Letters to the Editor

Smoker’s paradox in heart failure: might asymmetric dimethylarginine be the possible explanation?: reply

We are grateful to Dr Yilmaz et al.1 for their interest in our article on smoking and clinical outcomes in patients with heart failure. They raise several important issues, including an alternative explanation for the findings from OPTIMIZE-HF that smokers hospitalized with heart failure had lower risk adjusted in-hospital mortality, shorter length of stay, and similar early post discharge mortality compared with non-smokers. We concur that, in addition to the potential mechanisms discussed in the manuscript, lower levels of asymmetric dimethylarginine (ADMA) among smokers compared with non-smokers with heart failure is an additional potential explanation. Asymmetric dimethylarginine is elevated in acute and chronic heart failure and, in chronic heart failure, is associated with adverse outcomes. However, it is important to note that a recent prospective study of 118 patients hospitalized with heart failure failed to demonstrate a relationship between plasma ADMA concentration and subsequent clinical outcomes in this patient population. Additional studies are necessary to investigate the ADMA levels in patients hospitalized with heart failure as well as the potential mechanisms for the smoker’s paradox such as a pre-conditioning-like effect with smoking. We also concur that from a scientific standpoint any potential protective effects of smoking in hospitalized heart failure patients should not be refuted immediately, as suggested in the accompanying editorial.3 Paradoxical associations between traditional cardiovascular risk factors and clinical outcomes in patients with established heart failure have been well described, being referred to as ‘reverse epidemiology’.4 Heart failure patients with current or recent smoking exposure may have different intrinsic risk and/or responses to therapy compared with heart failure patients without recent exposure to smoking. Nevertheless, from a clinical perspective, effective smoking prevention and cessation methods should be vigorously employed in all individuals, including those hospitalized with heart failure.

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References

Gregg C. Fonarow

Department of Medicine
Division of Cardiology
Ahmanson-UCLA Medical Center
47-123 CHS, 10833 Le Conte Avenue
Los Angeles CA 90095-1679
USA
Fax: +1 310 206 9111
E-mail: gfonarow@mednet.ucla.edu

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Regrettably, on page 2272, in Figure 3, the line types of group 4 and group 5 were transposed. The corrected figure is printed below. The authors wish to apologize for this error.

Mehmet Birhan Yilmaz

Department of Cardiology
Cumhuriyet University Faculty of Medicine
Sivas
Turkey
Tel: +90 346 258 0828
Fax: +90 346 258 1305
Email: mehmet.birhan.yilmaz@tkd.org.tr

Osman Can Yontar

Department of Cardiology
Cumhuriyet University Faculty of Medicine
Sivas
Turkey

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References

Gregg C. Fonarow

Department of Medicine
Division of Cardiology
Ahmanson-UCLA Medical Center
47-123 CHS, 10833 Le Conte Avenue
Los Angeles CA 90095-1679
USA
Fax: +1 310 206 9111
E-mail: gfonarow@mednet.ucla.edu

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Mehmet Birhan Yilmaz

Department of Cardiology
Cumhuriyet University Faculty of Medicine
Sivas
Turkey
Tel: +90 346 258 0828
Fax: +90 346 258 1305
Email: mehmet.birhan.yilmaz@tkd.org.tr

Osman Can Yontar

Department of Cardiology
Cumhuriyet University Faculty of Medicine
Sivas
Turkey

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References
Ivabradine at 15–20 min of ischaemia

Infarct size (% area at risk)

Transmural blood flow per time (mL/min/g)

Group 4 (Ivabradine)
Group 5 (Placebo)
Group 6 (Ivabradine + Pacing)

$P < 0.05$

$r = -0.71$
$r = -0.61$
$r = -0.48$