Phospho-HSP27 (Ser82) (CB2) rabbit mAb

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Applications	Detection	Clonality	Isotype
Flow Cytometry,WB	Anti-Rabbit IgG	Monoclonal	Rabbit IgGk

Format: Unconjugated

Cross Reactivity: Predicted to work with mouse, rat and other homologues.

Formulation: 1X PBS, 0.02% NaN3, 50% Glycerol, 0.1% BSA

Preparation: Protein A+G

Reactivity: Human, Mouse

Recommended

Usage:

1μg/mL – 0.001μg/mL. It is recommended that the reagent be titrated for optimal

performance for each application. See product image legends for additional

information.

Immunogen: A synthetic phospho-peptide corresponding to residues surrounding Ser82 of

human phospho Hsp27

Description:

Human Hsp27 (also called HspB) is a heat shock protein whose level is upregulated when cells are exposed to conditions that alter protein folding. Hsp27 is an ATP-independent molecular chaperone involved in the protein refolding machinery (1). It functions as a tool to trap and store stress-induced mis-folded polypeptides to avoid their aggregation and to indirectly promote their refolding or proteolytic degradation (2,3). Hsp27 is also known for its control of cytoskeletal organization (4-6) and for its antiapoptotic and antioxidant properties (7-9).

Hsp27 phosphorylation level could be modified by agents such as phorbol ester, calcium ionophores, or stress conditions such as heat shock (10). Human Hsp27 contains three phosphorylatable serine sites (Ser15, Ser78, and Ser82) located in its N-terminal domain whereas murine Hsp27, also called Hsp25, has only two phosphorylated serine sites (Ser15 and Ser86). Several kinases phosphorylate Hsp27 (11) including mitogen-activated protein kinases associated protein kinases (MAPK/MK2,3), MK5-PRAK, PKCγ, and PKD. Moreover, threonine 143 (Thr143) of human Hsp27 appears to be phosphorylated by cGMP-dependent protein kinase. Therefore, Hsp27 often displays complex patterns of phosphorylation since several signaling pathways, including those induced by stress, mitogen, and differentiation, activate these kinases. Hsp27 is involved with cell differentiation (12). It also modifies apoptotic pathways through upstream and downstream of mitochondria by decreasing activity of caspase 9 and caspase 3 (13,14). These events are activated in heat shock, oxidative stress, and endoplasmic reticulum stress-induced apoptosis (15, 16).

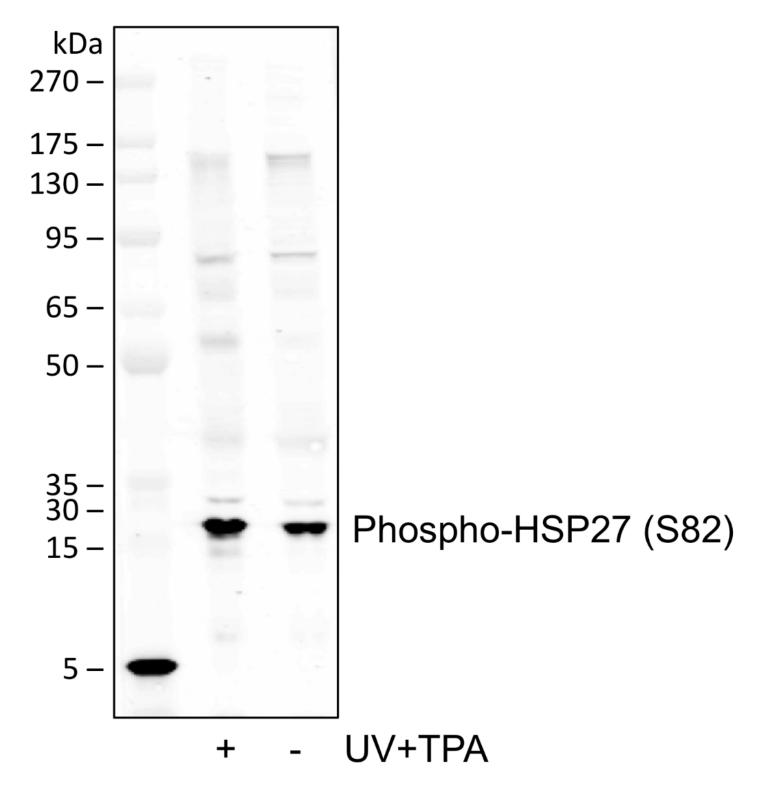
Hsp27 Phosphorylation:

Recently, it has been shown that Sec6 is involved with Hsp27 Ser78, and Ser82 phosphorylation through p38 MAPK promoting cell migration and inhibition of apoptosis induced by TNFa(17). MAPKAPK2 (MK2)/Hsp27 axis have been shown to promote tumorigenesis through phosphorylation of Hsp27 by MK2 (18). Hsp27 (Ser78/82) phosphorylation through PKCa (Thr638), p38MAPK (Thr180/Tyr182) is elevated by chemokine CC-motif ligand 2 (CCL2) in platelets suggesting that PKCa/pMAPK/Hsp27 axis may be target for antiplatelet drugs and the treatment for cardiovascular diseases (19).

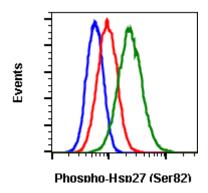
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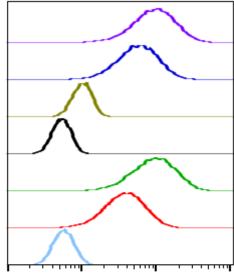




Western blot analysis of HeLa cell extract, untreated or treated with UV plus TPA using Phospho-HSP27 (Ser82) antibody HSP27S82-CB2 at 5 ng/mL. Cat. #2381



Flow cytometric analysis of HeLa cells unstained and untreated cells as as negative control (blue) or untreated (red) or treated with UV and TPA (green) using Phospho-HSP27-CB2 (Ser82) antibody HSP27S82-CB2 at 1.0 ng/mL. Cat. #2381.



\$WELLID	Treatment	Median : BL1-A
CB2 10 NP	UV TPA	9395
CB2 10 NP	CTRL	5673
CB2 10 PP	UV TPA	1016
CB2 10 PP	CTRL	536
CB2 10	UV TPA	9316
CB2 10	CTRL	3783
2'Ab	CTRL	571

Peptide blocking flow cytometric analysis of HeLa cells unstained and untreated as negative control (light blue) or stained and untreated (red) or treated with UV+ plus TPA (green) or untreated and blocked with phospho-peptide (black) or UV plus TPA blocked with phospho peptide (gold) or untreated and blocked with non-phospho peptide (dark blue) or UV plus TPA and blocked with non-phospho peptide (purple) using Phospho-HSP27 (Ser82) antibody HSP27S82-CB2 at 10 µg/mL, Cat#2381.