

# LIN-31 (cC-16): sc-9284

## BACKGROUND

Cell proliferation and development are carefully controlled in *C. elegans*, with each cell following a nearly invariant pattern of differentiation. Vulval development in particular provides a useful model for studying how cell fate is determined. In addition to cell signaling pathways such as Notch and Ras pathways, the establishment of cell polarity and the asymmetric distribution of certain receptors are also critical for proper cell fate determination. In *C. elegans*, activated Ras initiates a cascade of sequential phosphorylation events, including the protein kinases Raf, MEK, and MAP kinase. The Let-60 Ras-mediated signal transduction pathway controls vulval induction. LIN-45, a member of the Raf family of serine/threonine kinases, and LIN-31, an HNF-3 homologue, act downstream of the Ras protein as necessary components for vulval differentiation. LIN-1 contains an ETS domain and is thought to be a substrate for the ERK subfamily of MAP kinases. The *C. elegans* proteins MEK-2, a MAP kinase kinase, and SUR-2 also function in the Let-60 Ras-mediated vulval induction.

## REFERENCES

- Han, M., Golden, A., Han, Y. and Sternberg, P.W. 1993. *C. elegans* lin-45 Raf gene participates in let-60 Ras-stimulated vulval differentiation. *Nature* 363: 133-140.
- Eisenmann, D.M. and Kim, S.K. 1994. Signal transduction and cell fate specification during *Caenorhabditis elegans* vulval development. *Curr. Opin. Genet. Dev.* 4: 508-516.
- Wu, Y., Han, M. and Guan, K.L. 1995. MEK-2, a *Caenorhabditis elegans* MAP kinase kinase, functions in Ras-mediated vulval induction and other developmental events. *Genes Dev.* 9: 742-755.
- Singh, N. and Han, M. 1995. Sur-2, a novel gene, functions late in the let-60 Ras-mediated signaling pathway during *Caenorhabditis elegans* vulval induction. *Genes Dev.* 9: 2251-2265.
- Sommer, R.J. and Sternberg, P.W. 1996. Evolution of nematode vulval fate patterning. *Dev. Biol.* 173: 396-407.
- Sundaram, M. and Han, M. 1996. Control and integration of cell signaling pathways during *C. elegans* vulval development. *Bioessays* 18: 473-480.
- Kornfeld, K. 1997. Vulval development in *Caenorhabditis elegans*. *Trends Genet.* 13: 55-61.
- Jacobs, D., Beitel, G.J., Clark, S.G., Horvitz, H.R. and Kornfeld, K. 1998. Gain-of-function mutations in the *Caenorhabditis elegans* lin-1 ETS gene identify a C-terminal regulatory domain phosphorylated by ERK MAP kinase. *Genetics* 149: 1809-1822.

## SOURCE

LIN-31 (cC-16) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the C-terminus of LIN-31 of *Caenorhabditis elegans* origin.

## STORAGE

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

## 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-9284 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

## APPLICATIONS

LIN-31 (cC-16) is recommended for detection of LIN-31 of *Caenorhabditis elegans* origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), 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).

## RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) Immunofluorescence: use donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

## SELECT PRODUCT CITATIONS

- He, H., et al. 2009. Pot1b deletion and telomerase haploinsufficiency in mice initiate an ATR-dependent DNA damage response and elicit phenotypes resembling dyskeratosis congenita. *Mol. Cell. Biol.* 29: 229-240.

## RESEARCH USE

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

## PROTOCOLS

See our web site at [www.scbt.com](http://www.scbt.com) or our catalog for detailed protocols and support products.