10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Anti-CREB (Ser-133), Phosphospecific Antibody

Catalog # AN1724

Product Information

Application	WB, ICC
Primary Accession	P16220
Host	Mouse
Clonality	Mouse Monoclonal
Isotype	IgG1
Clone Names	M415
Calculated MW	35136

Additional Information

Gene ID	1385
Other Names	Cyclic AMP, CREB
Target/Specificity	CREB (cyclic AMP response element-binding protein) is a stimulus-induced transcription factor that plays pivotal roles in cell survival and proliferation. CREB is expressed in various tissues, and has important gene-regulating roles in the nervous system. The transactivation function of CREB is primarily regulated through Ser-133 phosphorylation by cAMP-dependent protein kinase A (PKA) and related kinases. CREB is phosphorylated at other sites in response to calcium influx and DNA damage. The DNA-damage responsive nuclear kinase, HIPK2, can phosphorylate Ser-271 but not Ser-133 in CREB, and this phosphorylation activates CREB transactivation function. Mutation of Ser-271 to Glu-271 potentiates the CREB transactivation function. Thus, phosphorylation of Ser-271 may be the mode of activation for CREB-dependent transcription in response to genotoxic stress.
Dilution	WB~~1:1000 ICC~~N/A
Format	Protein A Purified
Storage	Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	Anti-CREB (Ser-133), Phosphospecific Antibody is for research use only and not for use in diagnostic or therapeutic procedures.
Shipping	Blue Ice

Background

CREB (cyclic AMP response element-binding protein) is a stimulus-induced transcription factor that plays pivotal roles in cell survival and proliferation. CREB is expressed in various tissues, and has important gene-regulating roles in the nervous system. The transactivation function of CREB is primarily regulated

through Ser-133 phosphorylation by cAMP-dependent protein kinase A (PKA) and related kinases. CREB is phosphorylated at other sites in response to calcium influx and DNA damage. The DNA-damage responsive nuclear kinase, HIPK2, can phosphorylate Ser-271 but not Ser-133 in CREB, and this phosphorylation activates CREB transactivation function. Mutation of Ser-271 to Glu-271 potentiates the CREB transactivation function. Thus, phosphorylation of Ser-271 may be the mode of activation for CREB-dependent transcription in response to genotoxic stress.