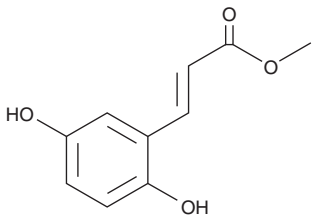
PRODUCT INFORMATION

Erbstatin analog

Item No. 10010238

CAS Registry No.:	63177-57-1
Formal Name:	3-(2,5-dihydroxyphenyl)-2-propenoic acid, methyl ester
Synonym:	Methyl 2,5-dihydroxycinnamate
MF:	$C_{10}H_{10}O_4$
FW:	194.2
Purity:	≥98%
UV/Vis.:	λ_{max}: 250, 280, 362 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

Erbstatin analog is supplied as a crystalline solid. A stock solution may be made by dissolving the erbstatin analog in the solvent of choice, which should be purged with an inert gas. Erbstatin analog is soluble in the organic solvent ethanol. The solubility of erbstatin analog in ethanol is approximately 10 mg/ml.

Erbstatin analog 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

Overactivity of the epidermal growth factor (EGF) receptor-associated tyrosine kinase has been associated with a number of cancers and thus tyrosine kinase inhibitors could be potential therapeutic agents to prevent malignant growth.[1] Erbstatin is a potent, small-molecule inhibitor of EGF receptor-associated tyrosine kinase. However, it is highly unstable, inactivating completely within 30 minutes in calf serum.[2] Erbstatin analog is a stable, potent inhibitor of EGFR kinase activity. It is four-times more stable than erbstatin in calf serum and inhibits EGFR tyrosine kinase *in vitro* similar to erbstatin with an IC_{50} value of 0.14 µg/ml.[2] Erbstatin analog inhibits EGF-stimulated growth in EGFR-overexpressing NIH3T3 cells with an IC_{50} value of 0.5 µg/ml and efficiently delays the onset of EGF-induced DNA synthesis.[3]

References

1. Sausville, E.A., Elsayed, Y., Monga, M., *et al.* Signal transduction-directed cancer treatments. *Annu. Rev. Pharmacol. Toxicol.* **43**, 199-231 (2003).
2. Umezawa, K., Hori, T., Tajima, H., *et al.* Inhibition of epidermal growth factor-induced DNA synthesis by tyrosine kinase inhibitors. *FEBS* **260(2)**, 198-200 (1990).
3. Umezawa, K., Sugata, D., Yamashita, K., *et al.* Inhibition of epidermal growth factor receptor functions by tyrosine kinase inhibitors in NIH3T3 cells. *FEBS* **314(3)**, 289-292 (1992).

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