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The Air We Breathe: Exploring the multifaceted impacts of Air Pollution on health and disease

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Abstract

Background: Air pollution poses a significant global health risk, affecting a wide range of conditions, including skin aging, atopic dermatitis, cardiovascular diseases, respiratory disorders, and cancer. Recent studies reveal its role in accelerating aging and exacerbating chronic conditions through mechanisms like oxidative stress and systemic inflammation. However, gaps remain in understanding pollutant-specific effects and their cumulative impact on health.

Methods: This review synthesizes findings from recent studies examining the effects of air pollution on skin and systemic health. A comprehensive analysis was conducted across multiple health conditions, with a focus on pollutant-specific mechanisms, including oxidative stress, DNA damage, immune dysregulation, and inflammatory responses.

Results: Findings highlight strong correlations between exposure to pollutants, such as PM2.5, SO2, NOx, and VOCs, with adverse health outcomes. Pollutants exacerbate conditions like wrinkles, atopic dermatitis, thrombosis, and lung cancer. Interestingly, some pollutants, like ozone, showed mixed effects on specific skin conditions, emphasizing the need for nuanced analysis.

Conclusion: Understanding the multifaceted impact of air pollution underscores the urgent need for targeted interventions and policies to mitigate health risks. Further research is essential to develop precise strategies to address the pollutant-specific effects and protect vulnerable populations.

Keywords: Air pollution, Skin aging, Atopic dermatitis, Cardiovascular health, Oxidative stress, Respiratory disease, Lung cancer

Introduction

Air pollution represents a critical global health challenge with far-reaching consequences across multiple domains of human health, including dermatology, cardiology, respiratory diseases, and

oncology (Hassan Bhat et al., 2021). The pervasive presence of pollutants such as particulate matter (PM2.5 and PM10), nitrogen oxides (NOx), sulfur dioxide (SO2), volatile organic compounds (VOCs), and ozone (O3) has been shown to significantly impact various physiological systems. Understanding these impacts is vital, not only for mitigating health risks but also for addressing the broader public health burden associated with air pollution (Bikis, 2023).

Research has increasingly highlighted the detrimental effects of air pollution on skin health, with pollutants linked to accelerated skin aging. Studies, such as the one conducted at Kaohsiung Medical University Hospital, have identified strong correlations between pollutants and adverse skin conditions, including wrinkles, pigmentation issues, and poor texture (Huang et al., 2022). These findings underscore the need to explore pollutant-specific mechanisms that influence skin aging, particularly in vulnerable populations.

In dermatology, air pollution has also been implicated in exacerbating atopic dermatitis (AD). The underlying mechanisms involve oxidative stress, immune dysregulation, and epidermal barrier dysfunction, aggravated by exposure to both outdoor and indoor pollutants. Despite advancements, questions remain regarding the precise causative relationships and effective interventions (Lai et al., 2023).

Cardiovascular health is similarly impacted by air pollution, with conditions such as thrombosis, dyslipidemia, atherogenesis, and ischemic heart disease strongly associated with exposure to PM2.5 and other pollutants. Evidence suggests that air pollution promotes systemic inflammation, oxidative stress, and endothelial dysfunction, which contribute to heightened cardiovascular morbidity and mortality (Alexeeff et al., 2021). However, the specific roles of different pollutants and their interactions remain inadequately understood, highlighting a significant research gap.

Respiratory diseases, including asthma and lung cancer, further exemplify the health consequences of air pollution (Santos et al., 2021). Traffic-related pollutants, second-hand smoke, and indoor contaminants exacerbate asthma symptoms, particularly in children and the elderly (Tiotiu et al., 2020). For lung cancer, especially non-small cell lung cancer (NSCLC), air pollutants have been identified as key contributors through mechanisms such as DNA damage, oxidative stress, and epigenetic changes (González-Ruíz et al., 2023). The role of extracellular vesicles (EVs) in mediating tumor progression presents a novel area of investigation, with promising implications for diagnostics and therapeutics.

Despite the breadth of research, critical gaps persist in understanding how specific pollutants impact diverse populations and interact with other environmental factors. Questions about dose-

response relationships, long-term exposures, and the combined effects of multiple pollutants are particularly pressing. Furthermore, the impact of climate change and increasing wildfires on pollutant levels and associated health risks necessitates urgent attention (Alahmad et al., 2023).

This article aims to provide a comprehensive synthesis of the current knowledge regarding air pollution's effects on human health, focusing on novel findings and identifying areas for future research. By integrating insights from dermatology, cardiology, and respiratory health, this work seeks to inform targeted interventions and public health policies that address the multifaceted challenges posed by air pollution. The findings presented here are not only significant for advancing scientific understanding but also crucial for guiding effective mitigation strategies to safeguard global health.

Methods

This comprehensive review evaluated the impact of air pollution on various health outcomes, focusing on dermatological, cardiovascular, respiratory, and oncological conditions. To ensure a broad and thorough understanding of the subject, a systematic search was conducted across reputable databases, including PubMed, using relevant keywords such as air pollution, skin aging, atopic dermatitis, thrombosis, dyslipidemia, asthma, cancer, and related terms.

The inclusion criteria were designed to incorporate a wide range of studies, including clinical trials, epidemiological research, mechanistic studies, meta-analyses, and systematic reviews. This comprehensive approach enabled a synthesis of evidence across diverse health conditions and pollutant types, such as particulate matter (PM2.5, PM10), nitrogen oxides (NOx), sulfur dioxide (SO2), ozone (O3), and volatile organic compounds (VOCs).

A total of 20 recent and high-quality studies were selected based on their methodological rigor, relevance to the outlined health outcomes, and detailed analysis of air pollution's effects on disease mechanisms. These studies were critically reviewed to examine the direct and indirect pathways through which pollutants contribute to oxidative stress, inflammation, immune dysregulation, and other biological disruptions linked to adverse health effects.

This review aimed to synthesize findings from diverse fields to provide a cohesive understanding of air pollution’s role in disease progression. Special emphasis was placed on studies exploring mechanisms of damage, including oxidative stress, immune modulation, and the exacerbation of pre-existing conditions. By integrating data from experimental and

observational research, the review highlights significant gaps in knowledge and opportunities for future investigation.

To ensure the reliability and accuracy of the conclusions, standardized evaluation criteria were applied to assess the quality of each study. This rigorous methodology allowed for the identification of consistent patterns and significant findings, ensuring a robust synthesis of the complex relationships between air pollution and human health.

Results

The findings of this review are presented across multiple sections, each addressing the distinct impacts of air pollution on various health conditions. These sections explore the mechanisms by which air pollution influences skin aging, atopic dermatitis, cardiovascular conditions like thrombosis and dyslipidemia, as well as respiratory and systemic diseases such as asthma and lung cancer. This structured approach provides a comprehensive understanding of how specific pollutants contribute to these health outcomes, highlighting pollutant-specific effects and identifying potential areas for targeted interventions and mitigation strategies.

1. Dyslipidemia

Dyslipidemia, a key contributor to atherosclerosis and cardiovascular disease, is increasingly linked to air pollution exposure. Epidemiological and mechanistic studies provide substantial evidence that air pollution adversely affects blood lipids, though findings vary due to differing methodologies. Air pollution impacts blood lipid profiles through mechanisms such as oxidative stress, inflammation, insulin resistance, mitochondrial dysfunction, hypothalamic hormone changes, and epigenetic modifications. These disruptions are biologically plausible, as air pollution is also associated with metabolic diseases like fatty liver disease, diabetes, and obesity conditions commonly accompanied by dyslipidemia (Zhang et al., 2023).

Particulate and gaseous pollutants exacerbate lipid imbalances by triggering systemic inflammation and impairing metabolic pathways. Vulnerable populations, such as the elderly and individuals with high-fat diets, obesity, or diabetes, are at heightened risk of experiencing the harmful effects of air pollution on lipid health (Gaio et al., 2019).

Despite the growing body of evidence, gaps remain in understanding the role of indoor air pollution, specific gaseous pollutants (e.g., NO2, SO2, CO, O3), and population susceptibility

to these effects. Additionally, challenges in studying the effects of mixed air pollutants complicate risk assessments. Future research must focus on these areas while developing effective interventions and policies to mitigate the health risks posed by air pollution. Enhanced methodologies and targeted studies will be essential for addressing the complexities of air pollution's impact on dyslipidemia and overall cardiovascular health.

2. **Atherogenesis**

Air pollution, particularly exposure to particulate matter less than 2.5 micrometers in diameter (PM2.5), is a significant contributor to atherogenesis and cardiovascular risk. Both short-term and long-term exposure to PM2.5 have been shown to impact cardiovascular health, with chronic exposure having a greater effect on the progression and severity of atherosclerosis compared to acute exposure (Bevan et el., 2021).

Recent epidemiological studies link ambient PM2.5 exposure to the presence and progression of atherosclerotic plaques in humans. These findings are supported by animal studies conducted over the past two decades, which demonstrate that chronic PM2.5 exposure accelerates plaque development and increases the vulnerability of atherosclerotic lesions to rupture. Mechanistic investigations suggest that PM2.5 exposure induces oxidative stress, systemic inflammation, and endothelial dysfunction, which are key drivers of atherogenesis (Lechner et al., 2019).

The relationship between PM2.5 and atherosclerosis highlights the broader public health implications, as billions of individuals are exposed to PM2.5 worldwide. Future research is needed to identify the specific constituents of air pollution that contribute to these effects and to explore how factors such as the gut microbiome and adaptive immunity modulate the impact of PM2.5 on atherogenesis. These findings underscore the growing need for policies and interventions to reduce air pollution exposure and mitigate its long-term cardiovascular effects, particularly its role in atherosclerosis progression and cardiovascular events.

3. **Ischemic Heart Disease**

Ambient air pollution is a critical but often underestimated risk factor for ischemic heart disease (IHD) and atherosclerosis. Evidence shows that both acute and chronic PM2.5 exposure significantly increase the burden of coronary atherosclerosis and elevate the risk of acute ischemic coronary events. These effects are linked to underlying biological mechanisms involving oxidative stress, systemic inflammation, and endothelial dysfunction, which accelerate the development and progression of atherosclerotic plaques (Montone et al., 2023).

Mitigation strategies are critical for reducing the cardiovascular impact of PM2.5. These include societal and governmental measures to lower emissions of air pollutants, traffic noise, and greenhouse gases. Additionally, personal interventions, such as protective equipment and lifestyle modifications, alongside pharmaceutical options targeting the molecular mechanisms of PM2.5-induced damage, are promising (Virani et al., 2023).

However, to establish effective interventions, there is a pressing need for randomized controlled clinical trials that evaluate the efficacy of pollution reduction strategies and specific therapeutic agents in lowering the incidence of IHD. Emerging concepts like the exposome emphasize the interplay of multiple environmental factors, including air pollution and climate change, in driving cardiovascular morbidity and mortality. Addressing these interconnected factors is essential for global cardiovascular health improvement.

4. **Stroke Events**

Fine particulate matter (PM2.5) has well-documented effects on cardiovascular morbidity and mortality. Long-term exposure to PM2.5 is strongly associated with increased risks of ischemic heart disease (IHD) mortality, cerebrovascular mortality, and cardiovascular events such as stroke and myocardial infarction (MI). A meta-analysis of 42 studies published by December 2019 quantified these risks, finding that a 10 $\mu g/m^3$ increase in long-term PM2.5 exposure was associated with a 23% increase in IHD mortality and a 24% increase in cerebrovascular mortality. Incident stroke risk rose by 13%, while the risk of incident MI increased by 8%, though this finding was not statistically conclusive (Kulick et al., 2023).

The study highlights the greater impact of long-term PM2.5 exposure on mortality compared to the incidence of non-fatal cardiovascular events, particularly for cerebrovascular and IHD-related deaths. The insufficient data on recurrent stroke and MI events underscores the need for further research in this area. The findings build on existing evidence that establishes a causal relationship between PM2.5 exposure and cardiovascular diseases (CVDs), offering robust quantitative evidence of the strength of these associations (Alexeeff et al., 2021).

This growing body of research reinforces the critical need for public health interventions to reduce long-term PM2.5 exposure. Measures aimed at mitigating air pollution, particularly in regions with high PM2.5 concentrations, could significantly reduce the burden of cardiovascular morbidity and mortality. Further investigations into recurrent events and incident MI will help refine our understanding and strengthen strategies for managing the cardiovascular risks posed by air pollution.

5. Atrial Fibrilation

Air pollution, a leading global environmental health risk, is linked to over 5 million premature deaths annually and poses significant threats to public health. Emerging evidence indicates that exposure to air pollution, particularly particulate matter (PM2.5 and PM10), increases the risk of atrial fibrillation (AF), a common cardiac arrhythmia associated with severe cardiovascular complications.

Both short- and long-term exposure to air pollution contribute to the development of AF. The mechanisms may involve oxidative stress, systemic inflammation, and vascular dysfunction, which exacerbate cardiovascular risks. High pollution levels in certain regions are particularly concerning, highlighting the need for targeted interventions (Błaszczyk et al., 2021) .

Efforts to reduce air pollution exposure are crucial for mitigating AF-related health risks. Further high-quality studies are essential to quantify the incidence of AF related to air pollution and to assess its broader public health impact. Reducing air pollution can improve cardiovascular health and prevent associated diseases like AF globally.

6. Thrombosis

Air pollution is a significant global health issue with profound effects on cardiovascular function, including an increased risk of thrombosis. Both acute and chronic exposure to particulate matter (PM2.5) has been associated with a pro-thrombotic state, which likely contributes to the heightened cardiovascular morbidity and mortality linked to air pollution. Recent studies reinforce the view that air pollution promotes thrombosis through multiple interrelated mechanisms, including platelet activation, oxidative stress, interleukin-6 interplay with tissue factor, and the involvement of circulating microvesicles and epigenetic changes (Robertson et al., 2018).

Acute exposure to fine particulate matter shifts the haemostatic balance towards coagulation, promoting thrombus formation. While data on the effects of gaseous pollutants on thrombosis remain insufficient, evidence strongly suggests that particulate pollutants induce oxidative stress and inflammatory responses that exacerbate pro-coagulant activity. The American Heart Association has previously highlighted these mechanisms as pivotal in linking air pollution to cardiovascular mortality (Tang et al., 2016).

The complexity of the pathophysiological processes suggests that multiple pathways act in concert, influencing both the initiation and progression of thrombotic events. Individuals with

pre-existing cardiovascular diseases are particularly vulnerable to these effects. However, further research is needed to clarify the role of gaseous pollutants, long-term exposure impacts, and dose-response relationships. Additionally, comparing susceptible populations to healthy individuals will help elucidate the mechanisms and enhance preventive strategies. Overall, the evidence underscores the significant pro-thrombotic impact of air pollution, emphasizing its contribution to cardiovascular

7. **Asthma**

Asthma, a chronic respiratory disease characterized by airflow obstruction, airway inflammation, and bronchial hyperresponsiveness, is significantly influenced by air pollution. Both indoor and outdoor pollutants negatively impact asthma outcomes, particularly in children and vulnerable populations such as the elderly (Madaniyazi et al., 2021) . Traffic-related air pollution (TRAP), nitrogen dioxide (NO2), and second-hand smoke (SHS) are prominent risk factors for the development of asthma in children, though a causal relationship with adult asthma remains inconclusive.

Outdoor pollutants like ozone (O3), sulfur dioxide (SO2), carbon monoxide (CO), and particulate matter (PM) can induce asthma symptoms, exacerbations, and hospitalizations. These effects are dose- and duration-dependent, with pollutants like O3, NO2, and PM frequently associated with declines in lung function. Indoor pollutants, including smoke from wood-burning stoves and NO2 from unvented gas heaters, also exacerbate asthma symptoms, reduce lung function, and increase rates of asthma exacerbation. Additionally, SHS is linked to poor asthma control, increased exacerbations, and greater healthcare utilization (Tiotiu et al., 2020).

Active tobacco smoking worsens asthma outcomes by accelerating lung function decline, increasing exacerbations, and reducing responsiveness to corticosteroids. Indoor molds, such as Aspergillus fumigatus and Penicillium spp., are associated with severe asthma symptoms, particularly in children and older adults.

Effective asthma management aligned with current guidelines can mitigate these effects. However, global measures to reduce exposure to air pollutants are essential for improving asthma outcomes. Future research should explore the impact of combined exposures (e.g., SHS and outdoor pollutants) and identify patterns of respiratory vulnerability to better address the health challenges posed by air pollution in asthma management.

8. **Lung Cancer**

Air pollution is a significant environmental factor contributing to the development of lung cancer, particularly non-small cell lung cancer (NSCLC). Particulate matter (PM2.5), nitrogen oxides (NOx), volatile organic compounds (VOCs), and other pollutants play a key role in this process by carrying carcinogens, inducing systemic inflammation, and generating oxidative stress. These factors result in DNA damage, mutations, epigenetic changes, and the promotion of tumor growth.

Recent research highlights the role of extracellular vesicles (EVs) in lung cancer progression, including NSCLC. EVs, released by both cancerous and non-cancerous lung cells, are key mediators of intercellular communication, facilitating tumor-promoting microenvironments through inflammatory signaling pathways. Their cargo, which includes miRNAs, lncRNAs, peptides, and metabolites, reflects the molecular landscape of tumors, making EVs valuable tools for understanding cancer phenotypes and for potential diagnostic and therapeutic applications.

The increasing frequency of wildfires and climate change exacerbates air pollution, amplifying its adverse effects on respiratory health and lung cancer risk. Mitigating these impacts requires a multipronged approach, including cleaner energy sources, stricter industrial and vehicle emission standards, and enhanced air quality regulations. Research into EV-based liquid biopsies holds promise for revolutionizing lung cancer diagnosis and treatment by providing non-invasive insights into tumor biology (González-Ruíz et al., 2023).

Addressing air pollution through public health policies and innovative medical approaches can reduce lung cancer incidence and improve outcomes for affected individuals, emphasizing the urgent need for action in combating air pollution-related health risks.

9. **Skin aging:**

Skin aging is influenced by various environmental factors, including air pollution, which significantly impacts skin health. A retrospective study conducted at Kaohsiung Medical University Hospital examined the correlation between air pollutants and skin aging in 389 patients aged 30 to 74 over a 13-year period. Using the VISIA Complexion Analysis System, eight skin conditions—spots, wrinkles, texture, pores, UV spots, brown spots, red areas, and porphyrin—were quantitatively analyzed against exposure to eight air pollutants: carbon monoxide (CO), non-methane hydrocarbons (NMHC), nitrogen oxides (NO, NO2, NOx), particulate matter (PM2.5 and PM10), sulfur dioxide (SO2), and ozone (O3).

The findings showed that pollutants such as CO, NMHC, NO2, NOx, PM2.5, PM10, and SO2 were strongly correlated with negative skin conditions, including increased brown spots, wrinkles, and poor texture. Specifically, PM10 and SO2 exposure were linked to the poorest skin quality in the most affected individuals. Interestingly, ozone (O3) was uniquely associated with improved scores for texture and pores, indicating potential pollutant-specific effects on skin aging (Huang et al., 2022).

The study highlighted that individuals over 45 years old experienced more pronounced skin aging effects from air pollution, particularly from PM10 and SO2. This research underscores the complex relationship between air pollution and skin aging and the need for further studies to investigate these effects in diverse populations. Understanding pollutant-specific impacts could inform better strategies for mitigating skin damage and slowing aging caused by environmental exposures.

10. **Atopic Dermatitis**

Atopic dermatitis (AD), a chronic inflammatory skin condition, is significantly influenced by environmental factors, including air pollution. AD arises from a combination of epidermal barrier dysfunction and immune dysregulation, and exposure to air pollutants exacerbates these underlying mechanisms. Outdoor pollutants such as particulate matter (PM), volatile organic compounds (VOC), gaseous compounds, and heavy metals, as well as indoor pollutants like tobacco smoke and fungal molds, have been linked to both the onset and severity of AD (Lai et al., 2023).

The impact of air pollution on AD primarily occurs through molecular pathways involving oxidative stress. Pollutants trigger the production of reactive oxygen species (ROS), leading to DNA damage, disrupted T-cell activity, and altered cytokine production. These effects aggravate immune dysregulation and compromise the skin barrier, exacerbating AD symptoms. For instance, pollutants like nitrogen dioxide (NO2) have been associated with an increased incidence of AD, although their precise causative role warrants further investigation.

Over the past two decades, research has expanded our understanding of the relationship between air pollution and AD. This knowledge highlights the need for targeted therapies that address pollutant-induced mechanisms, such as ROS production and immune dysregulation. Future studies are essential to establish more robust causal links and guide the development of novel treatments tailored to mitigate the effects of air pollution on AD. This comprehensive approach could significantly reduce the burden of AD in affected populations.

Discussion

This study underscores the multifaceted impact of air pollution on human health, demonstrating its role in conditions ranging from skin aging to cardiovascular, respiratory, and oncological diseases. These findings align with existing evidence, highlighting air pollution as a pervasive environmental health risk requiring urgent action and further investigation.

The correlation between air pollution and skin aging emphasizes the detrimental effects of pollutants like PM10 and SO2, which were linked to worsened skin conditions such as wrinkles and brown spots, particularly in individuals over 45. Interestingly, ozone (O3) exhibited a positive association with skin texture and pores, suggesting that pollutant-specific effects warrant further exploration. The strength of this study lies in its long-term data collection and advanced analysis methods. However, its scope is limited to a single population, necessitating replication in diverse demographics.

For atopic dermatitis (AD), the exacerbation of symptoms by pollutants like PM and VOCs is well-documented. These findings connect pollutant exposure to oxidative stress and immune dysregulation, aggravating AD severity. Despite advancements in understanding these mechanisms, gaps remain in establishing direct causality, particularly for indoor pollutants like molds. Future research should focus on targeted therapies addressing pollutant-induced oxidative stress.

The cardiovascular effects of air pollution are evident in conditions like thrombosis, dyslipidemia, and atherogenesis. PM2.5 exposure was strongly associated with increased thrombosis risk, lipid imbalances, and accelerated atherosclerosis. These outcomes stem from mechanisms like oxidative stress and inflammation, which promote endothelial dysfunction. The findings emphasize the need for population-specific studies, particularly to understand the effects of mixed pollutants and long-term exposures.

Respiratory conditions such as asthma and lung cancer also demonstrate significant susceptibility to air pollution. In asthma, both indoor and outdoor pollutants exacerbate symptoms and hospitalizations, while lung cancer risks are heightened by carcinogenic compounds in PM2.5 and NOx. Emerging tools like extracellular vesicle (EV)-based diagnostics hold promise for non-invasive cancer detection and personalized therapy.

While this review highlights the widespread health impacts of air pollution, several limitations exist. Most studies focus on isolated pollutants, leaving the effects of pollutant mixtures

underexplored. Additionally, vulnerable populations, including children and the elderly, require more targeted research.

Future studies should prioritize exploring combined exposures, dose-response relationships, and novel therapeutic interventions. Policymakers must enforce stricter air quality regulations, promote clean energy, and address global environmental changes to mitigate the long-term health burden of air pollution.

Conclusion

The findings synthesized in this article illustrate the far-reaching and multifaceted impacts of air pollution on human health, with implications for dermatological, cardiovascular, respiratory, and oncological conditions. These outcomes not only underscore the urgent need for comprehensive interventions but also highlight critical pathways and mechanisms through which pollutants exacerbate disease.

Skin aging, a visible marker of environmental stress, is significantly worsened by exposure to pollutants like PM10 and SO2, particularly in individuals over 45. The unexpected protective association of ozone with skin texture emphasizes the complex, pollutant-specific effects that merit further exploration. Similarly, atopic dermatitis is profoundly influenced by oxidative stress and immune dysregulation triggered by pollutants, pointing to the potential for targeted therapies to mitigate these effects.

In cardiovascular health, air pollution's role in promoting thrombosis, dyslipidemia, and atherogenesis demonstrates the systemic nature of its impact. Mechanistic insights into oxidative stress, inflammation, and endothelial dysfunction lay a foundation for developing interventions to address the pro-thrombotic and lipid-disrupting effects of pollutants.

Respiratory diseases like asthma and lung cancer reveal the dual indoor and outdoor risks posed by pollutants, with both direct (e.g., DNA damage) and indirect (e.g., immune modulation) pathways contributing to disease progression. Innovations such as extracellular vesicle-based diagnostics in lung cancer highlight opportunities for leveraging pollutant-induced biological changes to advance clinical care.

Moving forward, the interplay of pollutant mixtures, long-term exposures, and population vulnerabilities must be prioritized in research. Policymakers must address these findings with stringent regulations, cleaner energy initiatives, and global climate actions. The collective evidence positions air pollution not just as a public health issue but as a critical determinant of

global health outcomes. Addressing its effects requires an integrated, interdisciplinary effort to mitigate its burden and protect future generations.

Disclosure

Author's Contribution

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