EphA4 Protein, Mouse (528a.a, HEK293, His)

Cat. No.:	HY-P78293
Synonyms:	Sek; Sek1; EphA4; HEK8; SEK; TYRO1; EK8
Species:	Mouse
Source:	HEK293
Accession:	Q03137 (V20-T547)
Gene ID:	13838
Molecular Weight:	60-70 kDa

PROPERTIES

Biological Activity	The enzyme activity of this recombinant protein is testing in progress, we cannot offer a guarantee yet.
Appearance	Solution.
Formulation	Supplied as a 0.22 μm filtered solution of PBS, pH 7.4.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	N/A.
Storage & Stability	Stored at -80°C for 1 year. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles.
Shipping	Shipping with dry ice.

DESCRIPTION

Background	The EphA4 protein, a receptor tyrosine kinase, engages in contact-dependent bidirectional signaling with membrane-bound ephrin family ligands on adjacent cells. Distinguished by its high promiscuity, EphA4 uniquely binds and is physiologically activated by both GPI-anchored ephrin-A and transmembrane ephrin-B ligands, including EFNA1 and EFNB3. Upon activation by ephrin ligands, EphA4 modulates cell morphology and integrin-dependent cell adhesion through the regulation of Rac, Rap, and Rho GTPases activity. Crucial in the development of the nervous system, EphA4 controls various steps of axonal guidance, including the establishment of corticospinal projections and the segregation of motor and sensory axons during neuromuscular circuit development. In synaptic plasticity, EphA4 participates by phosphorylating CDK5 at 'Tyr-15,' leading to the regulation of RHOA and dendritic spine morphogenesis. Furthermore, EphA4 plays roles in repair after injury by preventing axonal regeneration and in angiogenesis, contributing to central nervous system vascular formation. Its promiscuity extends its involvement in various cell-cell signaling processes, regulating the development of the thymic epithelium and, during the development of the cochlear organ of Corti, facilitating pillar cell separation through the formation of a ternary complex with ADAM10 and CADH1, leading to the cleavage of CADH1 and disruption of adherens junctions. EphA4 also phosphorylates CAPRIN1, promoting CAPRIN1-dependent formation of a membraneless compartment.

Caution: Product has not been fully validated for medical applications. For research use only.

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