

DESCRIPTION

Source	<i>E. coli</i> -derived Ser21-Gly209, with an N-terminal Met Accession # Q9Z132
N-terminal Sequence Analysis	Met
Predicted Molecular Mass	21 kDa

SPECIFICATIONS

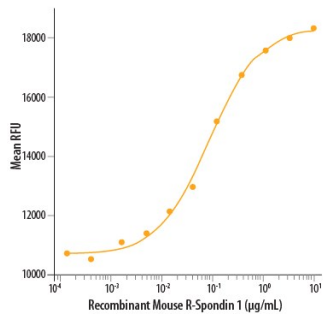
Activity	Measured by its ability to induce Topflash reporter activity in HEK293T human embryonic kidney cells. The typical ED ₅₀ is 50-200 ng/mL in the presence of 5 ng/mL recombinant mouse Wnt-3a.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 250 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	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <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. • 3 months, -20 to -70 °C under sterile conditions after reconstitution.

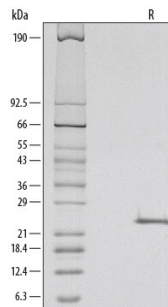
DATA

Bioactivity



Recombinant Mouse R-Spondin 1 (Catalog # 3474-RS) induces activation of β-catenin response in a Topflash Luciferase assay using HEK293T human embryonic kidney cells. The ED₅₀ for this effect is 50-200 ng/mL in the presence of 5 ng/mL of Recombinant Mouse Wnt-3a (Catalog # 1324-WN).

SDS-PAGE



1 µg/lane of Recombinant Mouse R-Spondin-1 was resolved with SDS-PAGE under reducing (R) conditions and visualized by silver staining, showing a single band at 24 kDa.

BACKGROUND

R-Spondin 1 (RSPO1, Roof plate-specific Spondin 1), also known as cysteine-rich and single thrombospondin domain containing protein 3 (Cristin 3), is a 27 kDa secreted protein that belongs to the R-Spondin family (1, 2). R-Spondins share around 40% aa identity. All regulate Wnt/ β -catenin signaling, but have distinct expression patterns (1-3). Like other R-spondins, R-Spondin 1 contains two adjacent cysteine-rich furin-like domains (amino acids (aa) 34-135) followed by a thrombospondin (TSP-1) motif (aa 147-207) and a region rich in basic residues (aa 211-263). Only the furin-like domains are needed for β -catenin stabilization (2, 4). A putative nuclear localization signal at the C-terminus may allow some expression in the nucleus (5). R-Spondin 1 contains one potential N-glycosylation site. Over aa 21 - 209, mouse R-Spondin 1 shares 98%, 94%, 94%, 93%, 92% and 88% aa identity with rat, human, horse, cow, goat and dog RSPO-1, respectively. R-Spondin 1 is expressed in early development at the roof plate boundary and is thought to contribute to dorsal neural tube development (3, 5). In humans, rare disruptions of the R-Spondin 1 gene are associated with tendencies for XX sex reversal (phenotypic male) or hermaphroditism, indicating a role for R-Spondin 1 in gender-specific differentiation (6, 7). Disruption is also associated with palmoplantar keratosis (6, 7). Postnatally, R-Spondin 1 is expressed by neuroendocrine cells in the intestine, adrenal gland and pancreas, and by epithelia in kidney and prostate (8). Injection of recombinant R-Spondin 1 in mice causes activation of β -catenin and proliferation of intestinal crypt epithelial cells, and ameliorates experimental colitis (8, 9). R-Spondin 1 appears to regulate Wnt/ β -catenin by competing with the Wnt antagonist DKK-1 for binding to the Wnt co-receptor, Kremen (10). This competition reduces internalization of DKK-1/LRP-6/Kremen complexes (10). Reports differ on whether R-Spondin 1 binds LRP-6 directly (10 - 12).

References:

1. Lowther, W. *et al.* (2005) *J. Virol.* **79**:10093.
2. Kim, K.-A. *et al.* (2006) *Cell Cycle* **5**:23.
3. Nam, J.-S. *et al.* (2007) *Gene Expr. Patterns* **7**:306.
4. Kazanskaya, O. *et al.* (2004) *Dev. Cell* **7**:525.
5. Kamata, T. *et al.* (2004) *Biochim. Biophys. Acta* **1676**:51.
6. Tomaselli, S. *et al.* (2008) *Hum. Mutat.* **29**:220.
7. Parma, P. *et al.* (2006) *Nat. Genet.* **38**:1304.
8. Kim, K.-A. *et al.* (2005) *Science* **309**:1256.
9. Zhao, J. *et al.* (2007) *Gastroenterology* **132**:1331.
10. Binnerts, M.E. *et al.* (2007) *Proc. Natl. Acad. Sci. USA* **104**:14700.
11. Nam, J.-S. *et al.* (2006) *J. Biol. Chem.* **281**:13247.
12. Wei, Q. *et al.* (2007) *J. Biol. Chem.* **282**:15903.