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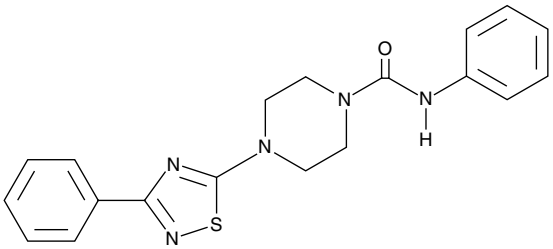
# Product Information

## 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.:** $\lambda_{max}$: 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 years.

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 ($IC_{50}$s = 34 and 33 nM in rat and human, respectively) that is able to cross the blood-brain barrier.[1] At 20 mg/kg, JNJ-1661010 has been shown to elevate levels of AEA in rat brain.[1] This compound has been used to examine the contribution of endocannabinoid signaling in experimental fibrosis.[2]

### References


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 $CB_1$. *Ann. Rheum. Dis.* **71**, 2051-2054 (2012).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