

LETTER TO THE EDITOR

DOES PHYSICAL EXERCISE IMPROVE ARTERIAL STRUCTURE AND FUNCTION IN SPINAL CORD-INJURED INDIVIDUALS?

Sir,

Jae et al. (1) reported recently that physically active spinal cord-injured (SCI) individuals have a preserved carotid artery function and structure compared with able-bodied controls. Therefore, the authors suggested a favourable effect of exercise on large elastic artery structure (intima-media thickness (IMT)) and function (arterial compliance). We believe that studies on vascular adaptations in SCI individuals potentially provide clinically as well as physiologically important information. Despite the fact that cardiovascular disease (CVD) is the leading cause of mortality in SCI individuals (2), traditional CVD risk factors cannot explain this strong association (3, 4). It was stated recently that changes in arterial function and structure may be related to CVD risk (5). Therefore, studies such as those performed by Jae et al. improve our understanding of the vascular adaptations after spinal cord injury that could eventually contribute to increased CVD risk. However, some methodological issues from Jae et al.'s study should be taken into consideration.

Our primary concern relates to the lack of a control group of normal active SCI individuals (i.e. who are not athletes or sedentary). Although a previous cross-sectional study reported higher carotid artery IMT in SCI individuals compared with able-bodied controls (6), a control group of SCI individuals examined under similar conditions and with similar data analysis techniques, is fundamental for the conclusions drawn by Jae et al. (1). Secondly, although all SCI individuals were wheelchair athletes, no information is provided about the frequency, duration and intensity of their physical activity, nor did the authors indicate for how long they were physically active and whether they participated in competitive or recreational sports. This information in SCI individuals, but also in controls, is essential for the interpretation of the results, as well as for future research on this topic. Finally, Jae et al. included only those SCI individuals with a spinal cord lesion below T6, which limits extrapolation to a larger group of SCI individuals. More importantly, the completeness of the spinal cord lesion varied from 17 SCI individuals with motor complete lesions (ASIA Impairment Scale (AIS) A/B (7)), 9 with motor incomplete lesions (AIS C/D) or even no loss of motor and sensory function at all (AIS E). Therefore, group heterogeneity in completeness of spinal cord lesion, which directly influences physical activity level, importantly impacted the overall findings.

Based on recent findings (3, 4), the markedly increased risk of developing CVD in SCI individuals (2) cannot be "simply" explained by the traditional CVD risk factors. The extreme physical inactivity below the spinal cord lesion may be re-

sponsible for adaptation in vascular function and structure. Another possibility relates to the loss of autonomic control of the cardiovascular system below the spinal cord lesion. The importance of the physiological relationships between the autonomic nervous system and vascular function, ageing and blood pressure should be considered. Taken together, despite a number of methodological issues, the results found by Jae et al. (1) in artery function and structure in physically active SCI individuals could potentially be relevant, as vascular adaptations in function and structure precede and may ultimately help us to understand the increased prevalence of CVD in SCI individuals.

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RESPONSE TO LETTER TO THE EDITOR BY JAN T. GROOTHUIS ET AL.
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INJURED INDIVIDUALS?

We thank Drs Groothuis, Hopman and Thijssen for their interest in our recently published paper (1). They are concerned about the lack of a control group comprising normal active individuals with spinal cord injury (SCI), the lack of information about participated exercise history, and group heterogeneity.

First, as Drs Groothuis et al. state, cardiovascular disease is much more prevalent in individuals with SCI than in able-bodied individuals (2, 3). It is also established in the general population that high levels of physical activity are associated with a low risk of subclinical atherosclerosis (4). However, these findings have been reported only in able-bodied persons and not in individuals with SCI. We thought that it was important to first compare active persons with SCI with an able-bodied control group (a control group with much more data available in the literature for comparison). However, we agree that our findings would have been strengthened by including a normally active group with SCI, and we believe that further studies are needed to clarify this association in both normally active and sedentary individuals with SCI. We have expressed this view in discussing the limitations in our study.

Secondly, the subjects with SCI had been participating in wheelchair track and field or wheelchair basketball on average 4–5 times per week and for at least 3 h per day for 8.3 (standard deviation (SD) 4.8) years.

Finally, we have acknowledged that the heterogeneity of our participants as a limitation of our study. This is a very valid point; however, participant heterogeneity is a common limitation in most research with individuals with SCI, as are low participant numbers. Considering our findings we do not consider that this heterogeneity detracts from our overall results, but in fact introduces a certain external validity to our findings. Active individuals with SCI, irrespective of injury

classification, did not differ from able-bodied individuals. We agree that autonomic dysfunction may be associated with atherosclerosis in individuals with SCI. We have reported previously that decreased autonomic function using heart rate recovery after exercise is associated with carotid atherosclerosis in able-bodied individuals (5). It is therefore possible that decreased autonomic function in persons with SCI would be associated with vascular dysfunction; however, this requires verification in future studies.

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