MBP-TcPINK1

Cat. # UB401

LifeSensors www.lifesensors.com

Background

PINK1 (PTEN-induced putative kinase 1) is a serine/threonine kinase that accumulates at the surface of depolarized mitochondria in response to mitochondrial damage. PINK1 is the only kinase known to phosphorylate ubiquitin at Ser65, as well as the UBL (ubiquitin-like) domain of the E3 ubiquitin ligase Parkin (PARK2) at the same residue. This phosphorylation is essential for recruiting and fully activating Parkin on damaged mitochondria. Activated Parkin ubiquitinates multiple mitochondrial outer membrane proteins, while additional phosphorylation of mono- and polyubiquitin chains by PINK1 generates a dense phospho-ubiquitin signal that promotes mitophagy, the selective autophagic removal of damaged mitochondria. Recombinant human PINK1 efficiently phosphorylates recombinant Parkin and ubiquitin in vitro. Mutations in PINK1 cause a familial form of Parkinson's disease known as autosomal recessive juvenile Parkinson's disease (AR-JP).

Alternate Names

BRPK, PTEN Induced Putative Kinase 1, PARK6, Protein Kinase BRPK

Application(s)

- Phosphorylation of ubiquitin, ubiquitin chains, and Parkin at the Ser65 residue
- Activation of Parkin E3 ligase in *in vitro* assays

Product Specifications

Tag	MBP
Purity	≥90% by SDS-PAGE
Molecular Weight	106 kDa
Quantity	50 µg
Species	*Tribolium castaneum*
Expression System	*E. coli*
Physical State	Liquid
Buffer	50 mM Tris pH 8.0, 150 mM NaCl, 1 mM DTT, 10% glycerol
Solubility	> 3 mg/mL
Storage	-80° C. Avoid repeated freeze/thaw cycles

Product QC

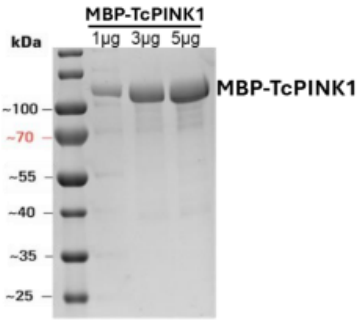

Figure 1. Commassie stained gel of MBP-TcPINK1. Purity is >90%.

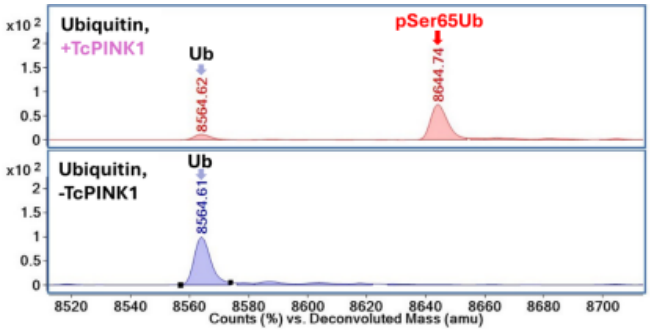

Figure 2. MBP-TcPINK1 efficiently phosphorylate Ser65 on Ubiquitin. Ubiquitin was incubated with 1µM MBP-TcPINK1 (top trace) or without MBP-TcPINK1 (bottom trace) in presence of 5mM ATP and samples were analyzed by LC-MS/MS. pSer65Ub was also confirmed by WB using anti-pSer65 Ub antibody.

References

1. Ge, P., et al., *Molecular neurodegeneration*, 2020;*15*(1), 1-18.
2. Kumar, A., et al., *Elife*, 2017; *6*, e29985.
3. Quinn, P. M., et al., *Acta Neuropathologica Communications*, 2020; *8*(1), 1-20.