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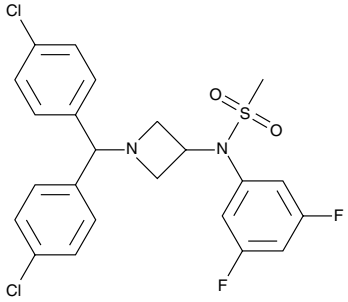
Product Information

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
Synonyms: Drinabant
MF: $C_{23}H_{20}Cl_2F_2N_2O_2S$
FW: 497.4
Purity: ≥98%
Stability: ≥2 years at -20°C
Supplied as: A crystalline solid
UV/Vis.: λ_{max}: 231 nm



Laboratory Procedures

For long term storage, we suggest that AVE-1625 be stored as supplied at -20°C. It should be stable for at least two years.

AVE-1625 is supplied as a crystalline solid. A stock solution may be made by dissolving the AVE-1625 in an organic solvent 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.

The central cannabinoid (CB_1) 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 CB_1 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.[5] At 1-3 mg/kg, AVE-1625 significantly improves the performance of rodents in working memory tasks.[5] 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**, 504-510 (1998).
2. Gómez, R., Navarro, M., Ferrer, B., *et al.* A peripheral mechanism for CB_1 cannabinoid receptor-dependent modulation of feeding. *J. Neurosci.* **22(21)**, 9612-9617 (2002).
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 CB_1 antagonist, as a co-treatment for Schizophrenia: Improvement in cognitive function and reduction of antipsychotic-side effects in animal models. *Neuropsychopharmachology* **30**, S77-S142 (2005).
5. Herling, A.W., Gossel, M., Haschke, G., *et al.* CB_1 receptor antagonist AVE1625 affects primarily metabolic parameters independently of reduced food intake in wistar rats. *Am. J. Physiol. Endocrinol. Metab.* **293**, E826-E832 (2007).

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