

GLUD2 (h): 293T Lysate: sc-116421

BACKGROUND

GLUD1 (glutamate dehydrogenase 1), also known as GDH, GDH1 or GLUD, and GLUD2 (glutamate dehydrogenase 2), also known as GDH2 or GLUDP1, are both mitochondrial matrix enzymes belonging to the Glu/Leu/Phe/Val dehydrogenases family. Existing as homohexamers, GLUD1 catalyzes the oxidative deamination of glutamate to α -ketoglutarate and ammonia while GLUD2 is involved in the recycling of glutamate during neurotransmission. GLUD1 is critical for regulating amino acid induced Insulin secretion and is allosterically activated by ADP and inhibited by GTP and ATP. Mutations in the gene encoding GLUD1 causes hyperinsulinism-hyperammonemia syndrome (HHS), which is an inherited condition characterized by high Insulin and ammonia levels in the blood. GLUD1 may also be involved in learning and memory reactions by increasing the turnover of the excitatory neurotransmitter glutamate. GLUD2 is expressed in testis and retina, with lower levels found in brain.

REFERENCES

1. Stanley, C.A., et al. 2000. Molecular basis and characterization of the hyperinsulinism/hyperammonemia syndrome: predominance of mutations in exons 11 and 12 of the glutamate dehydrogenase gene. HI/HA Contributing Investigators. *Diabetes* 49: 667-673.
2. Tanizawa, Y., et al. 2002. Unregulated elevation of glutamate dehydrogenase activity induces glutamine-stimulated Insulin secretion: identification and characterization of a GLUD1 gene mutation and Insulin secretion studies with MIN6 cells overexpressing the mutant glutamate dehydrogenase. *Diabetes* 51: 712-717.
3. Mastorodemos, V., et al. 2009. Human GLUD1 and GLUD2 glutamate dehydrogenase localize to mitochondria and endoplasmic reticulum. *Biochem. Cell Biol.* 87: 505-516.
4. Pajic, T., et al. 2009. Glutamate dehydrogenase activity in lymphocytes of B-cell chronic lymphocytic leukaemia patients. *Clin Biochem.* 42: 1677-1684.
5. Kapoor, R., et al. 2009. Hyperinsulinism-hyperammonemia (HI/HA) syndrome: novel mutations in the GLUD1 gene and genotype-phenotype correlations. *Eur. J. Endocrinol.* 161: 731-735.
6. Stagourakis, A., et al. 2009. Mutations in human GLUD2 glutamate dehydrogenase affecting basal activity and regulation. *J. Neurochem.* 109: 167-173.
7. Kapoor, R.R., et al. 2009. Advances in the diagnosis and management of hyperinsulinemic hypoglycemia. *Nat. Clin. Pract. Endocrinol. Metab.* 5: 101-112.
8. Zaganas, I., et al. 2009. The human GLUD2 glutamate dehydrogenase: localization and functional aspects. *Neurochem. Int.* 55: 52-63.
9. Plaitakis, A., et al. 2010. Gain-of-function variant in GLUD2 glutamate dehydrogenase modifies Parkinson's disease onset. *Eur. J. Hum. Genet.* 18: 336-341.

CHROMOSOMAL LOCATION

Genetic locus: GLUD2 (human) mapping to Xq24.

PRODUCT

GLUD2 (h): 293T Lysate represents a lysate of human GLUD2 transfected 293T cells and is provided as 100 μ g protein in 200 μ l SDS-PAGE buffer.

APPLICATIONS

GLUD2 (h): 293T Lysate is suitable as a Western Blotting positive control for human reactive GLUD2 antibodies. Recommended use: 10-20 μ l per lane.

Control 293T Lysate: sc-117752 is available as a Western Blotting negative control lysate derived from non-transfected 293T cells.

STORAGE

Store at -20° C. Repeated freezing and thawing should be minimized. Sample vial should be boiled once prior to use. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.