Recombinant Human DCX Protein (aa 45-150, GST Tag)

Catalog Number: PKSH030620

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

Description			
Species	Human		
Source	E coli-derived Human DCX protein Ala 45-Val 150, with an N-terminal GST		
Calculated MW	39.4 kDa		
Observed MW	36 kDa		
Accession	O43602-2		
Bio-activity	Not validated for activity		
Properties			
Purity	> 82 % as determined by reducing SDS-PAGE.		
Endotoxin	Please contact us for more information.		
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 t		
	°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of		
	reconstituted samples are stable at $< -20^{\circ}$ C for 3 months.		
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.		
Formulation	Lyophilized from sterile 20mM Tris, 1mM DTT, 10% glycerol, pH 7.5		
	Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants		
	before lyophilization.		
	Please refer to the specific buffer information in the printed manual.		
Reconstitution	Please refer to the printed manual for detailed information.		



KDa	MK	R
116		
66.2		
45.0		
35.0	-	
25.0	- 7	1
18.4 14.4	=	

> 82 % as determined by reducing SDS-PAGE.

Background

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DCX (doublecortin, N-GST chimera)contains 2 doublecortin domains and belongs to the doublecortin family. It is highly expressed in neuronal cells of fetal brain, but not expressed in other fetal tissues. In the adult, it is highly expressed in the brain frontal lobe, but very low expression in other regions of brain, and not detected in heart, placenta, lung, liver, skeletal muscles, kidney and pancreas. DCX is a microtubule-associated protein required for initial steps of neuronal dispersion and cortex lamination during cerebral cortex development. It may act by competing with the putative neuronal protein kinase DCAMKL1 in binding to a target protein. DCX may in that way participate in a signaling pathway that is crucial for neuronal interaction before and during migration, possibly as part of a calcium ion-dependent signal transduction pathway. It may be part with LIS-1 of a overlapping, but distinct, signaling pathways that promote neuronal migration. Defects in DCX are the cause of lissencephaly X-linked type 1 and subcortical band heterotopia X-linked.