

The Role of Air Pollution in Metabolic Syndrome: Mechanisms, Epidemiology, and Policy Implications

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Abstract

Metabolic Syndrome (MetS) is a cluster of conditions, including insulin resistance, obesity, dyslipidemia, and hypertension, and together pose a major risk of developing cardiovascular disease and type 2 diabetes. Recent research throws light on air pollution as one of the most important environmental factors contributing to the increase in MetS prevalence. MetS is pathophysiologically linked to key pollutants such as particulate matter (PM2.5 and PM10), nitrogen dioxide (NO₂), and ozone (O₃), via various pathways, including oxidative stress, systemic inflammation, endothelial dysfunction, deoxyribonucleic acid (DNA) methylation, and others. Disruption of metabolic dysregulation via these pathways leads to abnormal lipid and glucose metabolism tolerance and hormonal regulation.

Epidemiological studies confirm a very high correlation between total exposure to air pollutants and MetS prevalence, especially among urban and disadvantaged populations. Co-exposure to air and noise pollution can potentiate MetS risk, and this adds evidence to the need for environmental health strategies that integrate air, food, and noise pollution exposures. In fact, pollution exposure is also unequally distributed to vulnerable groups, such as children, poor, and minority communities, suggesting that quick and comprehensive public health action is urgently required. Strict air quality standards, innovative urban planning, and targeted policy measures are necessary to mitigate the health burden of air pollution. Individual vulnerability may be further reduced with public health campaigns regarding protective behaviors and dietary strategies. To inform future interventions, it is critical to bridge gaps in our understanding of the complex interactions between air pollution and metabolic health.

Keywords: metabolic syndrome, air pollution, epigenetics

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Metabolic Syndrome (MetS) is a cluster of conditions that together elevate the risk for heart disease, stroke, and type 2 diabetes, including increased blood pressure, high blood sugar, excess body fat around the waist, and abnormal cholesterol levels. Wide interest exists in the potential risk factors for the recent dramatic increase in MetS prevalence, where environmental factors, in particular air pollution, have drawn particular attention. Respiratory and cardiovascular diseases have been linked to substances like particles of PM2.5, PM10, NO₂, SO₂, and ozone (O₃) air pollution. There are an increasing number of reports linking long-term exposure to air pollution with MetS.^{1,2} For instance, a meta-analysis showed that a prolonged exposure to fine particulate matter (PM2.5) was associated with an increased risk for MetS.¹

There are various and complex mechanisms by which air pollution may contribute to MetS. Among possible pathways, one is DNA methylation, which is an epigenetic change in which DNA methylation controls the gene expression without affecting DNA sequence.¹ Air pollutants exposure could affect susceptibility of the cells to changes in DNA methylation patterns and, in turn, may promote the metabolic dis-regulation.² This review summarized the fact that air pollution influences DNA methylation, which may underlie its effects on MetS. The knowledge of the relationship between environmental exposures and regulation of genetics is important for public

health. DNA methylation can epigenetically modify gene activity without altering the DNA sequence. Research has shown that air pollution exposure is also associated with decreased DNA methylation levels which can have an effect on how genes are regulated.³

Epigenetic changes may lead directly to the development of metabolic syndrome through remodeling of metabolic pathways and an increased susceptibility to metabolic disease^{2,3}. Because environmental pollution has been rising all around the world and is so important to human health, while MetS prevalence has been steeply on the rise, it is imperative to understand the mechanisms by which environmental pollution may contribute to MetS.³ Developing effective public health strategies capable of reducing the impact of air pollution on metabolic health requires this knowledge.^{1,4}

The study evaluates available research that explores the impact of air pollution on DNA methylation as well as its connection to Metabolic syndrome disease development. Findings from this study can benefit environmental health researchers and public health policy makers and medical professionals who need to understand environmental pollutants' impact on metabolic health. Research into these mechanisms between air pollution and metabolic disorders has become essential because both conditions are escalating globally thereby demanding public health strategies which effectively address the pollution-metabolic link.

This review focuses on integrating existing research on air pollution and epigenetic impacts toward metabolic health to identify forthcoming research domains. New preventive measures based on our understanding of environmental pollution effects on MetS will help reduce metabolic health risks caused by pollution. More investigation must be conducted to identify the underlying mechanisms and create specific approaches to decrease the increased MetS risk stemming from air pollution exposure.

Objective

The research examines MetS developments produced by air pollution through analysis of biological pathways and studies of public records as well as policy recommendations. The research analyzes four main pollutants PM2.5, PM10, NO₂, and O₃ to examine why they generate oxidative stress along with systemic inflammation while affecting endothelial function and epigenetic change that causes metabolic dysregulation. The research emphasizes both the unfavorable conditions faced by vulnerable populations such as low-income and minority communities and the requirement of combined environmental health programs to reduce metabolic health effects from airborne pollution.

Air Pollution and Metabolic Dysregulation

The connection between air pollution and metabolic dysregulation has been extensively studied, revealing significant health implications. Metabolic dysregulation includes a variety

of conditions such as insulin resistance, dyslipidemia, and obesity, which are hallmarks of Metabolic Syndrome (MetS). Long-term exposure to ambient air pollutants such as particulate matter (PM2.5 and PM10), nitrogen dioxide (NO₂), and polycyclic aromatic hydrocarbons (PAHs) has been associated with the development of these metabolic disturbances.^{4,5}

The health impacts of long-term exposure to air pollution are profound, particularly for metabolic syndrome (MetS) and its components. Research shows that an intricate convergence of mechanisms, encompassing endothelial dysfunction, oxidative stress, inflammation, epigenetic remodeling, and hormonal effects, act in concert to derail vascular biology and compromise insulin sensitivity and lipid metabolism. MetS and related diseases, especially diabetes, obesity, and cardiovascular diseases, are associated with a significantly elevated risk for chronic exposure to fine particulate matter (PM2.5), especially nitrogen dioxide (NO₂).⁵

PM2.5 exposure leads to diminished endothelial function by blocking induction of Sarcoendoplasmic reticulum Ca²⁺-ATPase2 (SERCA2) expression in the aorta and vascular relaxation. In diabetic models, therapeutic activation of SERCA2 with CDN1163 has restored vascular function and glucose tolerance. In fact, chronic exposure to air pollution is coupled with cardiovascular morbidity and mortality risk factors such as arterial hypertension and chronic obstructive pulmonary disease (COPD). People living nearby large roads and within areas with higher PM10 and PM2.5 levels are at particularly high risk.⁵

Oxidative stress and inflammation, powerful drivers of metabolic dysfunction, are induced by air pollution. Elevated reactive oxygen species (ROS) exposure in liver and adipose tissues is a result of PM2.5 exposure and oxidative damage and metabolic process impairment.^{6,7} In addition, air pollution releases pro-inflammatory cytokines such as Interleukin 1 α (IL-1 α) and Interleukin 8, worsening systemic inflammation.⁷ This accelerated metabolic disturbance involves a chronic inflammatory state, induced by insulin resistance, obesity, and altered lipid profiles with high triglycerides and low high-density lipoprotein (HDL) cholesterol.^{7,8}

Epigenetic changes including DNA methylation occur long-term following air pollution exposure, particularly in genes with important roles in lipid metabolism, fatty acid oxidation, and circadian rhythm regulation. Addition of hypermethylated and hypomethylated DNA contributes to dysfunction and insulin resistance in brown adipose tissue (BAT). Furthermore, epigenetic alterations portrayed here are not related to metabolic alterations of current but rather predisposition to chronic noncommunicable diseases to occur later in life, supporting the The Developmental Origins of Health and Disease (DOHaD) hypothesis.^{7,9} Disruption of hormonal balance by air pollution, in particular, insulin and lipid metabolism, is of considerable interest. PM2.5 exposure chronically affects glucose tolerance and insulin sensitivity through perturbations of gluconeogenesis and lipogenesis enzyme expression. Such disruptions are responsible for

metabolic imbalances, including alterations in lipid profiles, i.e., increases in cholesterol and triglyceride levels. Additionally, early life exposure to environmental contaminants can have lasting effects on hormonal regulation and increase the risk of MetS, and ultimately other metabolic disorders as well.^{10,11}

Air pollution has fairly long-lasting effects on overall health and effects in many parts more metabolically (including endothelial disturbance, oxidative stress, inflammation, epigenetic changes, and hormonal disruption). These interconnected mechanisms raise the risk of MetS and chronic diseases (diabetes, obesity, and cardiovascular conditions). An effective air quality management and public health interventions against adverse effects of air pollution is vital to address these challenges.

Evidence from Epidemiological and Cohort Studies

Studies in epidemiology and cohort have repeatedly exposed a very strong association between air pollution exposure and MetS and its individual components. The new study consolidates many such studies by finding that there are significant associations between air pollutants and increased MetS risk, as reported in a systematic review and meta-analysis.¹ For example, among 93,771 participants in Taiwan (2006–2016) in a longitudinal cohort study, long-term exposure to PM2.5 and NO₂ was strongly associated with the development of MetS, demonstrating the chronic action of air pollution in affecting metabolic health.¹²

The combined effect of environmental factors was further explored. The study of a health checkup cohort explored the effect of co-exposures to air and noise pollution on MetS risk, demonstrating that we should account for multiple environmental stressors in their impacts on disease risk.¹³ A large-scale study in China shows that geographic variations in air pollution levels also matter. In this study, involving over 10.7 million adults, we found that patients living in northern regions have a higher burden of carotid atherosclerosis — a condition closely related to MetS.¹⁴

The environmental contribution to MetS risk is clearly demonstrated by the urban-rural divide. Urban women in Beijing with high exposure to outdoor PM2.5 with or without face masks had a significantly higher risk to develop MetS than rural residents. Our findings reveal how localized pollution sources and lifestyle factors influence metabolic health.¹⁵

Exposure to air pollution and its associated risk for the development of metabolic syndrome (MetS) depends on socioeconomic factors. Studies show differences across populations, but most obviously in lower-income groups and some racial and ethnic communities, which are hit harder. Residing in these environments with higher levels of air pollution makes these groups vulnerable to MetS and its components, all the more reason to advance urgently on this public health issue.¹⁶

Air pollution exposure is unequally distributed within income groups, with low-income communities suffering the highest exposures. These inequalities were highlighted by a comprehensive analysis of air pollution exposure in the United States. Communities with lower incomes tend to live next to industrial neighborhoods and busy intersections; have less green space available; and live in older housing with bad air filtration systems. But these environmental factors, especially for these populations, greatly increase the likelihood of developing MetS, starting a vicious cycle of health and economic inequality.¹⁶

Environmental pollutants and cardiovascular and metabolic disorders are disproportionately found among racial and ethnic minorities, especially African Americans. The prevalence of MetS was 22% in the African American subset of the National Health and Nutritional Examination Survey (NHANES), and women (25%) were more affected than men (18%).¹⁷ Moreover, living below the poverty line, African American women had 1.57 times the odds of developing MetS compared to those of higher incomes. These results point out the risk that multiple factors compounded by each other represent for minority groups.¹⁷

There are dramatic differences between rural and urban levels of air pollution and health impact from air pollution. MetS appears to be more common in urban regions, where the PM2.5 is higher and comes from sources such as traffic and industrial activities. A Beijing, China study found that exposed women who live in urban environments with high outdoor PM2.5 levels were at increased risk of MetS, unless protective measures, such as wearing face masks, were taken.¹⁸ But new facets of environmental exposure assessment have revealed that suburban and rural areas, traditionally thought of as less affected areas, are also at risk, questioning the divide between rural and urban.¹⁹

The MetS air pollution exposure requires an integrated urban planning plus policy changes and actions approach to reduce air pollution exposure to tackle MetS. On the community scale, smart urban design principles play a significant role. Among these are the design of high-density, mixed-use areas where walking and cycling are encouraged, paired with increased physical activity, alongside a reduction in vehicle emissions. Building safe pedestrian and cycling greenways away from major traffic arteries will reduce exposure to pollutants even more. In addition, investing in low-emission public transit systems assists will help with overall lowering of air pollution levels in urban settings.²⁰

Policy interventions are needed too. Important measures include stricter regulations on emissions from industrial sources and emissions from vehicles and greater incentives associated with the employment of cleaner technology in order to markedly lower air pollutant levels. Programs to help residents get cleaner-burning stoves can work in areas where wood burning is common. Advanced IoT and big data

technologies can enable public health officials to monitor the air pollution levels in real time and respond swiftly when the pollution levels peak.^{20,21}

Public health campaigns can inform people of the risks of air pollution and offer ways to avert it on an individual level. This might include recommendations for optimal times and locations for outdoor activities, in response to varying pollution levels seen with the rotation of the seasons and normal everyday schedules. As air pollution can lead to oxidative stress, and this may be mitigated through encouraging diets rich in omega-3 fatty acids and antioxidants. In addition, encouraging general cardiovascular health with recurrent activity and wholesome lifestyles may minimize an individual's vulnerability to the damaging consequences of air pollution on metabolic well-being.²⁰

Direct correlation has been made with specific pollutants like PM2.5 and NO₂, and metabolic disruptions. All components of MetS, such as elevated fasting glucose and triglycerides, decreased HDL cholesterol level, and higher blood pressure, are associated with long-term exposure to PM2.5.^{12,22,23} Like NO₂ exposure, it also corresponds to an increased risk of cardiovascular diseases. Not directly related to pollution, this marker of triglycerides to HDL cholesterol (TG/HDL-C) ratio is a good marker of MetS and insulin resistance, and it may serve as a tool to assess the metabolic impact of air pollution.^{12,23}

Current Regulations and Air Quality Standards

Developed Countries

Modern societies enforce rigorous air quality control standards together with sophisticated air quality tracking systems. The Environmental Protection Agency through its National Ambient Air Quality Standards (NAAQS) enforces regulations for PM2.5, PM10, ozone and nitrogen dioxide pollutants in the United States. The system for monitoring along with enforcement operates through a comprehensive set of regulations.²⁴ All vehicles throughout the South Korean territory comply with one set of uniform emission standards which apply to every vehicle and fuel regardless of type. The emissions rules in Thailand apply different standards to vehicles running on petrol and diesel fuel.²⁵ Hong Kong focuses on improving Indoor Air Quality (IAQ) especially in urban environments by establishing strict ventilation regulations which minimize health problems.²⁶

Developing Countries

Many developing countries face challenges in enforcing air quality standards, despite having monitoring systems in place. Thailand, for instance, has a regulatory framework for air pollution control, yet recent studies indicate that current PM2.5 standards may not be sufficient to protect public health, especially in high-risk areas like Chiang Mai.²⁷ The country also differentiates its vehicle emission standards by fuel type and weight classification, unlike South Korea's uniform

approach.²⁵ Similarly, Vietnam has separate emission standards for petrol and diesel vehicles, but struggles to implement consistent nationwide air quality regulations.²⁸ These countries often encounter difficulties in balancing environmental policies with economic growth, leading to regulatory gaps.

Emerging Economies with Progressive Policies

Several countries that are developing their economies have successfully improved their air quality management practices. Long-term PM10 reduction through successful efforts has been achieved in the Brazil metropolitan area known as São Paulo Metropolitan Area (MASP). Regulatory measures need to continue addressing efforts against tropospheric ozone because this air pollutant continues to be of increased concern.²⁹ China has established progressively stringent air quality regulations that affect its major cities including Beijing as well as Shanghai. The cities have implemented stricter air pollution regulations than other developing countries as China resolves to tackle its severe pollution crisis^{26,28}.

Comparative Analysis and Key Insights

An analysis between these countries demonstrates separate standards for regulation. The regulations for vehicle emissions follow different paths in each country because South Korea maintains uniform requirements but Thailand and Vietnam separate vehicles according to their fuel types and weight.^{25,28} Developed nations maintain tighter air quality standards than Thailand and Vietnam which are presently enhancing their air pollution control systems.^{27,30} The priority level for Indoor air quality (IAQ) stands at the forefront of Hong Kong yet many developing countries prioritise this less than other pressing concerns.²⁶

The greatest challenge we face today stems from the negative consequences that air pollution causes for community members. Current research indicates that public health suffers from PM2.5 pollutants even below the regulatory thresholds, particularly in children from rural Northern Thailand.²⁷ People who experience long-term air pollution exposure alongside restricted medical care are at risk of increased health risks from pollution according to scientific studies.³⁰ The reduction of air quality disparities must incorporate factors related to population vulnerability and exposure along with standards which protect everyone.

Proposed Interventions: Urban Planning and Design

A multi faceted approach is needed to combat the health impacts of air pollution. That's when smart urban planning and design can make all the difference.²⁰ High density, multi use areas where we can walk or cycle are promising in reducing reliance on vehicle and absolute emissions. Safe pedestrian and cycling greenways located away from major traffic arteries will also minimize exposure to noxious carcinogenic pollutants. Moreover, investments in low emission public transit systems reduce urban air pollution to levels far below

where they otherwise would be, improving air quality and public health.^{20,21}

Health Equity Measures

Protecting vulnerable populations means addressing inequities in air pollution exposure. Interventions should particularly aim to reach rural school children and marginalised groups²⁷ by providing resources that may help mitigate exposure and its health impact. These involve prioritizing access to healthcare and promoting knowledge of the harmful effects of exposure to air pollution and the use of community specific strategies to ensure their health is maintained.

Policy Recommendations

We need stronger policies to address air pollution. There should be stricter emission standards on cars and industries and these should be uniformly applied²⁸. With the advances in IoT and big data, it becomes possible to undertake improved, real time air quality monitoring and real time tracking and rapid interventions when pollution spikes occur.³¹ Also more public health campaigns are needed to educate the public on how to minimize exposure as well as give recommendations on how to enjoy outdoor activities at times when the pollution is lower.³⁰

Regional Collaboration and Indoor Air Quality

Another important way to fight back against air pollution is to improve indoor air quality. Indoor air quality management strategies should be comprehensive in public building and educational institutions.^{26,32} Further promotion of sustainable building practices, including use of low emitting materials, will contribute to improving indoor air quality.³³ Moreover, proper health impact assessments regularly conducted can ensure that people from all segments of the population get the benefit of air pollution reduction efforts.³⁴ As air pollution often extends beyond borders, collaboration across borders is critical to developing cohesive standard and policies across the region to tackle this problem.

The Need to Standardize Air Quality Index (AQI) for Global Public Health

Air Quality Index (AQI) systems differ across countries, resulting in inconsistent risk assessments and public health responses. For instance, Thailand's AQI includes a "Very Good" (blue) category that doesn't exist in international standards, potentially underestimating pollution risks.³⁵ Furthermore, pollutant concentration thresholds that should be classified as hazardous under WHO and US Environmental Protection Agency (EPA) guidelines remain at lower-risk levels in Thailand.³⁶

A standardized AQI would ensure uniform health advisories, enhance cross-border environmental policies, and increase public awareness. The US AQI and WHO guidelines serve as

benchmarks, employing stricter PM2.5 and PM10 limits that better reflect health risks.³⁷ Countries like Thailand, where PM2.5 thresholds are set higher, risk delaying essential public health interventions.³⁸

Global AQI standardization would enable governments to implement more effective pollution control measures and provide citizens with accurate, comparable data to make informed health decisions.³⁹ Harmonizing air quality metrics across nations is crucial in addressing pollution-related diseases and climate challenges on a global scale.

Conclusion

The linkage of air pollution and Metabolic Syndrome (MetS) underscores the interplay between environmental and metabolic health. MetS is induced through oxidative stress, inflammation, endothelial dysfunction, and epigenetic changes that collectively disrupt glucose and lipid metabolism. Air pollutants including PM2.5, NO₂, and ozone are responsible for this. Epidemiological evidence suggests that exposure to long-term air pollution is associated with increased MetS risk, with the effects most severe on urban and socioeconomically disadvantaged populations.

A multifaceted approach is necessary to address this growing public health challenge. This involves setting stricter air quality standards, enhancing urban design that reduces air pollution exposure, and developing low-emissions public transit systems. Targeted interventions to vulnerable populations such as public health campaigns to raise awareness and promote protective behaviors are also crucial. Further study of how the particular molecular pathways link between air pollution and MetS will be needed to develop innovative therapeutics and preventive strategies.

Integrated solutions based on environmental, urban planning, and policy can mitigate the impact of air pollution on metabolic health, reduce health disparities, and improve overall public well-being. Not only is addressing this an environmental priority, it's a crucial step toward promoting health equity and reducing the global burden of chronic disease.

Recommendation

A successful strategy to combat public health risks between air pollution and MetS depends on policymakers implementing multiple measures of better air quality rules alongside planned urban development and specific health intervention methods. Better emission controls combined with additional green space areas and promotion of active transportation systems will substantially decrease people's contact with dangerous pollutants. Public health initiatives should deliver education campaigns to educate at-risk individuals on how they can protect themselves from air pollution through dietary changes along with avoidance techniques. Further research is needed to refine our understanding of the molecular pathways linking air pollution to metabolic disorders, enabling the development of innovative preventive and therapeutic strategies.

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