Emapticap pegol

Cat. No.:	HY-148100
CAS No.:	1390630-22-4
Sequence:	Poly(oxy-1,2-ethanediyl), α-hydro-ω-methoxy-, 5′-ether with RNA β-L-(G-C-A-C-G-U-C-C-C-U-C-A-C-C-G-G-U-G-C-A-A-G-U-G-A-A-G-C-C-G-U-G-G-C-U-C-U-G-C-G) 5′-[6-[[2-[(2-hydroxyacetyl)(2-hydroxyethyl)amino]acetyl]amino]hexyl hydrogen phosphate] (2:1)
Target:	Others
Pathway:	Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

Emapticap pegol

SOLVENT & SOLUBILITY

In Vitro	H_2O : 50 mg/mL (Need ultrasonic)

BIOLOGICAL ACTIVITY

Description	Emapticap pegol is a inhibitor of pro-inflammatory chemokine C-C motif-ligand 2 (CCL2). Emapticap pegol is a 40-nucleotide oligonucleotide aptamer, displays different Spiegelmers (L-RNA aptamer) isform in human (NOX-E36) and mouse (mNOX-E36)[1][2][3].
IC_{50} & Target	chemokine C-C motif-ligand 2 (CCL2)[1]; MCP-1[3]
In Vitro	Spiegelmers are RNA-like molecules built from L-ribose units that are able to bind molecules such as peptides and proteins. NOX-E36, is human-specific CCL2 Spiegelmer; and mNOX-E36, is the mouse-specific CCL2 Spiegelmer[2]. NOX-E36 (1 nM) significantly inhibits CCL2-mediated migration in human monocytic leukemia cell line THP-1[2]. NOX-E36 inhibits monocyte chemotactic protein-1 (MCP-1), and blocks the inflammatory cell recruitment and differentiation of macrophages mediated by MCP-1[3]. mNOX-E36 inhibits the migration and signaling pathway activation in murine hematopoietic cells, and blocks CCL2 receptor expressing Ba/F3 cells (Ba/F3-CCR2) migration (~2000 fold than normal migration) in a dose-dependent manner[2]. mNOX-E36 abrogates the phosphorylation induced by CCL2 of AKT, ERK, p35-MAPK, respectively in mCCL2-stimulated cells (30 min)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Emapticap pegol (14.4 mg/kg, mNOX-E36; s.c.; three times per week, for 3 weeks) interferes the infiltration of M2-like macrophages into spleens of leukemia-bearing mice[2]. Emapticap pegol (20 mg/kg, mNOX-E36; s.c.; three times per week, for 4 weeks) reduces albuminuria and restores the glomerular endothelial glycocalyx in diabetic mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Non-irradiated immunocompetent C57BL/6 mice injected with syngeneic AML1/ETO9a-expressing primary murine leukemia cells[2]

Dosage:	14.4 mg/kg (mNOX-E36, Emapticap pegol of the mouse-specific CCL2 Spiegelmer)
Administration:	Subcutaneous injection; three times per week for 3 weeks
Result:	Abrogated this macrophage infiltration within the leukemia microenvironment.

Animal Model:	Male Apoe KO C57BL/6J mice rendered diabetic (6-week-old)[3]
Dosage:	20 mg/kg (mNOX-E36, Emapticap pegol of the mouse-specific CCL2 Spiegelmer)
Administration:	Subcutaneous injection; three times per week for 4 weeks
Result:	Reduced albumin/creatinine ratio without affecting blood glucose level and weight of mice. Reduced heparanase and cathepsin L expression.

REFERENCES

[1]. Menne J, et al. C-C motif-ligand 2 inhibition with emapticap pegol (NOX-E36) in type 2 diabetic patients with albuminuria. Nephrol Dial Transplant. 2017 Feb 1;32(2):307-315.

[2]. Rodrigo, et al. Effects of CCL2/CCR2 Blockade in Acute Myeloid Leukemia. Blood.

[3]. Boels MGS, et al. Systemic Monocyte Chemotactic Protein-1 Inhibition Modifies Renal Macrophages and Restores Glomerular Endothelial Glycocalyx and Barrier Function in Diabetic Nephropathy. Am J Pathol. 2017 Nov;187(11):2430-2440.

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