Efdamrofusp alfa

Cat. No.: HY-P99905

CAS No.: 2375661-82-6

Target: VEGFR

Pathway: Protein Tyrosine Kinase/RTK

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description

Efdamrofusp alfa is a bispecific fusion protein. Efdamrofusp alfa is capable of neutralizing both VEGF isoforms and C3b/C4b. Efdamrofusp alfa can be used for the research of neovascular age-related macular degeneration (nAMD) and other complement-related ocular conditions[1].

In Vitro

Efdamrofusp alfa (0.135 mg/mL; 0, 6, 12, or 24 h) suppresses endothelial cell migration and tube formation[1].
Efdamrofusp alfa (0-1000 μg/mL) inhibits complement activation in vitro[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Migration Assay [1]

Cell Line:	Human primary umbilical vein endothelial cell (HUVEC)
Concentration:	0.135 mg/mL
Incubation Time:	0, 6, 12, or 24 h
Result:	Showed a 20.91% reduction in migration.

In Vivo

Efdamrofusp alfa (13.5 μg; 3 days) inhibits activation ofthe complement system in a mouse model oflaser-induced CNV[1].
Efdamrofusp alfa (1.35 mg; single intravitreal injection) shows favorable safety profiles and exhibited antiangiogenetic efficacy in a nonhuman primate laser-induced CNV model[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6J mice[1]
Dosage:	13.5 μg; 13.0 μg
Administration:	3 days; 7days
Result:	Significantly reduced C3d deposition. Reduced vascular leakage at 7 days after laser-induced injury. Significantly suppressed CNV formation 7 days after laser-induced injury. Reduced the concentrations of vitreous VEGF-A.

Animal Model:	Rhesus monkeys[1]
Dosage:	1.35 mg
Administration:	Single intravitreal injection
Result:	Decreased the CNV leakage at 14 and 28 days and effectively reduced CNV volume 28 days.

CUSTOMER VALIDATION

- ArchaeGraph. 2023 Jul.

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REFERENCES

[1]. Shiqi Yang, et al. Targeting C3b/C4b and VEGF with a bispecific fusion protein optimized for neovascular age-related macular degeneration therapy. Sci Transl Med. 2022 Jun;14(647):eabj2177.

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