

Anti-RAD23B Antibody

Rabbit polyclonal antibody to RAD23B

Catalog # AP59684

Product Information

Application	WB, IP, IF/IC, IHC
Primary Accession	P54727
Other Accession	P54728
Reactivity	Human, Mouse, Rat, Monkey, Bovine
Host	Rabbit
Clonality	Polyclonal
Calculated MW	43171

Additional Information

Gene ID	5887
Other Names	UV excision repair protein RAD23 homolog B; HR23B; hHR23B; XP-C repair-complementing complex 58 kDa protein; p58
Target/Specificity	KLH-conjugated synthetic peptide encompassing a sequence within the N-term region of human RAD23B. The exact sequence is proprietary.
Dilution	WB~~WB (1/500 - 1/1000), IHC (1/100 - 1/200), IF/IC (1/100 - 1/500), IP (1/10 - 1/100) IP~~N/A IF/IC~~N/A IHC~~WB (1/500 - 1/1000), IHC (1/100 - 1/200), IF/IC (1/100 - 1/500), IP (1/10 - 1/100)
Format	Liquid in 0.42% Potassium phosphate, 0.87% Sodium chloride, pH 7.3, 30% glycerol, and 0.09% (W/V) sodium azide.
Storage	Store at -20 °C.Stable for 12 months from date of receipt

Protein Information

Name	RAD23B
Function	Multiubiquitin chain receptor involved in modulation of proteasomal degradation. Binds to polyubiquitin chains. Proposed to be capable to bind simultaneously to the 26S proteasome and to polyubiquitinated substrates and to deliver ubiquitinated proteins to the proteasome. May play a role in endoplasmic reticulum-associated degradation (ERAD) of misfolded glycoproteins by association with PNGase and delivering deglycosylated proteins to the proteasome. The XPC complex is proposed to represent the first factor bound at the sites of DNA damage and together with other core recognition factors, XPA, RPA and the TFIIH complex, is part of the pre-incision (or initial recognition) complex. The XPC complex recognizes a wide spectrum of damaged DNA characterized by distortions of the DNA helix

such as single-stranded loops, mismatched bubbles or single-stranded overhangs. The orientation of XPC complex binding appears to be crucial for inducing a productive NER. XPC complex is proposed to recognize and to interact with unpaired bases on the undamaged DNA strand which is followed by recruitment of the TFIIH complex and subsequent scanning for lesions in the opposite strand in a 5'-to-3' direction by the NER machinery. Cyclobutane pyrimidine dimers (CPDs) which are formed upon UV-induced DNA damage escape detection by the XPC complex due to a low degree of structural perturbation. Instead they are detected by the UV-DDB complex which in turn recruits and cooperates with the XPC complex in the respective DNA repair. In vitro, the XPC:RAD23B dimer is sufficient to initiate NER; it preferentially binds to cisplatin and UV-damaged double-stranded DNA and also binds to a variety of chemically and structurally diverse DNA adducts. XPC:RAD23B contacts DNA both 5' and 3' of a cisplatin lesion with a preference for the 5' side. XPC:RAD23B induces a bend in DNA upon binding. XPC:RAD23B stimulates the activity of DNA glycosylases TDG and SMUG1.

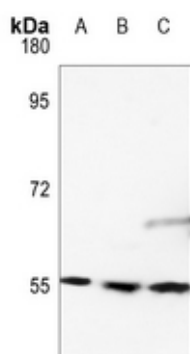
Cellular Location

Nucleus. Cytoplasm. Note=The intracellular distribution is cell cycle dependent. Localized to the nucleus and the cytoplasm during G1 phase. Nuclear levels decrease during S-phase; upon entering mitosis, relocalizes in the cytoplasm without association with chromatin

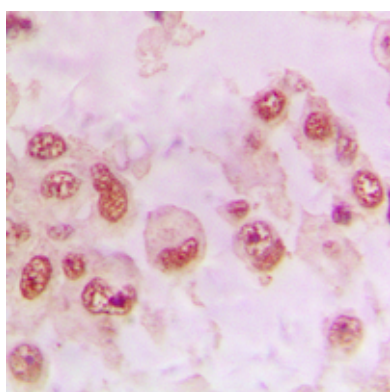
Background

KLH-conjugated synthetic peptide encompassing a sequence within the N-term region of human RAD23B. The exact sequence is proprietary.

Images

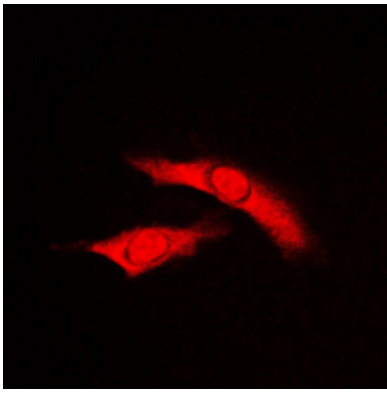


Western blot analysis of RAD23B expression in HEK293T (A), K562 (B), A549 (C) whole cell lysates.



Immunohistochemical analysis of RAD23B staining in human lung cancer formalin fixed paraffin embedded tissue section. The section was pre-treated using heat mediated antigen retrieval with sodium citrate buffer (pH 6.0). The section was then incubated with the antibody at room temperature and detected using an HRP conjugated compact polymer system. DAB was used as the chromogen. The section was then counterstained with haematoxylin and mounted with DPX.

Immunofluorescent analysis of RAD23B staining in HuvEc cells. Formalin-fixed cells were permeabilized with 0.1% Triton X-100 in TBS for 5-10 minutes and blocked with 3%



BSA-PBS for 30 minutes at room temperature. Cells were probed with the primary antibody in 3% BSA-PBS and incubated overnight at 4 °C in a humidified chamber. Cells were washed with PBST and incubated with a DyLight 594-conjugated secondary antibody (red) in PBS at room temperature in the dark.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.