Lead, Smoking, and Peripheral Vascular Function

To the Editor:

We read with interest the article by Argacha et al.1 dealing with acute effects of passive smoking on peripheral vascular function. The authors presented data concluding that passive smoking specifically increases aortic wave reflection through a nicotine-dependent pathway and impairs microvascular function, even after the end of the exposure. Both tobacco and nontobacco passive smoking inhalation increase plasma asymmetrical dimethyl-arginine levels.1 It is well documented that exposure to environmental tobacco smoke is a strong contributor to cardiovascular mortality, accounting for >50,000 deaths annually in the United States.2

In a recent study from Greece, a 12 times higher lead concentration was found in tobacco smokers than in nonsmokers living in the midaltitudes of the northern hemisphere.3 In this context, we showed that plasma and intracellular elevated lead concentrations contributed to the development of hypertension because of tobacco or nontobacco smoking or environmental exposure.4

In addition, in human lymphocytes, elevated lead concentrations with protein denaturation were also found in our studies.4 This is in concordance with the study from Prozialeck et al.5 showing the vascular system as a target of lead toxicity.

In conclusion, the article by Argacha et al.1 is excellent; however, the role of a lead overload resulting in acute and chronic effects on peripheral vascular function has to be taken in account, too. In addition, we agree that a tobacco-free cigarette cannot be considered as a safe cardiovascular smoke, especially because the role of fine particle matter or lead in the endothelial toxicity of smoke and air pollutants remains important.

Disclosures

None.

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