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\(^4\) 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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