Response to the Letters Regarding Article, “Can Intensive Exercise Harm the Heart? The Benefits of Competitive Endurance Training for Cardiovascular Structure and Function”

I thank Drs. Scott and Haykowsky for pointing out the important consideration of intensity in the overall characterization of a training stimulus. Of course, because the focus of this Controversies in Cardiology was on competitive athletes,1 it is important to note that virtually all athletes perform high-intensity exercise as part of their training regimens. Indeed, athletes could hardly be considered competitive if they did not perform high-intensity exercise. Most athletes engage in a periodized program of varied frequencies, durations, intensities (including recovery), and modes, which also vary day by day (microcycles), week by week (mesocycles), and month by month (macrocycles) as they train for a specific competition. I agree completely with Scott and Haykowsky that different types of exercise stimuli may evoke different adaptive responses and that these considerations are especially important for physicians who try to guide their patients on what kind of exercise is best for their overall health (as well as training goals). I am less convinced that athletes are especially more susceptible to injury from a given exercise training session. For example, in their cited study, it is not clear to me that the small differences between athletes and control subjects were meaningful physiologically even if they were demonstrable statistically. In addition, much of the literature in this field suggests that reductions in ventricular function are more likely to occur in untrained individuals than in trained individuals, particularly during shorter-duration (<3 hours) efforts.2 Clearly, extraordinary exercise efforts can cause fatigue of cardiac muscle in athletes and nonathletes alike. However, it is also clear that continued training at these levels, sufficient to sustain multiple Olympic competitions over decades, as shown by Pelliccia et al1 and cited in my article,1 does not lead to deterioration of cardiac structure or function.

I also agree with Dr. Möhlenkamp and colleagues that high-volume and -intensity training in the setting of pre-existing coronary artery disease or even remote and undiagnosed myocarditis can lead to adverse outcomes (as noted in my original article1), and I appreciate their emphasis that their cohort was a relatively high-risk one. Indeed, ≈50% of these runners were smokers,4 which is likely different from American marathoners. Moreover, nearly half of runners in whom late gadolinium enhancement was observed (5 of 12) had it in a distribution typical of patients with coronary artery disease.5 If I recalculate their statistics ($\chi^2$) using only those runners with a noncoronary pattern of late gadolinium enhancement (7 of 102 versus 2 of 102 control subjects), the probability that such a distribution could be observed simply by random chance sampling of the population more than doubles, from an already “nonsignificant” 0.072009;251:50–57. doi: 10.1148/radiol.2511081118.

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