# Product data sheet

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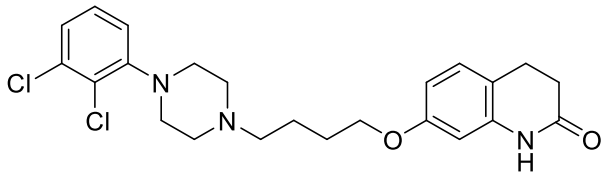
|---|---|
| MedKoo Cat#: 317201<br>Name: Aripiprazole<br>CAS#: 129722-12-9 (free base)<br>Chemical Formula: $C_{23}H_{27}Cl_2N_3O_2$<br>Exact Mass: 447.14803<br>Molecular Weight: 448.39 |  |
| Product supplied as: | Powder |
| Purity (by HPLC): | ≥ 98% |
| Shipping conditions | Ambient temperature |
| Storage conditions: | Powder: -20°C 3 years; 4°C 2 years. |
| | In solvent: -80°C 3 months; -20°C 2 weeks. |

**1. Product description:**
Aripiprazole, sold under the brand name Abilify among others, is an atypical antipsychotic. It is recommended and primarily used in the treatment of schizophrenia and bipolar disorder. Other uses include as an add-on treatment in major depressive disorder, tic disorders, and irritability associated with autism. Aripiprazole is a quinoline derivate and atypical anti-psychotic agent. Aripiprazole has partial agonistic activity at dopamine D2 receptors and serotonin 5-HT1A receptors, as well as potent antagonistic activity on serotonin 5-HT2A receptors.

**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 | 67.0 | 149.42 |
| DMF | 50.0 | 111.51 |

**4. Stock solution preparation table:**

| **Concentration / Solvent Volume / Mass** | **1 mg** | **5 mg** | **10 mg** |
|---|---|---|---|
| 1 mM | 2.23 mL | 11.15 | 22.30 mL |
| 5 mM | 0.45 mL | 2.23 mL | 4.46 mL |
| 10 mM | 0.22 mL | 1.12 mL | 2.23 mL |
| 50 mM | 0.04 mL | 0.22 mL | 0.45 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. Brust TF, Hayes MP, Roman DL, Watts VJ. New functional activity of aripiprazole revealed: Robust antagonism of D2 dopamine receptor-stimulated Gβγ signaling. Biochem Pharmacol. 2015 Jan 1;93(1):85-91. doi: 10.1016/j.bcp.2014.10.014. Epub 2014 Nov 7. PMID: 25449598; PMCID: PMC4276521.
2. Kato T, Mizoguchi Y, Monji A, Horikawa H, Suzuki SO, Seki Y, Iwaki T, Hashioka S, Kanba S. Inhibitory effects of aripiprazole on interferon-gamma-induced microglial activation via intracellular Ca2+ regulation in vitro. J Neurochem. 2008 Jul;106(2):815-25. doi: 10.1111/j.1471-4159.2008.05435.x. Epub 2008 Apr 19. PMID: 18429930.

In vivo study

1. Gil CH, Kim YR, Lee HJ, Jung DH, Shin HK, Choi BT. Aripiprazole exerts a neuroprotective effect in mouse focal cerebral ischemia. Exp Ther Med. 2018 Jan;15(1):745-750. doi: 10.3892/etm.2017.5443. Epub 2017 Nov 6. PMID: 29399080; PMCID: PMC5772374.

2. Sonnenschein SF, Gill KM, Grace AA. State-dependent effects of the D2 partial agonist aripiprazole on dopamine neuron activity in the MAM neurodevelopmental model of schizophrenia. Neuropsychopharmacology. 2019 Feb;44(3):572-580. doi: 10.1038/s41386-018-0219-1. Epub 2018 Sep 18. PMID: 30267014; PMCID: PMC6333840.

**7. Bioactivity**

Biological target:

Aripiprazole (OPC-14597) is a human 5-HT1A receptor partial agonist with a Ki of 4.2 nM.

In vitro activity

The 6-3 cells were pre-treated with DMSO (0.1%), aripiprazole (5, 10 and 20 μM) or D2 receptor full agonist, quinpirole (5, 10 and 20μM) for 12h, then the cells were treated with each drug and IFN-γ (50 U/mL) for 48h. Aripiprazole significantly inhibited the NO release dose-dependently in comparison with the positive control (DMSO + IFN-γ group). In order to confirm whether these effects are specific to IFN-γ–induced microglial activation or not, the effect of aripiprazole on LPS-induced microglial activation was measured. Aripiprazole significantly inhibited NO release by LPS-activated 6-3 microglia (Fig. 2b). These results suggest that the inhibitory effects of aripiprazole were not specific to IFN-γ receptor-mediated signaling. In addition, prepared rat primary microglial cells were also prepared in order to confirm the relevance of our results in these cells. Similar to what was observed in the 6-3 murine microglial cells, aripiprazole significantly inhibited NO release from LPS-activated rat primary microglia. Then. the 6-3 cells were pre-treated with DMSO (0.1%), aripiprazole (5, 10 and 20μM) or quinpirole (5, 10 and 20μM) for 12h, then the cells were treated with each drug and IFN-γ (50 U/mL) for 48h. Aripiprazole strongly inhibited the release of TNF-α dose-dependently. Therefore, the present results suggest that aripiprazole may ameliorate white matter disorders via inhibiting microglial activation in the brain of patients with schizophrenia.

Reference: J Neurochem. 2008 Jul;106(2):815-25. https://onlinelibrary.wiley.com/doi/full/10.1111/j.1471-4159.2008.05435.x

In vivo activity

Electrophysiological recordings were conducted from MAM rats and SAL rats, with each group receiving either VEH or ARI (aripiprazole ) (3 mg/kg or 10 mg/kg, p.o.). VEH-treated MAM rats (n = 6 rats, 63 neurons) exhibited the anticipated elevation in population activity with an average of 1.7 ± 0.1 cells/track compared to VEH-treated SAL rats (n = 8 rats, 61 neurons) which had an average of 1.1 ± 0.1 cells/track (Fig. 1a; 2-way ANOVA main effects: for MAM $F(2, 40) = 3.189$, $p = 0.010$; for ARI $F(2, 40) = 5.049$, $p = 0.011$; MAM-by-ARI interaction $F(2, 40) = 5.882$, $p = 0.005$; post hoc MAM control vs SAL control $p = 0.001$). Acute ARI treatment significantly reduced DA neuron population activity in MAM rats, both at 3 mg/kg (n = 6 rats, 37 neurons; post hoc MAM control vs MAM 3 mg/kg: $p = 0.007$) and 10 mg/kg (n = 10 rats, 63 neurons; post hoc MAM control vs MAM 10 mg/kg: $p = 0.001$) compared to VEH-treated MAM rats. Overall, this study demonstrates that ARI rapidly normalizes the hyperdopaminergic state observed in MAM rats without effect on DA neuron population activity in a normal system.

Reference: Neuropsychopharmacology. 2019 Feb; 44(3): 572–580. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6333840/