Author`s Reply

To the Editor,

I appreciate his interest in my study (1). He has pointed out about the rationalization of the concentrations of trimetazidine (200 μM) and coenzyme Q10 (200 mg/L) used in the ventricular myocytes. I performed the preliminary experiments to analyze the responses of rat cardiomyocytes to serial doses of TMZ (12.5–200 μM) or CoQ10 (12.5–200 mg/L). TMZ or CoQ10 attenuated cisplatin-induced cell toxicity in a dose-dependent manner using a CCK8 assay. However, statistical significance was only observed at a concentration of 200 μM TMZ or 200 mg/L CoQ10. Therefore, I chose to use 200 μM TMZ and 200 mg/L CoQ10 for subsequent experiments.

He has recommended measuring parameters, such as intracellular calcium levels and caspase 3 and caspase 9 activities. These parameters could be useful to investigate the mechanisms of the cardioprotective role of trimetazidine in chemotherapy-induced cardiotoxicity; however, my study focused on the upstream of caspase activities as described in the paper's introduction. ROS-mitochondrial dysfunction-Nrf2/CytoC-apoptosis was the major framework of my study. On the other hand, caspase-dependent apoptosis has been briefly dealt with in the paper's introduction and discussion.

Li Zhao
Department of Cardiology, Obstetrics and Gynecology Hospital of Fudan University; Shanghai-*China*

Reference

1. Zhao L. Protective effects of trimetazidine and coenzyme Q10 on cisplatin-induced cardiotoxicity by alleviating oxidative stress and mitochondrial dysfunction. Anatol J Cardiol 2019; 22: 232-9.

Address for Correspondence: Li Zhao, MD,
Department of Cardiology,
Obstetrics and Gynecology Hospital of Fudan University;
No. 419, Fangxie Road,
Huangpu District Shanghai-*China*
Phone: 86-21-33189900
E-mail: zhaoli20181212@163.com