Product Data Sheet: Phospho-Histone H2A.X (S139)

Cat. No:	ABP-0640
Conjugate:	Unconjugated
Size:	100 ug
Clone:	Poly
Concentration:	1mg/ml
Host:	Rb
Isotype:	IgG
Immunogen:	A phospho_specific peptide corresponding to residues surrounding S139 of human Histone H2AFX
Reactivity:	Hu, Ms, Rt
Applications:	Western Blot: 1:1,000 Immunohistochemistry: 1:50-1:200 Immunofluorescence: 1:50-1:200 Immunoprecipitation: 1:20-1: 50
Molecular:	15kDa
Purification:	Affinity purification
Background:	Histone H2A.X is a variant histone that represents approximately 10% of the total H2A histone proteins in normal human fibroblasts (1). H2A.X is required for checkpoint-mediated cell cycle arrest and DNA repair following double-stranded DNA breaks (1). DNA damage, caused by ionizing radiation, UV-light, or radiomimetic agents, results in rapid phosphorylation of H2A.X at Ser139 by PI3K-like kinases, including ATM, ATR, and DNA-PK (2,3). Within minutes following DNA damage, H2A.X is phosphorylated at Ser139 at sites of DNA damage (4). This very early event in the DNA-damage response is required for recruitment of a multitude of DNA-damage response proteins, including MDC1, NBS1, RAD50, MRE11, 53BP1, and BRCA1 (1). In addition to its role in DNA-damage repair, H2A.X is required for DNA fragmentation during apoptosis and is phosphorylated by various kinases in response to apoptotic signals. H2A.X is phosphorylated at Ser139 by DNA-PK in response to cell death receptor activation, c-Jun N-terminal Kinase (JNK1) in response to UV-A irradiation, and p38 MAPK in response to serum starvation (5-8). H2A.X is constitutively phosphorylated on Tyr142 in undamaged cells by WSTF (Williams-Beuren syndrome transcription factor) (9,10). Upon DNA damage, and concurrent with phosphorylation of Ser139, Tyr142 is dephosphorylated at sites of DNA damage by recruited EYA1 and EYA3 phosphatases (9). While phosphorylation at Ser139 facilitates the recruitment of DNA repair proteins and apoptotic proteins to sites of DNA damage, phosphorylation at Tyr142 appears to determine which set of proteins are recruited. Phosphorylation of H2A.X at Tyr142 inhibits the recruitment of DNA repair proteins and promotes binding of pro-apoptotic factors such as JNK1 (9). Mouse embryonic fibroblasts expressing only mutant H2A.X Y142F, which favors recruitment of DNA repair proteins over apoptotic proteins, show a reduced apoptotic response to ionizing radiation (9). Thus, it appears that the balance of H2A.X Tyr142 phosphorylation and dephosphorylation provides a switch mechanism to determine cell fate after DNA damage.
Form:	liquid
Buffer:	PBS with 0.02% sodium azide, 50% glycerol, pH7.3.
Storage:	Store at -20℃. Avoid freeze / thaw cycles.

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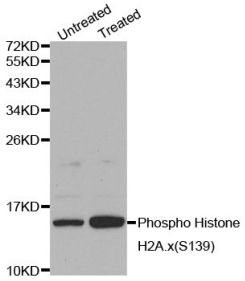
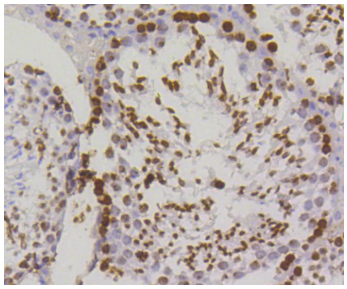


Western blot analysis of extracts of various cells, using Phospho-Histone H2AFX-S139 antibody Secondary antibody: HRP Goat Anti-Rabbit IgG (H+L) at 1:10,000 dilution. Lysates/proteins: 25ug per lane. Blocking buffer: 3% BSA



Immunohistochemistry of paraffin-embedded mouse testis using Phospho-Histone H2AFX-S139 antibody at dilution of 1:100 (40x lens)

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