

EDITORIAL COMMENT

At the Crossroads of Cardiac Beta-Adrenergic Receptor Signaling and Myocardial Glucose Metabolism*



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Heart failure remains a leading cause of global mortality. Despite recent advances in clinical therapy, long-term prognosis is poor. Beta-adrenergic receptors (βARs) are members of the G protein-coupled receptor (GPCR) family and play a central role in the progression of heart failure. In the heart, β2AR is a predominant isoform. Under pathological conditions, including myocardial ischemia and injury, βARs undergo GPCR kinase (GRK)-mediated desensitization and downregulation. Chronic desensitization of βARs leads to activation of the sympathetic nervous system, which in turn causes catecholamine toxicity resulting in ventricular dysfunction.¹

Cardiac energy metabolism is the process by which the heart produces the necessary amount of adenosine triphosphate (ATP) required to sustain contractile function.² Under normal physiological conditions, the adult heart generates most of its ATP from fatty acid oxidation³; however, glucose is also necessary for cardiac function in response to various physiological and pathological stimuli.^{4,5} It is well understood that adrenergic stimulation increases cardiac function, necessitating an increase for fuel and energy, but there is limited knowledge in how βAR signaling controls glucose uptake metabolism in the heart.

At the crossroads of cardiac βAR signaling and myocardial glucose metabolism is the recent study conducted by Jovanovic et al.,⁶ published in this issue of *JACC: Basic to Translational Science*, in which the investigators aimed to determine how β2AR controls cardiac glucose metabolism. Jovanovic et al.⁶ showed that the cardiac β2AR is required to stimulate glucose transporter type 4 (GLUT4)-mediated glucose uptake in 2 ex vivo model systems, isolated ventricular myocytes and isolated working hearts. Myocytes and hearts were isolated from mice with a cardiac-specific deletion of β2AR (β2AR cardiac knockout mice) and mice developed using Clustered Regularly Interspaced Short Palindromic Repeats knock-in technology, expressing endogenous β2AR but lacking the GRK2 phosphorylation site at Ser355/356 (β2AR GRK mutant mice). In the study, all mice exhibited normal and relatively similar cardiac function at baseline and after adrenergic stimulation with isoproterenol.⁶ Using these models, Jovanovic et al.⁶ first demonstrated that deletion of β2AR inhibited adrenergic stimulation of GLUT4-mediated glucose uptake in myocytes and glucose oxidation in working hearts. In the working heart model, glucose oxidation was measured at baseline and after infusion of isoproterenol, and deletion of β2AR prevented isoproterenol-induced increases in glucose oxidation.⁶ Additionally, membrane insertion of GLUT4 was blocked in the β2AR cardiac knockout mice working hearts. Jovanovic et al.⁶ then showed that stimulation of β2AR resulted in G_i-Akt-mediated GLUT4 translocation and glucose uptake in isolated myocytes from rat hearts. Adult myocytes were first pretreated with a β1AR or β2AR selective blocker and subsequently stimulated with isoproterenol, where pretreatment with the β2AR selective blocker prevented isoproterenol-induced phosphorylation of Akt and completely blocked GLUT4 membrane insertion.⁶ The

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