

Statistics and cardiovascular epidemiology: a different approach for the cardiovascular risk factors

Editorial

It is almost universally accepted that statistical estimate validates a large number of epidemiological, clinical and pathological findings in medical research, including cardiovascular disease due to the fact that in recent years a marked increase to apply statistical methodologies has been observed.

Strictly speaking, the term “statistics” indicates any function of sample data containing not yet defined parameters, which will be correctly evaluated when an appropriate statistical estimate will give them an evident significance identifying the specific role in the context of the studied hypothesis. Thus, it is worth noting that the choice and characteristics of the analyzed sample make interpreting results more reliable and, therefore, on line with the knowledge on a specific topic.

For instance, the large majority of observations associated with smoking exposure¹ as a risk factor for cardiovascular system come from epidemiological findings conducted on populations, different for demography, lifestyle, race, sex, socioeconomic conditions and several other characteristics, but clearly related to tobacco smoke.

Briefly, statistical methods allow establishing if those factors analyzed by epidemiological studies are related to a specific cardiovascular outcome as well as its rate. However, misinterpretation and/or even errors in the analysis of the results may be observed when applied methods of study are far to be exactly investigated.^{2,3}

From this premise, there is evidence that statistics could have some limits to analyze completely the characteristics of specific samples, although very important responses with regard to a cardiovascular event are usually provided and accepted.

Cardiovascular risk factors⁴⁻⁶ are a heterogeneous group because some exert their effects through a hemodynamic mechanism, and among these primarily arterial hypertension, some others like lipid alterations, diabetes mellitus and insulin resistance syndrome by metabolic properties, and, finally, others, mainly atherosclerosis and cigarette smoking, by a metabolic and hemodynamic mechanism associated.

When the adverse effects of the risk factors are examined, primarily the degree of harm of these factors on the studied population as well as the different responses displayed by different individuals should be carefully interpreted. Therefore, it is worth noting to establish whether a statistically evident association of the risk in the analyzed event exists and, in addition, its significance. Usually, the responses to this question derive from the observational findings conducted on larger samples of individuals. Thus, it is obvious that as largest is the analyzed sample as more correct is the statistical estimate.⁷ However, usually the observational studies involve populations affected by a single or, at a maximum, a limited number of risk factors associated, but not all the existing risk factors in their overall.^{8,9}

In an attempt to improve the statistical results meta-analysis findings have been proposed. Meta-analysis¹⁰ is a quantitative and epidemiological study designed to systematically assess previous research conclusions regarding similar patterns of study. Outcomes

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from a meta-analysis may include a more precise estimate of the effect of a risk factor for disease, or their outcomes, than any individual study contributing to the pooled analysis.

A large number of major cardiovascular risk factors have been better assessed by meta-analysis studies. Among these, it is worth noting that excellent papers¹¹⁻¹⁴ including several different reports have allowed cigarette smoking in both its form, active and passive smoking, and hypertension alone and/or associated with its complications to be demonstrated as factors of cardiovascular function impairment. However, meta-analysis show to be a promising but not a definite methodology able to absolutely solve statistical problems concerning cardiovascular epidemiology.

Following these concepts, a fundamental question arises: what is the true role of the statistics for a reliable estimate of cardiovascular epidemiology.

There is evidence that the epidemiological survey correlated to statistical methodology primarily contributes to the knowledge, and, consequently assessment of the distribution and rate of the cardiovascular disease, analyzing this outcome independently of the presence and/or absence of risk factors. Therefore, cardiovascular risk factors are investigated with regard to their role in developing the disease, but not own origin. However, clarifying this concept can have important implications in establishing both preventive measures and more appropriate therapy to reduce the incidence of a disease.

In addition, as can be seen, observational studies are the bases of the cardiovascular epidemiology. An observational study consists of collecting data belonging to the examined disease, whose analysis permits to identify those factors able to influence the onset and course of a disease by a statistical estimate. It is worth noting that a link of probability, but not certainty exists between characteristics of the disease and the examined parameter. Therefore, initially, the observations related to the disease provide the material to identify the role of a risk factor, but not vice versa. So, there is evidence that statistical estimate, strictly speaking, analyzes, at the beginning, the final results to identify those factors, which are responsible for the event. This fact should be a limit in the statistical interpretation since, usually, a mathematical approach, as should be considered applying

a statistical method, starts from the main factor to be analyzed in an attempt to identify its relationship with the event to be demonstrated.

Finally, there is evidence that both numbers and knowledge of cardiovascular risk factors as well as their interferences are continuously in progress, and this fact further demonstrates that statistical results are to be considered biased, although reliable with regard to the data showed.

Conclusion

In conclusion the results provided by the statistical estimate of cardiovascular risk factors should be taken carefully into account. In addition, although significant progresses of cardiovascular epidemiology on this topic cannot be denied, there is evidence that a conclusive validation of the statistical findings on cardiovascular risk factors certainly needs further studies, primarily starting from the question of what a cardiovascular risk factor really means.

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Conflicts of interest

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References

1. Leone A. Statistics for understanding damage. In: A Leone, editor. *Passive Smoking and Cardiovascular Pathology: Mechanisms and Physiopathological Bases of Damage*. 2nd ed. Nova Science Publishers, Inc., New York, USA. 2012. pp. 141–153.
2. White SJ. Statistical errors in papers in the British Journal of Psychiatry. *Br J Psychiatry*. 1979;135:336–342.
3. Harrell FE, Lee KL, Mark DB. Tutorial in Biostatistics multivariable prognostic models: Issues in developing models, evaluating assumption and adequacy, and measuring and reducing errors. *Stat Med*. 1996;15(4):361–387.
4. Leone A. Relationship between cigarette smoking and other coronary risk factors in atherosclerosis: risk of cardiovascular disease and preventive measures. *Curr Pharm Des*. 2003;9(29):2417–2423.
5. Leone A. Markers of atherosclerotic disease: What do they mean? Current opinion and future trends. *Curr Pharm Des*. 2015;22(1):7–17.
6. Leone A. Blood pressure outcome and quitting smoking: prevention or progression of cardiovascular damage? *JASH*. 2016;10 (Suppl 4):e47–e48.
7. Glantz SA. *Primer of Biostatistics*. Mc–Graw–Hill, New York, USA; 1992.
8. Akaike H. Information theory and extension of the maximum likelihood principles. In: S Kotz & NL Johnson, editors. *Break throughs in Statistics, Vol I, Foundations and Basic Theory*. *Springer series in Statistics*. 1992. p.199–213.
9. Keller A, Nesvizhskii AI, Kolker E, et al.. Empirical statistical model to estimate the accuracy of peptide identification made by MS/MS and database search. *Anal Chem*. 2002;74(20):5383–5392.
10. Haidich AB. Meta–analysis in medical research. *Hippokratia*. 2010;14(Suppl 1):29–37.
11. Wells AJ. Passive smoking as a cause of heart disease. *J Am Coll Cardiol*. 1994;24(2):546–554.
12. Law MR, Hackshaw AK. Environmental tobacco smoke. *Br Med Bull*. 1996;52(1):22–34.
13. Leone A, Landini L, Leone A. Epidemiology and costs of hypertension–related disorders. *Curr Pharm Des*. 2011;17(28):2955–2972.
14. Leone A, Landini L, Leone A. What is tobacco smoke? Sociocultural dimensions of the association with cardiovascular risk. *Curr Pharm Des*. 2010;16(23):2510–2517.