## BACKGROUND

The Akt family of protein kinases (also designated PKB) play a role in Insulin signaling, cellular survival and transformation. Genetic alterations resulting in an aberrant activation of the phosphoinositol-3-kinase (PI3K)/Akt signaling pathway frequently occur in glioblastomas. Such factors include phosphatase and tensin homolog (PTEN) mutation, epidermal growth factor receptor (EGFR) amplification and rearrangement, and carboxy-terminal modulator protein (CTMP) hypermethylation. CTMP binds to the C-terminal regulatory domain of Akt and acts as a negative regulator. CTMP inhibits phosphorylation of Akt on Serine 473 and Threonine 308, thereby reducing its activity. Decreased expression of CTMP by hypermethylation of its promoter has been linked to the pathogenesis of glioblastomas.

## REFERENCES

1. Maira, S.M., et al. 2001. Carboxyl-terminal modulator protein (CTMP), a negative regulator of PKB/Akt and v-Akt at the plasma membrane. Science 294: 374-380.
2. Knobbe, C.B., et al. 2004. Hypermethylation and transcriptional downregulation of the carboxyl-terminal modulator protein gene in glioblastomas. J. Natl. Cancer Inst. 96: 483-486. PMID: 15026474,
3. Knobbe, C.B., et al. 2005. Genetic alteration and expression of the phospho-inositol-3-kinase/Akt pathway genes PIK3CA and PIKE in human glioblastomas. Neuropathol. Appl. Neurobiol. 31: 486-490.
4. Chae, K.S., et al. 2005. Akt activation is necessary for growth factor-induced trafficking of functional $\mathrm{K}(\mathrm{Ca})$ channels in developing parasympathetic neurons. J. Neurophysiol. 93: 1174-1182.
5. Williams, D.L., et al. 2006. Modulation of the phosphoinositide 3-kinase signaling pathway alters host response to sepsis, inflammation, and ischemia/reperfusion injury. Shock 25: 432-439.
6. Martelli, A.M., et al. 2006. Intranuclear 3'-phosphoinositide metabolism and Akt signaling: new mechanisms for tumorigenesis and protection against apoptosis? Cell. Signal.18: 1101-1107.

## CHROMOSOMAL LOCATION

Genetic locus: Them4 (mouse) mapping to 3 F2.1.

## SOURCE

CTMP (M-128) is a rabbit polyclonal antibody raised against amino acids 40-167 mapping within an internal region of CTMP of mouse origin.

## PRODUCT

Each vial contains $200 \mu \mathrm{ggG}$ in 1.0 ml of PBS with $<0.1 \%$ sodium azide and $0.1 \%$ gelatin.

## STORAGE

Store at $4^{\circ} \mathrm{C}$, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

## APPLICATIONS

CTMP (M-128) is recommended for detection of CTMP of mouse and rat origin by Western Blotting (starting dilution 1:200, dilution range 1:1001:1000), immunoprecipitation [ $1-2 \mu \mathrm{~g}$ per $100-500 \mu \mathrm{~g}$ of total protein ( 1 ml of cell lysate)], 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).

Suitable for use as control antibody for CTMP siRNA (m): sc-142620, CTMP shRNA Plasmid ( m ): sc-142620-SH and CTMP shRNA ( m ) Lentiviral Particles: sc-142620-V.

Molecular Weight of CTMP: 26 kDa .

## RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use goat anti-rabbit IgG-HRP: sc-2004 (dilution range: 1:2000-1:100,000) or Cruz MarkerTM compatible goat antirabbit IgG-HRP: sc-2030 (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) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 ( 0.5 ml agarose $/ 2.0 \mathrm{ml}$ ). 3) Immunofluorescence: use goat anti-rabbit IgG-FITC: sc-2012 (dilution range: 1:100-1:400) or goat anti-rabbit IgG-TR: sc-2780 (dilution range: 1:100-1:400) with UltraCruz ${ }^{\text {M }}$ Mounting Medium: sc-24941.

## RESEARCH USE

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

## PROTOCOLS

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

