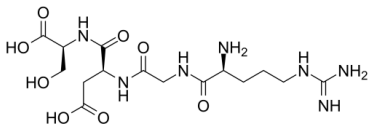
Data Sheet

Product Name:	Arg-Gly-Asp-Ser
Cat. No.:	CS-3266
CAS No.:	91037-65-9
Molecular Formula:	C15H27N7O8
Molecular Weight:	433.42
Target:	Integrin
Pathway:	Cytoskeleton
Solubility:	DMSO : ≥ 55 mg/mL (126.90 mM); H2O : ≥ 25 mg/mL (57.68 mM)



BIOLOGICAL ACTIVITY:

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]. **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-κB activity[3].

PROTOCOL (Extracted from published papers and Only for reference)

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. **Animal Administration:** Arg-Gly-Asp-Ser is formulated in sterile saline (0.9% NaCl).[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

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.

CAIndexNames:

L-Serine, L-arginylglycyl-L-α-aspartyl-

SMILES:

O=C(N[C@@H](CO)C(O)=O)[C@H](CC(O)=O)NC(CNC([C@@H](N)CCCNC(N)=N)=O)=O

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