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- Trockeneiszuschlag
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- Expressversand

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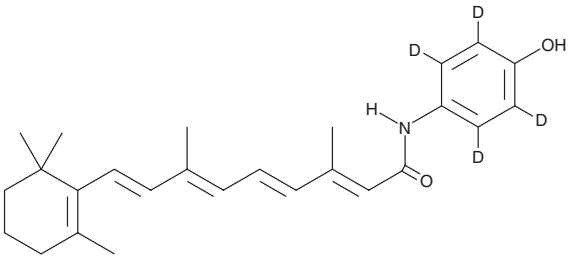
PRODUCT INFORMATION

Fenretinide-d_4

Item No. 26489

CAS Registry No.: 2118244-64-5
Formal Name: N-(4-hydroxyphenyl-2,3,5,6-d_4)-retinamide
Synonyms: 4-HPR-d_4, 4-Hydroxy(phenyl)retinamide-d_4, Retinoic Acid *p*-hydroxyphenylamide-d_4
MF: $C_{26}H_{29}D_4NO_2$
FW: 395.6
Chemical Purity: ≥98% (Fenretinide)
Deuterium Incorporation: ≥99% deuterated forms (d_1-d_4); ≤1% d_0
Supplied as: A solid
Storage: -20°C
Stability: ≥2 years



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

Laboratory Procedures

Fenretinide-d_4 is intended for use as an internal standard for the quantification of fenretinide (Item No. 17688) by GC- or LC-MS. The accuracy of the sample weight in this vial is between 5% over and 2% under the amount shown on the vial. If better precision is required, the deuterated standard should be quantitated against a more precisely weighed unlabeled standard by constructing a standard curve of peak intensity ratios (deuterated *versus* unlabeled).

Fenretinide-d_4 is supplied as a solid. A stock solution may be made by dissolving the fenretinide-d_4 in the solvent of choice. Fenretinide-d_4 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of fenretinide-d_4 in these solvents is approximately 10 mg/ml.

Description

Fenretinide is a synthetic derivative of retinoic acid (Item No. 11017) and an inhibitor of dihydroceramide desaturase (IC_{50} = 2.32 µM).[1] It promotes the generation of reactive oxygen species (ROS), the degradation of anti-apoptotic MCL-1, and the cleavage of pro-apoptotic PKCδ.[2] At 5-20 µM, fenretinide selectively activates retinoic acid receptor γ (RARγ) and is reported to inhibit the growth of Caov-3, OVCAR-3, and SKOV3 ovarian cancer cells with IC_{50} values of 3.7, 6.9, and 8.2 µM, respectively.[3,4] It also increases activity of serine palmitoyl transferase (SPT) by 1.75-fold and induces caspase-dependent apoptosis in BMEC cells (IC_{50} = 2.4 µM).[5] Fenretinide prevents lipid-induced reductions in insulin-stimulated glucose uptake in isolated rat soleus muscle.[6] *In vivo*, fenretinide (10 mg/ml in drinking water) reduces liver and soleus muscle neutral lipid content and inhibits high-fat diet-induced increases in dihydroceramide desaturase transcription.[6]

References

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2. Ruvolo, V.R., Karanjeet, K.B., Schuster, T.F., *et al. J. Signal Transduct.* **584657**, (2010).
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4. Liu, S., Brown, C.W., Berlin, K.D., *et al. J. Med. Chem.* **47(4)**, 999-1007 (2004).
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