Revision 1

Store at -20C

#23988

PhosphoPlus® ULK1 (Ser757) Antibody Duet

Cell Signaling TECHNOLOGY®

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

UniProt ID:	Entrez-Gene Id:
#O75385	8408

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
ULK1 (D8H5) Rabbit mAb	8054	100 µl	150 kDa	Rabbit IgG
Phospho-ULK1 (Ser757) (D7O6U) Rabbit mAb	14202	100 µl	140-150 kDa	Rabbit IgG

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

PhosphoPlus® Duets from Cell Signaling Technology (CST) provide a means to assess protein activation status. Each Duet contains an activation-state and total protein antibody to your target of interest. These antibodies have been selected from CST's product offering based upon superior performance in specified applications.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Background

Two related serine/threonine kinases, UNC-51-like kinase 1 and 2 (ULK1, ULK2), were discovered as mammalian homologs of the *C. elegans* gene *unc-51* in which mutants exhibited abnormal axonal extension and growth (1-4). Both proteins are widely expressed and contain an amino-terminal kinase domain followed by a central proline/serine rich domain and a highly conserved carboxy-terminal domain. The roles of ULK1 and ULK2 in axon growth have been linked to studies showing that the kinases are localized to neuronal growth cones and are involved in endocytosis of critical growth factors, such as NGF (5). Yeast two-hybrid studies found ULK1/2 associated with modulators of the endocytic pathway, SynGAP, and syntenin (6). Structural similarity of ULK1/2 has also been recognized with the yeast autophagy protein Atg1/Apg1 (7). Knockdown experiments using siRNA demonstrated that ULK1 is essential for autophagy (8), a catabolic process for the degradation of bulk cytoplasmic contents (9,10). It appears that Atg1/ULK1 can act as a convergence point for multiple signals that control autophagy (11), and can bind to several autophagy-related (Atg) proteins, regulating phosphorylation states and protein trafficking (12-16).

AMPK, activated during low nutrient conditions, directly phosphorylates ULK1 at multiple sites including Ser317, Ser555, and Ser777 (17,18). Conversely, mTOR, which is a regulator of cell growth and is an inhibitor of autophagy, phosphorylates ULK1 at Ser757 and disrupts the interaction between ULK1 and AMPK (17).

Background References

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