

Rusfertide

Cat. No.:	HY-P10272
CAS No.:	1628323-80-7
Molecular Formula:	C ₁₁₄ H ₁₈₁ N ₂₇ O ₂₈ S ₂
Molecular Weight:	2441.95
Sequence:	{Asp(N-(3-methyl-1-oxobutyl))}-Thr-His-Phe-Pro-Cys-Ile-[Lys(γGlu-C16 acid)]-Phe-Glu-Pro-Arg-Ser-Lys-Gly-Cys-Lys-NH ₂ (disulfide bridge: Cys6-Cys16)
Sequence Shortening:	{Asp(N-(3-methyl-1-oxobutyl))}-THFPCI-[Lys(γGlu-C16 acid)]-FEPRSKGCK-NH ₂ (disulfide bridge: Cys6-Cys16)
Target:	Ferroportin
Pathway:	Membrane Transporter/Ion Channel
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description	Rusfertide is a peptide mimetic of natural hepcidin, which targets and degrades ferroportin, reduces serum iron and transferrin-saturation, and thus regulates the production of red blood cells. Rusfertide ameliorates the polycythemia vera, β-thalassemia and hereditary hemochromatosis ^{[1][2]} .								
In Vivo	<p>Rusfertide limits the iron toxicity in red blood cells (RBCs) (1 mg/kg, s.c., once every two days, for 49 days) and transferrin-saturation (2.5 mg/kg, s.c., once every two days, for 2 weeks), improves oxygen carrying capacity of RBCs, attenuates the anemia and iron deposition in mice models for β-thalassemia and hereditary hemochromatosis^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table> <tr> <td>Animal Model:</td> <td>Hbb^{th3/+} mice model for β-thalassemia and hereditary hemochromatosis^[1]</td> </tr> <tr> <td>Dosage:</td> <td>1 and 2.5 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>s.c., once every two days, for 49 days (1 mg/kg); or for 2 weeks (2.5 mg/kg)</td> </tr> <tr> <td>Result:</td> <td>Improved the survival rate of RBCs in β-thalassemia model. Reduced transferrin-saturation and iron deposition.</td> </tr> </table>	Animal Model:	Hbb ^{th3/+} mice model for β-thalassemia and hereditary hemochromatosis ^[1]	Dosage:	1 and 2.5 mg/kg	Administration:	s.c., once every two days, for 49 days (1 mg/kg); or for 2 weeks (2.5 mg/kg)	Result:	Improved the survival rate of RBCs in β-thalassemia model. Reduced transferrin-saturation and iron deposition.
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REFERENCES

[1]. Taranath R, et al., Regulation of iron homeostasis by PTG-300 improves disease parameters in mouse models for beta-thalassemia and hereditary hemochromatosis[J]. Blood, 2019, 134: 3540.

[2]. Kremianskaya M, et al., PTG-300 eliminates the need for therapeutic phlebotomy in both low and high-risk polycythemia vera patients[J]. Blood, 2020, 136: 33-35.

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

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA