# Arg-Gly-Asp-Ser

**Cat. No.:** HY-12290

**CAS No.:** 91037-65-9

**Molecular Formula:** $C_{15}H_{27}N_7O_8$

**Molecular Weight:** 433.42

**Sequence:** Arg-Gly-Asp-Ser

**Sequence Shortening:** RGDS

**Target:** Integrin

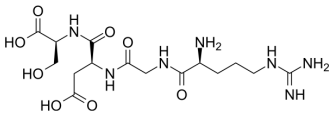
**Pathway:** Cytoskeleton

**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**

DMSO : 50 mg/mL (115.36 mM; Need ultrasonic)

$H_2O$ : ≥ 25 mg/mL (57.68 mM)

* "≥" means soluble, but saturation unknown.

| Preparing Stock Solutions | Mass / Solvent / Concentration | 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 $Ca^{2+}$·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 ($K_d$ 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].

| | |
|---|---|
| | 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.<br>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].<br>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-κB activity[3].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## 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.<br>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<br>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.

See more customer validations on www.MedChemExpress.com

## 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.

[4]. Aguzzi MS, et al. Intracellular targets of RGDS peptide in melanoma cells. Mol Cancer. 2010 Apr 22;9:84.



Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA