citations from the press were original quotations from us.

On the basis of the results in the literature, and as quoted in our article as well as by the authors, there are conflicting data as to whether addition of milk has an adverse effect on the beneficial effects of tea and dark chocolate.\(^1\)\(^2\)\(^3\) Most of these studies determined antioxidative capacities. We therefore decided to measure flow-mediated dilatation as a sensitive marker of endothelial function.\(^6\) 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\(^6\) and black tea.\(^6\)

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 effects measured, addition of milk blunts the beneficial effects that tea has on its own, in vitro and in vivo, on endothelial function. Certainly, future trials are necessary to confirm these findings, and we do not claim that our conclusions are universally valid for all physiological outcomes.

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References

We have read with great satisfaction that Rossenbacker and Priori,\(^1\) 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?’),\(^2\) 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 phenotypic characteristics. We must emphasize here that we fully agree with this latter strategy. Accordingly, we have discussed this issue at length in our manuscript, e.g. in Abstract and Discussion. Our Discussion states: ‘...finding a QTc duration in the upper range of normal in a relative of a LQTS proband should not provide the false reassurance that this individual will not carry LQTS. Previous studies also indicated reduced penetrance in LQTS (i.e. normal QTc values in mutation carriers). These observations clearly impart added importance to molecular genetic investigation, as DNA testing should be ordered in relatives of an LQTS proband, even if their QTc lie within the normal range’. The last sentence of our Abstract reads: ‘In genotyped families, genetic testing is the preferred diagnostic test’.

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

We have read with great interest the article by Ector *et al.\(^1\)* reporting ventricular arrhythmias (VA) in highly trained endurance athletes, originating from a mild right ventricular (RV) dysfunction. Of note, the