Racket sports as a model of studying vascular adaptations: a comeback after a quarter of a century

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TWENTY-FIVE YEARS AGO, SINOWAY et al. (13) ingeniously introduced the world of physiology to a simple yet unique model of investigating exercise-induced vascular adaptations. The model consisted of the examination of dominant and nondominant arms of tennis players to elucidate the localized impact of physical activity on the vasculature. This well-designed experimental paradigm provided evidence that vascular adaptations are preferentially confined to the trained limb. Specifically, the authors reported that forearm reactive hyperemia was 42% higher in the dominant vs. nondominant arm of tennis players (13). This experimental model has continued to be utilized sparingly to provide insight into exercise-induced vascular adaptations (3, 8).

In the present issue of the Journal of Applied Physiology, Rowley et al. (11) have employed this experimental paradigm to investigate the effect of chronic exercise training on not only local, but also systemic, adaptations in vascular structure. In particular, the authors compared diameter and intima media thickness (IMT) of the carotid, superficial femoral, and brachial arteries between elite squash players and healthy (untrained) controls. The authors found that all of the examined conduit arteries of squash players exhibited a significantly greater diameter than those of control subjects, with the exception of the carotid artery, which followed a similar trend. Furthermore, the diameter of the brachial artery was greater in the dominant vs. nondominant arm of tennis players (13). This experimental model has continued to be utilized sparingly to provide insight into exercise-induced vascular adaptations (3, 8).

In our opinion, the observation in Rowley et al. (11) that changes in arterial diameter occurred not only between dominant and nondominant arms of squash players, but also in comparisons that reflect systemic adaptations (Fig. 1), indicates that remodeling of arterial size cannot solely be attributed to localized effects. Likewise, the observation that changes in IMT are only present in comparisons that represent systemic vascular adaptations, the comparisons of brachial arteries in nondominant arms, as well as carotid arteries between squash players and controls, permits the evaluation of systemic (but not local) vascular adaptations.

Indeed, as illustrated in Fig. 1, all vessel comparisons of main interest in the present study can be organized into three categories, depending on whether they address local, systemic, or local + systemic adaptations to exercise training. We consider local adaptations to be isolated in vasculatures perfusing contracting muscles, whereas systemic adaptations occur in vasculatures feeding both contracting and noncontracting tissues. Based on these definitions, local and systemic adaptations can subsequently occur within a given vasculature. For instance, in the context of the present study, concurrent local and systemic adaptations are reflected by the comparison between dominant arms of squash players and control subjects. Conversely, while the comparison between dominant and nondominant arms of squash players allows for the examination of local (but not systemic) vascular adaptations, the comparisons of brachial arteries in nondominant arms, as well as carotid arteries between squash players and controls, permits the evaluation of systemic (but not local) vascular adaptations.

In the present study, the authors reported that forearm reactive hyperemia was 42% higher in the dominant vs. nondominant arm of tennis players. Thus this resourceful model employed by the authors theoretically allows one to decouple the relative contribution of local and systemic factors in the exercise-induced modulation of vascular structure.

The mechanisms underlying local adaptations to exercise training may include changes in the magnitude and/or profile of vascular shear stress, as well as external mechanical compressions induced by contracting muscles during the exercise bout. On the other hand, mechanisms contributing to systemic adaptations may include factors beyond shear stress, such as cyclic strain, transmural pressure, and/or circulating factors. Indeed, it has been reported that shear is elevated in vasculatures perfusing noncontracting tissues, including the carotid (6) and brachial (2, 12, 14) arteries during moderate-intensity

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lower limb exercise, and, therefore, vascular shear stress may be viewed as a systemic signal (4, 10). Given the existence of multiple and overlapping mechanisms potentially involved in local and systemic adaptations of exercise training, it becomes challenging when utilizing the current experimental design to dissect out the contribution of each mechanism. Future research should focus on elucidating and partitioning the fundamental mechanisms by which exercise training induces local and systemic vascular adaptations. This will inevitably require innovative approaches, including the use of complementary in vitro and in vivo animal and human experiments. Green and his colleagues have begun this journey of discovery and will undoubtedly continue down this path of exploration. We look forward to the upcoming breakthroughs in this field!

DISCLOSURES

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

REFERENCES


