Data Sheet (Cat.No.TMAB-09962)

TargetMol

Anti-PANK2 Polyclonal Antibody

Product Details

Ig Type:	IgG
Reactivity:	Human,Mouse (predicted:Rat,Dog,Pig,Cow,Horse)
Molecular Weight:	Theoretical: 57 kDa. Actual: 57-62 kDa.
Purification:	Protein A purified

Applications

Verified Activity:	1. Sample: Lane 1: HepG2 (Human) Cell Lysate at 30 µg Lane 2: U251 (Human) Cell Lysate at 30 µg Primary: Anti-PANK2 (TMAB-09962) at 1/1000 dilution Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution Predicted band size: 62/50 kD Observed band size: 62 kD 2. Sample: Bone (Mouse) Lysate at 40 µg Primary: Anti-PANK2 (TMAB-09962) at 1/1000 dilution Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution Predicted band size: 57 kD Observed band size: 57 kD
Application:	WB
Recommended	WB: 1:500-2000

Properties

Stability & Storage:	Store at 2°C-8°C for 1 month. Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	KLH conjugated synthetic peptide: human PANK2
Antigen Species:	Human
Gene ID:	80025
Uniprot ID:	Q9BZ23

Research Background

Defects in PANK2 are the cause of neurodegeneration with brain iron accumulation type 1 (NBIA1); also known as pantothenate kinase-associated neurodegeneration (PKAN) or Hallervorden-Spatz syndrome (HSS). It is an autosomal recessive neurodegenerative disorder associated with iron accumulation in the brain, primarily in the basal ganglia. Clinical manifestations include progressive muscle spasticity, hyperreflexia, muscle rigidity, dystonia, dysarthria, and intellectual deterioration which progresses to severe dementia over several years. It is clinically classified into classic, atypical, and intermediate phenotypes. Classic forms present with onset in the first decade, rapid progression, loss of independent ambulation within 15 years. Atypical forms have onset in the second decade,

slow progression, maintenance of independent ambulation up to 40 years later. Intermediate forms manifest onset in the first decade with slow progression or onset in the second decade with rapid progression. Patients with early onset tend to also develop pigmentary retinopathy, whereas those with later onset tend to also have speech disorders and psychiatric features. All patients have the 'eye of the tiger' sign on brain MRI.
Defects in PANK2 are the cause of hypoprebetalipoproteinemia, acanthocytosis, retinitis pigmentosa, and pallidal degeneration (HARP). HARP is a rare syndrome with many clinical similarities to NBIA1.

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