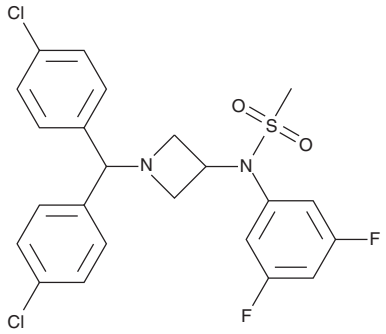
PRODUCT INFORMATION

AVE-1625

Item No. 10009021

CAS Registry No.:	358970-97-5
Formal Name:	N-[1-[*bis*(4-chlorophenyl)methyl]-3-azetidinyl]-N-(3,5-difluorophenyl)-methanesulfonamide
Synonym:	Drinabant
MF:	$C_{23}H_{20}Cl_2F_2N_2O_2S$
FW:	497.4
Purity:	≥98%
UV/Vis.:	λ_{max}: 231 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

AVE-1625 is supplied as a crystalline solid. A stock solution may be made by dissolving the AVE-1625 in the solvent of choice, which should be purged with an inert gas. AVE-1625 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of AVE-1625 in ethanol is approximately 0.15 mg/ml and approximately 15 mg/ml in DMSO and DMF.

AVE-1625 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, AVE-1625 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. AVE-1625 has a solubility of approximately 0.3 mg/ml in a 1:2 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

The central cannabinoid (CB1) receptor is a G protein-coupled receptor that is widely distributed in the central nervous system and several peripheral tissues and binds the active component of *cannabis*, Δ^9-tetrahydrocannabinol.[1] Signaling through the CB1 receptor is implicated in attentional and working memory deficits as well as obesity.[2-4] AVE-1625 is a highly potent, selective antagonist for the CB_1 receptor with K_i values of 0.16-0.44 nM.[4] At 1-3 mg/kg, AVE-1625 significantly improves the performance of rodents in working memory tasks.[4] At 30 mg/kg, AVE-1625 reduces caloric intake by more than 50% of controls and significantly increases lipolysis from fat tissues and reduces hepatic glycogen levels in rodents.[5]

References

1. Howlett, A.C., Song, C., Berglund, B.A., *et al.* Characterization of CB_1 cannabinoid receptors using receptor peptide fragments and site-directed antibodies. *Mol. Pharmacol.* **53(3)**, 504-510 (1998).
2. Williams, J.L., Kashfi, K., Ouyang, N., *et al.* NO-donating aspirin inhibits intestinal carcinogenesis in min (APC^{Minl+}) mice. *Biochem. Biophys. Res. Commun.* **313(3)**, 784-788 (2004).
3. Mackie, K. Cannabinoid receptors as therapeutic targets. Annu. Rev. Pharmacol. *Toxicol.* **46**, 101-122 (2006).
4. Borowsky, B., Stevens, R., Mark, B., *et al.* AVE1625, a cannabinoid CB1 antagonist, as a co-treatment for Schizophrenia: Improvement in cognitive function and reduction of antipsychotic-side effects in animal models. *Neuropsychopharmacology* **30**, S116-S117 (2005).
5. Herling, A.W., Gossel, M., Haschke, G., *et al.* The CB1 receptor antagonist AVE1625 affects primarily metabolic parameters independently of reduced food intake in wistar rats. *Am. J. Physiol. Endocrinol. Metab.* **293(3)**, E826-E832 (2007).

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