Product Data Sheet

Product Name: SRI-37240
Cat. No.: GC67789

Chemical Properties

Cas. No.	883956-47-6		
Formula	$C_{24}H_{23}N_3O_2$	M.Wt	385.46
Solubility	DMSO : 11.36 mg/mL (29.47 mM; ultrasonic and warming and heat to 60°C)	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	SRI-37240		

Background

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].

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].

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Western Blot Analysis[1]

Cell Line:	*CFTR*-G542X 16HBEge
Concentration:	10 μM
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 μM).

[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.

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