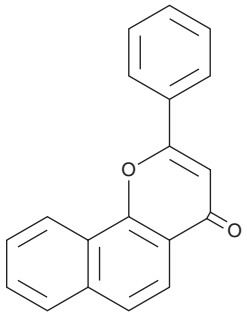
PRODUCT INFORMATION

α-Naphthoflavone

Item No. 16924

CAS Registry No.:	604-59-1
Formal Name:	2-phenyl-4H-naphtho[1,2-b]pyran-4-one
Synonyms:	7,8-Benzoflavone, NSC 407011
MF:	$C_{19}H_{12}O_2$
FW:	272.3
Purity:	≥98%
UV/Vis.:	λ_{max}: 225, 280, 344 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

α-Naphthoflavone is supplied as a crystalline solid. A stock solution may be made by dissolving the α-naphthoflavone in the solvent of choice, which should be purged with an inert gas. α-Naphthoflavone is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of α-naphthoflavone in these solvents is approximately 1, 10, and 20 mg/ml, respectively.

α-Naphthoflavone is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, α-naphthoflavone should first be dissolved in DMF and then diluted with the aqueous buffer of choice. α-Naphthoflavone has a solubility of approximately 0.1 mg/ml in a 1:4 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 aryl hydrocarbon receptor (AhR) is a ligand-activated transcription factor that promotes the expression of phase I and II xenobiotic chemical metabolizing enzyme genes, including the cytochrome P450 (CYP) isoforms CYP1A1 and CYP1A2.[1] α-Naphthoflavone is a flavone that modulates xenobiotic metabolism at several points. It antagonizes AhR, blocking the expression of phase I and II genes at nanomolar concentrations, although it can agonize AhR at higher concentrations (10 µM).[2,3] α-Naphthoflavone inhibits CYP19 (aromatase), CYP1A1, CYP1A2, and CYP1B1 (IC_{50}s = 500, 60, 6, and 5 nM, respectively), whereas it activates CYP3A4 (K_d = 7.4 µM).[4-6] Dietary α-naphthoflavone can contribute to carcinogenesis in the presence of synthetic estrogens.[7]

References

1. Denison, M.S. and Nagy, S.R. *Annu. Rev. Pharmacol. Toxicol.* **43**, 309-334 (2003).
2. Smith, K.J., Murray, I.A., Tanos, R., *et al. J. Pharmacol. Exp. Ther.* **338(1)**, 318-327 (2011).
3. Wilhelmsson, A., Whitelaw, M.L., Gustafsson, J.A., *et al. J. Biol. Chem.* **269(29)**, 19028-19033 (1994).
4. Bruno, R.D. and Njar, V.C.O. *Bioorg. Med. Chem.* **15(15)**, 5047-5060 (2007).
5. Campbell, D.R. and Kurzer, M.S. Flavonoid inhibition of aromatase enzyme activity in human preadipocytes. *J. Steroid Biochem. Mol. Biol.* **46(3)**, 381-388 (1993).
6. Sineva, E.V., Rumfeldt, J.A.O., Halpert, J.R., *et al. PLoS One* **8(12)**, 1-13 (2013).
7. Metzler, M., Blaich, G., and Tritscher, A.M. Role of metabolic activation in the carcinogenicity of estrogens: Studies in an animal liver tumor model. *Environ. Health Perspect.* **88**, 117-121 (1990).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.CAYMANCHEM.COM