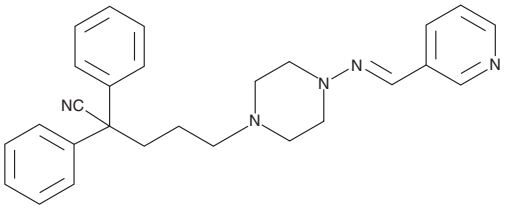
PRODUCT INFORMATION

SC-26196

Item No. 10792

CAS Registry No.: 218136-59-5
Formal Name: α,α-diphenyl-4-[(3-pyridinylmethylene) amino]-1-piperazinepentanenitrile
MF: $C_{27}H_{29}N_5$
FW: 423.6
Purity: ≥98%
UV/Vis.: λ_{max}: 303 nm
Storage: -20°C
Stability: ≥4 years



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

Laboratory Procedures

SC-26196 is supplied as a crystalline solid. A stock solution may be made by dissolving the SC-26196 in the solvent of choice, which should be purged with an inert gas. SC-26196 is soluble in organic solvents such as DMSO and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of SC-26196 in these solvents is approximately and 12 mg/ml, respectively.

SC-26196 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, SC-26196 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. SC-26196 has a solubility of approximately 0.2 mg/ml in a 1:4 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

The enzyme Δ^6 desaturase mediates the conversion of linoleic acid to γ-linolenic acid (GLA), which can then be elongated to dihomo-γ-linolenic acid (DGLA). DGLA can then be used as a substrate for Δ^5 desaturase to produce arachidonic acid (AA), the fatty acid that is used to generate eicosanoids. SC-26196 is an inhibitor of Δ^6 desaturase (IC_{50} = 0.2 µM in a rat liver microsomal assay) that completely blocks the conversion of linoleic acid to arachidonic acid (AA).[1] It is selective for Δ^6 desaturase, as IC_{50} values for Δ^5 and Δ^9 desaturases exceed 200 µM, and it has no effect on the conversion of dihomo-γ-linolenic acid to AA.[1,2] SC-26196 is orally active *in vivo*, decreasing edema in the carrageenan paw edema model in mice.[1] It also blocks aging-related increases in AA in myocardial cardiolipin in mice, attenuating contractile dysfunction without impacting mitochondrial oxidative stress.[3]

References

1. Obukowicz, M.G., Welsch, D.J., Salsgiver, W.J. , *et al.* Novel, selective Δ^6 or Δ^5 fatty acid desaturase inhibitors as anti-inflammatory agents in mice. J. Pharmacol. Exp. Ther. **287(1)**, 157-166 (1998).
2. Harmon, S.D., Kaduce, T.L., Manuel, T.D., et al. Effect of the Δ^6-desaturase inhibitor SC-26196 on PUFA metabolism in human cells. *Lipids* **38(4)**, 469-476 (2003).
3. Mulligan, C.M., Le, C.H., deMooy, A.B., *et al.* Inhibition of Δ^6 desaturase reverses cardiolipin remodeling and prevents contractile dysfunction in the aged mouse heart without altering mitochondrial respiratory function. *J. Gerontol. A Biol. Sci. Med. Sci.* **69(7)**, 799-809 (2014).

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