DRIVERS OF NEURODEGENERATION: Altered Cell Signaling

EuroClone®
serving science through innovation

Abnormal cell-cell communication, for example disrupted presynaptic input, as well as disrupted intracellular signaling contribute to the pathogenesis of neurodegenerative disease. Understanding the signal transduction pathways that regulate gene expression will help understand disease initiation and progression, thereby informing efforts to develop therapeutic interventions. Interested in better understanding Altered Cell Signaling?

Start with These Targets

CREB

CREB signaling is a cellular transcription factor that plays an important role in the formation of memories. Perturbed signaling has been observed in the brains of Alzheimer's disease mouse models, suggesting CREB signaling may be disrupted in human Alzheimer's disease brains as well. Disturbances in CREB function may also contribute to the development and progression of Huntington's disease.

CREB (48H2) Rabbit mAb #9197 – W, IP, IHC-P, IHC-F, IF-F, **IF-IC,** ChIP, ChIP-seq, F

Phospho-CREB (Ser133)

CREB is a cellular transcription factor activated when phosphorylated on Ser133. pCREB (Ser133) levels are reduced in the prefrontal cortex of patients with Alzheimer's disease, indicating a dysfunction in CREB signaling. Reduced pCREB levels in the peripheral blood mononuclear cells (PBMCs) of patients with Alzheimer's disease correlate with pCREB levels observed in postmortem Alzheimer's disease brains, suggesting pCREB expression in PBMCs may be a potential biomarker for disease progression.

Phospho-CREB (Ser133) (87G3) Rabbit mAb #9198 – W, IHC-P, **IF-IC,** IF-F, ChIP, ChIP-seq, F

GSK-3β

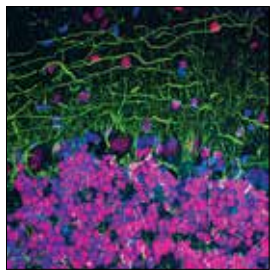
GSK-3β is known to interact with Tau, beta-amyloid, and α-synuclein and is implicated in the pathogenesis of Alzheimer's disease and Parkinson's disease. It is one of the kinases responsible for Tau hyperphosphorylation, resulting in neurofibrillary tangles. GSK-3β regulates several critical cellular events, such as axonal transport, microtubule dynamics, apoptosis, and inflammation, making GSK-3β a potential therapeutic target.

GSK-3β (D5C5Z) XP® Rabbit mAb #12456 – W, IP, **IHC-P**, IF-IC, F

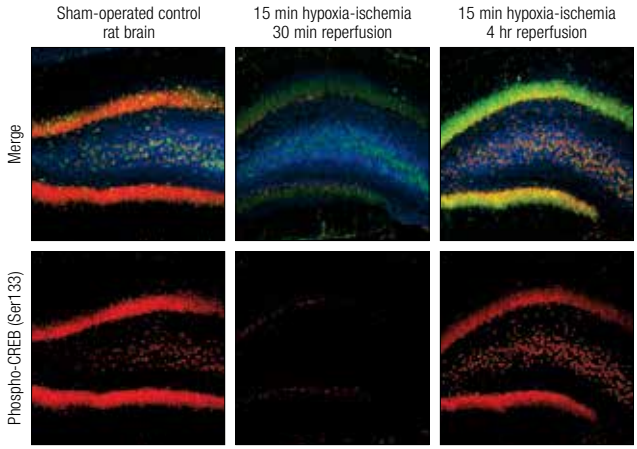
Phospho-GSK-3β (Ser9)

Phosphorylation of GSK-3β on Ser9 inactivates the protein, influencing its ability to regulate glycogen synthesis in response to insulin. In Alzheimer's disease mouse models, GSK-3β Ser9 phosphorylation may also reduce APP processing by β-secretase, decreasing Aβ production..

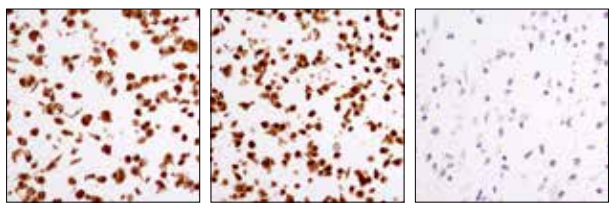
Phospho-GSK-3β (Ser9) (D85E12) XP® Rabbit mAb #5558 – W, IP, IF-IC, F



CREB (48H2) Rabbit mAb #9197: Confocal IF analysis of mouse cerebellum using #9197 (red) and Neurofilament-L (DA2) Mouse mAb #2835 (green). Blue pseudocolor =DRAQ5® #4084 (fluorescent DNA dye).



Phospho-CREB (Ser133) (87G3) Rabbit mAb #9198: Confocal IF images of dentate gyrus labeled with #9198 (red), Neurofilament-L (DA2) Mouse mAb #2835 (blue) and Phospho-S6 Ribosomal Protein (Ser235/236) (2F9) Rabbit mAb (Alexa Fluor® 488 Conjugate) #4854. Sections were obtained from a sham-operated control rat (left) or rats subjected to 15 min of hypoxia-ischemia followed by 30 min (left) or 4 h reperfusion (right).



GSK-3β (D5C5Z) XP® Rabbit mAb #12456: IHC analysis of paraffin-embedded MEF cell pellets, wild type (left), GSK-3α (-/-) (middle) and GSK-3β (-/-) (right) using #12456. (MEF wild type, GSK-3β (-/-), and GSK-3α (-/-) cells were kindly provided by Dr. Jim Woodgett, University of Toronto, Canada.)

Learn more about the Hallmarks of Neurodegeneration at:
cst-science.com/ndg-hallmarks

Cell Signaling TECHNOLOGY®

PR 951_CST_Cell_Signaling_1119