

Cardiovascular risk from smoking: doubt or absolute certainty?

Editorial

In the past, I wrote a research paper related to cardiovascular damage from smoking.¹ Consistent data demonstrated undoubtedly a clearly defined and reproducible harm to the heart and blood vessels caused by cigarette smoking. The amount of the damage varies according to different factors like type of smoking, duration of the exposure and health status of the individuals who met tobacco toxics. However, unanimous opinion did not exist in this statement.

Year after year, because of extensive and effective researches, as clinical and experimental findings, the topic achieved a significant result: the assessment of a cardiovascular damage from smoking characterized by establishing pathological alterations. These have shown to follow a typical course due to the effects of two main compounds of cigarette smoke, nicotine and carbon monoxide. The latter should not be considered as a product of tobacco leaf, but, on the contrary, as the combustion of manufactured tobacco and the paper of the cigarette. However, carbon monoxide has to be seen as the most dangerous chemical of smoking since it is mainly responsible of irreversible cardiovascular pathology in the time.^{2,3} Nicotine causes primarily a functional and reversible damage to the heart and blood vessels in both active smokers and individuals passively exposed to tobacco smoking.⁴ This substance, which reaches different concentrations according to the type of cigarettes smoked,⁵ exerts direct and mediated effects due to adrenergic and sympathetic stimulation of the heart with impaired cardiac performance and endothelium, which meets a dysfunction characterized by impaired endothelium-dependent vasodilatation due to reduced nitric oxide release and changes in platelet function. There is evidence that the alterations caused by nicotine and its metabolite cotinine are stably reproducible in those experiments exactly conducted with the same protocol of study. Therefore, this fact shows with no doubt the certainty of cardiovascular damage related to a smoking compound. In addition, nicotine burns at a temperature of 240°C below the boiling point of 247°C. Chemically, that means the alkaloid has an auto-ignition point less than that of boiling, allowing the cigarette to diffuse up to nicotine decomposition occurring at a boiling level with maximum of substance absorption. Thus, individuals anyway exposed to smoke feel the harmful effects at a high level of toxicity as well as to a prolonged addiction of the chemical.

Carbon monoxide has been documented to be immediately responsible of increased concentrations of carboxy hemoglobin. This alters oxygen transport to the tissues and cells, inducing hypoxia of various degrees, which is closely related to the metabolic demand of structures involved.³ Generally, carbon monoxide from a single cigarette smoked achieves a small concentration in the blood, which determines initially functional but transient effects. However, there is evidence of stronger ischemic responses caused by this chemical related to the hypoxia as documented by the reduced exercise tolerance during smoking exposure.⁶ On the contrary, chronic and prolonged exposure shows irreversible alterations of the heart and blood vessels as ischemic heart disease, stroke rate, and coronary and systemic

artery lesions consisting of multiple narrowing and thickening of the vascular wall with increased arterial stiffness.⁷

And, what about the cardiovascular damage? There is evidence that it is now as was before with a wide spectrum of alterations differently associated with no documented data of prevailing one type rather than another one.⁸ Myocardial infarction due to cigarette smoking recognizes two pathogenic mechanisms: coronarogenic as a result of coronary atherosclerosis and its complications, and toxic, which is a direct consequence of carbon monoxide. In addition, smokers usually display some altered coagulation parameters with increased blood fibrinogen levels. Generally, all pictures of ischemic heart disease may be observed with a significant major rate of myocardial infarction and chronic effort angina. It is worth noting that microcirculatory alterations,⁹ often neglected, play a strong role in the mechanisms of cardiovascular damage from smoking.

Microcirculation primarily involves resistance arteries and arterioles up to blood reflux in the great venous system. Functional and morphological alterations from cigarette smoking consist of the vascular lumen narrowing and thrombo-embolism as an effect of endothelial and blood cell changes. However, it is worth noting that conduit arteries mainly show degenerative atherosclerotic alterations, on the contrary the changes from smoking observed in the resistance arteries are of a hyperplastic type with organ damage and failure of different degree developed in a long time. In conclusion, my personal opinion is the certainty of cardiovascular damage from smoking. In the past I described a theorem, known as the theorem of Leone,¹⁰ which

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told as follows. “Active smoking injures the cardiovascular system chronically, causing structural lesions, which become, in the long run, irreversible alterations mainly related to coronary atherosclerosis. In contrast, passive smoking causes transiently impaired cardiac performance which may be considerably more harmful for those people with established ischemic heart disease..... Irreversible pathological alterations characterized chronic and prolonged exposure to passive smoking similarly to what occurs in active smokers.”

Finally, why do not include smoking as an etiologic factor of cardiovascular disease? Indeed, cigarette smoking constantly causes specific alterations, whose degree depends on the duration of the exposure. This statement should be seriously taken into account.

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Conflicts of interest

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