Resolvin D2
Item No. 10007279

CAS Registry No.: 810668-37-2
Formal Name: 7S,16R,17S-trihydroxy-4Z,8E,10Z,12E,14E,19Z-docosahexaenoic acid
Synonyms: 7(S),16(R),17(S)-Resolvin D2, RvD2
MF: C22H32O5
FW: 376.5
Purity: ≥95%
UV/Vis.: λ_max: 289, 302, 317 nm
Supplied as: A solution in ethanol
Storage: -80°C
Stability: As supplied, 1 year from the QC date provided on the Certificate of Analysis, when stored properly
Special Conditions: Light Sensitive

Laboratory Procedures

Resolvin D2 (RvD2) 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.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. If an organic solvent-free solution of RvD2 is needed, it can be prepared by evaporating the ethanol and directly dissolving the neat oil in aqueous buffers. The solubility of RvD2 in PBS, pH 7.2, is approximately 0.05 mg/ml. Aqueous solutions of RvD2 should be discarded immediately after use.

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). In addition to being anti-inflammatory, resolvins promote the resolution of the inflammatory response back to a non-inflamed state. 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. 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. Also, by stimulating nitric oxide production, RvD2 dose dependently decreases leukocyte-endothelial interactions. In a mouse model of sepsis, RvD2 reduces leukocyte and PMN infiltration, decreases production of pro-inflammatory cytokines, and promotes phagocyte-mediated bacterial clearance. Analytical and biological comparisons of synthetic RvD2 with endogenously derived RvD2 have confirmed its identity as matching the natural product.

References


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