Small Molecules

GlyH-101

Inhibits cystic fibrosis transmembrane conductance regulator (CFTR)

Catalog #100-0530	5 mg
100-0531	10 mg

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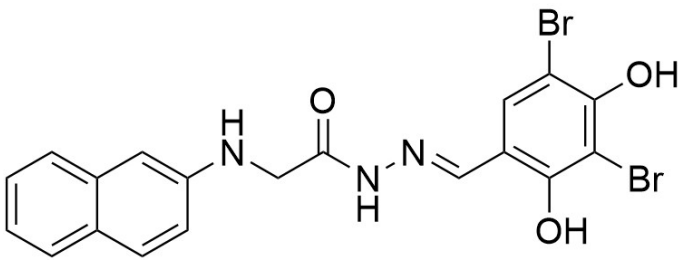
Product Description

GlyH-101 is a cell-permeable glycine hydrazide that selectively and reversibly blocks the cystic fibrosis transmembrane conductance regulator (CFTR) channel (K_i = 4.3 µM; Sonawane et al.). The CFTR protein is a chloride anion channel involved in the secretion of fluid in many epithelial tissues, such as the airways and intestine (Ma et al.). Defects in the CFTR gene alter ion transport, which can lead to cystic fibrosis (Dalli et al.; Ma et al.).

Molecular Name:	GlyH-101
Alternative Names:	CFTR inhibitor II
CAS Number:	328541-79-3
Chemical Formula:	$C_{19}H_{15}Br_2N_3O_3$
Molecular Weight:	493.2 g/mol
Purity:	≥ 95%
Chemical Name:	2-[(3,5-dibromo-2,4-dihydroxyphenyl)methylene]hydrazide N-2-naphthalenyl-glycine
Structure:	



Properties

Physical Appearance:	A crystalline solid
Storage:	Product stable at -20°C as supplied. Protect product from prolonged exposure to light. For long-term storage, store with a desiccant. Stable as supplied for 12 months from date of receipt.
Solubility:	• DMSO ≤ 50 mM For example, to prepare a 10 mM stock solution in DMSO, resuspend 1 mg in 203 µL of DMSO. Prepare stock solution fresh before use. Information regarding stability of small molecules in solution has rarely been reported, however, as a general guide we recommend storage in DMSO at -20°C. Aliquot into working volumes to avoid repeated freeze-thaw cycles. The effect of storage of stock solution on compound performance should be tested for each application. Compound has low solubility in aqueous media. For use as a cell culture supplement, stock solution should be diluted into culture medium immediately before use. Avoid final DMSO concentration above 0.1% due to potential cell toxicity.

Published Applications

CANCER RESEARCH
· Blocks CFTR and inhibits cell division by inducing hyperpolarization in human gastric cancer cells (Zhu et al.).

References

Dalli J et al. (2010) CFTR inhibition provokes an inflammatory response associated with an imbalance of the annexin A1 pathway. Am J Pathol 177(1): 176–86.
Ma T et al. (2002) Thiazolidinone CFTR inhibitor identified by high-throughput screening blocks cholera toxin-induced intestinal fluid secretion. J Clin Invest 110(11): 1651–8.
Sonawane ND et al. (2006) Luminally active, nonabsorbable CFTR inhibitors as potential therapy to reduce intestinal fluid loss in cholera. FASEB J 20(1): 130–2.
Zhu L et al. (2018) Involvement of AMP-activated protein kinase (AMPK) in regulation of cell membrane potential in a gastric cancer cell line. Sci Rep 8(1): 6028.

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