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**Product Data Sheet**

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Product Name: RR6  
Cat. No.: GC32740

**Chemical Properties**

Cas. No. 1351758-37-6

SMILES CC(C)(CO)[C@@H](O)C(NCCC(CC1=CC=CC=C1)=O)=O

Formula  $C_{16}H_{23}NO_4$  M.Wt 293.36

Solubility DMSO :  $\geq 100$  mg/mL (340.88 mM) Storage Store at  $-20^{\circ}C$

General tips For obtaining a higher solubility , please warm the tube at  $37^{\circ}C$  and shake it in the ultrasonic bath for a while. Stock solution can be stored below  $-20^{\circ}C$  for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

Structure

**Protocol****Cell experiment****[1]:**

Cell lines HepG2 cells

**Caution: Product has not been fully validated for medical applications. For research use only.**

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**Preparation Method** HepG2 cells were cultured in complete Dulbecco's Modified Eagle Medium (DMEM) supplemented with 4.5g/L glucose, 2mM L-glutamine, 1mM sodium pyruvate, 100U/mL penicillin-streptomycin, and 10% (v/v) fetal bovine serum (FBS) at 37°C in a humidified 5% CO<sub>2</sub> atmosphere. Cells were seed in a six-well plate. After the confluence rate was approximately 70%, 2µg of plasmid DNA (human Vanin-1 gene, cDNA clone expressing plasmid with the OFPSpark tag) and 2.5µL of lipofectamine 2000 were added to the serum-free medium. After 6h of culture, the culture medium was replaced with a complete medium for another 24h. Then cells were treated with or without 100µM RR6 for 30min. After treatments, all of the experimental cells were incubated with Cy-Pa at 37°C for 30min, and imaging was performed by laser scanning confocal microscopy.

**Reaction Conditions** 100µM; 30 min

**Applications** RR6 lowered intracellular Vanin-1 level and raised total GSH level in HepG2 cells.

**Animal experiment [2]:**

**Animal models** Male Zucker Diabetic Fatty rats

**Preparation Method** Male Zucker Diabetic Fatty (ZDF) rats of 8 weeks old were housed in a temperature (23±1°C) and humidity (50%) controlled animal facility with 12:12h light-dark cycle. Food and water were available ad libitum. Rats were treated with the pantetheinase (vanin) inhibitor RR6 (3mg/mL; dissolved in drinking water) or vehicle for 8 days. Rats were sacrificed at the end of the RR6 treatment (non-fasted; between 9 am and 11am) and blood and organs were collected for further analysis. Each test was repeated three times, and the data was averaged.

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Dosage form            3mg/mL; drinking water; 8 days

Applications            RR6 almost completely suppressed plasma vanin activity but did not alter hepatic steatosis, insulin sensitivity or glucose production in ZDF diabetic rats.

### References:

[1] Lu P, Zhang C, Fu L, et al. Near-Infrared Fluorescent Probe for Imaging and Evaluating the Role of Vanin-1 in Chemotherapy.

*Anal Chem.*

2021;93(29):10378-10387.

[2] van Diepen JA, Jansen PA, Ballak DB, et al. Genetic and pharmacological inhibition of vanin-1 activity in animal models of type 2 diabetes. *Sci Rep.* 2016;6:21906.

### Background

RR6 is a potent, selective, reversible, competitive and orally active vanin inhibitor with an IC<sub>50</sub> of 540nM for recombinant vanin-1<sup>[1]</sup>. Vanin is a membrane-bound pantetheinase that hydrolyzes pantetheine to cysteamine and regulates oxidative stress and inflammation<sup>[2]</sup>. RR6 is usually used in the studies of infection, inflammation and

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metabolism<sup>[3][4]</sup>.

In vitro, RR6 (100 $\mu$ M; 30min) lowered intracellular Vanin-1 level and raised total GSH level in HepG2 cells<sup>[5]</sup>.

In vivo, RR6 (3mg/mL; drinking water; 8 days) almost completely suppressed plasma vanin activity but did not alter hepatic steatosis, insulin sensitivity or glucose production in ZDF diabetic rats<sup>[6]</sup>. RR6 (3mg/mL; drinking water; 4 weeks) reduced neointima area and neointima/media ratio, and suppressed neointimal macrophage influx in Dark-Agouti-to-Brown-Norway rat aortic allografts<sup>[7]</sup>.

### References:

- [1] Jansen PA, van Diepen JA, Ritzen B, et al. Discovery of small molecule vanin inhibitors: new tools to study metabolism and disease. *ACS Chem Biol*. 2013;8(3):530-534.
- [2] Bartucci R, Salvati A, Olinga P, Boersma YL. Vanin 1: Its Physiological Function and Role in Diseases. *Int J Mol Sci*. 2019;20(16):3891.
- [3] Schalkwijk J, Jansen P. Chemical biology tools to study pantetheinases of the vanin family. *Biochem Soc Trans*. 2014;42(4):1052-1055.
- [4] Lv L, Wang T, Xie W, et al. Revealing VNN1: An Emerging and Promising Target for Inflammation and Redox Balance. *Immun Inflamm Dis*. 2025;13(10):e70274.
- [5] Lu P, Zhang C, Fu L, et al. Near-Infrared Fluorescent Probe for Imaging and Evaluating the Role of Vanin-1 in Chemotherapy. *Anal Chem*. 2021;93(29):10378-10387.
- [6] van Diepen JA, Jansen PA, Ballak DB, et al. Genetic and pharmacological inhibition of vanin-1 activity in animal models of type 2 diabetes. *Sci Rep*. 2016;6:21906.
- [7] Wedel J, Jansen PA, Botman PN, Rutjes FP, Schalkwijk J, Hillebrands JL. Pharmacological Inhibition of Vanin Activity Attenuates Transplant Vasculopathy in Rat Aortic Allografts. *Transplantation*. 2016;100(8):1656-1666.

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