Arg-Gly-Asp-Ser

Cat. No.: HY-12290
CAS No.: 91037-65-9
Molecular Formula: C₁₅H₂₇N₇O₈
Molecular Weight: 433.42
Sequence: Arg-Gly-Asp-Ser
Sequence Shortening: RGDS
Target: Integrin
Pathway: Cytoskeleton
Storage: Powder
-80°C  2 years
-20°C  1 year
In solvent
-80°C  6 months
-20°C  1 month

SOLVENT & SOLUBILITY

In Vitro
DMSO : ≥ 55 mg/mL (126.90 mM)
H₂O : ≥ 25 mg/mL (57.68 mM)
a "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

<table>
<thead>
<tr>
<th>Solvent</th>
<th>Concentration</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
</tr>
<tr>
<td>DMSO</td>
<td>1 mM</td>
<td>2.3072 mL</td>
<td>11.5362 mL</td>
<td>23.0723 mL</td>
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<tr>
<td></td>
<td>5 mM</td>
<td>0.4614 mL</td>
<td>2.3072 mL</td>
<td>4.6145 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.2307 mL</td>
<td>1.1536 mL</td>
<td>2.3072 mL</td>
</tr>
</tbody>
</table>

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

BIOLOGICAL ACTIVITY

Description
Arg-Gly-Asp-Ser is an integrin binding sequence that inhibits integrin receptor function, decreases systemic inflammation via inhibition of collagen-triggered activation of leukocytes and attenuates expression of inflammatory cytokines, iNOS and MMP-9.

In Vitro
The Arg-Gly-Asp-Ser-modified surface causes up-regulation of αvβ3 integrin. Attachment to the Arg-Gly-Asp-Ser-treated membrane completely abolishes apoptosis induced by staurosporine, the Ca²⁺·Pi ion pair, and sodium nitroprusside. Arg-Gly-Asp-Ser-dependent resistance to apoptosis is eliminated, when the activity of the phosphatidylinositol 3-kinase pathway is inhibited [1]. Arg-Gly-Asp-Ser interacts with survivin, as well as with procaspase-3, -8 and -9. Arg-Gly-Asp-Ser-peptide binding to survivin is found to be specific, at high affinity (Kₐ 27.5...
μM) and locates at the survivin C-terminus. Arg-Gly-Asp-Ser-survivin interaction appears to play a key role, since Arg-Gly-Asp-Ser lost its anti-mitogenic effect in survivin-deprived cells with a specific siRNA[4].

**In Vivo**

Arg-Gly-Asp-Ser (2.5 or 5 mg/kg, 1 h before LPS) significantly inhibits LPS-induced MMP-9 activity in BAL fluid 4 h post-LPS. Arg-Gly-Asp-Ser (1, 2.5 or 5 mg/kg, i.p.) administers 1 h before LPS inhibited LPS-induced increases in TNF-α and MIP-2 levels in BAL fluid at 4 h post-LPS[2]. Arg-Gly-Asp-Ser peptide significantly reduces tumor necrosis factor (TNF)-α and macrophage inflammatory protein (MIP)-2 production, and decreases myeloperoxidase (MPO) and NF-κ B activity[3].

**PROTOCOL**

**Cell Assay**[1]

Cell death is measured using the MTT analysis. This assay is based on the ability of mitochondrial dehydrogenases to oxidize thiazolyl blue (MTT), a tetrazolium salt (3-[4,5-dimethylthiazol-2-yl]-2,5-diphenylterazolium bromide), to an insoluble blue formazan product. The cells are incubated with the MTT reagent (120 μg/mL) at 37°C for 2 h. After the supernatant is removed, 400 μL of 0.04mol/LHCl in isopropanol is added to each well, and the optical density of the solution is read at 590 nm in an enzyme-linked immunosorbent assay plate reader. As the generation of the blue product is proportional to the dehydrogenase activity, a decrease in the absorbance at 590 nm provides a direct measurement of the number of viable cells. To determine the contribution of the PI3K pathway to inhibition of apoptosis, some cell populations are pretreated with 50 μM LY294002, a PI3K inhibitor. Following this pretreatment, cell death is determined as described above.

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

**Animal Administration**[2]

Mice pharyngeal aspiration is performed as described. Animals are anesthetized with a mixture of ketamine and xylazine (45 mg/kg and 8 mg/kg, i.p., respectively). Test solution (30 μL) containing LPS (1.5 mg/kg) is placed posterior in the throat and aspirated into the lungs. Control mice are administrated sterile saline (0.9% NaCl). Animals are administered with Arg-Gly-Asp-Ser or RGES peptide (1, 2.5 or 5 mg/kg, i.p.) once one hour before LPS treatment and sacrificed 4 h post-LPS. Animals are also administered Arg-Gly-Asp-Ser or RGES peptide (5 mg/kg, i.p.) once at different time points (1 h before or 2 h after LPS treatment) and sacrificed 24 h post-LPS. In addition, animals are administered with αvβ3-blocking mAbs, anti-αv, or anti-β3 (5 mg/kg, i.p.) once 1 h before and sacrificed 4 h post-LPS. Animals administered with these mAbs 2 h after LPS treatment are sacrificed 24 h post-LPS.

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**REFERENCES**

