

Product Data Sheet

Product Name: CT-179
Cat. No.: GC81499

Chemical Properties

Cas. No. 1996636-69-1

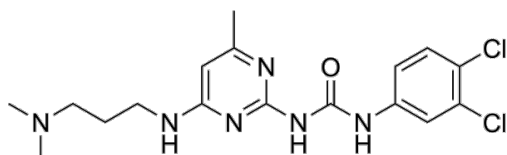
Formula $C_{17}H_{22}Cl_2N_6O$ M.Wt 397.3

Solubility DMSO: 12.5 mg/mL (31.46 mM); ultrasonic and warming and heat to 60°C) Storage Store at 4°C, protect from light

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available sizes: ship with RT, or blue ice upon request.

Structure



Background

CT-179 is a brain-penetrant and orally active OLIG2 inhibitor with a human IC_{50} of 1250 nM. CT-179 disrupts OLIG2 dimerization, phosphorylation, and DNA binding, blocking OLIG2-driven transcription. CT-179 induces G2/M phase arrest and increases G0 population. CT-179 induces apoptosis by reducing anti-apoptotic proteins and increasing cleaved caspase-3 and cleaved PARP. CT-179 can be used for the research of subgroup medulloblastoma [1].

Caution: Product has not been fully validated for medical applications. For research use only.

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In Vivo, CT-179 (50 mg/kg; i.p.; twice weekly; two weeks) as a single agent prolongs median event-free survival to 60 days, and combination with radiotherapy further extends median event-free survival to 75.5 days while delaying tumor growth in SHH-subgroup medulloblastoma-bearing NRG mice^[1]. CT-179 (75 mg/kg; p.o.; every other day) as a single agent prolongs median event-free survival to 76 days, and combination with radiotherapy further extends median event-free survival to 100.5 days in SHH-subgroup medulloblastoma-bearing NRG mice^[1]. CT-179 (80 mg/kg; i.p.; every other day) as a single agent reduces tumor proliferation, induces cell cycle arrest and neuronal differentiation, slows tumor growth, and improves event-free survival, while combination with radiotherapy further enhances event-free survival in SHH-subgroup medulloblastoma-bearing G-Smo mice^[1]. CT-179 (80 mg/kg; i.p.; every other day) combined with POx-Palbo enhances cell cycle arrest, increases apoptosis, and improves event-free survival more effectively than either single agent in SHH-subgroup medulloblastoma-bearing G-Smo mice^[1].

In Vitro, CT-179 (10 nM-10 μ M; 1 hour) dose-dependently disrupts OLIG2 dimerization in live HEK293 cells, with an IC₅₀ of 1250 nM^[1]. CT-179 (1-10 μ M; 1 hour) reduces OLIG2-DNA binding in live HEK293 cells, as shown by increased diffusion of DNA-bound OLIG2 and reduced fractional DNA-bound OLIG2 at concentrations of 1 μ M and 10 μ M^[1]. CT-179 (250 nM; 24 hours) blocks OLIG2-driven transcription in Daoy SHH-medulloblastoma cells, as measured by reduced activity of an LHX8 promoter luciferase reporter^[1]. CT-179 (1 μ M) shows minimal relevant off-target kinase inhibition in vitro; while it inhibits FLT3 with an IC₅₀ of 20 nM in a cell-free assay, the predicted in vivo cell potency is too low to be biologically meaningful^[1]. CT-179 (160 nM-2.5 μ M) shows minimal off-target effects in normal human primary cells at biologically relevant concentrations (160 nM, 630 nM), with only limited proliferation reduction observed at the supraphysiological concentration of 2.5 μ M^[1]. CT-179 (1 nM-10 μ M; 7 days) reduces viability in OLIG2-expressing SHH-medulloblastoma cell lines (Daoy, UW228, Med-813) in a manner correlating with OLIG2 expression, with IC₅₀ values ranging from 143.6 nM to 965.2 nM, and shows no activity in OLIG2-negative cell lines^[1]. CT-179 (1 μ M; 17 h-7 days) induces apoptosis, G2/M phase arrest, and mitotic disruption in Daoy SHH-medulloblastoma cells, and potentiates radiotherapy-induced apoptosis^[1]. CT-179 (1 μ M; 17-96 hours) induces apoptosis and disrupts mitotic mechanisms in Med-813 SHH-medulloblastoma cells, and potentiates radiotherapy-induced apoptosis^[1]. CT-179 (1 μ M; 48 hours) induces cell death in patient-derived medulloblastoma explant organoids (including SHH-subgroup R902), and when

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combined with radiotherapy, further reduces proliferation without increasing stem cell populations^[1].

References:

[1]. Li Y, et al. Suppressing recurrence in Sonic Hedgehog subgroup medulloblastoma using the OLIG2 inhibitor CT-179. Nat Commun. 2025;16(1):1091. Published 2025 Feb 4.

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