EDITORIAL FOCUS

Exercise is good for the heart, but the intensity matters

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IT IS WELL ESTABLISHED that exercise affords widespread and the most economic and effective health benefits for all individuals, especially those with noncommunicable chronic diseases, which are currently responsible for 65% of all deaths worldwide. Although exercise elicits coordinated multorgan responses, the heart is a primary responder to and effector of exercise. Exercise makes the heart strong. Intense exercise may increase cardiac output up to 5-fold and cardiac O2 consumption up to 10-fold to meet the higher demands of energy delivery (9). More importantly, moderate exercise reduces cardiovascular risk factors and confers direct protective effects on the cardiovascular system, including improving insulin sensitivity and enhancing survival signaling (16–18). Exercise is therefore known as an effective “polypill” for prevention and rehabilitation of cardiovascular disease, reducing cardiovascular morbidity and mortality (14). These benefits of exercise are usually associated with moderate intensity of exercise, as recommended in current European and American guidelines.

Long-term exercise largely increases cardiac metabolic flexibility and functional reserve along with a cardiac structural remodeling. In contrast to pathological remodeling, exercise induces adaptive growth that is characterized by normal cardiac function at rest and enhanced cardiac function during exercise (12). However, functional and biochemical cardiac abnormalities have also been observed during and after intense exercise (15). Such intense exercise may increase the incidence of cardiovascular events during physical activity, including acute myocardial infarction, arrhythmias, atrial fibrillation, coronary artery calcification, atherosclerosis, and sudden cardiac death (2). Individuals who are habitually inactive have the highest risk of adverse cardiovascular events during exercise, but even those who are habitually active have a higher incidence of adverse cardiovascular events during exercise than at rest (8, 13). A meta-analysis of 6 studies involving 655 athletes engaged in chronic exercise reported a 5-fold risk of atrial fibrillation compared with the sedentary population (1). These data suggest that the load (the intensity and duration) of exercise matters when evaluating the overall cardiovascular effects of exercise. However, the detailed cardiac remodeling, both structurally and functionally, in response to exercise load and its underlying mechanism are still unclear.

In a recent article in the American Journal of Physiology-Heart and Circulatory Physiology, Sanz-de la Garza et al. (11) provided direct evidence regarding the effects of training load on biventricular remodeling and function using a rat model of endurance training. They found that moderate training leads to a balanced biventricular dilation along with normal biventricular function, whereas intense training results in maladaptive right ventricular (RV) dilation along with functional impairment and myocardial fibrosis (Fig. 1). These results are consistent with the observation of RV structural and functional adaptations in some athletes (6, 10). These findings suggest that a low to moderate training load promotes RV remodeling within the physiological range and extreme training load aggravates RV remodeling away from a safety range. The authors suggest that this is a biphasic process and there is a cardiac loading “threshold” beyond which exercise is no longer good for the heart. It is understandable that this threshold varies among individuals. Appropriate and regular exercise training may elevate this threshold through physiological cardiac adaptive changes and thus make the heart stronger.

Another interesting finding of this study is that RV apical and basal segments exhibit different adaptations to varying intensities of exercise possibly because of the heterogeneous morphology of the RV. Exercise causes acute volume and pressure overload of the cardiac chambers and induces cardiac structural remodeling in response to chronic training, including increases in biventricular and biatrial cavity dimensions and volumes (6). Different from the left ventricle, the thin wall of the RV and its geometric shape make it more vulnerable to the deleterious consequences of pressure overload (7). More importantly, RV remodeling is highly associated with clinical outcomes (5). This makes it possible to use RV structural and functional adaptations as potential indexes for assessing the cardiac loading threshold of exercise. In fact, even though multiple modalities of assessment including echocardiography,
cardiac magnetic resonance imaging, genetic testing, and other approaches have been used in differentiation between physiological and pathological myocardial adaptations in response to exercise, practical evaluation of the threshold is still difficult. Given that the RV response to exercise is more sensitive and vulnerable, measurement of RV structural and functional adaptations may be a more efficient and sensitive approach for the assessment of the cardiac effects of exercise.

The limitations of this study, in addition to those mentioned by the authors (i.e., the difficulties in translating to humans hemodynamic results obtained from anesthetized young male rats), include that it did not monitor cardiac injury markers, such as plasma cardiac troponin I and creatine kinase-MB, during the exercise training. Since myocardial fibrosis, as found in the RV of intense exercise-trained rats in the present study, is usually the consequence of various cardiac injuries, plasma myocardial injury indexes may be potential candidates for biomarkers of exercise loading threshold. In addition, the exercise model of electric stimuli-forced treadmill running used in this study may cause stress in rats. It cannot be ignored that stress is associated with negative effects, such as more pronounced fibrosis, spontaneous ventricular arrhythmias, and sudden cardiac death, which might be avoided in voluntary wheel running (4). It would also be interesting to know whether the RV remodeling and dysfunction would be reversible after the cessation of endurance exercise training.

Collectively, Sanz-de la Garza et al. (11) demonstrated that endurance training affects biventricular remodeling and function in a load-dependent manner and intense training load leads to a maladaptive RV dilation and functional impairment, suggesting that special considerations are needed for athletes and exercise therapy practice in clinics. These findings provide new insights into the load-dependent effects of exercise on cardiac remodeling and function and provide a clue to the question of how exercise physiologists determine the optimal amount of exercise, especially for those diagnosed with cardiovascular disease, and minimize the adverse effects incurred by endurance exercise.

Of note, the well-accepted overall benefits of exercise should not be overwhelmed by the potential cardiac risks and damage of long-term overloading exercise training. Further investigation into the mechanisms responsible for cardiac remodeling and dysfunction, which would help to provide feasible assessment and supervision of exercise training, is merited. In addition, understanding the effects of the modality, duration, intensity, frequency, and timing of training on health-promoting benefits and the underlying mechanism of exercise will help exercise physiologists to determine the optimal individualized training protocols (3). There is rising interest in and endeavor to make personalized exercise prescriptions in recent years. More clinical studies are needed to further define the optimal training modality and load to maximize exercise-provided cardiovascular benefits to certain populations in health and disease.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

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