Product Data Sheet

Product Name: AVE-1625
Cat. No.: GC14115

Chemical Properties

Cas. No.	358970-97-5		
Chemical Name	N-[1-[bis(4-chlorophenyl)methyl]azetidin-3-yl]-N-(3,5-difluorophenyl)methanesulfonamide		
SMILES	CS(=O)(N(C1CN(C(C2=CC=C(Cl)C=C2)C3=CC=C(Cl)C=C3)C1)C4=CC(F)=CC(F)=C4)=O		
Formula	$C_{23}H_{20}Cl_2F_2N_2O_2S$	M.Wt	497.4
Solubility	≤0.15mg/ml in ethanol;15mg/ml in DMSO;15mg/ml in dimethyl formamide	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	AVE-1625		

Background

AVE-1625 is a highly potent, selective antagonist for the CB1 receptor [1].

The cannabinoid receptor type 1 (CB1) is a G protein-coupled receptor mainly expressed in the central and peripheral nervous system. The CB1 receptor is activated by the endocannabinoid neurotransmitters anandamide and 2-arachidonoylglycerol (2-AG). The CB1 receptor has been implicated in the maintenance of homeostasis in health and disease [2]. The CB1 receptor plays vital roles in modulating neurotransmitter release by preventing the development of excessive neuronal activity, reducing pain and other inflammatory symptoms [2].

AVE-1625 antagonized the CB1 receptor activity with the Ki values of 0.16-0.44 nM [1]. Treatment with AVE-1625 (1-3 mg/kg) significantly improved the performance of rodents in working memory tasks. At 30 mg/kg, AVE-1625 reduced caloric intake by more than 50% of controls and significantly increased lipolysis from fat tissues and reduced hepatic glycogen

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levels in rodents. In Wistar rats, postprandially administration of AVE1625 slightly increased the basal lipolysis in a dose-dependent manner. AVE1625 caused primary effects on metabolic blood and tissue parameters as well as metabolic rate [3]. As measured by indirect calorimetry, AVE1625 immediately increased the total energy expenditure and a transiently increased glucose oxidation [3].

References:

[1] Borowsky B, Stevens R, Mark B, et al. AVE1625, a cannabinoid CBI antagonist, as a co-treatment for schizophrenia: Improvement in cognitive function and reduction of antipsychotic-side effects in animal models[C]//Neuropsychopharmacology. Macmillan building, 4 crinan st, london n1 9xw, ENGLAND: NATURE PUBLISHING GROUP, 2005, 30: S116-S117.

[2] Herkenham M, Lynn A B, Little M D, et al. Cannabinoid receptor localization in brain[J]. Proceedings of the national Academy of sciences, 1990, 87(5): 1932-1936.

[3] Herling A W, Gossel M, Haschke G, et al. CB1 receptor antagonist AVE1625 affects primarily metabolic parameters independently of reduced food intake in Wistar rats[J]. American Journal of Physiology-Endocrinology and Metabolism, 2007, 293(3): E826-E832.