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Article

Ambient PM2.5 exposure is linked to elevated platelet count and reduced serum creatinine: a cross-sectional study in the UK Biobank

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Abstract
Air pollution, particularly fine particulate matter (PM2.5) is a major risk factor for cardiovascular and renal disease. Using UK Biobank data, we examined associations between World Health Organization PM2.5 classes and 18 blood biomarkers. We conducted (i) descriptive class-mean summaries and (ii) individual-level Pearson trend tests with Benjamini–Hochberg false discovery rate (BH-FDR) control across biomarkers. PM2.5 class showed a positive association with platelet count and a negative association with serum creatinine; both signals were consistent across descriptive trajectories and remained significant after BH-FDR adjustment. While several additional biomarkers reached statistical significance, effect sizes were uniformly small. Findings support the hypothesis that ambient air pollution is linked with systemic physiological changes relevant to cardiovascular (pro-thrombotic) and renal health and underscore the potential value of biomarker monitoring in population health assessments.

Keywords: *PM2.5, renal function, biomarkers, platelets, creatinine*

INTRODUCTION

Air pollution remains one of the most pervasive global environmental health threats; ambient (outdoor) air pollution is estimated to cause millions of premature deaths worldwide each year [1]. Among ambient pollutants, fine particulate matter (PM2.5) airborne particles less than 2.5 μm in diameter have garnered particular attention due to its strong associations with cardiovascular disease (CVD), renal dysfunction, and systemic inflammation. PM2.5 can penetrate deeply into the lungs, enter the bloodstream, and initiate oxidative stress, inflammatory responses, autonomic dysregulation, and endothelial dysfunction.

Recent advances in epidemiology and toxicology have highlighted the utility of specific blood-based biomarkers in both mediating and reflecting the adverse health effects of air pollution. Biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), and creatinine serve as quantifiable indicators of inflammation, oxidative stress, and renal function, respectively. Despite growing recognition of their importance, empirical studies that quantify the relationship between PM2.5 exposure and changes in these biomarkers within large population-based cohorts remain limited [2,3].

The present study seeks to address this gap by analysing the associations between PM2.5 classes and 18 blood-based biomarkers using robust data from the UK Biobank. Specifically, we pose the following research question: Which blood-based biomarkers most reliably reflect physiological responses to PM2.5 exposure in a large, representative population?

Air Pollution and cardiovascular disease

The relationship between air pollution and cardiovascular disease (CVD) is well-established in environmental epidemiology and public health research. Time-series evidence also shows that short-term increases in particulate air pollution are associated with increased hospital admissions among older adults [4]. Fine particulate matter (PM2.5), defined as airborne particles with a diameter less than 2.5 μm, has been identified as a major contributor to adverse cardiovascular outcomes, including ischemic heart disease, heart failure, arrhythmias, and stroke [5,6]. This association is mediated through multiple interrelated pathophysiological mechanisms, including systemic inflammation, oxidative stress, endothelial dysfunction, autonomic imbalance, altered platelet function, and renal impairment [7].

The small size of PM2.5 particles allows them to evade upper respiratory defenses, penetrate deeply into the alveolar spaces of the lungs, and enter the systemic circulation. Once in the bloodstream, PM2.5 triggers inflammatory responses characterized by elevated levels of biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α) [5,8]. These inflammatory markers have been directly implicated in the development of atherosclerosis and plaque instability [9].

Oxidative stress represents another key mechanism by which PM2.5 exerts cardiovascular effects. Inhaled particles stimulate the production of reactive oxygen species (ROS), leading to reduced nitric oxide bioavailability, increased vascular inflammation, and compromised endothelial function, which together contribute to arterial stiffness and hypertension [10,11]. In addition, PM2.5 exposure has been shown to disrupt autonomic nervous system regulation, as evidenced by increased sympathetic activity and reduced heart rate variability, further elevating the risk of arrhythmias and sudden cardiac events [5,6,12]. Particle composition (e.g., transition metals such as nickel) may modulate these vascular and inflammatory effects [13,14].

Emerging evidence also suggests that air pollution can adversely impact platelet function, an essential component of cardiovascular homeostasis. Epidemiological and experimental studies have linked PM2.5 exposure to increased platelet activation and aggregation, as reflected in elevated levels of biomarkers such as soluble P-selectin and β-thromboglobulin [15,16]. These pro-thrombotic changes are thought to be driven by systemic inflammation and oxidative stress, resulting in heightened platelet reactivity and a hypercoagulable state, thereby increasing the risk of myocardial infarction and stroke.

Renal biomarkers, particularly serum creatinine, have also emerged as relevant indicators of pollutant-induced systemic effects. PM2.5 may impair renal function via inflammatory and vascular pathways, leading to decreased glomerular filtration rate (GFR) and increased serum creatinine [17÷21]. Notably, reduced kidney function not only results from pollutant exposure but also amplifies cardiovascular risk, linking air pollution exposure to cardio-renal syndrome through shared pathophysiological pathways.

In summary, the link between PM2.5 exposure and CVD is commonly explained by interrelated mechanisms including systemic inflammation, oxidative stress, endothelial dysfunction, autonomic dysregulation, pro-thrombotic processes, and renal impairment. While mechanistic studies often assess markers such as IL-6, TNF-α, reactive oxygen species, nitric oxide bioavailability, and platelet activation markers (e.g., soluble P-selectin, β-thromboglobulin), the present study evaluates routinely available blood biomarkers that capture downstream inflammatory and thrombo-inflammatory signals (CRP, leukocyte indices, platelet count) and cardio-metabolic and renal pathways (lipids, glucose/HbA1c, creatinine, cystatin C). Collectively, these pathways provide a mechanistic basis for the increased cardiovascular risk observed in populations exposed to elevated levels of PM2.5 [22]. These mechanisms also underscore the relevance of blood-based biomarkers for characterizing early biological responses to exposure [2].

The aim of the study was to examine the associations between World Health Organization PM2.5 classes and 18 blood biomarkers using UK Biobank data.

EXPERIMENTAL PART

Data source

This study utilized data from the UK Biobank, a large-scale, prospective, population-based cohort. The dataset included annual PM2.5 exposure estimates for 2010, as well as 18 blood biomarker values for each participant. After preprocessing and removal of records with missing values, a total of 175,586 participants were included in the final analysis.

Exposure classification

Participants were categorized into five exposure groups according to the World Health Organization (WHO) 2021 PM2.5 classes air quality guidelines:

- Class 0: AQG (≤ 5 μg/m^3)
- Class 1: IT-4 ($5 \div 10$ μg/m^3)
- Class 2: IT-3 ($10 \div 15$ μg/m^3)
- Class 3: IT-2 ($15 \div 25$ μg/m^3)
- Class 4: IT-1 (> 25 μg/m^3)

No participants fell in Class 0 (AQG). Class sizes for Classes 1÷4 were: IT-4 = 86,820; IT-3 = 76,543; IT-2 = 125; IT-1 = 12,098.

Variables

The blood biomarkers analysed included: inflammatory markers (C-reactive protein (CRP), white blood bell count (WBC), neutrophils, lymphocytes, monocytes, and platelets); lipid profile markers (total cholesterol, high-density lipoprotein (HDL), low density lipoprotein (LDL), LDL cholesterol, Apolipoprotein A (Apo A), Apolipoprotein B (Apo B), lipoprotein(a), and triglycerides); metabolic and renal markers (glycated hemoglobin (HbA1c), glucose, creatinine, and cystatin C).

Statistical analysis

In the study were conducted two complementary analyses: descriptive (ecological) group-level summaries (Table 1), respectively primary inferential individual-level trend analysis (Table 2). For each PM2.5 class, we computed mean biomarker values. To characterize monotonic trends across exposure classes, we correlated the class index (1÷4) with the class means using Pearson's r. Because this analysis uses only four aggregated points, it is descriptive and subject to ecological limitations (Table 1).

At the participant level, we coded PM2.5 class as an ordered variable (1÷4) and computed Pearson correlations between class and each biomarker (two-sided tests). We controlled the false discovery rate across the 18 biomarkers using the Benjamini–Hochberg procedure (q-values), q denotes BH_FDR adjusted p-values (threshold $q < 0.05$). Given the ordinal exposure, Pearson's r serves as a linear trend test; results are interpretable as very small but population-level linear associations (Table 2).

The Pearson correlation coefficient (r) is calculated as follows:

$$r = \frac{\sum_{i=1}^{n}(x_i - \underline{x})(y_i - \underline{y})}{\sqrt{\sum_{i=1}^{n}(x_i - \underline{x})^2}\sqrt{\sum_{i=1}^{n}(y_i - \underline{y})^2}} \quad (1)$$

where: x_i and y_i are paired observations; $\underline{x}$ and $\underline{y}$ are sample means; n is the total number of paired observations.

Software and tools

All data processing and statistical analyses were performed using Python (version 3.12.0), employing libraries including NumPy, Pandas, and Matplotlib for data manipulation and visualization. Pearson correlations were computed with scipy.stats.pearsonr; multiple testing was controlled with statsmodels.stats.multitest.multipletests(method="fdr_bh"). All figures use standardized (Z-score) biomarkers for comparability across scales.

RESULTS AND DISCUSSION

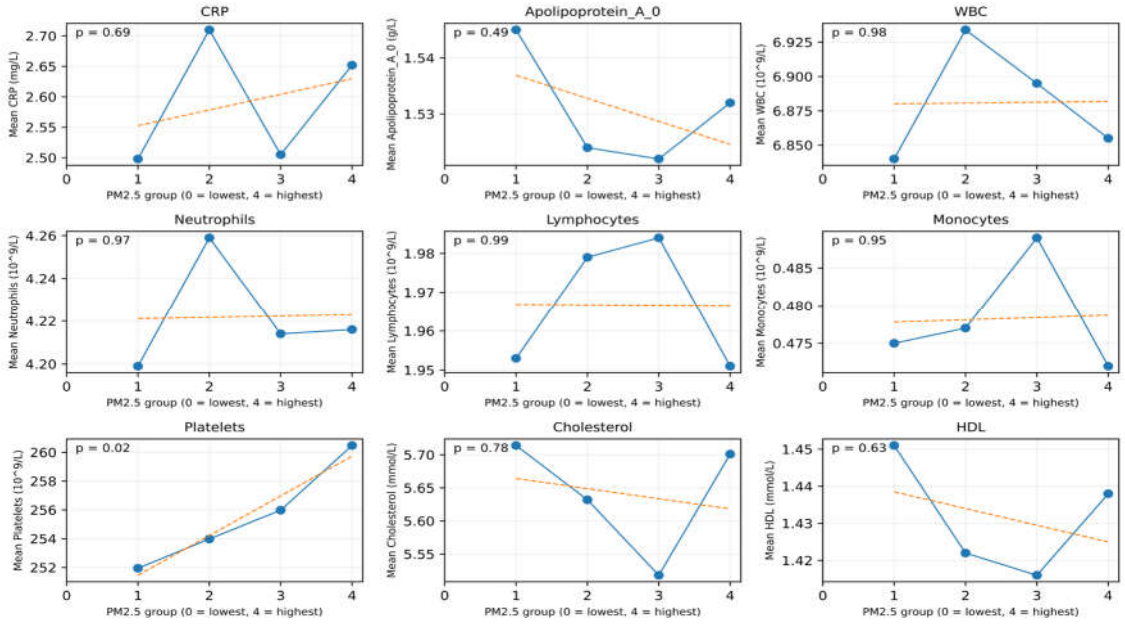
Descriptive patterns across PM2.5 classes

As summarized in Table 1, class-mean biomarker values were broadly stable across PM2.5 exposure classes. Visual trajectories in Figure 1 showed flat or minimally varying patterns for CRP, Apo A, WBC, neutrophils, lymphocytes, HDL, and LDL. Apo B, lipoprotein(a), and cholesterol exhibited small fluctuations without a consistent monotonic trend. In contrast, two markers displayed clear class gradients: platelets increased with higher PM2.5 class (251.95 → 260.48 from Class 1 to Class 4), while creatinine decreased (72.60 → 71.28). Corresponding ecological correlations based on class means indicated nominal significance for platelets ($r = 0.98$, $p = 0.02$) and creatinine ($r = -0.99$, $p = 0.01$); other markers were non-significant ($p > 0.05$). These statistics are descriptive and reflect trends in aggregated class means.

Table 1. Mean biomarker values by PM2.5 exposure class (WHO 2021), with Pearson correlation (r) and p-values across classes

Biomarker	Class 1	Class 2	Class 3	Class 4	r	p-value
CRP	2.50	2.71	2.50	2.65	0.31	0.69
Apo A	1.54	1.52	1.52	1.53	-0.51	0.49
WBC	6.84	6.93	6.89	6.86	0.02	0.98
Neutrophils	4.20	4.26	4.21	4.22	0.03	0.97
Lymphocytes	1.95	1.98	1.98	1.95	-0.01	0.99
Monocytes	0.47	0.48	0.49	0.47	0.05	0.95
Platelets	**251.95**	**253.98**	**255.98**	**260.48**	**0.98**	**0.02**
Cholesterol	5.71	5.63	5.52	5.70	-0.22	0.78
HDL	1.45	1.42	1.42	1.44	-0.37	0.63
LDL	3.58	3.53	3.45	3.58	-0.12	0.88
LDL Cholesterol	1.76	1.74	1.72	1.76	-0.31	0.69
Apo B	1.03	1.02	1.00	1.03	-0.17	0.83
Lipoprotein(a)	44.28	44.81	37.99	45.27	-0.15	0.85
HbA1c	35.88	36.26	37.36	35.92	0.23	0.77
Glucose	5.10	5.13	5.23	5.05	-0.09	0.91
Creatinine	**72.60**	**72.33**	**71.71**	**71.28**	**-0.99**	**0.01**
Cystatin C	0.90	0.91	0.89	0.91	-0.26	0.74
Triglycerides	1.73	1.75	1.75	1.72	-0.19	0.81

Notes: Columns "Class 1"÷"Class 4" correspond to WHO 2021[23] PM2.5 classes as implemented here: Class 1 = IT-4 (> 5–≤ 10 µg/m³), Class 2 = IT-3 (> 10–≤ 15 µg/m³), Class 3 = IT-2 (> 15–≤ 25 µg/m³), Class 4 = IT-1 (> 25 µg/m³). No participants met AQG (≤ 5 µg/m³). Values are class means. Pearson r and p summarize the linear trend across Classes 1–4 using these four classes means (descriptive only; no multiplicity adjustment). Class Ns: IT-4=86,820; IT-3=76,543; IT-2=125; IT-1=12,098.



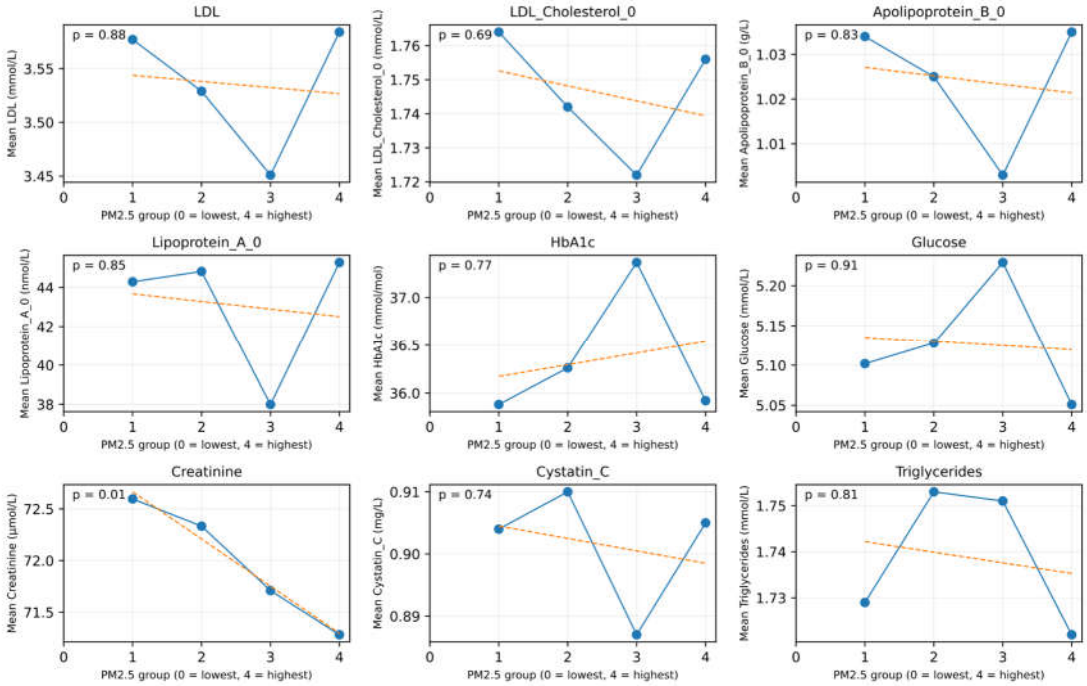


Fig. 1. Standardized (Z-score) trends for all measured biomarkers across PM2.5 exposure classes. Most biomarkers, including CRP, Apo A, WBC, neutrophils, lymphocytes, HDL, and LDL, remained relatively stable across exposure classes. Platelet count and creatinine, however, exhibited clear monotonic trends: platelet counts increased with higher PM2.5 exposure, while creatinine decreased.

Correlation analysis

We next evaluated individual-level trends by correlating ordered PM2.5 class (1÷4) with each biomarker and controlling the false discovery rate across 18 tests. Table 2 reports Pearson r, p, and BH–FDR q values. Directionally consistent with the descriptive findings, platelets showed a positive trend ($r = 0.035$, $q < 0.001$) and creatinine a negative trend ($r = -0.018$, $q < 0.001$). Several additional biomarkers reached $q < 0.05$ (BH–FDR) (e.g., CRP, Apo A, LDL, LDL Cholesterol, Apo B, Lipoprotein(a), HbA1c, Cystatin C); however, effect sizes were uniformly small ($|r| \approx 0.00 – 0.04$), indicating population-level associations of limited magnitude. Lymphocytes, glucose, and

Table 2. Individual-level associations between WHO 2021 PM2.5 class and blood biomarkers (Pearson trend across ordered classes IT-4 to IT-1)

Biomarker	r (Pearson, individual)	p	q (BH–FDR)	q <0.05
CRP	**0.017**	**2.95e-12**	**1.06e-11**	**Yes**
Apo A	**-0.025**	**3.84e-25**	**3.46e-24**	**Yes**
WBC	**0.010**	**1.42e-05**	**2.56e-05**	**Yes**
Neutrophils	**0.011**	**8.95e-06**	**1.79e-05**	**Yes**
Lymphocytes	0.004	8.03e-02	9.04e-02	No
Monocytes	-0.002	4.67e-01	4.67e-01	No
Platelets	**0.035**	**1.99e-49**	**3.58e-48**	**Yes**
Cholesterol	**-0.017**	**4.22e-12**	**1.27e-11**	**Yes**
HDL	**-0.023**	**7.10e-22**	**4.26e-21**	**Yes**
LDL	**-0.010**	**6.07e-05**	**9.93e-05**	**Yes**
LDL Cholesterol	**-0.014**	**8.07e-09**	**2.08e-08**	**Yes**
Apo B	**-0.007**	**5.02e-03**	**6.95e-03**	**Yes**
Lipoprotein(a)	**0.006**	**1.25e-02**	**1.60e-02**	**Yes**
HbA1c	**0.013**	**1.60e-07**	**3.60e-07**	**Yes**
Glucose	-0.004	7.42e-02	8.90e-02	No
Creatinine	**-0.018**	**9.45e-14**	**4.25e-13**	**Yes**
Cystatin C	**0.008**	**1.24e-03**	**1.86e-03**	**Yes**
Triglycerides	0.003	1.55e-01	1.64e-01	No

triglycerides did not pass FDR ($q \geq 0.05$). Class size imbalance (see notes in Table 2) does not affect the interpretation of r as an overall linear trend but should be considered when comparing classes.
Overall, our results are consistent with prior epidemiological and experimental evidence while clarifying that most exposure–biomarker associations are statistically detectable but small in magnitude at the individual level. We treat Table 2 the individual-level Pearson trends with BH–FDR control across 18 biomarkers as the primary inferential evidence and use Table 1 and Figures 1, 2 to provide descriptive and visual context.

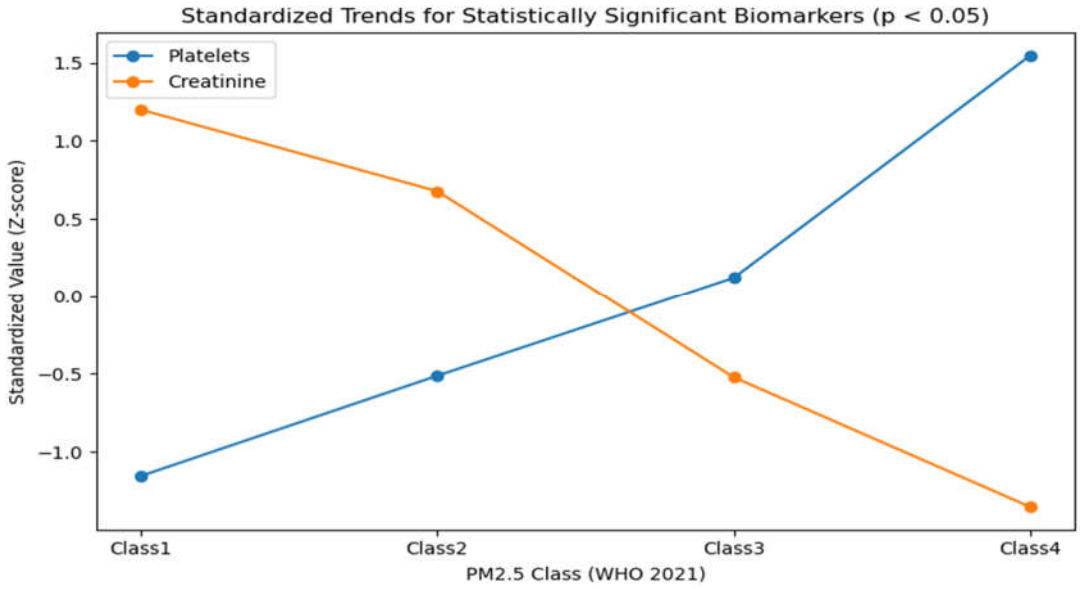


Fig. 2. Standardized (Z-score) trends for the two most consistent biomarkers with PM2.5 exposure: platelets (positive trend) and creatinine (negative trend) across exposure classes. These patterns highlight the strongest and most coherent biomarker responses observed in the study.

Platelets

The strong positive correlation between PM2.5 class and platelet count (ecological class-mean analysis: $r = 0.98$, $p = 0.02$; see Figure 1) supports prior evidence that particulate air pollution can promote a pro-thrombotic state via platelet activation and systemic inflammation [5,15,16]. At the individual level, this association remained robust after multiple-testing correction (Table 2: $r = 0.035$, $p = 1.99\times10^{-49}$, $q = 3.58\times10^{-48}$), indicating a population-level but small-magnitude increase in platelet count with higher PM2.5 class. Long-term exposure to ambient PM2.5 has been associated with higher platelet counts in adults, supporting the hypothesis that PM2.5 may enhance cardiovascular risk by influencing blood coagulation factors [15]. Multiple studies have linked elevated platelet counts and platelet reactivity to increased ambient particulate matter, suggesting a potential mechanism for increased cardiovascular events in polluted environments [5]. Experimental work further shows that PM2.5 promotes platelet activation and thrombosis through oxidative stress and mitochondrial dysfunction [16].

Creatinine

The negative correlation observed between PM2.5 class and creatinine (ecological class-mean analysis: $r = -0.99$, $p = 0.01$; see Figure 2) is less frequently documented, but growing evidence suggests air pollution may adversely affect renal function [17,18]. At the individual level, this association remained statistically significant after multiple-testing correction (Table 2: $r = -0.018$, $p = 9.45\times10^{-14}$, $q = 4.25\times10^{-13}$), indicating a small-magnitude decrease in creatinine with higher PM2.5 class. Long-term exposure to fine particulate matter has been linked to modest but significant declines in kidney function, including changes in serum creatinine in older men [17], and reviews indicate that chronic PM2.5 exposure may contribute to decreased kidney function and elevated chronic kidney disease risk, although mechanisms remain under investigation [18].

However, lower serum creatinine is not specific to improved renal clearance, because creatinine reflects both kidney filtration/excretion and creatinine generation. Creatinine generation depends strongly on muscle mass, diet/nutritional status, and upstream creatine metabolism, and impaired hepatic function may reduce creatine synthesis and thereby lower creatinine production. Therefore, the observed negative PM2.5 creatinine association could plausibly reflect reduced production (e.g., via differences in muscle mass or potential liver-related pathways) rather than a direct kidney-mediated effect alone. Because we did not include liver enzyme biomarkers (e.g., ALT, AST, ALP) in this analysis, we could not evaluate whether hepatic dysfunction contributed to the PM2.5 creatinine pattern; future work integrating liver biomarkers would help distinguish production-driven from clearance-driven explanations.

Other biomarkers

For markers such as CRP, WBC, and cholesterol fractions, the ecological class-mean patterns were weak or flat (Table 1; Figure 1). At the individual level, several of these biomarkers reached statistical significance after BH–FDR control (Table 2) for example, CRP ($r = 0.017$, $q = 1.06\times10^{-11}$), WBC ($r = 0.010$, $q = 2.56\times10^{-5}$), HDL ($r = -0.023$, $q = 4.26\times10^{-21}$), and LDL ($r = -0.010$, $q = 9.93\times10^{-5}$) yet the effect sizes were uniformly very small ($|r| \approx 0.00–0.02$), suggesting limited practical relevance. Other markers (e.g., LDL cholesterol, Apo B, lipoprotein(a), HbA1c, cystatin C) also showed statistically detectable but tiny trends, whereas lymphocytes, glucose, triglycerides, and monocytes did not pass FDR < 0.05. This overall pattern many statistically significant yet minimal associations alongside several nulls aligns with prior mixed findings, where differences in study design, populations, and residual confounding contribute to inconsistency across inflammatory and lipid markers [23].

Limitations

This cross-sectional analysis precludes causal inference and strict temporality. PM2.5 exposure was assigned from residential address and grouped into WHO 2021 PM2.5 classes; this does not capture time–activity patterns, mobility, indoor sources, or microenvironmental variability and may attenuate non-linear dose–response (likely biasing effects toward the null). Results are unadjusted for potential confounders (e.g., age, sex, BMI, smoking, medications, comorbidities, season, socioeconomic factors); the BH-FDR control used in Table 2 addresses multiplicity but not confounding. In addition, serum creatinine is influenced by non-renal determinants (e.g., muscle mass, diet/nutritional status, and creatinine production), and we did not include liver enzyme biomarkers (ALT, AST, ALP) to assess whether hepatic pathways contributed to the negative PM2.5 creatinine association; therefore, interpretation of this finding should remain cautious. Class sizes were imbalanced (e.g., sparse IT-2), and biomarkers were measured once, which may affect precision and introduce measurement error. Finally, the UK Biobank's volunteer composition may limit generalizability to other populations.

CONCLUSIONS

In this large population-based cohort, higher WHO 2021 PM2.5 class was associated with increased platelet counts and decreased serum creatinine. These signals were consistent across descriptive class-mean patterns (Table 1; Figures 1,2) and remained statistically significant at the individual level after BH–FDR correction (Table 2; platelets $r = 0.035$, $q < 0.001$; creatinine $r = -0.018$, $q < 0.001$). While several additional biomarkers reached nominal and FDR significance, effect sizes were uniformly small (most $|r| \lesssim 0.02$), suggesting limited practical impact for individual markers when considered in isolation. Taken together, the convergent platelet (↑) and creatinine (↓) trends support the growing evidence that long-term PM2.5 exposure relates to pathways relevant to cardiovascular (pro-thrombotic) and renal health. However, given the cross-sectional design, coarse exposure categorization, and unadjusted models, these findings should be interpreted as population-level associations rather than causal effects.

From a public health perspective, incorporating targeted biomarker monitoring (e.g., platelets and kidney function indices) into air-quality health assessments could aid early detection of pollution-

related physiological stress, especially in higher-exposure areas. Any risk communication should underscore that the observed associations are small in magnitude at the individual level but may be meaningful when aggregated across populations. Future work should (i) use longitudinal designs with continuous exposure estimates, (ii) fit covariate-adjusted and sensitivity models (e.g., Spearman, robust regression), (iii) examine dose–response and potential non-linearities, and (iv) explore mechanistic links (e.g., platelet activation, renal pathways).

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