CrossTalk opposing view: Prolonged intense exercise does not lead to cardiac damage

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Epidemiological evidence indicates that participation in competitive endurance sports decreases cardiac risk and increases life expectancy (Ruiz et al. 2011b). However, there is debate regarding whether strenuous endurance exercise for more than ∼1 h per day might induce deleterious effects on the healthy human heart (O’Keefe et al. 2012). In this cross-talk we highlight evidence that argues against this idea.

Is prolonged exercise training arrhythmogenic?

A primary observation that has been extrapolated to humans is that rats exposed to intense endurance exercise training via forced treadmill-running can develop arrhythmogenic areas of cardiac fibrosis (Benito et al. 2011). However, when the rat training programme is translated to the human lifespan, it appears that the exercise stimulus would equal training for several hours per day at ∼85–90% of maximum heart rate 5 days per week over a ∼10 year period (Ruiz et al. 2011a). These estimates are far higher than values that are sustained by elite endurance athletes. Importantly, it is not known if stress caused by the aversive stimulation (tail shock) used in the rat training programme might contribute to the exercise-associated findings. By contrast, studies with dogs show enhanced cardiac electrical stability and protection against ventricular fibrillation (Billman, 2009). While the findings by Benito et al. have added further controversy to the old debate (George et al. 2011), they may not be easily translatable to humans.

Marathon and cardiac arrest

One way to test the idea that either prolonged intense training or acute exercise is deleterious to the human heart is to evaluate catastrophic cardiac events that occur during the marathon (∼42 km). The growing popularity of this event (Lamppa, 2013) has been accompanied by reports of race-related cardiac arrests. Kim et al. (2012) estimated the incidence of sudden death in US marathons during 2000–2010, and showed that of 10.9 million runners, only 59 (51 men) had cardiac arrest. Additionally, underlying cardiovascular disease accounted for the majority of events and middle-aged men were the high-risk group, with hypertrophic cardiomyopathy being the major predisposing cause (Kim et al. 2012).

The high-risk group had minimal long-distance running experience and fatal events typically occurred during the last quarter of the race after about three or more hours of exercise. This high-risk group included inexperienced runners with limited exercise training histories, who followed a relatively low-intensity training regime focused on finishing the marathon in ∼4–5 h. Under these conditions, people with occult cardiovascular disease can remain asymptomatic. However, during the race, emotional stress and dehydration might raise heart rate well above normal training conditions for a longer time, leading to an increased risk. Exercise-induced increases in thrombotic factors could also lead to acute coronary thrombosis in runners with occult coronary artery disease (Albano et al. 2012). If lifelong, prolonged, intense training were responsible for these catastrophic events then a greater fraction should have been seen in individuals with longer and more intense training histories.

Post-race cardiac alterations: clinically relevant?

Although there is controversy (Lucia et al. 1999b), some data suggest post-race alterations in cardiac function. Neilan et al. (2006b) observed echocardiographic abnormalities including altered diastolic filling, increased pulmonary pressures and right-ventricular dimensions, and decreased right-ventricular systolic function shortly after completion of the Boston Marathon, with right-ventricular diastolic alterations persisting 3–4 weeks post-race (Neilan et al. 2006a). These
The marathoning heart: the trigger or the substrate for arrhythmias?

The main issue is whether arrhythmias seen in individuals who participate in endurance sports are due to undiagnosed underlying cardiac arrhythmogenic diseases, with long-term exercise being a triggering factor, or whether such exercise can actually be a primary cause of arrhythmia susceptibility. In men, lifelong endurance exercise training can be associated with increased risk of atrial fibrillation (AF), particularly lone AF (Karjalainen et al. 1998; Mont et al. 2002; Elosua et al. 2006; Molina et al. 2008).

A large prospective cohort study showed that frequency (days per week) of vigorous exercise was associated with an increased (+20%) risk of developing AF in young men and joggers, yet this risk disappeared in men aged ≥50 years (Aizer et al. 2009). There are several mechanisms through which frequent endurance exercise might influence AF risk, including left-atrial enlargement, left-ventricular hypertrophy or dilatation, and an increase in parasympathetic tone, which predisposes normal hearts to AF (Northcote et al. 1990; Hood & Northcote, 1999; Kasikcioglu et al. 2006).

Summary and perspective

Currently there is no strong evidence to support the idea that prolonged intense endurance exercise training increases permanent or pathological ventricular damage in humans. The protective effects of regular endurance exercise against cardiovascular disease and premature death would seem to offset any transient cardiac dysfunction that can be sometimes observed in less prepared middle-aged male runners after events such as the marathon. However, we believe this group deserves special attention, and the guidelines about medical examinations and exercise testing for individuals in this demographic group should be emphasized. A subject of debate that merits additional research is the incidence of arrhythmias, particularly AF, associated with long-term endurance exercise. Further research might elucidate whether ECG abnormalities in recreational marathon runners are the substrate for clinical events, or represent non-pathological adaptations to training. We also emphasize that the animal studies suggestive on long-term pathophysiological adaptations to prolonged intense endurance exercise training in some species are provocative, but their direct applicability to humans is questionable. Finally, in a world full of coronary artery disease let us not forget that ultra-marathoners with long histories of training have large coronary arteries with impressive vasodilator reserve (Haskell et al. 1993).

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References


Additional information

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