

## Anti-GABAA Receptor a5 Antibody

Our Anti-GABAA Receptor α5 rabbit polyclonal primary antibody from PhosphoSolutions is produced in-h Catalog # AN1395

## **Specification**

## Anti-GABAA Receptor a5 Antibody - Product Information

Application WB
Primary Accession P19969
Host Rabbit
Clonality Polyclonal
Isotype IgG
Calculated MW 52337

## **Anti-GABAA Receptor α5 Antibody - Additional Information**

Gene ID 29707

### **Other Names**

GAα5 antibody, GAA5 antibody, GABA(A) receptor subunit alpha-5 antibody, GABRα5 antibody, Gabra5 antibody, Gamma aminobutyric acid GABA A receptor alphα5 antibody, Gamma aminobutyric acid GABA A receptor alphα5 precursor antibody, Gamma aminobutyric acid receptor alphα5 subunit precursor GABA A receptor antibody, Gamma-aminobutyric acid receptor subunit alpha-5 antibody, GBRA5\_HUMAN antibody, GC138184 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  $\alpha$ s, four  $\beta$ s and four  $\gamma$ 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  $\alpha$ - and  $\beta$ -subunits results in the expression of functional GABA-A-Rs sensitive to GABA. However, coexpression of a  $\gamma$ -subunit is required for benzodiazepine modulation. The various effects of the benzodiazepines in brain may also be mediated via different  $\alpha$ -subunits of the receptor (McKernan et al., 2000; Mehta and Ticku, 1998; Ogris et al., 2004; Pöltl et al., 2003).

#### **Format**

Antigen Affinity Purified 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  $\alpha 5$  Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

# **Shipping**



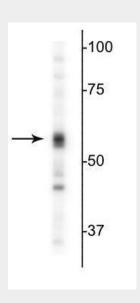
Blue Ice

# Anti-GABAA Receptor a5 Antibody - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

# Anti-GABAA Receptor α5 Antibody - Images



Western blot of mouse whole brain showing specific immunolabeling of the  $\sim$ 55 kDa  $\alpha$ 5-subunit of the GABAA-R.

## Anti-GABAA Receptor a5 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  $\alpha$ s, four  $\beta$ s and four  $\gamma$ 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  $\alpha$ - and  $\beta$ -subunits results in the expression of functional GABA-A-Rs sensitive to GABA. However, coexpression of a  $\gamma$ -subunit is required for benzodiazepine modulation. The various effects of the benzodiazepines in brain may also be mediated via different  $\alpha$ -subunits of the receptor (McKernan et al., 2000; Mehta and Ticku, 1998; Ogris et al., 2004; Pöltl et al., 2003).