

hHR23b Antibody

Purified Mouse Monoclonal Antibody (Mab)

Catalog # AP52817

Product Information

Application	WB, IHC, IHC-P, IHC-F, IF, ICC
Primary Accession	P54727
Reactivity	Human, Mouse, Rat
Host	Mouse
Clonality	Monoclonal
Isotype	IgG2b
Calculated MW	43171

Additional Information

Gene ID	5887
Other Names	hHR 23b;hHR23B;HR 23B;HR23B;mHR 23B;mHR23B;P58;RAD 23B;RAD23 (S. cerevisiae) homolog B; RAD23 homolog B (S. cerevisiae);RAD23 homolog B;RAD23 yeast homolog of B;RAD23B; RD23B_HUMAN;UV excision repair protein RAD23 homolog B;XP C repair complementing complex 58 kDa;XP C repair complementing complex 58 kDa protein;XP C repair complementing protein;XP-C repair-complementing complex 58 kDa protein;XPC repair complementing complex 58 kDa;XPC repair complementing complex 58 kDa protein;XPC repair complementing protein.
Dilution	WB~~1:1000 IHC~~1:100 IHC-P~~N/A IHC-F~~N/A IF~~1:50~200 ICC~~1:100
Format	Purified mouse monoclonal antibody in PBS(pH 7.4) containing with 0.09% (W/V) sodium azide and 50% glycerol.
Storage	Store at -20 °C.Stable for 12 months from date of receipt

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

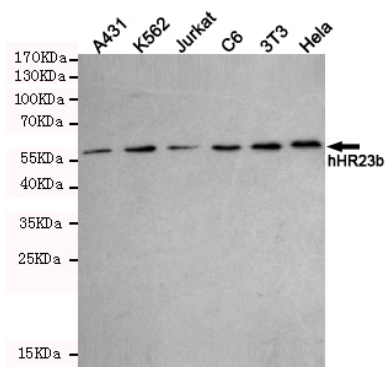
Background

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.

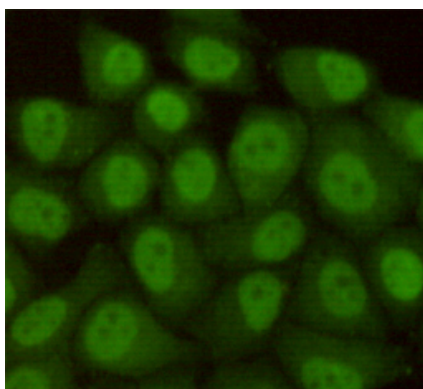
References

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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.

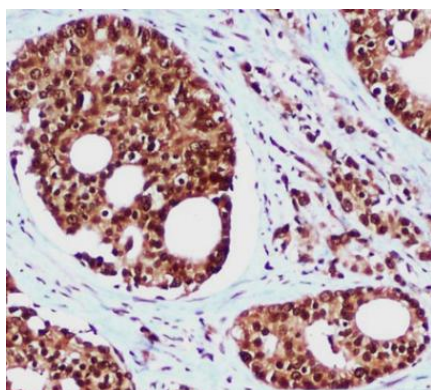
Images



Western blot detection of hHR23b in A431, K562, Jurkat, C6, 3T3 and HeLa cell lysates using hHR23b mouse mAb (1:1000 diluted). Predicted band size: 58 kDa. Observed band size: 58 kDa. Exposure time: 5 min.



Immunocytochemistry staining of HeLa cells fixed with 4% Paraformaldehyde and using anti-hHR23b antibody (dilution 1:100).



Immunohistochemical analysis of paraffin-embedded Prostate Cancer using hHR23b mouse mAb (1/100 dilution). Antigen retrieval was performed by pressure cooking in citrate buffer (pH 6.0).

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