

## Rabbit Anti-Beta arrestin 2/PE Conjugated antibody

SL1332R-PE

<b>Product Name</b>	Anti-Beta arrestin 2/PE
<b>Chinese Name</b>	PE 标记的 $\beta$ -抑制蛋白 2 抗体/ $\beta$ 休止蛋白 2/ $\beta$ -arrestin 抗体
<b>Alias</b>	Beta-arrestin 2; Beta Arrestin 2; ARB 2; ARB2; ARR 2; ARR2; ARRB 2; ARRB2; ARRB2_HUMAN; Arrestin 3; Arrestin beta 2; Arrestin beta-2; BARR2; DKFZp686L0365; HGNC:712; Beta-arrestin-2.
<b>Research Area</b>	Tumour Cell biology Neurobiology Signal transduction Growth factors and hormones Kinases and Phosphatases Channel protein The cell membrane 受体 G protein-coupled receptor Cytoskeleton
<b>Immunogen Species</b>	Rabbit
<b>Clonality</b>	Polyclonal
<b>React Species</b>	Human,Mouse,Rat(predicted:Chicken,Dog,Pig,Cow,Horse,Rabbit,GuineaPig) IF=1:100-500
<b>Applications</b>	not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
<b>Molecular weight</b>	45kDa
<b>Cellular localization</b>	The cell membrane
<b>Form</b>	Lyophilized or Liquid
<b>Concentration</b>	1mg/ml
<b>immunogen</b>	KLH conjugated synthetic peptide derived from human Beta-arrestin 2
<b>Lsotype</b>	IgG
<b>Purification</b>	affinity purified by Protein A
<b>Storage Buffer</b>	1M TBS(pH7.4) with 1% BSA, 3% Proclin300 and 50% Glycerol. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
<b>Storage</b>	
<b>Product Detail</b>	<b>background:</b>

Members of arrestin/beta arrestin protein family are thought to participate in agonist mediated desensitization of G protein coupled receptors and cause specific dampening of cellular responses to stimuli such as hormones, neurotransmitters, or sensory signals. Arrestin beta 2, like arrestin beta 1, was shown to inhibit beta adrenergic receptor function in vitro. It is expressed at high levels in the central nervous system and may play a role in the regulation of synaptic receptors

**Function:**

Functions in regulating agonist-mediated G-protein coupled receptor (GPCR) signaling by mediating both receptor desensitization and resensitization processes. During homologous desensitization, beta-arrestins bind to the GPRK-phosphorylated receptor and sterically preclude its coupling to the cognate G-protein; the binding appears to require additional receptor determinants exposed only in the active receptor conformation. The beta-arrestins target many receptors for internalization by acting as endocytic adapters (CLASPs, clathrin-associated sorting proteins) and recruiting the GPCRs to the adapter protein 2 complex 2 (AP-2) in clathrin-coated pits (CCPs). However, the extent of beta-arrestin involvement appears to vary significantly depending on the receptor, agonist and cell type. Internalized arrestin-receptor complexes traffic to intracellular endosomes, where they remain uncoupled from G-proteins. Two different modes of arrestin-mediated internalization occur. Class A receptors, like ADRB2, OPRM1, ENDRA, D1AR and ADRA1B dissociate from beta-arrestin at or near the plasma membrane and undergo rapid recycling. Class B receptors, like AVPR2, AGTR1, NTSR1, TRHR and TACR1 internalize as a complex with arrestin and traffic with it to endosomal vesicles, presumably as desensitized receptors, for extended periods of time. Receptor resensitization then requires that receptor-bound arrestin is removed so that the receptor can be dephosphorylated and returned to the plasma membrane. Mediates endocytosis of CCR7 following ligation of CCL19 but not CCL21. Involved in internalization of P2RY1, P2RY4, P2RY6 and P2RY11 and ATP-stimulated internalization of P2RY2. Involved in phosphorylation-dependent internalization of OPRD1 and subsequent recycling or degradation. Involved in ubiquitination of IGF1R. Beta-arrestins function as multivalent adapter proteins that can switch the GPCR from a G-protein signaling mode that transmits short-lived signals from the plasma membrane via small molecule second messengers and ion channels to a beta-arrestin signaling mode that transmits a distinct set of signals that are initiated as the receptor internalizes and transits the intracellular compartment. Acts as signaling scaffold for MAPK pathways such as MAPK1/3 (ERK1/2) and MAPK10 (JNK3). ERK1/2 and JNK3 activated by the beta-arrestin scaffold are largely excluded from the nucleus and confined to cytoplasmic locations such as endocytic vesicles, also called beta-arrestin signalosomes. Acts as signaling scaffold for the AKT1

pathway. GPCRs for which the beta-arrestin-mediated signaling relies on both ARRB1 and ARRB2 (codependent regulation) include ADRB2, F2RL1 and PTH1R. For some GPCRs the beta-arrestin-mediated signaling relies on either ARRB1 or ARRB2 and is inhibited by the other respective beta-arrestin form (reciprocal regulation). Increases ERK1/2 signaling in AGTR1- and AVPR2-mediated activation (reciprocal regulation). Involved in CCR7-mediated ERK1/2 signaling involving ligand CCL19. Is involved in type-1A angiotensin II receptor/AGTR1-mediated ERK activity. Is involved in type-1A angiotensin II receptor/AGTR1-mediated MAPK10 activity. Is involved in dopamine-stimulated AKT1 activity in the striatum by disrupting the association of AKT1 with its negative regulator PP2A. Involved in AGTR1-mediated chemotaxis. Appears to function as signaling scaffold involved in regulation of MIP-1-beta-stimulated CCR5-dependent chemotaxis. Involved in attenuation of NF-kappa-B-dependent transcription in response to GPCR or cytokine stimulation by interacting with and stabilizing CHUK. Suppresses UV-induced NF-kappa-B-dependent activation by interacting with CHUK. The function is promoted by stimulation of ADRB2 and dephosphorylation of ARRB2. Involved in p53/TP53-mediated apoptosis by regulating MDM2 and reducing the MDM2-mediated degradation of p53/TP53. May serve as nuclear messenger for GPCRs. Upon stimulation of OR1D2, may be involved in regulation of gene expression during the early processes of fertilization. Also involved in regulation of receptors others than GPCRs. Involved in endocytosis of TGFBR2 and TGFBR3 and down-regulates TGF-beta signaling such as NF-kappa-B activation. Involved in endocytosis of low-density lipoprotein receptor/LDLR. Involved in endocytosis of smoothed homolog/Smo, which also requires ADRBK1. Involved in endocytosis of SLC9A5. Involved in endocytosis of ENG and subsequent TGF-beta-mediated ERK activation and migration of epithelial cells. Involved in Toll-like receptor and IL-1 receptor signaling through the interaction with TRAF6 which prevents TRAF6 autoubiquitination and oligomerization required for activation of NF-kappa-B and JUN. Involved in insulin resistance by acting as insulin-induced signaling scaffold for SRC, AKT1 and INSR. Involved in regulation of inhibitory signaling of natural killer cells by recruiting PTPN6 and PTPN11 to KIR2DL1.

**Subcellular Location:**

Cytoplasm. Nucleus. Cell membrane. Membrane > clathrin-coated pit. Cytoplasmic vesicle. Translocates to the plasma membrane and colocalizes with antagonist-stimulated GPCRs.

**Post-translational modifications:**

Phosphorylated at Thr-382 in the cytoplasm; probably dephosphorylated at the plasma membrane. The phosphorylation does not regulate internalization and recycling of ADRB2, interaction with clathrin or AP2B1.

The ubiquitination status appears to regulate the formation and trafficking of beta-arrestin-GPCR complexes and signaling. Ubiquitination appears to occur GPCR-specific. Ubiquitinated by MDM2; the ubiquitination is required for rapid internalization of ADRB2. Deubiquitinated by USP33; the deubiquitination leads to a dissociation of the beta-arrestin-GPCR complex. Stimulation of a class A GPCR, such as ADRB2, induces transient ubiquitination and subsequently promotes association with USP33. Stimulation of a class B GPCR promotes a sustained ubiquitination.

**Similarity:**

Belongs to the arrestin family.

**Database links:**

[Entrez Gene: 409](#) Human

[Entrez Gene: 216869](#) Mouse

[Entrez Gene: 25388](#) Rat

[Olim: 107941](#) Human

[SwissProt: P32121](#) Human

[SwissProt: Q91YI4](#) Mouse

[SwissProt: P29067](#) Rat

[Unigene: 435811](#) Human

[Unigene: 203747](#) Mouse

[Unigene: 32973](#) Rat

**Important Note:**

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

$\beta$  抑制因子-2 又称  $\beta$  休止蛋白 2, 也是是调节 CD4+T 细胞存活和自身免疫性的关键因子, 与促进 Tlymphocyte 存活和自身免疫发病相关。经研究发现  $\beta$ -arrestin 不仅仅能阻断蛋白合成, 也能诱导蛋白合成, 参与信号传导。

对 Arrestins 家族的研究-探究  $\beta$ -arrestin 在 G protein-coupled receptor 信号

传导通路中的地位和作用，是当今生物学中信号传导 Research Area 的热门课题。

$\beta$  抑制因子-1 又称“胰岛素受体复合体”，近年来国内外科科研人员对  $\beta$ -arrestin 在 II 型 Diabetes 发生的研究机制方面有了新的突破，认为： $\beta$ -arrestin 缺少或下降可直接导致了胰岛素耐受和 II 型 Diabetes 的发生。 $\beta$ -arrestin1 蛋白  $\beta$ -arrestin2 蛋白有高度的同源性。