SRI-37240

MedChemExpress

Cat. No.:	HY-150089		
CAS No.:	883956-47-0	6	
Molecular Formula:	$C_{24}H_{23}N_{3}O_{2}$		
Molecular Weight:	385.46		
Target:	CFTR		
Pathway:	Membrane	Transpor	ter/Ion Channel
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month

SOLVENT & SOLUBILITY

In Vitro

DMSO : 11.36 mg/mL (29.47 mM; ultrasonic and warming and heat to 60°C)

	Solvent Mass Concentration	1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM	2.5943 mL	12.9715 mL	25.9430 mL
	5 mM	0.5189 mL	2.5943 mL	5.1886 mL
	10 mM	0.2594 mL	1.2972 mL	2.5943 mL

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

BIOLOGICAL ACTIV	
DIOLOGICAL ACTIV	
Description	SRI-37240 is a potent premature termination codons (PTCs) inhibitor. SRI-37240 suppresses CFTR nonsense mutations. SRI- 37240 alters cellular translation termination at PTCs in HEK293T cells. SRI-37240 can also restore CFTR function in primary bronchial epithelial cells when combination with G418 ^[1] .
IC ₅₀ & Target	Premature termination codons, CFTR ^[1]
In Vitro	 SRI-37240 (1, 3, 10 and 30 μM; 48 h) induces concentration-dependent increases in CFTR-dependent (Forskolin-stimulated and sensitive to the inhibitor CFTR_{Inh}-172) chloride conductance^[1]. SRI-37240 (10 μM; 72 h) significantly increases the amount of full-length, fully glycosylated form of CFTR protein, and the unprocessed, immature form of full-length CFTR protein in 16HBEge cells when co-treated with G418 (100 μM)^[1]. SRI-37240 (10 μM; 24 h) alters cellular translation termination at PTCs in HEK293T cells, also increases global densities of ribosomes at normal stop codons without affecting densities of ribosomes in 3-UTRs^[1]. SRI-37240 (10 μM; 72 h) restores CFTR function in primary bronchial epithelial cells when combination with G418^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. Western Blot Analysis^[1]

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Cell Line:	CFTR-G542X 16HBEge
Concentration:	10 μΜ
Incubation Time:	24 h
Result:	Significantly increased the amount of Band C CFTR protein, which represents the full-length, fully glycosylated form of CFTR and Band B, which represents the unprocessed immature form of full-length CFTR protein when combined with G418 (100 μ M).

REFERENCES

[1]. Sharma J, et al. A small molecule that induces translational readthrough of CFTR nonsense mutations by eRF1 depletion. Nat Commun. 2021 Jul 16;12(1):4358.

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

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