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Response to Tomoyuki Kawada, M.D.: „Smoking, systolic blood pressure, fasting plasma glucose and progression of carotid atherosclerosis“

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We would like to thank Dr Tomoyuki Kawada for his thoughtful comments on our recent article<sup>1</sup>.

In the main analysis of the CAROSS trial<sup>2</sup>, a smoking cessation trial assessing ultrasonographic visualisation of carotid plaques as a potential motivational intervention, we found a smoking cessation rate of 20% after 1 year, in both the intervention and control arms<sup>2</sup>, which is in line with other smoking cessation trials<sup>3</sup>. Our recent article is a secondary analysis of the intervention group (n=267 at inclusion) with an extended follow-up of 3 years, including a carotid ultrasound.

We agree with Dr Kawada that our study does not have enough statistical power to demonstrate a definite influence of smoking cessation and/or relapse on carotid intima-media thickness (CIMT) progression. A higher number of participants as well as a longer follow-up would be needed to demonstrate its impact on CIMT. Therefore, restricting our analysis to data between the 1-year and 3-year time points (i.e. 2 years' worth of data, n=208) instead of the complete dataset over 3 years of follow-up (n=276) would have led to even less statistical power and relevant information.

We used carotid ultrasound in the CAROSS trial<sup>2</sup> to let smokers visualize the impact of cigarette smoking on their arteries. We did not aim at assessing the nature of carotid plaques but focused on their presence vs. absence, using the commonly accepted method of the Rotterdam Study<sup>4</sup> and recommended by the US Task Force<sup>5</sup>. Additionally, we measured CIMT at one specific site within the carotid arteries<sup>6</sup> to obtain a precise outcome for detection of more subtle changes during follow-up.

We found that baseline fasting blood glucose seemed to have a significant negative association with 3-year CIMT progression in our study ( $p=0.03$ ), but it was likely a chance finding, as we found no association with baseline and 1-year CIMT. Furthermore, no time-dependent follow-up of fasting blood glucose was implemented.

In summary, our analysis including data of the complete 3-year follow-up allowed us to identify increased blood pressure as a risk factor on CIMT progression in smokers.

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