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Product datasheet for TA392569S

Histone H2A.X (H2AFX) Rabbit Polyclonal Antibody

Product data:

Product Type:	Primary Antibodies
Applications:	WB
Recommended Dilution:	WB: 1:1000~1:2000
Reactivity:	Human
Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Immunogen:	Synthetic phosphopeptide derived from human Histone H2A.X around the phosphorylation site of Serine 139.
Specificity:	Histone H2A.X (Phospho-Ser139) polyclonal antibody detects endogenous levels of Histone H2A.X protein only when phosphorylated at Ser139.
Formulation:	Rabbit IgG, 1mg/ml in PBS with 0.02% sodium azide, 50% glycerol, pH7.4.
Concentration:	1mg/ml
Conjugation:	Unconjugated
Storage:	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze-thaw cycles.
Stability:	1 year
Predicted Protein Size:	~ 17 kDa
Gene Name:	H2A histone family member X
Database Link:	Entrez Gene 3014 Human P16104

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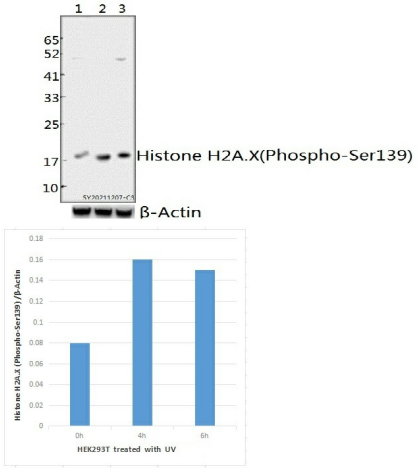
Background: Histone H2A.X is a variant histone that represents approximately 10% of the total H2A histone proteins in normal human fibroblasts. H2A.X is required for checkpoint-mediated cell cycle arrest and DNA repair following double-stranded DNA breaks. DNA damage, caused by ionizing radiation, UV-light, or radiomimetic agents, results in rapid phosphorylation of H2A.X at Ser139 by PI3K-like kinases, including ATM, ATR, and DNA-PK. Within minutes following DNA damage, H2A.X is phosphorylated at Ser139 at sites of DNA damage. 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. 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. H2A.X is constitutively phosphorylated on Tyr142 in undamaged cells by WSTF (Williams-Beuren syndrome transcription factor). Upon DNA damage, and concurrent with phosphorylation of Ser139, Tyr142 is dephosphorylated at sites of DNA damage by recruited EYA1 and EYA3 phosphatases. 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. 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. Thus, it appears that the balance of H2A.X Tyr142 phosphorylation and dephosphorylation provides a switch mechanism to determine cell fate after DNA damage.

Synonyms: H2a/x; H2AFX; H2AX; Histone H2A.X; Histone H2AX

Note: For research use only, not for use in diagnostic procedure.

Product images:



Western blot (WB) analysis of Histone H2A.X (Phospho-Ser139) polyclonal antibody at 1:1000 dilution Lane1:HEK293T whole cell lysate(40ug) Lane2:HEK293T treated with UV for 5 minutes then repair for 4 hours whole cell lysate(40ug) Lane3:HEK293T treated with UV for 5 minutes then repair for 6 hours whole cell lysate(40ug)