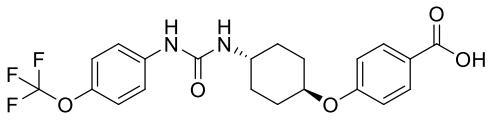
Product data sheet

MedKoo Cat#: 555121 Name: UC-1728 CAS#: 948304-40-3 Chemical Formula: $C_{21}H_{21}F_3N_2O_5$ Exact Mass: 438.1403 Molecular Weight: 438.4032	
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:
UC-1728, also known as t-TUCB, is a soluble epoxide hydrolase inhibitor. t-TUCB alleviates liver fibrosis and portal pressure in carbon tetrachloride-induced cirrhosis in rats. UC-1728 minimizes ischemia-reperfusion-induced cardiac damage in normal, hypertensive, and diabetic rats.

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	43.84	100.0

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.28 mL	11.41 mL	22.81 mL
5 mM	0.46 mL	2.28 mL	4.56 mL
10 mM	0.23 mL	1.14 mL	2.28 mL
50 mM	0.05 mL	0.23 mL	0.46 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. Overby H, Yang Y, Xu X, Graham K, Hildreth K, Choi S, Wan D, Morisseau C, Zeldin DC, Hammock BD, Wang S, Bettaieb A, Zhao L. Soluble Epoxide Hydrolase Inhibition by t-TUCB Promotes Brown Adipogenesis and Reduces Serum Triglycerides in Diet-Induced Obesity. Int J Mol Sci. 2020 Sep 24;21(19):7039. doi: 10.3390/ijms21197039. PMID: 32987880; PMCID: PMC7582898.
2. Kodani SD, Bhakta S, Hwang SH, Pakhomova S, Newcomer ME, Morisseau C, Hammock BD. Identification and optimization of soluble epoxide hydrolase inhibitors with dual potency towards fatty acid amide hydrolase. Bioorg Med Chem Lett. 2018 Feb 15;28(4):762-768. doi: 10.1016/j.bmcl.2018.01.003. Epub 2018 Jan 4. PMID: 29366648; PMCID: PMC5837813.

In vivo study

1. Minaz N, Razdan R, Hammock BD, Mujwar S, Goswami SK. Impact of diabetes on male sexual function in streptozotocin-induced diabetic rats: Protective role of soluble epoxide hydrolase inhibitor. Biomed Pharmacother. 2019 Jul;115:108897. doi: 10.1016/j.biopha.2019.108897. Epub 2019 May 15. PMID: 31102913; PMCID: PMC6893866.
2. Bastan I, Ge XN, Dileepan M, Greenberg YG, Guedes AG, Hwang SH, Hammock BD, Washabau RJ, Rao SP, Sriramarao P. Inhibition of soluble epoxide hydrolase attenuates eosinophil recruitment and food allergen-induced gastrointestinal inflammation. J Leukoc Biol. 2018 Jul;104(1):109-122. doi: 10.1002/JLB.3MA1017-423R. Epub 2018 Jan 17. PMID: 29345370; PMCID: PMC6020675.

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7. Bioactivity

Biological target:

UC-1728 (t-TUCB) is an epoxide hydrolase (sEH) Inhibitor (IC_{50} = 0.9 nM).

In vitro activity

Based on the sEH expression results, it was hypothesized that sEH might play a role in brown adipogenesis in vitro, and sEH inhibition may modulate the process. t-TUCB (Figure 3A), a selective inhibitor of sEH, was chosen for the studies. t-TUCB significantly increased oil red O (ORO) stained differentiated brown adipocyte morphology and lipid accumulation, as assessed by the ORO absorbance (Figure 3B). t-TUCB significantly increased mRNA levels of brown marker genes Ucp1, Pgc-1α, and cell death-inducing DFFA like effector A (Cidea) (Figure 3C). Pparγ mRNA level was significantly increased by t-TUCB only at 20 μM (Figure 3C). Protein expression of UCP1 and PGC-1α were also increased by t-TUCB (Figure 3D). Interestingly, t-TUCB also increased Ephx2 mRNA and sEH protein expression in a dose-dependent manner (Supplemental Figure S1), suggesting that there might be a feedback mechanism between t-TUCB and Ephx2 gene expression.

Reference: Int J Mol Sci. 2020 Oct; 21(19): 7039. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7582898/

In vivo activity

Treatment with t-TUCB significantly inhibited mucus secretion with levels similar to that noted in control mice (Fig. 4, A and B). Barrier function of the intestinal epithelium, which is tightly regulated by epithelial junction proteins, plays a crucial role in preventing passage of harmful agents, including dietary antigens, from the lumen of the gut into the mucosal tissues and circulatory system. This study examined expression of epithelial occludin, ZO-1 and E-cadherin in a model of SPI-induced GI allergy by immunofluorescence staining of jejunal sections (Fig. 4, C – E). Consistent with the phenotype associated with GI inflammation, SPI challenge resulted in a marked reduction in expression of epithelial occludin, ZO-1 and E-cadherin relative to control mice (Fig. 4, C and D, middle and top panels, respectively, Fig. 4, E, middle and left panels, respectively). In SPI-challenged mice treated with t-TUCB at 3 mg/kg, loss of occludin and E-cadherin expression was substantially prevented (Fig. 4, C, bottom panels, and E, right panels), while recovery of ZO-1 expression was less striking (Fig. 4, D, bottom panels). Overall, these studies suggest that sEH induced by food allergens such as soy proteins promotes GI allergic responses and inflammation and that inhibition of sEH can attenuate SPI-induced allergic responses.

Reference: J Leukoc Biol. 2018 Jul; 104(1): 109–122. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6020675/