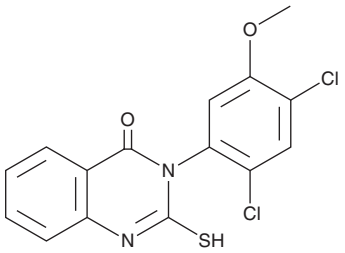
PRODUCT INFORMATION

Mdivi 1

Item No. 15559

CAS Registry No.:	338967-87-6
Formal Name:	3-(2,4-dichloro-5-methoxyphenyl)-2,3-dihydro-2-thioxo-4(1H)-quinazolinone
Synonym:	Mitochondrial Division Inhibitor 1
MF:	$C_{15}H_{10}Cl_2N_2O_2S$
FW:	353.2
Purity:	≥98%
UV/Vis.:	λ_{max}: 223, 295 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

Mdivi 1 is supplied as a crystalline solid. A stock solution may be made by dissolving the mdivi 1 in the solvent of choice, which should be purged with an inert gas. Mdivi 1 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of mdivi 1 in these solvents is approximately 20 mg/ml.

Mdivi 1 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, mdivi 1 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Mdivi 1 has a solubility of approximately 0.1 mg/ml in a 1:6 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Mdivi 1 is a mitochondrial division inhibitor.[1,2] It inhibits the GTPase activity of yeast, but not human, dynamin-1 (Dnm1; IC_{50} = ~1-10 µM for the recombinant yeast enzyme) and human dynamin-related protein 1 (DRP1) in A549 lung cancer cells when used at a concentration of 50 µM. Mdivi 1 (50 µM) inhibits mitochondrial fragmentation and DRP1 self-assembly induced by staurosporine (Item No. 81590) in COS cells overexpressing DRP1.[1] It induces apoptosis in, and inhibits colony formation of, A549 cells when used at concentrations of 25 and 50 µM, respectively.[2] Mdivi 1 (1 µM) decreases doxorubicin-induced increases in infarct-to-risk ratios in a Langendorff isolated perfused rat heart model of ischemia-reperfusion injury.[3] It reduces the severity of lung injury in a mouse model of LPS-induced acute lung injury when administered at a dose of 20 mg/kg.[4]

References

1. Cassidy-Stone, A., Chipuk, J.E., Ingerman, E., *et al*. Chemical inhibition of the mitochondrial division dynamin reveals its role in Bax/Bak-dependent mitochondrial outer membrane permeabilization. *Dev. Cell* **14(2)**, 193-204 (2008).
2. Wu, D., Dasgupta, A., Chen, K.-H., *et al*. Identification of novel dynamin-related protein 1 (Drp1) GTPase inhibitors: Therapeutic potential of Drpitor1 and Drpitor1a in cancer and cardiac ischemia-reperfusion injury. *FASEB J.* **34(1)**, 1447-1464 (2020).
3. Gharanei, M., Hussain, A., Janneh, O., *et al*. Attenuation of doxorubicin-induced cardiotoxicity by mdivi-1: A mitochondrial division/mitophagy inhibitor. *PLoS One* **8(10)**, e77713 (2013).
4. Deng, S., Zhang, L., Mo, Y., *et al*. Mdivi-1 attenuates lipopolysaccharide-induced acute lung injury by inhibiting MAPKs, oxidative stress and apoptosis. *Pulm. Pharmacol. Ther.* **62**, 101918 (2020).

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