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doi:10.1093/eurheartj/ehm109

Online publish-ahead-of-print 5 May 2007

Tea without milk: lifestyle advice based on a small lab study

In their article, Lorenz *et al.* report results of experiments in 16 healthy post-menopausal women and of additional *in vitro* experiments in rat tissue.[1] They infer that milk may counteract the known favourable effects of tea on vascular function. Further, they speculate that this finding may explain the lack of effect of tea on cardiovascular outcomes found in a previous population-based cohort study conducted in Caerphilly/Wales.[2] In this study, almost all participants added milk to their tea, and consequently, there was no control group of 'purist' tea drinkers.

The European Society of Cardiology accompanied the publication of this study with a press release that amplified the authors' conclusions.[3] As milk is in many tea-cups worldwide, it is not surprising that the 'bad news for tea-drinking nations such as the British' has spread rapidly and widely. Most related headlines left no room for doubt, e.g. 'Milk cancels health benefit of drinking tea: study'[4] or 'Tea is good for you, but skip the milk'.[5] Only few agencies took the time to produce a more balanced news piece by including additional information from an independent expert[6] or additional literature.[7] A simple explanation and some good advice sell better than the complexities of the real world. Also, it is well known that bad news is more likely to be published in newspapers than good news.[8]

Although the authors call for caution in the design of studies, they were less cautious when drawing inference from their data. The latter are derived from measurements in a few selected volunteers who drank tea in a laboratory and were not representative for any part of the population. Beyond the contentious question whether flavonoids in black tea are absorbed by milk proteins or not,[9,10] evidence from relevant population-based studies is not yet in sight. Instead of measuring vasorelaxation, such studies would certainly choose outcomes that are more meaningful to populations, for instance, incidence of ischaemic heart disease.[2] The present study could well serve as a starting point for the planning of such studies, but cannot replace them. It is hazardous to derive lifestyle advice from a single lab study. Such advice is likely to have a short shelf life when additional evidence is taken into account or new research data accumulate.

As doctors, we would not prescribe a new drug to patients if it was studied only in one small study. In analogy, milk abstinence should not be recommended to tea drinkers on the basis of evidence of similar strength. If science journalists carry news from the bench to excess, scientists should object. Publicity may help them along for a while, but they risk to be no longer heard by the public in the long term. Clearly, this story has not helped the case of public health. As long as the reported results are not confirmed in a fair number of humans who drink their tea outside the lab setting, we will continue to add milk to ours.

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doi:10.1093/eurheartj/ehm108

Online publish-ahead-of-print 5 May 2007

Tea without milk: lifestyle advice based on a small lab study: reply

We thank von Elm and Antes for their comments on our article 'Addition of milk prevents vascular protective effects of tea'.

In the first section of the letter, the authors critically comment on the newspaper coverage of our study, particularly on one-sided and unbalanced reporting in the press. They also note that only a few agencies took the time to give a more complex view and that bad news sells better than good news. We agree with the authors that any one-sided coverage of a story—whatever the topic might be—should be avoided and does not necessarily lead to improved information of the public. However, we would like to point out to the authors that we are not the writers of those news articles and hence cannot be held responsible for one-sided press coverage. This criticism, although correct, would be better addressed to the journalists composing the headlines and press articles. We also would like to state that not all the

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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.[1–3] Most of these studies determined antioxidative capacities. We therefore decided to measure flow-mediated dilation as a sensitive marker of endothelial function.[4] 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[5] 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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doi:10.1093/eurheartj/ehm118

Online publish-ahead-of-print 5 May 2007

Diagnostic 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,[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 induvidual will not carry LQTS. Previous studies also indicted 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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doi:10.1093/eurheartj/ehm121

Online publish-ahead-of-print 4 May 2007

Arrhythmias and the athlete: mechanisms and clinical significance

We 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

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