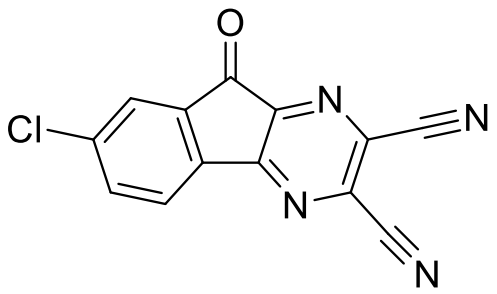
Product data sheet

MedKoo Cat#: 407460 Name: HBX-41108 CAS: 924296-39-9 Chemical Formula: $C_{13}H_3ClN_4O$ Exact Mass: 265.9995 Molecular Weight: 266.644	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
HBX 41108 is an inhibitor of ubiquitin-specific protease (USP) 7. HBX 41108 inhibits USP7 deubiquitinating activity with an IC(50) in the submicromolar range. HBX 41,108 was shown to affect USP7-mediated p53 deubiquitination in vitro and in cells. As RNA interference-mediated USP7 silencing in cancer cells, HBX 41,108 treatment stabilized p53, activated the transcription of a p53 target gene without inducing genotoxic stress, and inhibited cancer cell growth. HBX 41,108 induced p53-dependent apoptosis as shown in p53 wild-type and null isogenic cancer cell lines.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMF	25.0	93.76
DMSO	29.83	111.87
DMSO:PBS (pH 7.2) (1:40)	0.2	0.75

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.75 mL	18.75 mL	37.5 mL
5 mM	0.75 mL	3.75 mL	7.5 mL
10 mM	0.38 mL	1.88 mL	3.75 mL
50 mM	0.08 mL	0.38 mL	0.75 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Li X, Wang T, Tao Y, Wang X, Li L, Liu J. Inhibition of USP7 suppresses advanced glycation end-induced cell cycle arrest and senescence of human umbilical vein endothelial cells through ubiquitination of p53. Acta Biochim Biophys Sin (Shanghai). 2022 Mar 25;54(3):311-320. doi: 10.3724/abbs.2022003. PMID: 35538032.

In vivo study

1. Li X, Wang T, Tao Y, Wang X, Li L, Liu J. Inhibition of USP7 suppresses advanced glycation end-induced cell cycle arrest and senescence of human umbilical vein endothelial cells through ubiquitination of p53. Acta Biochim Biophys Sin (Shanghai). 2022 Mar 25;54(3):311-320. doi: 10.3724/abbs.2022003. PMID: 35538032.

7. Bioactivity
Biological target:

Product data sheet

HBX 41108 is an inhibitor of ubiquitin-specific protease (USP) 7 activity (IC_{50} = 424 nM). Also inhibits USP7-mediated p53 deubiquitination (IC_{50} = 0.8 μM).

In vitro activity

Moreover, the proportion of cells in the G0/G1 phase was significantly increased by AGEs, which was decreased by USP7 knockdown or HBX 41108, whereas that of cells in the S phase was significantly decreased by AGEs, which was increased by USP7 knockdown or HBX 41108 (Figure 2D,E). In addition, AGEs markedly increased the level of cell senescence, whereas the inhibition of USP7 by shUSP7 or HBX 41108 could reverse this effect (Figure 2F). p53 and p21 are markers of cell senescence. Western blot analysis revealed that the expressions of p53 and p21 were elevated by AGEs and decreased by shUSP7 and HBX 41108 (Figure 2G,H). These results indicated that inhibition of USP7 expression and its deubiquitinating activity could reduce cell cycle arrest and cell senescence in HUVECs.

Reference: Acta Biochim Biophys Sin (Shanghai). 2022 Mar 25;54(3):311-320. https://pubmed.ncbi.nlm.nih.gov/35538032/

In vivo activity

Treatment of diabetic rats with HBX 41108 decreased blood glucose levels on 14 post-injury (Figure 5A). STZ (streptozotocin) significantly reduced the wound healing rate, whereas the application of HBX 41108 could rescue the STZ-mediated effects (Figure 5B,C). HE staining of tissue sections showed that epithelial tissue was newly formed in wounds treated with HBX 41108 on day 7 post-injury (Figure 5D). Meanwhile, on day 7 and day 14, the expressions of USP7, p53, and p21 were found to be increased by STZ and decreased by the application of HBX 41108 (Figure 5E). These results showed that HBX 41108 could facilitate wound healing in diabetic rats through inhibiting USP7 deubiquitinating activity.

Reference: Acta Biochim Biophys Sin (Shanghai). 2022 Mar 25;54(3):311-320. https://pubmed.ncbi.nlm.nih.gov/35538032/