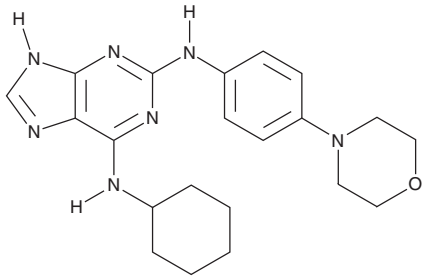
PRODUCT INFORMATION

Reversine

Item No. 10004412

CAS Registry No.:	656820-32-5
Formal Name:	N^6-cyclohexyl-N^2-[4-(4-morpholinyl) phenyl]-9H-purine-2,6-diamine
MF:	$C_{21}H_{27}N_7O$
FW:	393.5
Purity:	≥95%
UV/Vis.:	λ_{max}: 283 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



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

Laboratory Procedures

Reversine is supplied as a crystalline solid. A stock solution may be made by dissolving the reversine in the solvent of choice, which should be purged with an inert gas. Reversine is soluble in organic solvents such as DMSO and dimethyl formamide (DMF). The solubility of reversine in these solvents is approximately 10 and 20 mg/ml, respectively.

Reversine is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, reversine should first be dissolved in DMF and then diluted with the aqueous buffer of choice. Reversine has a solubility of approximately 0.5 mg/ml in a 1:8 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Reversine is a 2,6-disubstituted purine derivative that was originally found to induce dedifferentiation of C2C12 culture myoblast cells into stem cell progenitors when used at a concentration of 5 µM for four days.[1] Depending on cell type, reversine promotes either differentiation or dedifferentiation. For example, in NT2 neuronal and HL-60 human promyelocytic leukemia cells, it induces differentiation.[2] It inhibits the Aurora A, B, and C kinases with IC_{50} values of 98-876 nM and acts as an antagonist at the adenosine A_3 receptor with a K_i value of 0.66 µM.[2-4] Reversine is also used for studies of chromosome segregation. It inhibits the mitotic spindle checkpoint enzyme MPS1 with IC_{50} values of 6 and 2.8 nM for its kinase domain and full-length version, respectively).[4] Reversine induces autophagy in WRO human follicular thyroid cancer cells and decreases Akt/mTOR signaling.[5]

References

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2. D'Alise, A.M., Amabile, G., Iovino, M., *et al. Mol. Cancer Ther.* **7(5)**, 1140-1149 (2008).
3. Perreira, M., Jiang, J.K., Klutz, A.M., *et al. J. Med. Chem.* **48(15)**, 4910-4918 (2005).
4. Santaguida, S., Tighe, A., D'Alise, A.M., *et al. J. Cell. Biol.* **190(1)**, 73-87 (2010).
5. Lu, C.-H., Liu, Y.-W., Hua, S.-C., *et al. Biomed. Pharmacother.* **66(8)**, 642-647 (2012).

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