Vascular Dysfunction Even After 20 Years in Children Exposed to Passive Smoking
Alarming Results and Need For Awareness

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Smoking, either active or passive, is associated with increased cardiovascular risk with passive smoking alone reported to have a 30% to 60% higher event rate. Endothelial dysfunction, as a biomarker of atherosclerotic progression, is known to be affected by smoking. Long-term effects of childhood exposure to passive smoking on endothelial dysfunction in later life remains poorly understood but, if addressed, this is an issue that would be important not only for medical reasons but also for social reasons such as public awareness and health recommendations.

In the current issue, Juonala et al examined whether parental smoking in childhood is predictive of disrupted endothelial function (endothelium-dependent vasodilation) as assessed using brachial flow-mediated dilatation (FMD) methods more than 20 years later when the subjects reached adulthood. To test this hypothesis, the authors used 2 large independent cohorts followed up since childhood with follow-up periods of respectively 27 and 20 years: the Cardiovascular Risk in Young Finns Study in Finland (95% of patients were from this study) and the Childhood Determinants of Adult Health study in Australia. Results suggested that parental smoking status in childhood is associated with endothelial dysfunction over 20 years later in adulthood even when corrected for other confounding factors.

The authors should first be commended for their valiant efforts in continuing to address this important topic and, in the present study, address the long-term effects of passive smoking on endothelial dysfunction. The authors had previously addressed this issue in a limited cross-sectional study as did others, however to definitively investigate this topic in the present study, the authors examined a total of 2171 mostly Caucasian patients from well-documented Finnish and Australian cohorts over lengthy follow-up periods exceeding 20 years to provide for an elegant study design. FMD capacity was reduced reflecting endothelial dysfunction among participants who had parents that smoked in youth compared to those whose parents did not, thus strongly suggesting that passive exposure to cigarette smoke among children might cause long-term impairment in endothelium-dependent vasodilation.

Passive smoking in childhood might therefore be associated with persistent or permanent impairment of endothelial dysfunction. As described by the authors in previous studies, endothelial dysfunction due to passive smoking is presently thought to be at least partially reversible after cessation of exposure (eg, approximately 50% recover in 6 years for active smoking). However, the follow-up period of greater than 20 years in the present study suggests otherwise that there might remain long-term effects on endothelial dysfunction at least for children exposed to passive smoking; thus, adults who were exposed to passive smoking as a child are at risk for cardiovascular disease. The next arising question that stems from the present study is to epidemiologically test whether exposure to passive smoking as a child is in fact associated with increased onset of cardiovascular disease in later life. The authors examined FMD as an acceptable “surrogate” biomarker of atherosclerosis but did not ascertain association with cardiovascular events likely due to the still young age of the patients (mean age at time of study was in the third decade). If exposure to smoking as a child indeed shows permanent effects on endothelial dysfunction with increased risk of cardiovascular disease in later life, this would have grave implications and impact on social behavior possibly through stricter rules not only for public smoking but also in the home or in the presence of children.

It should be noted that the present study had limitations. The authors used only questionnaires to assess parental smoking. The present study also did not measure exogenous nitric oxide-mediated vasodilatation to confirm that the decreased FMD capacity observed is a consequence of endothelial dysfunction and not due to smooth muscle dysfunction. Further, a longer follow-up period and ideally a prospective study will be necessary to ascertain whether said effects are truly permanent or only take longer to reverse. Other limitations as noted by the authors should be taken into account on interpretation of results.

Clinical assessment of endothelial function has become increasingly readily available with newer and improved technologies. Although FMD, which uses ultrasound measurements of (brachial) arterial flow-mediated vasodilatation, remains the benchmark method, it requires accurate technique and expertise for reproducible results. Alternative methods...
such as the FDA-approved finger-tip based EndoPAT® device are available, but regardless of method, assessment of endothelial function in the clinic is becoming readily feasible as a routine procedure to assess atherosclerotic progression and cardiovascular risk. Results of the present study draw attention to need for awareness and possible testing of endothelial function (perhaps even serially, maybe not every year but every few years) if exposed to passive smoking in childhood regardless of time from cessation.

References

Key Words: atherosclerosis ■ endothelial function ■ smoking
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