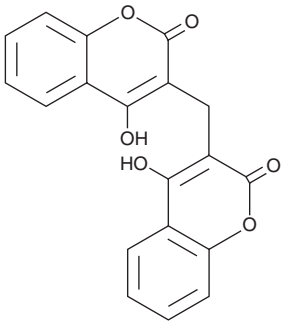
PRODUCT INFORMATION

Dicoumarol

Item No. 20764

CAS Registry No.:	66-76-2
Formal Name:	3,3'-methylenebis[4-hydroxy-2H-1-benzopyran-2-one
Synonyms:	NSC 17860, NSC 41834, NSC 221570
MF:	$C_{19}H_{12}O_6$
FW:	336.3
Purity:	≥98%
UV/Vis.:	λ_{max}: 309 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

Dicoumarol is supplied as a crystalline solid. A stock solution may be made by dissolving the dicoumarol in the solvent of choice. Dicoumarol is soluble in organic solvents such as DMSO and dimethyl formamide, which should be purged with an inert gas. The solubility of dicoumarol in these solvents is approximately 2.5 and 1.25 mg/ml, respectively. Dicoumarol is also slightly soluble in ethanol.

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

Description

Dicoumarol is a competitive inhibitor of NADH:quinone oxidoreductase (NQO1) with IC_{50} values of 2.6 and 404 nM in the absence and presence of 2 µM BSA, respectively.[1] It has antiproliferative activity against MIA PaCa-2 pancreas and HCT116 colon carcinoma cells (IC_{50}s = 52 and 19 µM, respectively, after a 96 hour incubation). Dicoumarol inhibits stress-activated protein kinase (SAPK) in HEK293 cells (IC_{50} = 19-33 µM) at a point upstream of MEKK1 and downstream of TNF receptor-associated factor 2 (TRAF2), and it inhibits TNF-α and LPS-induced NF-κB activation in HeLa cells.[2] It also has antiproliferative activity against FL5.12 lymphocytic and MCF-7 breast carcinoma cells (100 µM) by suppressing JNK activation.[3]

References

1. Nolan, K.A., Doncaster, J.R., Dunstan, M.S., *et al.* Synthesis and biological evaluation of coumarin-based inhibitors of NAD(P)H: Quinone oxidoreductase-1 (NQO1). *J. Med. Chem.* **52(22)**, 7142-7156 (2009).
2. Cross, J.V., Deak, J.C., Rich, E.A., *et al.* Quinone reductase inhibitors block SAPK/JNK and NFκB pathways and potentiate apoptosis. *J. Biol. Chem.* **274(44)**, 31150-31154 (1999).
3. Krause, D., Lyons, A., Fennelly, C., *et al.* Transient activation of Jun N-terminal kinases and protection from apoptosis by the insulin-like growth factor I receptor can be suppressed by dicumarol. *J. Biol. Chem.* **276(22)**, 19244-19252 (2001).

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