## PluriSIn 1

Cat. No.:	HY-15700		
CAS No.:	91396-88-2		
Molecular Formula:	C <sub>12</sub> H <sub>11</sub> N <sub>3</sub> O		
Molecular Weight:	213.24		
Target:	Stearoyl-CoA Desaturase (SCD); Apoptosis		
Pathway:	Metabolic Enzyme/Protease; Apoptosis		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year

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### SOLVENT & SOLUBILITY

	0, 1	DMSO : ≥ 100 mg/mL (468.96 mM) * "≥" means soluble, but saturation unknown.						
	Solvent Mass Concentration	1 mg	5 mg	10 mg				
	Preparing Stock Solutions	1 mM	4.6896 mL	23.4478 mL	46.8955 mL			
		5 mM	0.9379 mL	4.6896 mL	9.3791 mL			
		10 mM	0.4690 mL	2.3448 mL	4.6896 mL			
	Please refer to the solubility information to select the appropriate solvent.							
In Vivo	Solubility: ≥ 2.5 mg	ne by one: 10% DMSO >> 40% PEG ;/mL (11.72 mM); Clear solution ne by one: 10% DMSO >> 90% (20						
	Solubility: ≥ 2.5 mg	Solubility: ≥ 2.5 mg/mL (11.72 mM); Clear solution						

BIOLOGICAL ACTIVITY				
Description	PluriSIn 1 (NSC 14613) is an inhibitor of stearoyl-coA desaturase (SCD), and is a pluripotent cell-specific inhibitor.			
IC <sub>50</sub> & Target	SCD <sup>[1]</sup>			
In Vitro	PluriSIn 1, a small-molecule inhibitor of stearoyl-coA desaturase (SCD), on induced pluripotent stem cells (iPS)-derived cardiomyocytes (CM). PluriSIn 1 treatment significantly decreases the mRNA and protein level of Nanog, a marker for both cell pluripotency and tumor progression; importantly, we provide evidence that PluriSIn 1 treatment at 20 μM for 1 day significantly induces the apoptosis of Nanog-positive iPS derivates (iPSD). In addition, PluriSIn 1 treatment at 20 μM for 4 days diminished Nanog-positive stem cells in cultured iPSD while not increasing apoptosis of iPS-derived CM. To investigate			

# Product Data Sheet

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`ŅH HN whether PluriSIn 1 treatment prevents tumorigenicity of iPSD after cell transplantation, we intramyocardially injected PluriSIn 1- or DMSO-treated iPSD in a mouse model of myocardial infarction (MI). DMSO-treated iPSD readily formed Nanogexpressing tumors 2 weeks after injection, which is prevented by treatment with PluriSIn 1. Moreover, treatment with PluriSIn 1 does not change the expression of cTnI,  $\alpha$ -MHC, or MLC-2v, markers of cardiac differentiation (P>0.05, n=4). Importantly, PluriSIn 1-treated iPS-derived CM exhibits the ability to engraft and survive in the infarcted myocardium<sup>[1]</sup>. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

#### PROTOCOL

#### Cell Assay<sup>[1]</sup>

The differentiation of iPS cells to cardiomyocytes (CM) is induced by embryoid body (EB) formation. When iPS cells reached 70% confluency in 10-cm dishes, cells are digested using 0.25% trypsin/EDTA. Cell pellets are re-suspended in differentiation medium (DMEM with 20% FBS and 10 ng/mL BMP4) to a final concentration of 200,000 cells/mL. Cell suspensions are added to 6-well plates with Ulta-Low Attachment surfaces for 4 d to initiate EB formation. On day 5, EBs are cultured on 0.1% gelatin-coated dishes for 14 d using CF culture medium for the outgrowth of cardiac structures. At this stage, iPS cells undergoing EB formation are termed iPS derivates (iPSD)<sup>[1]</sup>.

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

#### **CUSTOMER VALIDATION**

- Mol Cell. 2019 Mar 7;73(5):1001-1014.e8.
- Sci China Life Sci. 2021 May 27;1-21.
- Int J Biol Macromol. 2023 Dec 4:128583.

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#### REFERENCES

[1]. Zhang L, et al. Inhibition of stearoyl-coA desaturase selectively eliminates tumorigenic Nanog-positive cells: improving the safety of iPS cell transplantation to myocardium. Cell Cycle. 2014;13(5):762-71.

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

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA