Aflibercept

Cat. No.: HY-108801

CAS No.: 862111-32-8

Target: VEGFR

Pathway: Protein Tyrosine Kinase/RTK

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

BIOLOGICAL ACTIVITY

Description

Aflibercept (VEGF Trap) is a soluble decoy VEGFR constructed by fusing the Ig domains of VEGFR1 and VEGFR2 with the Fc region of human IgG1. Aflibercept inhibits VEGF signaling by reducing VEGF-regulated processes. Aflibercept can be used for thr research of age-related macular degeneration (AMD) and cardiovascular disease[1][2][3].

In Vitro

Aflibercept (500 μg/mL; 24 h and 7 d) shows no toxicity on RPE cells, neither in MTT-assay nor in trypan blue exclusion assay [1].

Aflibercept (500 μg/mL; 24 h) shows a statistically significant effect on wound healing compared with control in the confluent RPE cell layer with three wounds[1].

Aflibercept (500 μg/mL; 7 d) displays a significantly diminished phagocytosis of opsonised latex beads compared to untreated control[1].

Aflibercept (1 and 10 μg/mL; 10 h) inhibits VEGF signaling by reducing VEGF-regulated processes, such as permeability and angiogenesis[2].

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

In Vivo

Aflibercept (10 mg/kg; 3 h post-middle cerebral artery occlusion (MCAO)) reduces stroke-induced VEGF-A and VEGFR2 expression, and brain edema, and BBB disruption and improves poststroke survival in obese mice[2].

Aflibercept (18.2 mg/kg and 36.4 mg/kg; i.v. once) affects BP, ROS and eNOS production in mice[3].

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

Animal Model:	Male C57BL/6 mice[3]
Dosage:	18.2 mg/kg and 36.4 mg/kg
Administration:	Intravenous injection; 18.2 mg/kg and 36.4 mg/kg once
Result:	Rapidly and dose-dependently elevated BP in mice and markedly impaired endothelial-dependent relaxation (EDR) and resulted in NADPH oxidases 1 (NOX1)- and NADPH oxidases 4 (NOX4)-mediated generation of ROS, decreased the activation of protein kinase B (Akt) and endothelial nitric oxide synthase (eNOS) concurrently with a reduction in nitric oxide (NO) production and elevation of ET-1 levels in mouse aortas.

REFERENCES

[1]. Klettner A, et al. Effects of aflibercept on primary RPE cells: toxicity, wound healing, uptake and phagocytosis. Br J Ophthalmol. 2014 Oct;98(10):1448-52.

[2]. Kim ID, et al. Aflibercept, a VEGF (Vascular Endothelial Growth Factor)-Trap, Reduces Vascular Permeability and Stroke-Induced Brain Swelling in Obese Mice. Stroke. 2021 Aug;52(8):2637-2648.

[3]. Dong ZC, et al. The vascular endothelial growth factor trap aflibercept induces vascular dysfunction and hypertension via attenuation of eNOS/NO signaling in mice. Acta Pharmacol Sin. 2021 Sep;42(9):1437-1448.

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