Product Data Sheet

Product Name: Leukadherin 1
Cat. No.: GC17263

Chemical Properties

Cas. No.	344897-95-6
Chemical Name	(Z)-4-(5-((3-benzyl-4-oxo-2-thioxothiazolidin-5-ylidene)methyl)furan-2-yl)benzoic acid
SMILES	O=C(N(C1=S)CC2=CC=CC=C2)/C(S1)=C([H])/C(O3)=CC=C3C4=CC=C(C(O)=O)C=C4

Formula	$C_{22}H_{15}NO_4S_2$	M.Wt	421.49
Solubility	DMF: 1 mg/ml,DMSO: 1 mg/ml,DMSO:PBS(pH 7.2) (1:2): 0.3 mg/ml	Storage	Store at -20°C

General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

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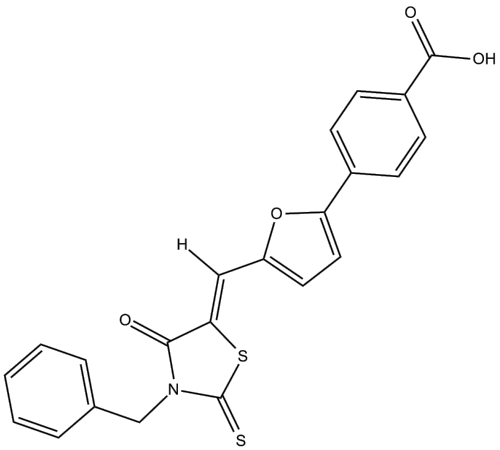
Structure



Protocol

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Cell experiment: 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.

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.

Background

Leukadherin 1 is an activator of CD11b/CD18 [1].

CD11b/CD18 is a cell surface adhesion receptor that mediates immune functions and leukocyte migration and is expressed primarily in the cells of the innate immune system.

Leukadherin 1 is a CD11b/CD18 activator. Leukadherin 1 increased CD11b/CD18-dependent cell adhesion to fibrinogen with EC50 value of 4 µM [1]. Leukadherin 1 increased CD11b/CD18-dependent cell adhesion to its ligand ICAM-1. In K562 cells, leukadherin 1 increased the binding of CD11b/CD18 to ICAM-1 through the formation of long membrane tethers [2].

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In the acute peritonitis mice model, leukadherin 1 significantly reduced neutrophil accumulation by 40% [1]. In a MHC mismatched orthotopic kidney transplantation mouse model, leukadherin 1 reduced interstitial leukocyte infiltration, glomerular damage, neointimal hyperplasia and prolonged survival from 48.5% (CsA only) to 100% (CsA and LA1) [3]. In newborn rats exposed to hyperoxia (85% O2), leukadherin 1 reduced both macrophage and neutrophil infiltration in the lungs [4].

References:

[1]. Maiguel D, Faridi MH, Wei C, et al. Small molecule-mediated activation of the integrin CD11b/CD18 reduces inflammatory disease. Sci Signal, 2011, 4(189): ra57.
[2]. Celik E1, Faridi MH, Kumar V, et al. Agonist leukadherin-1 increases CD11b/CD18-dependent adhesion via membrane tethers. Biophys J, 2013, 105(11): 2517-2527.
[3]. Khan SQ, Guo L, Cimbaluk DJ, et al. A small molecule b2 integrin agonist improves chronic kidney allograft survival by reducing leukocyte recruitment and accompanying vasculopathy. Front Med (Lausanne), 2014, 1: 45.
[4]. Jagarapu J, Kelchtermans J, Rong M, et al. Leukadherin-1 Attenuates Hyperoxia-induced Lung Injury in Neonatal Rats. Am J Respir Cell Mol Biol, 2015.

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