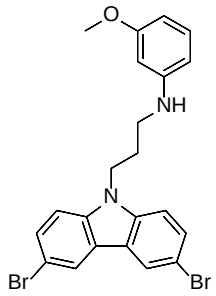
Product data sheet

MedKoo Cat#: 522438 Name: P7C3A20-analog CAS: unknown as of 10/13/2015 Chemical Formula: $C_{22}H_{20}Br_2N_2O$ Exact Mass: 485.9942 Molecular Weight: 488.223	
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:

P7C3A20-analog, as known as defluoro-P7C3-A20, is a structural analogue of P7C3-A20. Compared to P7C3-A20 structure, P7C3-A20 analog has no fluorine atom in the 1,3-diaminopropane-bridge. P7C3-A20 analog was synthesized by mistake during a process to make P7C3-A20. The bioactivity of P7C3-A20 analog is unknown. P7C3-A20 analog may be used for research to compare with P7C3-A20. P7C3-A20 is a proneurogenic, neuroprotective agent. P7C3-A20 displayed increased activity and an improved toxicity profile compared to P7C3. P7C3-A20 demonstrated greater proneurogenic efficacy than a wide spectrum of currently marketed antidepressant drugs. P7C3-A20 showed neuroprotective properties in rodent models of Parkinson's disease, amyotrophic lateral sclerosis, traumatic brain injury and age-related cognitive decline.

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
TBD	TBD	TBD

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.05 mL	10.24 mL	20.48 mL
5 mM	0.41 mL	2.05 mL	4.10 mL
10 mM	0.21 mL	1.02 mL	2.05 mL
50 mM	0.04 mL	0.21 mL	0.41 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. Wang SN, Xu TY, Wang X, Guan YF, Zhang SL, Wang P, Miao CY. Neuroprotective Efficacy of an Aminopropyl Carbazole Derivative P7C3-A20 in Ischemic Stroke. CNS Neurosci Ther. 2016 Sep;22(9):782-8. doi: 10.1111/cns.12576. Epub 2016 Jun 23. PMID: 27333812; PMCID: PMC6492790.

In vivo study

1. Vázquez-Rosa E, Shin MK, Dhar M, Chaubey K, Cintrón-Pérez CJ, Tang X, Liao X, Miller E, Koh Y, Barker S, Franke K, Crosby DR, Schroeder R, Emery J, Yin TC, Fujioka H, Reynolds JD, Harper MM, Jain MK, Pieper AA. P7C3-A20 treatment one year after TBI in mice repairs the blood-brain barrier, arrests chronic neurodegeneration, and restores cognition. Proc Natl Acad Sci U S A. 2020 Nov 3;117(44):27667-27675. doi: 10.1073/pnas.2010430117. Epub 2020 Oct 21. PMID: 33087571; PMCID: PMC7959512.
2. Hill CS, Menon DK, Coleman MP. P7C3-A20 neuroprotection is independent of Wallerian degeneration in primary neuronal culture. Neuroreport. 2018 Dec 12;29(18):1544-1549. doi: 10.1097/WNR.0000000000001146. PMID: 30334859; PMCID: PMC6250284.

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7. Bioactivity

Biological target:

P7C3A20-analog, as known as defluoro-P7C3-A20, is a structural analogue of P7C3-A20.

In vitro activity

The aim of this study was to investigate the effect of a potential NAMPT activator, P7C3-A20, an aminopropyl carbazole derivative, on ischemic stroke. In vitro study, neuron protection effect of P7C3-A20 was investigated by co-incubation with primary neurons subjected to oxygen-glucose deprivation (OGD) or oxygen-glucose deprivation/reperfusion (OGD/R) injury.
In order to explore the role of P7C3 - A20 in cerebral ischemia, this study cultured primary cortical neurons and established neuronal OGD model to mimic in vitro ischemic stress. Primary cortical neurons were exposed to OGD condition for 1.5 and 12 h, respectively. This study found that neuronal injuries were growing worse following prolonged duration of OGD. Prolonged administration of P7C3-A20 may have positive effects for the recovery of chronic stroke. Furthermore, P7C3-A20 may benefit poststroke recovery because of its role in neurogenesis, given evidence that P7C3-A20 could rescue NPAS3$^{-/-}$-induced loss of hippocampal neurogenesis, and augment hippocampal neurogenesis.

CNS Neurosci Ther. 2016 Sep; 22(9): 782–788. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6492790/

In vivo activity

This laboratory model produces neurodegeneration and neurobehavioral deficits reminiscent of TBI in people. Mice were treated and analyzed beginning 1 y after a single injury, as outlined in Fig. 1A. Two-month-old mice were subjected to TBI or sham-TBI and housed under standard conditions for 1 y. Mice were then administered either vehicle (TBI-Veh, Sham-Veh) or P7C3-A20 (10 mg·kg−1·day−1 intraperitoneally [IP]; TBI-P7C3-A20) for 4 wk. Animals were then housed under standard conditions with no treatment for four additional months (Fig. 1A). At 19 mo of age (17 mo postinjury), TBI-Veh mice exhibited impaired learning (Fig. 1B) and memory (Fig. 1C) whereas TBI-P7C3-A20 mice again performed as well as Sham-Veh animals. P7C3-A20 directly protects brain microvascular endothelial cells in vivo in mice and in cultured human cells. Taken together, the results suggest that BBB deterioration may be a major contributor to chronic neurodegeneration after remote TBI and that its repair may halt this pathology.

Proc Natl Acad Sci U S A. 2020 Nov 3; 117(44): 27667–27675. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7959512/

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.