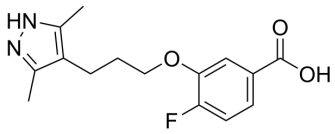
Acoramidis

Cat. No.:	HY-109165
CAS No.:	1446711-81-4
Molecular Formula:	$C_{15}H_{17}FN_2O_3$
Molecular Weight:	292.31
Target:	Transthyretin (TTR)
Pathway:	Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Acoramidis (AG10) is an orally active and selective kinetic stabilizer of WT and V122I-TTR (transthyretin). Acoramidis (AG10) is used in the study for transthyretin amyloidosis[1][2].

In Vitro

Acoramidis (AG10, 0.1-10 μM for TTR, 5 μM) stabilizes V122I- and WT-TTR equally well and also exceeds their efficacy to stabilize WT and mutant TTR in whole serum[1].

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Acoramidis (AG10) stimulates the mitochondrial QO2 in a concentration-dependent manner between 10 and 100 μM[3].

Acoramidis (AG10) has very minimal inhibition of two common off-targets in drug discovery, the potassium ion channel hERG (IC_{50} > 100 μM) and a number of cytochrome P450 isozymes (IC_{50} > 50 μM) (low toxicity)[1].

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

Western Blot Analysis[1].

Cell Line:	Human serum (TTR 5 μM).
Concentration:	0.1 and 10 μM.
Incubation Time:	72 h.
Result:	Was significantly more effective than tafamidis in stabilizing TTR. The concentration of AG10 to 10 μM resulted in stabilization of almost all of TTR in serum.

REFERENCES

[1]. Sravan C Penchala, et al. AG10 inhibits amyloidogenesis and cellular toxicity of the familial amyloid cardiomyopathy-associated V122I transthyretin. Proc Natl Acad Sci U S A. 2013 Jun 11;110(24):9992-7.

[2]. Jonathan C Fox, et al. First-in-Human Study of AG10, a Novel, Oral, Specific, Selective, and Potent Transthyretin Stabilizer for the Treatment of Transthyretin Amyloidosis: A Phase 1 Safety, Tolerability, Pharmacokinetic, and Pharmacodynamic Study in Healthy Adult Volunteers. Clin Pharmacol Drug Dev. 2020 Jan;9(1):115-129.

[3]. Stephen P Soltoff, et al. Evidence that tyrphostins AG10 and AG18 are mitochondrial uncouplers that alter phosphorylation-dependent cell signaling. J Biol Chem. 2004 Mar 19;279(12):10910-8.