Product Data Sheet

Product Name: Ranibizumab
Cat. No.: GC19801

Chemical Properties

Cas. No.	347396-82-1		
Formula		M.Wt	
Solubility	Soluble in water	Storage	-80°C, avoid multiple freeze-thaws.
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.		
Structure			

Protocol

Cell experiment [1]:

Cell lines	Müller cells (the major type of glial cells in the retina)

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Preparation Method	Clinical doses of Aflibercept (0.5mg/mL) and Ranibizumab (0.125mg/mL) were dissolved in starvation media containing 1% FCS, various concentrations of glucose along with and without $CoCl_2$ (200μM). Müller cells and Y79 photoreceptors were incubated in 12 starvation media containing 1% FCS and other elements including: (1) low glucose (LG); (2) LG+Aflibercep; (3) LG+Ranibizumab; (4) LG+$CoCl_2$ (200μM; (5) LG+$CoCl_2$+Aflibercep; (6) LG+$CoCl_2$+Ranibizumab; (7) high glucose (HG), (8) HG+Aflibercep; (9) HG+Ranibizumab; (10) HG+$CoCl_2$; (11) HG+$CoCl_2$+Aflibercep; (12) HG+$CoCl_2$+Ranibizumab. The effects of Aflibercept and Ranibizumab on cell survival, differential expression of cell stress markers and the production of NT3, BDNF and PEDF as well as IRBP were assessed 24h after incubation.
Reaction Conditions	0.125mg/mL; 24h
Applications	Both Aflibercept and Ranibizumab inhibited the production of IRBP in Y79 photoreceptors. Aflibercept and Ranibizumab are not toxic to Müller cells. Aflibercept and Ranibizumab did not significantly affect the production of neurotrophic factors (NT3, BDNF and PEDF) by Müller cells under the stress conditions we tested.

Animal experiment [2]:

Animal models	C57BL/6J mice

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Preparation Method	PBS, 10mg/kg of Aflibercept, or 2.5mg/kg or 10mg/kg of Ranibizumab was intraperitoneally injected into C57BL/6J mouse neonates daily from P3 to P6. To minimize the effects of individual differences, littermates were divided into the above four groups. The mice were weighed at all time points of injection. At P6, eyes and kidneys were harvested 6h after the last injection.
Dosage form	2.5, 10mg/kg/day; i.p.
Applications	Body weight gain in pups treated with Aflibercept was significantly impaired comparing with other three groups. The body weight gain in pups treated with 2.5mg/kg or 10mg/kg of Ranibizumab was comparable with that in PBS treated pups.

References:

[1]Shen W, Yau B, Lee S R, et al. Effects of ranibizumab and aflibercept on human Müller cells and photoreceptors under stress conditions[J]. International journal of molecular sciences, 2017, 18(3): 533.
[2]Ichiyama Y, Matsumoto R, Obata S, et al. Assessment of mouse VEGF neutralization by ranibizumab and aflibercept[J]. PloS one, 2022, 17(12): e0278951.

Background

Ranibizumab is a humanized anti-vascular endothelial growth factor (VEGF) monoclonal antibody Fab fragment that can recognize all VEGF-A isoforms (including VEGF110, VEGF121, and VEGF165)[1]. Ranibizumab inhibits pathological angiogenesis and vascular leakage by blocking the interaction between VEGF-A and receptors (VEGFR1 and VEGFR2). It is mainly used in the treatment of wet age-related macular degeneration (wAMD), diabetic macular edema (DME) and retinal vein occlusion (RVO)[2, 3]. Ranibizumab has high intraocular penetration and low immunogenicity[4].

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In vitro, Ranibizumab (0.125mg/mL) treated retinal glial cells (Müller cells) for 24h inhibited the production of inter-photoreceptor compound binding protein (IRBP) in Y79 photoreceptors, without affecting cell viability, the production of neurotrophic factors and the expression of cell stress markers[5]. In vitro, Ranibizumab (0.02-5.17nM) treated human umbilical vein endothelial cells (HUVEC) inhibited rhVEGF165, rhVEGF121, and rhVEGF110-induced HUVEC proliferation in a dose-dependent manner, with IC_{50} values of 0.44nM, 0.56nM, and 0.23nM, respectively[6].

In vivo, Ranibizumab (2.5, 10mg/kg/day) treated newborn mice by intraperitoneal injection for 4 days had no effect on the weight gain of mice, while the weight gain of mice treated with Aflibercept was significantly impaired[7].

References:

[1] Lien S, Lowman H B. Therapeutic anti-VEGF antibodies[J]. Therapeutic antibodies, 2008: 131-150.
[2] Ferro Desideri L, Barra F, Ferrero S, et al. Clinical efficacy and safety of ranibizumab in the treatment of wet age-related macular degeneration[J]. Expert opinion on biological therapy, 2019, 19(8): 735-751.
[3] Amadio M, Govoni S, Pascale A. Targeting VEGF in eye neovascularization: What's new?: A comprehensive review on current therapies and oligonucleotide-based interventions under development[J]. Pharmacological research, 2016, 103: 253-269.
[4] Stewart M W. Intraocular drugs: pharmacokinetic strategies and the influence on efficacy and durability[J]. Expert Opinion on Drug Metabolism & Toxicology, 2024, 20(10): 977-987.
[5] Shen W, Yau B, Lee S R, et al. Effects of ranibizumab and aflibercept on human Müller cells and photoreceptors under stress conditions[J]. International journal of molecular sciences, 2017, 18(3): 533.
[6] Lowe J, Araujo J, Yang J, et al. Ranibizumab inhibits multiple forms of biologically active vascular endothelial growth factor in vitro and in vivo[J]. Experimental eye research, 2007, 85(4): 425-430.
[7] Ichiyama Y, Matsumoto R, Obata S, et al. Assessment of mouse VEGF neutralization by ranibizumab and aflibercept[J]. PloS one, 2022, 17(12): e0278951.

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