

Glypican 3 (GPC3) Antibody (Center) Blocking peptide
Synthetic peptide
Catalog # BP6339c**Specification**

Glypican 3 (GPC3) Antibody (Center) Blocking peptide - Product InformationPrimary Accession [P51654](#)**Glypican 3 (GPC3) Antibody (Center) Blocking peptide - Additional Information****Gene ID** 2719**Other Names**

Glypican-3, GTR2-2, Intestinal protein OCI-5, MXR7, Secreted glypican-3, GPC3, OCI5

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP6339c](/product/products/AP6339c) was selected from the Center region of human GPC3. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Glypican 3 (GPC3) Antibody (Center) Blocking peptide - Protein Information**Name** GPC3**Synonyms** OCI5**Function**

Cell surface proteoglycan (PubMed:[14610063](http://www.uniprot.org/citations/14610063)). Negatively regulates the hedgehog signaling pathway when attached via the GPI- anchor to the cell surface by competing with the hedgehog receptor PTC1 for binding to hedgehog proteins (By similarity). Binding to the hedgehog protein SHH triggers internalization of the complex by endocytosis and its subsequent lysosomal degradation (By similarity). Positively regulates the canonical Wnt signaling pathway by binding to the Wnt receptor Frizzled and stimulating the binding of the Frizzled receptor to Wnt ligands (PubMed:[16227623](http://www.uniprot.org/citations/16227623), PubMed:[24496449](http://www.uniprot.org/citations/24496449)). Positively regulates the non-canonical Wnt signaling pathway (By similarity). Binds to CD81 which decreases

the availability of free CD81 for binding to the transcriptional repressor HHEX, resulting in nuclear translocation of HHEX and transcriptional repression (By similarity). Inhibits the dipeptidyl peptidase activity of DPP4 (PubMed:17549790). Plays a role in limb patterning and skeletal development by controlling the cellular response to BMP4 (By similarity). Modulates the effects of growth factors BMP2, BMP7 and FGF7 on renal branching morphogenesis (By similarity). Required for coronary vascular development (By similarity). Plays a role in regulating cell movements during gastrulation (By similarity).

Cellular Location

Cell membrane; Lipid-anchor, GPI-anchor {ECO:0000250|UniProtKB:P13265}; Extracellular side {ECO:0000250|UniProtKB:P13265}

Tissue Location

Detected in placenta (at protein level) (PubMed:32337544). Highly expressed in lung, liver and kidney

Glypican 3 (GPC3) Antibody (Center) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

Glypican 3 (GPC3) Antibody (Center) Blocking peptide - Images**Glypican 3 (GPC3) Antibody (Center) Blocking peptide - Background**

GPC3 is a cell surface proteoglycan that bears heparan sulfate. This protein may be involved in the suppression/modulation of growth in the predominantly mesodermal tissues and organs, and may play a role in the modulation of IGF2 interactions with its receptor and thereby modulate its function. Members of the glypican-related integral membrane proteoglycan family contain a core protein anchored to the cytoplasmic membrane via a glycosyl phosphatidylinositol (GPI) linkage. These proteins may play a role in the control of cell division, growth regulation, and tumor predisposition. Deletion mutations in GPC3 are the cause of Simpson-Golabi-Behmel syndrome (SGBS), also known as Simpson dysmorphia syndrome (SDYS). SGBS is a condition characterized by pre- and postnatal overgrowth (gigantism) with visceral and skeletal anomalies.

Glypican 3 (GPC3) Antibody (Center) Blocking peptide - References

Nakatsura, T., et al., Clin. Cancer Res. 10(19):6612-6621 (2004).Boily, G., et al., Br. J. Cancer 90(8):1606-1611 (2004).Wichert, A., et al., Oncogene 23(4):945-955 (2004).Midorikawa, Y., et al., Int. J. Cancer 103(4):455-465 (2003).Sung, Y.K., et al., Cancer Sci. 94(3):259-262 (2003).