Myocardial Adaptation in Response to Marathon Training
Do Short-Term Benefits Translate Into Long-Term Prognosis?

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Regular physical activity at low to moderate intensity confers many health benefits, such as reduced morbidity from cardiovascular and other chronic diseases as well as reduced cardiovascular and all-cause mortality. In the past decades, many mechanisms of benefit have been revealed down to a subcellular level. As little as 15 minutes of exercise daily may improve prognosis, and the greatest increase in health status is observed when moving from none or little to at least some physical activity.

In recent years, the dose–response relationship at the other end of the spectrum of physical activity has come into focus. It is well established that acute bouts of vigorous exercise may trigger cardiovascular events not only in an otherwise sedentary population but also in endurance athletes. The observation of increases in cardiac troponin in ≈50% of marathon runners has raised concerns of a link between such vigorous exercise and acute or chronic myocardial injury. However, the usually small and transient cardiac troponin elevation has led most researchers to conclude that it is likely of no prognostic relevance. This is supported by echocardiographic studies shortly before and after a marathon showing usually small and transient right or biventricular systolic and diastolic functional impairment, but no sustained short- or medium-term myocardial dysfunction. Furthermore, a 9-year follow-up of 114 young (age, 22±4 years) Olympic athletes with evidence of physiological cardiac remodeling (athlete’s heart) at baseline demonstrated no evidence of abnormalities in global or regional left ventricular structure and function and cardiovascular symptoms or events.

In this issue of Circulation: Cardiovascular Imaging, Zilinski et al addressed the potential cardiovascular health benefits of marathon training in 45 middle-aged apparently healthy male recreational (nonelite) athletes. During the structured 18-week marathon-training program, exercise volume doubled from 14 to 24 miles per week, and running speed was mainly performed in aerobic condition, which explains that training primarily improved aerobic capacity but not so much maximal exercise capacity. At the same time, left ventricular function and morphology changed in a direction typical for athlete’s heart with moderate dilatation of left and right ventricles as well as left atrial volume and a subsequent increase in myocardial mass. These adaptive changes were also accompanied by an improved risk factor profile. Furthermore, Zilinski et al distinguished more experienced (n=22; >5 marathons; mean, 14±11) from less experienced recreational runners (n=23; ≤5 marathons; mean, 1±1). With similar training mileage during the 6 months prior to enrollment (ie, 14±12 miles per week), both groups were comparable at baseline with respect to cardiorespiratory exercise testing data, all (but one) echocardiographic variables and risk factor values, possibly as a result of detraining.

There are several important findings in this. First, we learn that myocardial adaptation to volume overload by high-volume endurance training—and from previous studies also adaptation to detraining—is inducible and reversible within 4 to 6 months even in middle-aged nonelite athletes. Second, endurance training performed at aerobic intensity (ie, an intensity that leads to improved aerobic capacity with little effect on maximal exercise capacity) seems to induce myocardial remodeling typical of the athlete’s heart. Third, this study confirms that these adaptations can even be induced in persons with cardiovascular risk factors and can also reverse pathological changes of diastolic function. Fourth, an exercise volume threshold for cardiac adaptation of athlete’s heart may be postulated as remodeling was also observed in trained individuals.

Currently, the full phenotype of the athlete’s heart is expected in elite endurance athletes induced by long-term training over years, but less frequently if at all in recreational athletes. However, exercise training volumes (exercise time×exercise intensity) as needed for finishing a marathon in a decent time (eg, ≈4 hours) seem to match endurance training volumes of elite athletes in sports such as soccer in whom similar cardiac remodeling is frequently observed. The present study by Zilinski et al reveals that the current definition of elite athletes in sports cardiology (including those exercising competitively irrespective of training volume) is obviously too vague and that exercise capacity as well as exercise volume during the preceding 6 months should be included in preparticipation cardiovascular work-up even in recreational athletes.

The 16% prevalence of diastolic dysfunction in these middle-aged athletes at baseline may be related to their...
cardiovascular risk profile of hypertension in 29%, dyslipidemia in 54%, previous syncope in 29%, as well as medication in 31%. Reversing diastolic dysfunction has been previously observed over 3 months in a combined endurance and resistance exercise training 3 times per week in sedentary individuals and was confirmed in the current study. This shows that myocardial dysfunction can be reversed in physically active individuals when they double their training volume. This observation certainly deserves additional research to better understand the long-term health benefits of marathon training.

But do we have to continuously run 24 miles per week to sustain the health benefits of running? Will we already miss some benefit of exercise when reducing training effort to a not inconsiderable 14 miles per week for ≈2 months? And does marathon training with all its adaptive myocardial and nonmyocardial responses truly translate into measurable long-term cardiovascular health benefits?

Based on their findings of improved cardiovascular risk factors in addition to myocardial adaptation, Zilinski et al proposed marathon training as a putative preventive strategy. This is supported by improved long-term survival in Olympic athletes. Also, in a 12-month rehabilitation program in older patients with coronary artery disease, increasing energy expenditure gradually led to attenuation of coronary atherosclerotic lesions. The latter study shows the benefit of moderate exercise on prevalent cardiovascular disease, but not of high-volume marathon training. Olympic athletes may be a genetically privileged cohort, which may not be representative for recreational athletes.

In our view, a general recommendation of marathon running for preventing cardiovascular disease should be seen with caution because of potentially increased cardiovascular risk in marathon runners. Support for this comes from our own cohort of experienced recreational marathon runners: a much lower risk factor burden in marathon runners than in age-matched controls from the general population did not translate into less coronary artery disease, as measured by coronary artery calcium. When compared with age- and risk factor-matched controls, that is, to men that presumably have had a favorable risk factor profile throughout their lives, the coronary artery calcium burden was even higher in runners than controls. We also observed an unexpectedly higher prevalence of myocardial fibrosis in runners. Importantly, the number of marathon race participations was predictive of myocardial fibrosis, and runners with myocardial fibrosis had higher increases in cardiac troponin during a marathon than those without fibrosis, suggesting a causal relation between the frequency of marathon-induced myocardial injury and eventually detectable myocardial fibrosis. Finally, both coronary atherosclerosis and myocardial fibrosis were also predictors of long-term cardiac events. These findings may not all be causally attributable to marathon running. Indeed, event rates in our runners might have been much higher if the individuals had not engaged in marathon running. But these data cast a shadow of uncertainty on the health benefits of regular marathon training and competition, especially in persons with prevalent but unknown cardiovascular disease. An increased cardiovascular mortality with excessive exercise volume has recently been described for persons who survived myocardial infarction, and a U-shaped dose–response relationship of exercise is also supported by epidemiological data. In the Copenhagen City Heart Study, 1878 runners and >10000 controls were followed for >35 years. All-cause mortality in runners was 44% lower than in controls. The largest risk reduction was observed at 1 to 2.5 weekly hours of exercise. However, at training volumes >20 miles per week, all-cause mortality was comparable to that in controls. Similar observations were reported in a long-term study in >50000 Americans. Thus, marathon running may be less beneficial than cross-sectional or short-term risk factor analysis, echocardiography, or cardiorespiratory function assessment may suggest.

In summary, most studies agree with Zilinski et al that there is no need at present to discourage an otherwise healthy person from marathon training. However, this should not translate into public propagation of marathon training as a preventative strategy because this may distract from established cardiovascular prevention recommendations that focus on sedentary individuals to increase their activities to 30 minutes of exercise on most days of the week or 3×30 minutes of structured exercise. At the other end of the spectrum, it seems prudent that the cause–effect and dose–response relationships between high-volume endurance exercise and cardiovascular disease are studied further in different cohorts of athletes.

Finally, a word of gratitude: runners in this study were recruited in 2013 from the Dana-Farber (Cancer Institute) Marathon Challenge (DFMC), the largest charity group in the Boston marathon, running its 26th year in 2015. These runners experienced the devastating bombings during the 2013 Boston Marathon. The authors must be congratulated to have continued their research and all marathon researchers and readers alike deeply acknowledge the runners’ contribution to cancer research (via DFMC) and marathon research: keep it running (or at least walking)!

Disclosures
None.

References


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