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Adrenal G-protein coupled receptor kinase-2 in regulation of sympathetic nervous system activity in heart failure

Katie A McCrink, Ava Brill, Anastasios Lymperopoulos

Abstract

Heart failure (HF), the number one cause of death in the western world, is caused by the insufficient performance of the heart leading to tissue underperfusion in response to an injury or insult. It comprises complex interactions between important neurohormonal mechanisms that try but ultimately fail to sustain cardiac output. The most prominent such mechanism is the sympathetic (adrenergic) nervous system (SNS), whose activity and outflow are greatly elevated in HF. SNS hyperactivity confers significant toxicity to the failing heart and markedly increases HF morbidity and mortality via excessive activation of adrenergic receptors, which are G protein-coupled receptors. Thus, ligand binding induces their coupling to heterotrimeric G proteins that transduce intracellular signals. G protein signaling is turned-off by the agonist-bound receptor phosphorylation courtesy of G-protein coupled receptor kinases (GRKs), followed by βarrestin binding, which prevents the GRK-phosphorylated receptor from further interaction with the G proteins and simultaneously leads it inside the cell

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