Mechanistic role of β₂ adrenergic receptor in glucose homeostasis

Gaetano Santulli and Guido Iaccarino
Published on April 24, 2017

Wang, Liu, Fu and colleagues report that β₂-adrenergic receptor (β₂AR) plays a key role in hyperinsulinaemia-induced cardiac dysfunction (1). Overall, the data are very interesting and compelling. However, we noticed that in this paper β₂AR⁻/⁻ mice do not exhibit glucose intolerance; in fact, they seem to have a response to intraperitoneal glucose that is even better than wild-type mice (though a statistical analysis comparing these two groups is not provided). Although surprisingly not reported by the Authors, mounting evidence indicates that the deletion of β₂AR has detrimental effects on glucose metabolism (2-4). Indeed, we have demonstrated that β₂AR⁻/⁻ mice display impaired insulin release and significant glucose intolerance (2). Muzzi and colleagues found that the ablation of βARs mechanistically underlies impaired glucose homeostasis (3). Other groups have confirmed these results, also showing that β₂AR⁻/⁻ mice develop diabetic-related microvascular complications (i.e. retinopathy) (4). Nonetheless, the Authors fail to at least discuss previous relevant literature describing the alterations in glucose metabolism observed in β₂AR⁻/⁻ mice and do not accurately circumstantiate their findings. Furthermore, the Authors do not provide any measurement (not in vivo nor in isolated islets) of insulin levels following glucose challenge, showing just baseline serum levels. We believe that for the sake of scientific appropriateness the Readers of Circulation will appreciate a clarification, in particular regarding the fact that pertinent literature in the field has been overlooked.

References


Competing Interests: None declared.