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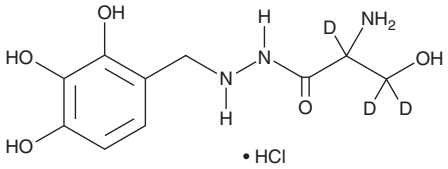
PRODUCT INFORMATION

Benserazide-d_3 (hydrochloride)

Item No. 29643

Formal Name:	2-amino-3-hydroxy-N'-(2,3,4-trihydroxybenzyl)propanehydrazide-2,3,3-d_3, monohydrochloride
MF:	$C_{10}H_{12}D_3N_3O_5 \bullet HCl$
FW:	296.7
Chemical Purity:	≥95% (Benserazide)
Deuterium Incorporation:	≥99% deuterated forms (d_1-d_3); ≤1% d_0
Supplied as:	A solid
Storage:	-20°C
Stability:	≥2 years



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

Laboratory Procedures

Benserazide-d_3 (hydrochloride) is intended for use as an internal standard for the quantification of benserazide (Item No. 20298) 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).

Description

Benserazide is a peripherally restricted inhibitor of aromatic L-amino acid decarboxylase (AADC; IC_{50} = 0.53 µM).[1] It also inhibits tryptophan oxygenase and kynureninase (K_is = 41.8 and 26.4 µM, respectively, in rat liver homogenates), as well as hexokinase 2 (HK2), HK1, and HK4 (IC_{50}s = 5.5, 25.1, and 40.5 µM, respectively, for the recombinant human enzymes).[2,3] Benserazide (50-400 µM) is cytotoxic to SW480 colorectal cancer cells, an effect that can be reversed by *HK2* siRNA knockdown, and inhibits proliferation of SW480, LoVo, HCT116, MCF-7, and SMMC-7721 cancer cells with IC_{50} values of 143, 151, 181.4, 186, and 210.4 nM, respectively.[3] It reduces tumor growth in an SW480 mouse xenograft model when administered at doses of 300 and 600 mg/kg per day for 16 days. Oral administration of benserazide (8.8 mg/kg) inhibits AADC activity in rat liver, kidney, and brain by approximately 90, 90, and 25%, respectively, and increases striatal dopamine levels following administration of L-DOPA (Item No. 13248) in rats. Benserazide (10 and 50 mg/kg) also inhibits striatal AADC and increases striatal dopamine levels following administration of L-DOPA in a 6-OHDA (Item No. 25330) mouse model of Parkinson's disease.[4]

References

1. Schultz, E. L-dopa as substrate for human duodenal catechol-*O*-methyltransferase and aromatic L-amino acid decarboxylase. *Biomed. Chromatogr.* **4(6)**, 242-244 (1990).
2. Bender, D.A. Inhibition *in vitro* of the enzymes of the oxidative pathway of tryptophan metabolism and of nicotinamide nucleotide synthesis by benserazide, carbidopa and isoniazid. *Biochem. Pharmacol.* **29(5)**, 707-712 (1980).
3. Li, W., Zheng, M., Wu, S., *et al.* Benserazide, a dopadecarboxylase inhibitor, suppresses tumor growth by targeting hexokinase 2. *J. Exp. Clin. Cancer Res.* **36:58**, (2017).
4. Shen, H., Kannari, K., Yamato, H., *et al.* Effects of benserazide on L-DOPA-derived extracellular dopamine levels and aromatic L-amino acid decarboxylase activity in the striatum of 6-hydroxydopamine-lesioned rats. *Tohoku J.Exp.Med.* **199(3)**, 149-159 (2003).

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