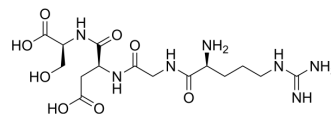


## Arg-Gly-Asp-Ser

Cat. No.:	HY-12290
CAS No.:	91037-65-9
Molecular Formula:	C <sub>15</sub> H <sub>27</sub> N <sub>7</sub> O <sub>8</sub>
Molecular Weight:	433.42
Sequence:	Arg-Gly-Asp-Ser
Sequence Shortening:	RGDS
Target:	Integrin
Pathway:	Cytoskeleton
Storage:	Sealed storage, away from moisture
	Powder    -80°C    2 years
	-20°C    1 year



\* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)

### SOLVENT & SOLUBILITY

#### In Vitro

H<sub>2</sub>O : ≥ 25 mg/mL (57.68 mM)

\* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	2.3072 mL	11.5362 mL	23.0723 mL
	5 mM	0.4614 mL	2.3072 mL	4.6145 mL
	10 mM	0.2307 mL	1.1536 mL	2.3072 mL

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. Arg-Gly-Asp-Ser directly and specifically bind pro-caspase-8, pro-caspase-9 and pro-caspase-3, while it does not bind pro-caspase-1.

#### 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 Ca<sup>2+</sup>-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<sup>[1]</sup>. 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 (K<sub>d</sub> 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<sup>[4]</sup>.

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

## 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.) administered 1 h before LPS inhibited LPS-induced increases in TNF- $\alpha$  and MIP-2 levels in BAL fluid at 4 h post-LPS<sup>[2]</sup>. Arg-Gly-Asp-Ser peptide significantly reduces tumor necrosis factor (TNF)- $\alpha$  and macrophage inflammatory protein (MIP)-2 production, and decreases myeloperoxidase (MPO) and NF- $\kappa$ B activity<sup>[3]</sup>. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## PROTOCOL

### Cell Assay <sup>[1]</sup>

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  $\mu$ g/mL) at 37°C for 2 h. After the supernatant is removed, 400  $\mu$ L 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  $\mu$ M 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 <sup>[2]</sup>

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  $\mu$ L) containing LPS (1.5 mg/kg) is placed posterior in the throat and aspirated into the lungs. Control mice are administered 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  $\alpha$ v $\beta$ 3-blocking mAbs, anti- $\alpha$ v, or anti- $\beta$ 3 (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. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## CUSTOMER VALIDATION

- Theranostics. 2020 Oct 26;10(26):12127-12143.
- iScience. 22 December 2022, 105642.
- Sci Rep. 2021 Jan 25;11(1):2141.
- Mol Hum Reprod. 2021 Feb 24;gaab014.
- J Oral Pathol Med. 2016 Nov;45(10):730-739.

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## REFERENCES

- [1]. Grigoriou V, et al. Apoptosis and survival of osteoblast-like cells are regulated by surface attachment. *J Biol Chem*. 2005 Jan 21;280(3):1733-9.
- [2]. Moon C, et al. Synthetic RGDS peptide attenuates lipopolysaccharide-induced pulmonary inflammation by inhibiting integrin signaled MAP kinase pathways. *Respir Res*. 2009 Mar 9;10:18.
- [3]. Yin X, et al. Synthetic RGDS peptide attenuated lipopolysaccharide/D-galactosamine-induced fulminant hepatic failure in mice. *J Gastroenterol Hepatol*. 2014 Jun;29(6):1308-15.

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

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