

# Recombinant Human MUSK Kinase Protein (aa 433-783, His & GST Tag)



Catalog Number:PKSH030345

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

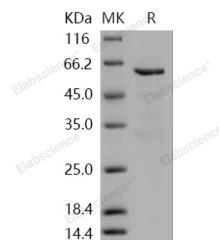
## Description

Synonyms	CMS9;FADS
Species	Human
Expression Host	Baculovirus-Insect Cells
Sequence	Arg 433-Val 783
Accession	O15146-2
Calculated Molecular Weight	68.0 kDa
Observed molecular weight	58 kDa
Tag	N-His-GST

## Properties

Purity	> 90 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per $\mu$ g of the protein as determined by the LAL method.
Storage	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < - 20°C.
Formulation	Supplied as sterile solution of 20mM Tris, 500mM NaCl, pH 7.4, 10mM GSH.
Reconstitution	Not Applicable

## Data



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## Background

Muscle, skeletal receptor tyrosine-protein kinase, also known as Muscle-specific tyrosine-protein kinase receptor, Muscle-specific kinase receptor, and MUSK, is a single-pass type I membrane protein which belongs to the protein kinase superfamily and tyrosine kinase family. MUSK contains one FZ (frizzled) domain, three Ig-like C2-type (immunoglobulin-like) domains and one protein kinase domain. This protein is a muscle-specific tyrosine kinase receptor and it may play a role in clustering of the acetylcholine receptor in the postsynaptic neuromuscular junction. MUSK expression is increased in muscle cells stimulated with Wnt or at conditions when the Wnt signaling was activated. MUSK is a muscle-specific receptor tyrosine kinase that is activated by agrin. It has a critical role in neuromuscular synapse formation. MUSK is a receptor tyrosine kinase that is a key mediator of agrin's action and is involved in neuromuscular junction (NMJ) organization. Defects in MUSK encoding gene is a cause of autosomal recessive congenital myasthenic syndrome (CMS). Congenital myasthenic syndromes are inherited disorders of neuromuscular transmission that stem from mutations in presynaptic, synaptic, or postsynaptic proteins. MUSK mutations lead to decreased agrin-dependent AChR aggregation, a critical step in the formation of the neuromuscular junction. Mutations in this receptor encoding

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gene also have been associated with congenital myasthenic syndrome.

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