

# XPA (B-1): sc-28353

## 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.

## CHROMOSOMAL LOCATION

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

## SOURCE

XPA (B-1) is a mouse monoclonal antibody raised against amino acids 1-273 representing full length XPA of human origin.

## PRODUCT

Each vial contains 200 µg IgG<sub>1</sub> kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

XPA (B-1) is available conjugated to agarose (sc-28353 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-28353 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-28353 PE), fluorescein (sc-28353 FITC), Alexa Fluor® 488 (sc-28353 AF488), Alexa Fluor® 546 (sc-28353 AF546), Alexa Fluor® 594 (sc-28353 AF594) or Alexa Fluor® 647 (sc-28353 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-28353 AF680) or Alexa Fluor® 790 (sc-28353 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

## APPLICATIONS

XPA (B-1) is recommended for detection of XPA of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

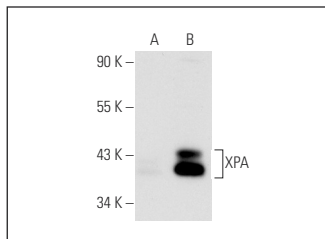
Suitable for use as control antibody for XPA siRNA (h): sc-36853, XPA siRNA (m): sc-36854, XPA shRNA Plasmid (h): sc-36853-SH, XPA shRNA Plasmid (m): sc-36854-SH, XPA shRNA (h) Lentiviral Particles: sc-36853-V and XPA shRNA (m) Lentiviral Particles: sc-36854-V.

Molecular Weight of XPA: 40 kDa.

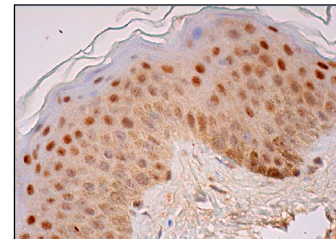
## STORAGE

Store at 4° C, **\*\*DO NOT FREEZE\*\***. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

## DATA



XPA (B-1): sc-28353. Western blot analysis of XPA expression in non-transfected: sc-117752 (A) and mouse XPA transfected: sc-126255 (B) 293T whole cell lysates.



XPA (B-1): sc-28353. Immunoperoxidase staining of formalin fixed, paraffin-embedded human skin tissue showing nuclear staining of fibroblasts, keratinocytes, Langerhans cells and melanocytes.

## SELECT PRODUCT CITATIONS

- Lu, S.Y., et al. 2006. Ripe areca nut extract induces G<sub>1</sub> phase arrests and senescence-associated phenotypes in normal human oral keratinocyte. *Carcinogenesis* 27: 1273-1284.
- Dong, L., et al. 2010. Melanocyte-stimulating hormone directly enhances UV-induced DNA repair in keratinocytes by a xeroderma pigmentosum group A-dependent mechanism. *Cancer Res.* 70: 3547-3556.
- Fong, Y.W., et al. 2011. A DNA repair complex functions as an oct4/sox2 coactivator in embryonic stem cells. *Cell* 147: 120-131.
- Fishelevich, R., et al. 2011. Imiquimod-induced TLR7 signaling enhances repair of DNA damage induced by ultraviolet light in bone marrow-derived cells. *J. Immunol.* 187: 1664-1673.
- Kemp, M.G., et al. 2012. Mechanism of release and fate of excised oligonucleotides during nucleotide excision repair. *J. Biol. Chem.* 287: 22889-22899.
- Hu, J., et al. 2013. Nucleotide excision repair in human cells: fate of the excised oligonucleotide carrying DNA damage *in vivo*. *J. Biol. Chem.* 288: 20918-20926.
- Yüce, Ö. and West, S.C. 2013. Senataxin, defective in the neurodegenerative disorder ataxia with oculomotor apraxia 2, lies at the interface of transcription and the DNA damage response. *Mol. Cell. Biol.* 33: 406-417.
- Gao, C., et al. 2016. A functional polymorphism (rs10817938) in the XPA promoter region is associated with poor prognosis of oral squamous cell carcinoma in a Chinese Han population. *PLoS ONE* 11: e0160801.

## RESEARCH USE

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

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