SANTA CRUZ BIOTECHNOLOGY, INC.

RelB (N-17): sc-30887

BACKGROUND

The NFκB transcription factor was originally identified as a protein complex consisting of a DNA binding subunit and an associated protein. The DNA binding subunit is functionally related to c-Rel p75 and Rel B p68. The p50 subunit was initially believed to be a functionally unique protein derived from the amino terminus of a precursor designated p105. A second protein designated p52 (previously referred to as p49) has been identified that can act as an alternative NFκB subunit. Rel B does not bind with high affinity to NFkB sites, but heterodimers between Rel B and p50 bind with an affinity comparable to that of p50 NFκB homodimers. However, Rel B/p50 heterodimers, in contrast to NFκB heterodimers, transactivates transcription of promotors containing κB binding sites.

CHROMOSOMAL LOCATION

Genetic locus: RELB (human) mapping to 19q13.32; Relb (mouse) mapping to 7 A3.

SOURCE

RelB (N-17) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of RelB of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-30887 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

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

APPLICATIONS

RelB (N-17) is recommended for detection of RelB of mouse, rat and human origin by Western Blotting (starting dilution 1:200, 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) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

RelB (N-17) is also recommended for detection of RelB in additional species, including bovine.

Suitable for use as control antibody for RelB siRNA (h): sc-36402, RelB siRNA (m): sc-36403, RelB shRNA Plasmid (h): sc-36402-SH, RelB shRNA Plasmid (m): sc-36403-SH, RelB shRNA (h) Lentiviral Particles: sc-36402-V and RelB shRNA (m) Lentiviral Particles: sc-36403-V.

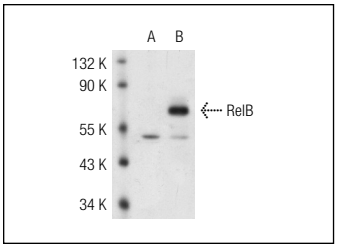
Molecular Weight of RelB: 68 kDa.

Positive Controls: RelB (h): 293T Lysate: sc-114651, NIH/3T3 whole cell lysate: sc-2210 or KNRK nuclear extract: sc-2141.

RESEARCH USE

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

DATA



RelB (N-17): sc-30887. Western blot analysis of RelB expression in non-transfected: sc-117752 (**A**) and human RelB transfected: sc-114651 (**B**) 293T whole cell lysates.

SELECT PRODUCT CITATIONS

1. Reiley, W.W., et al. 2007. Deubiquitinating enzyme CYLD negatively regulates the ubiquitin-dependent kinase Tak1 and prevents abnormal T cell responses. J. Exp. Med. 204: 1475-1485.
2. Naujokat, C., et al. 2007. Proteasomal chymotrypsin-like peptidase activity is required for essential functions of human monocyte-derived dendritic cells. Immunology 120: 120-132.
3. Jaskoll, T., et al. 2008. Cytomegalovirus induces abnormal chondrogenesis and osteogenesis during embryonic mandibular development. BMC Dev. Biol. 8: 33.
4. Jaskoll, T., et al. 2008. Cytomegalovirus inhibition of embryonic mouse tooth development: a model of the human amelogenesis imperfecta phenocopy. Arch. Oral Biol. 53: 405-415.
5. Jonassen, J.A., et al. 2008. Deletion of IFT20 in the mouse kidney causes misorientation of the mitotic spindle and cystic kidney disease. J. Cell Biol. 183: 377-384.
6. Jonassen, J.A., et al. 2012. Disruption of IFT complex A causes cystic kidneys without mitotic spindle misorientation. J. Am. Soc. Nephrol. 23: 641-651.

PROTOCOLS

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