Supplemental Figure S1. The cAMP signaling and pharmacological interventions used in this study. Stimulation of Gαs-coupled receptors by epinephrine, glucagon or other ligands leads to activation of adenylyl cyclase (AC), which results in an increase of cAMP synthesis. The levels of cAMP is also controlled by phosphodiesterases (PDE). Epac and PKA are two major effectors of cAMP. Binding of cAMP to Epac results in activation of Epac and its downstream effector Rap proteins. Under basal condition, regulatory (R) subunits and catalytic (C) subunits of PKA forms a complex and the kinase activity of PKA is restricted. Upon binding of cAMP to R subunits, C subunits are released from the complex, resulting in PKA activation. Pharmacological inhibitors or activators of AC, PDE or PKA were shown in blue boxes.