Age-Related Increase in Femoral Intima-Media Thickness in Healthy Humans

To the Editor:

We read with interest the recent article by Kornet et al., who reported that wall shear stress of the common femoral artery did not change, whereas intima-media thickness (IMT) increased significantly with advancing age. Their findings in general are consistent with our contention that age-related increases in IMT of the common femoral artery observed in healthy humans may not be due to diffusive atherosclerosis but rather due to hypertrophy and/or hyperplasia of vascular smooth muscle cells in the medial layer of the arterial wall. Insight into this issue comes from studies in experimental animals. Arterial walls thicken with age, even in animals in which the incidence of atherosclerosis is very low.2 In other species that are known to develop atherosclerosis (eg, rabbits), the age-related arterial wall thickening is not associated with an increase in fibroatheromas or fatty streaks unless it is accompanied by experimentally induced hyperlipidemia.2,3 In this context, we recently reported that the IMT of the common femoral artery increases with age in a healthy, rigorously screened, nonsmoking population.4 Additionally, we reported that the IMT of the common femoral artery is strongly and positively related to arterial wall thickness of the common femoral artery with advancing age. Thus, the data reported by Kornet et al5 complement our data and further suggest that modest increases in arterial wall thickness of the common femoral artery with advancing age in healthy humans may not be due to diffusive atherosclerosis within the intimal layer but may reflect smooth muscle hypertrophy/hyperplasia within the medial layer of the arterial wall.

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Response

We thank Dr Tanaka and his colleagues for their valuable comments to our study published in Arteriosclerosis, Thrombosis, and Vascular Biology.1 We could not agree more that increases in intima-media thickness (IMT) are not necessarily related to atherosclerotic changes. The correlations found between increases in IMT and the occurrence of atherosclerosis, even when corrected for age, in a variety of epidemiological studies2,3 could be explained by 2 independent processes developing at the same rate. It cannot be excluded, however, that very pronounced increases in IMT may be indicative of atherosclerosis,4 but even in this situation, a causal relation has still to be established.

Arterial diameter increases with age to maintain such hemodynamic forces as wall shear stress and pulse pressure near baseline levels. We have always been in favor of the idea that the increase in arterial diameter, combined with the increase in mean arterial pressure and pulse pressure, lead to arterial wall thickening to maintain tensile stress of the arterial wall near baseline level with increasing age.5–7

In a recent multiparametric study, it was shown that not only diastolic arterial wall diameter, mean arterial pressure, and pulse pressure but also age was an independent variable in determining IMT of the common carotid artery.8 In this respect, the observations of Dinenno and colleagues9 that age-associated arterial wall thickening is related to enhanced sympathetic activity, causing hypertrophy and/or hyperplasia of smooth muscle cells, is of interest. This enhanced activity may indeed contribute to the increase in IMT with age, especially because this increase was found to be unrelated to wall shear stress.1 However, one should keep in mind that locally, IMT is dependent on wall shear stress, being larger in areas where this stress is lower.1 That elevated sympathetic activity may also influence arterial wall properties in the femoral artery is indicated by the observation that this artery shows a stiffer behavior with a full than with an empty bladder.10

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Arterioscler Thromb Vase Biol. 2000;20:2172
doi: 10.1161/01.ATV.20.9.2172
Arteriosclerosis, Thrombosis, and Vascular Biology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1079-5642. Online ISSN: 1524-4636

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://atvb.ahajournals.org/content/20/9/2172

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