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

Anti-GABAA Receptor α1, N-Terminus Antibody

Our Anti-GABAA Receptor α1, N-Terminus primary antibody from PhosphoSolutions is rabbit polyclonal.

Catalog # AN1391

Specification

Anti-GABAA Receptor α1, N-Terminus Antibody - Product Information

Application	**WB**
Primary Accession	P62813
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**IgG**
Calculated MW	**51754**

Anti-GABAA Receptor α1, N-Terminus Antibody - Additional Information

Gene ID	**29705**

Other Names

ECA4 antibody, EIEE19 antibody, EJM antibody, EJM5 antibody, Gaba receptor alpha 1 polypeptide antibody, GABA(A) receptor antibody, GABA(A) receptor subunit alpha 1 antibody, GABA(A) receptor subunit alpha-1 antibody, GABA(A) receptor, alpha 1 antibody, GABRA 1 antibody, GABRα1 antibody, Gamma aminobutyric acid (GABA) A receptor alpha 1 antibody, Gamma aminobutyric acid A receptor alpha 1 antibody, Gamma aminobutyric acid type A receptor alphα1 subunit antibody

Target/Specificity

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).

Dilution

WB~~1:1000

Format

Antigen Affinity 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

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Anti-GABAA Receptor α1, N-Terminus Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

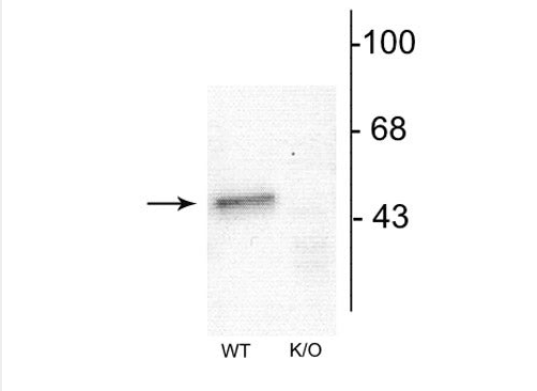
Shipping
Blue Ice

Anti-GABAA Receptor α1, N-Terminus Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Anti-GABAA Receptor α1, N-Terminus Antibody - Images



Western blot of mouse forebrain lysates from Wild Type (WT) and α1-knockout (K/O) animals showing specific immunolabeling of the ~51 kDa α1-subunit of the GABAA-R. The labeling was absent from a lysate prepared from α1-knockout animals.

Anti-GABAA Receptor α1, N-Terminus Antibody - Background

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).