



Total dietary antioxidant capacity and risk of type 2 diabetes

Niels van der Schaft¹ · Trudy Voortman¹

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We extend our gratitude to Dr Kawada for the comments on our paper on total dietary antioxidant capacity (TAC) and risk of type 2 diabetes [1, 2].

The author remarks that risk assessment for type 2 diabetes in relation to coffee intake must be precisely reviewed in light of our finding that the association between TAC and risk of type 2 diabetes was, to an extent, driven by the contribution of coffee intake to TAC. Indeed, coffee contributed to 49% of TAC in our study and is therefore one of the most important sources of antioxidants in the diet of our study population. Given the widely documented health benefits of antioxidants, we hypothesize that the previously reported protective associations between coffee consumption and risk of type 2 diabetes might to no small degree be driven by the antioxidants contained in coffee [3, 4].

We agree with Dr Kawada that the association between TAC and non-communicable diseases of both physical and psychiatric nature is an important topic that merits further investigation. Building upon the findings with relation to risk of type 2 diabetes, previous studies have also demonstrated that higher TAC is also associated with lower risk of myocardial infarction [5, 6]. Other studies have investigated TAC in relation to cause-specific and all-cause mortality, but the results of these studies have not been unequivocal [7, 8]. Similarly, studies on TAC and incident stroke have reported conflicting results [9, 10]. Furthermore, as highlighted in the letter by Dr Kawada, there have been some studies that investigated TAC with outcomes related to psychiatric illness, although there is a paucity of longitudinal studies on this topic. Taken together, the studies on TAC and health-related outcomes suggest that a dietary pattern rich in antioxidants could be an important and modifiable determinant of a wide range of physical and mental diseases, but further well-designed studies are needed to better understand

its properties as a risk factor and the corresponding pathophysiological pathways.

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✉ Trudy Voortman
trudy.voortman@erasmusmc.nl

¹ Department of Epidemiology, Erasmus University Medical Center, Rotterdam, The Netherlands