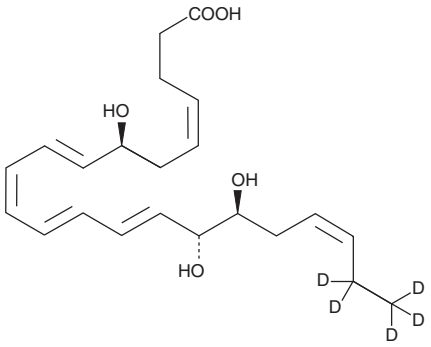
PRODUCT INFORMATION

Resolvin D2-d_5

Item No. 11184

CAS Registry No.: 1881277-33-3
Formal Name: 7S,16R,17S-trihydroxy-4Z,8E,10Z,12E,14E,19Z-21,21',22,22,22-d_5 docosahexaenoic acid
Synonyms: 7(S),16(R),17(S)-Resolvin D2-d_5, RvD2-d_5
MF: $C_{22}H_{27}D_5O_5$
FW: 381.5
Chemical Purity: ≥95% Resolvin D2
Deuterium Incorporation: ≥99% deuterated forms (d_1-d_5); ≤1% d_0
UV/Vis.: λ_{max}: 289, 302, 316 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 D2-d_5 (RvD2-d_5) is intended for use as an internal standard for the quantification of resolvin D2 (Item No. 10007279) 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).

RvD2-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 DMF or DMSO. If diluted with an aqueous buffer, this product should be discarded immediately after use.

Description

Resolvins are a family of potent lipid mediators derived from both eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).[1] In addition to being anti-inflammatory, resolvins promote the resolution of the inflammatory response back to a non-inflamed state.[2] RvD2 is produced physiologically from the sequential oxygenation of DHA by 15- and 5-lipoxygenase and functions to dampen excessive neutrophil trafficking to sites of inflammation.[3] It reduces zymosan-stimulated PMN infiltration by 70% at doses as low as 10 pg per mouse and significantly reduces PAF-stimulated leukocyte adherence and emigration at 1 nM.[3] Also, by stimulating nitric oxide production, RvD2 dose dependently decreases leukocyte-endothelial interactions. In a murine model of sepsis, RvD2 reduces leukocyte and PMN infiltration, decreases production of pro-inflammatory cytokines, and promotes phagocyte-mediated bacterial clearance.[3]

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 Immunol*. **28(4)**, 176-183 (2007).
3. Spite, M., Norling, L.V., Summers, L., *et al*. *Nature* **461(7268)**, 1287-1291 (2009).

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