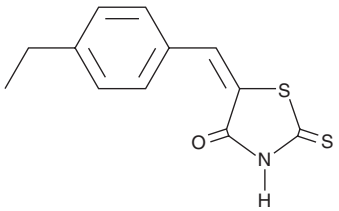
PRODUCT INFORMATION

10058-F4

Item No. 15929

CAS Registry No.:	403811-55-2
Formal Name:	5-[(4-ethylphenyl)methylene]-2-thioxo-4-thiazolidinone
MF:	$C_{12}H_{11}NOS_2$
FW:	249.4
Purity:	≥95%
UV/Vis.:	λ_{max}: 233, 277, 379 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

10058-F4 is supplied as a crystalline solid. A stock solution may be made by dissolving the 10058-F4 in the solvent of choice, which should be purged with an inert gas. 10058-F4 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of 10058-F4 in these solvents is approximately 0.3, 25, and 30, respectively.

10058-F4 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, 10058-F4 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. 10058-F4 has a solubility of approximately 0.5 in a 1:1 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

The Myc proto-oncogenes interact with Max to form a dimer that regulates gene transcription. The protein c-Myc, in particular, promotes gene expression relevant to cell growth and thus drives cancer. 10058-F4 is a cell-permeable thiazolidinone that inhibits the dimerization of c-Myc and Max at 64 µM, preventing c-Myc-dependent gene expression and cell proliferation.[1] It induces cell cycle arrest, apoptosis, and myeloid differentiation at 100 µM in human acute myeloid leukemia cells.[2] 10058-F4 is rapidly metabolized in mice when given intravenously, limiting its effects on tumors *in vivo*.[3] In addition to c-Myc, 10058-F4 inhibits the nuclear Myc protein, N-Myc, at 50 µM, inducing arrest, apoptosis, and differentiation in neuroblastoma cells overexpressing the gene for N-Myc.[4] This compound can be used to delineate novel actions of Myc proteins, especially those related to lipid and glucose metabolism.[4,5]

References

1. Yin, X., Giap, C., Lazo, J.S., *et al.* Low molecular weight inhibitors of Myc-Max interaction and function. *Oncogene* **22(40)**, 6151-6159 (2003).
2. Huang, M.-J., Cheng, Y.-C., Liu, C.-R., *et al.* A small-molecule c-Myc inhibitor, 10058-F4, induces cell-cycle arrest, apoptosis, and myeloid differentiation of human acute myeloid leukemia. *Exp. Hematol.* **34(11)**, 1480-1489 (2006).
3. Guo, J., Parise, R.A., Joseph, E., *et al.* Efficacy, pharmacokinetics, tissue distribution, and metabolism of the Myc-Max disruptor, 10058-F4 [Z,E]-5-[4-ethylbenzylidine]-2-thioxothiazolidin-4-one, in mice. *Cancer Chemother. Pharmacol.* **63(4)**, 615-625 (2009).
4. Zirath, H., Frenzel, A., Oliynyk, G., *et al.* MYC inhibition induces metabolic changes leading to accumulation of lipid droplets in tumor cells. *Proc. Natl. Acad. Sci. USA* **110(25)**, 10258-10263 (2013).
5. Zhang, P., Metukuri, M.R., Bindom, S.M., *et al.* c-Myc is required for the ChREBP-dependent activation of glucose-responsive genes. *Mol. Endocrinol.* **24(6)**, 1274-1286 (2010).

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