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Product Information

Prostaglandin H₁

Product Number **P 6492**
Storage Temperature $-70\text{ }^{\circ}\text{C}$

CAS# 52589-22-7
Synonym: PGH₁

Product Description

Formula: C₂₀H₃₄O₅
Formula weight: 354.5

Prostaglandins are a family of fatty acid derivatives possessing a variety of potent biological activities, which are hormonal or regulatory in nature.¹ Prostaglandin H₁ (PGH₁) is the precursor to all 1-series prostaglandins and thromboxanes, and is a suicide inhibitor of platelet thromboxane synthase, possessing a K_i of 28 mM.² PGH₁ also acts on the aryl hydrocarbon receptor (AhR) by stimulating AhR transformation and DNA binding *in vitro*. It also induces AhR-dependent reporter gene expression in mouse hepatoma cells in culture.³

Reagent

Prostaglandin H₁ is supplied in an acetone solution.

Precautions and Disclaimer

This product is for laboratory research use only. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Preparation Instructions

Solutions may be prepared in DMSO, ethanol, or aqueous buffer (PBS) by evaporating the acetone using a stream of nitrogen with the product cooled on ice. The solvent of choice should be added immediately. Prostaglandin H₁ is soluble in DMSO and ethanol up to 100 mg/ml and in PBS at more than 2 mg/ml. Aqueous solutions of PGH₁ are stable for no more than 10 minutes and should be prepared immediately prior to use.

Storage/Stability

Store at $-70\text{ }^{\circ}\text{C}$. The product as supplied is stable at least two years when stored properly. Solutions in DMSO or ethanol are stable for up to 6 months when stored at $-70\text{ }^{\circ}\text{C}$.

References

1. Biochemistry, Second Ed., Lehninger, A.L., ed., Worth Publishers, (New York, NY: 1978) p. 300.
2. Jones, D.A. and Fitzpatrick, F.A., Suicide inactivation of thromboxane A₂ synthase. Characteristics of mechanism-based inactivation with isolated enzyme and intact platelets. J. Biol. Chem., **265**, 20166-20171 (1990).
3. Seidel, S.D., et al., Activation of the Ah receptor signaling pathway by prostaglandins. J. Biochem. Mol. Toxicol., **15**, 187-196 (2001).

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