Reply to “Letter to the editor: ‘Deconstructing the dogma of sympathetic restraint and its role in the cardiovascular response to exercise’”

Ilkka Heinonen,1,2,6 Maria Wendelin-Saarenhovi,3 Kimmo Kaskinoro,4 Juhani Knuuti,1 Mika Scheinin,5 and Kari K. Kalliokoski1

1Turku PET Centre, University of Turku and Turku University Hospital, Turku, Finland; 2Research Centre of Applied and Preventive Cardiovascular Medicine, University of Turku and Turku University Hospital, Turku, Finland; 3Department of Clinical Physiology and Nuclear Medicine, University of Turku and Turku University Hospital, Turku, Finland; 4Department of Anesthesiology, University of Turku and Turku University Hospital, Turku, Finland; 5Department of Pharmacology, Drug Development and Therapeutics, University of Turku and Turku University Hospital, Turku, Finland; and 6Division of Experimental Cardiology, Department of Cardiology, Thoraxcenter, Erasmus Medical Center, University MC Rotterdam, Rotterdam, The Netherlands

REPLY: We thank Dr. Tschakovsky (3) for his interest and enthusiasm regarding our recent study (1) dealing with sympathetic control of blood flow in different tissues of the leg in humans at rest and during exercise. Dr. Tschakovsky importantly emphasises that exercise-evoked vasodilatation cannot directly spread from the active quadriceps muscle group to the inactive hamstring muscles. This was not what we literally meant, and although according to our understanding ascending vasodilatation and its spread to inactive tissues could be contributing to some marginal extent, his concern in this regard was apparently mainly raised from our integration of our findings with those of Moore and colleagues (2), together suggesting that α-adrenergic-mediated sympathetic constraint is important for optimization of blood flow and oxygen extraction to the most metabolically active tissues of the limb. We also fully agree with Dr. Tschakovsky that investigating blood flow in active versus inactive muscle groups is not the same thing as investigating blood flow around active versus inactive muscle fibers within an exercising muscle. We tried to emphasise this notion already in the introduction of our paper (1).

Regarding the findings from α-adrenergic blockade during exercise, we indeed could not document any change in blood flow in the exercising muscle. Increased blood flow occurred in inactive muscles and other tissues, indeed very much challenging the common belief that there is sympathetic constraint in the vasculature of active muscles during exercise, as pointed out by Dr. Tschakovsky (3). Moreover, to the best of our knowledge, previous research in this area has relied on rather indirect assessments and estimations. Our study is one of the first to directly and quantitatively address this topic in humans. However, as also discussed in the original report (1), we made our observations during fairly low-intensity exercise, when also an only fairly small mass of active muscle was exercising. Thus, even if methodologically quite challenging, it still remains to be tested whether the findings are similar at higher exercise intensities and particularly when performing whole body exercise. In those conditions, it might well be that some sympathetic restraint is needed also in active muscle vasculature. At this moment, there is little human experimental evidence to support this idea, despite some indirect estimations. In all, we think that the insightful comments of Dr. Tschakovsky will help vascular and exercise physiologists to better understand sympathetic regulation of cardiovascular function during exercise in humans.

DISCLOSURES
No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS
I.H. drafted manuscript; I.H., M.W.-S., K.K., J.K., M.S., and K.K.K. edited and revised manuscript; I.H., M.W.-S., K.K., J.K., M.S., and K.K.K. approved final version of manuscript.

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