

Editorial

World Heart Day - creating heart healthy environments

The conventional risk factors for cardiovascular diseases (CVD) like hypertension, dyslipidaemia, diabetes mellitus and smoking have largely overshadowed and perhaps precluded any sincere efforts towards discovery of new risk factors. However, environmental influences are waiting in the pipeline to be accounted for an increased chance of development of cardiovascular diseases. Many large epidemiological studies have reported a definite link between the exposure to fine air particles and an increase in cardiovascular mortality¹. Statistically significant correlations are now available for both short and long term exposure and correlations with heart failure and arrhythmias^{1,2}. As per the WHO around 7 million people died, one in eight of total global deaths, as a result of air pollution exposure. Regionally, low- and middle-income countries in the WHO South-East Asia and Western Pacific Regions had the largest air pollution-related burden in 2012, with a total of 3.3 million deaths linked to indoor air pollution and another 2.6 million to outdoor air pollution³. This finding more than doubles the previous estimates and confirms that air pollution is now perhaps the world's largest single environmental health risk⁴.

Long term effects

The Six Cities Study², by Harvard environmental epidemiologists group, addressed this vital issue. In this study, 8000 participants from six US cities with varying levels of air pollution, were followed up for 14-16 yr and reported a 26 per cent increase in all cause mortality (mostly cardiovascular) in the most heavily polluted city when compared to the least polluted one. In another study, Pope *et al*⁵ covering more than half a million people over 16 years noted that fine

particulates surprisingly were more strongly linked with cardiovascular deaths compared to respiratory causes. The air pollutants like ozone, carbon monoxide, nitrogen oxides, sulphur dioxides and lead also appear to have evident links with cardiovascular disease (CVD).

In another large study, The Women's Health Initiative Observational study⁶, database of more than 65,000 post-menopausal women without prior CVD was studied for the relation between long term exposure to air pollutants and the risk for a first cardiovascular event. After correction for all confounding factors, it was concluded that for each 10 µg/m³ increase in pollution concentration, there were significant increases in the risk of any cardiovascular event, death from CVD and of cerebrovascular events (hazard ratios 1.24, 1.76 and 1.35 respectively)⁶. The American Health Association (AHA) Scientific Statement (2004)⁷ has finally acknowledged that air pollutants pose a "serious public health problem" for CVD.

Short-term effects: In addition to long-term risk, short-term exposure to air pollutants (both ozone and fine particulate matter) has been associated with acute coronary ischaemic events. In a study of 12,000 patients, a short-term increase in fine ambient particulate matter had shown a positive correlation with an increase in acute ischaemic coronary events². In a systematic review and meta-analysis of data from 34 studies, carbon monoxide, nitrogen dioxide, sulphur dioxide, and small particulate matter (<10 and <2.5 microns) were all associated with an increased risk of myocardial infarction, with the overall population attributable risk ranging from 1 to 5 per cent⁸.

Environmental noise like road or air traffic has also been correlated to an increased risk of developing CVD⁹. This effect is hypothesized to be due to stress-related dysregulation of the autonomic nervous system, leading to an increase in hypertension and subsequent CVD.

Pathophysiological links: Numerous theories have been postulated to establish causal association between environmental toxins and cardiovascular adverse events. A few of these untoward effects may be mediated via atherosclerosis, vasoconstriction and changes in heart rate variability, blood pressure, coagulation, abnormal platelet activation, endothelial dysfunction with their consequent acute and chronic clinical sequelae^{10,11}.

Possible mechanisms by which fine particulate air pollution may increase the risk of CVD include (i) an increase in mean resting arterial blood pressure through an increase in sympathetic tone and/or the modulation of basal systemic vascular tone¹⁰; (ii) an increase in the likelihood of intravascular thrombosis through transient increases in plasma viscosity and impaired endothelial dysfunction¹¹; and (iii) the initiation and promotion of atherosclerosis^{12,13}.

Preventive measures: The preceding facts and figures lead us to a sufficiently irrevocable conclusive link between the environmental pollution - be it in the air we breathe, food we eat, soil we grow, fuel we burn, noise we hear and even the stress we bear, and cardiovascular disease progression. The evidences are sufficiently strong for us to start taking preventive measures against this all pervasive hazard.

In the western and advanced nations, environmental cardiology appears to be increasingly becoming a factor in research and public policy discussion. Strict implementations of air quality regulation and pollution levels are possibly contributing to a meaningful increase in life expectancy and reduction in cardiovascular mortality.

The obvious impediment in the developing countries is a lack of awareness coupled with a non aggressive policy for the prevention of non communicable diseases. Some of the suggestions mentioned below can help us progress in this direction: (i) Air pollution levels displays should be available in each city at crowded places, to increase awareness.

(ii) People should be discouraged to walk/exercise at places with high air pollution levels.

(iii) The use of vehicle emanating fewer pollutants is already being encouraged by the Government. It should be made mandatory and strictly enforced.

(iv) Environmental factors should be referred to as reversible risk factors for atherosclerotic coronary artery disease in the documents, textbooks and in a national health compendium.

(v) Awareness of such a hazard should be prominently displayed at par with smoking and tobacco in various Government sponsored advertisements.

(vi) Posters and slides at places of entertainment like multiplexes and shopping malls can support this message.

(vii) Innovative catchy cartoons and drawings can be displayed at school fetes and functions.

(viii) In any future environmental study, cardiovascular health should be included as an endpoint.

(ix) The drug industry should be advised to adopt this aspect as a thrust area for research and innovation. Regional differences in disease patterns may be correlated and highlighted to expose this insidious link.

Creating a heart healthy environment, being the result of a slow and collective process without many immediate tangible benefits holds us back from working towards this goal with a zest and zeal which it merits. However, knowledge, awareness and conviction regarding its importance and utility and multiple benefits should be a sufficient motivator for our present day thinkers and administrators.

To summarize, the environmental toxins are significantly contributing to the adverse cardiovascular events. The gravity of this issue should be appreciated. The target is to create a heart healthy environment for all.

Nakul Sinha^{1,*}, R.K. Saran² & Mansoor Hasan³

¹Sahara Hospital, Former Head
Department of Cardiology
Sanjay Gandhi Postgraduate
Institute of Medical Sciences

²Department of Cardiology &

³Professor Emeritus, King George's
Medical University, Lucknow 226 003, India

*For correspondence:
sinha.nakul@gmail.com

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