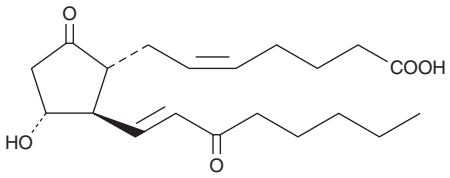
PRODUCT INFORMATION

15-keto Prostaglandin E_2

Item No. 14720

CAS Registry No.: 26441-05-4
Formal Name: 9,15-dioxo-11α-hydroxy-prosta-5Z,13E-dien-1-oic acid
Synonyms: 15-keto PGE_2, 15-oxo PGE_2
MF: $C_{20}H_{30}O_5$
FW: 350.5
Purity: ≥98%
UV/Vis.: λ_{max}: 229 nm
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥4 years



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

Laboratory Procedures

15-keto Prostaglandin E_2 (15-keto PGE_2) is supplied as a crystalline solid. 15-keto PGE_2 is sparingly soluble in water but freely soluble in organic solvents such as ethanol, DMSO, or dimethyl formamide. The solubility of 15-keto PGE_2 in these solvents is at least 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 15-keto PGE_2 can be prepared by directly dissolving the crystalline compound in aqueous buffers. The solubility of 15-keto PGE_2 in PBS, pH 7.2, is approximately 5 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

15-keto PGE_2 is a metabolite of PGE_2 (Item No. 14010) formed by 15-hydroxy prostaglandin dehydrogenase (15-PGDH).[1] Unlike PGE_2, 15-keto PGE_2 does not bind effectively to the PGE_2 receptors EP_2 and EP_4 expressed in CHO cells (K_is = 2.6 and 15 µM, respectively) or induce adenylate cyclase activity in the same cells (EC_{50}s = 1.8 and >33 µM, respectively). However, it does bind to EP_2 and EP_4 in HEK cells expressing these receptors (IC_{50}s = 0.117 and 2.82 µM, respectively), as well as induces cAMP formation (EC_{50}s = 0.137 and 0.426 µM, respectively) and the transcriptional activity of β-catenin/TCF in the same cells.[2] 15-keto PGE_2 inhibits CD3-CD28-MHC-I-induced proliferation of isolated human $CD4^+$ T cells in a concentration-dependent manner.[3] It also reduces mortality in a mouse model of LPS-induced sepsis when administered at a dose of 15 mg/kg.[4]

References

1. Nishigaki, N., Negishi, M., and Ichikawa, A. Two G_s-coupled prostaglandin E receptor subtypes, EP2 and EP4, differ in desensitization and sensitivity to the metabolic inactivation of the agonist. *Mol. Pharmacol.* **50(4)**, 1031-1037 (1996).
2. Endo, S., Suganami, A., Fukushima, K., *et al.* 15-Keto-PGE_2 acts as a biased/partial agonist to terminate PGE_2-evoked signaling. *J. Biol. Chem.* **295(38)**, 13338-13352 (2020).
3. Schmidleithner, L., Thabet, Y., Schönfeld, E., *et al.* Enzymatic activity of HPGD in Treg cells suppresses Tconv cells to maintain adipose tissue homeostasis and prevent metabolic dysfunction. *Immunity* **50(5)**, 1232-1248 (2019).
4. Chen, I.-J., Hee, S.-W., Liao, C.-H., *et al.* Targeting the 15-keto-PGE2-PTGR2 axis modulates systemic inflammation and survival in experimental sepsis. *Free Radic. Biol. Med.* **115**, 113-126 (2018).

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