

# Product data sheet



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| MedKoo Cat#: 597971<br>Name: TT2-32 acetate<br>CAS#: TT2-32 acetate<br>Chemical Formula: C <sub>47</sub> H <sub>62</sub> N <sub>10</sub> O <sub>11</sub> S <sub>2</sub><br>Exact Mass: 946.383<br>Molecular Weight: 1007.192 |   |
| Product supplied as:   | Powder  |
| Purity (by HPLC):  | ≥ 98%   |
| Shipping conditions  | Ambient temperature   |
| Storage conditions:  | Powder: -20°C 3 years; 4°C 2 years.<br>In solvent: -80°C 3 months; -20°C 2 weeks. |

## 1. Product description:

TT2-32, also known as TLN-232 and CAP232, is a somatostatin structural derivative with antitumor activity. TT-232 inhibited tyrosine kinase activity of tumor cell lines and this inhibition correlated well with the inhibition of cell proliferation of a large number of cancer cell lines in vitro and reduces the size of different tumors in animal models in vivo. The antitumor efficacy of TT-232 has been found to be associated with the induction of apoptosis in tumor cells, resulting in highly selective elimination of tumor tissue. TT-232 was found to be devoid of GH release inhibitory activity but to possess strong antitumor effects. It binds with a high affinity to SSTR1 and SSTR4. This compound was also found to inhibit inflammation in a number of experimental models.

## 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    | TBD             | TBD          |

## 4. Stock solution preparation table:

| Concentration / Solvent Volume / Mass | 1 mg    | 5 mg    | 10 mg   |
|---------------------------------------|---------|---------|---------|
| 1 mM                                  | 0.99 mL | 4.96 mL | 9.93 mL |
| 5 mM                                  | 0.20 mL | 0.99 mL | 1.99 mL |
| 10 mM                                 | 0.10 mL | 0.50 mL | 0.99 mL |
| 50 mM                                 | 0.02 mL | 0.10 mL | 0.20 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. Vántus T, Kéri G, Krivickiene Z, Valius M, Steták A, Keppens S, Csermely P, Bauer PI, Bökönyi G, Declercq W, Vandennebeele P, Merlevede W, Vandennebeele JR. The somatostatin analogue TT-232 induces apoptosis in A431 cells: sustained activation of stress-activated kinases and inhibition of signalling to extracellular signal-regulated kinases. *Cell Signal*. 2001 Oct;13(10):717-25. doi: 10.1016/s0898-6568(01)00194-2. PMID: 11602182.

### In vivo study

1. Elekes K, Helyes Z, Kereskai L, Sándor K, Pintér E, Pozsgai G, Tékus V, Bánvölgyi A, Németh J, Szuts T, Kéri G, Szolcsányi J. Inhibitory effects of synthetic somatostatin receptor subtype 4 agonists on acute and chronic airway inflammation and hyperactivity in the mouse. *Eur J Pharmacol*. 2008 Jan 14;578(2-3):313-22. doi: 10.1016/j.ejphar.2007.09.033. Epub 2007 Oct 5. PMID: 17961545.

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## 7. Bioactivity

### Biological target:

TT 232 is a peptide agonist for sst1/sst4 somatostatin receptors.

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### In vitro activity

The present report describes TT-232-induced signalling events in A431 cells, where a 4-h preincubation with the peptide irreversibly induced a cell death program. Early intracellular signals of TT-232 include a transient two-fold activation of the extracellular signal-regulated kinase (ERK2) and a strong and sustained activation of the stress-activated protein kinases c-Jun NH(2)-terminal kinase (JNK)/SAPK and p38MAPK. Blocking the signalling to ERK or p38MAPK activation had no effect on the TT-232-induced cell killing. At the commitment time for inducing cell death, TT-232 decreased EGFR-tyrosine phosphorylation and prevented epidermal growth factor (EGF)-induced events like cRaf-1 and ERK2 activation. Signalling to ERK activation by FCS, phorbol 12-myristate 13-acetate (PMA) and platelet-derived growth factor (PDGF) was similarly blocked. The data suggest that TT-232 triggers an apoptotic type of cell death, concomitant with a strong activation of JNK and a blockade of cellular ERK2 activation pathways.

Reference: Cell Signal. 2001 Oct;13(10):717-25. <https://pubmed.ncbi.nlm.nih.gov/11602182/>

### In vivo activity

The aim of this study was to examine the effects of TT-232, a heptapeptide sst(4)/sst(1) receptor agonist in airway inflammation models in the mouse. Acute pneumonitis was evoked by intranasal lipopolysaccharide 24 h before measurement. Chronic inflammation was induced by ovalbumin inhalation on days 28, 29 and 30 after i.p. sensitization on days 1 and 14. TT-232 induced an about 50% inhibitory action on bronchoconstriction after administration of a single 500 µg/kg i.p. dose. Inhalation of increasing concentrations (5.5–22 mM) of the muscarinic receptor agonist carbachol evoked a concentration-dependent bronchoconstriction shown by the Penh curves. TT-232 (500 µg/kg i.p.) significantly inhibited endotoxin-induced airway hyperreactivity after a single acute administration and after repeated injections. These results suggest that stable, somatostatin sst(4) receptor-selective agonists could be potential candidates for the development of a completely novel group of anti-inflammatory drugs for the treatment of airway inflammation and hyperresponsiveness.

Reference: Eur J Pharmacol. 2008 Jan 14;578(2-3):313-22. <https://pubmed.ncbi.nlm.nih.gov/17961545/>

*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.*