

## Letter to the Editor

# Coffee intake, glucose metabolism and gene polymorphisms

Robertson *et al.*<sup>(1)</sup> conducted a randomised study to investigate the effects of regular coffee intake on markers of glucose and lipid metabolism with special reference to *rs762551* SNP in the CYP1A2 gene. Before coffee intake, the AC genotype subjects with slow caffeine metabolism presented higher baseline glucose and NEFA than the AA genotype subjects with fast caffeine metabolism. Post-intervention, reduced postprandial glycaemia and NEFA were observed in the AC genotype subjects, which significantly differed from the change in the AA genotype subjects. The authors were cautious with regard to one-size-fits-all recommendation for coffee drinking and development of type 2 diabetes (T2D), because genotype and intervention were closely related to the association. I have a concern about their study related to type 2 diabetes mellitus (T2DM).

Denden *et al.*<sup>(2)</sup> conducted a meta-analysis to clarify the association between the CYP1A2 *rs762551* polymorphism and habitual coffee intake with particular reference to sex and ethnicity. OR of genotypes AA against AC+CC for coffee intake was 1.13 (95% CI 1.03, 1.24). In subgroup analyses, OR of genotypes AA against AC+CC in male, younger and Caucasian subjects for coffee intake were 1.21 (95% CI 1.08, 1.35), 1.71 (95% CI 1.18, 2.48) and 1.29 (95% CI 1.12, 1.49), respectively. They concluded that the *rs762551* AA genotype was closely associated with higher coffee intake, especially in the three presented subgroups. I suppose that the advantage of the AA genotype subjects for rapid caffeine metabolism would be lead to diminish the suppression of insulin sensitivity by habitual coffee intake, which would be supported by the preventive effect of decaffeinated coffee on incident T2DM<sup>(3)</sup>. Glucose-lowering effect by chlorogenic acids is explained by the suppression of hepatic glucose-6-phosphate activity<sup>(4)</sup>, and the reduced risk of developing T2DM by regular coffees would be explained by the combination of chlorogenic acids and caffeine intake<sup>(5)</sup>.

Robertson *et al.* also pointed out the interventional effect in the AC genotype subjects of the postprandial glycaemic and lipaemic responses to chronic coffee consumption. Mechanisms

of interventional effect on both glucose and NEFA should be specified by further study.

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