

Anti-GABAA Receptor α 1, N-Terminus Antibody

Our Anti-GABAA Receptor α 1, N-Terminus primary antibody from PhosphoSolutions is rabbit polyclonal.

Catalog # AN1391

Specification**Anti-GABAA Receptor α 1, N-Terminus Antibody - Product Information**

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

Anti-GABAA Receptor α 1, 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, GABR α 1 antibody, Gamma aminobutyric acid (GABA) A receptor alpha 1 antibody, Gamma aminobutyric acid A receptor alpha 1 antibody, Gamma aminobutyric acid type A receptor alpha1 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 α s, four β s and four γ s, 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; Pörtl et al., 2003).

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.

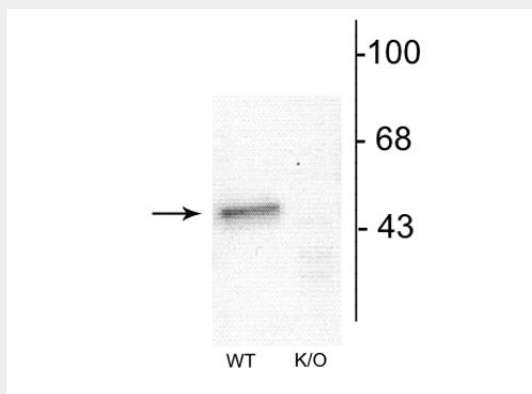
Precautions

Anti-GABAA Receptor α 1, N-Terminus Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Shipping
Blue Ice**Anti-GABAA Receptor α 1, 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 Cytometry](#)
- [Cell Culture](#)

Anti-GABAA Receptor α 1, N-Terminus Antibody - 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 GABAA-R. The labeling was absent from a lysate prepared from α 1-knockout animals.

Anti-GABAA Receptor α 1, 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 α s, four β s and four γ s, 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; Pölzl et al., 2003).