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Association between Exposure to Toll Highway Air Pollution and Overweight and Obesity Risk in Dakar: A Cross-Sectional Study

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Abstract

Introduction: In sub-Saharan Africa, air pollution is a growing public health concern, primarily linked to cardiorespiratory diseases, while its role in metabolic disorders such as type 2 diabetes and obesity remains insufficiently understood. This study assessed the association between traffic-related air pollution from toll highways and body composition in Dakar's toll booth workers (Senegal). **Methods:** A cross-sectional study was conducted from March 2018 to October 2020 involving 122 employees of the Eiffage company at the Thiaroye and Rufisque toll booths. Data on sociodemographic, occupational, and lifestyle factors were collected via a structured questionnaire. Body composition was assessed using bioelectrical impedance analysis to estimate fat mass, lean mass, and visceral fat. Body mass index (BMI) was calculated. Air pollution levels were obtained from the Air Quality Management Center (CGQA). **Results:** The mean age was 35.7 ± 7.1 years, with a mean BMI of 23.5 ± 3.7 kg/m^2. Nine percent were smokers, working an average of 48 hours per week. No history of overweight or obesity was reported. Sixty-five percent had normal weight, 23% were overweight, 7% underweight, and 5% moderately obese. Men exhibited lower-than-reference lean mass, whereas women had excessive fat mass. Smoking was associated with normal lean and fat mass. BMI correlated positively with fat mass (r = 0.77) and abdominal fat (r = 0.89). PM_{10} concentrations ranged from 180 to 220 µg/m^3, and $PM_{2.5}$ from 60 to 120 µg/m^3. **Conclusion:** Chronic exposure to air pollution from high-traffic toll highways is associated to overweight and obesity by increasing fat mass.

Keywords

Air Pollution, Obesity, Body Composition, Toll Highway, Dakar

1. Introduction

Air pollution represents one of the most pressing global public health challenges. It is estimated to cause over 6.8 million deaths each year, accounting for approximately 12% of all global deaths and contributing substantially to the worldwide burden of noncommunicable diseases [1]-[3]. Chronic exposure to air pollution, whether outdoor or indoor, is now recognized as a significant risk factor for the development of numerous chronic diseases, including respiratory illnesses, type 2 diabetes, overweight, and obesity.

Sub-Saharan Africa is particularly affected by this issue [4]. In this region, pollution arises from multiple sources, often linked to specific human activities and a vulnerable environmental context. The combustion of biomass such as firewood, charcoal, and agricultural residues for cooking and heating remains the primary source of both indoor and outdoor pollution, in both urban and rural settings [5] [6]. Moreover, in many low-income countries, the absence of structured waste management systems frequently leads to open-air incineration, releasing fine particulate matter (PM) and toxic gases that significantly degrade air quality [7].

Road traffic is another major contributor to worsening air pollution in sub-Saharan Africa. The widespread importation of used vehicles often poorly maintained and equipped with outdated engines leads to elevated emissions of fine particles and nitrogen oxides (NOx). In addition, rapid and unregulated urbanization, occurring without proper planning or infrastructure, further exacerbates environmental pollution. Uncontrolled urban sprawl is closely associated with the deteriorating air quality in many cities across the region [8] [9]. In West Africa, for instance, the proportion of the population living in urban areas rose from 33% in 2008 to 42.5% in 2021 [9] [10], thereby increasing pressure on transportation systems and urban infrastructure.

In Senegal, air pollution is particularly alarming in the capital, Dakar, where pollution levels frequently exceed recommended safety thresholds. The city's vehicle fleet is largely composed of old, poorly maintained cars with outdated and inefficient combustion systems, which significantly contribute to the degradation of ambient air quality [11]. In response to this issue, the Senegalese government made substantial efforts during the 2010s to improve urban mobility, notably through the construction of new road infrastructures such as the Dakar toll highway, operated by the French company Eiffage.

This toll highway is heavily trafficked daily by thousands of vehicles, including trucks, taxis, and public transport buses running on diesel or gasoline, and represents a significant source of both air and noise pollution. Toll booth operators, in particular, experience prolonged exposure to these pollutants due to the static na-

ture of their work, which takes place in enclosed but poorly insulated cabins located adjacent to high-traffic lanes. This daily exposure, compounded by occupational physical inactivity, may promote metabolic disturbances such as overweight or obesity.

We therefore hypothesize that chronic exposure to traffic-related environmental pollution may represent a risk factor for the development of overweight and/or obesity. The objective of this study is to investigate potential associations between exposure to automobile pollution and the risk of developing overweight or obesity among Eiffage employees working on the Dakar toll highway.

2. Methods

2.1. Study Design and Participants

This was a descriptive and analytical cross-sectional study conducted between March 2018 and October 2020 in Dakar, Senegal. The study population consisted of toll booth workers employed by the French company Eiffage on the Dakar toll highway, specifically at the Thiaroye and Rufisque toll stations.

Participants were recruited systematically, following the provision of written, informed consent. To be eligible for inclusion, individuals had to be aged 18 years or older and have worked for Eiffage for at least one year, with a minimum work schedule of five days per week and eight hours per day.

Prior to participation, each subject was informed about the objectives of study and procedures, and gave their free and informed consent. Participants were invited to attend morning sessions for physical examinations and pulmonary function assessments. Data collection began with a standardized questionnaire designed to gather information on age, job tenure in toll booth operations, estimated duration of daily exposure to traffic-related air pollution, and the presence of pre-existing chronic conditions such as obesity/overweight, type 2 diabetes, asthma, or chronic obstructive pulmonary disease (COPD) prior to employment in this sector. Smoking status was also recorded.

Following the interview, a physical examination was conducted, focusing primarily on the respiratory and cardiovascular systems. Anthropometric measurements, including body weight and height, were recorded. Body mass index (BMI) was then calculated using the Quetelet formula:

BMI (kg/m^2) = weight (kg)/height^2 (m^2).

BMI categories were defined according to the World Health Organization (WHO) criteria:

1) Underweight: BMI < 18.5 kg/m^2

2) Normal weight: 18.5 ≤ BMI < 25 kg/m^2

3) Overweight: 25 ≤ BMI < 30 kg/m^2

4) Obesity: BMI ≥ 30 kg/m^2

Body composition parameters including lean mass, total body fat, and visceral fat were assessed using multifrequency bioelectrical impedance analysis (BIA) with the segmental body Bioelectrical impedance analyzer (BIA) device, (OM-

RON BF-511). This device provides segmental estimations of body composition by independently analyzing different body regions (upper limbs, lower limbs, and trunk), thereby enhancing measurement accuracy, particularly for visceral fat assessment.

Prior to each measurement, participants were asked to remove their shoes and socks and to stand barefoot on the footplate electrodes of the device. They were then instructed to grasp the hand-held electrodes located on either side of the analyzer, with arms extended horizontally in front of the body. This posture ensured optimal electrical conductivity throughout the entire body.

The BIA method relies on the application of a low-intensity alternating electric current (typically between 500 and 800 μA) through the body via so-called "source" electrodes. This current flows more readily through tissues with high water and electrolyte content, such as skeletal muscle, and is impeded by tissues with low water content, such as adipose tissue and bone. The method is based on the differences in electrical conductivity across various body compartments.

Two key electrical parameters are measured:

1) Resistance (R): This reflects the opposition to current flow through body tissues. A higher proportion of adipose tissue, which is a poor electrical conductor, results in greater resistance values.

2) Reactance (Xc): This represents the capacitive properties of cell membranes, which temporarily store electrical energy, thereby reflecting both cell membrane integrity and active cell mass.

The combination of resistance and reactance allows for the calculation of **impedance (Z)**. Based on integrated predictive algorithms, the device estimates the distribution of various body compartments:

1) Lean mass (LM): including skeletal muscle, intracellular and extracellular fluids, and organ tissue.

2) Fat mass (FM): comprising both subcutaneous and deep adipose tissues.

3) Visceral fat (VF): representing fat accumulation around abdominal organs, a critical indicator of cardiometabolic risk.

Measurements are completed within a few seconds under standardized conditions (e.g., in a fasted state or several hours after meals, with no recent physical activity or caffeine intake) to minimize variability due to hydration status.

In parallel, ambient air pollution levels in the study area were continuously monitored over the period 2018 to 2020, based on data collected by the Dakar Air Quality Management Center (Centre de Gestion de la Qualité de l'Air, CGQA). These data included daily average concentrations of particulate matter ($PM_{2.5}$ and PM_{10}), nitrogen dioxide (NO_2), ozone (O_3), and sulfur dioxide (SO_2), allowing for accurate estimation of population exposure to ambient air pollutants in the study zones.

2.2. Statistical Analysis of the Data

Qualitative variables were expressed as proportions, while quantitative variables

were presented as means ± standard deviations. An analysis was also conducted to assess the association between overweight or obesity (as the dependent variable) and independent variables such as personal characteristics (e.g., sex, weight, height) and smoking status, using Fisher's exact test.

The odds ratio (OR) with a 95% confidence interval (CI) was calculated to estimate the risk of altered body composition between smokers and non-smokers. Statistical analyses were performed using GraphPad Prism version 6 and p-values were calculated to assess the statistical significance of the findings. A p-value of less than 0.05 was considered statistically significant.

3. Results

3.1. Participant Characteristics and Body Composition Profile

Participant characteristics and body composition profile participant characteristics are presentetd in Table 1 and body composition profile is detailed in Table 2. The study population had a mean age of 36 ± 7.14 years, with ages ranging from 22 to 59 years. The average body mass index (BMI) was 23.6 ± 3.68 kg/m^2. Regarding tobacco use, 9% of participants were current smokers, 9% were former smokers, and 82% had never smoked. Participants reported an average work schedule of 8 hours per day and 6 days per week (Table 1).

Based on BMI classification, 65% of individuals had a normal weight status, while 23% were overweight, 7% were underweight, and 5% presented with moderate obesity. In male participants, the mean percentage of lean body mass was below the reference threshold of 40%, indicating a reduced muscular component. However, both mean total body fat and abdominal fat levels remained within established normative values.

Among female participants, the mean lean body mass was 26.77%, which is lower than the reference range of 29% to 33%. Conversely, the mean body fat percentage was 36.08%, exceeding the upper limit of the normal range (21% - 35%). Despite this, abdominal fat measures in women were within standard reference limits (Table 2).

Distribution of Body Composition Parameters in the Study Population (Figure 1)

In our study population, 53% of participants exhibited high body fat levels, while 42% had normal fat levels, and only 5% presented with low body fat.

Regarding lean mass, 69% of individuals were below normal values, 20% had lean mass within the normal range, and 11% showed elevated levels.

As for visceral fat, the majority of participants (94%) had values within the normal range, whereas 6% exceeded the recommended thresholds.

3.2. Association between Smoking and Body Composition Profiles

The data from the association analysis between smoking and the risk of overweight/obesity and body composition disorders are presented in Table 3. No statistically significant differences were observed between smokers and non-smokers

across the categories of Body Mass Index (BMI). However, notable distinctions emerged regarding body composition, particularly fat mass and lean mass.

Smokers were significantly less likely to have high fat mass and more likely to present with normal fat mass compared to non-smokers. Specifically, they were nearly twice as likely to have normal fat mass (OR = 1.912; 95% CI: 1.089 - 3.356; p = 0.033), and their risk of having high fat mass was significantly lower (OR = 0.460; 95% CI: 0.261 - 0.812; p = 0.0104).

With respect to lean mass, smokers were significantly less likely to have low lean mass (OR = 0.334; 95% CI: 0.186 - 0.599; p = 0.0003) and were approximately four times more likely to have normal lean mass compared to non-smokers (OR = 3.995; 95% CI: 2.077 - 7.682; p = 0.0001).

Finally, no significant differences were found between smokers and non-smokers in terms of abdominal fat distribution.

Pollution Levels in the Study Area, Figure 2

The chart below illustrates the monthly concentrations of PM_{10} and $PM_{2.5}$ in 2019 measured at the Thiaroye and Rufisque toll stations. The values are compared against the World Health Organization (WHO) guidelines and Senegalese national air quality standards [7].

Environmental data collected from the study sites specifically the Thiaroye and Rufisque toll stations revealed high levels of air pollution. Monthly average concentrations of particulate matter with a diameter less than 10 micrometers (PM_{10}) significantly exceeded both the World Health Organization (WHO) annual guideline value of 20 μg/m³ and the Senegalese national standard (NS-05-062), which allows up to 80 μg/m³ on an annual basis. Similarly, fine particulate matter ($PM_{2.5}$) levels remained elevated, surpassing the WHO reference value of 10 μg/m³ and the national standard of 25 μg/m³. In 2019, monthly PM_{10} concentrations ranged from 180 to 220 μg/m³, while $PM_{2.5}$ levels fluctuated between 60 and 120 μg/m³ in these areas [12].

Table 1. Participant characteristics parameters.

Variables	Mean ± SD	[Min - Max]
Age (years)	36 ± 7.14	[22 - 59]
Weight (kg)	73 ±1 2.3	[49 - 105]
Height (cm)	176.3 ± 8.4	[157 - 196]
BMI (kg/m²)	23.6 ± 3.68	[16.3 - 31.7]
Current smokers (%)	9	-
Former smokers (%)	10	-
Non-smokers (%)	81	
Workdays per week (days)	5. 6 ± 0.40	(5 - 6)
Working hours per day (h)	7.85 ± 0.54	(7 – 9)

Table 2. Body composition parameters.

	Mean ± SD	[Min - Max]
Study population		
Lean mass (%)	33.7 ± 6.3	[19.1 - 46.9]
Fat mass (%)	25.4 ± 10.3	[7.4 - 51.8]
Abdominal fat (%)	6.3 ± 3.6	[1 - 15]
Women population		
Lean mass (%)	26.77 ± 5.3	[19.1 - 40.2]
Fat mass (%)	36.08 ± 9.4	[11.9 - 51.8]
Abdominal fat (%)	5.58 ± 2.3	[1 - 11]
Men population		
Lean mass (%)	36.05 ± 4.7	[34.3 - 46.9]
Fat mass (%)	22.09 ± 7.7	[19.6 - 38.9]
Abdominal fat (%)	6.62 ± 3.9	[5 - 15]

Table 3. Association between smoking and body composition profiles.

	Smokers	Non smokers	p-value	Odds Ratio (95% CI)
BMI				
Low	9	7	0.7953	1.314 (0.4694 - 3.678)
Normal	73	68	0.5353	1.272 (0.6916 - 2.341)
High	18	25	0.3017	0.6585 (0.3329 - 1.303)
Fat mass				
Low	9	5	0.4068	1.879 (0.606 - 5.82)
Normal	55	39	0.033	1.912 (1.089 - 3.356)
High	36	55	0.0104	0.4602(0.261 - 0.812)
Lean mass				
Low	45	71	0.0003	0.334(0.186 - 0.599)
Normal	45	17	0.0001	3.995 (2.077 - 7.682)
High	9	12	0.6455	0.725 (0.291 - 1.807)
Abdominal fat				
Low	0	0	-	-
Normal	91	94	0.5928	0.645(0.221 - 1.887)
High	9	6	0.5928	1.549 (0.53 - 4.53)

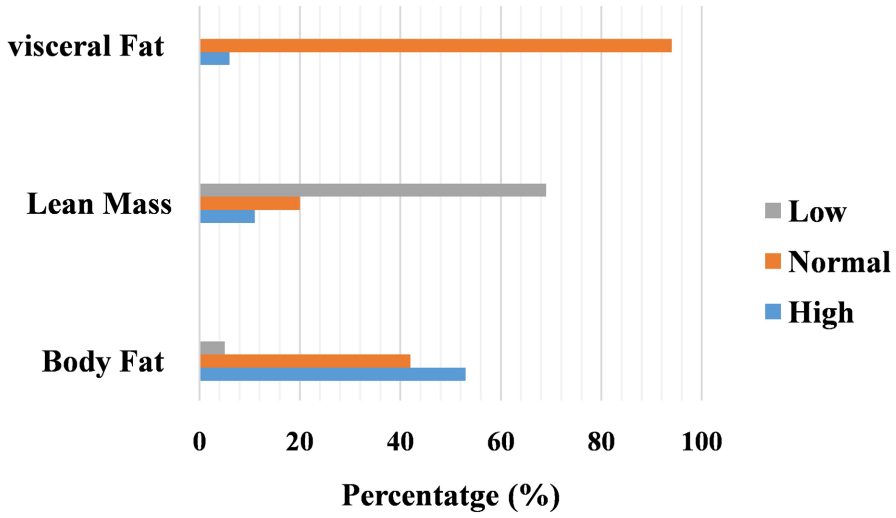

Figure 1. Distribution of body composition parameters in the study population.

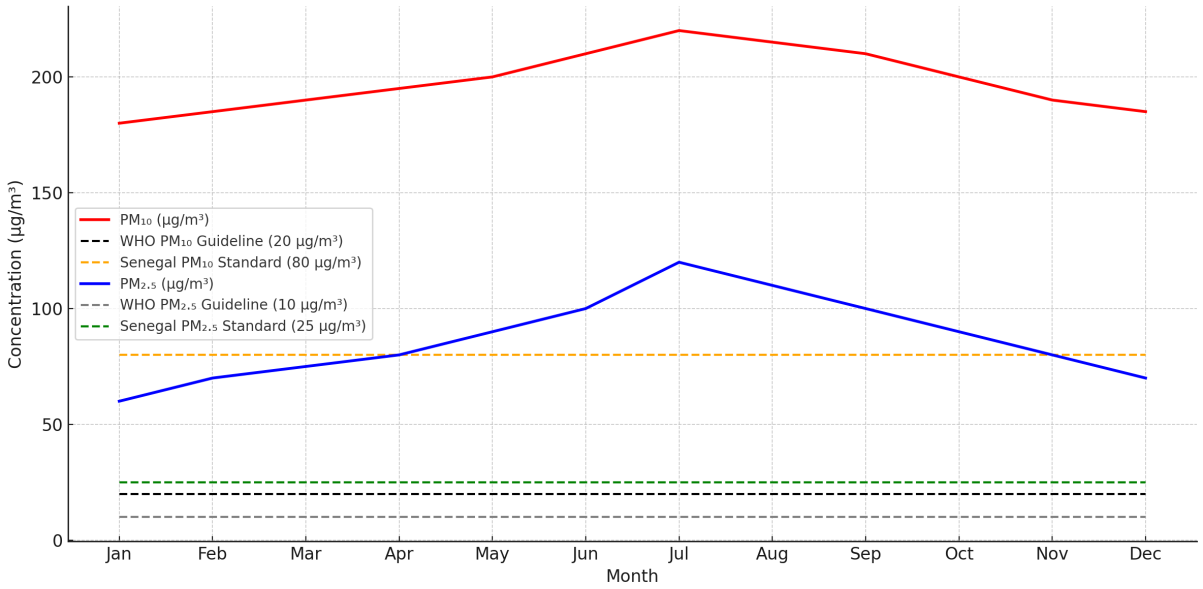

Figure 2. Monthly concentrations of PM_{10} and $PM_{2.5}$ compared to WHO and Senegalese standards.

4. Discussion

The findings of this study, conducted in a relatively young and professionally active population (mean age: 35.7 ± 7.1 years), reveal subtle but clinically meaningful alterations in body composition that are not adequately captured by conventional anthropometric indicators such as BMI. Although the average BMI fell within the normal range according to World Health Organization (WHO) guidelines (23.5 kg/m^2 ± 3.7 kg/m^2), a considerable proportion of participants (35%) were outside this range 7% were underweight, 23% overweight, and 5% moderately obese, highlighting the well-known limitations of BMI as a standalone indicator of cardiometabolic risk. Indeed, BMI fails to distinguish between FM and fat-free mass FFM, and does not account for fat distribution, both of which are

critical determinants of metabolic health.

In our study, BMI only partially reflected actual adiposity, as evidenced by strong positive correlations with total fat mass ($r = 0.77$) and visceral fat ($r = 0.89$) (data not shown). Paradoxically, these associations also suggest that lower BMI values were, in some individuals, linked to disproportionately elevated fat depots, particularly visceral fat. This underscores the complex and at times counterintuitive relationship between BMI and true body composition, emphasizing the need for more precise tools in assessing metabolic risk, especially in populations exposed to environmental stressors such as air pollution.

In men, lean mass values were lower than expected for their age, raising concern for incipient sarcopenia or insufficient muscle development. While sarcopenia is typically associated with aging, emerging evidence suggests it may also arise prematurely due to chronic systemic stressors such as low-grade inflammation and environmental toxicity [13]. Exposure to fine airborne particulate matter, particularly $PM_{2.5}$, has been shown to trigger systemic inflammation by activating oxidative stress pathways and elevating pro-inflammatory cytokines such as IL-6, TNF-α, and CRP [14] [15]. These mediators contribute to muscle catabolism via activation of the ubiquitin-proteasome and autophagy-lysosome systems, while simultaneously impairing muscle regeneration by disrupting satellite cell function [16].

Conversely, women in the study displayed fat mass percentages exceeding recommended thresholds, suggesting a predisposition to adiposity that may be amplified by environmental endocrine-disrupting compounds (EDCs) often adsorbed onto particulate pollutants. Substances such as polycyclic aromatic hydrocarbons (PAHs), dioxins, and heavy metals (e.g., cadmium) can bind to nuclear receptors like PPARγ and estrogen receptors, promoting adipocyte differentiation and central fat accumulation [17]. This pattern of fat distribution is metabolically active, associated with elevated secretion of inflammatory adipokines, and linked to insulin resistance, dyslipidemia, and systemic inflammation [18].

Interestingly, smoking status though reported by only 9% of participants was associated with higher odds of both normal lean mass and normal fat mass. Nicotine has complex metabolic effects: in the short term, it suppresses appetite, increases resting metabolic rate through sympathetic nervous system activation, and promotes lipolysis via catecholaminergic β-adrenergic pathways [19] [20]. These effects may transiently support lower fat mass and preserved lean mass. However, long-term tobacco use is associated with muscle wasting, mitochondrial dysfunction, and elevated oxidative stress, all of which contribute to sarcopenia and metabolic dysregulation [21]. The paradoxical associations observed here may reflect the predominance of moderate or recent exposure in this young adult population, rather than the cumulative effects of chronic smoking.

The observed positive correlations between BMI and both total fat mass ($r = 0.77$, $p < 0.05$) and abdominal adiposity ($r = 0.89$, $p < 0.05$) reaffirm the partial utility of BMI in population-level assessments, particularly when cross-validated

with measures of visceral fat. However, these findings also highlight the inadequacy of BMI to discern harmful fat distribution patterns. Visceral adipose tissue, in contrast to subcutaneous fat, acts as an endocrine organ, secreting pro-inflammatory cytokines (e.g., TNF-*a*, IL-6) and reducing protective adipokines like adiponectin, thereby promoting insulin resistance, vascular dysfunction, and hypertension [22]. The strong association between BMI and abdominal fat in this cohort suggests that incorporating waist circumference or waist-to-hip ratio could provide more accurate insights into cardiometabolic risk profiles in similar populations [23].

Air pollution emerged as a critical environmental factor in this study. The ambient concentrations of PM_{10} (180 μg/m^3 - 220 μg/m^3) and $PM_{2.5}$ (60 μg/m^3 - 120 μg/m^3) far exceeded the WHO-recommended limits of 45 μg/m^3 and 15 μg/m^3, respectively [24]. Chronic exposure to such pollutants is increasingly recognized as a key driver of metabolic dysfunction. Particulate matter generates reactive oxygen species (ROS), depletes antioxidant reserves like glutathione, and impairs mitochondrial bioenergetics [25]. In skeletal muscle, these effects culminate in reduced ATP synthesis, muscle fiber atrophy, and defective regeneration. In adipose tissue, $PM_{2.5}$ promotes adipocyte hypertrophy, perturbs adipokine signaling, and aggravates insulin resistance [26]. Additionally, there is growing evidence that particulate pollutants alter gut microbiota composition and increase intestinal permeability, leading to metabolic endotoxemia [27] [28]. This mechanism, involving the translocation of lipopolysaccharides into the bloodstream, has been linked to obesity, type 2 diabetes, and low-grade systemic inflammation [29].

Taken together, the patterns observed reduced lean mass in men, excess fat mass in women, and high levels of abdominal adiposity in both sexes suggest the emergence of a novel "environmental metabolic phenotype", defined as a distinct pattern of metabolic traits shaped by prolonged environmental exposures. This phenotype is characterized by disproportionate tissue distribution, subclinical inflammation, and early metabolic alterations in the absence of overt obesity. It may serve as an early harbinger of metabolic syndrome, particularly in urban populations subjected to sustained exposure to air pollution and other environmental stressors. Recognizing this phenotype calls for a more integrative model of risk assessment, one that includes environmental and occupational exposures alongside conventional clinical metrics [30].

From a public health perspective, the findings underscore the need to adopt a multidimensional approach to metabolic health screening and prevention. First, reliance on BMI alone should be avoided; instead, tools such as bioelectrical impedance analysis (BIA) or dual-energy X-ray absorptiometry (DEXA) should be implemented to differentiate fat from lean mass. Second, environmental exposure assessments, whether through personal monitoring devices or geospatial pollution mapping should be incorporated into primary care and occupational health protocols. Third, interventions must account for sex-specific susceptibilities to environmental toxins and their distinct metabolic consequences. Finally, reducing air pol-

lution through environmental policies could yield far-reaching benefits by attenuating the early onset and progression of non-communicable metabolic diseases.

In conclusion, this study sheds light on the intricate interplay between environmental exposures, body composition, and metabolic risk. It emphasizes the necessity of moving beyond traditional anthropometric indices to embrace a more nuanced, context-sensitive approach to public health and clinical risk stratification.

5. Conclusion

This study suggests the emergence of an environmental metabolic phenotype characterized by an imbalance between reduced muscle mass and excess fat mass, likely linked to prolonged exposure to air pollution. This phenotype may increase the risk of cardiometabolic disorders, even in individuals whose BMI remains within normal ranges. Longitudinal studies are needed to confirm these observations and better understand their long-term consequences. Furthermore, recognizing and addressing this phenotype through appropriate public health strategies and strengthened environmental regulations is essential to limit the metabolic impact of pollution.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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