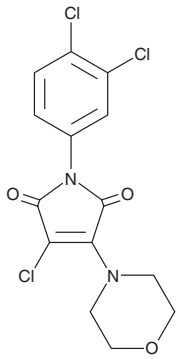
PRODUCT INFORMATION

RI-1

Item No. 24182



CAS Registry No.:	415713-60-9
Formal Name:	3-chloro-1-(3,4-dichlorophenyl)-4-(4-morpholinyl)-1H-pyrrole-2,5-dione
MF:	$C_{14}H_{11}Cl_3N_2O_3$
FW:	361.6
Purity:	≥98%
UV/Vis.:	λ_{max}: 255, 397 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

RI-1 is supplied as a crystalline solid. A stock solution may be made by dissolving the RI-1 in the solvent of choice. RI-1 is soluble in organic solvents such as DMSO and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of RI-1 in these solvents is approximately 2 and 13 mg/ml, respectively.

RI-1 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, RI-1 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. RI-1 has a solubility of approximately 0.3 mg/ml in a 1:2 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

RI-1 is an irreversible inhibitor of RAD51 (IC_{50}s = 5-30 µM), a protein that is central to homologous recombination during DNA repair and is overexpressed in a wide range of human cancer cell types.[1,2] RI-1 selectively inhibits homologous recombination without inhibiting single strand annealing.[1] It binds directly to the C319 thiol of RAD51 and inhibits oligomerization of RAD51 filaments. *In vitro*, RI-1 sensitizes cancer cells to chemotherapeutics that generate interstrand DNA crosslinks, including HEK293 cells co-treated with mitomycin C (LD_{50} = 16.62 µM).[1,2]

References

1. Budke, B., Logan, H.L., Kalin, J.H., *et al.* RI-1: A chemical inhibitor of RAD51 that disrupts homologous recombination in human cells. *Nucleic Acids Res.* **40(15)**, 7347-7357 (2012).
2. Budke, B., Kalin, J.H., Pawlowski, M., *et al.* An optimized RAD51 inhibitor that disrupts homologous recombination without requiring Michael acceptor reactivity. *J. Med. Chem.* **56(1)**, 254-263 (2013).

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