ROBO3 Polyclonal Antibody

catalog number: E-AB-67631

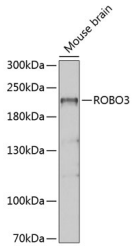
Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

Description

Reactivity	Mouse
Immunogen	Recombinant fusion protein of human ROBO3 (NP_071765.2).
Host	Rabbit
Isotype	IgG
Purification	Affinity purification
Buffer	Phosphate buffered solution, pH 7.4, containing 0.05% stabilizer and 50% glycerol.

Applications	Recommended Dilution
WB	1:500-1:2000

Data



Western blot analysis of extracts of Mouse brain using ROBO3 Polyclonal Antibody at dilution of 1:1000.

Observed-MV:200 kDa

Calculated-MV:110 kDa/148 kDa

Preparation & Storage

Storage	Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles.
Shipping	The product is shipped with ice pack,upon receipt,store it immediately at the temperature recommended.

Background

This gene is a member of the Roundabout (ROBO) gene family that controls neurite outgrowth, growth cone guidance, and axon fasciculation. ROBO proteins are a subfamily of the immunoglobulin transmembrane receptor superfamily. SLIT proteins 1-3, a family of secreted chemorepellants, are ligands for ROBO proteins and SLIT/ROBO interactions regulate myogenesis, leukocyte migration, kidney morphogenesis, angiogenesis, and vasculogenesis in addition to neurogenesis. This gene, ROBO3, has a putative extracellular domain with five immunoglobulin (Ig)-like loops and three fibronectin (Fn) type III motifs, a transmembrane segment, and a cytoplasmic tail with three conserved signaling motifs: CC0, CC2, and CC3 (CC for conserved cytoplasmic). Unlike other ROBO family members, ROBO3 lacks motif CC1. The ROBO3 gene regulates axonal navigation at the ventral midline of the neural tube. In mouse, loss of Robo3 results in a complete failure of commissural axons to cross the midline throughout the spinal cord and the hindbrain. Mutations ROBO3 result in horizontal gaze palsy with progressive scoliosis (HGPPS); an autosomal recessive disorder characterized by congenital absence of horizontal gaze, progressive scoliosis, and failure of the corticospinal and somatosensory axon tracts to cross the midline in the medulla. Alternative transcript variants have been described but have not been experimentally validated.

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