

p-4E-BP1 (62.Ser 65): sc-293124

BACKGROUND

The multisubunit eukaryotic translation initiation factor (eIF) 4F recruits 40S ribosomal subunits to the 5' end of mRNA. The eIF4F subunit eIF4E interacts directly with the mRNA 5' cap structure. Assembly of the eIF4F complex is inhibited by a family of repressor polypeptides, the eIF4E-binding proteins (4E-BPs). 4E-BP1 (also known as PHAS-1) normally binds eIF4E, inhibiting cap-dependent translation. Hyperphosphorylation of 4E-BP1 disrupts this binding, activating cap-dependent translation. The PI 3-kinase/Akt pathway and the FRAP/mTOR kinase regulate 4E-BP1. 4E-BP1 is phosphorylated *in vivo* on multiple residues and phosphorylation by FRAP/mTOR on Threonine 37 and Threonine 46 of human 4E-BP1 may prime it for subsequent phosphorylation at sites including Serine 65 and Threonine 70. The corresponding rat residues include Threonine 36, Threonine 45, Serine 64 and Threonine 69. *In vitro*, 4E-BP1 is also phosphorylated by ataxia telangiectasia (Atm) at human Serine 112 (rat Serine 111) in response to an increase in Insulin levels.

CHROMOSOMAL LOCATION

Genetic locus: EIF4EBP1 (human) mapping to 8p11.23; Eif4ebp1 (mouse) mapping to 8 A2.

SOURCE

p-4E-BP1 (62.Ser 65) is a mouse monoclonal antibody raised against a short amino acid sequence containing Ser 65 phosphorylated 4E-BP1 of human origin.

PRODUCT

Each vial contains 200 µg IgG_{2b} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

p-4E-BP1 (62.Ser 65) is available conjugated to agarose (sc-293124 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; and to HRP (sc-293124 HRP), 200 µg/ml, for WB, IHC(P) and ELISA.

APPLICATIONS

p-4E-BP1 (62.Ser 65) is recommended for detection of Ser 65 phosphorylated 4E-BP1 of human origin, and correspondingly Ser 64 phosphorylated 4E-BP1 of mouse and rat 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)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for 4E-BP1 siRNA (h): sc-29594, 4E-BP1 siRNA (m): sc-29595, 4E-BP1 shRNA Plasmid (h): sc-29594-SH, 4E-BP1 shRNA Plasmid (m): sc-29595-SH, 4E-BP1 shRNA (h) Lentiviral Particles: sc-29594-V and 4E-BP1 shRNA (m) Lentiviral Particles: sc-29595-V.

Molecular Weight of p-4E-BP1: 21 kDa.

Positive Controls: 4E-BP1 (h): 293T Lysate: sc-116590, HeLa whole cell lysate: sc-2200 or K-562 whole cell lysate: sc-2203.

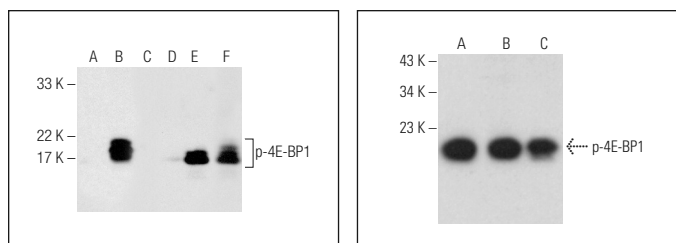
STORAGE

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

RESEARCH USE

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

DATA



Western blot analysis of 4E-BP1 phosphorylation in non-transfected: sc-117752 (A,D), untreated human 4E-BP1 transfected: sc-116590 (B,E) and lambda protein phosphatase (sc-200312A) treated human 4E-BP1 transfected: sc-116590 (C,F) 293T whole cell lysates. Antibodies tested include p-4E-BP1 (62.Ser 65): sc-293124 (A,B,C) and 4E-BP1 (11G12C11): sc-81149 (D,E,F).

p-4E-BP1 (62.Ser 65): sc-293124. Western blot analysis of 4E-BP1 phosphorylation in HeLa (A), K-562 (B) and HEL 92.1.7 (C) whole cell lysates.

SELECT PRODUCT CITATIONS

- Wang, X., et al. 2017. Sestrin2 and sestrin3 suppress NK-92 cell-mediated cytotoxic activity on ovarian cancer cells through AMPK and mTORC1 signaling. *Oncotarget* 8: 90132-90143.
- Lee, M.K., et al. 2018. *Pyropia yezoensis* protein supplementation prevents dexamethasone-induced muscle atrophy in C57BL/6 mice. *Mar. Drugs* 16: 328.
- Shokrzadeh, N., et al. 2019. Calcitonin administration improves endometrial receptivity via regulation of LIF, Muc-1 and microRNA Let-7a in mice. *J. Cell. Physiol.* 234: 12989-13000.
- Shariati, M.B.H., et al. 2019. Administration of dexamethasone disrupts endometrial receptivity by alteration of expression of miRNA 223, 200a, LIF, Muc1, SGK1, and ENaC via the ERK1/2-mTOR pathway. *J. Cell. Physiol.* 234: 19629-19639.
- Hesam Shariati, M.B., et al. 2019. The effect of fludrocortisone on the uterine receptivity partially mediated by ERK1/2-mTOR pathway. *J. Cell. Physiol.* 234: 20098-20110.
- Lv, X.F., et al. 2020. TMEM16A ameliorates vascular remodeling by suppressing autophagy via inhibiting Bcl-2-p62 complex formation. *Theranostics* 10: 3980-3993.
- Sousa, M.I., et al. 2020. Metabolic characterization of a paused-like pluripotent state. *Biochim. Biophys. Acta Gen. Subj.* 1864: 129612.
- Chen, Z., et al. 2020. A high-throughput drug combination screen identifies an anti-glioma synergism between TH588 and PI3K inhibitors. *Cancer Cell Int.* 20: 337.

PROTOCOLS

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