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Molecular basis of endothelial dysfunction due to mercury exposure: An interface between nitric oxide and oxidative stress

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Mercury, a heavy metal belonging to the transition element series of the periodic table, is widespread and persistent in the environment as an industrial pollutant. Any pollutant affecting human is transported to various organs by the cardiovascular system. Epidemiological and animal studies have suggested a strong association between the environmental and occupational exposure to mercury and risk of cardiovascular diseases (CVD). Toxicity from mercury is associated with *in vivo* oxidative stress. Mercury exposure induce the generation of reactive oxygen species (ROS) with subsequent oxidative damage in several organs and systems, as well as alter the antioxidant defence system in cells (6-10).Vascular endothelium, is very sensitive to oxidative stress and plays a vital role in the organization and function of the blood vessel and maintains homeostasis of the circulatory system and normal arterial function. Functional disruption of the endothelium is recognized as the beginning event that triggers the development of consequent cardiovascular disease (CVD) including atherosclerosis and coronary heart disease. Oxidative stress which results in endothelial dysfunction and loss of endothelium dependent vasorelaxation is one of the most commonly observed cardiovascular effects of mercury exposure. The endothelium can evoke relaxations and contractions of the underlying smooth muscle, by releasing vasoactive agents. Nitric oxide (NO), formed by endothelial NO synthase (eNOS), is the best characterized endothelium derived relaxing factor (EDRF). The release of NO is down regulated/upregulated by factors like oxidative stress, estrogen and diseases like diabetes and hypercholelestrolemia etc. The inhibition/activation of eNOS by mercury, affecting the NO release is majorly regulated by superoxide anions and minorly may be by insulin, estrogen, omega 3 unsaturated fatty acid and hypercholesterolemia and is one of the proposed mechanisms for mercury-induced vascular diseases. In addition, during exposure to mercury, overproduction of reactive oxygen species (ROS) can occur, resulting in oxidative stress, which is another major risk factor for endothelial dysfunction.

Conclusion: NO signaling mechanism and oxidative stress play a vital function in the mercury –induced cardiovascular diseases in the populations exposed to mercury.

Biography

Swati Omanwar completed her PhD in 2008 in Medical Physiology from Faculty of Medical Sciences, University of Delhi. She is presently as Scientist at School of Sciences, Indira Gandhi National Open University, New Delhi. She has published more than 17 papers in reputed journals and has been serving as reviewer for many.

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