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ERAS Antibody (N-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP1470a

Product Information

Application	WB, IHC-P, IF, FC, E
Primary Accession	Q7Z444
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB12690
Calculated MW	25287
Antigen Region	13-42

Additional Information

Gene ID	3266
Other Names	GTPase ERas, E-Ras, Embryonic stem cell-expressed Ras, ERAS, HRAS2, HRASP
Target/Specificity	This ERAS antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 13-42 amino acids from the N-terminal region of human ERAS.
Dilution	WB~~1:1000 IHC-P~~1:100~500 IF~~1:10~50 FC~~1:10~50 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	ERAS Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	ERAS
Synonyms	HRAS2, HRASP
Function	Ras proteins bind GDP/GTP and possess intrinsic GTPase activity. Plays an important role in the tumor-like growth properties of embryonic stem cells

(By similarity).

Cellular Location Cell membrane; Lipid-anchor; Cytoplasmic side

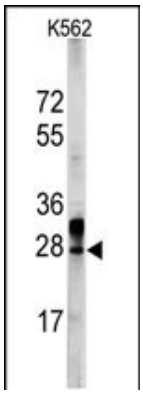
Background

Ras proteins bind GDP/GTP and possess intrinsic GTPase activity. Point mutations of several amino acids of human RAS, including gly12, ala59, or glu63, render the protein constitutively active. Embryonic stem cell-expressed Ras (ERAS) has serine, alanine, and asparagine at the positions corresponding to gly12, ala59, and glu63 of human RAS, suggesting that it is constitutively active.The PI3K (phosphoinositide 3-kinase) pathway is important for proliferation, survival and maintenance of pluripotency in ES cells. The PI3K pathway is activated by growth factors and cytokines including insulin and leukaemia inhibitory factor. In addition to these exogenous factors, the PI3K pathway is endogenously activated by the constitutively active Ras family protein ERas (ES cell-expressed Ras). ERas null ES cells maintained pluripotency but show significantly reduced growth and tumorigenicity, which can be rescued by expression of ERas cDNA or by activated phosphatidylinositol 3-hydroxykinase. The transforming oncogene ERAS appears to be important in the tumor-like growth properties of ES cells.

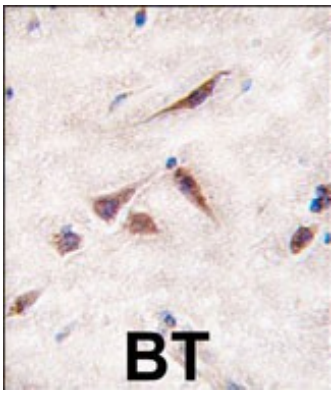
References

Kameda,T., Stem Cells 23 (10), 1535-1540 (2005)
Takahashi,K., Nature 423 (6939), 541-545 (2003)
Miyoshi,J., Nucleic Acids Res. 12 (4), 1821-1828 (1984)

Images

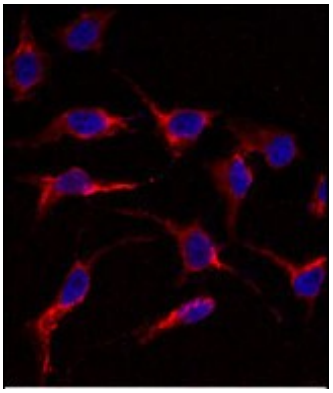


Western blot analysis of ERAS Antibody (N-term) (Cat.#AP1470a) in K562 cell line lysates (35ug/lane). ERAS (arrow) was detected using the purified Pab.

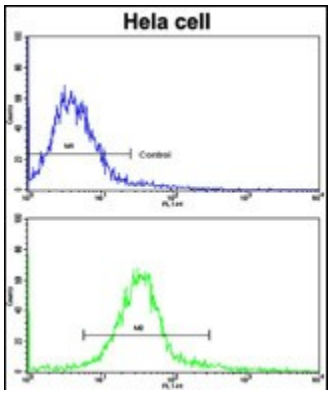


Formalin-fixed and paraffin-embedded human brain tissue reacted with ERAS antibody (N-term) (A28)(Cat.#AP1470a), which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated.

Immunofluorescence analysis of anti-ERAS Antibody (N-term) in HeLa cells. 0.025 mg/ml primary antibody was followed by Alexa-Fluor-546-conjugated donkey anti-rabbit lgG (H+L). Alexa-Fluor-546 emits orange



fluorescence. Blue counterstaining is DAPI.



Flow cytometric analysis of hela cells using ERAS Antibody (N-term)(bottom histogram) compared to a negative control cell (top histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

Citations

- ERas regulates cell proliferation and epithelial-mesenchymal transition by affecting Erk/Akt signaling pathway in pancreatic cancer
- BRAF inhibitor resistance mediated by the AKT pathway in an oncogenic BRAF mouse melanoma model.
- Insertional mutagenesis in a HER2-positive breast cancer model reveals ERAS as a driver of cancer and therapy resistance.

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