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Product Datasheet

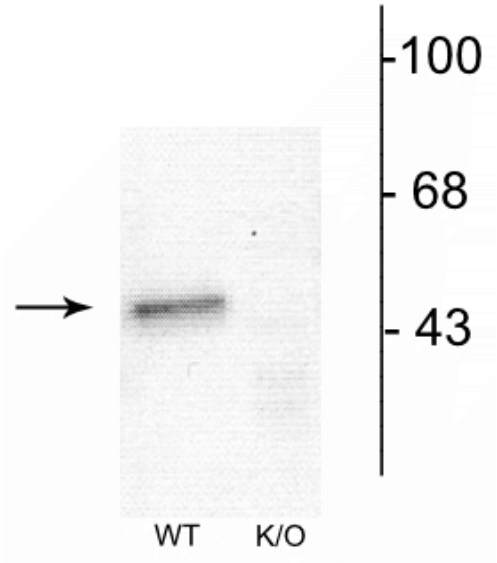
Anti-GABA$_A$ Receptor α1, N-Terminus

KO Validated

Overview

Catalog #	812-GA1N
Host Species	Rabbit Polyclonal
Format	Antigen Affinity Purified
Applications	WB 1:1000 IHC 1:100
Species Tested	Mouse, Rat
Expected Reactivity	Bovine, Canine, Human, Non-Human Primate
Immunogen	Synthetic peptide corresponding to amino acid residues from the N-terminal region of the α1 subunit of rat GABA$_A$, conjugated to keyhole limpet hemocyanin (KLH).
Molecular Weight	51 kDa
Cite this Antibody	PhosphoSolutions Cat# 812-GA1N, RRID:AB_2492100

Images



Western blot of mouse forebrain lysates from Wild Type (WT) and α_1-knockout (K/O) animals showing specific immunolabeling of the ~51 kDa α_1-subunit of the GABA$_A$-R. The labeling was absent from a lysate prepared from α_1-knockout animals.

Details

Target Description	Gamma-aminobutyric acid (GABA) is the primary inhibitory neurotransmitter in the central nervous system, causing a hyperpolarization of the membrane through the opening of a Cl− channel associated with the $GABA_A$ receptor ($GABA_A$-R) subtype. $GABA_A$-Rs are important therapeutic targets for a range of sedative, anxiolytic, and hypnotic agents and are implicated in several diseases including epilepsy, anxiety, depression, and substance abuse. The $GABA_A$-R is a multimeric subunit complex. To date six αs, four βs and four γs, plus alternative splicing variants of some of these subunits, have been identified (Olsen and Tobin, 1990; Whiting et al., 1999; Ogris et al., 2004). Injection in oocytes or mammalian cell lines of cRNA coding for α- and β-subunits results in the expression of functional $GABA_A$-Rs sensitive to GABA. However, coexpression of a γ-subunit is required for benzodiazepine modulation. The various effects of the benzodiazepines in brain may also be mediated via different α-subunits of the receptor (McKernan et al., 2000; Mehta and Ticku, 1998; Ogris et al., 2004; Pöltl et al., 2003).
Specificity	Specific for endogenous levels of the ~51 kDa α1-subunit of the $GABA_A$ receptor. Immunolabeling is absent in α1-subunit knockout animals.
Production/Purification	Prepared from pooled rabbit serum by affinity purification using a column to which the peptide immunogen was coupled.
Quality Control	Western blots performed on each lot.
Buffer	10 mM HEPES (pH 7.5), 150 mM NaCl, 100 µg per ml BSA and 50% glycerol.
Storage	Storage at -20°C is recommended, as aliquots may be taken without freeze/thawing due to presence of 50% glycerol.
Stability	After date of receipt, stable for at least 1 year at -20°C.

Significant Citations

Agoglia, A.E., Zhu, M., Ying, R., Sidhu, H., Natividad, L.A., Wolfe, S.A., Buczynski, M.W., Contet, C., Parsons, L.H., Roberto, M. and Herman, M.A., 2020. Corticotropin-Releasing Factor Receptor-1 Neurons in the Lateral Amygdala Display Selective Sensitivity to Acute and Chronic Ethanol Exposure. *Eneuro*, *7(2)*.

Engin, E., Zarnowska, E. D., Benke, D., Tsvetkov, E., Sigal, M., Keist, R., Bolshakov, V.Y., Pearce, R.A., & Rudolph, U. (2015). Tonic Inhibitory Control of Dentate Gyrus Granule Cells by α5-Containing GABAA Receptors Reduces Memory Interference. *The Journal of Neuroscience*, *35(40)*, 13698-13712.

Wyatt, L. R., Finn, D. A., Khoja, S., Yardley, M. M., Asatryan, L., Alkana, R. L., & Davies, D. L. (2014). Contribution of P2X4 receptors to ethanol intake in male C57BL/6 mice. *Neurochemical Research*, *39(6)*, 1127-1139.

Herman, M. A., & Roberto, M. (2014). Cell-type-specific tonic GABA signaling in the rat central amygdala is selectively altered by acute and chronic ethanol. *Addiction Biology*. Aug. 29.

Kurano, Y., Nakamura, M., Ichiba, M., Matsuda, M., Mizuno, E., Kato, M., Izumo, S. and Sano, A., 2006. Chorein deficiency leads to upregulation of gephyrin and GABAA receptor. *Biochemical and biophysical research communications*, *351(2)*, pp.438-442.

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