PRODUCT INFORMATION

Prostaglandin E₂ Ethanolamide
Item No. 14012

CAS Registry No.: 194935-38-1
Formal Name: N-(2-hydroxyethyl)-9-oxo-11α,15S-
dihydroxy-prosta-5Z,13E-dien-1-amide
Synonyms: Dinoprostone Ethanolamide,
PGE₂-EA, Prostamide E₂
MF: C₂₂H₄₇NO₅
FW: 395.5
Purity: ≥98%
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥1 year

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

Laboratory Procedures

Prostaglandin E₂ ethanolamide (PGE₂-EA) is supplied as a crystalline solid. A stock solution may be made by dissolving the PGE₂-EA in the solvent of choice. PGE₂-EA is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of PGE₂-EA in these solvents is approximately 100 mg/ml.

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. Organic solvent-free aqueous solutions of PGE₂-EA can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of PGE₂-EA in PBS, pH 7.2, is approximately 12 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

PGE₂-EA is an analog of PGE₂ (Item No. 14010) with improved water solubility and stability. PGE₂-EA is formed via COX-2 metabolism of arachidonoyl ethanolamide (AEA; Item No. 90050) and acts as an agonist at E prostanoid (EP) receptors 1-4 (Kᵢₛ = 2.45, 0.46, 0.2, and 0.51 μM, respectively). It also inhibits indoleamine 2,3-dioxygenase-1 (IDO-1) in THP-1 cells and human monocytes (IC₅₀ₛ = 5.7 and 4.7 μM, respectively). PGE₂-EA (10 μM) prevents morphological changes and F-actin rearrangement as well as reduces L-homocysteine-induced NLRP3 inflammasome formation and activation in podocytes. Ex vivo, PGE₂-EA reduces luminal damage and lymphocyte infiltration in a human mucosal explant colitis model.

References