

Product datasheet for **AP12171PU-N**

AGL (C-term) Rabbit Polyclonal Antibody

Product data:

| | |
|-----------------------|--|
| Product Type: | Primary Antibodies |
| Applications: | IF, WB |
| Recommended Dilution: | ELISA: 1/1,000. Western Blot: 1/100-1/500. Immunofluorescence: See: Cheng, Alan, et al. Genes & Dev. 2007 Oct 01;21(19):2399-2409. |
| Reactivity: | Human |
| Host: | Rabbit |
| Isotype: | Ig |
| Clonality: | Polyclonal |
| Immunogen: | KLH conjugated synthetic peptide between 1487~1516 amino acids from the C-terminal region of Human AGL |
| Specificity: | This antibody is specific to AGL/GDE (C-term). |
| Formulation: | PBS with 0.09% (W/V) Sodium Azide as preservative. State: Purified State: Liquid purified Ig fraction. |
| Concentration: | lot specific |
| Purification: | Protein A Chromatography, eluted with high and low pH buffers and neutralized immediately, followed by dialysis against PBS. |
| Conjugation: | Unconjugated |
| Storage: | Store the antibody undiluted at 2-8°C for one month or (in aliquots) at -20°C for longer. Avoid repeated freezing and thawing. |
| Stability: | Shelf life: one year from despatch. |
| Gene Name: | amylo-alpha-1, 6-glucosidase, 4-alpha-glucanotransferase |
| Database Link: | Entrez Gene 178 Human P35573 |



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Background:

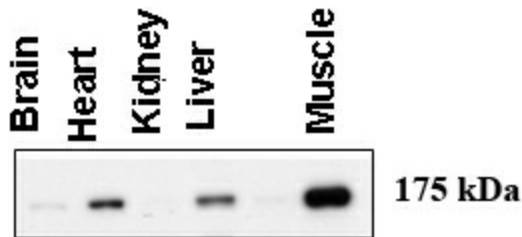
AGL is a glycogen debrancher enzyme which is involved in glycogen degradation. This enzyme has two independent catalytic activities which occur at different sites on the protein: a 4- α -glucotransferase activity and a amylo-1,6-glucosidase activity. Mutations in the AGL gene are associated with glycogen storage disease although a wide range of enzymatic and clinical variability occurs which may be due to tissue-specific alternative splicing.

Synonyms:

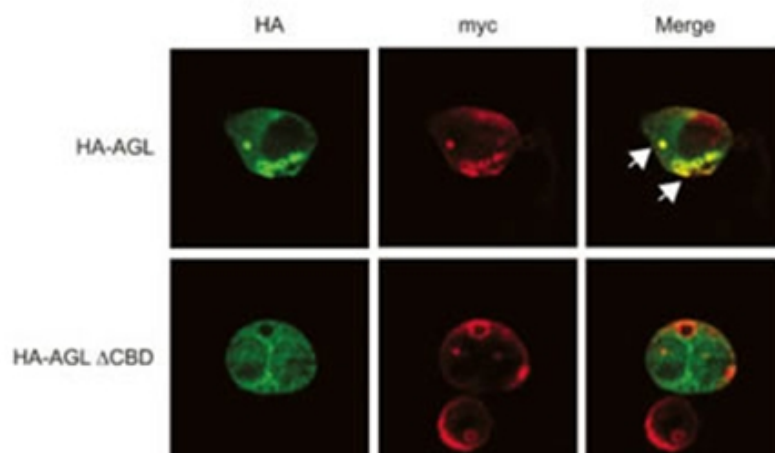
Glycogen debranching enzyme

Note:

Predicted Molecular weight: 174764 Da

Product images:


Western blot using anti-AGL (C-term) antibody at 1:500 dilution. A total of 20 μ g of lysates was loaded for each tissue. Data courtesy of Dr. Alan Cheng, Department of Internal Medicine, Life Sciences Institute, University of Michigan Medical Center, Ann Arbor, Michigan.



Expression of myc-GS causes wild type but not the Δ CBD mutant of AGL to aggregate around the PAS-stain-positive inclusions. HepG2 cells were transfected with either HA-tagged wild-type AGL (HA-AGL) or HA-AGL Δ CBD. Cells were fixed in formalin and processed for IF using anti-HA (green) and anti-myc (red) antibodies. White arrows indicate colocalization of HA-AGL and myc-GS.