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RAD23B Antibody

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP50068

Product Information

Application	WB
Primary Accession	P54727
Reactivity	Human, Mouse, Rat
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, RAD23B
Dilution	WB~~ 1:1000
Format	Rabbit IgG in phosphate buffered saline (without Mg2+ and Ca2+), pH 7.4, 150mM NaCl, 0.09% (W/V) sodium azide and 50% glycerol.
Storage Conditions	-20℃

Protein Information

Name	RAD23B (HGNC:9813)
Function	Multifunctional protein that participates in histone H4K20 demethylation, DNA repair, ubiquitin-dependent protein degradation and transcriptional regulation (PubMed:10488153, PubMed:32209475, PubMed:9372924). Specifically demethylates mono-, di- and trimethylated 'Lys-20' of histone H4 (H4K20me1, H4K20me2, H4K20me3, respectively) into unmethylated forms. Activates the transcription of coding genes by demethylating H4K20me1 and the transcription of repetitive elements by demethylating H4K20me3 (PubMed:32209475). 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 (PubMed:10488153, PubMed:19435460). May play a role in endoplasmic reticulum-associated degradation (ERAD) of misfolded glycoproteins by association with PNGase and delivering deglycosylated proteins to the proteasome (PubMed:15358861). Involved in global genome nucleotide excision repair (GG-NER) by acting as component of the XPC complex, a nucleotide- excision repair complex that is involved in damage

	sensing during global genome nucleotide excision repair. In vitro, the XPC:RAD23B dimer is sufficient to initiate NER; it preferentially binds to cisplatin and UV-damaged double-stranded DNA. Recognizes a wide spectrum of damaged DNA characterized by distortions of the DNA helix including single-stranded loops, mismatched bubbles or single-stranded overhangs. Cooperatively with CETN2 appears to stabilize XPC (PubMed:10873465, PubMed:12815074, PubMed:9372924).
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
Tissue Location	[Isoform 2]: Highly expressed in the testis and in ejaculated spermatozoa.

Background

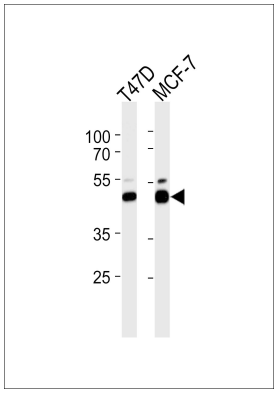
Multiubiquitin chain receptor involved in modulation of proteasomal degradation. Binds to polyubiquitin chains. Proposed to be capable to bind simultaneously to the 26S proteasome and to polyubiquitinated substrates and to deliver ubiquitinated proteins to the proteasome. May play a role in endoplasmic 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 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.

References

Masutani C.,et al.EMBO J. 13:1831-1843(1994).
Huang X.,et al.J. Androl. 25:363-368(2004).
Ota T.,et al.Nat. Genet. 36:40-45(2004).
Humphray S.J.,et al.Nature 429:369-374(2004).
Mural R.J.,et al.Submitted (JUL-2005) to the EMBL/GenBank/DDBJ databases.

Images

Western blot analysis of lysate from T47D,MCF-7 cell line,using RAD23B Antibody(C19671). C19671 was diluted at 1:1000 at each lane. A goat anti-rabbit IgG H&L(HRP) at 1:5000 dilution was used as the secondary antibody.Lysate at 35ug per lane.



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