

PRODUCT INFORMATION



ADH-1

Item No. 29981

CAS Registry No.: 229971-81-7

Formal Name: N-acetyl-L-cysteiny-L-histidyl-L-alanyl-L-valyl-L-cysteinamide, cyclic (1→5)-disulfide

Synonym: N-Ac-CHAVC-NH₂

MF: C₂₂H₃₄N₈O₆S₂

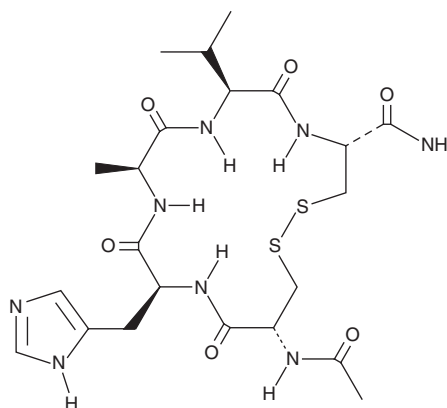
FW: 570.7

Purity: ≥98%

Supplied as: A solid

Storage: -20°C

Stability: ≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

ADH-1 is supplied as a solid. A stock solution may be made by dissolving the ADH-1 in the solvent of choice, which should be purged with an inert gas. ADH-1 is soluble in DMSO and aqueous acid (5% HCl in H₂O).

Description

ADH-1 is a cyclic peptide antagonist of N-cadherin.¹ It inhibits neurite outgrowth in cerebellar neurons cultured on N-cadherin-expressing 3T3 cell monolayers (IC₅₀ = 0.323 mM). ADH-1 (0.2 mg/ml) inhibits cell scattering and motility induced by collagen I in Capan-1 cells and wild-type and N-cadherin-overexpressing BxPC-3 cells.² It induces apoptosis in N-cadherin-overexpressing, but not knockdown, BxPC-3 cells when used at a concentration of 1 mg/ml. ADH-1 (50 mg/kg) reduces tumor growth in an N-cadherin-overexpressing BxPC-3 mouse xenograft model.

References

1. Williams, E., Williams, G., Gour, B.J., *et al.* A novel family of cyclic peptide antagonists suggests that N-cadherin specificity is determined by amino acids that flank the HAV motif. *J. Biol. Chem.* **275**(6), 4007-4012 (2000).
2. Shintani, Y., Fukumoto, Y., Chaika, N., *et al.* ADH-1 suppresses N-cadherin-dependent pancreatic cancer progression. *Int. J. Cancer* **122**(1), 71-77 (2008).

WARNING

THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA

This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the [complete](#) Safety Data Sheet, which has been sent via email to your institution.

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