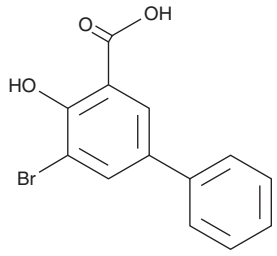
PRODUCT INFORMATION

3-bromo-5-phenyl Salicylic Acid

Item No. 13574

CAS Registry No.: 4906-68-7
Formal Name: 5-bromo-4-hydroxy-[1,1'-biphenyl]-3-carboxylic acid
Synonym: NSC 109116
MF: $C_{13}H_9BrO_3$
FW: 293.1
Purity: ≥95%
UV/Vis.: λ_{max}: 233, 326 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

3-bromo-5-phenyl Salicylic acid is supplied as a crystalline solid. A stock solution may be made by dissolving the 3-bromo-5-phenyl salicylic acid in the solvent of choice, which should be purged with an inert gas. 3-bromo-5-phenyl Salicylic acid is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of 3-bromo-5-phenyl salicylic acid in these solvents is approximately 0.1, 12.5, and 15 mg/ml, respectively.

3-bromo-5-phenyl Salicylic acid is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, 3-bromo-5-phenyl salicylic acid should first be dissolved in DMF and then diluted with the aqueous buffer of choice. 3-bromo-5-phenyl Salicylic acid has a solubility of approximately 0.1 mg/ml in a 1:9 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 aldo-keto reductase (AKR) enzymes constitute a family of related NADPH-dependent oxidoreductases. The 1C subfamily (AKR1C) includes four human hydroxysteroid dehydrogenases, with AKR1C1 being a 20α-HSD and the other three being 3α-HSDs. AKR1C1 metabolizes progesterone to an inactive progestin, 20α-hydroxy progesterone.[1] In addition, AKR1C1 actions have been implicated in cancer and in the processing of neuroactive steroids involved in brain function.[2-5] 3-bromo-5-phenyl Salicylic acid selectively inhibits AKR1C1 (K_i = 4 nM) over AKR1C2 (K_i = 87 nM), AKR1C3 (K_i = 4.2 µM), and AKR1C4 (K_i = 18.2 µM).[6] Moreover, it potently inhibits the metabolism of progesterone by bovine aortic endothelial cells overexpressing AKR1C1 (IC_{50} = 460 nM).[6]

References

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3. Wang, H.W., Lin, C.P., Chiu, J.H., *et al. Int. J. Cancer* **120(9)**, 2019-2027 (2007).
4. Belelli, D., Herd, M.B., Mitchell, E.A., *et al. Neuroscience* **138(3)**, 821-829 (2006).
5. Usami, N., Yamamoto, T., Shintani, S., *et al. Biol. Pharm. Bull.* **25(4)**, 441-445 (2002).
6. El-Kabbani, O., Scammells, P.J., Gosling, J., *et al. J. Med. Chem.* **52(10)**, 3259-3264 (2009).

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