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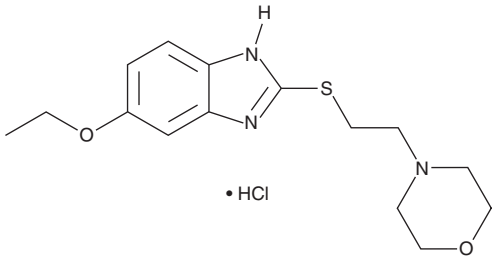
PRODUCT INFORMATION

Afobazole (hydrochloride)

Item No. 22923

CAS Registry No.:	173352-39-1
Formal Name:	6-ethoxy-2-[[2-(4-morpholinyl) ethyl]thio]-1H-benzimidazole, monohydrochloride
Synonym:	SM-346
MF:	$C_{15}H_{21}N_3O_2S \bullet HCl$
FW:	343.9
Purity:	≥98%
UV/Vis.:	λ_{max}: 299 nm
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

Afobazole (hydrochloride) is supplied as a solid. A stock solution may be made by dissolving the afobazole (hydrochloride) in the solvent of choice, which should be purged with an inert gas. Afobazole (hydrochloride) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of afobazole (hydrochloride) in these solvents is approximately 50 mg/ml.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of afobazole (hydrochloride) can be prepared by directly dissolving the solid in aqueous buffers. The solubility of afobazole (hydrochloride) in PBS, pH 7.2, is approximately 10 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Afobazole is a multi-targeted anxiolytic drug with neuroprotective activities.[1,2] It binds to the sigma-1, melatonin MT_1, and MT_3 receptors, as well as monoamine oxidase A (MAO-A; K_is = 5.9, 160, 0.97, and 3.6 µM, respectively, in a radioligand binding assay).[1] Afobazole (5 mg/kg) decreases the latency to enter, as well as increases the number of entries into and percentage of time spent in, the open arms of the elevated plus maze, indicating anxiolytic-like activity in passive stress-coping BALB/c, but not active stress-coping C57BL/6, mice.[3] It decreases stroke volume and neuronal and oligodendroglial cell death in the brain in a rat model of ischemia induced by middle cerebral artery occlusion (MCAO) when administered at doses of 0.3 and 3 mg/kg.[2]

References

1. Seredenin, S.B., and Voronin, M.V. [Neuroreceptor mechanisms of the afobazole effect]. *Eksp. Klin. Farmakol.* **72(1)**, 3-11 (2009).
2. Katnik, C., Garcia, A., Behensky, A.A., *et al.* Treatment with afobazole at delayed time points following ischemic stroke improves long-term functional and histological outcomes. *Neurobiol. Dis.* **62**, 354-364 (2014).
3. Anderzhanova, E.A., Bächli, H., Buneeva, O.A., *et al.* Strain differences in profiles of dopaminergic neurotransmission in the prefrontal cortex of the BALB/C vs. C57Bl/6 mice: Consequences of stress and afobazole. *Eur. J. Pharmacol.* **708(1-3)**, 95-104 (2013).

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