openheart

Air pollution and cardiovascular risk: is it time to change guidelines?

Marco Bernardi ,[1] Giuseppe Biondi-Zoccai ,[2,3] Francesco Versaci[4]

> "One must be a sea, to receive a polluted stream without becoming impure."
> Friedrich Nietzsche, Thus Spoke Zarathustra

To cite: Bernardi M, Biondi-Zoccai G, Versaci F. Air pollution and cardiovascular risk: is it time to change guidelines?. *Open Heart* 2022;**9**:e001961. doi:10.1136/openhrt-2022-001961

Accepted 11 March 2022

Linked

► http://dx.doi.org/10.1136/openhrt-2021-001891

Check for updates

[1]Department of Clinical, Internal, Anesthesiology and Cardiovascular Sciences, University of Rome La Sapienza, Rome, Lazio, Italy
[2]Department of Medical-Surgical Sciences and Biotechnologies, University of Rome La Sapienza, Rome, Lazio, Italy
[3]Mediterranea Cardiocentro, Napoli, Campania, Italy
[4]UOC UTIC Emodinamica e Cardiologia, Ospedale Santa Maria Goretti, Latina, Lazio, Italy

Correspondence to
Dr Giuseppe Biondi-Zoccai; giuseppe.biondizoccai@uniroma1.it

Environmental pollution is a great challenge of our times that has been neglected for too long, despite its ongoing worsening and established adverse impact on health. The Global Burden Disease (GBD) study estimated that 9 million people died worldwide due to pollution in 2019, in most cases because of cardiovascular disease.[1] Realistically, health implications of the totality of all forms of pollution—the so-called pollutome—are even more remarkable.[2]

Among heterogeneous air pollutants, particulate matter (PM) plays a major and well-established detrimental role in human health and disease, in general, especially cardiovascular disease, mainly when its diameter is equal or less than 2.5 µm ($PM_{2.5}$).[3 4] Particles of this size may reach the alveolocapillary barrier, enter the systemic circulation and activate pathogenetic patterns such as inflammation, oxidative stress and dysregulation of the autonomic nervous system.[5] All of these effects can, for instance, increase plaque progression and favour its rupture and coronary instability.[6] Even short-term changes in levels of $PM_{2.5}$ can impact on cardiovascular events, as poignantly epitomised by recent studies performed in the COVID-19 pandemic: during the lockdown, critical cardiovascular events decreased, in line with the reduction of polluting emissions.[7–9]

The endless war against individual cardiovascular risk factors significantly reduced the burden of cardiovascular diseases, thanks to the identification of patient-level risk factors such as hypercholesterolaemia, diabetes, hypertension, smoking and obesity. However, cardiovascular risk factors can also be collective, involving large groups of individuals or even entire populations living in a city, region or nation, such as environmental pollution and weather. Thus, solely by extending prevention plans against collective risk factors, it will be possible to save other lives. Expanding the evidence base on the mechanistic link between is crucial to reinforce the need to act proactively against the detrimental effect of pollution. Indeed, it is established that $PM_{2.5}$ exposure may trigger acute cardiovascular events such as ST-elevation myocardial infarction and stroke.[9] Still, there is no clear evidence regarding the association between less dramatic changes in daily $PM_{2.5}$ concentrations and subclinical non-acute cardiomyocytes damage outside of the realm of acute cardiovascular events qualifying as a clinical myocardial infarction (MI).

In this issue of the journal, Wyatt *et al* originally report a wide retrospective cohort study of 4226 MI survivors in North Carolina, USA, evaluating the cardiac impact of short-term exposure to increased levels$PM_{2.5}$, and focusing on serum levels of cardiac troponin I (cTnI), a specific myocardial damage biomarker, capable of detecting even subclinical myocardial injury.[10] Strengths of this work include the assessment of daily $PM_{2.5}$ exposure based on merging data from satellite and land measurements, the linking of patient data with environmental data based on residential address at the time of the troponin assay and the use of sophisticated modelling technique specifying different timeframes (immediate, lag 0, as well as delayed lags, lag 1–4). They intriguingly found that even a tiny 10 µg/m^3 elevation in $PM_{2.5}$ 3 days before was associated with an average increase of 0.06 ng/mL higher in cTnI levels, demonstrating consistent cardiotoxic effects of this pollutant (figure 1).

Indeed, inadequate air quality may cause continual injury to the cardiac tissue and thus increase the risk of cardiovascular disease and accelerated cardiovascular disease in apparently healthy individuals as well as those with prior MI. Notably, this association was stronger in white males, with age at first MI≥65 years, and, paradoxically, those living in rural areas. Evidently, this study is currently the most substantial proof of the association between poor air quality and troponin rise out of the contest of MI, underlying a potential continuum of cardiac tissue damage. Until

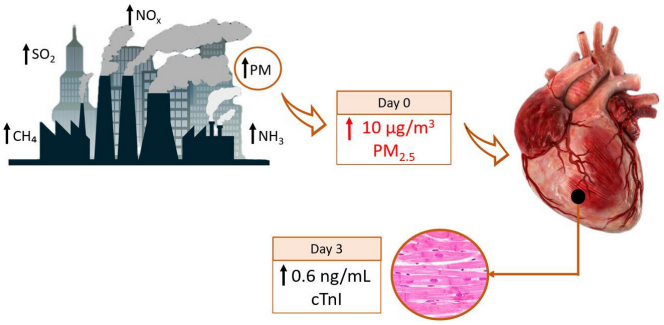

Figure 1 Impact of pollutants, and especially of particulate matter with diameter equal or smaller than 2.5 µm ($PM_{2.5}$), on risk of subclinical cardiomyocyte injury, as measured by cardiac troponin I (cTnI) levels. CH_4, methane; NH_3, ammonia; NO_X, nitric oxides; SO, sulfur dioxide.

now, only smaller studies and preclinical experimentations had shown a similar correlation.[11]

Perhaps the subpopulation with the highest troponin increase should have been further investigated, because it is known that advanced age (inflammaging), coexisting diseases, other individual cardiovascular risk factors and immunosuppression may increase air pollution sensitivity.[3] Furthermore, it would be interesting to understand the reasons behind the less dramatic troponin increase in patients living in a highly urbanised area after the same $PM_{2.5}$ exposure. It could be argued that people who live in urban areas seem to be less sensitive to prolonged exposure: is there any conditioning on them? In addition, the study did not consider some other key variables such as the already known cardiovascular risk factors, other previous diseases and socioeconomic status, which could impact significantly on this association. Finally, the single-centre study and the specific geographical area limit the generalisability of this study's results. Although a Chinese paper provided similar outcomes,[11] a multicentre study should be considered to explore out potential geographical differences in the association between $PM_{2.5}$ exposure and cTnI levels. This holds even truer as low-income and middle-income countries have drastically higher pollutant levels and fewer resources to provide renewable energy, so inevitably pollution may risk selectively killing especially the poorest and the most vulnerable.[2 3]

This global threat needs a global solution, as low-income and middle-income countries provide 55% of the worldwide population, and has become even more relevant given the Russian invasion of Ukraine. This article is another piece of increasingly scientific literature on air pollution harmful effects on cardiovascular health.

As a scientific community, we must spread awareness through the population and the clinical practice, and we must be proactive towards decision-makers to promote clean energy transition. Simple actions like traffic bans had significantly reduced coronary atherothrombotic events.[12] It is also necessary to reduce the disproportion between different parts of our planet and support along this process the low-income country where the pollution kills more people. In addition to population interventions, we also need to change clinical practice to guarantee individualised care and personalised medicine. To do that, it is necessary to revise current guidelines, including $PM_{2.5}$ exposure as a significant risk factor of cardiovascular disease, and fight it both as a collective and an individual risk factor as smoking, dyslipidaemia, hypertension or obesity.

Twitter Marco Bernardi @MarcoBernardiMD

Collaborators Not applicable.

Contributors All authors have equally contributed to design, drafting and final approval of the manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests GB-Z has consulted for Cardionovum, Innovheart, Meditrial, Opsens Medical and Replycare. All other authors report no conflict of interest.

Patient consent for publication Not applicable.

Ethics approval Not applicable.

Provenance and peer review Commissioned; internally peer reviewed.

Data availability statement No analytic data were used to prepare the present editorial.

ORCID iDs
Marco Bernardi http://orcid.org/0000-0001-9269-8829
Giuseppe Biondi-Zoccai http://orcid.org/0000-0001-6103-8510

REFERENCES

1 GBD 2019 Risk Factors Collaborators. Global burden of 87 risk factors in 204 countries and territories, 1990-2019: a systematic analysis for the global burden of disease study 2019. *Lancet* 2020;396:1223–49.
2 Landrigan PJ, Fuller R, Acosta NJR, *et al*. The Lancet Commission on pollution and health. *Lancet* 2018;391:462–512.
3 Rajagopalan S, Landrigan PJ. Pollution and the heart. *N Engl J Med* 2021;385:1881–92.
4 Biondi-Zoccai G, Frati G, Gaspardone A, *et al*. Impact of environmental pollution and weather changes on the incidence of ST-elevation myocardial infarction. *Eur J Prev Cardiol* 2021;28:1501–7.
5 Miller MR. Oxidative stress and the cardiovascular effects of air pollution. *Free Radic Biol Med* 2020;151:69–87.
6 Montone RA, Camilli M, Russo M. Air pollution and coronary plaque vulnerability and instability: an optical coherence tomography study. *JACC Cardiovasc Imaging* 2021:00692–6.
7 Kwon OK, Kim S-H, Kang S-H, *et al*. Association of short- and long-term exposure to air pollution with atrial fibrillation. *Eur J Prev Cardiol* 2019;26:1208–16.
8 Calcagno S, Di Pietro R, Dei Giudici A, *et al*. Air pollution, climate changes and cardiovascular diseases: a nightmare threesome! *Minerva Cardioangiol* 2020;68:282–4.
9 Biondi-Zoccai G, Rodriguez-Granillo GA, Mercadé JM, *et al*. Interplay between climate, pollution and COVID-19 on ST-elevation myocardial infarction in a large metropolitan region. *Minerva Med* 2021.
10 Wyatt L, Kamat G, Moyer J, *et al*. Associations between short-term exposure to air pollution and cardiomyocyte injury in myocardial infarction survivors in North Carolina. *Open Heart* 2022. doi:10.1136/openhrt-2021-001891
11 Xu H, Brook RD, Wang T, *et al*. Short-term effects of ambient air pollution and outdoor temperature on biomarkers of myocardial damage, inflammation and oxidative stress in healthy adults. *Environ Epidemiol* 2019;3:e078.
12 Versaci F, Gaspardone A, Danesi A, *et al*. Impact of temporary traffic bans on the risk of acute coronary syndromes in a large metropolitan area. *Panminerva Med* 2020;62:252–9.