# **Product data sheet**



MedKoo Cat#: 525808				
Name: Oxfenicine				
CAS#: 32462-30-9				
Chemical Formula: C <sub>8</sub> H <sub>9</sub> NO <sub>3</sub>				
Exact Mass: 167.0582				
Molecular Weight: 167.16				
Product supplied as:	Powder			
Purity (by HPLC):	$\geq$ 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:

Oxfenicine, also known as UK-25842, is a carnitine palmitoyltransferase I (CPT-1) inhibitor involved in fatty acid metabolism in the heart. In animal studies oxfenicine acts as a cardioprotective agent during ischemia. Oxfenicine produces changes in myocardial substrate utilisation and is attributed to the protection of ischemic stressed myocardium by shifting the cardiac metabolism with reduction of FFA (Free Fatty Acid) utilisation. This may be favourable in circumstances with limited oxygen supply.

#### 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	4.55	27.22
H2O	1.00	5.98

#### 4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	5.98 mL	29.91 mL	59.82 mL
5 mM	1.20 mL	5.98 mL	11.96 mL
10 mM	0.60 mL	2.99 mL	5.98 mL
50 mM	0.12 mL	0.60 mL	1.20 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. Sepa-Kishi DM, Wu MV, Uthayakumar A, Mohasses A, Ceddia RB. Antilipolytic and antilipogenic effects of the CPT-1b inhibitor oxfenicine in the white adipose tissue of rats. Am J Physiol Regul Integr Comp Physiol. 2016 Oct 1;311(4):R779-R787. doi: 10.1152/ajpregu.00243.2016. Epub 2016 Aug 24. PMID: 27558315; PMCID: PMC5142162.

In vivo study

1. Sepa-Kishi DM, Wu MV, Uthayakumar A, Mohasses A, Ceddia RB. Antilipolytic and antilipogenic effects of the CPT-1b inhibitor oxfenicine in the white adipose tissue of rats. Am J Physiol Regul Integr Comp Physiol. 2016 Oct 1;311(4):R779-R787. doi: 10.1152/ajpregu.00243.2016. Epub 2016 Aug 24. PMID: 27558315; PMCID: PMC5142162.

2. Drake-Holland AJ, Passingham JE. The effect of Oxfenicine on cardiac carbohydrate metabolism in intact dogs. Basic Res Cardiol. 1983 Jan-Feb;78(1):19-27. doi: 10.1007/BF01923190. PMID: 6847579.

#### 7. Bioactivity

### Biological target:

Oxfenicine (L-p-Hydroxyphenylglycine) is a carnitine palmitoyltransferase-1 inhibitor that inhibits the oxidation of fatty acid in heart. Oxfenicine protects heart from necrotic tissue damage during ischaemia

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### In vitro activity

This study is the first to examine the in vitro effects of oxfenicine-induced CPT-1b inhibition on adipocyte metabolism In isolated adipocytes from both the Epid and SC Ing fat depots treated with 1 mM oxfenicine, palmitate oxidation was significantly reduced by 50% compared with control cells (Fig. 6, A and B). Isoproterenol-stimulated lipolysis was significantly decreased by 20% and 12% in Epid adipocytes following treatment with 100  $\mu$ M and 1 mM oxfenicine, respectively (Fig. 6C). Similarly, in SC Ing adipocytes treatment with 100  $\mu$ M and 1 mM of oxfenicine decreased stimulated lipolysis by 8% and 18%, respectively (Fig. 6D). There was no effect of oxfenicine on basal rates of lipolysis in Epid and SC Ing adipocytes (Fig. 6, C and D). Basal and insulin-stimulated glucose incorporation into lipids, the measure for lipogenesis, was reduced by 39% and 31%, respectively, in Epid adipocytes following treatment with 1 mM oxfenicine (Fig. 7A). In adipocytes from the SC Ing fat depot, the incorporation of glucose into lipids was also reduced by 41% under insulin-stimulated conditions, following 1 mM oxfenicine treatment (Fig. 7B). This suggests that fat cells adjusted their metabolism to compensate for the increased circulating NEFAs seen with inhibition of  $\beta$ -oxidation.

Reference: Am J Physiol Regul Integr Comp Physiol. 2016 Oct 1; 311(4): R779–R787. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5142162/

#### In vivo activity

This study investigated whether oxfenicine-induced inhibition of FA oxidation affected adiposity and WAT metabolism in rats fed either low (LF) or high-fat (HF) diets. Following 8 wk of dietary intervention, male Sprague-Dawley rats were given a daily intraperitoneal injection of oxfenicine (150 mg/kg body wt) or vehicle (PBS) for 3 wk. To examine the in vivo effects of the oxfenicine treatment, animals were placed in the CLAMS for 24 h following oxfenicine treatment and their  $\dot{V}o2$ , ambulatory activity, and respiratory exchange ratio (RER) were measured. Treatment with oxfenicine had no effect on the RER of animals fed the LF diet, whereas those fed a HF diet had significantly higher RER values, particularly during the dark cycle (Fig. 1, C and D). This is indicative of the effectiveness of oxfenicine at inhibiting fatty acid  $\beta$ -oxidation, resulting in an increased reliance on carbohydrate oxidation. This was more pronounced during the dark cycle (1900 to 0700), when the animals were the most active and ate the most food (0.830 ± 0.00271  $\dot{V}co2/\dot{V}o2$  vs. 0.796 ± 0.00244,  $\dot{V}co2/\dot{V}o2$ , Fig. 1, C and D). Fasting plasma NEFAs were also 1.45- and 1.46-fold higher in the LF- and HF-fed animals, respectively, following 3 wk of oxfenicine treatment (Fig. 1E). An increase in circulating fatty acids provides further evidence of the effectiveness of the oxfenicine treatment in reducing fatty acid oxidation. This is the first study to examine the adaptive changes in adipose tissue metabolism that occur as a result of suppressing fatty acid oxidation through CPT-1b inhibition in vivo and provides novel additional information regarding this potential obesity and T2D therapy.

Reference: Am J Physiol Regul Integr Comp Physiol. 2016 Oct 1; 311(4): R779–R787. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5142162/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.