

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived
Gln22-Gly205, with a C-terminal 6-His tag
Accession # NP_848660

N-terminal Sequence Analysis No results obtained: Gln22 predicted

Predicted Molecular Mass 22 kDa

SPECIFICATIONS

SDS-PAGE 25-29 kDa, reducing conditions

Activity Measured by its ability to induce Topflash reporter activity in HEK293T human embryonic kidney cells.
The typical ED₅₀ is 0.7-2.8 ng/mL in the presence of 5 ng/mL recombinant mouse Wnt-3a.

Endotoxin Level <0.01 EU per 1 µg of the protein by the LAL method.

Purity >90%, by SDS-PAGE under reducing conditions and visualized by silver stain.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 200 µg/mL in sterile PBS.

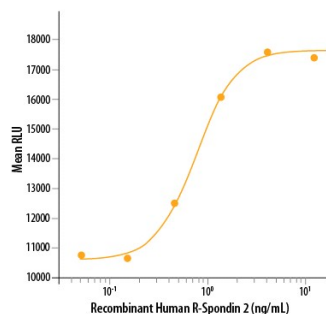
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

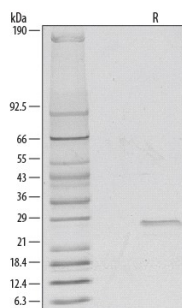
DATA

Bioactivity



Recombinant R-Spondin 2 (Catalog # 3266-RS/CF) induces activation of β-catenin response in a Topflash Luciferase assay using HEK293T human embryonic kidney cells. The ED₅₀ for this effect is 0.7-2.8 ng/mL in the presence of 5 ng/mL of Recombinant Mouse Wnt-3a (Catalog # 1324-WN).

SDS-PAGE



1 µg/lane of Recombinant Human R-Spondin 2 was resolved with SDS-PAGE and visualized by silver staining under reducing (R) conditions, showing a single band at 28 kDa.

BACKGROUND

Roof plate-specific Spondin 2 isoform 1 (R-Spondin 2, RSPO2), also known as cysteine-rich and single thrombospondin domain containing protein 2 (Cristin 2), is a 33 kDa secreted protein that belongs to the R-Spondin family (1-3). The four R-Spondins regulate Wnt/ β -catenin signaling and overlap in expression and function (1-3). Like other R-Spondins, RSPO2 contains two adjacent cysteine-rich furin-like domains (aa 90-134) followed by a thrombospondin (TSP-1) motif (aa 144-204) and a C-terminal region rich in basic residues (aa 207-243). The basic region binds heparin and mediates cell surface retention and extracellular matrix attachment while the furin-like domains are required for Wnt/ β -catenin signaling (1, 3, 4). RSPO2 contains one potential N-glycosylation site. Mature human RSPO2 shares 97-98% aa identity with mouse, rat, equine, canine and bovine RSPO2 and ~40% aa identity with RSPO1, RSPO3 and RSPO4. Of the three reported splice isoforms of human R-Spondin 2, isoform 2 lacks residues 1 - 67 of isoform 1, while isoform 3 has a glycine substitution for residues 32-95 of isoform 1 (5). Human RSPO2 is expressed in organs of endodermal origin in adults, including intestine and lung, and is down-regulated in tumors of these tissues (1). In the embryonic mouse, RSPO2 expression is concentrated in the apical epidermal ridge, hippocampus, and developing muscle, teeth and bones (1, 6). Deletion of RSPO2 results in down-regulation of Wnt activity in these areas, malformations of the facial skeleton and limbs, and respiratory failure at birth (7-9). RSPO2 is an extracellular potentiator of Wnt/ β -catenin signaling (3, 4). It functions at least in part by binding LRP-6, stimulating its long-term phosphorylation and down-regulating its internalization (3, 4). RSPO proteins, especially RSPO2 and RSPO3, also antagonize DKK1 activity by interfering with DKK1-mediated LRP-6 and Kremen association (10).

References:

1. Kazanskaya, O. *et al.* (2004) *Dev. Cell* **7**:525.
2. Kim, K.-A. *et al.* (2006) *Cell Cycle* **5**:23.
3. Nam, J.-S. *et al.* (2006) *J. Biol. Chem.* **281**:13247.
4. Li, S.-J. *et al.* (2009) *Cell Signal.* **21**:916.
5. Swiss-Prot Accession # Q6UXX9.
6. Nam, J.-S. *et al.* (2007) *Gene Expr. Patterns* **7**:306.
7. Yamada, W. *et al.* (2009) *Biochem. Biophys. Res. Commun.* **381**:453.
8. Jin, Y.-R. *et al.* (2011) *Dev. Biol.* **352**:1.
9. Nam, J.-S. *et al.* (2007) *Dev. Biol.* **311**:124.
10. Kim, K.-A. *et al.* (2007) *Mol. Biol. Cell* **19**:2588.