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SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

www.szabo-scandic.com

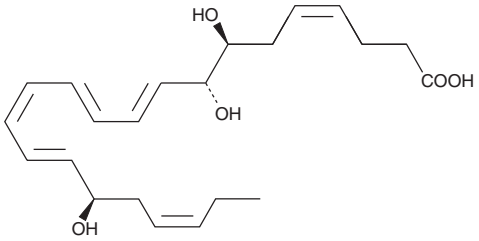
linkedin.com/company/szaboscandic

PRODUCT INFORMATION

17(R)-Resolvin D1

Item No. 13060

CAS Registry No.: 528583-91-7
Formal Name: 7S,8R,17R-trihydroxy-4Z,9E,11E,13Z,15E19Z-docosahexaenoic acid
Synonyms: Aspirin-triggered Resolvin D1, AT-RvD1, 17-*epi*-Resolvin D1, 17(R)-RvD1
MF: $C_{22}H_{32}O_5$
FW: 376.5
Purity: ≥95%
Stability: ≥1 year at -80°C
Supplied as: A solution in ethanol
Special Conditions: Light Sensitive
UV/Vis.: λ_{max}: 302 nm



Laboratory Procedures

For long term storage, we suggest that 17(R)-resolvin D1 (17(R)-RvD1) be stored as supplied at -80°C. It should be stable for at least one year.

17(R)-RvD1 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.

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 17(R)-RvD1 is needed, it can be prepared by evaporating the ethanol and directly dissolving the neat oil in aqueous buffers. The solubility of 17(R)-RvD1 in PBS, pH 7.2, is approximately 0.05 mg/ml. Aqueous solutions of 17(R)-RvD1 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).[1] In addition to being anti-inflammatory, resolvins promote the resolution of the inflammatory response back to a non-inflamed state.[2] RvD1 (Item No. 10012554) is produced physiologically from the sequential oxygenation of DHA by 15- and 5-lipoxygenase.[1] 17(R)-RvD1 is an aspirin-triggered epimer of RvD1 that reduces human polymorphonuclear leukocyte (PMN) transendothelial migration, the earliest event in acute inflammation, with equipotency to RvD1 (EC_{50} = ~30 nM).[3] 17(R)-RvD1 exhibits a dose-dependent reduction in leukocyte infiltration in a mouse model of peritonitis with maximal inhibition of ~35% at a 100 ng dose.[3] In contrast to RvD1, the aspirin-triggered form resists rapid inactivation by eicosanoid oxidoreductases. Analytical and biological comparisons of synthetic 17(R)-RvD1 with endogenously derived 17(R)-RvD1 have confirmed its identity as matching the natural product.[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 Immunol.* **28(4)**, 176-183 (2007).
3. Sun, Y.-P., Oh, S.F., Uddin, J., *et al. J. Biol. Chem.* **282(13)**, 9323-9334 (2007).
4. Serhan, C. Personal Communication.

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM