



EDITORIAL COMMENT

Exercise and sports revisited: Is too much exercise bad for your heart?

Exercício e desporto revisitados: o excesso de exercício é mau para o coração?

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The study by Pereira et al. published in this issue of the journal¹ raises pertinent questions about the interaction between physical activity, plaque morphology, and risk of cardiovascular disease.

The possible relationship between physical activity and the development of coronary artery disease (CAD) has been neglected for many years. However, the topic of coronary atherosclerosis in athletes is today very important for two reasons: first, because the number of high-volume training athletes is increasing; second, because CAD is the most prevalent cause of exercise-related cardiac events in individuals over 35 years.²

After the landmark study of Mohlenkamp et al.³ demonstrating that in athletes the use of traditional calculators underestimates cardiovascular risk, other authors have included the coronary calcium score (CCS) to optimize the prediction of cardiovascular risk in this population,⁴ as well as to predict it based on the type, intensity, and duration of exercise.^{5,6}

Based on these studies, an important question has raised – should the CCS be used in an asymptomatic and highly

trained population to better stratify their cardiovascular risk?

If, on the one hand, CCS is increased in middle-aged athletes who perform greater volume of physical load per week, on the other hand, the prognostic studies carried out with this parameter are derived from the general population, that do not present high levels of physical activity.⁷ This doubt remains since it is not known whether higher CCS in populations with high-training volumes is related to a greater number of cardiovascular events and mortality. The classic relation between the CCS score and coronary events seen in the general population may not be true in high exercise volume athletes in whom the atherosclerotic plaque is a consequence of increased thrombogenicity, sympathetic activation, electrolyte imbalance, hyperdynamic circulation with “kinking” and spasm, shear stress and imbalance between the antioxidant and oxidative effects of exercise.⁸ This protective concept was supported by older studies that described the plaques of these athletes as being more calcified and therefore more stable, not prone to rupture, which could give them a lower risk of events.⁵ In opposition, more recent research has shown that the level of physical exercise increased both calcified and non-calcified plaques in athletes.^{9,10}

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The work of Pereira et al. suggests that, in athletes, specific cardiovascular risk assessment strategies must be developed, because of a synergistic atherosclerotic effect between classic atherosclerotic risk factors and oxidative stress derived from high levels of exercise. This is in line with a recent multicenter prospective cohort study (Master@Heart), in which lifelong athletes had a higher atherosclerotic burden, more significant proximal CAD, more unstable plaques and in which the authors suggest a possible reverse J-shaped dose–response relationship between resistance training and coronary atherosclerosis.⁹

However, in athletes with low to moderate cardiovascular risk this association no longer exists, and higher volume training may even be protective of greater atherosclerotic burden. The group that presents a proportion of individuals with a CAC score >100 and a segment involvement score (SIS) >5 was the one with the least training volume, in line with previous studies that indicate that moderate to high regular physical exercise results in a lower number of total and non-calcified plaques and fewer high-risk plaques.⁹ Of course, this will be influenced by the fact that these athletes, who exercised less, had a higher prevalence of at least one cardiovascular risk factor, showing a higher BMI (body mass index), even in the absence of a higher SCORE2.

It is important to highlight the fact that the presence of dyslipidemia and active or previous smoking in this population is quite high, especially in people who exercise and want to be healthier.¹¹ Remarkably, the average value of LDL (low-density lipoproteins) cholesterol (considered the main cardiovascular risk factor) was, in this population, above than the desired for individuals at low to moderate risk and comparable to recent studies in athletes.⁹ This raises an important point and a growing concern that other lifestyle behaviors (such as a healthy diet), should be adopted by these individuals,

When appropriate, statins (and/or other ant lipidic drugs) should be prescribed to keep LDL cholesterol at therapeutic targets for the inherent cardiovascular risk. In fact, we can expect that if these athletes do not lower their cholesterol, in less than 10 years, there will be a high percentage of individuals with a calcium score >100, with a greater atherosclerotic burden and potentially with more unstable coronary disease.^{9,12} Even so, vigorous exercise can have the same effects as statins on the composition of the atheroma plaque, leading to its progressive calcification, decreasing its size and minimizing cardiovascular risk.¹³

These results cannot be extrapolated to other subgroups, such as female athletes (as the participants were all male), knowing that there is much less evidence of coronary disease in female athletes compared to their male counterparts. However, they appear to have, at most, a number and type of atherosclerotic plaques similar to controls¹⁴ or even less atherosclerotic burden.¹⁵ Additionally, the results cannot be extrapolated to patients with known cardiovascular disease (for example post-acute myocardial infarction), diabetic or chronic kidney disease patients, which were exclusion criteria from the study.

The elite exercise paradox will have to be unraveled in the future and the dose-response relationship between physical activity and cardiovascular health still needs better understanding.

Despite the favorable data linking the benefit effect of physical activity to all-cause mortality regardless of the calcium score,^{16,17} we do not know yet whether this burden of coronary atherosclerosis translates into a greater risk of cardiovascular events, specifically an increase in cardiovascular mortality in veteran athletes. More prospective studies with clinical endpoints are needed to evaluate these data. It will therefore be interesting to monitor follow-up data from a unique investigation into this topic – the Master@Heart study.

Conflicts of interest

The authors have no conflicts of interest to declare.

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