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GLUT2 Antibody
Catalog # ASM10554

Specification

GLUT2 Antibody - Product Information

Application	**WB**
Primary Accession	P11168
Other Accession	NP_000331.1
Host	**Rabbit**
Reactivity	**Human**
Clonality	**Polyclonal**

Description
Rabbit Anti-Human GLUT2 Polyclonal

Target/Specificity
Predicted molecular weight at ~57.5kDa. Observed molecular weight at 60-70kDa and 38-45kDa.

Other Names
Glucose Transporter 2 Antibody, Glucose Transporter GLUT2 Antibody, BLUT-2 Antibody, GTR_Human Antibody, SLC2a2 Antibody, Solute Carrier Family 2 (facilitated glucose transporter) member 2 Antibody

Immunogen
Synthetic peptide from the C-terminal of human GLUT2

Purification
Peptide Affinity Purified

Storage	**-20ºC**

Storage Buffer
PBS, 50% glycerol, 0.09% sodium azide

Shipping Temperature	**Blue Ice or 4ºC**

Certificate of Analysis
A 1:1000 dilution of SPC-697 was sufficient for detection of GLUT2 on 293T Rapamycin-treated lysates using Goat anti-rabbit IgG:HRP as the secondary antibody.

Cellular Localization
Membrane

GLUT2 Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence

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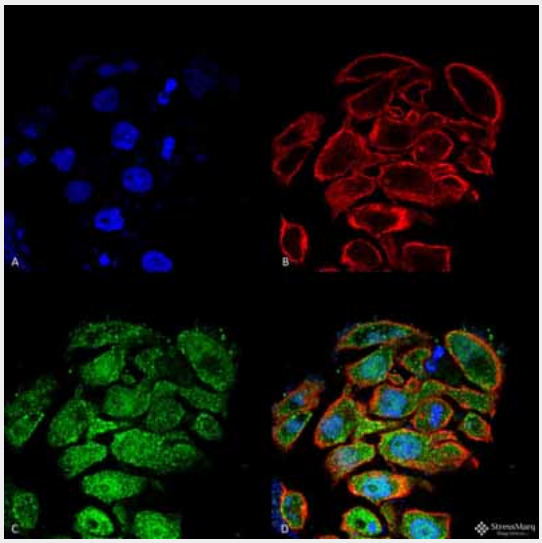
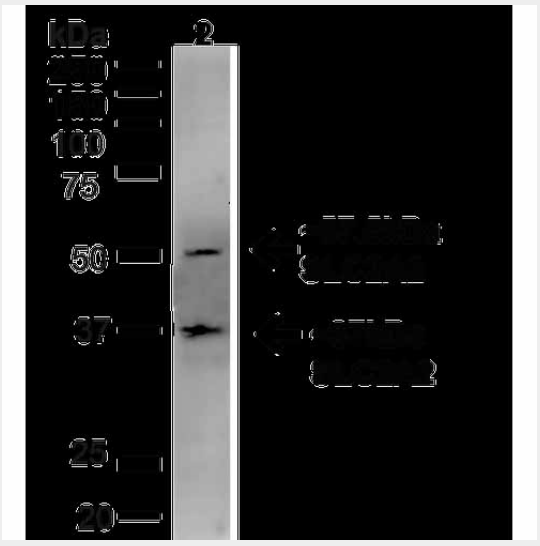
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

GLUT2 Antibody - Images



Immunocytochemistry/Immunofluorescence analysis using Rabbit Anti-GLUT2 Polyclonal Antibody (ASM10554). Tissue: Colon cancer cell line (HT-29). Species: Human. Fixation: 4% Formaldehyde for 15 min at RT. Primary Antibody: Rabbit Anti-GLUT2 Polyclonal Antibody (ASM10554) at 1:100 for 60 min at RT. Secondary Antibody: Goat Anti-Rabbit ATTO 488 at 1:100 for 60 min at RT. Counterstain: Phalloidin Texas Red F-Actin stain; DAPI (blue) nuclear stain at 1:1000, 1:5000 for 60min RT, 5min RT. Localization: Cytoplasm, membrane. Magnification: 60X. (A) DAPI (blue) nuclear stain (B) Phalloidin Texas Red F-Actin stain (C) GLUT2 Antibody (D) Composite.



Western blot analysis of Rat Liver showing detection of ~57.5kDa GLUT2 protein using Rabbit Anti-GLUT2 Polyclonal Antibody (ASM10554). Lane 1: MW Ladder. Lane 2: Rat Liver (20 µg). Load: 20 µg. Block: 5% milk + TBST for 1 hour at RT. Primary Antibody: Rabbit Anti-GLUT2 Polyclonal Antibody (ASM10554) at 1:1000 for 1 hour at RT. Secondary Antibody: Goat Anti-Rabbit: HRP at 1:2000 for 1 hour at RT. Color Development: TMB solution for 12 min at RT. Predicted/Observed Size: ~57.5kDa. Other Band(s): ~37kDa.

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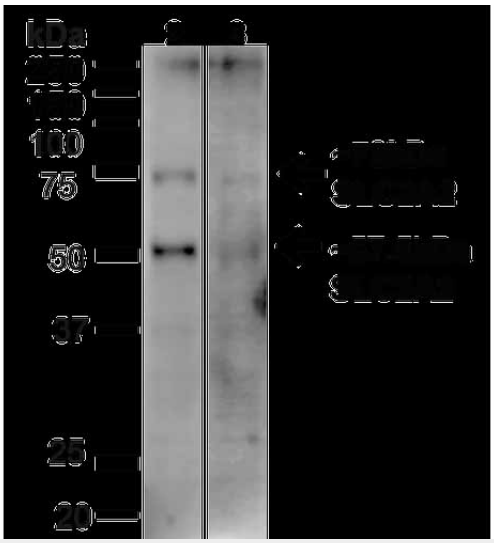

Western blot analysis of Human HeLa and 293T cell lysates showing detection of ~57.5kDa GLUT2 protein using Rabbit Anti-GLUT2 Polyclonal Antibody (ASM10554). Lane 1: MW Ladder. Lane 2: Human HeLa (20 µg). Lane 3: Human 293T (20 µg). Load: 20 µg. Block: 5% milk + TBST for 1 hour at RT. Primary Antibody: Rabbit Anti-GLUT2 Polyclonal Antibody (ASM10554) at 1:1000 for 1 hour at RT. Secondary Antibody: Goat Anti-Rabbit: HRP at 1:2000 for 1 hour at RT. Color Development: TMB solution for 12 min at RT. Predicted/Observed Size: ~57.5kDa. Other Band(s): ~72kDa.

GLUT2 Antibody - Background

Glucose transporter 2 (GLUT2) also known as solute carrier family 2 (facilitated glucose transporter), member 2 (SLC2A2) is a transmembrane carrier protein that enables protein facilitated glucose movement across cell membranes. It is the principal transporter for transfer of glucose between liver and blood, and has a role in renal glucose reabsorption (1). Mutations in SLC2A2 lead to Fanconi-Bickel syndrome (FBS), which results in hepatorenal glycogen accumulation, proximal renal tubular dysfunction, and impaired utilization of glucose and galactose. Recent studies have shown that mutations in SLC2A2 can cause neonatal diabetes, and therefore may contribute to human insulin secretion (2). Novel SLC2A2 mutations have also been discovered and are being investigated to determine their roles in FBS as well (3).

GLUT2 Antibody - References

1. Kellett G.L., Brot-Laroche E. (2005) Diabetes. 54(10): 3056-3062.
2. Sansbury F.H., et al. (2012) Diabetologia. 55(9): 2381-2385.
3. Su Z., et al. (2011) J Perdiatr Endocrinol Metab. 24(9-10): 749-753.