# Abstract

β-Adrenergic receptors (β-ARs) are G-protein-coupled receptors (GPCR), widely present in the animal organism and mediate catecholamine pathways leading to diverse physiological responses. The family of β-ARs consists of $\beta_1$-AR, $\beta_2$-AR and $\beta_3$-AR, which are distinguished by their affinity to adrenaline and noradrenaline. A typical model of β-AR signalling includes binding of the ligand, G-protein coupling, activation of adenylyl cyclase (AC) resulting in production of the second messenger cAMP and activation of protein kinase A (PKA) that phosphorylates downstream proteins leading to physiological responses. Beacause excessive catecholamine signalling can cause undesirable consequences, a mechanism has evolved, which attenuates the function of β-ARs in spite of further stimulation, so called desensitization. The classic course of desensitization consists of characteristic steps including phosphorylation of the receptor, β-arrestin attachement and uncoupling of the G-protein from β-AR. Restoration of the signalling ability is allowed through resensitization of β-AR when the receptor is sequestrated and dephosphorylated. Given that β-ARs are structurally and genetically different, it is reasonable to consider that each step of the desenstization process may happen differently among the different subtypes of β-ARs.



### Keywords

β-Adrenergic receptor, G-protein, β-arrestin, adenylyl cyclase, desensitization