

TP53BP1 Polyclonal Antibody

catalog number: E-AB-92405

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

Description

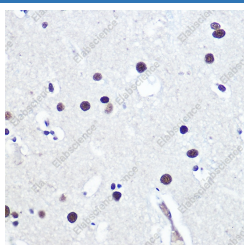
Reactivity	Human
Immunogen	Recombinant fusion protein of human TP53BP1
Host	Rabbit
Isotype	Rabbit IgG
Purification	Affinity purification
Buffer	Phosphate buffered solution, pH 7.4, containing 0.05% stabilizer and 50% glycerol.

Applications

Recommended Dilution

IHC	1:50-1:200
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Data



Immunohistochemistry of paraffin-embedded human brain using 53BP1 Polyclonal Antibody at dilution of 1:100 (40x lens). Perform high pressure antigen retrieval with 10 mM citrate buffer pH 6.0 before commencing with IHC staining protocol.

Preparation & Storage

Storage	Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles.
Shipping	The product is shipped with ice pack, upon receipt, store it immediately at the temperature recommended.

Background

Double-strand break (DSB) repair protein involved in response to DNA damage, telomere dynamics and class-switch recombination (CSR) during antibody genesis. Plays a key role in the repair of double-strand DNA breaks (DSBs) in response to DNA damage by promoting non-homologous end joining (NHEJ)-mediated repair of DSBs and specifically counteracting the function of the homologous recombination (HR) repair protein BRCA1. In response to DSBs, phosphorylation by ATM promotes interaction with RIF1 and dissociation from NUDT16L1/TIRR, leading to recruitment to DSBs sites. Recruited to DSBs sites by recognizing and binding histone H2A monoubiquitinated at 'Lys-15' (H2AK15Ub) and histone H4 dimethylated at 'Lys-20' (H4K20me2), two histone marks that are present at DSBs sites. Required for immunoglobulin class-switch recombination (CSR) during antibody genesis, a process that involves the generation of DNA DSBs. Participates in the repair and the orientation of the broken DNA ends during CSR (By similarity). In contrast, it is not required for classic NHEJ and V(DJ) recombination (By similarity). Promotes NHEJ of dysfunctional telomeres via interaction with PAXIP1.

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