

# **Product** Data Sheet

## LQZ-7F

Cat. No.:HY-122678CAS No.:354543-09-2Molecular Formula: $C_{14}H_7N_9O_3$ Molecular Weight:349.26

Target: Survivin; Apoptosis

Pathway: Apoptosis

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: 5 mg/mL (14.32 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent Mass Concentration	1 mg	5 mg	10 mg
	1 mM	2.8632 mL	14.3160 mL	28.6320 mL
	5 mM	0.5726 mL	2.8632 mL	5.7264 mL
	10 mM	0.2863 mL	1.4316 mL	2.8632 mL

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

### **BIOLOGICAL ACTIVITY**

Description

LQZ-7F, a survivin dimerization inhibitor, induces spontaneous apoptosis and synergizes with Docetaxel in prostate cancer cells. LQZ-7F dose-dependently inhibits survival of both PC-3 and C4-2 cells with IC<sub>50</sub>s of 2.99 and 2.47  $\mu$ M, respectively<sup>[1]</sup>.

In Vitro

LQZ-7F could be hudrolyzed under acidic conditions  $^{[1]}$ .

LQZ-7F (2.5  $\mu$ M, 72 hours) displays cytotoxicity towards human cancer cells (PC-3, C4-2) with the IC<sub>50</sub>s of 2.99  $\mu$ M and 2.74  $\mu$ M, respectively<sup>[1]</sup>.

LQZ-7F effectively inhibits survival of all cancer cell lines with IC<sub>50</sub> values ranging between 0.4 and 4.4 mM<sup>[2]</sup>.

LQZ-7F (2  $\mu$ M, 24 hours) disrupts microtubule structure and cause mitotic arrest in P3 cells [2].

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

Apoptosis Analysis<sup>[1]</sup>

Cell Line:	PC3, HL-60 cells
Concentration:	0, 5, 10 μΜ

Incubation Time:		
Result:	Caused 54% to 69% apoptosis for PC3 and 66% to 98% apoptosis for HL-60 cells.	
Cell Cytotoxicity Assay <sup>[1</sup>		
Cell Line:	PC-3, DU145	
Concentration:	10, 20 μΜ	
Incubation Time:	72 hours	
Result:	Showed that the IC <sub>50</sub> in human PC3 and DU145 cells was approximately 25 $\mu$ M.	

#### In Vivo

 $\label{eq:LQZ-7F} \ \text{LQZ-7F (i.p. injection; 25 mg/kg once every 3 days; 24 days) inhibits growth of xenograft tumor and may be due to its induction of surviving degradation in vivo $^{[1]}$.}$ 

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Animal Model:	4- to 6-week-old male NSG (NOD/SCID/IL-2Rg null) mice <sup>[2]</sup> .	
Dosage:	25 mg/kg	
Administration:	24 days	
Result:	Inhibited growth of xenograft tumors by inhibiting surviving.	

#### **REFERENCES**

[1]. Robert Peery, et al. A novel survivin dimerization inhibitor without a labile hydrazone linker induces spontaneous apoptosis and synergizes with docetaxel in prostate cancer cells. Bioorg Med Chem. 2022 Jul 1;65:116761.

[2]. Qi Jing, et al. Effective Targeting of the Survivin Dimerization Interface with Small-Molecule Inhibitors. Cancer Research. 2016 Jan. 76(2):453-462.

 $\label{lem:caution:Product} \textbf{Caution: Product has not been fully validated for medical applications. For research use only.}$ 

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