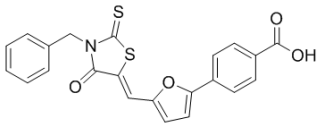
Leukadherin-1

Cat. No.:	HY-15701
CAS No.:	344897-95-6
Molecular Formula:	$C_{22}H_{15}NO_4S_2$
Molecular Weight:	421.49
Target:	Complement System; Integrin
Pathway:	Immunology/Inflammation; Cytoskeleton
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5 mg/mL (11.86 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3725 mL	11.8627 mL	23.7254 mL
5 mM	0.4745 mL	2.3725 mL	4.7451 mL
10 mM	0.2373 mL	1.1863 mL	2.3725 mL

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

BIOLOGICAL ACTIVITY

Description

Leukadherin-1 is a specific agonist of complement receptor 3 (**CR3**) and the leukocyte surface **αMβ2 integrin CD11b/CD18**.

In Vitro

Leukadherin-1 (LA1) modulates natural killer (NK) cell inflammatory cytokine secretion. The SLE-associated CD11b-R77H variant does not influence NK cell response to Leukadherin-1. Leukadherin-1 does not modulate Syk activation in NK cells. Leukadherin-1 (LA1) does not modulate signal transducer and activator of transcription (STAT)-4 phosphorylation. Leukadherin-1 modulates TLR-2 and TLR-7/8-induced monocyte cytokine secretion[1].

In Vivo

Leukadherin-1 decreases macrophage infiltration in the lungs during hyperoxia. Furthermore, treatment with Leukadherin-1 improves alveolarization and angiogenesis and decreases pulmonary vascular remodeling and PH. Targeting leukocyte trafficking using Leukadherin-1, an integrin agonist, is beneficial in preventing lung inflammation and protecting alveolar and vascular structures during hyperoxia[2].

PROTOCOL

Cell Assay [1]

Supernatant cytokines are quantified after stimulation and culture for 18 h (monocytes) or 24 h (NK cells). Except for bead-based stimulation, all experiments are conducted using 100 µL cells in a 96-well plate format. NK cell stimuli are added as follows: (1) Syk inhibitor (1 µM), (2) Leukadherin-1 or dimethylsulphoxide (DMSO) (vector control) (7.5 µM). Shown to induce~82% of maximum response with negligible off-target effect, (3) anti-CD210 or isotype control (5 µg/mL), (4) 30-45 min after Leukadherin-1 NK cells are stimulated with combinations of IL-12 (10 ng/mL), IL-15 (30 ng/mL) or IL-18 (10 ng/mL): either IL-12 + IL-15 or IL-12 + IL-18. Monocytes are stimulated using pam3csk4 (TLR-2 agonist, 300 ng/mL) or R848 (TLR-7/8 agonist, 2 µg/mL). Supernatants are stored at -80ºC for < 1 month before quantification. To exclude non-specific Leukadherin-1-mediated cytotoxicity, the cell viability is assayed at 24 h using the CellTitre-Glo reagent. No significant loss of viability in comparison with the DMSO control is seen, concurring with published data in other cell types.

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

REFERENCES

[1]. Roberts AL, et al. The complement receptor 3 (CD11b/CD18) agonist Leukadherin-1 suppresses human innate inflammatory signalling. Clin Exp Immunol. 2016 Sep;185(3):361-71.

[2]. Jagarapu J, et al. Efficacy of Leukadherin-1 in the Prevention of Hyperoxia-Induced Lung Injury in Neonatal Rats. Am J Respir Cell Mol Biol. 2015 Dec;53(6):793-801.

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