
Product Data Sheet

Product Name: BAY 38-7271

Cat. No.: GC61778

Chemical Properties

Cas. No. 212188-60-8

SMILES O=S(CCCC(F)(F)F)(OC1=CC=CC(OC2=CC=CC3=C2C[C@H](CO)C3)=C1)=OFormula $C_{20}H_{21}F_3O_5S$ M.Wt 430.44Solubility Storage Store at $-20^{\circ}C$

General tips For obtaining a higher solubility , please warm the tube at $37^{\circ}C$ and shake it in the ultrasonic bath for a while. Stock solution can be stored below $-20^{\circ}C$ 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

Background

BAY 38-7271 is selective and highly potent and cannabinoid CB1/CB2 receptor agonist, with K_{is} of 1.85 nM and 5.96 nM for recombinant human CB1 receptor and CB2 receptor, respectively. BAY 38-7271 has strong neuroprotective properties[1].

BAY 38-7271 shows only minor interactions at the micromolar range with other binding sites such as adenosine A3 receptor ($IC_{50} = 7.5 \mu M$), peripheral GABAA benzodiazepine receptor ($IC_{50} = 971 \text{ nM}$), melatonin ML1 receptor ($IC_{50} = 3.3 \mu M$), and at the monoamine transporter ($IC_{50} = 1.7 \mu M$)[1].

BAY 38-7271 ($Ed_{50} = 0.02 \text{ mg/kg}$; i.v. and 0.5 mg/kg ; i.p.) induces a potent and dose-dependent reduction in core body temperature[1]. BAY 38-7271 has low physical dependence liability and is not essentially different from that of other cannabinoid CB1 receptor agonists[1]. BAY 38-7271 ($1-1000 \text{ ng/kg/h}$; i.v. infusion; for 4 hours) shows neuroprotective efficacy in the rat SDH model[1]. BAY 38-7271 also has neuroprotective efficacy in models of transient and permanent occlusion of the middle cerebral artery and brain edema models[1]. Animal Model: Wistar rat ,TBI rat models (acute subdural hematoma, SDH)[1]

Caution: Product has not been fully validated for medical applications. For research use only.

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[1]. Mauler F, et al. BAY 38-7271: a novel highly selective and highly potent cannabinoid receptor agonist for the treatment of traumatic brain injury. CNS Drug Rev. 2003 Winter;9(4):343-58.

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