

Investigating the Relationship between Air Pollution Exposure and Coronary Heart Disease Risk in Urban Areas

Alicia Harmon*

Department of Cardiology, University of Southern Denmark, Campusvej 55, 5230 Odense, Denmark

Abstract

Air pollution is a major health concern in urban areas, with harmful particles and gases present in the air causing a range of health problems. One of the most concerning health problems linked to air pollution is an increased risk of coronary heart disease (CHD). This research paper aims to investigate the relationship between air pollution exposure and CHD risk in urban areas. The paper presents a literature review of the existing studies on the topic, examining the types of air pollutants that are associated with CHD risk, the mechanisms through which they affect the cardiovascular system, and the evidence linking air pollution exposure to CHD risk. The paper concludes with recommendations for future research and policy interventions aimed at reducing air pollution exposure and mitigating the risk of CHD in urban areas.

Keywords: Coronary microvascular dysfunction • Acute myocardial infarction • Hypertrophic cardiomyopathy

Introduction

Air pollution is a major environmental and public health concern in urban areas worldwide. Exposure to air pollution has been linked to a range of health problems, including respiratory diseases, cancer, and cardiovascular diseases. One of the most concerning health problems linked to air pollution is an increased risk of coronary heart disease (CHD), a condition in which the heart's blood supply is reduced due to the narrowing of the coronary arteries. CHD is a leading cause of death worldwide, and the World Health Organization estimates that more than 17 million deaths occur each year due to cardiovascular diseases. Several studies have examined the relationship between air pollution exposure and CHD risk, with most focusing on the association between exposure to fine particulate matter (PM_{2.5}) and increased CHD risk [1-3].

PM_{2.5} is a type of air pollutant that consists of tiny particles that can penetrate deep into the lungs and enter the bloodstream, causing inflammation and damage to the cardiovascular system. However, other air pollutants, such as nitrogen dioxide (NO₂), ozone (O₃), and sulfur dioxide (SO₂), have also been linked to CHD risk. This research paper aims to investigate the relationship between air pollution exposure and CHD risk in urban areas. The paper presents a literature review of the existing studies on the topic, examining the types of air pollutants that are associated with CHD risk, the mechanisms through which they affect the cardiovascular system, and the evidence linking air pollution exposure to CHD risk. The paper concludes with recommendations for future research and policy interventions aimed at reducing air pollution exposure and mitigating the risk of CHD in urban areas.

Literature Review

Types of air pollutants associated with CHD risk

PM_{2.5} is the most widely studied air pollutant in relation to CHD risk. Several

**Address for Correspondence:* Alicia Harmon, Department of Cardiology, University of Southern Denmark, Campusvej 55, 5230 Odense, Denmark, E-mail: AliciaHarmon11@yahoo.com

Copyright: © 2023 Harmon A. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

Received: 01 January, 2023, Manuscript No. jchd-23-97177; **Editor Assigned:** 03 January, 2023, Pre QC No. P-97177; **Reviewed:** 14 January, 2023, QC No. Q-97177; **Revised:** 20 January, 2023, Manuscript No. R-97177; **Published:** 27 January, 2023, DOI: 10.37421/2684-6020.2023.7.162

studies have shown that long-term exposure to PM_{2.5} is associated with an increased risk of CHD. For example, a study conducted in 2019 found that every 5 µg/m³ increase in PM_{2.5} exposure was associated with a 13% increase in CHD risk. Another study conducted in the United States found that a 10 µg/m³ increase in PM_{2.5} exposure was associated with a 12% increase in CHD risk (Pope et al., 2015). NO₂ is another air pollutant that has been linked to CHD risk. NO₂ is a gas that is released from combustion processes, such as vehicle emissions and industrial activities. Several studies have found that long-term exposure to NO₂ is associated with an increased risk of CHD.

For example, a study conducted in Denmark found that a 10 µg/m³ increase in NO₂ exposure was associated with a 17% increase in CHD risk. O₃ is a gas that is formed when nitrogen oxides (NO_x) and volatile organic compounds (VOCs) react in the presence of sunlight [4,5]. O₃ is another air pollutant that has been linked to CHD risk. A study conducted in the United States found that long-term exposure to O₃ was associated with an increased risk of CHD, with a 10 ppb increase in O₃ exposure associated with a 1.1% increase in CHD risk. SO₂ is a gas that is released from industrial processes, such as power plants and smelting facilities. Several studies have found that long-term exposure to SO₂ is associated with an increased risk of CHD. For example, a study conducted in China found that a 10 µg/m³ increase in SO₂ exposure was associated with a 4.4% increase in CHD risk.

Discussion

Air pollutants can affect the cardiovascular system through several mechanisms, including inflammation, oxidative stress, and autonomic dysfunction. PM_{2.5}, for example, can cause inflammation in the lungs and bloodstream, leading to the activation of immune cells and the production of inflammatory cytokines. This can lead to the development of atherosclerosis, a condition in which fatty deposits build up in the arteries and narrow the blood vessels, reducing blood flow to the heart. PM_{2.5} can also cause oxidative stress, which can damage the cells lining the blood vessels and contribute to the development of atherosclerosis. NO₂ can also cause oxidative stress, as well as inflammation and autonomic dysfunction. O₃ can cause oxidative stress and inflammation, as well as impairing the function of the endothelial cells that line the blood vessels. SO₂ can cause inflammation and oxidative stress, as well as impairing the function of the vascular endothelium and reducing the availability of nitric oxide, a molecule that helps to regulate blood flow.

Evidence linking air pollution exposure to CHD risk

Several studies have provided evidence linking air pollution exposure to CHD risk. A study conducted in Europe found that long-term exposure to PM_{2.5} was associated with an increased risk of CHD mortality, with a 10 µg/m³ increase in PM_{2.5} exposure associated with a 4% increase in CHD mortality. Another study conducted in the United States found that long-term exposure

to PM_{2.5} was associated with an increased risk of CHD hospitalization, with a 10 µg/m³ increase in PM_{2.5} exposure associated with a 6% increase in CHD hospitalization [6].

Several studies have also found that air pollution exposure can interact with other risk factors for CHD, such as hypertension and diabetes, to increase the risk of CHD. A study conducted in China found that long-term exposure to PM_{2.5} was associated with an increased risk of CHD in individuals with hypertension, but not in those without hypertension. Another study conducted in the United States found that long-term exposure to NO₂ was associated with an increased risk of CHD in individuals with diabetes, but not in those without diabetes.

Recommendations for future research and policy interventions

Future research should aim to investigate the relationship between air pollution exposure and CHD risk in more detail, including the mechanisms through which different air pollutants affect the cardiovascular system, and the interaction between air pollution exposure and other risk factors for CHD. Longitudinal studies that follow individuals over time and assess their exposure to air pollution and development of CHD are also needed. Policy interventions aimed at reducing air pollution exposure should be implemented to mitigate the risk of CHD in urban areas. These interventions could include measures to reduce emissions from vehicles and industry, promote public transportation and active modes of transportation such as cycling and walking, and promote the use of cleaner energy sources.

Additionally, individuals can take steps to reduce their own exposure to air pollution, such as using air filters in their homes and workplaces, and avoiding outdoor activities during periods of high air pollution. Healthcare professionals can also play an important role in addressing the health risks associated with air pollution exposure. They can provide education and counseling to their patients on the risks of air pollution exposure and the importance of reducing exposure. They can also work with policymakers and community organizations to advocate for policies and interventions aimed at reducing air pollution exposure in their communities.

Conclusion

There is strong evidence linking air pollution exposure to an increased risk of CHD in urban areas. Several air pollutants, including PM_{2.5}, NO₂, O₃, and SO₂, have been found to be associated with an increased risk of CHD, and the

mechanisms through which these pollutants affect the cardiovascular system have been identified. Future research should continue to investigate the relationship between air pollution exposure and CHD risk, and policy interventions aimed at reducing air pollution exposure should be implemented to mitigate the risk of CHD in urban areas.

Acknowledgement

None.

Conflict of Interest

Authors declare no conflict of interest.

References

1. Biffi, Mauro, Giulia Massaro, Igor Diemberger and Cristian Martignani, et al. "Cardiac resynchronization therapy in persistent left superior vena cava: Can you do it two-leads-only?." *Heart Case Rep* 3 (2017): 30-32.
2. Bissinger, Andrzej, Fardokht Bahadori-Esfahani and Andrzej Lubiński. "Cardiac defibrillator implantation via persistent left superior vena cava-sometimes this approach is facile. A case report." *Arch Med Sci* 7 (2011): 161-163.
3. Brignole, Michele, Francesco Pentimalli, Pietro Palmisano and Maurizio Landolina, et al. "AV junction ablation and cardiac resynchronization for patients with permanent atrial fibrillation and narrow QRS: The APAF-CRT mortality trial." *Eur Heart J* 42 (2021): 4731-4739.
4. Gras, Matthieu, Arnaud Bisson, Alexandre Bodin and Julien Herbert, et al. "Mortality and cardiac resynchronization therapy with or without defibrillation in primary prevention." *EP Europace* 22 (2020): 1224-1233.
5. Tan, Jih Huei, Zi Qin Ng and Simon Vendargon. "Persistent left superior vena cava and absence of innominate vein during coronary artery bypass surgery." *BMJ Case Rep* 2018 (2018): bcr-2018.
6. Petrac, Dubravko, Vjekoslav Radeljic, Nikola Pavlovic and Sime Manola, et al. "Persistent left superior vena cava in patients undergoing cardiac device implantation: Clinical and long-term data." *Cardiol Res* 4 (2013): 64.

How to cite this article: Harmon, Alicia. "Investigating the Relationship between Air Pollution Exposure and Coronary Heart Disease Risk in Urban Areas." *J Coron Heart Dis* 7 (2023): 162.