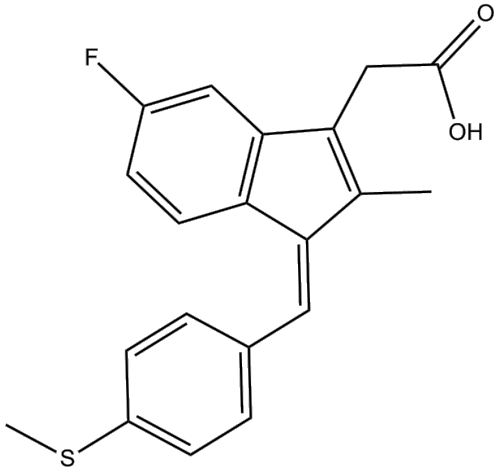
Product Data Sheet

Product Name: Sulindac sulfide
Cat. No.: GC15059

Chemical Properties

Cas. No.	49627-27-2		
Chemical Name	(Z)-2-(5-fluoro-2-methyl-1-(4-(methylthio)benzylidene)-1H-inden-3-yl)acetic acid		
SMILES	FC1=CC2=C(/C(C(C)=C2CC(O)=O)=C\C3=CC=C(SC)C=C3)C=C1		
Formula	$C_{20}H_{17}FO_2S$	M.Wt	340.41
Solubility	2mg/mL in ethanol, 30mg/mL in DMSO, or in DMF	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Background

Sulindac is a non-steroidal anti-inflammatory drug that has an extensive epidemiology documenting reduced human colorectal cancer. In mouse models, sulindac was found not only to inhibit the enzymatic activity of polyp-associated COX-2, but also to downregulate the expression of colonic COX-2 protein to control levels.[1] Sulindac sulfide is a metabolite of sulindac that has diverse

activities.[2],[3] It inhibits both COX-1 and COX-2 (IC50s = 1.9 and 1.21 μM, respectively), whereas the parent compound, sulindac, is much less effective (IC50 = 58 μM for COX-2 and > 100 μM for COX-1).[4] Sulindac sulfide also inhibits aldose reductase (IC50 = 279 nM), blocking NADPH-dependent reduction of glucose to sorbitol, and reducing type 2 diabetic complications.[5] It increases the expression and activity of NAD(P)H quinone oxidoreductase [1].[6] Sulindac sulfide inhibits colorectal cancer growth both in vitro and in vivo.[7]

Reference:

[1]. Boolbol, S.K., Dannenberg, A.J., Chadburn, A., et al. Cyclooxygenase-2 overexpression and tumor formation are blocked by sulindac in a murine model of familial adenomatous polyposis. Cancer Research 56, 2556-2560 (1996).
[2]. Kitamura, S., and Tatsumi, K. In vitro metabolism of sulindac and sulindac sulfide: Enzymatic formation of sulfoxide and sulfone. Japanese Journal of Pharmacology 32(5), 833-838 (1982).
[3]. Brunell, D., Sagher, D., Kesaraju, S., et al. Studies on the metabolism and biological activity of the epimers of sulindac. Drug Metabolism and Disposition 39(6), 1014-1021 (2011).
[4]. Warner, T.D., Giuliano, F., Vojnovic, I., et al. Nonsteroid drug selectivities for cyclo-oxygenase-1 rather than cyclo-oxygenase-2 are associated with human gastrointestinal toxicity: A full in vitro analysis. Proc. Nat. Acad. Sci. USA 96(13), 7563-7568 (1999).
[5]. Zheng, X., Zhang, L., Zhai, J., et al. The molecular basis for inhibition of sulindac and its metabolites towards human aldose reductase. FEBS Letters 586(1), 55-59 (2012).
[6]. Kung, H.N., Weng, T.Y., Liu, Y.L., et al. Sulindac compounds facilitate the cytotoxicity of β-lapachone by up-regulation of NAD(P)H quinone oxidoreductase in human lung cancer cells. PLoS One 9(2), 1-15 (2014).
[7]. Williams, C.S., Goldman, A.P., Sheng, H., et al. Sulindac sulfide, but not sulindac sulfone, inhibits colorectal cancer growth. Neoplasia 1(2), 170-176 (1999).