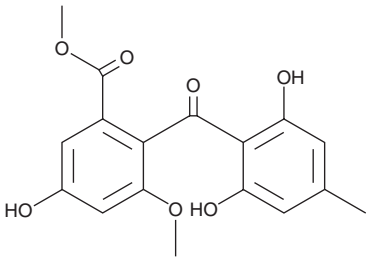
PRODUCT INFORMATION

Sulochrin

Item No. 21797

CAS Registry No.:	519-57-3
Formal Name:	2-(2,6-dihydroxy-4-methylbenzoyl)-5-hydroxy-3-methoxy-benzoic acid, methyl ester
MF:	$C_{17}H_{16}O_7$
FW:	332.3
Purity:	≥99%
Supplied as:	A solid
Storage:	-20°C
Stability:	≥4 years
Item Origin:	Fungus/*Aspergillus terreus*



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Sulochrin is supplied as a solid. A stock solution may be made by dissolving the sulochrin in the solvent of choice. Sulochrin is soluble in organic solvents such as ethanol, methanol, DMSO, and dimethyl formamide, which should be purged with an inert gas.

Sulochrin is sparingly soluble in aqueous solutions. To enhance aqueous solubility, dilute the organic solvent solution into aqueous buffers or isotonic saline. If performing biological experiments, ensure the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. We do not recommend storing the aqueous solution for more than one day.

Description

Sulochrin is a fungal metabolite produced by *A. terreus* and *Penicillium* that exhibits antiallergenic, anti-angiogenic, and antiviral activities. Sulochrin inhibits eosinophil degranulation.[1] It prevents leukotriene C_4 (LTC_4) release, O_2^- production, and IL-8 production (IC_{50}s = 0.03, 8.2, 9.6, and 8.7 µM, respectively) and inhibits migration of human and guinea pig eosinophils (IC_{50} = 0.2-0.4 µM). Sulochrin inhibits VEGF-induced capillary tube formation of human umbilical vein endothelial cells (HUVEC) *in vitro* (70% inhibition at 10 mg/ml).[2] It also inhibits hepatitis C viral infection (IC_{50} = 24.4 µM) *in vitro*.[3]

References

1. Ohashi, H., Motegi, Y., Kita, H., *et al.* Sulochrin inhibits eosinophil activation and chemotaxis. *Inflamm. Res.* **47(10)**, 409-415 (1998).
2. Lee, H.J., Lee, J.H., Hwang, B.Y., *et al.* Fungal metabolites, asterric acid derivatives inhibit vascular endothelial growth factor (VEGF)-induced tube formation of HUVECs. *J. Antibiot. (Tokyo)* **55(6)**, 552-556 (2002).
3. Nakajima, S., Watashi, K., Kamisuki, S., *et al.* Specific inhibition of hepatitis C virus entry into host hepatocytes by fungi-derived sulochrin and its derivatives. *Biochem. Biophys. Res. Commun.* **440(4)**, 515-520 (2013).

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