

Produktinformation



Forschungsprodukte & Biochemikalien



Zellkultur & Verbrauchsmaterial



Diagnostik & molekulare Diagnostik



Laborgeräte & Service

Weitere Information auf den folgenden Seiten! See the following pages for more information!



Lieferung & Zahlungsart

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Product Information

JNJ-1661010

Item No. 14497

CAS Registry No.: 681136-29-8

Formal Name: N-phenyl-4-(3-phenyl-1,2,4-thiadiazol-

5-yl)-1-piperazinecarboxamide

MF: $C_{19}H_{19}N_5OS$ FW: 365.5 **Purity:** ≥98%

Stability: ≥2 years at -20°C Supplied as: A crystalline solid UV/Vis.: λ_{max} : 245 nm

Laboratory Procedures

For long term storage, we suggest that JNJ-1661010 be stored as supplied at -20°C. It should be stable for at least two

JNJ-1661010 is supplied as a crystalline solid. A stock solution may be made by dissolving the JNJ-1661010 in the solvent of choice. JNJ-1661010 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of JNJ-1661010 in these solvents is approximately 1.5, 20, and 25 mg/ml, respectively.

JNJ-1661010 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, JNJ-1661010 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. JNJ-1661010 has a solubility of approximately 0.16 mg/ml in a 1:5 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Fatty acid amide hydrolase (FAAH) degrades N-acyl ethanolamines, including the endocannabinoid arachidonoyl ethanolamide (AEA). JNJ-1661010 is a selective inhibitor of FAAH (IC₅₀s = 34 and 33 nM in rat and human, respectively) that is able to cross the blood-brain barrier. At 20 mg/kg, JNJ-1661010 has been shown to elevate levels of AEA in rat brain. This compound has been used to examine the contribution of endocannabinoid signaling in experimental fibrosis. 2

- 1. Keith, J.M., Apodaca, R., Xiao, W., et al. Thiadiazolopiperazinyl ureas as inhibitors of fatty acid amide hydrolase. Bioorg. Med. Chem. Lett. 18(17), 4838-4843 (2008).
- 2. Palumbo-Zerr, K., Horn, A., Distler, A., et al. Inactivation of fatty acid amide hydrolase exacerbates experimental fibrosis by enhanced endocannabinoid-mediated activation of CB₁. Ann. Rheum. Dis. 71, 2051-2054 (2012).

Related Products

For a list of related products please visit: www.caymanchem.com/catalog/14497

WARNING: This product is for laboratory research only: not for administration to humans. Not for human or veterinary DIAGNOSTIC OR THERAPEUTIC USE.

This material should be considered hazardous until information to the contrary becomes available. Do not ingest, swallow, or inhale. Do not get in eyes, on skin, or on clothing. Wash thoroughly after handling. This information contains some, but not all, of the information required for the safe and proper use of this material. Before use, the user must review the complete Safety Data Sheet, which has been sent via email to your institution.

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at the time of delivery.

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For further details, please refer to our Warranty and Limitation of Remedy located on our website and in our catalog.

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