Uridine triphosphate

**Cat. No.:** HY-107372  
**CAS No.:** 63-39-8  
**Molecular Formula:** C₉H₁₅N₂O₁₅P₃  
**Molecular Weight:** 484.14  
**Target:** Endogenous Metabolite  
**Pathway:** 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  
H₂O : ≥ 150 mg/mL (309.83 mM)  
* “≥” means soluble, but saturation unknown.  

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Concentration</th>
<th>Mass</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mM</td>
<td></td>
<td>2.0655 mL</td>
<td>10.3276 mL</td>
<td>20.6552 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td></td>
<td>0.4131 mL</td>
<td>2.0655 mL</td>
<td>4.1310 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td></td>
<td>0.2066 mL</td>
<td>1.0328 mL</td>
<td>2.0655 mL</td>
</tr>
</tbody>
</table>

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

**BIOLOGICAL ACTIVITY**

**Description**  
Uridine triphosphate (UTP; Uridine 5’-triphosphate) is a nucleotide that regulates the functions of the pancreas in endocrine and exocrine secretion, proliferation, channels, transporters, and intracellular signaling under normal and disease states.

**IC₅₀ & Target**  
Human Endogenous Metabolite

**In Vitro**  
Uridine triphosphate treatment induces Schwannoma cell migration through activation of P2Y2 receptors and through the increase of extracellular matrix metalloproteinase-2 (MMP-2) activation and expression[^1^]. Uridine triphosphate-induced proliferation is mediated by protein kinase D, Src-family tyrosine kinase, Ca/calmodulin-dependent protein kinase II, phosphatidylinositol 3-kinase (PI3K), Akt, and phospholipase D. Uridine triphosphate increases phosphorylation of Akt through protein kinase C, Src-family tyrosine kinase, Ca/calmodulin-dependent protein kinase II, and PI3K[^2^].
**In Vivo**

<table>
<thead>
<tr>
<th>PROTOCOL</th>
<th>Uridine triphosphate reduces mitochondrial calcium levels following hypoxia. Early or late uridine triphosphate preconditioning is effective to reduce infarct size and superior myocardial function(^3). Uridine triphosphate treatment increases the number of monocytes and macrophages infiltrating the pouch and up-regulates the gene expression of IL-4 and IL-13 in the regional lymph nodes(^4).</th>
</tr>
</thead>
</table>

**PROTOCOL**

**Cell Assay**\(^2\)

Pancreatic duct epithelial cells are treated with (0.1 μM-1 mM) uridine triphosphate. BrdU is added after different time points (12-24 hours). Proliferation is measured using the BrdU incorporation assay in triplicate\(^2\).

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**Animal Administration**\(^3\)

Rats: Four main groups are tested: (1) sham without LAD ligation, (2) LAD ligation, (3) injected with UTP (0.44 μg/kg i.v.) 30 min before MI, (4) UTP injection (4.4 μg/kg i.v.) 24 h prior to MI. Several different concentrations of UTP are tested (0.044–44.4 μg/kg). Left ventricular systolic pressure (LVSP), end-diastolic pressure (LVEDP) and heart rates are monitored before, after UTP treatment and post MI\(^3\).

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**REFERENCES**


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**Caution:** Product has not been fully validated for medical applications. For research use only.