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 squash players. In contrast, squash players demonstrated lower IMT across all three vasculatures compared with the control individuals, with no differences between contralateral limbs. The authors interpreted these findings as evidence that remodeling of arterial size induced by habitual exercise is driven by local factors (e.g., vascular shear stress), whereas alterations in wall thickness are influenced by systemic factors (e.g., arterial pressure) (11).

Over the past two decades, a plethora of research describing exercise-mediated structural alterations to the vasculature has been reported in the literature. Certainly, a number of studies have documented increased vessel diameter and reduction in wall thickness in conduit arteries perfusing contracting skeletal muscle (1, 5). For example, Dinanno et al. (1), using both a cross-sectional and longitudinal experimental design, demonstrated that regular walking/running increases resting femoral artery diameter and decreases IMT. In addition, changes in IMT have also been observed in vasculatures feeding tissues beyond the active muscle beds, including the carotid artery (7, 9, 15, 16). The current investigation by Rowely et al. (11) has eloquently synthesized these previous findings into a single study that takes advantage of the dominant vs. nondominant arm paradigm of racket sports. Thus this resourceful model employed by the authors theoretically allows one to uncouple the relative contribution of local and systemic factors in the exercise-induced modulation of vascular structure.

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 simultaneously 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 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 adaptations suggests that modulation of wall thickness is strictly dependent on systemic influences. However, it should be noted that the lack of difference in brachial artery IMT between dominant and nondominant arms of squash players may be ascribed to an inability of a vessel wall, with an already minimal wall thickness, to undergo further remodeling, resulting in an even smaller IMT. Therefore, the possibility that alterations in wall thickness may also be attributed to local effects in vasculatures of individuals who have not reached their lower limits (e.g., physically inactive subjects) cannot be excluded at this time.

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...
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**


