In Reply—Caffeine Effects on Arterial Stiffness: To Drink or Not to Drink

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excreted in urine. However, the concentration of these metabolites in urine is subjected to many confounding factors. For example, it is reported that the rate of caffeine metabolism is variable, with caffeine consumers being slow, intermediate, or fast metabolizers. This is highly attributable to genetic factors related to the CYP1A2 phenotype and liver function in general, and it is important to be taken into account when assessing caffeine health effects using urinary caffeine metabolites. Also, variability in excretion highly depends on the timing of the last intake of caffeine and previous habitual consumption. Ponte et al assessed the timing of the last intake of a caffeinated beverage, but it was not very clear whether this parameter was used as a confounding factor in the analysis. Also, habitual caffeine consumption should be taken into account. Previous studies have shown that people are more sensitive to the diuretic effect of caffeine after a short period of abstinence from caffeinated beverages compared with regular consumers; this results in an increased rate of caffeine metabolism and consequently to more accurately assess their association with PP and PWV. In our view and on the basis of available data, we have a lot more to learn concerning the effects of caffeine intake on arterial wall properties and blood pressure.

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To the Editor: We thank Karatzis et al for their letter and the issues they raised. The authors stressed the fact that the rate of caffeine metabolism is variable and that it is highly attributable to genetic factors related to the CYP1A2 phenotype and liver function in general. We cannot agree more and have actually acknowledged this in the Limitation section of our article as follows: “although we considered major factors, the biological half-life of caffeine is highly variable among individuals (2–10 hours) and is influenced by several genetic and nongenetic determinants (eg, liver function) that we could not account for.” It is true that information on habitual caffeine consumption, period of abstinence, and types of coffee could have been useful to further control for potential confounding.

We fully agree with Karatzis et al who underscore that questionnaires on caffeine intake are subject to recall bias. This is the reason why we have collected in our study objective information using caffeine urinary excretion instead of self-reported intake. Although Karatzis et al suggested that 24-hour dietary recall or even food diaries would offer better information regarding caffeine intake, we believe that these questionnaires are not
much more immune to bias as compared with other questionnaires. In addition, the use of these questionnaires would not help us in deciphering the role of caffeine metabolites, namely, paraxanthine, theobromine, and theophylline, in cardiovascular health. Using data from urinary excretion instead of questionnaires, we found strong positive associations of paraxanthine and theophylline with arterial stiffness. This finding is in line with the fact that paraxanthine is a very potent methylxanthine, and even slightly more potent than caffeine.

Regarding the timing of the last caffeinated beverage, it was standardized, but indeed we have not adjusted the analyses for that parameter. We also have not systematically collected information on the different types of coffee.

Finally, we fully agree with the authors that we all have a lot more to learn concerning the effects of caffeine intake on arterial wall properties and blood pressure. We hope that our study will encourage further research to increase knowledge in this field.

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