How does to Quit Smoking Influence the Effects of Other Major Cardiovascular Risk Factors?

Editorial

Volume 3 Issue 6 - 2015

Aurelio Leone[1,2,3]*

[1]*Fellow of the American Society of Hypertension (ASH), USA*
[2]*Fellow of the Royal Society for Promotion of Health (FRSPH), United Kingdom*
[3]*Editor-in-Chief of the Journal of Cardiology & Current Research, USA*

***Corresponding author:** Aurelio Leone, Via Provinciale 27, 19030 Castelnuovo Magra (SP), Italy, Tel: +393472272215; Email: reliol@libero.it

Received: November 14, 2015 | **Published:** November 17, 2015

Editorial

It has been recognized that a strong interaction exists between the major cardiovascular risk factors [1-3], which may exert their harmful effects on cardiovascular system either independently or variably associated with statistical, probabilistic evidence, in this case, an exponential increased rate of disease and, consequently, more powerful response. A different matter concerns the effects of lack or changing a cardiovascular risk factor, being this occurrence not yet well established. On the contrary that should be carefully known since there are several methods, if applied, to control the specific characteristics of those factors potentially harmful for the cardiovascular system.

The detrimental effects of the tobacco undoubtedly could be absolutely avoided by quitting smoking [4,5]. How does it modify the characteristics of the other major risk factors? This is the goal of the editorial. Four major cardiovascular risk factors, respectively diabetes mellitus, abnormalities of lipid metabolism, obesity and hypertension rather than others play synergistic effects with cigarette smoking. The first three belong to the group of metabolic disorders, while the fourth identifies also changes in arterial tone and blood flow. It is a great pleasure for me in this editorial to briefly discuss the basic concepts that emerge about the relationship between quitting smoking and other major cardiovascular risk factors to establish its importance to prevent and reduce the rate of heart disease. If so, quitting smoking should be considered a habit to follow not only with regard to itself, but also for the favorable effects, still variably interpreted by the different reports, on the group of other major risk factors.

In spite ofwhat's believed in the past, overwhelming evidence indicates that quitting smoking quickly takes back smokers to the status of a never smokers [5,6] with a reduction in the rate and events of cardiovascular disease. The risk of coronary pathology in ex-smokers declines rapidly after quitting and within 2–3 years is similar to the risk for non-smokers. A controversial opinion still exists about the direct effects of cigarette smoking on metabolic disorders with regard to the exponential reduction-rate of cardiovascular events.

Diabetes mellitus, alone, increases the cardiovascular risk up to 10% with a highly major rate when is associated with other factors, including cigarette smoking. In quitting smokers there would be a cardiovascular risk equal to that of diabetes mellitus alone. Really, this does not exactly occur. Two relatively recent papers [7,8] achieved completely opposite conclusions. The one [7] emphasized that a significant metabolic improvement together with cardiovascular risk reduction was seen in diabetic patients who stopped smoking, particularly when they were pharmacologically treated, while another [8] reported a worsening in glycemic control in similar patients with no change in cardiovascular risk. Generally, all the metabolic parameters that characterize metabolic syndrome are variably influenced by smoking cessation.

Lipid metabolism disorders are more strongly associated with cigarette smoking than that of diabetes mellitus. Thus, lipid metabolism alterations, primarily increased concentrations of LDL-Cholesterol, actively contribute to atherosclerotic plaque formation as cigarette smoking does, while hyperglycemia of diabetes mellitus exerts primarily its effects on both the arterioles, which mainly show fibro-sclerotic lesions accompanied by progressive organ damage and failure, and conduit arteries, which may develop atherosclerotic lesions. Therefore, micro and macro vascular pathology are a typical pattern of diabetes mellitus also in nonsmoker individuals.

It is worth noting that the main compounds of cigarette smoking, nicotine and carbon monoxide, exert both direct and mediated effects of catecholamine release and sympathetic nervous system stimulation, which enhance LDL-Cholesterol concentrations. There is evidence that oxidized LDL-Cholesterol is the major precursor of atherogenesis because it leads monocytes to binding endothelial cells, which incorporate fat droplets and migrate within the vessel wall [9]. However, smoking cessation favorably influences a reduction of atherogenic effects due to LDL-Cholesterol [10] through an improved HDL-Cholesterol, total HDL, and large HDL particle concentrations. In addition, a gain in body weight has been observed in quitting smokers.

These observations allow analyzing the role of obesity, which is widely debated with regard to the type of fat, its significance and distribution. However, of the two risk factors already discussed, diabetes mellitus and disorders of lipid metabolism, undoubtedly the first is more strongly correlated to the pathology of obese individuals being often a metabolic disorder associated with metabolic syndrome. Although health benefits of smoking cessation are undeniable, this habit may be associated with a

small increase in the prevalence of overweight, whose significance is still far to be established [11]. However, opinions support that the gain in body weight could be a temporary occurrence, and physical activity and appropriate diet should control this outcome [12].

My personal and foreseen opinion to explain the gain in body weight following smoking cessation is it significantly improves the health status of the individuals, who, therefore, show a major attention to other gratifications of the life like wholesome food and recreational activities. Appropriate dietetic measures and fitness practice undoubtedly could be able to correct body weight gain, which, generally, does not adversely influence cardiovascular risk. However, as aforementioned, all the features related to overweight and obesity need further explanation, also including smoking cessation, in an attempt to properly classify the true risk of obese individuals. Thus, the evidence indicates that body fat distribution, white and/or brown adipose tissue and genetic patterns play a strong role as determinants of the metabolic imbalance [13-15].

With regard to hypertension, the results observed after quitting smoking are certainly clearer than that of other major cardiovascular risk factors. It is worth noting that smoking and hypertension exert not only an independent, but also additive effects to harm the heart and blood vessels [1]. However, conflicting opinions exist on this concept [16,17], some emphasizing lowering while some others an increase in blood pressure following smoking cessation, although is commonly accepted a significant reduction of cardiovascular events and mortality in this group of patients. Apart the blood pressure outcome, the evidence indicates an improvement in the arterial stiffness and decrease in adrenergic and sympathetic stimulation in habitual smokers who quit.

Conclusion

There is evidence that different opinions characterize the interpretation and outcome of major cardiovascular risk factors when one of them, including cigarette smoking, stops its effects. In addition, the degree of damage of heart and blood vessels could not be changed, unless forwhat's due to the activity of the specific factor removed if reversible alterations still co-exist [18]. However, whatever results are obtained, a reduction in rate of cardiovascular morbidity and mortality has been certainly seen. Therefore, efforts to help smokers to change their habit quitting smoking should be encouraged independently of the interaction with other risk factors.

References

1. Leone A (2011) Smoking and hypertension: independent or additive effects to determining vascular damage. Curr Vasc Pharmacol 9(5): 585-593.
2. Leone A (2014) Endothelial dysfunction in passive smokers. J Cardiol Curr Res 1(7): 00039.
3. Leone A (2012) Passive smoking, endothelial dysfunction and related markers in healthy individuals: An update. Curr Hypertens Rev 8(2): 141-150.
4. Critchley JA, Capewell S (2003) Mortality risk reduction associated with smoking cessation in patients with coronary heart disease: A systematic Review. JAMA 290(1): 86-97.
5. Dobson AJ, Alexander HM, Heller RF, Lloyd DM (1991) How soon after quitting smoking does risk of heart attack decline? J Clin Epidemiol 44(11): 1247-1253.
6. Ockene IS, Miller NH (1997) Cigarette Smoking, Cardiovascular Disease, and Stroke.A Statement for Healthcare Professionals From the American Heart Association. American Heart Association Task Force on Risk Reduction. Circulation 96(9): 3243-3247.
7. Sherman JJ (2005) The Impact of Smoking and Quitting Smoking on Patients With Diabetes. Diabetes Spectrum 18(4): 202-208.
8. Lino K, Iwase M, Tsutsu N, lida M (2004) Smoking cessation and glycaemic control in type 2 diabetic patients. Diabetes Obes Metab 6(3): 181-186.
9. Kwiterovich PO (1998) The anti atherogenic role of high-density lipoprotein cholesterol. Am J Cardiol 82(2): 13- 21.
10. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, et al. (2011) Effects of smoking and smoking cessation on lipids and lipoproteins: Outcome from a randomized clinical trial. Am Heart J 161(1): 145-151.
11. Flegal KM, Troiano RP, Pamuk ER, Kuczmarski RJ, Campbell SM (1995) The influence of smoking cessation on the prevalence of overweight in the United States. N Engl J Med 333(18): 1165-1170.
12. Dare S, Mackay DF, Pell JP (2015) Relationship between smoking and obesity: a cross-sectional study of 499,504 middle-aged adults in the UK general population. Plos One 10(4): e0123579.
13. Chiolero A, Faeh D, Paccaud F, Cornuz J (2008) Consequences of smoking for body weight, body fat distribution, and insulin resistance. Am J Clin Nutr 87(4): 801-809.
14. Vanni H, Kazeros A, Wang R, Harvey BG, Ferris B, et al. (2009) Cigarette smoking induces over expression of a fat depleting gene AZGP1 in the human. Chest 135(5): 1197-1208.
15. Harms M, Seale P (2013) Brown and beige fat: development, function, and therapeutic potential. Nature Medicine 19(10): 1252-1263.
16. Minami J, Ishimitsu T, Matsuoka H (1999) Effects of smoking cessation on blood pressure and heart rate variability in habitual smokers. Hypertension 33(1 Pt 2): 586-590.
17. Hee Lee D, Hwa Ha M, Rak Kim J, Jacobs DR (2001) Effects of smoking cessation on changes in blood pressure and incidence of hypertension. A 4-year follow-up study. Hypertension 37(2): 194-198.
18. Leone A (2003) Relationship between cigarette smoking and other coronary risk factors in atherosclerosis: risk of cardiovascular disease and preventive measures. Curr Pharm Des 9(29): 2417-2423.

Citation: Leone A (2015) How does to Quit Smoking Influence the Effects of Other Major Cardiovascular Risk Factors?. J Cardiol Curr Res 3(6): 00125. DOI: 10.15406/jccr.2015.03.00125