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## Product Information

### Anti-phospho-GABA<sub>B</sub> Receptor (pSer<sup>892</sup>) R2 Subunit

produced in rabbit, affinity isolated antibody

Catalog Number **G5293**

#### Product Description

Anti-phospho-GABA<sub>B</sub> Receptor (pSer<sup>892</sup>) R2 Subunit (GABA<sub>B</sub> R2, GABABR2; GABBR2) is produced in rabbit using a synthetic phosphorylated peptide derived from the region of rat GABA<sub>B</sub> receptor R2 subunit that is phosphorylated on serine 892 as immunogen. The antiserum is affinity purified using epitope-specific affinity chromatography. The antibody is preadsorbed to remove any reactivity towards a non-phosphorylated GABA<sub>B</sub> R2 protein and phospho-serine GABA<sub>B</sub> R2 irrespective of the sequence. Anti-phospho-GABA<sub>B</sub> R2 (pSer<sup>892</sup>) specifically recognizes GABA<sub>B</sub> R2 phosphorylated at Ser<sup>892</sup> (110 kDa). The antibody detects human and rat GABA<sub>B</sub> R2 (pSer<sup>892</sup>). It is used in immunoblotting<sup>1</sup>, ELISA, immunocytochemistry, immunoprecipitation and immunofluorescence.

GABA ( $\gamma$ -aminobutyric acid) receptors are a family of proteins involved in the GABAergic neurotransmission of the mammalian central nervous system. GABA signals through two distinct types of pre- and post-synaptic receptors referred to as GABA<sub>A</sub> and GABA<sub>B</sub> receptors. The GABA<sub>B</sub> receptors are heterodimeric G protein-coupled receptors that mediate slow synaptic inhibition in the central nervous system via regulation of the release of neurotransmitters and activation of ion channels and adenylyl cyclase.

In 1998 White et al., identified a human G protein-coupled receptor 51 (GPR51) cDNA, which they called GABA<sub>B</sub>R2. Recombinant GPR51 expressed in COS cells has an apparent molecular mass of 130 kDa, thought to be a glycosylated 110 kDa GPR51.<sup>2-4</sup> The expression of GABA<sub>B</sub> R1 and GABA<sub>B</sub> R2 was detected in fetal and adult human brain. Native GABA<sub>B</sub> receptors function as heteromeric proteins, the most abundant form being the GABA<sub>B</sub> R1/GABA<sub>B</sub> R2 coupled receptor targeted to plasma membrane. The interaction of these receptors appears to be crucial for important physiologic effects of GABA and provides a mechanism in receptor signaling pathways that involve a heterotrimeric GTP-binding protein.<sup>5</sup>

Cyclic AMP-dependent protein kinase (PKA) directly phosphorylates a single serine residue (Ser<sup>892</sup>) in the cytoplasmic tail of GABA<sub>B</sub> R2. Basal phosphorylation of this residue is evident in rat brain membranes and in cultured neurons. Phosphorylation of Ser<sup>892</sup> is modulated positively by pathways that elevate cAMP concentration, such as those involving forskolin and  $\beta$ -adrenergic receptors. PKA activity enhances the functional coupling of GABA receptors and PKA signaling pathways may have significant effect on the efficacy of synaptic inhibition mediated through GABA<sub>B</sub> receptors. Phosphorylation of Ser<sup>892</sup> also enhances the membrane stability of GABA<sub>B</sub> receptors.<sup>1</sup>

#### Reagent

Supplied, at approximately 0.1 mg/ml, as a solution in 10 mM HEPES, pH 7.5, 150 mM NaCl, 100  $\mu$ g/mL BSA and 50% glycerol.

#### Storage/Stability

Store at  $-20^{\circ}\text{C}$ . For extended storage, freeze in working aliquots. Due to the viscosity of glycerol the solutions needs to be mixed well prior to aliquoting. Do not store in frost-free freezers. Avoid repeated freezing and thawing to prevent denaturing the antibody. Working dilution samples should be discarded if not used within 12 hours. The antibody is stable for at least 6 months when stored appropriately.

#### Product Profile

Immunoblotting: a recommended working dilution of 1:1,000 is determined using rat brain membranes.  
Dot blot: use dilution of 1:1,000.

**Note:** In order to obtain best results in different techniques and preparations we recommend determining optimal working concentration by titration test.

## References

1. Couve, A., et al., Cyclic AMP-dependent protein kinase phosphorylation facilitates GABA<sub>B</sub> receptor-effector coupling. *Nature Neurosci.*, **5**, 415-424 (2002).
2. White, J. H., et al., Heterodimerization is required for the formation of a functional GABA-B receptor. *Nature*, **396**: 679-682, 1998.
3. Grifa, A., et al., GABA ( $\gamma$ -amino-butyric acid) neurotransmission: identification and fine mapping of the human GABA-B receptor gene. *Biochem. Biophys. Res. Commun.*, **250**, 240-245 (1998).
4. Martin, S. C., et al., Molecular identification of the human GABA<sub>B</sub> R1: cell surface expression and coupling to adenylyl cyclase in the absence of GABA<sub>B</sub> R2. *Mol. Cell. Neurosci.*, **13**, 180-191 (1999).
5. Kuner, R., et al., Role of heteromer formation in GABA-B receptor function. *Science*, **283**, 74-77 (1999).

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