

## 5071

## **ELP1/IKBKAP Antibody**



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## For Research Use Only. Not for Use in Diagnostic Procedures.

<b>Applications:</b> W, IP	<b>Reactivity:</b> H Mk	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 150	Source/Isotype: Rabbit	<b>UniProt ID:</b> #O95163	Entrez-Gene Id: 8518
Product Usage Information		<b>Application</b> Western Blotting Immunoprecipitation			<b>Dilution</b> 1:1000 1:50	
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 $\mu$ g/ml BSA and 50% glycerol. Store at – 20°C. Do not aliquot the antibody.				
Specificity/Sensitivity		ELP1/IKBKAP Antibody recognizes endogenous levels of total ELP1/IKBKAP protein.				
Source / Purification		Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues near the carboxy terminus of human ELP1/IKBKAP protein. Antibodies are purified by protein A and peptide affinity chromatography.				
Background		Elongator is a highly conserved transcription elongation factor complex that was first identified in yeast as part of the hyperphosphorylated RNA polymerase II (RNAPII) holoenzyme (1). The Elongator complex consists of 6 subunits, ELP1-6, and has been shown to have acetyltransferase activity (2). The acetylation targets of Elongator include histone H3, which is linked to the transcription elongation function of the complex, and α-tubulin, which is associated with regulation of migration and maturation of projection neurons (3-6).  The ELP1/IKBKAP subunit of Elongator was initially thought to function as a scaffolding protein within the NFκB signaling pathway (7). It contains several WD40 domains and is critical for the formation of the Elongator complex (8). Investigators have determined that mutations in ELP1 are the cause of Familial Dysautonomia (FD), an autosomal recessive neurodegenerative disorder (9). Research studies have demonstrated that defects in Elongator function upon ELP1 mutation affect transcription elongation of several genes involved in cell motility and neuronal development that may be the underlying cause of the neuropathology of FD patients (10,11).				
Background References		<ol> <li>Otero, G. et al. (1999) Mol Cell 3, 109-18.</li> <li>Creppe, C. and Buschbeck, M. (2011) J Biomed Biotechnol 2011, 924898.</li> <li>Wittschieben, B.O. et al. (1999) Mol Cell 4, 123-8.</li> <li>Hawkes, N.A. et al. (2002) J Biol Chem 277, 3047-52.</li> <li>Kim, J.H. et al. (2002) Proc Natl Acad Sci USA 99, 1241-6.</li> <li>Creppe, C. et al. (2009) Cell 136, 551-64.</li> <li>Cohen, L. et al. (1998) Nature 395, 292-6.</li> <li>Frohloff, F. et al. (2003) J Biol Chem 278, 956-61.</li> <li>Anderson, S.L. et al. (2001) Am J Hum Genet 68, 753-8.</li> <li>Close, P. et al. (2006) Mol Cell 22, 521-31.</li> <li>Cohen-Kupiec, R. et al. (2011) PLos One 6, e19147.</li> </ol>				

**Species Reactivity** 

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

**Western Blot Buffer** 

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at  $4^{\circ}$ C with gentle shaking, overnight.

**Applications Key** 

W: Western Blotting IP: Immunoprecipitation

**Cross-Reactivity Key** 

H: Human Mk: Monkey

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