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Datasheet for 200-4158-0100

Glutamate Dehydrogenase Antibody

Overview

Description:	Anti-Glutamate Dehydrogenase (Bovine Liver) (RABBIT) Antibody - 200-4158-0100
Item No.:	200-4158-0100
Size:	100 µg
Applications:	ELISA, WB
Reactivity:	Bovine
Host Species:	Rabbit

Product Details

Background:	Glutamate Dehydrogenase, mitochondrial, converts L-glutamate into alpha-ketoglutarate. It plays a key role in glutamine anaplerosis by producing alpha-ketoglutarate, which is an important intermediate in the tricarboxylic acid cycle. It may be involved in learning and memory reactions by increasing the turnover of the excitatory neurotransmitter glutamate. It is subject to allosteric regulation. It is activated by ADP, inhibited by GTP and ATP, and ADP can occupy the NADH binding site and activate the enzyme.
Synonyms:	rabbit anti-Glutamate Dehydrogenase Antibody, Glutamate dehydrogenase 1 mitochondrial, GDH 1
Host Species:	Rabbit
Clonality:	Polyclonal
Format:	IgG

Target Details

Gene Name:	GLUD1
Reactivity:	Bovine
Immunogen Type:	Native Protein
Immunogen:	Glutamate Dehydrogenase [Bovine Liver]

Purity/Specificity:	Anti-Glutamate Dehydrogenase is an IgG fraction antibody purified from monospecific antiserum by a multi-step process which includes delipidation, salt fractionation and ion exchange chromatography followed by extensive dialysis against the buffer stated above. Assay by immunoelectrophoresis resulted in a single precipitin arc against anti-Rabbit Serum as well as purified and partially purified Glutamate Dehydrogenase [Bovine Liver]. Cross reactivity against Glutamate Dehydrogenase from other tissues and species may occur but have not been specifically determined.
Relevant Links:	• UniProtKB - P00366 • NCBI - 32880221 • GeneID - 281785

Application Details

Tested Applications:	ELISA, WB
Application Note:	Glutamate Dehydrogenase antibody has been tested by ELISA and western blot and is assayed against 1.0 ug of Glutamate Dehydrogenase [Bovine Liver] in a standard ELISA using Peroxidase conjugated Affinity Purified anti-Rabbit IgG [H&L] (Goat) code #611-1302 and (ABTS (2,2'-azino-bis-[3-ethylbenthiazoline-6-sulfonic acid]) code # ABTS-100 as a substrate for 30 minutes at room temperature. A working dilution of 1:5,000 to 1:20,000 is suggested for this product.
Assay Dilutions:	All assays should be optimized by the user. Recommended dilutions (if any) may be listed below.
ELISA:	1:5,000 - 1:20,000
IP:	1:100
WB:	1:500 - 1:5,000

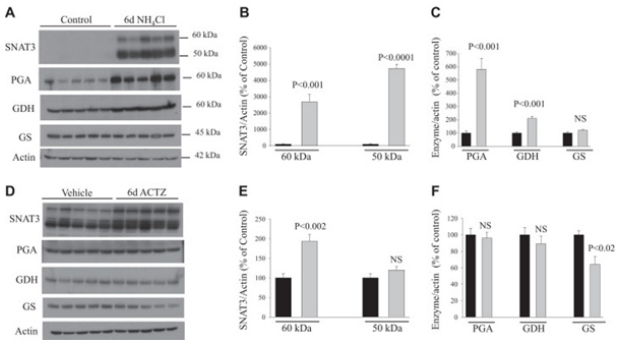
Formulation

Physical State:	Lyophilized
Concentration:	1 mg/ml by UV absorbance at 280 nm
Buffer:	0.02 M Potassium Phosphate, 0.15 M Sodium Chloride, pH 7.2
Preservative:	0.01% (w/v) Sodium Azide
Stabilizer:	None
Reconstitution Volume:	100 µL
Reconstitution Buffer:	Restore with deionized water (or equivalent)

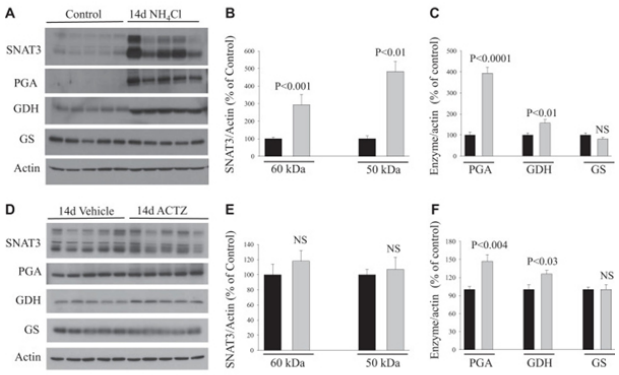
Shipping & Handling

Shipping Condition:	Ambient
Storage Condition:	Store vial at 4° C prior to restoration. For extended storage aliquot contents and freeze at -20° C or below. Avoid cycles of freezing and thawing. Centrifuge product if not completely clear after standing at room temperature. This product is stable for several weeks at 4° C as an undiluted liquid. Dilute only prior to immediate use.
Expiration:	Expiration date is one (1) year from date of receipt.

Images



Western Blot
Adaptation of glutamine transporter and ammoniagenic enzymes in the kidney cortex of rats treated with acetazolamide (ACTZ) or NH4Cl loading for 6 days. A and D: tissue homogenates (enzymes) and membrane fractions (SNAT3) were isolated from the kidney cortex of rats treated with NH4Cl loading (A) or ACTZ (D) versus their respective control or vehicle treatment and were used for immunoblot analysis using specific antibodies for each protein. B, C, E, and F: corresponding average means ± SE of the densitometry analysis of SNAT3 (B and E) and ammoniagenic enzymes (C and F) in response to NH4Cl loading versus control (B and C) or in ACTZ-treated versus vehicle-treated rats (E and F). The abundance of these proteins was normalized to actin used for the control of gel loading. As shown, SNAT3, phosphate-dependent glutaminase (PGA), and glutamate dehydrogenase (GDH) were sharply upregulated in NH4Cl-loaded rats but not in ACTZ-treated rats. n = 5 rats in each group. Each lane was loaded with 10–30 µg protein from the kidney cortex. Note that the exposure time for SNAT3 in vehicle versus ACTZ (D) was much higher than that in control versus 6 days of NH4Cl loading (A). GS, glutamine synthetase; NS, not significant. Fig 4. PMID: 32657159



Western Blot

Adaptation of glutamine transporter and ammoniagenic enzymes in the kidney cortex of rats treated with acetazolamide (ACTZ) or NH4Cl loading for 2 wk. A and D: tissue homogenates (enzymes) and membrane fractions (SNAT3) were isolated from the kidney cortex of rats treated with NH4Cl loading (A) or ACTZ (D) versus their respective control or vehicle treatment and were used for immunoblot analysis using specific antibodies for each protein. B, C, E, and F: corresponding average means ± SE of the densitometry analysis of SNAT3 (B and E) and ammoniagenic enzymes (C and F) in response to NH4Cl loading versus control (B and C) or in ACTZ-treated versus vehicle-treated rats (E and F). The abundance of these proteins was normalized to actin used for the control of gel loading. As shown, SNAT3 was sharply upregulated in NH4Cl-loaded rats but not ACTZ-treated rats for 2 wk. Phosphate-dependent glutaminase (PGA) and glutamate dehydrogenase (GDH) were slightly, but significantly, upregulated in ACTZ-treated rats for 2 wk. The magnitude of PGA induction was greater in NH4Cl loading versus ACTZ treatment. n = 5 rats in each group. Each lane was loaded with 10−30 µg protein from the kidney cortex. Note that the exposure time for SNAT3 in vehicle versus ACTZ (D) was much higher than that in control versus 6 days of NH4Cl loading (A). GS, glutamine synthetase; NS, not significant. Fig 5. PMID: 32657159

References

- Alam P et al. Acetazolamide causes renal HCO−3 wasting but inhibits ammoniagenesis and prevents the correction of metabolic acidosis by the kidney. *Am J Physiol Renol Physiol.* (2020)
- Wilkie MP et al. Shifting Patterns of Nitrogen Excretion and Amino Acid Catabolism Capacity during the Life Cycle of the Sea Lamprey (Petromyzon marinus). *Physiol Biochem Zool.* (2006)

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