/j.envres.2022.114806.

[23] M.M. Mukaka, Statistics corner: A guide to appropriate use of correlation coefficient in medical research, Malawi Med. J. 24 (3) (2012) 69–71.

[24] J. Lepeule, A.A. Litonjua, A. Gasparrini, P. Koutrakis, D. Sparrow, P.S. Vokonas, et al., Lung function association with outdoor temperature and relative humidity and its interaction with air pollution in the elderly, Environ. Res. 165 (2018) 110–117, https://doi.org/10.1016/j.envres.2018.03.039.

[25] M.C. Mccormack, L.M. Paulin, C.E. Gummerson, R.D. Peng, G.B. Diette, N.N. Hansel, Colder temperature is associated with increased copd morbidity, Eur. Respir. J. 49 (6) (2017) 1601501. https://doi.org/10.1183/13993003.01501-2016.

[26] Y. Lin, T. Ho, Y. Wang, Mortality risk associated with temperature and prolonged temperature extremes in elderly populations in Taiwan, Environ. Res. 111 (8) (2011) 1156–1163, https://doi.org/10.1016/j.envres.2011.06.008.

[27] Y. Zhao, Z. Huang, S. Wang, J. Hu, J. Xiao, X. Li, et al., Morbidity burden of respiratory diseases attributable to ambient temperature: A case study in a subtropical city in China, Environ. Health 18 (1) (2019) 89. https://doi.org/10.1186/s12940-019-0529-8.

[28] A. Ponjoan, J. Blanch, L. Alves-Cabratosa, R. Martí-Lluch, M. Comas-Cufí, D. Parramon, et al., Effects of extreme temperatures on cardiovascular emergency hospitalizations in a Mediterranean region: A self-controlled case series study, Environ. Health 16 (1) (2017) 32–39. https://doi.org/10.1186/s12940-017-0238-0.

[29] L. Deng, P. Ma, Y. Wu, Y. Ma, X. Yang, Y. Li, et al., High and low temperatures aggravate airway inflammation of asthma: Evidence in a mouse model, Environ. Pollut. 256 (2020) 113433, https://doi.org/10.1016/j.envpol.2019.113433.

[30] H. Lin, S. Dai, W. Liu, X. Lin, B. Yu, S. Chen, et al., Effects of prolonged cold-ischemia on autophagy in the graft lung in a rat orthotopic lung transplantation model, Life Sci 268 (2021) 118820, https://doi.org/10.1016/j.lfs.2020.118820.

[31] Y. Zhang, Y. Lin, X. Li, L. Zhang, W. Pan, H. Zhu, et al., Silica dioxide nanoparticles combined with cold exposure induce stronger systemic inflammatory response, Environ. Sci. Pollut. Res. 24 (1) (2017) 291–298. https://doi.org/10.1007/s11356-016-7649-2.

[32] K. Zhang, L. Guo, Q. Wei, Q. Song, J. Liu, J. Niu, et al., Copd rat model is more susceptible to cold stress and $PM_{2.5}$ exposure and the underlying mechanism, Environ. Pollut. 241 (2018) 26–34. https://doi.org/10.1016/j.envpol.2018.05.034.

[33] C. Lu, W. Yang, M. Lan, B. Li, F. Wang, Effects of intrauterine and postnatal exposure to meteorological factors on childhood pneumonia, Build. Environ. 244 (2023) 110800, https://doi.org/10.1016/j.buildenv.2023.110800.

[34] W. Qiu, H. He, L. Fan, X. Feng, M. Li, C. Dong, et al., Ambient temperature exposure causes lung function impairment: The evidence from controlled temperature study in healthy subjects (ctshs), Int. J. Hyg Environ. Health 252 (2023) 114214, https://doi.org/10.1016/j.ijheh.2023.114214.

[35] H. He, B. Wang, M. Zhou, L. Cao, W. Qiu, G. Mu, et al., Systemic inflammation mediates the associations between abdominal obesity indices and lung function decline in a Chinese general population, Diabetes Metab. Syndr. Obes. 13 (2020) 141–150. https://doi.org/10.2147/DMSO.S229749.

[36] J.T. Liu, B. Luo, X.T. He, L.Y. Li, S.G. Xu, The protective effects of vitamin E on lung injury caused by high temperature and $PM_{2.5}$ in copd rats, Zhongguo Ying Yong Sheng Li Xue Za Zhi 35 (4) (2019) 293–296. https://doi.org/10.12047/j.cjap.5788.2019.061.

[37] M.S. Davis, C.C. Williams, J.H. Meinkoth, J.R. Malayer, C.M. Royer, K.K. Williamson, et al., Influx of neutrophils and persistence of cytokine expression in airways of horses after performing exercise while breathing cold air, Am. J. Vet. Res. 68 (2) (2007) 185–189. https://doi.org/10.2460/ajvr.68.2.185.

[38] K.A. Jetha, S. Egginton, G.B. Nash, Changes in the integrin-mediated adhesion of human neutrophils in the cold and after rewarming, Biorheology 44 (1) (2007) 37–49.

[39] G.B. Nash, K.B. Abbitt, K. Tate, K.A. Jetha, S. Egginton, Changes in the mechanical and adhesive behaviour of human neutrophils on cooling in vitro, Pflügers Archiv 442 (5) (2001) 762–770. https://doi.org/10.1007/s004240100598.

[40] A.S. Sabnis, C.A. Reilly, J.M. Veranth, G.S. Yost, Increased transcription of cytokine genes in human lung epithelial cells through activation of a trpm8 variant by cold temperatures, Am. J. Physiol. Lung Cell Mol. Physiol. 295 (1) (2008) L194–L200. https://doi.org/10.1152/ajplung.00072.2008.

[41] J.H. Zheng, B.T. Gao, Z.M. Jiang, X.Q. Yu, Z.W. Xu, Evaluation of early macrophage activation and nf-kappab activity in pulmonary injury caused by deep hypothermia circulatory arrest: an experimental study, Pediatr. Cardiol. 31 (2) (2010) 215–221. https://doi.org/10.1007/s00246-009-9586-z.

[42] Y. Geng, R. Li, S.X. He, H.H. Yang, Q.T. Deng, X.Y. Shao, et al., Dexmedetomidine attenuates acute lung injury induced by heatstroke and improve outcome, Shock 52 (5) (2019) 532–539. https://doi.org/10.1097/SHK.0000000000001289.

[43] Y.C. Hsieh, R.F. Chen, Y.S. Yeh, M.T. Lin, J.H. Hsieh, S.H. Chen, Kynurenic acid attenuates multiorgan dysfunction in rats after heatstroke, Acta Pharmacol. Sin. 32 (2) (2011) 167–174. https://doi.org/10.1038/aps.2010.191.

[44] W. Sun, Z. Wang, J. Cao, X. Wang, Y. Han, Z. Ma, Enhanced production of nitric oxide in A549 cells through activation of TRPA1 ion channel by cold stress, Nitric Oxide-Biol, Chem 40 (2014) 31–35, https://doi.org/10.1016/j.niox.2014.04.009.

[45] W. Sun, Z. Wang, J. Cao, H. Cui, Z. Ma, Cold stress increases reactive oxygen species formation via trpa1 activation in A549 cells, Cell Stress Chaperones 21 (2) (2016) 367–372, https://doi.org/10.1007/s12192-015-0663-3.

[46] S.S. Chauhan, P. Celi, B.J. Leury, F.R. Dunshea, High dietary selenium and vitamin E supplementation ameliorates the impacts of heat load on oxidative status and acid-base balance in sheep, J. Anim. Sci. 93 (7) (2015) 3342–3354. https://doi.org/10.2527/jas.2014-8731.

[47] N. Mosleh, T. Shomali, F. Nematollahi, Z. Ghahramani, M.S. Ahrari Khafi, F. Namazi, Effect of different periods of chronic heat stress with or without vitamin C supplementation on bone and selected serum parameters of broiler chickens, Avian Pathol. 47 (2) (2018) 197–205. https://doi.org/10.1080/03079457.2017.1401212.

[48] G.Y. Tan, L. Yang, Y.Q. Fu, J.H. Feng, M.H. Zhang, Effects of different acute high ambient temperatures on function of hepatic mitochondrial respiration, antioxidative enzymes, and oxidative injury in broiler chickens, Poultry Sci 89 (1) (2010) 115–122, https://doi.org/10.3382/ps.2009-00318.

[49] J. Yang, C.Q. Ou, Y. Ding, Y.X. Zhou, P.Y. Chen, Daily temperature and mortality: A study of distributed lag non-linear effect and effect modification in guangzhou, Environ. Health 11 (2012) 63. https://doi.org/10.1186/1476-069X-11-63.

[50] J. Yang, P. Yin, M. Zhou, C. Ou, M. Li, Y. Liu, et al., The effect of ambient temperature on diabetes mortality in China: a multi-city time series study, Sci. Total Environ. 543 (Pt A) (2016) 75–82, https://doi.org/10.1016/j.scitotenv.2015.11.014.

[51] T. Long, R. Wang, J. Wang, F. Wang, Y. Xu, Y. Wei, et al., Plasma metals and cardiovascular disease in patients with type 2 diabetes, Environ. Int. 129 (2019) 497–506, https://doi.org/10.1016/j.envint.2019.05.038.

[52] D.D. Sin, R.L. Jones, S.F.P. Man, Obesity is a risk factor for dyspnea but not for airflow obstruction, Arch. Intern. Med. 162 (13) (2002) 1477. https://doi.org/10.1001/archinte.162.13.1477.

[53] J. Han, J. Shao, Q. Chen, H. Sun, L. Guan, Y. Li, et al., Transcriptional changes in the hypothalamus, pituitary, and mammary gland underlying decreased lactation performance in mice under heat stress, Faseb. J. 33 (11) (2019) 12588–12601. https://doi.org/10.1096/fj.201901045R.

[54] G. Laurendi, C. Donfrancesco, L. Palmieri, D. Vanuzzo, G. Scalera, S. Giampaoli, et al., Association of lifestyle and cardiovascular risk factors with lung function in a sample of the adult Italian population: A cross-sectional survey, Respiration 89 (1) (2015) 33–40. https://doi.org/10.1159/000369035.

[55] J. Dai, X. Wu, Y. Bai, W. Feng, S. Wang, Z. Chen, et al., Effect of thallium exposure and its interaction with smoking on lung function decline: A prospective cohort study, Environ. Int. 127 (2019) 181–189, https://doi.org/10.1016/j.envint.2019.03.034.

[56] J.H. Sisson, J.A. Stoner, D.J. Romberger, J.R. Spurzem, T.A. Wyatt, J. Owens-Ream, et al., Alcohol intake is associated with altered pulmonary function, Alcohol 36 (1) (2005) 19–30, https://doi.org/10.1016/j.alcohol.2005.05.002.

[57] M.M. Vasquez, D.L. Sherrill, T.D. Levan, W.J. Morgan, J.H. Sisson, S. Guerra, Persistent light to moderate alcohol intake and lung function: A longitudinal study, Alcohol 67 (2018) 65–71, https://doi.org/10.1016/j.alcohol.2017.08.013.

[58] J. Park, M. Seo, H.C. Jung, J. Song, J. Lee, Association between health-related physical fitness and respiratory diseases in adolescents: An age- and gender-matched study, Int. J. Environ. Res. Publ. Health 18 (12) (2021) 6655. https://doi.org/10.3390/ijerph18126655.

[59] K. Aoltys, L. Lendvorský, I. Hric, E. Baranovičová, A. Penesová, I. Mikula, et al., Strenuous physical training, physical fitness, body composition and bacteroides to prevotella ratio in the gut of elderly athletes, Front. Physiol. 12 (2021). https://doi.org/10.3389/fphys.2021.670989.