



Letter to the Editor

Letter to the editor regarding “Role of cytochrome epoxygenase (CYP2J2) in the pathophysiology of coronary artery disease in South Indian Population.”

Sir,

We read with great interest a research article published in *Indian heart Journal* titled “Role of cytochrome epoxygenase (CYP2J2) in the pathophysiology of coronary artery disease in South Indian Population.” by tantray et al.¹ I would like to congratulate the authors for their research in this novel field of finding the association between cytochrome epoxygenase and coronary artery disease (CAD). These studies would help us expand the horizon of causal agents of CAD and hence implement better preventive measures. However, after reading the research article, we have the following observation to make.

1. The authors in their research have studied the genotypic distribution of CYP2J2 G-50T polymorphism in patients with CAD and controls, and the data so obtained were analysed using an odds ratio between genotypic polymorphism and CAD. The research also studied the demographic and biochemical difference between the case and control group. According to the study results, the authors were able to establish a significant difference in the demographic and biochemical parameters of the case and control group. For example, compared with the controls, the patients with CAD had a higher prevalence of smoking (65% vs 36.4%, P value = 0.001), alcohol use (20% vs 4.54%, P value = 0.001), hypertension (35% vs 13.6%, P value = 0.004), diabetes (30% vs 8.2%, P values = 0.001) and family history of premature CAD (24% vs 11.8%, P value = 0.02). There was also a significant different in biochemical parameters, for example, difference was significant for total cholesterol (TC), low density lipoprotein cholesterol (LDL-C) and triglyceride (TG) when cases of CAD were compared with controls. TC (236 ± 15.5 vs 202 ± 10.3 mg/dL; $P < 0.0001$), LDL-C (140.8 ± 5.7 vs 102 ± 5.5 mg/dL; $P < 0.001$), TGs (193 ± 2.4 vs 121 ± 2.6 mg/dL; $P < 0.0001$), CHO/high-density lipoprotein-cholesterol (HDL-C) ratio (3.2 ± 1.4 Vs 3.8 ± 2.2 ; $P < 0.001$) and LDL/HDL-C ratio (5.3 ± 2.3 Vs 4.2 ± 1.1 ; $P < 0.001$) were higher in patients with CAD, whereas levels of HDL-C (40 ± 2.9 vs 40.5 ± 2.8 mg/dL; $P < 0.20$) were lower. The demographic and biochemical factors mentioned by the authors have been well established as the risk factors of CAD. Hence, we opine that an adjusted odds ratio, for all these variables, would be a better

representation of the association between CYP2J2 G-50T polymorphism and CAD.² One of the articles cited by the authors, by spiecker et al, has used adjusted odds ratio in analysing the data.³

2. In Table 5, odds ratio for G and T is mentioned as 0.16–0.61 and 1.62–5.9, respectively. How is odds ratio a range? In the same table, confidence intervals are mentioned as a single number.

3. To study the effect of CYP2J2 G-50T polymorphism as a risk factor for CAD, research with Mendelian randomisation in future would provide a more unbiased association between variables free of confounders.

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Conflicting interest

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ihj.2019.02.001>.

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