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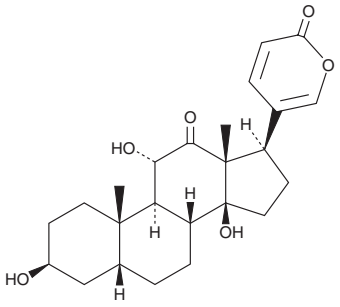
PRODUCT INFORMATION

Arenobufagin

Item No. 30695

CAS Registry No.:	464-74-4
Formal Name:	(3β,5β,11α)-3,11,14-trihydroxy-12-oxo-bufa-20,22-dienolide
MF:	$C_{24}H_{32}O_6$
FW:	416.5
Purity:	≥95%
UV/Vis.:	λ_{max}: 299 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥2 years
Item Origin:	Plant/*Bufonis venenum*



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

Laboratory Procedures

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

Arenobufagin is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, arenobufagin should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Arenobufagin has a solubility of approximately 0.11 mg/ml in a 1:8 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Arenobufagin is a cardiotonic steroid that has been found in the skin secretions of the toad *B. gargarizans* and has anticancer activity.[1-4] It inhibits Na^+/K^+-ATPase activity in isolated guinea pig ventricular myocytes with an IC_{50} value of 290 nM.[1] Arenobufagin (20-500 nM) induces cleavage of poly(ADP-ribose) polymerase (PARP), caspase-3, and caspase-9, as well as induces apoptosis in HepG2 hepatocellular carcinoma cells.[2] It inhibits proliferation of HepG2, Hep3B, BEL-7402, and MCF-7 cancer cells (IC_{50}s = 101.9, 39.8, 416.3, and 143.7 nM, respectively), as well as multidrug-resistant HepG2/adm and MCF-7/adr cancer cells (IC_{50}s = 730 and 67.5 nM, respectively). Arenobufagin (1 mg/kg once per day, i.v.) reduces tumor growth, the number and size of lung metastases, and tumor levels of the epithelial-to-mesenchymal transition (EMT) markers vimentin and β-catenin in a PC3 mouse xenograft model.[3] Arenobufagin (5 and 10 µM/plug) inhibits VEGF-induced angiogenesis in a Matrigel™ plug assay in mice.[4]

References

1. dos Santos Cruz, J. and Matsuda, H. Arenobufagin, a compound in toad venom, blocks Na+-K+ pump current in cardiac myocytes *Eur. J. Pharmacol.* **239(1-3)**, 223-226 (1993).
2. Zhang, D.-M., Liu, J.-S., Deng, L.-J., *et al.* Arenobufagin, a natural bufadienolide from toad venom, induces apoptosis and autophagy in human hepatocellular carcinoma cells through inhibition of PI3K/Akt/ mTOR pathway. *Carcinogenesis* **34(6)**, 1331-1342 (2013).
3. Chen, L., Mai, W., Chen, M., *et al.* Arenobufagin inhibits prostate cancer epithelial-mesenchymal transition and metastasis by down-regulating β-catenin. *Pharmacol. Res.* **123**, 130-142 (2017).
4. Li, M.W., S., Liu, Z., Zhang, W., *et al.* Arenobufagin, a bufadienolide compound from toad venom, inhibits VEGF-mediated angiogenesis through suppression of VEGFR-2 signaling pathway. *Biochem. Pharmacol.* **83(9)**, 1251-1260 (2012).

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