

# Recombinant Human 14-3-3 tau/14-3-3 theta/YWHAQ Protein (GST Tag)



Catalog Number: PKSH031392

**Note:** Centrifuge before opening to ensure complete recovery of vial contents.

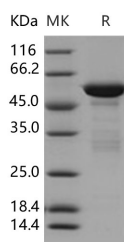
## Description

<b>Synonyms</b>	1C5;41701;HS1
<b>Species</b>	Human
<b>Expression Host</b>	E.coli
<b>Sequence</b>	Met 1-Asn 245
<b>Accession</b>	P27348
<b>Calculated Molecular Weight</b>	54.6 kDa
<b>Observed molecular weight</b>	53 kDa
<b>Tag</b>	N-GST

## Properties

<b>Purity</b>	> 88 % as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	Please contact us for more information.
<b>Storage</b>	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
<b>Shipping</b>	This product is provided as lyophilized powder which is shipped with ice packs.
<b>Formulation</b>	Lyophilized from sterile 20mM Tris, 0.15m NaCl, 20mM GSH, pH 7.5 Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
<b>Reconstitution</b>	Please refer to the printed manual for detailed information.

## Data



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## Background

G protein-coupled receptor kinase 5, also known as G protein-coupled receptor kinase GRK5 and GRK5, is a member of the protein kinase superfamily, AGC Ser/Thr protein kinase family and GPRK subfamily. GRKs specifically phosphorylate agonist-occupied G protein-coupled receptors at the inner surface of the plasma membrane (PM), leading to receptor desensitization. GRKs utilize a variety of mechanisms to bind tightly, and sometimes reversibly, to cellular membranes. GRKs play an important role in mediating agonist-specific desensitization of numerous G protein-coupled receptors. GRK5 contains one AGC-kinase C-terminal domain, one protein kinase domain and one RGS domain. GRK5 specifically phosphorylates the activated forms of G protein-coupled receptors. Phospholipid-stimulated autophosphorylation may represent a novel mechanism for membrane association and regulation of GRK5 activity. GRK5

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deficiency significantly exaggerates microgliosis and astrogliosis in the presence of an inflammatory initiator, such as the excess fibrillar Abeta and the subsequent active inflammatory reactions. GRK5 deficiency has been linked to early Alzheimer's disease in humans and mouse models of the disease.

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