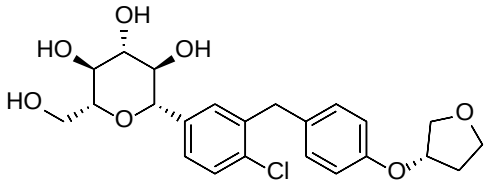
Product data sheet

MedKoo Cat#: 314251 Name: Empagliflozin CAS#: 864070-44-0 Chemical Formula: $C_{23}H_{27}ClO_7$ Exact Mass: 450.1445 Molecular Weight: 450.91	
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:

Empagliflozin, also known as BI10773 (trade name Jardiance), is drug approved for the treatment of type 2 diabetes in adults in 2014. It was developed by Boehringer Ingelheim and Eli Lilly and Company. Empagliflozin is an inhibitor of the sodium glucose co-transporter-2 (SGLT-2), and causes sugar in the blood to be absorbed by the kidneys and eliminated in urine. Empagliflozin is an inhibitor of the sodium glucose co-transporter-2 (SGLT-2), which is found almost exclusively in the proximal tubules of nephronic components in the kidneys. SGLT-2 accounts for about 90 percent of glucose reabsorption into the blood.

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
DMSO	56.67	125.68
DMF	30.0	66.53
Ethanol	30.0	66.53
Ethanol:PBS (pH 7.2) (1:1)	0.50	1.11

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.22 mL	11.09 mL	22.18 mL
5 mM	0.44 mL	2.22 mL	4.44 mL
10 mM	0.22 mL	1.11 mL	2.22 mL
50 mM	0.04 mL	0.22 mL	0.44 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. Lee YH, Kim SH, Kang JM, Heo JH, Kim DJ, Park SH, Sung M, Kim J, Oh J, Yang DH, Lee SH, Lee SY. Empagliflozin attenuates diabetic tubulopathy by improving mitochondrial fragmentation and autophagy. Am J Physiol Renal Physiol. 2019 Oct 1;317(4):F767-F780. doi: 10.1152/ajprenal.00565.2018. Epub 2019 Aug 7. PMID: 31390268.
2. Ng KM, Lau YM, Dhandhania V, Cai ZJ, Lee YK, Lai WH, Tse HF, Siu CW. Empagliflozin Ammeliorates High Glucose Induced-Cardiac Dysfuntion in Human iPSC-Derived Cardiomyocytes. Sci Rep. 2018 Oct 5;8(1):14872. doi: 10.1038/s41598-018-33293-2. PMID: 30291295; PMCID: PMC6173708.

In vivo study

1. Li C, Zhang J, Xue M, Li X, Han F, Liu X, Xu L, Lu Y, Cheng Y, Li T, Yu X, Sun B, Chen L. SGLT2 inhibition with empagliflozin attenuates myocardial oxidative stress and fibrosis in diabetic mice heart. Cardiovasc Diabetol. 2019 Feb 2;18(1):15. doi: 10.1186/s12933-019-0816-2. PMID: 30710997; PMCID: PMC6359811.

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2. Lee YH, Kim SH, Kang JM, Heo JH, Kim DJ, Park SH, Sung M, Kim J, Oh J, Yang DH, Lee SH, Lee SY. Empagliflozin attenuates diabetic tubulopathy by improving mitochondrial fragmentation and autophagy. Am J Physiol Renal Physiol. 2019 Oct 1;317(4):F767-F780. doi: 10.1152/ajprenal.00565.2018. Epub 2019 Aug 7. PMID: 31390268.

7. Bioactivity

Biological target: Empagliflozin (BI 107730) is a sodium glucose cotransporter-2 (SGLT-2) inhibitor with an IC50 of 3.1 nM.

In vitro activity

The effects of empagliflozin on mitochondrial quality control and autophagy in renal tubular cells in a diabetic environment were examined in vitro. Human renal proximal tubular cells (hRPTCs) were incubated under high-glucose conditions. Improvements in mitochondrial biogenesis and balanced fusion-fission protein expression were noted in hRPTCs after treatment with empagliflozin in high-glucose media. Empagliflozin also increased autophagic activities in renal tubular cells in the high-glucose environment, which was accompanied with mammalian target of rapamycin inhibition. Moreover, reduced mitochondrial reactive oxygen species production and decreased apoptotic and fibrotic protein expression were observed in hRPTCs after treatment with empagliflozin, even in the hyperglycemic circumstance. Importantly, empagliflozin restored AMP-activated protein kinase-α phosphorylation and normalized levels of AMP-to-ATP ratios in hRPTCs subjected to a high-glucose environment, which suggests the way that empagliflozin is involved in mitochondrial quality control.

Reference: Am J Physiol Renal Physiol. 2019 Oct 1;317(4):F767-F780. https://journals.physiology.org/doi/full/10.1152/ajprenal.00565.2018?rfr_dat=cr_pub++0pubmed&url_ver=Z39.88-2003&rfr_id=ori%3Arid%3Acrossref.org

In vivo activity

To investigate the effect of empagliflozin on myocardium injury and the potential mechanism in type 2 diabetic KK-Ay mice, thirty diabetic KK-Ay mice were administered empagliflozin (10 mg/kg/day) by oral gavage daily for 8 weeks. Results showed that empagliflozin improved diabetic myocardial structure and function, decreased myocardial oxidative stress and ameliorated myocardial fibrosis. Further study indicated that empagliflozin suppressed oxidative stress and fibrosis through inhibition of the transforming growth factor β/Smad pathway and activation of Nrf2/ARE signaling.

Reference: Cardiovasc Diabetol. 2019 Feb 2;18(1):15. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6359811/