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Produktinformation

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- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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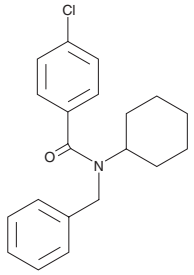
PRODUCT INFORMATION

FPS-ZM1

Item No. 11909

CAS Registry No.:	945714-67-0
Formal Name:	4-chloro-N-cyclohexyl-N-(phenylmethyl)-benzamide
MF:	$C_{20}H_{22}ClNO$
FW:	327.9
Purity:	≥98%
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

FPS-ZM1 is supplied as a crystalline solid. A stock solution may be made by dissolving the FPS-ZM1 in the solvent of choice, which should be purged with an inert gas. FPS-ZM1 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of FPS-ZM1 in these solvents is approximately 15, 25, and 30 mg/ml, respectively.

FPS-ZM1 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, FPS-ZM1 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. FPS-ZM1 has a solubility of approximately 0.12 mg/ml in a 1:7 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

FPS-ZM1 is a high-affinity inhibitor of the receptor for advanced glycation end products (RAGE; K_i = 25 nM).[1] It blocks the binding of amyloid β (Aβ) protein to RAGE and inhibits Aβ40- and Aβ42-induced cellular stress in RAGE-expressing cells.[1] FPS-ZM1 is functional in mice when administered intraperitoneally and penetrates the blood-brain barrier (BBB), inhibiting β-secretase activity, Aβ production, microglia activation, and neuroinflammation.[1,2] FPS-ZM1 is used to elucidate the role of RAGE-mediated signaling in diverse physiological processes *in vivo*, including cancer tumor growth, angiogenesis, and metastasis, as well as BBB damage, edema, and inflammation after intracerebral hemorrhage.[3,4]

References

1. Deane, R., Singh, I., Sagare, A.P., *et al*. A multimodal RAGE-specific inhibitor reduces amyloid β-mediated brain disorder in a mouse model of Alzheimer disease. *J. Clin. Invest.* **122(4)**, 1377-1392 (2012).
2. Hong, Y., Shen, C., Yin, Q., *et al*. Effects of RAGE-specific inhibitor FPS-ZM1 on amyloid-β metabolism and AGEs-induced inflammation and oxidative stress in rat hippocampus. *Neurochem. Res.* **41(5)**, 1192-1199 (2016).
3. Kwak, T., Drews-Elger, K., Ergonul, A., *et al*. Targeting of RAGE-ligand signaling impairs breast cancer cell invasion and metastasis. *Oncogene* **36(11)**, 1559-1572 (2016).
4. Yang, F., Wang, Z., Zhang, J.H., *et al*. Receptor for advanced glycation end-product antagonist reduces blood-brain barrier damage after intracerebral hemorrhage. *Stroke* **46(5)**, 1328-1336 (2015).

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