Cotinine and Blood Pressure Levels: Variability Omitted Once Again

To the Editor:

We read with interest the well-written article by Alshaarawy et al. that investigated the association of hypertension with second-hand smoke in 2889 never smokers from the National Health and Nutrition Examination Survey. Authors demonstrated that serum cotinine was positively associated with both systolic blood pressure (BP) and prevalence of hypertension. Although the determinant as depicted by serum cotinine levels—at first sight—seems appropriate to evaluate the exposure of participants to passive smoke, there are some obscured points that need to be clarified.

It is not reported whether cotinine assessment was performed on the same day of BP measurements. We should acknowledge that beyond BP variability evaluation completely lacking in the present study, there might also be variability in cotinine levels. This latter phenomenon could not be ruled out by the design of the present study, as exposure was not evaluated by structured questionnaires examining the periodicity of the exposure. The integration of passive smoking characteristics (ie, intensity, duration, environment of exposure) with cotinine levels would have provided a more integrated approach of passive smoking dynamics. For example, if the measurement of cotinine (plasma half-life of 19–24 hours) was performed on the first working day after a weekend in a subject exposed to secondhand smoke only at workplace, the measurement might be by far different of another performed in a midweek working day. Thus, authors’ statement that cotinine is an objective marker of secondhand smoke might be relevant for the clinical practice and research, only if combined with the investigation of individual passive smoking dynamics.

Additionally, BP measurement was performed in a single visit only, and represents a snapshot of the hemodynamic load of each participant. However, because BP measurement was performed 3 times during the same visit, it would be interesting to know the in-visit BP variability, given that subjects with high cotinine levels might demonstrate a higher variability with respect to those residing in the low cotinine levels group. Finally, passive smoking was previously found correlated with masked hypertension, and the possibility that the short or perhaps long wait for an appointment in a smoke-free clinic environment might be sufficient for the effects of passive smoking on BP to wear off, as suggested for the effects of active smoking.

In these lines, the association of a clinically guided serum cotinine measurement with BP levels could not be definitive without ambulatory BP evaluation.

Beyond the above considerations, the study by Alshaarawy et al. further confirms that passive smoking should be addressed in combination with other demographic and lifestyle characteristics during the usual clinic BP evaluation. Measurement of cotinine levels might not have clinical importance when measured separately of structured questionnaires evaluating smoking dynamics, but potentially could refine the clinical research in the field.

Disclosures

None.

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