Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Arg-Gly-Asp-Ser

Catalog No: tcsc3266

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Specifications

CAS No:
91037-65-9

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

Pathway:
Cytoskeleton

Target:
Integrin

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 55 mg/mL (126.90 mM); H2O : ≥ 25 mg/mL (57.68 mM)

Alternative Names:
RGDS peptide; Fibronectin tetrapeptide

Observed Molecular Weight:
433.42

Product Description

Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

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 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].