

RAD23B / HR23B Antibody (aa163-176)
Goat Polyclonal Antibody
Catalog # ALS11350**Specification**

RAD23B / HR23B Antibody (aa163-176) - Product Information

Application	WB, IHC-P, IF, E
Primary Accession	P54727
Reactivity	Human
Host	Goat
Clonality	Polyclonal
Calculated MW	43kDa KDa
Dilution	WB~~1:1000 IHC-P~~N/A IF~~1:50~200 E~~N/A

RAD23B / HR23B Antibody (aa163-176) - 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

Target/Specificity

aa 163-176 of human HR23B protein.

Reconstitution & Storage

Long term: -20°C; Short term: +4°C. Avoid repeat freeze-thaw cycles.

Precautions

RAD23B / HR23B Antibody (aa163-176) is for research use only and not for use in diagnostic or therapeutic procedures.

RAD23B / HR23B Antibody (aa163-176) - 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

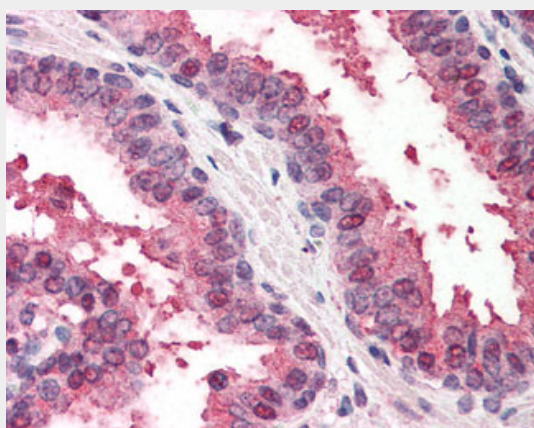
Nucleus. Cytoplasm. Note=The intracellular distribution is cell cycle dependent. Localized to the nucleus and the cytoplasm during G1 phase. Nuclear levels decrease during S-phase; upon entering mitosis, relocates in the cytoplasm without association with chromatin

RAD23B / HR23B Antibody (aa163-176) - 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 (aa163-176) - Images



Anti-RAD23B antibody IHC of human prostate.

RAD23B / HR23B Antibody (aa163-176) - Background

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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 (aa163-176) - References

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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).
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