Vosoritide acetate

Cat. No.: HY-P3503A

Molecular Formula: $C_{176}H_{290}N_{56}O_{51}S_3.C_2H_4O_2$

Molecular Weight: 4162.78

Sequence: Pro-Gly-Gln-Glu-His-Pro-Asn-Ala-Arg-Lys-Tyr-Lys-Gly-Ala-Asn-Lys-Lys-Gly-Leu-Ser-Lys-Gly-Cys-Phe-Gly-Leu-Lys-Leu-Asp-Arg-Ile-Gly-Ser-Met-Ser-Gly-Leu-Gly-Cys (Disulfide bridge:Cys23-Cys39)

PGQEHPNARKYKGANKKGLSKGCFGLKLDRIGSMSGLGC (Disulfide bridge:Cys23-Cys39) (Acetate salt)

Sequence Shortening: PGQEHPNARKYKGANKKGLSKGCFGLKLDRIGSMSGLGC (Disulfide bridge:Cys23-Cys39)

Target: FGFR

Pathway: Protein Tyrosine Kinase/RTK

Storage: Sealed storage, away from moisture and light, under nitrogen

Powder -80°C 2 years

-20°C 1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light, under nitrogen)

SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 100 mg/mL (24.02 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	0.2402 mL	1.2011 mL	2.4022 mL
	5 mM	0.0480 mL	0.2402 mL	0.4804 mL
	10 mM	0.0240 mL	0.1201 mL	0.2402 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

Vosoritide (BMN 111) acetate is a natriuretic peptide receptor 2 (NPR2) agonist that acts on the proliferation and differentiation of chondrocytes to promote bone growth[1].

In Vitro

Vosoritide (0.1 μM; 1 h) acetate decreases NPR2 phosphorylation in chondrocytes[2].

Vosoritide (0.1 μM; 6 d) acetate improves chondrocyte differentiation and increases the proliferative growth plate area of cultured $Fgfr3^{Y367C/+}$ femurs[2].

Vosoritide (10 μM; overnight) acetate reduces ERK1/2 activation in ACH growth-plate chondrocytes[3].

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

Western Blot Analysis[2]

Cell Line:	Chondrocyte cultures
Concentration:	0.1 μM
Incubation Time:	1 hour
Result:	Led to reduction in NPR2 phosphorylation.

Western Blot Analysis[3]

Cell Line:	Chondrocyte
Concentration:	10 μM
Incubation Time:	Overnight
Result:	Prevented FGF-mediated increase in ERK1/2 phosphorylation.

In Vivo

Vosoritide (subcutaneous injection; 800 μg/kg; once daily; 20 d) acetate treatment leads to improvement in skeletal parameters in Fgfr3 gain-of-function mutation mouse[3].

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

Animal Model:	$Fgfr3^{Y367C/+}$ mice[3]
Dosage:	800 μg/kg
Administration:	Subcutaneous injection; 800 μg/kg; once daily; 20 days
Result:	Observed phenotypic changes including flattening of the skull, elongation of the snout, improvement of the anterior crossbite, larger paws and digits, and longer and straightened tibias and femurs.

REFERENCES

[1]. Leia C. Shuhaibar, et al. Phosphatase inhibition by LB-100 enhances BMN-111 stimulation of bone growth. JCI Insight. 2021 May 10;6(9):e141426.

[2]. Shuhaibar LC, et al. Phosphatase inhibition by LB-100 enhances BMN-111 stimulation of bone growth. JCI Insight. 2021 May 10;6(9):e141426.

[3]. Lorget F, et al. Evaluation of the therapeutic potential of a CNP analog in a Fgfr3 mouse model recapitulating achondroplasia. Am J Hum Genet. 2012 Dec 7;91(6):1108-14.

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