BIBLIOGRAPHIC INFORMATION SYSTEM

Journal Full Title: Journal of Biomedical Research & Environmental Sciences
Journal NLM Abbreviation: J Biomed Res Environ Sci
Journal Website Link: https://www.jelsciences.com
Journal ISSN: 2766-2276
Category: Multidisciplinary
Subject Areas: Medicine Group, Biology Group, General, Environmental Sciences
Topics Summation: 130
Issue Regularity: Monthly
Review Process: Double Blind
Time to Publication: 21 Days
Indexing catalog: Visit here
Publication fee catalog: Visit here

DOI: 10.37871 (CrossRef)
Plagiarism detection software: iThenticate
Managing entity: USA
Language: English
Research work collecting capability: Worldwide
Organized by: SciRes Literature LLC

IndexCopernicus ICV 2020: 53.77

Manuscript should be submitted in Word Document (.doc or .docx) through

Online Submission

form or can be mailed to support@jelsciences.com

Vision: Journal of Biomedical Research & Environmental Sciences main aim is to enhance the importance of science and technology to the scientific community and also to provide an equal opportunity to seek and share ideas to all our researchers and scientists without any barriers to develop their career and helping in their development of discovering the world.

JOURNAL OF BIOMEDICAL RESEARCH & ENVIRONMENTAL SCIENCES
ISSN: 2766-2276
SUBJECT AREA(S): CARDIOVASCULAR DISEASES | PUBLIC HEALTH | CLINICAL CARDIOLOGY | NANOTECHNOLOGY
DOI: 10.37871

REVIEW ARTICLE

Exposure to Urban Air Pollution Nanoparticles: Oxidative Stress and Cardiovascular Disease

Mojtaba Ehsanifar[1]*, Zeinab Montazeri[2] and Mehravar Rafati[3]

[1]Anatomical Sciences Research Center, Kashan University of Medical Sciences, Kashan, Iran
[2]Institute of Endocrinology and Metabolism Research and Training Center, Iran University of Medical Sciences, Tehran, Iran
[3]Department of Medical Physics and Radiology, Faculty of Paramedicine, Kashan University of Medical Sciences, Kashan, Iran

ABSTRACT

It is estimated that more than two-thirds of air pollution-related deaths are due to cardiovascular causes. Significant studies have now indicated that exposure to urban air pollutants is known to be a source of oxidative stress and inflammation that causes cardiovascular disease. Nitrogen oxides, Particulate Matter (PM) such as coarse particle (PM10, PM <10μm), fine particles (PM2.5, PM <2.5μm) and Ultra-Fine Particles (UFPs or PM0.1, PM <0.1μm,), ozone and transition metals are oxidant potent capable of producing Reactive Oxygen Species (ROS). Although several biological mechanisms are involved in cardiovascular disease, oxidative stress is an important observation in many levels of cardiovascular failure due to exposure to air pollutants. This mini-review cites evidence that oxidative stress is a key pathway for various cardiovascular measures of exposure to air pollution.

INTRODUCTION

Exposure to air pollutants has far-reaching implications for biodiversity, and its human health effects have made it internationally important. Air pollutants exposure is the fifth risk factor for all-cause mortality and the number one environmental risk factor for mortality [1]. Recently, air pollutants it has been revealed to have effects all over the human body [2,3], however, their effects cardiovascular system, especially are disproportionate in terms of disease and mortality. In fact, due to the high prevalence of the cardiovascular disease worldwide and the inherent mortality of many cardiovascular diseases, more than 65% of air pollution deaths are due to cardiovascular causes, especially cerebrovascular disease and ischemic heart disease [1,4]. In addition, cardiovascular complications may play an important role in the air pollution impact on the progression of the diseases in other organs [5]. Oxidative stress has been shown to play a major role in cardiovascular disease of air pollution. This topic was examined in detail in 2012 [6], and the 2010-2016 data were expertly reviewed [7]. The aim of this study is to provide a mini-review of the oxidative stress role in cardiovascular effects of exposure to urban air pollution nanoparticles by surveying evidence in various aspects of cardiovascular pathophysiology.

Exposure to air pollution

Air pollution involves a range of materials derived from various sources and the chemical reactions in atmosphere. Airborne pollutants can come from both human resources (e.g. power plants, industry, traffic, cooking, home heating, construction, agriculture, mechanical wear, etc.) and natural sources (e.g. volcanic

***Corresponding author**

Mojtaba Ehsanifar, Anatomical Sciences Research Center, Kashan University of Medical Sciences, Kashan, Iran

E-mail: ehsanifar@gmail.com

DOI: 10.37871/jbres1461

Submitted: 26 April 2022

Accepted: 29 April 2022

Published: 29 April 2022

Keywords

- Urban air pollution
- Nanoparticles
- Oxidative stress
- Cardiovascular disease

MEDICINE GROUP

CARDIOVASCULAR DISEASES | PUBLIC HEALTH | CINICAL CARDIOLOGY | NANOTECHNOLOGY

VOLUME: 3 ISSUE: 4 - APRIL, 2022

How to cite this article: Ehsanifar M, Montazeri Z, Rafati M. Exposure to Urban Air Pollution Nanoparticles: Oxidative Stress and Cardiovascular Disease. J Biomed Res Environ Sci. 2022 Apr 29; 3(4): 429-435. doi: 10.37871/jbres1461, Article ID: JBRES1461, Available at: https://www.jelsciences.com/articles/jbres1461.pdf

eruptions, forest resources, airborne dust and mildew) [8,9]. Although indoor air pollution has been particularly important in terms of disease burden in developing countries, ambient air pollution refers to outdoor air pollutants and has historically received the most attention [9-11]. The many studies on ambient air pollution focus on urban air pollution; for other reasons, higher levels of traffic emissions, high urban population densities, and increasing urbanization of communities around the world. Urban air pollution is a complex mixture of chemicals. Gaseous pollutants such as CO_2, SO_2, CO, NO_2, and O_3 that are present in varying amounts, have potential to cause short-term and long-term health effects, possibly as additives manner to particulate matter [9,12,13]. Many of these gases have oxidative properties and the induction of oxidative stress (along with inflammation) is a possible mechanism through which it can affect human health [14]. Semi-volatile species such as naphthalene, formaldehyde, Poly Aromatic Hydrocarbons (PAHs), benzene, exist as liquid droplets, but can also be transported between gas phases and particulate phases of the air pollution [15]. In addition, there are many sources of PM. PM is classified according to the size of the particle. Coarse particle is particle with a diameter of 10µm or less (PM10), fine particles with a diameter of 2.5µm or less (PM2.5), and Ultrafine Particles (UFPs or nanoparticles) with a diameter of 100 nanometers or less. (PM0.1) (Figure 1). Particulate matters are monitored in environment through fixed monitoring networks that measure PM2.5 and PM10. At present, it is not possible to measure very fine PM using very large monitoring networks in environment. Organic and elemental carbon is an important part of ambient PM (especially those obtained from traffic combustion) but non-carbon compounds such as sea salt, various mineral dust, ammonium, sulfates, nitrates, etc. are also present [16,17]. Particle composition is one of the key physicochemical properties that determine the biological response to PM inhalation. Organic carbon species (PAHs, nitro PAHs, quinones, alkanes, alkenes, alkyl benzenes, etc.) and active oxidation transfer metals often play a role in health effects of ambient PM and availability of the these chemicals on PM surface has a biological effect on these particles [18,19]. In addition, the sizes of particles are a very important factor for PM health effects. The size is a determinant of penetration into the lungs and the ability (or inability) mechanisms of biological clearance to remove inhaled PM [20,21]. The particle size also has a very important effect on PM relative surface area, with small particles having an area greater than mass equivalent to a particle larger than the same substance (although this is partly due to the agglomeration of particles in air and the biological fluids). Accordingly, PM2.5 is often more important to health associations than PM10 [9,22]. There is a perception that UFPs can pose a greater health risk due to their greater reactive relative surface area and their ability to deep penetrate into the lungs alveoli and bloodstream [23,24]. Vehicle exhaust is an eminent source of UFPs in urban air pollutants, and Diesel Exhaust Particles (DEPs) is of particular interest due to its higher PM ratio compared to petrol/ gasoline engine emissions as well as the tendency to correlate with high levels of co-pollutants such as NO_2 [25,26]. Many of the above air pollutants and their constituent compound have the ability to induce health effects. However, in relation to the cardiovascular system, the composition of the PM is more important for epidemiological associations [9,27]. For this reason, this study will focus on cardiovascular measures of PM and in particular the vehicle emissions as outstanding sources of UFPs.

Epidemiological evidence overview of air pollution and cardiovascular disease

The air pollution exposure cardiovascular effects were highlighted in the early 1990s. Examining the relationship between exposure to PM2.5 and death from cardiovascular disease, in 1993, a completely linear relationship was observed between the level of PM2.5 and the rate of cardiovascular disease and mortality [28]. In the USA, in polluted cities with level of PM2.5 (11 - 30µg / m^3), the adjusted cardiopulmonary mortality ratio was 1.37 (95% CI: 1.11-1.68) [29]. In the other study in the United States, it

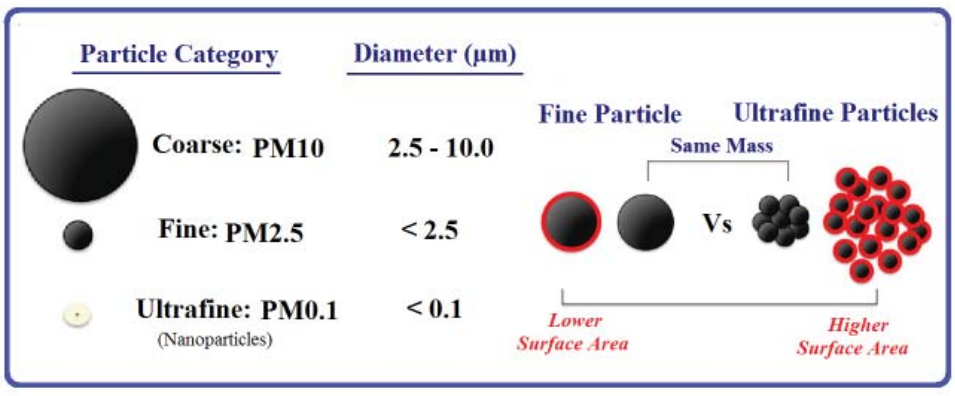

Figure 1 Categories of air pollution Particles Matter (PM) based on different sizes.

was estimated that a decrease of 10μg/m³ PM2.5 was cause increase the average life expectancy by approximately 7 months. In addition, a study on women in the USA indicated that long-term air pollution exposure caused a 24% increase in risk of the cardiovascular event and alarming 76% increase in the cardiovascular disease death risk [30]. Finally, a basic study in across Los Angeles, USA, showed an association between exposure to PM2.5 and atherosclerosis (chronic vascular disease that underlies coronary artery disease and many other cardiovascular diseases). A difference of 10μg /m³ PM2.5 was related to a 4-6% increase in Carotid-Intima Thickness (CIMT) [31]. Long-term and short-term air pollution exposure is related to mortality and or cardiovascular events (such as heart attack or stroke) Using data from the three large US cohorts. Prolonged air pollution exposure (1-4 years) was related with an 8-18% increase in the cardiovascular mortality per 30μg / m³ PM2.5 [32].

An 8-year follow-up of people living in near to a main road indicates almost a doubling of risk of the cardiovascular death [33]. In the short term exposure, increased coronary events [34] and cardiovascular mortality [35,36] were associated with air pollution PM on the same day and before. The trac air pollution coronary effects may occur even earlier, as people with a heart attack may have been in traffic 1-2 hours beforehand [37,38].

Our previous studies have well demonstrated the effects of urban air pollution nanoparticles exposure on oxidative stress and inflammation in the central nervous system and neurotoxicity and behavioral changes associated with anxiety and depression and impaired memory and learning [39-44].

However, dose-response relationship between the cardiovascular mortality and exposure to PM, especially at low and high doses, needs further investigation. Current evidence suggests that there is a linear relationship between exposure to moderate PM levels and mortality, followed by an increase in mortality rates to higher levels ("superliner") [45-47].

It is important to note that relatively low levels of air pollution can also increase cardiovascular disease in the long run [46]. However, this is true for PM2.5 levels, which are currently lower than international guidelines (e.g. World Health Organization; annual PM2.5 <10μg / m³) [48,49]. The volume of epidemiological evidence goes far beyond mortality and widespread criteria for complications. Indeed, air pollution has been shown to be associated with most cardiovascular disorders, including coronary artery disease [30,50,51], arrhythmias and cardiac arrest [52,53], acute myocardial infarction [54,55] Related are heart failure [54,56], cerebrovascular disease [13,50,57], peripheral arterial disease [58,59] and venous thromboembolism [60,61]. Comprehensive review of evidence suggests that there is a strong causal link between a wide range of the cardiovascular endpoints and exposure to air pollution [62]. "Evidence suggests a causal relationship between PM2.5 exposure and cardiovascular morbidity and mortality" [9]. Recent studies have strengthened the weight of this evidence. Another study concluded that "there is now ample evidence that exposure to urban air pollution contributes to cardiovascular disease and mortality, supported by credible evidence of a number of possible mechanisms." It also emphasizes that "air pollution should be considered as one of several major modifiable risk factors in the prevention and management of cardiovascular disease" [63].

Oxidative stress following exposure to air pollution in cardiovascular disease

Oxidative stress was proven as a key pathway in the field of cardiovascular effects of exposure to air pollution [64-66]. Both controlled exposure and epidemiological studies in humans have provided the strong evidence for oxidative pathways, and these foundations have been established by a network of mechanical studies in cellular and animals models [7]. The AHA statement in 2010 concluded that "at the molecular level, oxidative stress as a very important cause and effect of PM-mediated cardiovascular effects has a good empirical basis" [9]. This mini-review emphasizes the role of the oxidative stress in the cardiovascular disease of exposure to air pollutants nanoparticles (Figure 2). Extensive effects of UFPs-induced oxidative stress have been investigated [65,67]. Studies have identified linking both cardiovascular function and air pollution exposure to the mechanical evidence for oxidative stress (such as identifying the source of free radicals, measuring the biomarker of oxidative stress, assessing the oxidative potential of a

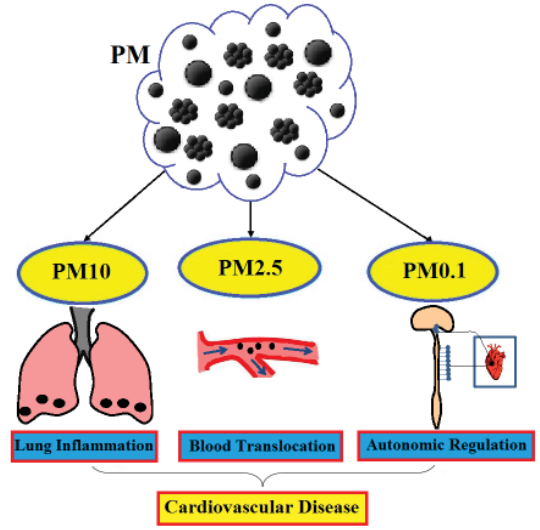

Figure 2 Air pollution nanoparticles and cardiovascular disease. The role of oxidative stress in the mechanisms by which inhaled PM disrupts cardiovascular function. A complex set of interconnected mechanisms underlies the effects of inhaled PM on cardiovascular complications.

contaminant, exploring genetic polymorphisms that cause altered sensitivity to oxidative stress, or preventing/reversing antioxidant compounds). In addition, based on an examination of various aspects of the cardiovascular system, it is clear that the oxidative stress is an important and common mechanism in many different processes that link cardiovascular mortality to air pollution exposure.

Air pollution nanoparticles and cardiovascular disease

Many studies have indicated that fine PM exposure causes coagulation changes and systemic inflammation predisposing to ischemic cardiovascular disease, as measured by elevated C - Reactive Protein (CRP), platelets, circulating polymorphonuclear leukocytes, plasma viscosity, fibrinogen and other markers.

PM promotes vascular inflammation, endothelial dysfunction and atherosclerosis [3]. Previous findings mainly have attributed this effect to PM2.5, but most studies show a far greater effect for PM0.1. In fact, new researches show that PM0.1 has a main role in essentially all of these factors [68]. PM0.1 also causes loss of sympathovagal balance, increased heart rate variability, hemostatic function, and altered inflammatory in exposed humans [69]. Even short-term exposure to PM0.1 can cause cardiovascular effects. In the middle-aged patients with metabolic syndrome following PM0.1 exposure for 2h, have been observed decreased blood plasminogen, electrocardiographic changes, thrombomodulin, increased serum CRP, and amyloid A [70]. Many studies have indicated an association between heart disease and chronic UFPs exposure. A 33,831 Dutch residents prospective study found that prolonged UFPs exposure was related to an increased risk of myocardial infarction, cardiovascular disease, and heart failure [71]. Among Toronto residents from 1996 to 2012, an increase in exposure to PM0.1 in adults was related to an increased acute myocardial infarction incidence and heart failure. Although NO_2 was also independently related to an increased incidence of heart failure, adjustment for NO_2 and PM2.5 did not change this association [72]. Mobile monitoring has shown that annually-averaged number of airborne particles exposure is associated with hypertension, ischemic heart disease, and stroke [73]. Other research have also report an increase in thrombotic and ischemic stroke with exposure to PM0.1 [74] And worse microvascular function and increased blood pressure with UFPs (PM0.1) but not with fine (PM2.5) and coarse (PM10) particles [68,75]. Particle size is correlated with total cardiovascular mortality. As the particle size decreases, this correlation becomes stronger. UFPs (PM <0.50μm) have the highest correlation [76,77]. No correlation was found for mass concentrations coarse particles ($PM_{2.5}$ and larger) [77].

Particles 10-50 nm mostly indicate this finding that the number of particles is correlated with the cardiovascular disease related emergency department visits, with a delay of 4 to 10 days. Exposure to PM0.1 is reported to account for more than 7% of the emergency department visits [78]. The strongest immediate effect correlate was found with PM 30–100-nm (within 2 days), despite a concentration of small mass. The immediate effect associated with mass concentration was with the 1–5 μm particles, which had a similar delayed effect to PM0.1 number [78]. Another study in California of more than 100,000 women found that the mortality from ischemic cardiac disease is more strongly related to PM0.1 than to PM2.5 [79]. Repeated biweekly submaximal exercise tests on adult patients with stable coronary cardiovascular disease showed that exposure to PM0.1 has been related to electrocardiographic ST-segment depression of >0.1 mV. Findings show that PM0.1 effect was independent of the PM2.5. CO and NO_2 were also related to risk for ST-segment depression, but PM10 and PM2.5 were not [80].

CONCLUSION

Awareness of the air pollution health effects is increasing worldwide and is now a priority on environmental and political health agenda. This awareness is reinforced by global data revealing the staggering extent of the air pollution effects on health; reaches the peak of more than a few million deaths annually. A vital stimulus for action is the wider understanding that air pollution effects are not limited to the lungs, but have effects on what each organ appears to be. The cardiovascular effects of contamination are increasingly being recognized by the mainstream. Given the high prevalence of cardiac disease worldwide and its high mortality rate, the infection effects on cardiovascular system will remain crucial. In addition, there is now strong human evidence that inhaled nanoparticles can enter the bloodstream [11,24,66], circulation is not only a means of transporting transported particles throughout the body, but the direct effect of pollutants on cardiovascular function can also contribute to these effects.

Air pollution affects various organs of the body (for example, through high blood pressure, impaired organ perfusion, changes in vascular growth, etc.) [81]. Significant progress has been made in determining biological mechanisms for air pollution cardiovascular effects. A set of interactive mechanisms has been elucidated; however, oxidative stress is a key mechanism for pathophysiological application of contamination to various aspects of cardiovascular system. It is noteworthy that oxidative stress appears as a mechanism in cardiovascular actions of air pollutants using different study approaches. The complementary findings at numerous endpoints and a variety of studies argue that oxidative stress is a vital mechanism in the association between cardiovascular disease and air pollution exposure. Whether oxidative stress is a key trigger or not just a contributing factor is challenging. However, its presence undoubtedly exacerbates the disease. The close

interaction between inflammations indicates and oxidative stress a possible means by which air pollution activities can be enhanced to produce pathophysiological effects in several organs. In addition, due to the obvious prooxidative effects of many air pollutants, and the capacity of oxidative stress to disrupt various aspects of cardiovascular function, oxidative stress may play a key mediator, not just an early phenomenon in later stages of the disease.

Reducing pollution sources should be a key strategy to reduce the burden of urban air pollution on human health. Today, given the challenges of reducing human pollution in the face of increasing urbanization and lifestyle changes, there is room for interventions that can protect against environmental pollution in the medium term. As a result, oxidative stress is the key mechanism by which air pollution exposure causes cardiovascular complications and mortality. The burden of cardiovascular disease and other diseases caused by oxidative pathways is expected to be reduced by developing strategies that reduce the production of air pollutants.

REFERENCES

1. Cohen AJ. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: An analysis of data from the Global Burden of Diseases Study 2015. The Lancet. 2017;389(10082):1907-1918. https://tinyurl.com/yeywta3b
2. Schraufnagel DE, Balmes JR, Cowl CT, De Matteis S, Jung SH, Mortimer K, Perez-Padilla R, Rice MB, Riojas-Rodriguez H, Sood A, Thurston GD, To T, Vanker A, Wuebbles DJ. Air pollution AND noncommunicable diseases: a review BY THE forum OF international respiratory societies' environmental committee, Part 1: The Damaging Effects of Air Pollution. Chest. 2019 Feb;155(2):409-416. doi: 10.1016/j.chest.2018.10.042. Epub 2018 Nov 9. PMID: 30419235; PMCID: PMC6904855.
3. Schraufnagel DE, Balmes JR, Cowl CT, De Matteis S, Jung SH, Mortimer K, Perez-Padilla R, Rice MB, Riojas-Rodriguez H, Sood A, Thurston GD, To T, Vanker A, Wuebbles DJ. Air pollution and noncommunicable diseases: a review by the forum of international respiratory societies' environmental committee, Part 2: Air Pollution and Organ Systems. Chest. 2019 Feb;155(2):417-426. doi: 10.1016/j.chest.2018.10.041. Epub 2018 Nov 9. PMID: 30419237; PMCID: PMC6904854.
4. Lelieveld J. Cardiovascular disease burden from ambient air pollution in Europe reassessed using novel hazard ratio functions. European heart journal. 2019;40(20):1590-1596. https://tinyurl.com/2p99zvht
5. Raftis JB, Miller. Nanoparticle translocation and multi-organ toxicity: A particularly small problem. Nano Today. 2019;26:8-12. https://tinyurl.com/y88685fw
6. Miller MR, Shaw CA, Langrish JP. From particles to patients: Oxidative stress and the cardiovascular effects of air pollution. Future Cardiol. 2012 Jul;8(4):577-602. doi: 10.2217/fca.12.43. PMID: 22871197.
7. Kelly FJ, Fussell JC. Role of oxidative stress in cardiovascular disease outcomes following exposure to ambient air pollution. Free Radic Biol Med. 2017 Sep;110:345-367. doi: 10.1016/j.freeradbiomed.2017.06.019. Epub 2017 Jun 29. PMID: 28669628.
8. Jonidi A, Ehsanifar. The share of different vehicles in air pollutant emission in tehran, using 2013 traffic information. Caspian Journal of Health Research. 2016;2(2):28-36. https://tinyurl.com/4dm63y3w
9. Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F, Hong Y, Luepker RV, Mittleman MA, Peters A, Siscovick D, Smith SC Jr, Whitsel L, Kaufman JD; American Heart Association Council on Epidemiology and Prevention, Council on the Kidney in Cardiovascular Disease, and Council on Nutrition, Physical Activity and Metabolism. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation. 2010 Jun 1;121(21):2331-2378. doi: 10.1161/CIR.0b013e3181dbece1. Epub 2010 May 10. PMID: 20458016.
10. Ehsanifar M, Rafati, Yavari. Indoor air pollution and behavioral factors affecting to COVID-19 transition. 2022. https://tinyurl.com/2rmndd62
11. Ehsanifar M, Banihashemian, Farokhmanesh. Exposure to urban air pollution nanoparticles and CNS disease. On J Neur & Br Disord. 2021;5(5):520-526. https://tinyurl.com/yckpyvyv
12. Ehsanifar M, Montazeri, Rafati. Neurotoxicity related exposure to ambient nanoparticles. 2022. https://tinyurl.com/mztnuarb
13. Ehsanifar M, Banihashemian, Farokhmanesh. Exposure to Ambient Ultra-Fine Particles and Stroke. 2021. https://tinyurl.com/3b9kb4ey
14. Auerbach A, Hernandez ML. The effect of environmental oxidative stress on airway inflammation. Curr Opin Allergy Clin Immunol. 2012 Apr;12(2):133-139. doi: 10.1097/ACI.0b013e32835113d6. PMID: 22306553; PMCID: PMC3319111.
15. Liu C, Zhang Y, Weschler CJ. The impact of mass transfer limitations on size distributions of particle associated SVOCs in outdoor and indoor environments. Sci Total Environ. 2014 Nov 1;497-498:401-411. doi: 10.1016/j.scitotenv.2014.07.095. Epub 2014 Aug 19. PMID: 25146909.
16. Lighty JS, Veranth JM, Sarofim AF. Combustion aerosols: factors governing their size and composition and implications to human health. J Air Waste Manag Assoc. 2000 Sep;50(9):1565-1618; discussion 1619-1622. doi: 10.1080/10473289.2000.10464197. PMID: 11055157.
17. Ehsanifar M. Airborne aerosols particles and COVID-19 transition. Environ Res. 2021 Sep;200:111752. doi: 10.1016/j.envres.2021.111752. Epub 2021 Jul 22. PMID: 34302822; PMCID: PMC8295061.
18. Cui X, Zhou T, Shen Y, Rong Y, Zhang Z, Liu Y, Xiao L, Zhou Y, Li W, Chen W. Different biological effects of $PM_{2.5}$ from coal combustion, gasoline exhaust and urban ambient air relate to the PAH/metal compositions. Environ Toxicol Pharmacol. 2019 Jul;69:120-128. doi: 10.1016/j.etap.2019.04.006. Epub 2019 Apr 17. PMID: 31026736.
19. O'Driscoll CA, Owens LA, Gallo ME, Hoffmann EJ, Afrazi A, Han M, Fechner JH, Schauer JJ, Bradfield CA, Mezrich JD. Differential effects of diesel exhaust particles on T cell differentiation and autoimmune disease. Part Fibre Toxicol. 2018 Aug 24;15(1):35. doi: 10.1186/s12989-018-0271-3. PMID: 30143013; PMCID: PMC6109291.
20. Donaldson K, Stone V. Current hypotheses on the mechanisms of toxicity of ultrafine particles. Ann Ist Super Sanita. 2003;39(3):405-10. PMID: 15098562.
21. Delfino RJ, Sioutas C, Malik S. Potential role of ultrafine particles in associations between airborne particle mass and cardiovascular health. Environ Health Perspect. 2005 Aug;113(8):934-946. doi: 10.1289/ehp.7938. PMID: 16079061; PMCID: PMC1280331.
22. Liu C. Ambient particulate air pollution and daily mortality in 652 cities. New England Journal of Medicine. 2019;381(8):705-715. https://tinyurl.com/yckredut
23. Ohlwein S, Kappeler R, Kutlar Joss M, Künzli N, Hoffmann B. Health effects of ultrafine particles: A systematic literature review update of epidemiological evidence. Int J Public Health. 2019 May;64(4):547-559. doi: 10.1007/s00038-019-01202-7. Epub 2019 Feb 21. PMID: 30790006.
24. Miller MR, Raftis JB, Langrish JP, McLean SG, Samutrtai P, Connell SP, Wilson S, Vesey AT, Fokkens PHB, Boere AJF, Krystek P, Campbell CJ, Hadoke PWF, Donaldson K, Cassee FR, Newby DE, Duffin R, Mills NL. Inhaled nanoparticles accumulate at sites of vascular disease. ACS Nano. 2017 May 23;11(5):4542-4552. doi: 10.1021/acsnano.6b08551. Epub 2017 Apr 26. Erratum in: ACS Nano. 2017 Oct 24;11(10):10623-10624. PMID: 28443337; PMCID: PMC5444047.
25. Steiner S, Bisig C, Petri-Fink A, Rothen-Rutishauser B. Diesel exhaust: Current knowledge of adverse effects and underlying cellular mechanisms. Arch Toxicol. 2016 Jul;90(7):1541-1553. doi: 10.1007/s00204-016-1736-5. Epub 2016 May 10. PMID: 27165416; PMCID: PMC4894930.
26. Geller MD. Physicochemical and redox characteristics of Particulate Matter (PM) emitted from gasoline and diesel passenger cars. Atmospheric Environment. 2006;40(36):6988-7004. https://tinyurl.com/53bc2ccn
27. Münzel T, Gori T, Al-Kindi S, Deanfield J, Lelieveld J, Daiber A, Rajagopalan S. Effects of gaseous and solid constituents of air pollution on endothelial function. Eur Heart J. 2018 Oct 7;39(38):3543-3550. doi: 10.1093/eurheartj/ehy481. PMID: 30124840; PMCID: PMC6174028.
28. Dockery DW, Pope CA 3rd, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr, Speizer FE. An association between air pollution and mortality in six U.S. cities. N Engl J Med. 1993 Dec 9;329(24):1753-1759. doi: 10.1056/NEJM199312093292401. PMID: 8179653.
29. Pope CA 3rd, Ezzati M, Dockery DW. Fine-particulate air pollution and life expectancy in the United States. N Engl J Med. 2009 Jan 22;360(4):376-386. doi: 10.1056/NEJMsa0805646. PMID: 19164188; PMCID: PMC3382057.
30. Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, Kaufman JD. Long-term exposure to air pollution and incidence of cardiovascular events in women. N Engl J Med. 2007 Feb 1;356(5):447-458. doi: 10.1056/NEJMoa054409. PMID: 17267905.
31. Künzli N, Jerrett M, Mack WJ, Beckerman B, LaBree L, Gilliland F, Thomas D, Peters J, Hodis HN. Ambient air pollution and atherosclerosis in Los Angeles. Environ Health Perspect. 2005 Feb;113(2):201-216. doi: 10.1289/ehp.7523. PMID: 15687058; PMCID: PMC1277865.

32. Pope CA 3rd, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. Circulation. 2004 Jan 6;109(1):71-77. doi: 10.1161/01.CIR.0000108927.80044.7F. Epub 2003 Dec 15. PMID: 14676145.

33. Hoek G, Brunekreef B, Goldbohm S, Fischer P, van den Brandt PA. Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. Lancet. 2002 Oct 19;360(9341):1203-1209. doi: 10.1016/S0140-6736(02)11280-3. PMID: 12401246.

34. Pope CA 3rd, Muhlestein JB, May HT, Renlund DG, Anderson JL, Horne BD. Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. Circulation. 2006 Dec 5;114(23):2443-2448. doi: 10.1161/CIRCULATIONAHA.106.636977. Epub 2006 Nov 13. PMID: 17101851.

35. Zanobetti A, Schwartz J, Samoli E, Gryparis A, Touloumi G, Peacock J, Anderson RH, Le Tertre A, Bobros J, Celko M, Goren A, Forsberg B, Michelozzi P, Rabczenko D, Hoyos SP, Wichmann HE, Katsouyanni K. The temporal pattern of respiratory and heart disease mortality in response to air pollution. Environ Health Perspect. 2003 Jul;111(9):1188-1193. doi: 10.1289/ehp.5712. PMID: 12842772; PMCID: PMC1241573.

36. Dominici F, McDermott A, Daniels M, Zeger SL, Samet JM. Revised analyses of the national morbidity, mortality, and air pollution study: Mortality among residents of 90 cities. J Toxicol Environ Health A. 2005 Jul 9-23;68(13-14):1071-1092. doi: 10.1080/15287390590935932. PMID: 16024489.

37. Peters A, von Klot S, Mittleman MA, Meisinger C, Hörmann A, Kuch B, Wichmann HE. Triggering of acute myocardial infarction by different means of transportation. Eur J Prev Cardiol. 2013 Oct;20(5):750-758. doi: 10.1177/2047487312446672. Epub 2012 Apr 26. PMID: 22544548.

38. Bhaskaran K. The effects of hourly differences in air pollution on the risk of myocardial infarction: case crossover analysis of the MINAP database. Bmj. 2011:343. https://tinyurl.com/23peu2rf

39. Ehsanifar M, Jafari AJ, Montazeri Z, Kalantari RR, Gholami M, Ashtarinezhad A. Learning and memory disorders related to hippocampal inflammation following exposure to air pollution. J Environ Health Sci Eng. 2021 Jan 22;19(1):261-272. doi: 10.1007/s40201-020-00600-x. PMID: 34150234; PMCID: PMC8172730.

40. Ehsanifar M, Montazeri Z, Taheri MA, Rafati M, Behjati M, Karimian M. Hippocampal inflammation and oxidative stress following exposure to diesel exhaust nanoparticles in male and female mice. Neurochem Int. 2021 May;145:104989. doi: 10.1016/j.neuint.2021.104989. Epub 2021 Feb 12. PMID: 33582162.

41. Ehsanifar M, Tameh AA, Farzadkia M, Kalantari RR, Zavareh MS, Nikzaad H, Jafari AJ. Exposure to nanoscale diesel exhaust particles: Oxidative stress, neuroinflammation, anxiety and depression on adult male mice. Ecotoxicol Environ Saf. 2019 Jan 30;168:338-347. doi: 10.1016/j.ecoenv.2018.10.090. Epub 2018 Nov 2. PMID: 30391838.

42. Ehsanifar M, Jafari AJ, Nikzad H, Zavareh MS, Atlasi MA, Mohammadi H, Tameh AA. Prenatal exposure to diesel exhaust particles causes anxiety, spatial memory disorders with alters expression of hippocampal pro-inflammatory cytokines and NMDA receptor subunits in adult male mice offspring. Ecotoxicol Environ Saf. 2019 Jul 30;176:34-41. doi: 10.1016/j.ecoenv.2019.03.090. Epub 2019 Mar 25. PMID: 30921694.

43. Ehsanifar M, Yavari Z, Rafati M. Exposure to urban air pollution particulate matter: Neurobehavioral alteration and hippocampal inflammation. Environ Sci Pollut Res Int. 2022 Mar 3. doi: 10.1007/s11356-022-19367-9. Epub ahead of print. PMID: 35237914.

44. Ehsanifar M. Anxiety and depression following diesel exhaust nano-particles exposure in male and female mice. J Neurophysiol Neurol Disord, 2020;8:1-8. https://tinyurl.com/2p9782bd

45. Vodonos A, Awad YA, Schwartz J. The concentration-response between long-term $PM_{2.5}$ exposure and mortality; A meta-regression approach. Environ Res. 2018 Oct;166:677-689. doi: 10.1016/j.envres.2018.06.021. Epub 2018 Aug 1. PMID: 30077140.

46. Papadogeorgou G, Kioumourtzoglou MA, Braun D, Zanobetti A. Low levels of air pollution and health: Effect estimates, methodological challenges, and future directions. Curr Environ Health Rep. 2019 Sep;6(3):105-115. doi: 10.1007/s40572-019-00235-7. PMID: 31090042; PMCID: PMC7161422.

47. Pappin AJ, Christidis T, Pinault LL, Crouse DL, Brook JR, Erickson A, Hystad P, Li C, Martin RV, Meng J, Weichenthal S, van Donkelaar A, Tjepkema M, Brauer M, Burnett RT. Examining the shape of the association between low levels of fine particulate matter and mortality across three cycles of the canadian census health and environment cohort. Environ Health Perspect. 2019 Oct;127(10):107008. doi: 10.1289/EHP5204. Epub 2019 Oct 22. PMID: 31638837; PMCID: PMC6867181.

48. Beelen R, Raaschou-Nielsen O, Stafoggia M, Andersen ZJ, Weinmayr G, Hoffmann B, Wolf K, Samoli E, Fischer P, Nieuwenhuijsen M, Vineis P, Xun WW, Katsouyanni K, Dimakopoulou K, Oudin A, Forsberg B, Modig L, Havulinna AS, Lanki T, Turunen A, Oftedal B, Nystad W, Nafstad P, De Faire U, Pedersen NL, Östenson CG, Fratiglioni L, Penell J, Korek M, Pershagen G, Eriksen KT, Overvad K, Ellermann T, Eeftens M, Peeters PH, Meliefste K, Wang M, Bueno-de-Mesquita B, Sugiri D, Krämer U, Heinrich J, de Hoogh K, Key T, Peters A, Hampel R, Concin H, Nagel G, Ineichen A, Schaffner E, Probst-Hensch N, Künzli N, Schindler C, Schikowski T, Adam M, Phuleria H, Vilier A, Clavel-Chapelon F, Declercq C, Grioni S, Krogh V, Tsai MY, Ricceri F, Sacerdote C, Galassi C, Migliore E, Ranzi A, Cesaroni G, Badaloni C, Forastiere F, Tamayo I, Amiano P, Dorronsoro M, Katsoulis M, Trichopoulou A, Brunekreef B, Hoek G. Effects of long-term exposure to air pollution on natural-cause mortality: An analysis of 22 European cohorts within the multicentre ESCAPE project. Lancet. 2014 Mar 1;383(9919):785-795. doi: 10.1016/S0140-6736(13)62158-3. Epub 2013 Dec 9. PMID: 24332274.

49. Wan G, Rajagopalan S, Sun Q, Zhang K. Real-world exposure of airborne particulate matter triggers oxidative stress in an animal model. Int J Physiol Pathophysiol Pharmacol. 2010 Mar 15;2(1):64-68. PMID: 21383899; PMCID: PMC3047275.

50. Chen H, Goldberg MS, Burnett RT, Jerrett M, Wheeler AJ, Villeneuve PJ. Long-term exposure to traffic-related air pollution and cardiovascular mortality. Epidemiology. 2013 Jan;24(1):35-43. doi: 10.1097/EDE.0b013e318276c005. PMID: 23222554.

51. Cesaroni G, Forastiere F, Stafoggia M, Andersen ZJ, Badaloni C, Beelen R, Caracciolo B, de Faire U, Erbel R, Eriksen KT, Fratiglioni L, Galassi C, Hampel R, Heier M, Hennig F, Hilding A, Hoffmann B, Houthuijs D, Jöckel KH, Korek M, Lanki T, Leander K, Magnusson PK, Migliore E, Ostenson CG, Overvad K, Pedersen NL, J JP, Penell J, Pershagen G, Pyko A, Raaschou-Nielsen O, Ranzi A, Ricceri F, Sacerdote C, Salomaa V, Swart W, Turunen AW, Vineis P, Weinmayr G, Wolf K, de Hoogh K, Hoek G, Brunekreef B, Peters A. Long term exposure to ambient air pollution and incidence of acute coronary events: prospective cohort study and meta-analysis in 11 European cohorts from the ESCAPE Project. BMJ. 2014 Jan 21;348:f7412. doi: 10.1136/bmj.f7412. PMID: 24452269; PMCID: PMC3898420.

52. Raza A, Bellander T, Bero-Bedada G, Dahlquist M, Hollenberg J, Jonsson M, Lind T, Rosenqvist M, Svensson L, Ljungman PL. Short-term effects of air pollution on out-of-hospital cardiac arrest in Stockholm. Eur Heart J. 2014 Apr;35(13):861-868. doi: 10.1093/eurheartj/eht489. Epub 2013 Dec 2. PMID: 24302272.

53. Watkins A, Danilewitz M, Kusha M, Massé S, Urch B, Quadros K, Spears D, Farid T, Nanthakumar K. Air pollution and arrhythmic risk: the smog is yet to clear. Can J Cardiol. 2013 Jun;29(6):734-741. doi: 10.1016/j.cjca.2012.09.005. Epub 2012 Dec 6. PMID: 23219609.

54. Bai L, Shin S, Burnett RT, Kwong JC, Hystad P, van Donkelaar A, Goldberg MS, Lavigne E, Copes R, Martin RV, Kopp A, Chen H. Exposure to ambient air pollution and the incidence of congestive heart failure and acute myocardial infarction: A population-based study of 5.1 million Canadian adults living in Ontario. Environ Int. 2019 Nov;132:105004. doi: 10.1016/j.envint.2019.105004. Epub 2019 Aug 3. PMID: 31387019.

55. Malik AO. Association of long-term exposure to particulate matter and ozone with health status and mortality in patients after myocardial infarction. Circulation: Cardiovascular Quality and Outcomes. 2019;12(4):e005598. https://tinyurl.com/5c38nhxj

56. Atkinson RW, Carey IM, Kent AJ, van Staa TP, Anderson HR, Cook DG. Long-term exposure to outdoor air pollution and incidence of cardiovascular diseases. Epidemiology. 2013 Jan;24(1):44-53. doi: 10.1097/EDE.0b013e318276ccb8. Erratum in: Epidemiology. 2013 Mar;24(2):339. PMID: 23222514.

57. Lee KK, Miller MR, Shah ASV. Air pollution and stroke. J Stroke. 2018 Jan;20(1):2-11. doi: 10.5853/jos.2017.02894. Epub 2018 Jan 31. PMID: 29402072; PMCID: PMC5836577.

58. Hoffmann B, Moebus S, Kröger K, Stang A, Möhlenkamp S, Dragano N, Schmermund A, Memmesheimer M, Erbel R, Jöckel KH. Residential exposure to urban air pollution, ankle-brachial index, and peripheral arterial disease. Epidemiology. 2009 Mar;20(2):280-288. doi: 10.1097/EDE.0b013e3181961ac2. PMID: 19194299.

59. Peng RD, Chang HH, Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F. Coarse particulate matter air pollution and hospital admissions for cardiovascular and respiratory diseases among Medicare patients. JAMA. 2008 May 14;299(18):2172-2179. doi: 10.1001/jama.299.18.2172. PMID: 18477784; PMCID: PMC3169813.

60. Baccarelli A, Martinelli I, Pegoraro V, Melly S, Grillo P, Zanobetti A, Hou L, Bertazzi PA, Mannucci PM, Schwartz J. Living near major traffic roads and risk of deep vein thrombosis. Circulation. 2009 Jun 23;119(24):3118-24. doi: 10.1161/CIRCULATIONAHA.108.836163. Epub 2009 Jun 8. PMID: 19506111; PMCID: PMC2895730.

61. Franchini M, Guida A, Tufano A, Coppola A. Air pollution, vascular disease and thrombosis: linking clinical data and pathogenic mechanisms. J Thromb Haemost. 2012 Dec;10(12):2438-2451. doi: 10.1111/jth.12006. PMID: 23006215.

62. Zhang P, Dong G, Sun B, Zhang L, Chen X, Ma N, Yu F, Guo H, Huang H, Lee YL, Tang N, Chen J. Long-term exposure to ambient air pollution and mortality due to

cardiovascular disease and cerebrovascular disease in Shenyang, China. PLoS One. 2011;6(6):e20827. doi: 10.1371/journal.pone.0020827. Epub 2011 Jun 10. PMID: 21695220; PMCID: PMC3112212.

63. Newby DE, Mannucci PM, Tell GS, Baccarelli AA, Brook RD, Donaldson K, Forastiere F, Franchini M, Franco OH, Graham I, Hoek G, Hoffmann B, Hoylaerts MF, Künzli N, Mills N, Pekkanen J, Peters A, Piepoli MF, Rajagopalan S, Storey RF; ESC Working Group on Thrombosis, European Association for Cardiovascular Prevention and Rehabilitation; ESC Heart Failure Association. Expert position paper on air pollution and cardiovascular disease. Eur Heart J. 2015 Jan 7;36(2):83-93b. doi: 10.1093/eurheartj/ehu458. Epub 2014 Dec 9. PMID: 25492627; PMCID: PMC6279152.

64. Wilson SJ, Miller MR, Newby DE. Effects of diesel exhaust on cardiovascular function and oxidative stress. Antioxid Redox Signal. 2018 Mar 20;28(9):819-836. doi: 10.1089/ars.2017.7174. Epub 2017 Jul 14. PMID: 28540736.

65. Kelly FJ, Fussell JC. Linking ambient particulate matter pollution effects with oxidative biology and immune responses. Ann N Y Acad Sci. 2015 Mar;1340:84-94. doi: 10.1111/nyas.12720. Epub 2015 Feb 25. PMID: 25716617.

66. Ehsanifar M, Banihashemian, Ehsanifar. Exposure to air pollution nanoparticles: Oxidative stress and neuroinfl ammation. J Biomed Res Environ Sci. 2021;2(10):964-976. https://tinyurl.com/2jmvthvw

67. Weichenthal SA, Godri-Pollitt K, Villeneuve PJ. PM2.5, oxidant defence and cardiorespiratory health: a review. Environ Health. 2013 May 4;12:40. doi: 10.1186/1476-069X-12-40. PMID: 23641908; PMCID: PMC3652795.

68. Olsen Y, Karottki DG, Jensen DM, Bekö G, Kjeldsen BU, Clausen G, Hersoug LG, Holst GJ, Wierzbicka A, Sigsgaard T, Linneberg A, Møller P, Loft S. Vascular and lung function related to ultrafine and fine particles exposure assessed by personal and indoor monitoring: a cross-sectional study. Environ Health. 2014 Dec 15;13:112. doi: 10.1186/1476-069X-13-112. PMID: 25512042; PMCID: PMC4290094.

69. Samet JM, Rappold A, Graff D, Cascio WE, Berntsen JH, Huang YC, Herbst M, Bassett M, Montilla T, Hazucha MJ, Bromberg PA, Devlin RB. Concentrated ambient ultrafine particle exposure induces cardiac changes in young healthy volunteers. Am J Respir Crit Care Med. 2009 Jun 1;179(11):1034-1042. doi: 10.1164/rccm.200807-1043OC. Epub 2009 Feb 20. PMID: 19234105.

70. Devlin RB, Smith CB, Schmitt MT, Rappold AG, Hinderliter A, Graff D, Carraway MS. Controlled exposure of humans with metabolic syndrome to concentrated ultrafine ambient particulate matter causes cardiovascular effects. Toxicol Sci. 2014 Jul;140(1):61-72. doi: 10.1093/toxsci/kfu063. Epub 2014 Apr 9. PMID: 24718702.

71. Downward GS, van Nunen EJHM, Kerckhoffs J, Vineis P, Brunekreef B, Boer JMA, Messier KP, Roy A, Verschuren WMM, van der Schouw YT, Sluijs I, Gulliver J, Hoek G, Vermeulen R. Long-term exposure to ultrafine particles and incidence of cardiovascular and cerebrovascular disease in a prospective study of a dutch cohort. Environ Health Perspect. 2018 Dec;126(12):127007. doi: 10.1289/EHP3047. PMID: 30566375; PMCID: PMC6371648.

72. Bai L, Weichenthal S, Kwong JC, Burnett RT, Hatzopoulou M, Jerrett M, van Donkelaar A, Martin RV, Van Ryswyk K, Lu H, Kopp A, Chen H. Associations of long-term exposure to ultrafine particles and nitrogen dioxide with increased incidence of congestive heart failure and acute myocardial infarction. Am J Epidemiol. 2019 Jan 1;188(1):151-159. doi: 10.1093/aje/kwy194. PMID: 30165598.

73. Li Y, Lane KJ, Corlin L, Patton AP, Durant JL, Thanikachalam M, Woodin M, Wang M, Brugge D. Association of long-term near-highway exposure to ultrafine particles with cardiovascular diseases, diabetes and hypertension. Int J Environ Res Public Health. 2017 Apr 26;14(5):461. doi: 10.3390/ijerph14050461. PMID: 28445425; PMCID: PMC5451912.

74. Andersen ZJ, Olsen TS, Andersen KK, Loft S, Ketzel M, Raaschou-Nielsen O. Association between short-term exposure to ultrafine particles and hospital admissions for stroke in Copenhagen, Denmark. Eur Heart J. 2010 Aug;31(16):2034-2040. doi: 10.1093/eurheartj/ehq188. Epub 2010 Jun 10. PMID: 20538735.

75. Pieters N, Koppen G, Van Poppel M, De Prins S, Cox B, Dons E, Nelen V, Panis LI, Plusquin M, Schoeters G, Nawrot TS. Blood pressure and same-day exposure to air pollution at school: Associations with nano-sized to coarse pm in children. Environ Health Perspect. 2015 Jul;123(7):737-742. doi: 10.1289/ehp.1408121. Epub 2015 Mar 10. PMID: 25756964; PMCID: PMC4492263.

76. Meng X, Ma Y, Chen R, Zhou Z, Chen B, Kan H. Size-fractionated particle number concentrations and daily mortality in a Chinese city. Environ Health Perspect. 2013 Oct;121(10):1174-1178. doi: 10.1289/ehp.1206398. Epub 2013 Aug 13. PMID: 23942310; PMCID: PMC3801202.

77. Stölzel M, Breitner S, Cyrys J, Pitz M, Wölke G, Kreyling W, Heinrich J, Wichmann HE, Peters A. Daily mortality and particulate matter in different size classes in Erfurt, Germany. J Expo Sci Environ Epidemiol. 2007 Aug;17(5):458-67. doi: 10.1038/sj.jes.7500538. Epub 2006 Nov 15. PMID: 17108895.

78. Liu L, Breitner S, Schneider A, Cyrys J, Brüske I, Franck U, Schlink U, Marian Leitte A, Herbarth O, Wiedensohler A, Wehner B, Pan X, Wichmann HE, Peters A. Size-fractioned particulate air pollution and cardiovascular emergency room visits in Beijing, China. Environ Res. 2013 Feb;121:52-63. doi: 10.1016/j.envres.2012.10.009. Epub 2013 Jan 30. PMID: 23375554.

79. Ostro B, Hu J, Goldberg D, Reynolds P, Hertz A, Bernstein L, Kleeman MJ. Associations of mortality with long-term exposures to fine and ultrafine particles, species and sources: Results from the California Teachers Study Cohort. Environ Health Perspect. 2015 Jun;123(6):549-556. doi: 10.1289/ehp.1408565. Epub 2015 Jan 23. PMID: 25633926; PMCID: PMC4455590.

80. Pekkanen J, Peters A, Hoek G, Tiittanen P, Brunekreef B, de Hartog J, Heinrich J, Ibald-Mulli A, Kreyling WG, Lanki T, Timonen KL, Vanninen E. Particulate air pollution and risk of ST-segment depression during repeated submaximal exercise tests among subjects with coronary heart disease: The Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air (ULTRA) study. Circulation. 2002 Aug 20;106(8):933-938. doi: 10.1161/01.cir.0000027561.41736.3c. PMID: 12186796.

81. Miller MR. Newby, air pollution and cardiovascular disease: Car sick. Cardiovascular Research. 2020;116(2):279-294. https://tinyurl.com/yckmkrmp

How to cite this article: Ehsanifar M, Montazeri Z, Rafati M. Exposure to Urban Air Pollution Nanoparticles: Oxidative Stress and Cardiovascular Disease. J Biomed Res Environ Sci. 2022 Apr 29; 3(4): 429-435. doi: 10.37871/jbres1461, Article ID: JBRES1461, Available at: https://www.jelsciences.com/articles/jbres1461.pdf