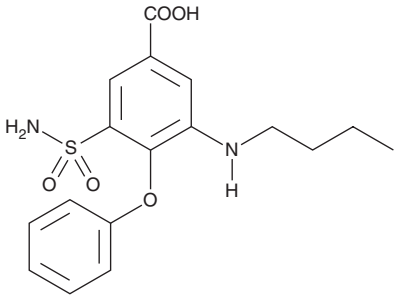
PRODUCT INFORMATION

Bumetanide

Item No. 14630



CAS Registry No.: 28395-03-1
Formal Name: 3-(aminosulfonyl)-5-(butylamino)-4-phenoxy-benzoic acid
Synonyms: PF-1593, Ro 10-6338
MF: $C_{17}H_{20}N_2O_5S$
FW: 364.4
Purity: ≥98%
UV/Vis.: λ_{max}: 228, 262, 339 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

Bumetanide is supplied as a crystalline solid. A stock solution may be made by dissolving the bumetanide in the solvent of choice, which should be purged with an inert gas. Bumetanide is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of bumetanide in these solvents is approximately 14, 25, and 33 mg/ml, respectively.

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

Description

Bumetanide is an inhibitor of Na-K-2Cl cotransporter 1 (NKCC1; IC_{50} = 0.68 µM).[1] It is selective for NKCC1 over NKCC2 (IC_{50} = 4 µM). Bumetanide is an agonist of G protein-coupled receptor 35 (GPR35; EC_{50} = 10 µM).[2] It also inhibits various carbonic anhydrases.[3] Bumetanide (0.1 mg/kg) increases urine flow and sodium and potassium excretion, as well as decreases sodium reabsorption, in anesthetized dogs.[4] It reduces brain edema and infarct size in a rat model of stroke induced by permanent middle cerebral artery occlusion (MCAO) when administered at doses ranging from 7.6 to 30.4 mg/kg.[5] Formulations containing bumetanide have been used in the treatment of edema associated with congestive heart failure and hepatic and renal diseases.

References

1. Lykke, K., Töllner, K., Feit, P.W., *et al.* The search for NKCC1-selective drugs for the treatment of epilepsy: Structure-function relationship of bumetanide and various bumetanide derivatives in inhibiting the human cation-chloride cotransporter NKCC1A. *Epilepsy Behav.* **59**, 42-49 (2016).
2. Yang, Y., Fu, A., Wu, X., *et al.* GPR35 is a target of the loop diuretic drugs bumetanide and furosemide. *Pharmacology* **89(1-2)**, 13-17 (2012).
3. Temperini, C., Cecchi, A., Scozzafava, A., *et al.* Carbonic anhydrase inhibitors. Interaction of indapamide and related diuretics with 12 mammalian isozymes and X-ray crystallographic studies for the indapamide-isozyme II adduct. *Bioorg. Med. Chem. Lett.* **18(8)**, 2567-2573 (2008).
4. Cohen, M.R., Hinsch, E., Vergona, R., *et al.* A comparative diuretic and tissue distribution study of bumetanide and furosemide in the dog. *J. Pharmacol. Exp. Ther.* **197(3)**, 697-702 (1976).
5. O'Donnell, M.E., Tran, L., Lam, T.I., *et al.* Bumetanide inhibition of the blood-brain barrier Na-K-Cl cotransporter reduces edema formation in the rat middle cerebral artery occlusion model of stroke. *J. Cereb. Blood Flow Metab.* **24(9)**, 1046-1056 (2004).

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