

Product Information

Anti-Glutamine Synthetase

produced in rabbit, IgG fraction of antiserum

Catalog Number **G2781**

Product Description

Anti-Glutamine Synthetase is produced in rabbit using a synthetic peptide corresponding to the C-terminus of mouse glutamine synthetase (GS) (amino acids 357-373 with N-terminally added lysine) conjugated to KLH as immunogen. This sequence is identical in human, bovine, rat, hamster and pig GS, and highly conserved in chicken GS (single amino acid substitution). Whole antiserum is purified to provide an IgG fraction of antiserum.

Anti-Glutamine synthetase recognizes rat glutamine synthetase (GS) (45 kDa). Applications include the detection and localization of GS by immunoblotting and immunohistochemistry. Staining of GS in immunoblotting is specifically inhibited with the GS immunizing peptide (amino acids 357-373 with N-terminally added lysine).

Glutamine synthetase (GS) catalyzes the amidation of glutamate to form glutamine. It is found in mammals as an octamer of identical 45 kDa subunits.¹ In the brain, GS is considered a key enzyme participating in the recycling of the excitatory neurotransmitter glutamate released from neurons and termination of the neurotransmitter signal, as well as in ammonia detoxification.^{2,3} Glutamate, the major excitatory transmitter in the mammalian central nervous system, is known to be neurotoxic when present in excess at the synapses. Two major mechanisms protect neurons from glutamate-induced toxicity: (a) removal of synaptic glutamate via a high affinity uptake carried out by excitatory amino acid transporters (EAAT); and (b) metabolism and recycling of glutamate by synaptic astrocytes via GS, in an ATP-requiring reaction.

GS is localized in glial cells of various species. It is present in the brain primarily in astrocytes and in the retina in Muller glial cells that support neurons using glutamate as a neurotransmitter.²⁻⁶ GS activity is a useful marker for astrocytes, and an important differentiation feature in the retina.^{6,7} GS is found in several brain regions including hippocampus, cerebral cortex, neostriatum and cerebellar granular layer, corresponding to sites with high glutamate receptor density.⁶ Upregulation of GS is a hallmark of reactive astrocytosis and GS activity level increases following spinal cord injury.^{8,9} The GS expression in astrocytes is diminished in brains affected by Alzheimer's disease, particularly in the vicinity of senile plaques.¹⁰⁻¹² GS is upregulated in a subset of human hepatocellular carcinomas, suggesting that its expression may be related to tumor recurrence, since tumors expressing GS are independent of the extracellular glutamine supply.¹³

Reagent

Supplied in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2-8 °C for up to one month. For extended storage, freeze in working aliquots at -20 °C. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

Immunoblotting: a minimum working antibody dilution of 1:10,000 is determined using a rat brain cytosolic fraction extract.

Immunohistochemistry: a minimum working antibody dilution of 1:10,000 is determined using formalin-fixed, paraffin-embedded sections of rat brain.

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

References

1. Pahuja, S.L., et al., *Exp. Eye Res.*, **40**, 61 (1985).
2. Martinez-Hernandez A., et al., *Science*, **195**, 1356 (1977).
3. Norenberg, M.D., et al., *Brain Res.*, **161**, 303 (1979).
4. Riepe, R.E., and Norenberg, M.D., *Exp. Eye Res.*, **27**, 435 (1978).
5. Lewis, G.P., et al., *Exp. Eye Res.*, **47**, 855 (1988).
6. Norenberg, M.D., *J. Histochem. Cytochem.*, **27**, 756 (1979).
7. Kentroti, S., et al., *J. Neurosci. Res.*, **28**, 497 (1991).
8. Pike, C.J., et al., *Exp. Neurol.*, **139**, 167 (1996).
9. Benton, R.L., et al., *Neurosci. Lett.*, **291**, 1 (2000).
10. Hensley, K., et al., *J. Neurochem.*, **65**, 2146 (1995).
11. Aksenov, M.Y., et al., *J. Neurochem.*, **66**, 2050 (1996).
12. Robinson, S.R., *Neurochem. Int.*, **36**, 471 (2000).
13. Osada, T, et al., *Cancer*, **85**, 817 (1999).

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