Original Article

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Environmental Pollution and Population Disorders: A Brief Communication

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Abstract

Most diseases in society have a complicated epidemiology including various chemical influences such as biology, diet and environmental pollution (EP). "The most dangerous contaminants included particulate matter (PMs), nitrogen oxides, polycyclic aromatic hydrocarbons (PAHs), heavy metals, pesticides, hormones, and polychlorinated biphenyls (PCBs)". Indeed, there are countless potential contaminants and most have never been assessed as toxic and health hazards, particularly when new chemicals are constantly being developed as a result of interactions with existing chemicals. The effects of these new substances on wellbeing are almost difficult to assess. Previous reports show a wide range of pollution-related diseases. EP has been linked to an elevated prevalence of some malignancies, an increase in all-cause mortality, coronary disease progression, recurring illnesses, "disrupted intellectual and psychomotor development in infants, type 2 diabetes, breathing and immune system as well as brain-degenerative disorders." EP is a significant reason of mortality and morbidity around the globe, initiating high expenditures in health care. Ecological, biological and toxicological testing is needed to determine the environmental toxins and at what amounts are most dangerous to animals and humans. It will only be possible to enhance environmental security by interdisciplinary collaboration and public awareness-raising programs.

Keywords: Environmental Pollution, Particulate Matter, Chronic Diseases, Health Hazards.
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Introduction

The rise of the EP is a major public health sign. While about 12,6 million deaths annually are due to a complicated environmental issue, the "World Health Organization (WHO)" has given report that factors contribute to a broad range of diseases as well as mortality cannot be accurately calculated.[1,2] Today, amid improved understanding of public health risks and stronger prophylaxis and health treatment, the health impacts of pollution are noticeable not only in poor and medium income countries however in high-income countries. It is now crystal clear that exposure to toxic contaminants in one's lifetime cannot be avoided. While the EP has a close association with wellbeing, government health programs globally have not solved or even minimized this issue adequately. The domestic tools for regulation of emissions are not commensurate with the scale of the problem. Interdisciplinary study teams who investigate EP therefore have the moral responsibility to sensitize and improve strategies to reduce pollutants and public knowledge of this problem. This short communication discusses most frequently correlated humanity disorders with environmental chemicals exposure." cvMost diseases in civilization have a diverse etiology that has genetic, lifestyle and environmental causes.[3] The connection between EP and health is like a chain reaction of health effects from exposure to pollutant sources.[4] Chemical contaminants are mainly generated by humans by different household practices, transport, agriculture, industry, energy manufacturing, waste management as well as biological sources.[4] The pollutants derived from different sources get into the air, water, and soil, and thus, to our food. There are a thousand potentially toxic contaminants, including large toxins of particulate matter (PM), nitrogen oxides, poly-cyclic aromatic hydrocarbons (PAHs) as well as heavy metals, pesticides, bisphenol A

plasticizer, polychlorinated biphenyls (PCBs)". Human exposure to these compounds occurs principally through the gastrointestinal, digestive and skin processes. In reality, there are countless possible contaminants and most were never tested for toxicity or health,[4] especially because of interactions with existing ones, new chemicals are constantly emerging. A wide range of diseases has been found to be linked to EP. Previous findings have shown a rise in the prevalence of malignancies; developing or exacerbating major coronary complications; several brain disorders; foetal complications; infancy; cardiac, endocrine and immune disorders; and elevated all-cause death rates.[1–5]

Allergies:" Air pollution (AP) is among the first five significant reasons of increased susceptibility to allergies, which are now becoming an epidemic.[6] Many chemicals may be combined with neutral, allergic substances and thus mild allergens," and therefore become more active and cause harder allergic reactions.[7,8] The children of women living in big urban communities suffered from childhood and adult allergies more frequently than children born to mothers in rural areas.[9] Pollutants penetrating the human body induce inflammation by cell activation or antigens that are involved in allergic or inflammatory reactions. It also boosts bronchial reactivity and causes coughingandasthma. Many studies have shown that susceptibility to prenatal and neonatal toxins already change the immune response to allergens during these cycles.[8]

Cardiovascular Disorders: They remain a leading cause of death despite their advances in the diagnosis and treatment of cardiovascular diseases." The classic risk factors include hereditary predisposition, ageing, diabetes, high blood pressure, emotions, lipid abnormalities, smoking, and obesity. However, the interests of chemical EP have grown in recent years, which in the field of cardiology has until now been somewhat undervalued. The London Great Smog has now shown the potential for a higher risk in hospitals and death due to cardiovascular and respiratory problems from sudden exposure to AP components. In clinical trials too, this correlation was verified.[10–12] There is also investigation of the processes underlying the impact of AP on the cardiovascular system. PM2.5 was suggested to infiltrate the blood from the pulmonary system and then to inner organs owing to their limited aerodynamic diameter, which leads to "systemic inflammation, oxidative stress, elevated development of pro-inflammatory cytokine, coagulation disruptions, atherosclerotic dislocation" of the plaques, and an increasing systemic vascular resistance.[10] In the other hand, there are contradictory findings from research that evaluate the effects of AP on cardiovascular deaths. Other contaminants such as chronic organic compounds, other than "PM 2.5 and gas components in" AP, can also damage the cardiovascular system (POPs). Dioxins, for example, can "cause endothelial cell toxicity, increase oxidative stressand cause inflammation and atherosclerosis, are an example of POPs.[3,13,14] The accumulation of such pesticides and cardiovascular diseases in farmworkers was also positively linked in a report.[15]

Central Nervous System Disorders: Experimental and clinical trials have found that AP even in the foetal phase can affect the brain. During birth, PAH and PM2.5 exposure effects the brain of foetuses adversely, impairing the psychomotor and intellectual growth and causing deficiency of concentration or excess.[16–18] A further research has shown that neurodegenerative brain conditions such as Alzheimer's and Parkinson's diseases can correspond to "PM2.5, nanoparticles, heavy metals, PAH's and various other materials found in AP.[19] The precise processes that underlie AP's detrimental impact on the brain are currently still investigated although certain "chemical substances (e.g. nanoparticles and PM2.5) can permeate the brain where they lead to local inflammation, oxidative stress, and brain gliosis.[20–22]

Pulmonary Disorders: The increased prevalence of respiratory exacerbations, as well as subsequent hospitalizations, was correlated with "short-term exposure to significant air contaminants (O3, CO, NO2, SO2, PM10, and PM2.5),[23–26] especially in patients with asthma and chronic obstructive pulmonary disease (COPD). The rise of 10 μg/m3 in PM2.5 levels" in short-run exposure was corresponding to a 2.5% improvement in COPD associated risk for admissions to hospitals (95% CI, 1.6% –3.4%); 10 μg/m3 increased in NO2, "with a 4.2% increase in hospital admits""(95% CI, 2.5% -6.0%), and an increase in 10 $\mu g/m^3$ in" the level of SO_2 coincided with a correspondingly high increase in hospital admissions.[27] In addition, the increased risk of death in these patients by each exacerbation of COPD requiring hospitalization.[23,24] Furthermore, AP patients report higher rates of bacterial and viral respiratory infections, as well as longer infection time leading to respiratory inflammation and the degradation of the mucosal barrier by chemicals.[25]

Type 2 Diabetes: A growing number of evidence suggests that certain common chemical compounds may help to establish type 2 diabetes and a condition usually associated with lack of physical exercise, obesity or consumption of excessively packaged foods. Such compounds include POPs such as dioxins, PCBs and contaminants that occur in fatty tissue in humans and animals. These accumulated substances can, among other consequences, lead to resistance to insulin and thereby contribute to diabetes.[28,29] Similar trials were carried out in many food products including drinking water, fish and rice for heavy metals (especially arsenic and mercury.[28] Bisphenol is also thought to be a contributing substance to diabetes. It is widely used for plastic production, "including mineral water bottles, as well as epoxy resins that coat" within food tanks. It is also common in the atmosphere and in our bodies. Bisphenol influences insulin and glucagon, inhibiting cell development and apoptosis, and

affecting cells, muscles, and liver function, causing insulin resistance. Bisphenol impairs insulin secretion.[28,29]

Malignant Neoplasm: There has been a dynamic change worldwide in the occurrence of malignant neoplasm.[6] The number of cancer morbidity will soon excelled the number of deaths due to cardiovascular diseases.[6] Although there is no doubt about the environmental causes in carcinogenesis, it is also impossible to assess the degree to which toxicity involves cancer etiology. Since no person can trace or forecast any potential associations between all hazardous substances he or she has been exposed to since the foetal age. In addition, certain genetic predispositions can increase the vulnerability to the effects of environmental pollutants.[30] Another research showed a possible correlation between dietary nitrosamine contamination and the occurrence of certain GMC, with a relative risk (RR) of 2.12 consumption quartiles being the highest to the lowest (95% CI, 1.04–4.33).[31] A major study in 12 cohorts of six European countries has shown that the relationship between adult malignant brain tumors and PM2.5 absorption is positive, but insignificant. (HR = 1.67; 95% CI, 0.89–3.14 per 10 μg/m3), as well as a weak positive or lack of associations with other air pollutants.[32] Radon radiation is currently thought to be a significant risk factor in lung cancer as well as hematopoietic neoplasm. Researchers from Korea found that a 10-Bq/m3 the increase in the concentration of indoor radon in women less than 20 years of age was linked to a 7% increase in the prevalence of non-Hodgkin lymphoma while no combinations have been identified with leukemia.[33] Some studies concluded that an adequate diet rich in vegetables and fruit can exert some protective anticancer effects (3), which may be counterbalanced by EP.

Conclusion

Today, we are damaging the world more quickly than will regenerate and EP contributes significantly to global morbidity and mortality, resulting in high health care costs. EP-related diseases often evolve over a long period of time and may be permanent. Global EP challenges the traditional recommendation" that, while there is no ecological certification, vegetables, fruit and fish be consumed daily in the form of a balanced diet. Awareness of some possible contaminants is still lacking, since most chemical pollutants were never assessed as harmful and risk to health. Research in ecology, biotechnology and toxicology is essential to decide which environmental pollutants are and at which levels are the most dangerous to animals and people. Reducing pollution, primarily because of poverty in many nations, is difficult. Interdisciplinary coordination and public awareness-raising activities may contribute to better environmental security. In order to contribute to deforestation and to protect the environment, international collaboration with government and non-governmental organisations is also essential.

References

1. ; 2016. Available from: https://www.who.int/quantifying_ehimpacts/publications.
2. Remoundou K, Koundouri P. Environmental Effects on Public Health: An Economic Perspective. Int J Environ Res Public Health. 2009;6(8):2160–2178. Available from: https://dx.doi.org/10.3390/ijerph6082160.
3. Hennig B, Petriello MC, Gamble MV, Surh YJ, Kresty LA, Frank N. The role of nutrition in influencing mechanisms involved in environmentally mediated diseases. Rev Environ Health. 2018;33(1):87–97. Available from: https://dx.doi.org/10.1515/reveh-2017-0038.
4. Briggs D. Environmental pollution and the global burden of disease. Br Med Bull. 2003;68:1–24. Available from: https://doi.org/10.1093/bmb/ldg019.
5. Kelishadi R. Environmental Pollution: Health Effects and Operational Implications for Pollutants Removal. Int J Environ Res. 2012;2012:1–2. Available from: https://dx.doi.org/10.1155/2012/341637.
6. Wojciechowska U, Didkowska J, Polsce ZIZW, Krajowyrejestrnowotworów. Centrum Onkologii - Instytutim Mari-iSkłodowskiej - Curie Available;.
7. Parsa N. Environmental factors inducing human cancers. Iran J Publ Health. 2012;41(11):1–9.
8. Oliveira PA, Colaço A, Chaves R, Guedes-Pinto H, P LFDLC, Lopes C. Chemical carcinogenesis. An Acad Bras Cienc. 2007;79(4):593–616. Available from: https://dx.doi.org/10.1590/s0001-37652007000400004.
9. ; 2018. Available from: http://www.who.int/news-room/fact-sheets/detail/pesticide-residues-in-food.
10. Newby DE, Mannucci PM, Tell GS, Baccarelli AA, Brook RD, Donaldson K. Expert position paper on air pollution and cardiovascular disease. Eur Heart J. 2015;36(2):83–93. Available from: https://dx.doi.org/10.1093/eurheartj/ehu458.
11. Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, et al. Long-Term Exposure to Air Pollution and Incidence of Cardiovascular Events in Women. N Engl J Med. 2007;356(5):447–458. Available from: https://dx.doi.org/10.1056/nejmoa054409.
12. Konduracka E, Niewiara Ł, Guzik B, Kotynia M, Szolc P, Gajos G, et al. Effect of short-term fluctuations in outdoor air pollution on the number of hospital admissions due to acute myocardial infarction among inhabitants of Krakow, Poland. Pol Arch Intern Med. 2019;129(2):88–96. Available from: https://dx.doi.org/10.20452/pamw.4424.
13. Humblet O, Birnbaum L, Rimm E, Mittleman MA, Hauser R. Dioxins and Cardiovascular Disease Mortality. Environ Health Perspect. 2008;116(11):1443–1448. Available from: https://dx.doi.org/10.1289/ehp.11579.
14. Burstyn I, Kromhout H, Partanen T, Svane O, Lang??rd S, Ahrens W, et al. Polycyclic Aromatic Hydrocarbons and Fatal Ischemic Heart Disease. Epidemiology. 2005;16(6):744–750. Available from: https://dx.doi.org/10.1097/01.ede.0000181310.65043.2f.

15. Sekhotha M, Monyeki K, Sibuyi M. Exposure to Agrochemicals and Cardiovascular Disease: A Review. Int J Environ Res Public Health. 2016;13(2):229. Available from: https://dx.doi.org/10.3390/ijerph13020229.
16. Jedrychowski WA, Perera FP, Camann D, Spengler J, Butscher M, Mroz E, et al. Prenatal exposure to polycyclic aromatic hydrocarbons and cognitive dysfunction in children. Environ Sci Pollut Res. 2015;22(5):3631–3639. Available from: https://dx.doi.org/10.1007/s11356-014-3627-8.
17. Edwards SC, Jedrychowski W, Butscher M, Camann D, Kieltyka A, Mroz E, et al. Prenatal Exposure to Airborne Polycyclic Aromatic Hydrocarbons and Children's Intelligence at 5 Years of Age in a Prospective Cohort Study in Poland. Environ Health Perspect. 2010;118(9):1326–1331. Available from: https://dx.doi.org/10.1289/ehp.0901070.
18. Perera FP, Wheelock K, Wang Y, Tang D, Margolis AE, Badia G, et al. Combined effects of prenatal exposure to polycyclic aromatic hydrocarbons and material hardship on child ADHD behavior problems. Environ Res. 2018;160:506–513. Available from: https://dx.doi.org/10.1016/j.envres.2017.09.002.
19. Chin-Chan M, Navarro-Yepes J, Quintanilla-Vega B. Environmental pollutants as risk factors for neurodegenerative disorders: Alzheimer and Parkinson diseases. Front Cell Neurosci. 2015;9:124–129. Available from: https://dx.doi.org/10.3389/fncel.2015.00124.
20. Calderón-Garcidueñas L, Vojdani A, Blaurock-Busch E, Busch Y, Friedle A, Franco-Lira M, et al. Air Pollution and Children: Neural and Tight Junction Antibodies and Combustion Metals, the Role of Barrier Breakdown and Brain Immunity in Neurodegeneration. J Alzheimer's Dis. 2014;43(3):1039–1058. Available from: https://dx.doi.org/10.3233/jad-141365.
21. Maher BA, Ahmed IAM, Karloukovski V, MacLaren DA, Foulds PG, Allsop D, et al. Magnetite pollution nanoparticles in the human brain. Proc Natl Acad Sci. 2016;113(39):10797–10801. Available from: https://dx.doi.org/10.1073/pnas.1605941113.
22. Duffy C, Swanson J, Northrop W, Nixon J, Butterick T. Microglial Immune Response to Low Concentrations of Combustion-Generated Nanoparticles: An In Vitro Model of Brain Health. Nanomaterials. 2018;8(3):155. Available from: https://dx.doi.org/10.3390/nano8030155.
23. Li J, Sun S, Tang R, Qiu H, Huang Q. Major air pollutants and risk of COPD exacerbations: a systematic review and meta-analysis. Int J Chron Obstruct Pulmon Dis. 2016;11:3079–91. Available from: https://doi.org/10.2147/copd.s122282.
24. Badyda A, Gayer A, Czechowski P, Majewski G, Dąbrowiecki P. Pulmonary Function and Incidence of Selected Respiratory Diseases Depending on the Exposure to Ambient PM10. Int J Mol Sci. 2016;17(11):1954. Available from: https://dx.doi.org/10.3390/ijms17111954.
25. Samoliński B, Raciborski F, Lipiec A, Tomaszewska A, Krzych-Fałta E, Samel-Kowalik P, et al. Epidemiologia Chorób Alergicznych w Polsce (ECAP). Alergologia Polska - Polish J Allergology. 2014;1(1):10–18. Available from: https://dx.doi.org/10.1016/j.alergo.2014.03.008.
26. Takizawa H. Impact of air pollution on allergic diseases. Korean J Intern Med. 2011;26(3):262–73. Available from: https://dx.doi.org/10.3904/kjim.2011.26.3.262.
27. DeVries R, Kriebel D, Sama S. Outdoor Air Pollution and COPD-Related Emergency Department Visits, Hospital Admissions, and Mortality: A Meta-Analysis. J Chron Obstruct Pulmon Dis. 2017;14(1):113–121. Available from: https://dx.doi.org/10.1080/15412555.2016.1216956.
28. Provvisiero D, Pivonello C, Muscogiuri G, Negri M, de Angelis C, Simeoli C, et al. Influence of Bisphenol A on Type 2 Diabetes Mellitus. Int J Environ Res Public Health. 2016;13(10):989. Available from: https://dx.doi.org/10.3390/ijerph13100989.
29. Konduracka E, Krzemieniecki K, Gajos G. Relationship between everyday use cosmetics and female breast cancer. Pol Arch Intern Med. 2014;124(5):264–269. Available from: https://dx.doi.org/10.20452/pamw.2257.
30. Parsa N. Environmental factors inducing human cancers. Iran J Publ Health. 2012;41(11):1–9.
31. Knekt P, Jrvinen R, Dich J, Hakulinen T. Risk of colorectal and other gastro-intestinal cancers after exposure to nitrate, nitrite andN-nitroso compounds: a follow-up study. Int J Cancer. 1999;80(6):852–856. Available from: https://dx.doi.org/10.1002/(sici)1097-0215(19990315)80:6<852::aid-ijc9>3.0.co;2-s.
32. Andersen ZJ, Pedersen M, Weinmayr G, Stafoggia M, Galassi C, Jørgensen JT. Long-term exposure to ambient air pollution and incidence of brain tumor: the European Study of Cohorts for Air Pollution Effects (ESCAPE). NeuroOncol. 2018;20:420–452. Available from: https://doi.org/10.1093/neuonc/nox163.
33. Ha M, Hwang SS, Kang S, Park NW, Chang BU, Kim Y. Geographical correlations between indoor radon concentration and risks of lung cancer, non-Hodgkin's lymphoma, and leukemia during 1999-2008 in Korea. Int J Environ Res Public Health. 2017;14(4):344–350. Available from: https://doi.org/10.3390/ijerph14040344.

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