

DESCRIPTION

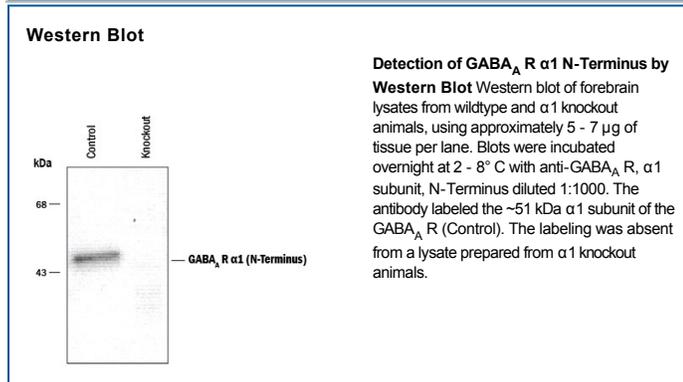
Species Reactivity	Mouse/Rat/Bovine
Specificity	Mouse and rat ~51 kDa GABA _A R α1 subunit, N-Terminus
Source	Polyclonal Rabbit IgG
Purification	Antigen Affinity-purified
Immunogen	N-terminus peptide of GABA _A R α1 subunit conjugated to keyhole limpet hemocyanin (KLH)
Formulation	100 µL in 10 mM HEPES (pH 7.5), 150 mM NaCl, 100 µg/mL BSA and 50% glycerol. See Certificate of Analysis for details.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	1:1000 dilution	See Below

DATA



PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	For long-term storage, ≤ -20° C is recommended. Product is stable at ≤ -20° C for at least 1 year.

BACKGROUND

GABA_A (γ-aminobutyric acid-type A) receptors are members of the cysteine-loop family of neurotransmitter-gated ion channels. GABA binding to A-type receptors induces anion-selective ion channel opening. These receptors are the principal fast inhibitory neurotransmitter receptors in the CNS. GABA_A receptors are heteropentamer combinations of seven subunit types; α, β, γ, δ, ε, θ, and π. Three subunits, α, β, and γ, have at least three separate gene products in mammals, and typical GABA_A receptors have some combination of α, β, and γ subunits. The rat α1 isoform is a 50 - 52 kDa, 428 amino acid (aa), 4 transmembrane protein with two terminal extracellular regions. The ligand-binding region is in the N-terminus (aa 15 - 222). As with many receptors, phosphorylation is used as a regulatory mechanism. CaM kinase II is known to phosphorylate the α1 subunit and regulate benzodiazepine binding. α1 subunits are particularly abundant in the cerebellum and may contribute to GABA receptor distribution. In the hippocampus and amygdala, the α1 subunit may contribute to amnesia.

References:

1. Darlison, M.G. et al. (2005) Cell. Mol. Neurobiol. 25:607.
2. Akabas, M.H. (2004) Int. Rev. Neurobiol. 62:1.
3. Churn, S.B. et al. (2002) J. Neurochem. 82:1065.
4. Kralic, J.E. et al. (2006) J. Comp. Neurol. 495:408.
5. Sonner, J.M. et al. (2006) Mol. Pharmacol. 68:61.