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EDITOR'S NOTE

JAHA Spotlight on Air Pollution and Cardiovascular Disease: A Call for Urgent Action

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The World Health Organization (WHO) estimates that nearly 90% of the world's population is exposed to ambient air pollution, which accounts for up to 7 million deaths annually and loss of 147 million healthy life years.[1,2] These numbers are perhaps an underestimate, with another recent study reporting nearly 10 million annual deaths directly attributable to the long-term exposure to fine particulate matter.[3]

Several observational studies have established the relationship between the short- and long-term exposure to ambient pollution (including fine particulate matter [$PM_{2.5}$], ozone, and vehicular pollution) and the risk of cardiovascular morbidity and mortality.[4] However, there are several gaps in this rapidly accumulating evidence.

In this issue of the *Journal of the American Heart Association* (*JAHA*), we feature several publications that elucidate the critical relationship between ambient air pollution with cardiovascular risk factors, such as hypertension, and address some of the existing knowledge gaps. Some of these studies have linked their findings with an urgent need to re-evaluate regulations and policies, while another study for the first time, has provided important mechanistic insight.

Huang et al evaluated the impact of both acute and long-term exposure to air pollutants (PM_{10}, $PM_{2.5}$ and nitrogen dioxide [NO_2]) on both systolic and diastolic blood pressure (BP) among children and adolescents.[5] In their meta-analysis involving 14 studies, they found that acute exposure to increasing concentrations of PM_{10} is associated with an increase in systolic BP. The adverse impact on systolic BP was significantly and numerically more with long-term exposure than short-term exposure. Moreover, both systolic and diastolic BP increase with the long-term exposure to $PM_{2.5}$ and PM_{10}.

Guo and colleagues evaluated the impact of short-term exposure to various sizes of the particulate matter and its constituents on BP in healthy participants.[6] They found that acute exposure to any particulate size between 0.2 to 2.5 μm and their constituents, such as chloride or vanadium, is associated with increased systolic BP. These findings are consistent with the findings from other studies in this Spotlight.[5] Furthermore, this study highlighted that it is not only the size of the particulate matter that adversely impact cardiovascular health, but the constituents of particulate matters also have a significant impact on cardiovascular well-being.

Liao and colleagues[7] and Ward-Caviness et al[8] have evaluated some of these relationships in at-risk populations. Liao et al evaluated the risk of a cardiovascular events with increased exposure to fine particulate matter among those with a previous history of myocardial infarction or stroke. Their findings suggest that, similar to healthy populations, these high-risk populations are also susceptible to the adverse cardiovascular impact on exposure to a higher concentration of particulate matter.[7] Interestingly, the cut-off levels of concentration of these particulate matters (ie, the levels when there is an increased cardiovascular risk) vary for those in the high-risk groups, and it seems the current cut-offs may

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not optimally protect those with pre-existing cardiovascular disease.

Ward-Caviness et al have cleverly linked the electronic health records with the environmental exposure data to study the readmission rates amongst recently discharged patients with heart failure.[8] They found that for each unit increase in exposure to $PM_{2.5}$ levels, there was about a 14% significant increase in the risk of 30-day readmission and about a nine per cent increase in the risk of a hospital visit. These findings also illustrate the scope and extent of the adverse health impact of air pollutants, particularly amongst at-risk populations.

One of the most exciting studies in this issue is by Rankin and colleagues.[9] They investigated the primary mechanism of increase in BP with the acute exposure to vehicular pollutants, such as diesel fumes. In their elegantly designed study, they exposed healthy adults to diluted diesel exhaust in a controlled manner and measured their sympathetic nervous system activity. They found that acute exposure to diesel exhaust pollution is associated with increased muscle sympathetic nerve activity. This study is possibly the first direct biological evidence for autonomic imbalance as a mediating pathway, whereby air-pollutants cause excessive sympathoexcitation. This may explain not only the increase in BPs seen in some of the studies[5,6] but also, perhaps may support some of the studies that have shown the relationship between acute exposure to the air pollutants from traffic and the risk of acute cardiovascular events, such as myocardial infarction.[10]

Brook and Rajagopalan, in their editorial, have explained the importance of these findings, mainly using them to call for the changes to the regulatory framework.[11] They believe there is an urgent need to review current regulatory standards by considering not only daily or annual levels of PMs but also some criteria that can categorize the risk posed by the acute rise in the levels of these particulate matters in a shorter time window, which is not reflected in daily or annual standards.

Aside from the air pollutants, this Spotlight also includes a Viewpoint article reviewing existing evidence on the relationship between the environmental exposure to lead or cadmium and the risk of cardiovascular disease. In their Viewpoint, Lamas et al have argued that some metal-based environmental contaminants, particularly lead and cadmium, should now be recognised amongst the traditional cardiovascular risk factors.[12] This article brings together the evidence that almost all environmental contaminants adversely affect cardiovascular health in one way or another.

In summary, this Spotlight has brought into the limelight not only the adverse health impact of both short-term and long-term exposure to ambient air pollution, but it also has underscored the need for urgent action both from policymakers and public health specialists. There is an urgent need to review the policy regarding the acute but transient rise in the concentration of these pollutants; to broaden some policy decisions to limit this adverse exposure to at-risk populations and children; and to rapidly expand the definition of modifiable cardiovascular risk factors to not only include air pollution as one of the modifiable risk factors but also to include other environmental pollutants. Identification of these novel and modifiable risk factors at the same level as that of traditional cardiovascular risk factors will force policymakers to adopt stringent criteria and robust measures to limit their exposure amongst general and at-risk populations. This may help reduce the hidden cardiovascular toll associated with them and perhaps reduce further the already plateaued population-level cardiovascular disease burden.

ARTICLE INFORMATION

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