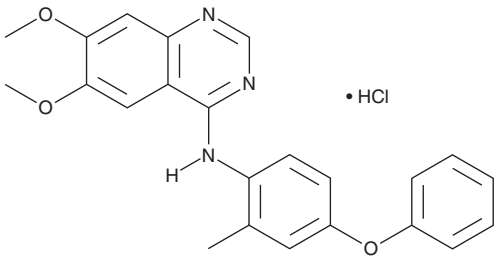
PRODUCT INFORMATION

APS-2-79 (hydrochloride)

Item No. 22137

CAS Registry No.:	2002381-31-7
Formal Name:	6,7-dimethoxy-N-(2-methyl-4-phenoxyphenyl)-4-quinazolinamine, monohydrochloride
MF:	$C_{23}H_{21}N_3O_3 \bullet HCl$
FW:	423.9
Purity:	≥95%
UV/Vis.:	λ_{max}: 220, 252, 342 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

APS-2-79 (hydrochloride) is supplied as a crystalline solid. A stock solution may be made by dissolving the APS-2-79 (hydrochloride) in the solvent of choice, which should be purged with an inert gas. APS-2-79 (hydrochloride) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of APS-2-79 (hydrochloride) in these solvents is approximately 1, 25, and 10 mg/ml, respectively.

APS-2-79 (hydrochloride) is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, APS-2-79 (hydrochloride) should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. APS-2-79 (hydrochloride) has a solubility of approximately 0.3 mg/ml in a 1:2 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

APS-2-79 is an inhibitor of MAPK signaling dependent on kinase suppressor of Ras (KSR).[1] It inhibits ATP^{biotin} binding to KSR2 in KSR2-MEK1 complexes (IC_{50} = 120 nM), stabilizes KSR2 in an inactive state, and reduces interactions between B-RAF and KSR2-MEK1 complexes.[1,2] APS-2-79 (5 µM) inhibits KSR-stimulated phosphorylation of MEK and ERK in HEK293H cells.[1] It enhances the efficacy of the MEK inhibitor trametinib (Item No. 16292), further reducing cell viability in HCT116 and A549 cell lines containing K-Ras mutations, but not B-RAF mutant SK-MEL-239 and A375 cell lines, when used at a concentration of 1 µM.

References

1. Dhawan, N.S., Scopton, A.P., and Arvin, C.D. Small molecule stabilization of the KSR inactive state antagonizes oncogenic Ras signalling. *Nature* **537(7618)**, 112-116 (2016).
2. Neilsen, B.K., Frodyma, D.E., Lewis, R.E., *et al*. KSR as a therapeutic target for Ras-dependent cancers. *Expert Opin. Ther. Targets*. **21(5)**, 499-509 (2017).

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