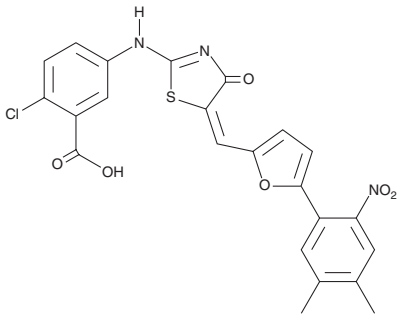
PRODUCT INFORMATION

PT1

Item No. 21335

CAS Registry No.:	331002-70-1
Formal Name:	2-chloro-5-[[5-[[5-(4,5-dimethyl-2-nitrophenyl)-2-furanyl]methylene]-4,5-dihydro-4-oxo-2-thiazolyl]amino]-benzoic acid
MF:	$C_{23}H_{16}ClN_3O_6S$
FW:	497.9
Purity:	≥95%
UV/Vis.:	λ_{max}: 272, 399 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

PT1 is supplied as a crystalline solid. A stock solution may be made by dissolving the PT1 in the solvent of choice, which should be purged with an inert gas. PT1 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of PT1 in DMSO and DMF is approximately 1 mg/ml. PT1 is sparingly soluble in ethanol.

PT1 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, PT1 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. PT1 has a solubility of approximately 0.2 mg/ml in a 1:4 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

PT1 is an activator of AMP-activated protein kinase (AMPK) that directly activates the inactive truncated forms of AMPK monomers $\alpha1_{335}$, $\alpha1_{394}$, and $\alpha2_{398}$ in a dose-dependent manner (EC_{50}s = ~ 8, 8, and 12 µM, respectively).[1] It stimulates the α1β1γ1 AMPK heterotrimer with an EC_{50} value of 0.3 µM.[1] PT1 dose-dependently increases phosphorylation of AMPK and its downstream substrate, acetyl-CoA carboxylase, without increasing the cellular AMP:ATP ratio in L6 myotubes.[1] In HepG2 liver cells, PT1 lowers lipid content through AMPK activation. PT1 induces autophagy in cardiomyocytes after oxygen glucose deprivation/reoxygenation, resulting in improved cell survival.[2]

References

1. Pang, T., Zhang, Z.-S., Gu, M., *et al*. Small molecule antagonizes autoinhibition and activates AMP-activated protein kinase in cells. *J. Biol. Chem.* **283(23)**, 16051-16060 (2008).
2. Huang, L., Dai, K.H., Chen, M., *et al*. The AMPK agonist PT1 and mTOR inhibitor 3HOI-BA-01 protect cardiomyocytes after ischemia through induction of autophagy. *J. Cardiovasc. Pharmacol. Ther.* **21(1)**, 70-81 (2016).

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