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LETTER TO THE EDITOR

TOBACCO SMOKING, A MODIFIABLE CARDIOVASCULAR RISK FACTOR

EL HÁBITO DE FUMAR, UN FACTOR DE RIESGO CARDIOVASCULAR MODIFICABLE

Hector Roche Molina, MSc.[1]; Omaida J. López Bernal, MD.[2]

1. Master in Primary Health Care Emergencies. Bachelor in Nursing. Assistant Professor. Cardiocentro "Ernesto Che Guevara". Villa Clara, Cuba.
2. First Degree Specialist in Pathology. Instructor Professor. Jose Luis Miranda Pediatric Hospital. Villa Clara, Cuba.

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To the Editor:

Every day there are more cases with a high number of adverse health risk factors[1]. These are accentuated in the care of patients with heart disease, with greater damage for the patients and increased costs related to health complications, hospital stay, and reintegration into society: tobacco smoking is one of these factors[2].

Although estimates of life expectancy reflect how old a person expects to live, they do not specify the "expected" state of health in life, due to mortality rates for certain environments. Mortality statistics by themselves are insufficient to fully describe and compare the health of diverse populations because they underestimate the burden of "poor health" caused by chronic diseases and provide little information on non-fatal health outcomes[1].

According to the World Health Organization, cardiovascular diseases caused 17.5 million deaths worldwide each year and account for half of all deaths in the United States and other developed countries, and also in developing countries[1,3]. As a whole, they constitute the leading cause of death in adults[3]. There are coronary risk factors such as smoking, that affect health. The striking thing about this situation is that it is a modifiable factor, and patients are unaware of the impact of this bad habit on the vascular endothelium[4].

Aladro Miranda[5] suggests that ischemic heart disease increases proportionally with tobacco smoking, because this habit is linked at least to 25% of all cases of cardiovascular disease, and the likelihood of suffering from heart failure is, among smokers, 1.2 to 1.4 times higher than in nonsmokers. The author notes that only 10 years after smoking cessation, the risk of cardiac ischemia reaches the same level of nonsmokers,

✉ *MSc. H Roche Molina*
Cardiocentro "Ernesto Che Guevara"
Cuba 610, entre Barcelona y Capitán Velazco
Santa Clara, CP 50200, Villa Clara, Cuba

but this assertion is controversial, because atherosclerosis is a chronic inflammatory disease[6,7], and tobacco noxious substances (carbon monoxide, tar and nicotine) damage the vascular endothelium and favor the accumulation of macrophages, foam cells, lymphocytes and platelets[8], leading to the formation of fatty streak which is the first injury that appears in the arterial intima[6]. Subsequently, smooth muscle cell proliferation triggers and eventually atheromatous plaques develop[7-9]. Once this complex process has developed, there is no possibility of "back to top", but only to limit -after smoking cessation- the progression of atherogenesis, reduce the size of already formed plaques and reduce the risk of atherothrombosis[10].

Studies in smokers have shown that heart diseases are their biggest problem and there is a significant causal relationship, since tobacco smoking increases several times the risk for ischemic heart disease[10] because it decreases high-density lipoproteins (HDL), increases platelet aggregation, carboxyhemoglobin levels and direct irritation of the endothelium by nicotine and carbon monoxide,[7-9,11] and all this promotes vasospasm, thrombosis, and increased myocardial oxygen consumption by increasing production of catecholamines and heart rate[12].

Furthermore, carbon monoxide and hemoglobin form a stable compound, carboxyhemoglobin, which has high affinity for oxygen and does not give it to the oxidation/reduction systems of the tissue[13]. Hypoxia increases endothelial permeability and allows the passage of cholesterol-rich lipoproteins contributing to the formation of the atheroma plaque, and restenosis, in the case of patients with angioplasty. The endothelial injury breaks prostacyclin-thromboxane balance in favor of the latter, and predisposes to platelet aggregation due to the release of thrombogenic factors, and secondarily, smooth muscle cells are stimulated and migrate and become myo-intimal elements that acquire diapedesis capacity, phagocytosis and proteoglycans and collagen secretion, which are also constituents of the atheroma plaque[7-9,14-17].

Nicotine has facilitation effects on postsynaptic ganglionic transmission and increases noradrenaline and adrenaline release, which have proven ability to damage the vascular endothelium, are inducers of platelet aggregation, increase levels of free fatty acids and hepatic synthesis of very low density lipoproteins (VLDL)[11,16] which are, in turn, precursors of low-density lipoproteins (LDL)[7]. The increase in sympathetic tone induced by nicotine also increases myocardial oxygen consumption, which may result in an imbalance between increased demand and reduced supply, in the event of coronary atheromatosis[5].

Smokers are 70% more likely to die from cardiovascular disease than nonsmokers[5]. According to statistics, smokers live 6 years less than non-smokers and a nonsmoker can suffer a heart attack, but if he or she had been a smoker it would have happened 6 or 7 years before.[18] Those who consume 20 or more cigarettes per day have a mortality rate from cardiovascular disease 2 to 3 times greater than nonsmokers[5,18].

Smoking is an undeniable risk of restenosis in angioplasty patients, and occlusion of the grafts in those who underwent revascularization surgery[19,20].

And as the vascular endothelium (considered as the second largest organ in the body after the skin) is only one, smoking also increases the risk of stroke and peripheral arterial disease, which can cause disabling brain injury, lower limb amputations or death[21].

Since 1987, every May 31st the "World No-Snuff" is celebrated as an example of the scientific community's effort to point out, educate and prevent the risks of tobacco consumption for the health. However, we need greater attempts, on the part of the Government, -as has been done in Spain and other European countries[22]- to promote more effective policies for reducing this important cardiovascular risk factor.

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