

Anti-GABAA Receptor δ , N-Terminus Antibody

Our Anti-GABAA Receptor δ , N-Terminus primary antibody from PhosphoSolutions is rabbit polyclonal. I
Catalog # AN1402

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

Application	WB, IHC
Primary Accession	P18506
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	50566

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

Gene ID **29689**

Other Names

GABA(A) receptor subunit delta antibody, Gabrd antibody, Gamma aminobutyric acid GABA A receptor delta antibody, Gamma aminobutyric acid receptor delta subunit precursor GABA A receptor antibody, Gamma-aminobutyric acid receptor subunit delta antibody, GBRD_HUMAN antibody, MGC45284 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, co-expression 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). More recently there have been a number of studies demonstrating that the δ -subunit of the receptor may affect subunit assembly (Korpi et al., 2002) and may also confer differential sensitivity to neurosteroids and to ethanol (Wallner et al., 2003; Wohlfarth et al., 2002).

Dilution

WB~~1:1000
IHC~~1:100~500

Format

Antigen Affinity Purified from Pooled Serum

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 δ , N-Terminus Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Shipping

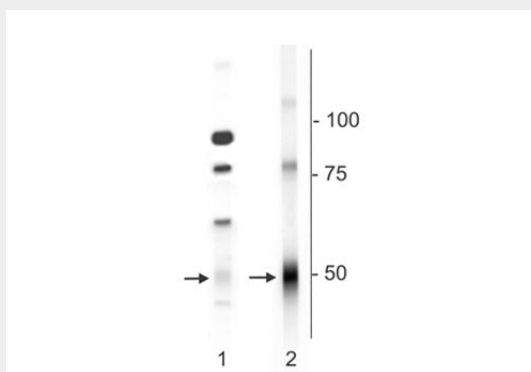
Blue Ice

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

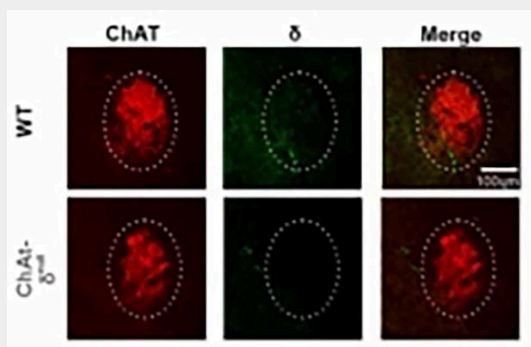
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

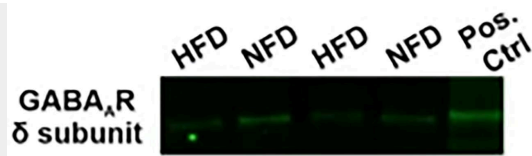
Anti-GABAA Receptor δ , N-Terminus Antibody - Images



Western blot of mouse whole brain (1) and mouse synaptic plasma membrane (2) lysates showing specific immunolabeling of the ~50 kDa δ -subunit of the GABAA-R.



Immunostaining of a novel ChAT- δ knock down mouse brain labeling GABAA(δ)R (Cat no 868A-GDN, 1:50, green) in WT c57Bl/6 mouse brain, and confirmation of a negative signal in the ChAT- δ knock down mouse brain. Image from publication CC-BY-4.0. PMID: 37085567



Anti-GABAA Receptor δ , N-Terminus Antibody

Anti-GABAA Receptor δ , 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, co-expression 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). More recently there have been a number of studies demonstrating that the δ -subunit of the receptor may affect subunit assembly (Korpi et al., 2002) and may also confer differential sensitivity to neurosteroids and to ethanol (Wallner et al., 2003; Wohlfarth et al., 2002).