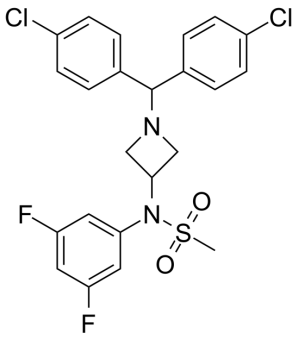
Drinabant

Cat. No.:	HY-14788
CAS No.:	358970-97-5
Molecular Formula:	$C_{23}H_{20}Cl_2F_2N_2O_2S$
Molecular Weight:	497.38
Target:	Cannabinoid Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	-20°C, stored under nitrogen * In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



BIOLOGICAL ACTIVITY

Description

Drinabant (AVE1625) is an orally active CB1 receptor antagonist. Drinabant (AVE1625) inhibits the agonist-stimulated calcium signal with IC_{50} values of 25 nM and 10 nM for the hCB1-R and rCB1-R, respectively, and is ineffective for the hCB2-R [1].

IC_{50} & Target

hCB1-R	rCB1-R	CB2
25 nM (IC_{50})	10 nM (IC_{50})	10000 nM (IC_{50})

In Vivo

AVE1625 (10 mg/kg orally once daily), combined with Olanzapine (HY-14541) attenuates body weight gain, diminishing the enhanced food intake while maintaining increased energy expenditure and decreased motility[2].

AVE1625 (1, 3, and 10 mg/kg ip), reverses abnormally persistent LI induced by MK-801 (HY-15084B) or neonatal nitric oxide synthase inhibition in rodents, and improves both working and episodic memory[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Rats[1].
Dosage:	30 mg/kg.
Administration:	Oral gavage, single dose.
Result:	Had free access to food during the preceding night (postprandial state) caused a pronounced reduction of food intake during the subsequent 10-12 h without differences intheir locomotor activity relative to that of the control group. Caused an increase in FFA and glycerol, indicating increased lipolysis from fat tissue. Immediately resulted in a pronounced increase in V_{CO2} and V_{O2}, indicating increased oxidation of energetic substrates and increased TEE.

Animal Model:	Female Hanover Wistar rats weighing 225 ± 8.6 g[2].
Dosage:	10 mg/kg.
Administration:	Orally once daily.
Result:	Reduced their weight markedly within the first 3 days of treatment where upon animals

	maintained lower body weight, although they lost about 7.3 ± 1.3 g fat during the 12 days of treatment.

REFERENCES

[1]. Andreas W Herling, et al. CB1 receptor antagonist AVE1625 affects primarily metabolic parameters independently of reduced food intake in Wistar rats. Am J Physiol Endocrinol Metab. 2007 Sep;293(3):E826-32.

[2]. Michaela Liebig, et al. Profiling of energy metabolism in olanzapine-induced weight gain in rats and its prevention by the CB1-antagonist AVE1625. Obesity (Silver Spring). 2010 Oct;18(10):1952-8.

[3]. Mark D Black, et al. AVE1625, a cannabinoid CB1 receptor antagonist, as a co-treatment with antipsychotics for schizophrenia: improvement in cognitive function and reduction of antipsychotic-side effects in rodents. Psychopharmacology (Berl). 2011 May;215

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA