

Anti-Phospho-FOXO3 (Ser253) Antibody (7U72)

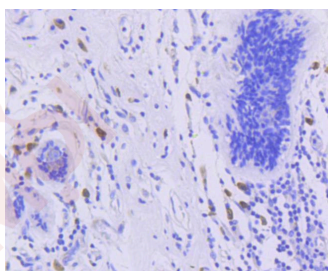
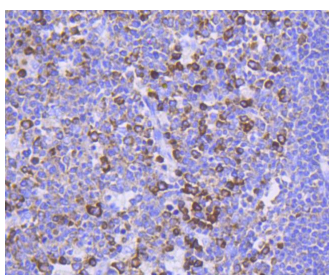
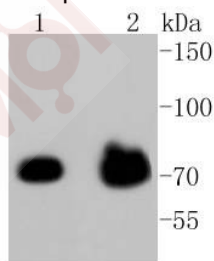
Product Details

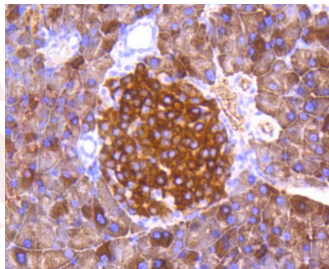
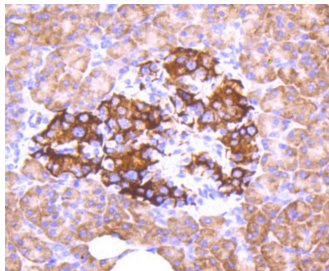
Ig Type:	IgG
Reactivity:	Human,Mouse,Rat
Conjugation:	Unconjugated
Molecular Weight:	Theoretical: 71 kDa.
Clone:	7U72
Purification:	ProA affinity purified

Applications

Verified Activity:

1. Western blot analysis of Phospho-FOXO3a (S253) on different lysates using anti-Phospho-FOXO3a (S253) antibody at 1/1,000 dilution. Positive control: Lane 1: HeLa, Lane 2: PC12.
2. Immunohistochemical analysis of paraffin-embedded human tonsil tissue using anti-Phospho-FOXO3a (S253) antibody. Counter stained with hematoxylin.
3. Immunohistochemical analysis of paraffin-embedded human breast carcinoma tissue using anti-Phospho-FOXO3a (S253) antibody. Counter stained with hematoxylin.
4. Immunohistochemical analysis of paraffin-embedded human pancreas tissue using anti-Phospho-FOXO3a (S253) antibody. Counter stained with hematoxylin.
5. Immunohistochemical analysis of paraffin-embedded mouse pancreas tissue using anti-Phospho-FOXO3a (S253) antibody. Counter stained with hematoxylin.





Application: IHC,WB
Recommended WB: 1:1000-5000; IHC: 1:50-200

Properties

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

Antigen Details

Immunogen: A synthesized phosphopeptide: human FOXO3a around the phosphorylation site of Ser253
Antigen Species: Human
Uniprot ID: O43524
Synonyms: FOXO3 (p-S253);p-FOXO3 (S253);p-FOXO3 (Ser253);FOXO3 (p-Ser253)

Research Background

FKHRL1 (for forkhead in rhabdomyosarcoma) is a member of the FKHR subfamily of forkhead transcription factors. Transcriptional activation of FKHR proteins is regulated by the serine/threonine kinase Akt1, which phosphorylates FKHRL1 at threonine-32 and serine-253. Phosphorylation by Akt1 negatively regulates FKHRL1 by promoting its export from the nucleus. Phosphorylated FKHRL1 associates with 14-3-3 proteins and this complex is retained in the cytoplasm. Growth factor withdrawal stimulates FKHRL1 dephosphorylation and nuclear translocation, leading to FKHR-induced gene-specific transcriptional activation. Within the nucleus, dephosphorylated FKHRL1 triggers apoptosis by inducing the expression of genes that are critical for cell death.

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