

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human GFR α -1/GDNF R α -1 in direct ELISAs and Western blots. In direct ELISAs, approximately 10% cross-reactivity with recombinant rat GFR α -1 is observed and less than 5% cross-reactivity with recombinant human (rh) GFR α -2, rhGFR α -3, and rhGFR α -4 is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human GFR α -1/GDNF R α -1 Met1-Lys429 Accession # NP_665736
Endotoxin Level	<0.10 EU per 1 μ g of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 μ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 μ m filtered solution in PBS.

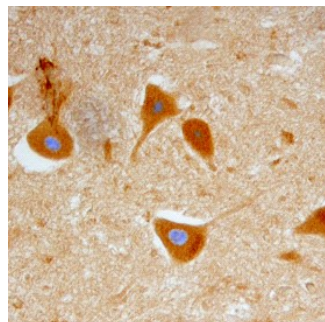
APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 μ g/mL	Recombinant Human GFR α -1/GDNF R α -1 Fc Chimera (Catalog # 714-GR)
Immunohistochemistry	5-15 μ g/mL	See Below

DATA

Immunohistochemistry



GFR α -1/GDNF R α -1 in Human Spinal Cord. GFR α -1/GDNF R α -1 was detected in immersion fixed paraffin-embedded sections of human spinal cord using Goat Anti-Human GFR α -1/GDNF R α -1 Antigen Affinity-purified Polyclonal Antibody (Catalog # AF714) at 15 μ g/mL overnight at 4 °C. Tissue was stained using the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). Specific staining was localized to ventral horn motoneurons. View our protocol for [Chromogenic IHC Staining of Paraffin-embedded Tissue Sections](#).

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Glial cell line-derived growth factor (GDNF), neurturin (NTN), artemin and persephin are distant members of the TGF- β superfamily. They function as neurotrophic factors for a variety of neuronal populations in the central and peripheral nervous systems. The bioactivities of GDNF and NTN are mediated through a receptor complex composed of the non ligand-binding signaling subunit (c-Ret receptor tyrosine kinase) and either of two ligand binding subunits [GDNF receptor α -1 (GFR α -1) or GFR α -2]. GFR α -1 and -2 are members of a family of at least four cysteine-rich glycosyl-phosphatidylinositol (GPI)-linked cell surface proteins that share conserved placements of many of their cysteine residues. Binding of GDNF to membrane-associated GFR α -1 or GFR α -2 initiates the association with and activation of the Ret tyrosine kinase. Soluble GFR α s released enzymatically from the cell surface-associated protein with phosphatidylinositol phospholipase C, as well as recombinantly produced soluble GFR α -1, can also bind with high-affinity to GDNF and trigger the activation of Ret tyrosine kinase.

Human GFR α -1 cDNA encodes a 465 amino acid (aa) residue protein with an N-terminal 24 aa residue hydrophobic signal peptide. Like other GPI-linked proteins, human GFR α -1 has a C-terminal hydrophobic region which is preceded by a three aa residue (ASS) GPI-binding site. Human GFR α -1 shares 93% aa identity with rat GFR α -1. The expression of the various GFR α s are differentially regulated in the central and peripheral nervous system, suggesting complementary roles for the GFR α s in mediating the activities of the GDNF family of neurotrophic factors.

References:

1. Thompson, J. *et al.* (1998) Mol. Cell Neurosci. **11**:117.
2. Trupp, M. *et al.* (1998) Mol. Cell Neurosci. **11**:47.
3. Baloh, R.H. *et al.* (1998) Proc. Natl. Acad. Sci. USA **95**:5801.