

Authors' response to comment on: Less sodium and more potassium to reduce cardiovascular risk and the PURE study

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We thank Campbell *et al.*¹ for their interest in our recent commentary.² Our reply to the main point they raised is as follows: engaging in the controversy over the validity of the PURE study was beyond the scope of our article. Nevertheless, we acknowledge again the well-known limitations of spot urine sampling to estimate 24 h urinary sodium excretion, which have been widely emphasized by numerous experts and confirmed by more recent research. Such limitations have also been clearly stated in our commentary, describing the J-shaped relationship between salt consumption and incidence of cardiovascular events reported in some studies, including the PURE study.² We agree with Campbell *et al.* that, to support the dose–response relationship between dietary sodium consumption and blood pressure (BP), results from robust, randomized controlled trials based on 24 h urinary sodium estimation must be cited as well. Research conducted on a population with high sodium intake showed that the association between sodium intake and BP varied significantly depending on the methods used to estimate urinary sodium excretion, suggesting to consider the strengths of such methods before looking for associations between sodium intake and BP or other cardiovascular outcomes. Nevertheless, based on two population-based studies including almost 3000 patients, it was found that this relationship was similar regardless of the method used to estimate urinary sodium excretion, namely 24 h urine sample collection or fasting morning spot urine sampling and indirect 24 h urine sodium estimation through the Kawasaki equation. Although 24 h urine sample collection remains the method of choice to estimate sodium intake, further studies are needed to definitely clarify these issues. In their letter, Campbell *et al.* stated that the PURE study provides a good example that using an inappropriate methodology can

produce spurious results, even with a large sample size.¹ Beyond this, we believe that the debate engendered by this study has highlighted few crucial aspects: the need for simpler and more reliable methods to estimate sodium intake at the individual and population levels that can answer fundamental questions in the different health domains, including hypertension and cardiovascular risk management and the strong necessity to improve diet modification by both reducing sodium and increasing potassium consumption.³

References

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