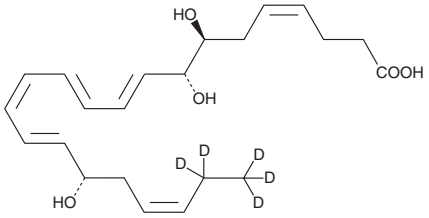
PRODUCT INFORMATION

Resolvin D1-d_5

Item No. 11182

CAS Registry No.: 1881277-32-2
Formal Name: 7S,8R,17S-trihydroxy-4Z,9E,11E,13Z,15E,19Z-21,21',22,22,22-d_5 docosahexaenoic acid
Synonyms: 17(S)-Resolvin D1-d_5, RvD1-d_5
MF: $C_{22}H_{27}D_5O_5$
FW: 381.5
Chemical Purity: ≥95%
Deuterium Incorporation: ≥99% deuterated forms (d_1-d_5); ≤1% d_0
UV/Vis.: λ_{max}: 289, 302, 317 nm
Supplied as: A solution in ethanol
Storage: -80°C
Stability: ≥1 year
Special Conditions: Light sensitive



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

Laboratory Procedures

Resolvin D1-d_5 (RvD1-d_5) is intended for use as an internal standard for the quantification of RvD1 (Item No. 10012554) by GC- or LC-mass spectrometry. 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).

RvD1-d_5 is supplied as a solution in ethanol. To change the solvent, simply evaporate the ethanol under a gentle stream of nitrogen and immediately add the solvent of choice. It is recommended that this product be stored and handled in an ethanol solution. Resolvins can isomerize and degrade when put into freeze thaw conditions and/or in solvents such as dimethyl formamide or DMSO.

Description

Resolvins are a family of potent lipid mediators derived from both eicosapentaenoic acid (EPA; Item No. 90110) and docosahexaenoic acid (DHA; Item No. 90310).[1] In addition to being anti-inflammatory, resolvins promote the resolution of the inflammatory response back to a non-inflamed state.[2] RvD1 is produced physiologically from the sequential oxygenation of DHA by 15- and 5-lipoxygenase.[1] A 17(R)-epimer of RvD1 can also be generated in aspirin-treated samples.[3] Both RvD1 and its 17(R) configuration reduce human polymorphonuclear leukocyte (PMNL) transendothelial migration, the earliest event in acute inflammation, with EC_{50} values of ~30 nM.[4] RvD1 and its aspirin-triggered form also exhibit a dose-dependent reduction in leukocyte infiltration in a mouse model of peritonitis with a maximal inhibition of ~35% at a 10-100 ng dose.[4]

References

1. Hong, S., Gronert, K., Devchand, P.R., *et al. J. Biol. Chem.* **278(17)**, 14677-14687 (2003).
2. Ariel, A. and Serhan, C.N. *TRENDS in Immunology* **28(4)**, 176-183 (2007).
3. Serhan, C.N., Hong, S., Gronert, K., *et al. J. Exp. Med.* **196(8)**, 1025-1037 (2002).
4. Sun, Y.-P., Oh, S.F., Uddin, J., *et al. J. Biol. Chem.* **282(13)**, 9323-9334 (2007).

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