

# RAD23B Antibody

Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP50068

## Product Information

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Application	WB
Primary Accession	<a href="#">P54727</a>
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	polyclonal
Calculated MW	43171

## Additional Information

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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 Mg <sup>2+</sup> and Ca <sup>2+</sup> ), pH 7.4, 150mM NaCl, 0.09% (W/V) sodium azide and 50% glycerol.
Storage Conditions	-20°C

## Protein Information

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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

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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.

## References

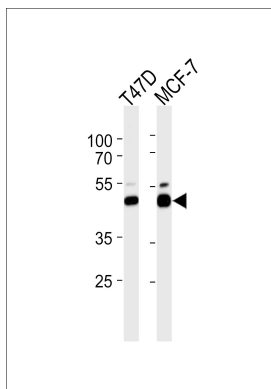
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Masutani C., et al. EMBO J. 13:1831-1843(1994).  
Huang X., et al. J. Biol. Chem. 279:363-368(2004).  
Ota T., et al. Nat. Genet. 36:40-45(2004).  
Humphray S.J., et al. Nature 429:369-374(2004).  
Mural R.J., et al. Submitted (JUL-2005) to the EMBL/GenBank/DBJ databases.

## Images

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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 35 µg per lane.



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