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Letter to the Editor

A novel, potential therapeutic target in diabetic cardiomyocytes: ROCK2

Suiyang Tong [a], Liangliang Zhang [b], Jacob Joseph [b], Xuejun Jiang [c,*]

[a] Department of Cardiology, Suizhou Hospital, Hubei University of Medicine, Suizhou, Hubei, PR China
[b] Division of Cardiovascular Medicine, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, United States of America
[c] Department of Cardiology, Renmin Hospital of Wuhan University, Wuhan, Hubei, PR China

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Dear Editor,

We read the recently published article titled "ROCK2 promotes ryanodine receptor phosphorylation and arrhythmic calcium release in diabetic cardiomyocytes" by Soliman H et al. with great interest [1]. Their results revealed that ROCK inhibition improved some parameters of contractile function and Ca^{2+} handling in cardiomyocytes from diabetic rat hearts.

Diabetic patients with cardiomyopathy show a higher incidence of arrhythmia and sudden death. Koyani et al. found that impairing the CaMKII-phospholamban-sarcoplasmic reticulum Ca^{2+}-ATPase 2a axis and protein kinase C activity in cardiomyocytes leads to impaired cardiac contractility and electrophysiological function [2]. Lin et al. demonstrated that over-activation of ROCK2 contributes to diabetic cardiomyopathy via multiple mechanisms, including direct phosphorylation and activation of PKCβ2, interference with PDK-1-mediated phosphorylation and activation of AKT, and translocation of GLUT4. These findings suggest that ROCK2 is a critical node in the development of diabetic cardiomyopathy and may be an effective therapeutic target for improving cardiac function in diabetes [3]. Sunamura et al. reported that cardiomyocyte-specific ROCK1 deficiency promoted pressure-overload-induced cardiac dysfunction, whereas cardiomyocyte-specific ROCK2 deficiency improved cardiac function [4]. Shimizu et al. demonstrated that ROCK2 may show therapeutic effects in patients with left ventricular diastolic dysfunction by reducing left ventricular wall thickness and fibrosis, and improving isovolumetric relaxation [5].

According to the present findings, ROCK2 plays a pivotal role in cardiac function by impairing cardiac Ca^{2+} homeostasis and may have important clinical implications for the treatment of diabetes-related dysrhythmias.

Conflict of interest

The authors report no relationships that could be construed as conflicts of interest.

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* Corresponding author at: Department of Cardiology, Renmin Hospital of Wuhan University, Cardiovascular Research Institute, Wuhan University, Jiefang Road 238, Wuhan 430060, PR China.
E-mail address: xjjiang@whu.edu.cn (X. Jiang).

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