COMMENT

Running for health: how much running for how much health?

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ABSTRACT

Increasing physical activity has been shown to reduce physiological markers of cardiovascular disease, such as high blood pressure, vascular endothelial cell reactivity and arterial stiffness. In this issue of Clinical Science, Hägg and colleagues have chosen the spontaneously hypertensive rat model to investigate the effect of exercise on vascular function. They found that spontaneous running increased aortic compliance and antioxidant capacity with decreased oxidative stress in mesenteric arteries, presenting support for the cardiovascular protective effects of physical activity. Two important aspects of their study include the magnitude of the running stimulus and the choice of artery to evaluate.

Key words: cardiovascular disease, hypertensive rat, physical exercise, spontaneous running, vascular effect.
Abbreviation: SHR, spontaneously hypertensive rats.
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rats into high (13.4 km/day), moderate (7.2 km/day) and low (3.2 km/day) achievers [4]. The other question is whether SHR behave differently from other rat strains. This does not seem to be the case, as previous studies report running distances for SHR that are similar to normotensive rats (subjected to the same age and exposure conditions) [7]. Thus it seems that the SHR in the study by Hägg et al. [3] were running as one might expect a young rat to run on initial exposure to a voluntary running wheel. The intensity of running is important as previous studies have suggested that the blood pressure response of SHR to running may depend on running intensity [7].

How does this study by Hägg et al. translate to human health care delivery? They clearly demonstrated that voluntary running will improve several markers of vascular health in hypertensive rats, but the exercise stimulus has to be considered to be far greater in terms of time/day, times/week, and even exercise intensity than the typically recommended exercise programmes for humans (30 min of exercise three times/week, at a moderate intensity). The strength of the study by Hägg et al. [3] is the information on what vascular biochemical changes maybe associated with exercise, but it will remain to be determined if the same biochemical changes occur with a lower intensity training stimulus [8]. Perhaps we should consider ‘activating the wheel’ for selected time periods to more closely mimic human exercise patterns?

Another interesting issue presented by Hägg et al. [3] is the choice of arteries to evaluate. They chose the thoracic aorta as a conduit artery and the second- or third-order branch of the mesenteric artery as a resistance artery. A number of studies have suggested that central conduit arteries provide the best link between arterial stiffness and future cardiovascular events [9]. However, it is important to consider the impact of the exercise on the arteries being studied [10]. The aortic artery should have increased blood flow in response to exercise as it conducts blood to the exercising limbs. However, Laughlin et al. [11] found that mesenteric arteries decreased blood flow during exercise and, furthermore, exercise training did not significantly alter mesentery artery blood flow. This suggests that changes in the mesentery arteries reported by Hägg et al. [3] may well reflect non-specific responses to exercise training. This highlights the importance of considering the role the artery plays during exercise as well as the general function of artery when evaluating the impact of exercise on arterial structure and function.

REFERENCES


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