

Anti-GABAA Receptor α 6 Antibody

Our Anti-GABAA Receptor α 6 primary antibody from PhosphoSolutions is rabbit polyclonal. It detects m
Catalog # AN1396

Specification**Anti-GABAA Receptor α 6 Antibody - Product Information**

Primary Accession	P30191
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	51184

Anti-GABAA Receptor α 6 Antibody - Additional Information

Gene ID **29708**

Other Names

GABA A antibody, GABA A Receptor α 6 polypeptide antibody, GABA A receptor α 6 antibody, GABA A receptor subunit α 6 antibody, GABA subunit A receptor α 6 antibody, GABA(A) receptor subunit α -6 antibody, GABRA6 antibody, Gamma aminobutyric acid A receptor α 6 antibody, Gamma aminobutyric acid GABA A receptor α 6 antibody, Gamma aminobutyric acid receptor subunit α 6 antibody, Gamma-aminobutyric acid receptor subunit α -6 antibody, GBRA6_HUMAN antibody, MGC116903 antibody, MGC116904 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). Lastly, phosphorylation of β -subunits of the receptor has been shown to modulate GABAA-R function (Brandon et al., 2003).

Format

Neat 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 α 6 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

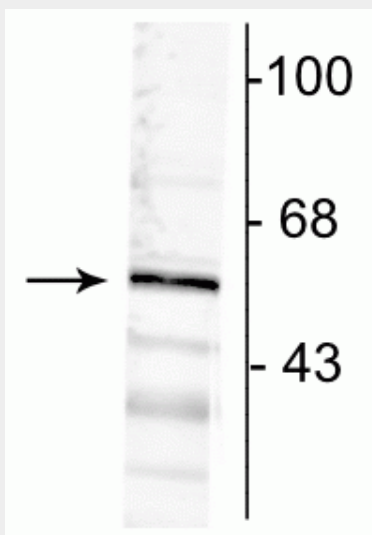
Shipping
Blue Ice

Anti-GABAA Receptor $\alpha 6$ 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 $\alpha 6$ Antibody - Images



Western blot of rat cortical lysate showing specific immunolabeling of the ~57 kDa $\alpha 6$ -subunit of the GABAA-R.

Anti-GABAA Receptor $\alpha 6$ 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; Pörtl et al., 2003). Lastly, phosphorylation of β -subunits of the receptor has been shown to modulate GABAA-R function (Brandon et al., 2003).