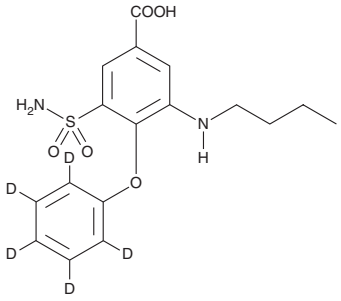
PRODUCT INFORMATION

Bumetanide-d_5

Item No. 26773

CAS Registry No.: 1216739-35-3
Formal Name: 3-(aminosulfonyl)-5-(butylamino)-4-phenoxy-d_5-benzoic acid
MF: $C_{17}H_{15}D_5N_2O_5S$
FW: 369.4
Chemical Purity: ≥98% (Bumetanide)
Deuterium Incorporation: ≥99% deuterated forms (d_1-d_5); ≤1% d_0
Supplied as: A 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-d_5 is intended for use as an internal standard for the quantification of bumetanide (Item No. 14630) by GC- or LC-MS. The accuracy of the sample weight in this vial is between 5% over and 2% under the amount shown on the vial. If better precision is required, the deuterated standard should be quantitated against a more precisely weighed unlabeled standard by constructing a standard curve of peak intensity ratios (deuterated *versus* unlabeled).

Bumetanide-d_5 is supplied as a solid. A stock solution may be made by dissolving the bumetanide-d_5 in the solvent of choice, which should be purged with an inert gas. Bumetanide-d_5 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of bumetanide-d_5 in these solvents is approximately 14, 25, and 33 mg/ml, respectively.

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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