Data Sheet (Cat.No.TMAH-00085)

TargetMol

Anti-ATF-4 Antibody (4W890)

Product Details

Ig Type:	Rabbit IgG
Reactivity:	Human
Conjugation:	Unconjugated
Clone:	4W890
Purification:	Affinity-chromatography

Applications

Verified Activity:

1. Western Blot
-Positive WB detected in: Hela whole cell lysate, MCF-7 whole cell lysate, 293 whole cell lysate, HepG2 whole cell lysate, Jurkat whole cell lysate, K562 whole cell lysate
-All lanes: ATF4 antibody at 1.6μg/ml
-Secondary: Goat polyclonal to rabbit IgG at 1/50000 dilution
-Predicted band size: 39 KDa
-Observed band size: 50 KDa
2. IHC image of TMAH-00085 diluted at 1:160 and staining in paraffin-embedded human breast cancer performed on a Leica BondTM system. After dewaxing and hydration, antigen retrieval was mediated by high pressure in a citrate buffer (pH 6.0). Section was blocked with 10% normal goat serum 30min at RT. Then primary antibody (1% BSA) was incubated at 4°C overnight. The primary is detected by a biotinylated secondary antibody and visualized using an HRP conjugated SP system.
3. Immunofluorescence staining of HepG2 cells with TMAH-00085 at 1:53, counter-stained with DAPI. The cells were fixed in 4% formaldehyde, permeabilized using 0.2% Triton X-100 and blocked in 10% normal Goat Serum. The cells were then incubated with the antibody overnight at 4°C. The secondary antibody was Alexa Fluor 488-congugated AffiniPure Goat Anti-Rabbit IgG (H+L).
4. Immunoprecipitating ATF4 in Hela whole cell lysate
-Lane 1: Rabbit control IgG instead of TMAH-00085 in Hela whole cell lysate.
For western blotting, a HRP-conjugated Protein G antibody was used as the secondary antibody (1/2000)
-Lane 2: TMAH-00085 (3μg) + Hela whole cell lysate (500μg)
-Lane 3: Hela whole cell lysate (20μg)
5. Overlay histogram showing Hela cells stained with TMAH-00085 (red line) at 1:50. The cells were fixed with 70% Ethylalcohol (18h) and then permeabilized with 0.3% Triton X-100 for 2 min. The cells were then incubated in 1x PBS /10% normal goat serum to block non-specific protein-protein interactions followed by primary antibody for 1 h at 4°C. The secondary antibody used was FITC goat anti-rabbit IgG (H+L) at 1/200 dilution for 1 h at 4°C. Control antibody (green line) was used under the same conditions. Acquisition of >10,000 events was performed.

Application:	ELISA, WB, IHC, IF, FCM, IP
Recommended	WB:1:500-1:5000; IHC:1:50-1:200; IF:1:20-1:200; IP:1:200-1:1000.

Properties

Stability & Storage:	Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	A synthetic peptide: Human ATF4
Antigen Species:	Human
Gene ID:	468
Uniprot ID:	P18848
Biology Area:	Epigenetics and Nuclear Signaling

Research Background

Transcription factor that binds the cAMP response element (CRE) (consensus: 5'-GTGACGT[AC][AG]-3') and displays two biological functions, as regulator of metabolic and redox processes under normal cellular conditions, and as master transcription factor during integrated stress response (ISR). Binds to asymmetric CRE's as a heterodimer and to palindromic CRE's as a homodimer. Core effector of the ISR, which is required for adaptation to various stress such as endoplasmic reticulum (ER) stress, amino acid starvation, mitochondrial stress or oxidative stress. During ISR, ATF4 translation is induced via an alternative ribosome translation re-initiation mechanism in response to EIF2S1/eIF-2-alpha phosphorylation, and stress-induced ATF4 acts as a master transcription factor of stress-responsive genes in order to promote cell recovery. Promotes the transcription of genes linked to amino acid sufficiency and resistance to oxidative stress to protect cells against metabolic consequences of ER oxidation. Activates the transcription of NLRP1, possibly in concert with other factors in response to ER stress. Activates the transcription of asparagine synthetase (ASNS) in response to amino acid deprivation or ER stress. However, when associated with DDIT3/CHOP, the transcriptional activation of the ASNS gene is inhibited in response to amino acid deprivation. Together with DDIT3/CHOP, mediates programmed cell death by promoting the expression of genes involved in cellular amino acid metabolic processes, mRNA translation and the terminal unfolded protein response (terminal UPR), a cellular response that elicits programmed cell death when ER stress is prolonged and unresolved. Together with DDIT3/CHOP, activates the transcription of the IRS-regulator TRIB3 and promotes ER stress-induced neuronal cell death by regulating the expression of BBC3/PUMA in response to ER stress. May cooperate with the UPR transcriptional regulator QRICH1 to regulate ER protein homeostasis which is critical for cell viability in response to ER stress. In the absence of stress, ATF4 translation is at low levels and it is required for normal metabolic processes such as embryonic lens formation, fetal liver hematopoiesis, bone development and synaptic plasticity. Acts as a regulator of osteoblast differentiation in response to phosphorylation by RPS6KA3/RSK2: phosphorylation in osteoblasts enhances transactivation activity and promotes expression of osteoblast-specific genes and post-transcriptionally regulates the synthesis of Type I collagen, the main constituent of the bone matrix. Cooperates with FOXO1 in osteoblasts to regulate glucose homeostasis through suppression of beta-cell production and decrease in insulin production. Activates transcription of SIRT4. Regulates the circadian expression of the core clock component PER2 and the serotonin transporter SLC6A4. Binds in a circadian time-dependent manner to the cAMP response elements (CRE) in the SLC6A4 and PER2 promoters and periodically activates the transcription of these genes. Mainly acts as a transcriptional activator in cellular stress adaptation, but it can also act as a transcriptional repressor: acts as a regulator of synaptic plasticity by repressing transcription, thereby inhibiting induction and maintenance of long-term memory. Regulates synaptic functions via interaction with DISC1 in neurons, which inhibits ATF4 transcription factor activity by disrupting ATF4 dimerization and DNA-binding. (Microbial infection) Binds to a Tax-responsive enhancer element in the long terminal repeat of HTLV-I.

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