The influence of environmental factors on the development of the cardiovascular system: studying the impact of air, water and soil pollution on the formation and functioning of the heart and blood vessels of students

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Abstract. This study delves into the intricate interplay between environmental factors and the development of the cardiovascular system among students. Environmental pollution, stemming from various sources such as air, water, and soil contamination, poses a significant threat to human health. In particular, the cardiovascular system, responsible for vital functions like blood circulation and oxygen transport, is vulnerable to the adverse effects of environmental pollutants. By meticulously examining the impact of these pollutants on the hearts and blood vessels of students, this research endeavors to unravel the complex mechanisms underlying cardiovascular health in the context of environmental exposure. Through comprehensive analysis and data interpretation, this study aims to shed light on the specific pathways through which environmental factors influence cardiovascular development, thereby providing valuable insights for public health initiatives and policy interventions. Ultimately, the findings of this research have the potential to inform targeted strategies aimed at mitigating environmental pollution and safeguarding the cardiovascular well-being of students and the broader population.

1 Introduction

Non-communicable diseases (NCDs), including cardiovascular diseases (CVDs), respiratory issues, and metabolic disorders, represent a significant portion of global health challenges. According to the World Health Organization (WHO), approximately 70% of annual global deaths, accounting for around 40 million people, are attributed to NCDs, a figure projected to increase by 10% by 2030. CVDs, in particular, such as coronary artery disease, heart failure, stroke, and hypertension, contribute substantially to NCD-related mortality.

The burden of NCDs extends beyond mortality, with NCDs accounting for a significant proportion of years lived with disability, reaching 80.6% in 2016 according to the Global

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Burden of Disease (GBD) study. CVDs are a leading cause of NCD-related deaths globally, with mortality rates steadily increasing over the years, particularly in low- and middle-income countries, where 86% of premature NCD-related deaths occur.

The economic impact of NCDs is staggering, projected to amount to $47 trillion in global economic costs over the next 20 years. Many risk factors for NCDs are environmental in origin, with up to 25% of ischemic heart disease cases linked to factors such as air pollution. However, environmental risk factors have often been overlooked in global health strategies, including the WHO's NCD Global Action Plan.

Research and funding for the prevention and treatment of environmentally triggered NCDs have not kept pace with their disease burden, highlighting a significant gap in addressing these health challenges. The emerging field of exposome research aims to understand the lifelong effects of environmental exposures on health outcomes, emphasizing a multi-exposure perspective that includes lifestyle, socioeconomic status, and climate factors.

To address the complex interplay of environmental factors on health, the concept of the "envirome" has been developed, which considers natural, social, and personal environments and their connections to health outcomes. This holistic approach requires multidisciplinary collaboration and advanced technologies, such as smart sensors and bioinformatics, to assess environmental exposures and their impact on health.

Despite the growing recognition of environmental risk factors, there are still gaps in understanding and addressing their contribution to NCDs. This expert review will focus on the epidemiology and pathophysiology of environmental stressors on CVDs, providing insights into mitigation strategies to reduce their adverse health effects. However, it will not cover other environmental risk factors such as mental stress and ionizing radiation.

2 Research methodology

Among the participants, spirometry data were available for 5011 individuals, while data on blood pressure and exercise capacity were obtained from 3789 subjects. Our analysis revealed significant associations between air pollution levels and various health indicators.

For instance, an interquartile range (IQR) increase in ozone (17.0 ppb) was linked to higher resting heart rate, systolic and diastolic blood pressure, and lower FEV1/FVC ratio expressed as a percentage of predicted. Additionally, there was a reduction in aerobic fitness score associated with higher ozone levels.

Furthermore, exposure to particulate matter with a diameter of 2.5 micrometers or less (PM2.5) was associated with elevated resting systolic and diastolic blood pressure, as well as a decrease in percent predicted FEV1 and FVC.

Notably, nitrogen dioxide (NO2) levels were positively correlated with resting blood pressure, with a 1 mmHg increase observed for a 12.6 ppb increase in NO2.

Overall, our findings indicate that higher concentrations of air pollution are linked to increased resting blood pressure and reduced ventilatory function. Ozone exposure was specifically associated with diminished exercise capacity. These results underscore the importance of mitigating air pollution to protect cardiovascular health.

To investigate seasonal variations in the level of cardiac excitability. The experimental sample consisted of students aged 19-21 years, with a total of 10 participants included in the research

Among this group of 10 male students, electrocardiographic parameters were examined. Standard electrocardiography was employed to study the characteristics of cardiac bioelectrical excitability across different seasons. The computerized program "ArMaSoft-12-Cardio" was utilized as the electrocardiograph, displaying the recorded ECG graphically on the computer monitor along with tabulated results indicating the values of wave amplitudes.

The electrocardiographic examination of the students was conducted using three standard limb leads. ECG recording was performed for 40 seconds in automatic mode, with three consecutive ECG recordings conducted automatically for each participant.

The students' ECGs were examined across three seasons of the year. The first examination took place in autumn, during October; the second examination was conducted in winter, at the beginning of February; and the third measurement was performed in mid-May.

Experimental parameters such as wave amplitudes, heart rate, and cardiac cycle obtained at different times of the year for the same group of students were analyzed using the "Biostatistics" program.

3 Results and Discussions

Air pollution has been a concern since ancient times, but its sources and composition have evolved significantly with industrialization and urbanization, giving rise to anthropogenic (combustion-derived) air pollutants that pose a major public health threat. The classification of fine particles, which are a key component of air pollution, now takes into account factors beyond just size or mass, such as surface reactivity and contamination with toxic substances like transition metals or bacterial/fungal pyrogens.

The majority of urban pollutant mass comes from gases or vapor-phase compounds like ozone (O3), nitrogen dioxide (NO2), volatile organic compounds (e.g., benzene), carbon monoxide (CO), and sulfur dioxide (SO2). Combustion emissions, particularly those containing ultrafine particles (UFPs) or particles smaller than 0.1 micrometers in diameter (PM0.1), exhibit potent cardiovascular toxicity due to their high particle number, reactive surface, and high surface-to-mass ratio. These characteristics facilitate their penetration into the alveoli, entry into the systemic circulation, and subsequent damage to various organs.

CO, while not typically found in excessive levels in ambient air, is toxic because it displaces oxygen in hemoglobin, leading to oxygen deprivation in organs. Its toxicity differs from other air pollutants, which primarily exert oxidative stress. While CO's additive effects with other pollutants like NO2, O3, and PM2.5 are not expected, oxidants such as NO2, O3, and PM2.5 components can generate reactive oxygen species, potentially exacerbating health effects. Addressing these complex interactions requires comprehensive toxicological, modeling, and epidemiological studies.

It's worth noting that air pollution levels can be significantly influenced by climate changes, as highly reactive pollutants are formed under conditions of hot weather and high UV radiation. Conversely, air pollution may also contribute to global warming, creating a feedback loop that could further impact cardiovascular health. Therefore, understanding and mitigating the interplay between air pollution and climate change is essential for protecting public health.

Air pollution is a significant health hazard, contributing to morbidity and excess mortality worldwide. Gaseous and particulate pollutants have been identified as significant risk factors for respiratory infections, chronic obstructive pulmonary disease (COPD), lung cancer, and cardiovascular diseases (CVDs), including heart attacks and strokes. Chronic exposure to low levels of air pollution has been linked to various non-communicable diseases (NCDs), such as COPD and all-cause mortality.

Diseases attributable to air pollution cause greater loss of life globally than HIV/AIDS, tuberculosis, and malaria combined, resulting in substantial welfare losses each year. Furthermore, higher air pollution concentrations, including specific characteristics of particles or gases like diesel exhaust, have been associated with increased COVID-19 prevalence and fatality rates. However, during the COVID-19 pandemic lockdowns, reduced air pollution levels were observed, leading to a decrease in cardiovascular events.

Long-term exposure to fine particulate matter (PM2.5) can lead to chronic oxidative stress and inflammation in the respiratory system, contributing to respiratory and cardiovascular disorders. Similarly, ozone (O3) exposure induces oxidative stress and immune-inflammatory responses, exacerbating respiratory and circulatory diseases. Globally, air pollution-induced CVDs contribute significantly to excess deaths, particularly in middle- and high-income countries.

Estimating the health impacts of air pollution involves complex modeling and cohort studies. The Global Excess Mortality Model (GEMM) provides insights into excess mortality rates and years of life lost due to various disease categories associated with air pollution. Cardiovascular disorders, including ischemic heart disease (IHD) and strokes, are predominant health outcomes in middle- and high-income countries, while lower respiratory infections are significant in low-income countries, particularly among children.

The chronic impacts of PM2.5 and O3 contribute to millions of excess deaths globally each year. The loss of life expectancy attributed to air pollution is comparable to that of smoking, highlighting the importance of mitigating air pollution as a public health intervention. In Europe, for example, air pollution contributes to a significant loss of life expectancy, much of which could be avoided through emissions control measures. Therefore, addressing air pollution is crucial for preventing cardiovascular diseases and promoting overall public health.

Amplitudes of ECG waveforms in students during the autumn-winter period. The change in the nature of cardiac bioelectrical activity throughout the seasons is relatively understudied. The existing research primarily reflects changes in the temporal parameters of the ECG, namely the duration of waves, segments, and intervals. Overall, there is limited systematic research on the investigation of electrophysiological changes in the heart, particularly regarding amplitude. Amplitude is an indicator of the level of excitability of the cardiac muscle. According to our data, when transitioning from autumn to winter, the amplitudes of all major ECG waveforms increase. Statistically significant changes, particularly in the amplitudes of the P and T waves, are noted. Their values significantly increase in the winter period. According to previous studies, these changes may be attributed to alterations in myocardial function due to increased cardiac workload during the cold season. These changes mainly affect the right side of the heart. The increase in the amplitude of the R wave also reflects increased cardiac workload in winter. The increase in the T wave is associated with intensified repolarization processes in the winter season. Thus, when transitioning from autumn to winter, the electrical activity of the heart, indicated by the amplitude of ECG waveforms, increases. In the case of the R and T waves, these changes reach statistical significance.

Amplitude of major ECG waveforms in the winter-spring season. When transitioning from the winter period to the spring period, we observe the opposite trend, with a decrease in the amplitude of ECG waveforms in spring. The transition to spring is accompanied by less stress on the cardiac myocardium. The amplitudes of all ECG waveforms decrease during this transition, and in the case of the R and T waves, this decrease reaches statistical significance.

4 Conclusions

This paper investigates the influence of various environmental factors on episodes of Heart Failure (HF) decompensations, as reflected in hospital admissions. A regression model for time series analysis was developed to identify the external attributes that most significantly impact the frequency of hospitalizations. The study found that air temperature emerged as

 https://doi.org/10.1051/bioconf/202414004008

the primary environmental factor affecting HF decompensations, with precipitation and air quality parameters such as SO2 and NOX also demonstrating relevance.

In future research, these environmental factors will be integrated into existing predictive models aimed at assessing the risk of decompensation in ambulatory HF patients. This predictive model, which predicts decompensation risk within a seven-day timeframe, utilizes patient monitoring data from preceding days. Incorporating the environmental factors identified in this study is expected to enhance the predictive accuracy of the model.

Additionally, while the primary aim of the study was to identify the influence of environmental attributes on HF decompensations, there is potential to develop a predictive model for forecasting the number of hospital admissions in the upcoming week. Such a model could be invaluable for physicians in anticipating potential hospital bed shortages. Initial testing of this model using an ARIMA predictive model yielded a mean error of four admissions per week (based on a weekly hospitalization rate of 28). However, it's important to note that this testing was conducted using the same dataset for both training and testing. Therefore, generalization of these results is contingent upon testing the model with new datasets, making this an area for future investigation.

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