

RAD23B / HR23B Antibody (aa27-76)
Rabbit Polyclonal Antibody
Catalog # ALS17002**Specification**

RAD23B / HR23B Antibody (aa27-76) - Product Information

Application	WB, IHC-P, E
Primary Accession	P54727
Other Accession	5887
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	43171
Dilution	WB~~1:1000 IHC-P~~N/A E~~N/A

RAD23B / HR23B Antibody (aa27-76) - Additional Information**Gene ID** 5887**Other Names**

RAD23B, HR23B, p58, RAD23, yeast homolog of, B, HHR23B

Target/Specificity

RAD23B Antibodyantibody detects endogenous levels of RAD23B.

Reconstitution & Storage

PBS, pH 7.4, 150 mM sodium chloride, 0.02% sodium azide, 50% glycerol. Store at -20°C.

Precautions

RAD23B / HR23B Antibody (aa27-76) is for research use only and not for use in diagnostic or therapeutic procedures.

RAD23B / HR23B Antibody (aa27-76) - 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

Volume

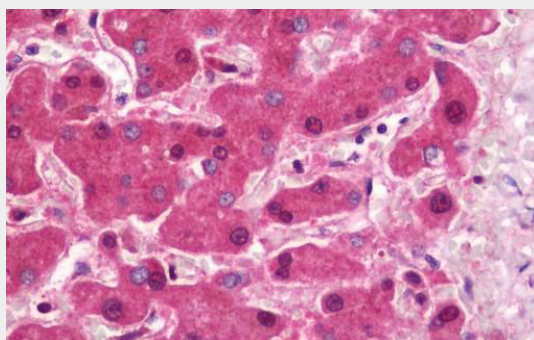
50 µl

RAD23B / HR23B Antibody (aa27-76) - Protocols

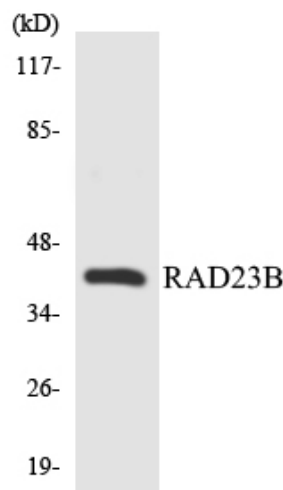
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

RAD23B / HR23B Antibody (aa27-76) - Images



Anti-RAD23B / HR23B antibody IHC staining of human liver.



Western blot of the lysates from Jurkat cells using RAD23B antibody.

RAD23B / HR23B Antibody (aa27-76) - 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 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.

RAD23B / HR23B Antibody (aa27-76) - 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.