Endurance Exercise and Myocardial Fibrosis
Let Us Keep the Risk in Perspective

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If exercise could be packed in a pill, it would be the single most widely prescribed and beneficial medicine in the nation.

—Robert Butler, MD, Chair of the National Institute of Aging

As captured by Robert Butler’s quote, there is no disputing the wide-reaching health benefits of exercise training. Despite this, the health burden associated with the diseases of inactivity continues to rise. Each year millions of dollars are invested to promote physical activity, yet uptake remains relatively low as highlighted by data showing that only 20.2% of the US population meet the physical activity guidelines for aerobic and muscle strengthening exercise.1 Given these facts, it is imperative that any research that questions the benefits of exercise, or indeed suggests that exercise is deleterious to health, needs to be handled responsibly and placed within appropriate context.

See Article by Abdullah et al

A logical question following on from Butler’s analogy is that if exercise acts as a medication, is it possible to overdose? This idea has received significant scientific attention, especially in relation to the impact of prolonged exercise on the heart.2–4 A body of work has emerged showing that prolonged exercise, such as that completed by elite or competitive amateur endurance athletes, results in acute reductions in right and left ventricular functions,5–7 a transient release of cardiac biomarkers8 and potentially the development of an exercise-induced cardiomyopathy.9 Several studies have shown in a small number of elite or veteran athletes evidence of focal myocardial fibrosis.10,11 These authors suggest that this is evidence of the Phidippides Cardiomyopathy,9 with a cause related to the exceptionally high volume and intensity of exercise completed by these athletes. These studies have prompted scientific concern12,13 and attracted much media attention.

In this issue of Circulation: Cardiovascular Imaging, Abdullah et al14 report findings from a relatively large study examining the incidence of focal myocardial fibrosis using late gadolinium enhancement in 4 distinct groups of veterans with divergent levels of fitness and training histories. Importantly, the groups were well characterized in relation to their fitness and training history and were also highly screened so as to exclude the potential confounding influence of cardiovascular comorbidities (eg, hypertension and atherosclerosis). Across all 4 veteran groups, there was virtually no evidence of focal myocardial fibrosis, even in the high fit cohort who were extremely well trained (mean VO2peak of 39.8 mL/kg per minute) and had at least 20 years of competitive experience. The authors, therefore, conclude that increasing levels of lifelong fitness are not associated with focal myocardial fibrosis. These findings build on those recently published by Bohm et al15 similarly showing a lack of fibrosis in a younger cohort of elite athletes. Taken together, these findings should provide reassurance that high levels of competitive physical activity do not typically result in myocardial fibrosis. The current data are further supported by the relative low cardiac risk associated with competing in endurance exercise. Based on a sample population of 10.9 million runners in marathons and half-marathons, Kim et al16 have calculated a relative risk of 0.54 per 100,000 participants. If exercise-induced cardiac fibrosis, or the so-called Phidippides Cardiomyopathy, were a common consequence of completing endurance exercise, based on a sample population of 10.9 million runners in marathons and half-marathons, Kim et al16 have calculated a relative risk of 0.54 per 100,000 participants.

Although the data from Abdullah et al14 are reassuring to endurance athletes and their physicians, there are many important caveats. The high degree of clinical screening adopted by the authors, to specifically partition the effects of exercise from cardiovascular comorbidities, may have excluded veteran athletes who would have been identified by late gadolinium enhancement. It is also important to note that both the study by Abdullah et al14 and the previous publication by Bohm et al15 are cross-sectional in design and in real terms only assessed a small number of older individuals. Bohm et al15 examining elite masters athletes (mean age 29±8 years) and Abdullah et al14 examining veteran athletes, who may have come to exercise later in life and thus not have trained to a high level during their early developmental years. Accordingly, to definitively answer whether the late gadolinium enhancement observed in a small number of athletes is because of an exercise overdose, there is need for a large, well-designed prospective study conducted over the course of an elite athlete’s entire career and beyond. Importantly, any such study would need to consider and control for many confounding issues (eg, genetic substrate, systemic infections, and the use of regulated and unregulated drugs).

Until a carefully conducted longitudinal study is completed, it is impossible to establish whether extreme doses of intense exercise, in isolation, can cause myocardial fibrosis, or whether the phenomenon is explained by epigenetic interactions in the

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small number of athletes it seems to effect. Irrespective, the public health message related to physical activity should not be undermined by a phenomenon that while clinically relevant to a few, and scientifically interesting, seems not to be widespread among the majority of highly active individuals.

Disclosures

None.

References


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