

## Product datasheet for TP525799

### Arrb1 (NM\_178220) Mouse Recombinant Protein

#### Product data:

Product Type:	Recombinant Proteins
Description:	Purified recombinant protein of Mouse arrestin, beta 1 (Arrb1), transcript variant b, with C-terminal MYC/DDK tag, expressed in HEK293T cells, 20ug
Species:	Mouse
Expression Host:	HEK293T
Expression cDNA Clone or AA Sequence:	>MR225799 representing NM_178220 <b>Red</b> =Cloning site <b>Green</b> =Tags(s)
	<p>MGDKGTRVFKKASPNGKLTVYLGKRDFVDHIDLVDLPVDGWLVDPEYLKERRVYVTLTCAFRYGREDLDV LGLTFRKDLFVANVQSFPAPEDKKPLTRLQERLIKKLGEHACPFTFEIPPNLPCSVTLQPGPEDTGKAC GVDYEVKAFCAENLEEKIHKRNSVRLVIRKQYAPERPGPQPTAETTRQFLMSDKPLHLEASLDKEIYYH GEPISVNVHVTNNTNKTVKKIKISVRQYADICLFNTAQYKCPVAMEEADDNVAPSSTFCKVYTLTPFLAN NREKRGLALDGKLVKHEDTNLSSTLLREGANREILGIIVSYKVKVCLVSRGGDVAVELPFTLMHPKPKE EPPHREVPESETPVDTNLIELDTNDDDIVFEDFARQRLKGMKDDKDEEDDGTGSPHLNNR</p> <p><b>TRTRPLEQKLISEEDLAANDILDYKDDDDKV</b></p>
Tag:	C-MYC/DDK
Predicted MW:	46.7 kDa
Concentration:	>0.05 µg/µL as determined by microplate BCA method
Purity:	> 80% as determined by SDS-PAGE and Coomassie blue staining
Buffer:	25 mM Tris-HCl, 100 mM glycine, pH 7.3, 10% glycerol
Note:	For testing in cell culture applications, please filter before use. Note that you may experience some loss of protein during the filtration process.
Storage:	Store at -80°C after receiving vials.
Stability:	Stable for 12 months from the date of receipt of the product under proper storage and handling conditions. Avoid repeated freeze-thaw cycles.
RefSeq:	<a href="#">NP_835738</a>
Locus ID:	109689
UniProt ID:	<a href="#">Q8BWG8</a>



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RefSeq Size:	7088
Cytogenetics:	7 54.09 cM
RefSeq ORF:	1230
Synonyms:	1200006I17Rik; AW208571; G430100A01Rik

**Summary:** 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. Involved in internalization of P2RY4 and UTP-stimulated internalization of P2RY2. Involved in phosphorylation-dependent internalization of OPRD1 and subsequent recycling. Involved in the degradation of cAMP by recruiting cAMP phosphodiesterases to ligand-activated receptors. 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). ERK1/2 activated by the beta-arrestin scaffold is largely excluded from the nucleus and confined to cytoplasmic locations such as endocytic vesicles, also called beta-arrestin signalosomes. Recruits c-Src/SRC to ADRB2 resulting in ERK activation. 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). Inhibits ERK1/2 signaling in AGTR1- and AVPR2-mediated activation (reciprocal regulation). Is required for SP-stimulated endocytosis of NK1R and recruits c-Src/SRC to internalized NK1R resulting in ERK1/2 activation, which is required for the antiapoptotic effects of SP. Is involved in proteinase-activated F2RL1-mediated ERK activity. Acts as signaling scaffold for the AKT1 pathway. Is involved in alpha-thrombin-stimulated AKT1 signaling. Is involved in IGF1-stimulated AKT1 signaling leading to increased protection from apoptosis. Involved in activation of the p38 MAPK signaling pathway and in actin bundle

formation. Involved in F2RL1-mediated cytoskeletal rearrangement and chemotaxis. Involved in AGTR1-mediated stress fiber formation by acting together with GNAQ to activate RHOA. 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. May serve as nuclear messenger for GPCRs. Involved in OPRD1-stimulated transcriptional regulation by translocating to CDKN1B and FOS promoter regions and recruiting EP300 resulting in acetyla