Healthful Plant-Based Dietary Patterns, $PM_{2.5}$ Exposure, and the Risk of Heart Failure: a Population Based Cohort Study

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Abstract

Fine particulate matter ($PM_{2.5}$) is a known risk factor for heart failure (HF), while plant-based dietary patterns may help reduce HF risk. This study examined the combined impact of $PM_{2.5}$ exposure and a plant-based diet on HF incidence. A total of 190,092 participants from the UK Biobank were included in this study. HF cases were identified through linkage to the UK National Health Services register, with follow-up lasting until October 2022 in England, August 2022 in Scotland, and May 2022 in Wales. Annual mean $PM_{2.5}$ concentration was obtained using a land use regression model, while the healthful plant-based diet index (hPDI) was calculated using the Oxford WebQ tool based on two or more 24-hour dietary assessments of 17 major food groups. Cox proportional hazard models assessed the associations of $PM_{2.5}$ and hPDI with HF risk, and interactions were evaluated on additive and multiplicative scales. During a median of 13.4-year follow-up, 4,351 HF cases were recorded. Participants in the highest $PM_{2.5}$ tertile had a 23% increased HF risk (HR: 1.23, 95% CI: 1.14–1.32) compared to those in the lowest tertile. Moderate or high hPDI was associated with reduced HF risk relative to low hPDI. The lowest HF risk was observed in individuals with high hPDI and low $PM_{2.5}$ exposure, underscoring the protective role of a plant-based diet, particularly in areas with lower $PM_{2.5}$ levels. A healthy plant-based diet may mitigate HF risk, especially in populations exposed to lower $PM_{2.5}$ levels..

Keywords: Healthful Plant-Based Dietary Patterns; $PM_{2.5}$; Heart Failure; Cohort study

Introduction

Heart failure (HF), defined as a clinical syndrome characterized by typical symptoms, such as breathlessness, ankle swelling, and fatigue, is increasingly becoming a major global public health problem. Even though the incidence of HF has been declining since 2000, it is still highly prevalent and increases direct and indirect costs to the healthcare system owing to the aging of the population. It is suggested that there are an estimated of 56.19 million people globally suffered from HF [(1)]. The Global Burden of Disease Study suggested that air pollution was responsible for approximately 6.6 million premature deaths in 2019, which was mainly attributed to fine particulate matter ($PM_{2.5}$). The potential mechanism may be that air pollutants could increase the risk of cardio-metabolic diseases by eliciting inflammatory changes and oxidative stress [(2)].

Emerging evidence increasingly indicates that lifestyle factors, such as dietary habits, may influence the relationship between air pollution and human health. For example, a large prospective cohort study conducted in the United States found that adherence to a Mediterranean diet could alter the link between air pollution and cardiovascular disease (CVD) mortality[(3)]. Participants with higher scores on the alternative Mediterranean Diet Index showed a reduced risk of CVD mortality associated with air pollution exposure. Additionally, several short-term intervention studies have highlighted the potential role of specific foods or nutrients in modifying the impact of air pollution on cardiopulmonary health[(4; 5)]. Plant foods, usually rich in antioxidant vitamins, flavonoids, polyphenols, play a pivotal role in reducing inflammation and oxidative stress [(6)]. Previous studies suggested that plant-based dietary pattern might be associated with better neurological health and a reduced risk of cardiovascular risk factors, such as obesity[(7)], hypertension[(8)], and diabetes mellitus [(9)]. In considering the negative relationship between plant-based dietary patterns and HF and the adverse effect of air pollution and HF, it is plausible to hypothesize that plant-based diet may modify the association between air pollution and HF. Our study aimed to test this hypothesis and add additional dimensions to food, air pollution, and HF.

Methods

Study population

The UK Biobank is an ongoing national health resource carried out in the United Kingdom aimed at preventing, diagnosing, and treating a wide range of diseases and promoting health. This cohort includes over 500,000 participants aged between 40 and 69 years at recruiting time (2006–2010). Participants attended 1 of 22 assessment centres located across England, Scotland, and Wales, where they completed a comprehensive baseline assessment. Further details of the study protocol have been described elsewhere[10]. For this cohort study, participants who withdrew consent during follow-up, had missing data on diet, with uncompleted $PM_{2.5}$ exposure information, with HF or other cardiovascular disease at baseline, or had implausible energy intakes (>17 573 or <3347 kJ for men and >14644 or <2092 kJ for women) were excluded from analyses (Figure S1). The UK Biobank study received ethical approval from the NHS North-West Multi-Centre Research Ethics Committee (Ref. 11/NW/0382), and all participants provided written informed consent for their participation. Finally, a total of 190,092 participants were available for this analysis.

Assessment of HF

The assessment of HF were conducted using both linked with hospital inpatient data and death register records according to the International Classification of Disease 10th revision (ICD-10) codes: I11.0, I13.0, I13.2, I50.0, I50.1, and I50.9[11; 12]. Hospital admissions data were available until October 2022 in England, August 2022 in Scotland and May 2022 in Wales. Participants were followed-up from the date they attended the assessment center until the date of death, diagnosis, or last date of follow-up, whichever came first.

Healthful Plant-Based Dietary Patterns

The Oxford WebQ tool was adopted to evaluated the healthful plant-based diet index (hPDI) based on mean food intakes taken from a minimum of two 24-hour dietary assessments[13; 14]. The hPDI consisted 17 food groups[13]: whole grains, fruits, vegetables, nuts, legumes and vegetarian protein alternatives, tea and coffee, fruit juices, refined grains, potatoes, sugar-sweetened beverages, sweets and desserts, animal fat, dairy, eggs, fish or seafood, meat, and miscellaneous animal-derived foods. The 17 food groups were divided

into quintiles based on consumption levels, with scores assigned either positively (Q1=1 to Q5=5) or in reverse (Q5=1 to Q1=5). Healthy foods were assigned positive scores, while less healthy foods were assigned reverse scores. For positive scoring, the highest quintile received a score of 5, decreasing incrementally to a score of 1 for the lowest quintile. For reverse scoring, this pattern was flipped (details in Table S1). The scores for all 17 food groups were then summed to calculate the hPDI for each individual. A higher hPDI reflects a diet characterized by greater consumption of healthy plant-based foods, along with lower intake of less healthy plant-based foods and animal products, indicating better overall dietary quality. We categorized the participants into three groups based on the tertile cut-off values of hPDI as low, moderate and high hPDI, respectively. Previous research demonstrated stable food intake levels over 4 years following the baseline assessment in this cohort[15]. Additionally, a strong correlation (Pearson correlation coefficient =0.88; $P < 0.0001$) was found between the baseline hPDI and the averaged hPDI based on repeated measurements from 2009 to 2012[13]. Therefore, the earliest dietary intake data were used in this study to maximize follow-up time for participants who completed more than one dietary assessment.

Assessment of ambient air pollution

The annual average concentrations of $PM_{2.5}$ were estimated with a Land Use Regression (LUR) model developed from the European Study of Cohorts for Air Pollution Effects project [16; 17]. The spatial variations of annual average air pollutant concentrations were calculated using the LUR model including the geospatial predictor variables generated from the Geographic Information System such as traffic, land use, and topography. Air pollution exposures of all participants in the UK Biobank were linked to the records through residential addresses given at the baseline visit. The exposure data for $PM_{2.5}$ were collected in 2010. The annual average concentrations of $PM_{2.5}$ in 2010 for each participant were linked to their residential addresses provided at the baseline visit (2006–2010). Both the continuous data (per 10 $\mu g/m^3$) and categorical data [defined as low, moderate and high by the tertile cut-off points] for $PM_{2.5}$ were used for data analysis.

Covariates

Covariates were selected a priori, mainly based on literature review[13; 18]. Anthropometric measurements and biological samples were collected from all study participants by trained staff. In this study, several covariates were considered, including age, sex [19], ethnicity, educational level, BMI (in kg/m^2), smoking status [20], alcohol consumption [21], physical activity (PA)[22], and employment.

Statistical analysis

The baseline characteristics of the study population were presented by incident HF. Continuous variables were expressed as mean (SD) and categorical variables were presented as number (percentage), respectively.

Multivariable Cox regression models were used to analyse the relationships of hPDI and $PM_{2.5}$ with the risk of incident HF. Three models were used: Model 1 was the crude model; Model 2 was adjusted for age and sex; Model 3 was further adjusted for ethnicity, educational level, BMI, smoking status, drinking status, PA, and employment. To evaluate the combined effects of hPDI and $PM_{2.5}$, participants were categorized into nine groups according to hPDI and $PM_{2.5}$ exposure level: low hPDI-high $PM_{2.5}$, low hPDI -moderate $PM_{2.5}$, low hPDI -low $PM_{2.5}$, moderate hPDI-high $PM_{2.5}$, moderate hPDI -moderate $PM_{2.5}$, moderate hPDI-low $PM_{2.5}$, high hPDI-high $PM_{2.5}$, high hPDI -moderate $PM_{2.5}$, and high hPDI -low $PM_{2.5}$. The hazard ratios (HRs) with their 95% confidence interval (CI) of HF in model 3 were calculated with low hPDI and high $PM_{2.5}$ exposure as the reference. The interaction effects of $PM_{2.5}$ exposure and hPDI were analysed using both additive and multiplicative interaction models. For additive interactions, a new variable was constructed to categorize individuals into nine groups based on combinations of $PM_{2.5}$ exposure levels (low, moderate, and high) and hPDI levels (low, moderate, and high), with individuals in the low hPDI and high $PM_{2.5}$ group serving as the reference. The relative excess risk due to interaction (RERI) was calculated to quantify the interaction: RERI=0 indicated no additive interaction, RERI<0 suggested a negative additive interaction, and RERI>0 reflected a positive additive interaction. In this study, a significant positive RERI suggested that hPDI could influence the effects of $PM_{2.5}$ exposure on HF risk. For multiplicative interactions, the likelihood ratio test was performed

by comparing models with and without an interaction term between $PM_{2.5}$ and hPDI. A P-value for interaction below 0.05 was considered evidence of a significant multiplicative interaction.

A total of 4 sensitivity analyses were carried out to examine the robustness of the relationships by 1) excluding individuals with less than 2 years of follow-up to minimize the reverse casualty; 2) restricting analyses among the participants without hypertension, diabetes, and cancer at baseline to minimize the potential influence of comorbidities; 3) restricting analyses among the male participants to determine if the relationships were consistent across different sexes; 4) excluding participants aged less than 60 years old to investigate whether the results were consistent across different age-groups; 5) further adjusting for diabetes, hypertension, dyslipidaemia, and cancer at baseline minimize the potential influence of health status on diet habits.

All statistical analyses were conducted using R (version 4.0.3). A 2-tailed $P < 0.05$ was considered as statistical significance.

Results

Table 1 presents the baseline characteristics of 190,092 study participants by HF status. The mean age of the participants at baseline was 56.42 years (SD: 7.94 years) and 48.7% were males; 4351 cases of HF were identified during the follow-up. Participants who developed HF were older, and more likely to be male, unemployed, and current smokers in comparison with those without depression.

The main effects of hPDI and chronic $PM_{2.5}$ exposure on incident HF are depicted in Table 2. A higher hPDI was related to a lower risk of incident HF. Compared with the low hPDI levels, the HR (95% CI) of incident HF was 0.88 (95% CI: 0.82, 0.94) and 0.84 (95% CI: 0.78, 0.91) for moderate and high levels of hPDI, respectively. Whereas, individuals exposed to higher level of $PM_{2.5}$ were associated with a higher risk of HF. Compared with the low-$PM_{2.5}$ group, the moderate- and high-$PM_{2.5}$ groups had HRs of 1.13 (95% CI: 1.05, 1.21) and 1.23 (95% CI: 1.14, 1.32), respectively.

The results of subgroup analysis are shown in Table 3. A higher hPDI was associated with a lower HF risk in moderate and high $PM_{2.5}$ exposure, whilst a higher level of $PM_{2.5}$

exposure was associated with a higher HF risk in each hPDI stratum.

The combined effects of hPDI and long-term exposure to $PM_{2.5}$ on HF risk are shown in Table 4 and Figure S1. Individuals with a high level of hPDI and exposed to low level of $PM_{2.5}$ exhibited the lowest risk of incident HF, whilst those who were in low hPDI group and exposed to a high level of $PM_{2.5}$ were associated with the highest risk of HF. Compared with participants were in low hPDI group and exposed to a high level of $PM_{2.5}$, those with a high level of hPDI and exposed to low level of $PM_{2.5}$ had a HR of 0.68 (95% CI: 0.60, 0.78). The trend patterns of the effects of hPDI and $PM_{2.5}$ exposure were evident in each stratum of $PM_{2.5}$ exposure or hPDI. A significant positive RERI was observed, indicating the existence of positive additive interactions. For individuals with Moderate hPDI and low $PM_{2.5}$ exposure, the RERI were 0.15 (95% CI: 0.01, 0.29), suggesting that a 0.11 relative excess risk was due to the additive interaction. However, no significant additive interactions for participants with high hPDI and low $PM_{2.5}$ exposure.

The sensitivity analysis generally yield similar results (Tables S2-S5).

Discussion

To the best of our knowledge, this is the first study to evaluate both the independent and combined effects of adherence to healthy plant-based dietary pattern and long-term $PM_{2.5}$ exposure on the risk of HF. Our study found that higher hPDI was associated with lower risk of HF. Whilst, living in areas with higher $PM_{2.5}$ was associated with higher risk of HF. $PM_{2.5}$ exposure may increase the risk of HF and more greatly in individuals with low hPDI than those with high hPDI. Our findings indicated that higher adherence to the plant-based dietary pattern might be beneficial to the risk of HF induced by chronic exposure to $PM_{2.5}$.The negative relationships between a plant-based diet and HF can be attributed to several factors. First, plant-based diets are typically rich in dietary fibre, which has been consistently associated with a reduced risk of cardiovascular diseases, including HF. The high fibre content in these diets promotes satiety, helps maintain healthy body weight, and reduces the risk of obesity and metabolic disorders, all of which are risk factors for HF[23; 24]. Second, plant-based diets are abundant in antioxidant compounds, such as vitamins C and E, carotenoids, and flavonoids, that have been shown to possess anti-inflammatory and

cardioprotective properties [25]. These antioxidants help minimize oxidative stress and inflammation, which play crucial roles in the development and progression of HF. Elevated serum levels of inflammation increase risk factors for HF, as well as incident HF [26; 27]. Plant-based diets decrease serum levels of inflammation and may be protective. One potential explanation is the differing factors related to the reduction of oxidative stress and inflammation [28; 29]. Furthermore, the consumption of plant-based proteins instead of animal-based proteins in a plant-based diet may contribute to its beneficial effects on heart health. Plant-based proteins, derived from sources like legumes, soy, and nuts, are typically lower in saturated fat and cholesterol compared to animal-based proteins. Substituting animal proteins with plant-based proteins has been associated with improved lipid profiles, decreased blood pressure, and reduced cardiovascular risk, which may ultimately lower the risk of HF[30].

Moreover, our study investigated the potential role of $PM_{2.5}$ exposure in HF risk. It is well known that $PM_{2.5}$ exposure is associated with higher risk of incident HF. Our results reported a positive association between $PM_{2.5}$ exposure and HF, which is in line with previous studies[31; 32].. Several potential mechanisms might be underlying the observed relations between air pollution and HF. For instance, exposure to air pollution can lead to oxidative stress, systemic inflammation, and autonomic imbalance and then increase blood pressure [33] and decrease cardiac output [34]. Prolonged or repeated stimulation of these pathways may further result in the progression of endothelial dysfunction, atherosclerosis, diastolic dysfunction, left ventricular hypertrophy, and myocardial fibrosis[35; 36; 37], which could eventually increase the risk of HF. In addition, a randomized controlled study of HF showed that a filter intervention might reduce endothelial dysfunction and B-type natriuretic peptide increases associated with short-term exposure to diesel exhaust exposure in patients with HF.[38; 39] Existing evidence suggests the potential mechanisms for the risk of HF associated with a higher $PM_{2.5}$ exposure strengthened by a low hPDI score.

We observed a synergistic interaction on the additive scale between hPDI and $PM_{2.5}$ exposure in relation to HF risk. The RERI is widely regarded as the standard metric for assessing additive interactions, as it better represents biological interaction compared to

measures on the multiplicative scale[40]. Assessing interactions on the additive scale is particularly useful for determining whether two risk factors act synergistically-meaning their combined excess risk exceeds the sum of their individual excess risks-and is especially relevant for evaluating public health implications[41]. In this study, the significant RERI suggested that moderate hPDI combined with low $PM_{2.5}$ exposure reduced HF risk more than expected based on their independent effects. These findings imply that hPDI may modify the relationship between $PM_{2.5}$ exposure and HF risk, potentially attenuating the negative impact of air pollution in individuals with higher hPDI scores. Further research is required to confirm and expand upon these observations.To our knowledge, this is the first study to evaluate the combined effects of plant-based dietary patterns and $PM_{2.5}$ on HF risk. The prospective study design and the large sample size were the two main strengths of this study. The current study also has several potential limitations. First, the results from our observational study were based on a retrospective sub-analysis of the data from the UK Biobank. Thus, the causality of the results should be interpreted with caution. Second, the dietary assessment was based on 24-hour recall, which might be subjected to measurement error and lead to misclassification [16]. Third, only 17 food groups were used to construct the PDIs due to the unavailability of vegetable oils in the current study, which was included in the original paper describing the PDIs by Satija et al. [42]. Fourth, the PDIs treat all animal-based foods equally without discrimination by assigning opposite scores, which may ignore benefits from some food components, such as dairy products and seafood. However, the results of our sensitivity analyses were stable by considering dairy products and seafood as healthful food groups. Fifth, only a single measurement of air pollution is available in the UK Biobank since the home addresses of the participants are unavailable during follow-up. Further studies with repeated measurements are needed to confirm the findings. Sixth, even though we had controlled the majority of confounders, the residual confounding from unmeasured or unknown factors might remain. Finally, our analyses were conducted among Europeans, limiting the extrapolation of our findings to other ethnic groups.

In conclusion, our findings from the UK Biobank Study suggested that the association between long-term $PM_{2.5}$ exposure and the risk of developing HF were lower among the

participants with higher hPDI. Our findings suggest that higher adherence to the plant-based dietary pattern may benefit the risk of HF induced by long-term $PM_{2.5}$ exposure. Promoting the plant-based dietary pattern may be a strategy to reduce the effects of $PM_{2.5}$ on cardiovascular disease.

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None of the authors has any conflicts of interest to declare.

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YJZ and LL designed research; YJZ conducted the data cleaning and data analysis; SSZ, XQZ and YJZ drafted the manuscript; ZKW YFJ, WDW, JXZ, XLZ, and YCB revised the manuscript. YJZ and LL had primary responsibility for final content. All authors read and approved the final manuscript.

Figure Legends

Figure S1 Flowchart of participants selection

Figure S2 Combined effects of hPDI and $PM_{2.5}$ on risk of heart failure

Abbreviations: $PM_{2.5}$ particulate matter with aerodynamic diameter < 2.5 μm, hPDI: Healthful Plant-Based Diet Index

The results were adjusted by age, sex, ethnicity, physical activity, education, smoking status, drinking status, and BMI

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Table 1 Baseline characteristics of the study participants by heart failure

Characteristics	Overall N=190092	Incident HF N=4351	Non-HF N= 185741
Age(years)	56.42 (7.94)	62.22 (6.13)	56.28 (7.93)
Sex			
Female	106938 (56.3)	1723 (39.6)	105215 (56.6)
Male	83154 (43.7)	2628 (60.4)	80526 (43.4)
BMI, kg/m^2	26.91 (4.64)	29.09 (5.64)	26.86 (4.61)
Ethnic			
White	181036 (95.2)	4187 (96.2)	176849 (95.2)
Others	9056 (4.8)	164 (3.8)	8892 (4.8)
Physical activity			
Low	34941 (18.4)	921 (21.2)	34020 (18.3)
Moderate	80215 (42.2)	1811 (41.6)	78404 (42.2)
High	74936 (39.4)	1619 (37.2)	73317 (39.5)
Employment			
No	72443 (38.1)	2695 (61.9)	69748 (37.6)
Yes	117649 (61.9)	1656 (38.1)	115993 (62.4)
Education qualification			
<high school	109547 (57.6)	2949 (67.8)	106598 (57.4)
≥ high school	80545 (42.4)	1402 (32.2)	79143 (42.6)
Smoking status			
Never	108457 (57.1)	1938 (44.5)	106519 (57.3)
Former	66730 (35.1)	1891 (43.5)	64839 (34.9)
Current	14905 (7.8)	522 (12.0)	14383 (7.7)
Drinking status			
Never	6213 (3.3)	171 (3.9)	6042 (3.3)
Former	5680 (3.0)	221 (5.1)	5459 (2.9)
Current	178199 (93.7)	3959 (91.0)	174240 (93.8)
$PM_{2.5}$, $\mu g/m^{-3}$	9.91 (1.02)	9.95 (1.01)	9.91 (1.02)
Comorbidities at baseline			
Cancer	14337 (7.5)	509 (11.7)	13828 (7.4)
Hypertension	7708 (4.1)	422 (9.7)	7286 (3.9)
Diabetes	7367 (3.9)	512 (11.8)	6855 (3.7)

Abbreviations: $PM_{2.5}$:particulate matter with aerodynamic diameter < 2.5 μm, HF: Heart failure, BMI: Body Mass index

Table 2 Associations of plant-based diet and long-term $PM_{2.5}$ exposure and risk of HF

	Model 1		Model2		Model 3	
	HR (95 % CI)	*P*	HR (95 % CI)	*P*	HR (95 % CI)	*P*
hPDI						
Low-hPDI	Ref		Ref		Ref	
Moderate-hPDI	0.87(0.81 - 0.94)	<0.001	0.83(0.77 - 0.89)	<0.001	0.88(0.82 - 0.94)	0.003
High-hPDI	0.78(0.72 - 0.83)	<0.001	0.77(0.72 - 0.83)	<0.001	0.84(0.78 - 0.91)	<0.001
$PM_{2.5}$ levels[a]						
Low-$PM_{2.5}$	Ref		Ref		Ref	
Moderate-$PM_{2.5}$	1.12(1.04 - 1.20)	0.0034	1.17(1.08 - 1.26)	<0.001	1.13(1.05 - 1.21)	0.0014
High-$PM_{2.5}$	1.11(1.03 - 1.20)	0.0045	1.29(1.20 - 1.39)	<0.001	1.23(1.14 - 1.32)	<0.001
Per IQR increment	1.04(1.01 - 1.07)	0.0136	1.11(1.08 - 1.14)	<0.001	1.08(1.05 - 1.12)	<0.001

Abbreviations: $PM_{2.5}$:particulate matter with aerodynamic diameter < 2.5 μm, HR: hazard ratio, CI: confidence interval, hPDI: Healthful Plant-Based Diet Index, BMI: Body Mass index

Model 1 was crude model; Model 2 was adjusted for age and sex; Model 3 was further adjusted for ethnicity, physical activity, education, smoking status, drinking status, and BMI.

Table 3 Subgroup analyses on the association of HF with hPDI and $PM_{2.5}$ exposure stratified by the categories of $PM_{2.5}$ or hPDI

Stratified by $PM_{2.5}$	**Low-$PM_{2.5}$**		**Moderate-$PM_{2.5}$**		**High-$PM_{2.5}$**	
	Hazard Ratio	***P***	**Hazard Ratio**	***P***	**Hazard Ratio**	***P***
Low-hPDI	Ref	–	Ref	–	Ref	–
Moderate-hPDI	0.98(0.86 - 1.11)	0.714	0.83(0.73 - 0.94)	0.003	0.84(0.74 - 0.95)	0.006
High-hPDI	0.90(0.79 - 1.03)	0.128	0.82(0.72 - 0.93)	0.002	0.82(0.72 - 0.93)	0.002
Stratified by hPDI	**Low-hPDI**		**Moderate-hPDI**		**High-hPDI**	
	Hazard Ratio	***P***	**Hazard Ratio**	***P***	**Hazard Ratio**	***P***
Low-$PM_{2.5}$	Ref	–	Ref	–	Ref	–
Moderate-$PM_{2.5}$	1.21(1.08 - 1.36)	<0.001	1.04(0.91 - 1.19)	0.591	1.10(0.96 - 1.26)	0.171
High-$PM_{2.5}$	1.31(1.17 - 1.47)	<0.001	1.15(1.00 - 1.32)	0.044	1.19(1.04 - 1.37)	0.013

Abbreviations: $PM_{2.5}$:particulate matter with aerodynamic diameter < 2.5 μm, HR: hazard ratio, CI: confidence interval, hPDI: Healthful Plant-Based Diet Index, BMI: Body Mass index

The results were adjusted by age, sex, ethnicity, physical activity, education, smoking status, drinking status, and BMI.

Table 4 Combined effects of hPDI, $PM_{2.5}$ and the risk of HF

hPDI	$PM_{2.5}$ levels (HR, 95% CI)			RERI		*P* for interaction
	High-$PM_{2.5}$	Moderate-$PM_{2.5}$	Low-$PM_{2.5}$	Moderate-$PM_{2.5}$	Low-$PM_{2.5}$	
						0.178
Low-hPDI	Ref	0.92(0.83 - 1.02)	**0.75(0.67 - 0.84)**			
Moderate-hPDI	**0.84(0.74 - 0.95)**	**0.76(0.67 - 0.86)**	**0.74(0.65 - 0.84)**	0.00(-0.15 - 0.15）	**0.15 (0.01 - 0.29)**	
High-hPDI	**0.81(0.72 - 0.92)**	**0.75(0.67 - 0.85)**	**0.68(0.60 - 0.78)**	0.02(-0.12 - 0.17)	0.12 (-0.02 - 0.16)	

Abbreviations: $PM_{2.5}$:particulate matter with aerodynamic diameter < 2.5 μm, HR: hazard ratio, CI: confidence interval, hPDI: Healthful Plant-Based Diet Index, BMI: Body Mass index

The results were adjusted by age, sex, ethnicity, physical activity, education, smoking status, drinking status, and BMI.

The estimates of RERI were calculated based on the reference group with low hPDI and high $PM_{2.5}$