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## CREB-H (D10D8) Rabbit mAb

For Research Use Only. Not for Use in Diagnostic Procedures.

<b>Applications:</b> W, IP	<b>Reactivity:</b> H M R Mk	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 75	<b>Source/Isotype:</b> Rabbit IgG	<b>UniProt ID:</b> #Q68CJ9	<b>Entrez-Gene Id:</b> 84699
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### Product Usage Information

#### Application

Western Blotting  
Immunoprecipitation

#### Dilution

1:1000  
1:50

### Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

### Specificity/Sensitivity

CREB-H (D10D8) Rabbit mAb recognizes endogenous levels of total and cleaved CREB-H proteins.

### Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Leu93 of human CREB-H protein.

### Background

CREB-H belongs to the bZIP transmembrane transcription factor family that activates transcription by binding to cAMP responsive elements (1,2). CREB-H interacts with ATF-6 and binds to conserved elements in the APR genes to synergistically activate transcription (2-4). Evidence suggests that CREB-H is activated by cleavage upon ER stress, inflammatory stimuli (2-5), and metabolic stress (5,6). Known chemical activators of ER stress, such as tunicamycin and thapsigargin, have been shown to induce cleavage of the full-length 75 kDa from of CREB-H, releasing the 50 kDa N-terminal fragment, which translocates to the nucleus (1-4). Upon ER stress, the transmembrane domain of CREB-H is cleaved by Golgi proteases, which allows subsequent translocation to the nucleus. Liberated nuclear CREB-H plays a crucial role in the acute systemic inflammatory response by activating transcription of genes that encode serum amyloid P-component (SAP) and C-reactive protein (CRP) (2,3). Recent studies suggest that activated CREB-H functions as a crucial metabolic regulator of hepatic lipogenesis, fatty acid (FA) oxidation, and lipolysis (5,6). Metabolic stress inducers, such as saturated fatty acids, insulin, and atherogenic high-fat diets have been shown to activate CREB-H in the liver (5-7).

### Background References

- Omori, Y. et al. (2001) *Nucleic Acids Res* 29, 2154-62.
- Chin, K.T. et al. (2005) *Nucleic Acids Res* 33, 1859-73.
- Zhang, K. et al. (2006) *Cell* 124, 587-99.
- DeBose-Boyd, R.A. et al. (1999) *Cell* 99, 703-12.
- Zhang, C. et al. (2012) *Hepatology* 55, 1070-82.
- Lee, A.H. (2012) *Curr Opin Lipidol* 23, 141-6.
- Lee, J.H. et al. (2011) *Nat Med* 17, 812-5.

### Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

### Western Blot Buffer

**IMPORTANT:** For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween@ 20 at 4°C with gentle shaking, overnight.

### Applications Key

**W:** Western Blotting **IP:** Immunoprecipitation

### Cross-Reactivity Key

**H:** Human **M:** Mouse **R:** Rat **Mk:** Monkey

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