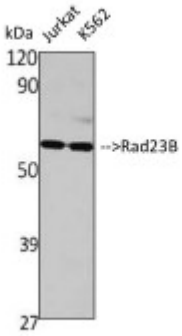
Anti-Rad23B antibody



Description	Rabbit polyclonal to Rad23B.

Model	STJ95327
Host	Rabbit
Reactivity	Human, Mouse, Rat
Applications	ELISA, IF, IHC, WB
Immunogen	Synthesized peptide derived from human Rad23B
Immunogen Region	10-90 aa, N-terminal
Gene ID	5887
Gene Symbol	RAD23B
Dilution range	WB 1:500-1:2000IHC 1:100-1:300IF 1:200-1:1000ELISA 1:20000
Specificity	Rad23B Polyclonal Antibody detects endogenous levels of Rad23B protein.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Note	For Research Use Only (RUO).
Protein Name	UV excision repair protein RAD23 homolog B HR23B hHR23B XP-C repair-complementing complex 58 kDa protein p58
Molecular Weight	55 kDa
Clonality	Polyclonal
Conjugation	Unconjugated

Isotype	IgG
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Concentration	1 mg/ml
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:9813OMIM:600062
Alternative Names	UV excision repair protein RAD23 homolog B HR23B hHR23B XP-C repair-complementing complex 58 kDa protein p58
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.; Involved in global genome nucleotide excision repair (GG-NER) by acting as component of the XPC complex. Cooperatively with CETN2 appears to stabilize XPC. May protect XPC from proteasomal degradation.; 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 esacpe detection by the XPC complex due to a low degree of structural perurbation. 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.
Sequence and Domain Family	The ubiquitin-like domain mediates interaction with ATXN3.
Cellular Localization	Nucleus. Cytoplasm. 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.