SANTA CRUZ BIOTECHNOLOGY, INC.

XPA (1-273): sc-4331 WB

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Xeroderma pigmentosum (XP) is an autosomal recessive disorder characterized by a genetic predisposition to sunlight-induced skin cancer due to deficiencies in the DNA repair enzymes. The most frequent mutations are found in the XP genes of group A through G and group V, which encode nucleotide excision repair proteins. Nucleotide excision repair (NER) is the normal cellular response to DNA damage induced by UV irradiation and is disrupted in patients with XP. Xeroderma pigmentosum group A (XPA) is an essential NER factor that coordinates the collection of a preincision complex during the processing of DNA damage. XPA may also have a role in the repair of oxidized DNA bases. XPA is sensitive not only to the structure of the DNA double helix, but also to bulky groups incorporated into DNA. XPA forms a homodimer in the absence of DNA, but binds to DNA in both monomeric and dimeric forms. The dimerically bound XPA is much more efficient, so cells probably regulate XPA activity in a concentration-dependent manner. XPA deficient organisms cannot repair UV-induced DNA damage and thus acquire skin cancers by UV irradiation very easily.

REFERENCES

1. Tateishi, S., et al. 1995. Separation of protein factors that correct the defects in the seven complementation groups of xeroderma pigmentosum cells. J. Biochem. 118: 819-824.
2. Nakane, H., et al. 1995. High incidence of ultraviolet-B-or chemical-carcinogen-induced skin tumours in mice lacking the xeroderma pigmentosum group A gene. Nature 377: 165-168.
3. Li, L., et al. 1995. Mutations in XPA that prevent association with ERCC1 are defective in nucleotide excision repair. Mol. Cell. Biol. 15: 1993-1998.
4. Kuraoka, I., et al. 1996. Identification of a damaged-DNA binding domain of the XPA protein. Mut. Res. 362: 87-95.
5. Cappelli, E., et al. 1999. The DNA helicases acting in nucleotide excision repair, XPD, CSB and XPB, are not required for PCNA-dependent repair of abasic sites. Eur. J. Biochem. 259: 325-330.
6. Riou, L., et al. 1999. The relative expression of mutated XPB genes results in xeroderma pigmentosum/Cockayne's syndrome or trichothiodystrophy cellular phenotypes. Hum. Mol. Genet. 8: 1125-1133.
7. Constantinou, A., et al. 1999. Conserved residues of human XPG protein important for nuclease activity and function in nucleotide excision repair. J. Biol. Chem. 274: 5637-5648.
8. Masutani, C., et al. 1999. The XPV (xeroderma pigmentosum variant) gene encodes human DNA polymerase eta. Nature 399: 700-704.

CHROMOSOMAL LOCATION

Genetic locus: XPA (human) mapping to 9q22.33; Xpa (mouse) mapping to 4 B1.

SOURCE

XPA (1-273) is expressed in *E. coli* as a 40 kDa tagged fusion protein corresponding to amino acids 1-273 of XPA of human origin.

PRODUCT

XPA (1-273) is purified from bacterial lysates (>98%) by column chromatography; supplied as 10 µg in 0.1 ml SDS-PAGE loading buffer.

APPLICATIONS

XPA (1-273) is suitable as a Western blotting control for sc-853, sc-28353, sc-48711, sc-48712, sc-48713, sc-48715, sc-53467, sc-53468, sc-56497, sc-56813, sc-73272 and sc-73273.

Molecular Weight of XPA: 40 kDa.

SELECT PRODUCT CITATIONS

1. Guthrie, O.W., et al. 2008. Cisplatin induces cytoplasmic to nuclear translocation of nucleotide excision repair factors among spiral ganglion neurons. Hear. Res. 239: 79-91.

STORAGE

Store at -20° C; stable for one year from the date of shipment.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.