Acute effects of tea and dark chocolate. Most of these studies determined antioxidative capacities. We therefore decided to measure flow-mediated dilation as a sensitive marker of endothelial function. We do not agree with the authors that the sample size in our study was rather small. We obtained highly significant results after measurement of FMD in 16 volunteers. Comparable studies measuring FMD in humans after consumption of beverages comprised a sample size similar to our study and yielded statistically significant results: e.g. after consumption of red wine and black tea.

Nevertheless, we concur with the authors that a single study cannot replace larger studies involving a comprehensive cross-section of the population. The aim of our study was to evaluate the immediate impact that addition of milk to tea has on a single, cardiovascular relevant parameter, the endothelial function. The rationale for drinking tea in a lab setting was that only under these conditions could the influence of other beverages and food be controlled for. This setting accordingly allowed us to closely study the interaction of milk with tea. On the basis of our results and for the purposes of this study, we concur with the authors that the sample size in our study is statistically significant: e.g. after consumption of red wine and black tea.

Diabetic criteria for congenital long QT syndrome in the era of molecular genetics: do we need a scoring system?

We have read with great satisfaction that Rossenbacker and Priori, in their editorial to our article (Diagnostic criteria for congenital long QT syndrome in the era of molecular genetics: do we need a scoring system?), have provided supportive evidence for our conclusion that presently used diagnostic criteria for inherited long QT syndrome (LQTS) have insufficient diagnostic power. Unfortunately, we must rectify an interpretation of our work by Rossenbacker and Priori, which is clearly erroneous. Rossenbacker and Priori state that we propose in our article that when molecular diagnosis is available in a family, 'it would be worthwhile to use clinical criteria to select individuals suitable for molecular screening'. These authors provide reasons why such a strategy should not be followed. Instead, genetic testing should be conducted in all relatives, regardless of penetrance. We fully agree with this latter strategy. Rossenbacker and Priori state that we do not claim that our conclusions are universally valid for all physiological outcomes.

References


authors conclude that endurance exercise may act as a promotor for RV changes and a resultant trigger for VA. In part, we agree with the authors’ findings; however, we have some comments regarding the postulated cause and effect relationship between RV dysfunction and VA.

An increased prevalence of arrhythmias in endurance-trained athletes has been noted previously, predominantly in veteran athletes. Several forms of idiopathic VA have been identified in athletes, which, by definition, originate in hearts without structural abnormalities. Unpublished observations from our laboratory at the CRY Centre for Sport Cardiology support these findings. The differentiation of pathological VA and benign VA originating in the right ventricle is important clinically when discussing prognosis and management options. This is of particular importance in the differentiation of right ventricular outflow tract-ventricular tachycardia (RVOT-VT) and arrhythmogenic right ventricular cardiomyopathy (ARVC) given the association of the latter with sudden death in athletes. We concur with hypothesis 1 proposed by Ector et al. in the difficulties associated with the differentiation of RVOT-VT and ARVC, particularly when the early concealed phase of ARVC is considered. A recent case in our laboratory highlighted this difficulty; however, the EP study was instrumental in the differential diagnosis, a finding that concurs with previous work in the field.

The mechanism(s) underlying VA in endurance-trained athletes is unclear. Our group, and others, has demonstrated an elevation in cardiac troponins following endurance exercise and a concomitant but unrelated reduction in cardiac function. Although the presence of cardiac troponins is pathognomonic of cardiac damage, the rapid return of cardiac troponins to baseline (<24 h) has led to the suggestion that this phenomenon is physiological in nature. The impact of multiple episodes of prolonged exercise as experienced by highly trained and life-long endurance athletes however is not fully understood. Previous studies have reported myocardial fibrosis and heart failure in highly trained athletes. Our group recently investigated a case of sudden cardiac death in an experienced veteran Marathon runner. Post-mortem examination revealed widespread idiopathic interstitial myocardial fibrosis. In the absence of any other cause, we postulate that life-long, repetitive bouts of arduous physical activity may result in fibrous replacement of the myocardium, resulting in a pathological substrate for the propagation of arrhythmias. This proposed mechanism is supported in studies in non-ischaemic cardiomyopathy where myocardial damage leading to fibrosis has been implicated in myocardial re-entry leading to VA. Furthermore, previous studies have supported the view that conduction system abnormalities and arrhythmias in athletes may be associated with myocardial damage. Although these findings may be somewhat supportive of hypothesis 2 proposed by Ector et al., we feel some care is warranted in the interpretation of the observed RV dysfunction. Long-standing VA can result in ventricular hypokinesia, leading to dysfunction. Following cardioversion through pharmacological or non-pharmacological means, a normal function is often restored. Ector et al. did not report the intervention employed or outcome in those athletes with VA, and this may be informative for the journal readership.

In contrast to the conclusion of the authors, we propose a third hypothesis that the burden of long-standing sustained or non-sustained VA may lead to RV dysfunction. Furthermore, the mechanism(s) underlying the increased arrhythmia prevalence observed in endurance-trained athletes remains elusive. Future studies should aim to identify the potential mechanism(s), including interstitial myocardial fibrosis.

References
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