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Editorial

Is Childhood Exposure to Parental Smoking a Risk Factor for Future Cardiovascular Disease?

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The American Heart Association reported that exposure to second-hand tobacco smoke (SHS) in childhood, mainly SHS through parental smoking, was associated with future adverse cardiovascular health[1]. However, the patho-physiological mechanism by which SHS affects cardiovascular health is not fully understood.

In this issue of the Journal of Atherosclerosis and Thrombosis, Wang *et al.* used 31-year follow-up data for 2,511 participants from the Cardiovascular Risk in Young Finns Study, to explore the association between childhood exposure to parental smoking and low-grade inflammation in adulthood, which was defined as a high-sensitivity C-reactive protein (hsCRP) level of greater than 0.3 mg/dl[2]. Based on information obtained from a self-reported parental questionnaire, they defined passive smoking at baseline as one or both parents smoking. Compared with off-springs with non-smoking parents, those who had a smoking mother or father showed an increased risk of elevated hsCRP level (relative risk (RR) adjusted for cardiovascular risk factors and participants' own smoking status in childhood and adulthood = 1.3, with a 95% confidence interval (CI) of 1.0-1.8). In addition, the authors observed that the high hsCRP risk for mother-only smoking (RR = 2.4; 95% CI = 1.3-4.2) was greater than that for father-only smoking (RR = 1.6; 95% CI = 1.2-2.3) and for both-parent smoking (RR = 1.4; 95% CI = 0.9-2.0).

An elevated CRP level, which is a systemic inflammatory marker, is known to reflect the development of atherosclerotic lesions[3, 4]. The J-STARS trial showed that statin treatments led to a decrease in the hs-CRP level among patients with a history of non-cardiogenic ischemic stroke[5]. As CRP is a marker of atherosclerosis, an increase in the CRP level typically precedes the occurrence of cardiovascular disease[3, 6]. In addition, there is evidence that hsCRP was associated with the risk of chronic kidney disease[7]. Therefore, the results reported by Wang et al suggested that exposure to parental smoking in childhood has the potential to cause future cardiovascular disease via the development of atherosclerosis. However, their study had some limitations. First, the reason behind the stronger effect of mother-only smoking compared with both-parent smoking was unclear. Second, the authors did not obtain information on maternal smoking during pregnancy (exposure to SHS in utero). The statement from the American Heart Association noted that it was still unclear whether exposure to SHS in utero, or in childhood, or in both stages, was important for future cardiovascular health[1]. In addition, there were unmeasured factors that may have confounded the observed association; among them, the use of anti-inflammatory medication and exposure to air pollutants[8]. Notwithstanding these limitations, however, the study by Wang *et al.* provided additional evidence regarding future cardiovascular effects of childhood exposure to SHS.

To our knowledge, few prospective Japanese studies have examined the effect of exposure to SHS in childhood on cardiovascular sequence. The Longitudinal Survey of Babies in the 21st Century, conducted by the Japanese Ministry of Health, Labour and Welfare, registered approximately 47,000

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6-month-old children in 2001, and followed them until the age of 13. Compared with children not exposed to parental smoking when they were 6 months old, children whose mothers or fathers had smoked indoors had an elevated risk of overweightness/obesity from 2 to 13 years old[9]. Epidemiological evidence, in this regard, is expected for Japanese individuals whose cardiovascular risk profile differs from that of the Western population.

Conflict of Interest

None.

References

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