**Orotic acid**

**Cat. No.:** HY-N0157  
**CAS No.:** 65-86-1  
**Molecular Formula:** C$_5$H$_4$N$_2$O$_4$  
**Molecular Weight:** 156.1  
**Target:** Nucleoside Antimetabolite/Analog; Endogenous Metabolite  
**Pathway:** Cell Cycle/DNA Damage; Metabolic Enzyme/Protease  
**Storage:**  
- Powder: -20°C 3 years  
- 4°C: 2 years  
- In solvent: -80°C 6 months  
- -20°C: 1 month  

### SOLVENT & SOLUBILITY

**In Vitro**  
DMSO: 32 mg/mL (205.00 mM; Need ultrasonic and warming)

<table>
<thead>
<tr>
<th>Solvent Concentration</th>
<th>Mass 1 mg</th>
<th>Mass 5 mg</th>
<th>Mass 10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>6.4061 mL</td>
<td>32.0308 mL</td>
<td>64.0615 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>1.2812 mL</td>
<td>6.4061 mL</td>
<td>12.8123 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.6406 mL</td>
<td>3.2031 mL</td>
<td>6.4061 mL</td>
</tr>
</tbody>
</table>

Please refer to the solubility information to select the appropriate solvent.

### BIOLOGICAL ACTIVITY

**Description**  
Orotic acid (OA) is an intermediate in pyrimidine metabolism. IC$_{50}$ Value: Target: Nucleoside antimetabolite/analog in vitro: OA increases cell proliferation and decreases apoptosis in serum-starved SK-Hep1 hepatocellular carcinoma cells, which may ascribe to the inhibition of AMP-activated protein kinase (AMPK) phosphorylation and thus activation of mammalian target of rapamycin complex 1 (mTORC1) [1]. In vivo: male Fischer 344 rats (130-150 g) to two-thirds PH in the absence or in the presence of OA (a 300-mg tablet of OA methyl ester implanted intraperitoneally at the time of two-thirds PH). Treatment with OA resulted in a near-100% inhibition of RNR induced by two-thirds PH in rat liver, as measured by enzyme activity and protein level [2]. The increases of hepatic OA and betaine levels in OA feeding rats was also found when compared to the normal rats [3]. Feeding 1% OA with diet decreased the phosphorylation of AMPK and increased the maturation of SREBP-1 and the expression of SREBP-responsive genes in the rat liver. OA-induced lipid accumulation was also completely inhibited by rapamycin. Mouse hepatocytes and mice were resistant to OA-induced lipogenesis because of little if any response in AMPK and downstream effectors [4].

**IC$_{50}$ & Target**  
Human Endogenous Metabolite
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REFERENCES


