Dr Zingg and colleagues conducted a 3-year follow-up study of 260 smokers included in a smoking cessation trial to elucidate factors related to the progression of carotid atherosclerosis. The authors evaluated carotid atherosclerosis using the maximum value of the intima-media thickness (CIMT) and analyzed the association between traditional cardiovascular risk factors, high-sensitivity C-reactive protein (hs-CRP), smoking cessation, and CIMT progression using a general linear mixed model with repeated measures. As the main result, the systolic blood pressure was found to be a significant predictor of the progression of CIMT after 3 years, but no significant effect of smoking abstinence on CIMT progression was seen. I have some questions regarding their study.

First, the authors described the numbers of participants with 1-year relapse, 1-year abstinent/3-year relapse, and 3-year continuous abstinence as 208, 13, and 39, respectively. This means that 80% of the participants failed to achieve smoking cessation within 1 year. The authors understood that the statistical power was insufficient, and they did not conclude a no-effect of smoking abstinence on CIMT progression. As their study design is not suitable for evaluating the effect of smoking cessation on CIMT progression, the progress of carotid atherosclerosis in current smokers after 2–3 years could be simply evaluated using the 208 participants.

Second, the authors used the max value of CIMT for the evaluation of carotid atherosclerosis. I understand that the authors calculated the mean value of the maximal CIMT for the right and left carotid artery bifurcation according to the criteria of the US Taskforce. I also reported, using the same method, that aging, metabolic components, and the serum hs-CRP level were independent predictors of CIMT in healthy subjects. However, the mean value of the CIMT at three different positions of the carotid arteries on each side has also been used in combination with the number, magnitude, and hardness of the plaque in some epidemiological studies. The author cited the Rotterdam study, in which the carotid plaque was also used as an indicator of carotid atherosclerosis. As a further study, the investigation of other indicators of carotid atherosclerosis using ultrasound is also recommended.

Third, the author described a significant negative estimate value of the fasting plasma glucose at baseline for CIMT progression at a 3-year follow-up in Table 2. A higher fasting plasma glucose level at baseline suppresses CIMT progression, although the authors did not comment on this result. Smoking is harmful to vascular endothelial cells, and the combination of smoking and glucose intolerance has severe adverse effects on vascular damage. Naya et al. reported that smoking, body mass index, fasting serum insulin, systolic blood pressure, and fasting blood sugar were significant risk factors for early atherosclerosis development using the max CIMT. After substituting the max CIMT with the average CIMT, the significance of fasting blood sugar disappeared. Together, these results suggest that damage to the carotid artery as a result of the fasting plasma glucose is controversial. Anyway, the inverse relationship between CIMT progression at a 3-year follow-up and impaired fasting glucose should be explored in a future study.

References