

Product Datasheet

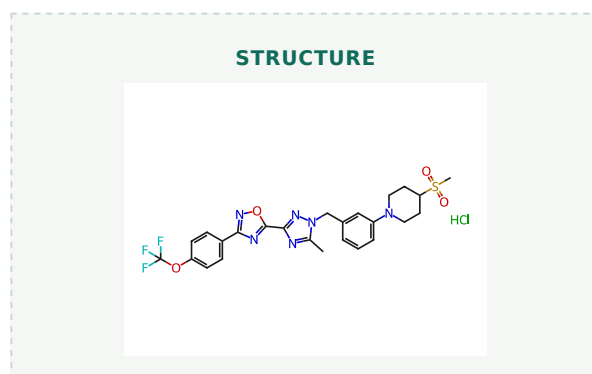
IACS-010759 HCl

Catalog No. **BP-01900A** · CAS **1807523-99-4** · Form **salt** · **salt** · Physical state **solid** · Color **white to off-white** · Version **1.0** · Revision **2026-09-03**

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Target Oxidative Phosphorylation; Mitochondrial Metabolism; Apoptosis	Research area Cancer
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Molecular formula	C₂₅H₂₆ClF₃N₆O₄S
Molecular weight	599.0250



DESCRIPTION

IACS-010759 hydrochloride is an orally active, potent mitochondrial complex I of oxidative phosphorylation (OXPHOS) inhibitor. IACS-010759 hydrochloride inhibits proliferation and induces apoptosis in models of brain cancer and acute myeloid leukemia (AML) reliant on OXPHOS. IACS-010759 hydrochloride has the potential for relapsed/refractory AML and solid tumors research.

BIOLOGICAL ACTIVITY

In Vitro

IACS-010759 hydrochloride (4 or 5 days) reduces viability and induces apoptosis in primary AML. IACS-010759 hydrochloride (72 hurs) robustly inhibits both OCR and galactose-dependent H460 cell viability and has nearly identical IC₅₀ values of 1.4?nM. IACS-010759 hydrochloride is similarly active in mouse (average IC₅₀?=?5.6?nM), rat (IC₅₀?=?12.2?nM), and cynomolgus monkey (IC₅₀?=?8.7?nM) cell lines.

In Vivo

IACS-010759 hydrochloride (5, 10, 25?mg/kg/day; oral; for 21 d) results in tumor regression with minimal body weight loss at the 5 or 10?mg/kg dose in mice bearing NB-1 (PGD-null) subcutaneous xenografts. IACS-010759 at the 25?mg/kg dose is not tolerated. IACS-010759 HCl (10?mg/kg; orally; QD (daily) or QD×5 (5 d on/2 d off); for 35 d) increases median survival from 28 d to longer than 60 d, whereas less-frequent dosing schedules (Q2D or Q3D) enhances survival to a lesser extent. IACS-010759 hydrochloride (0.3?mg/kg for iv; 1?mg/kg for oral) has low plasma clearance with a high volume of distribution, resulting in a prolonged terminal half-life (>24h).

SOLUBILITY

Soluble in DMSO : 60 mg/mL

H₂O : < 0.1 mg/mL

REFERENCES

[1]. Protopopova M. IACS-10759: A novel OXPHOS inhibitor which selectively kill tumors with metabolic vulnerabilities. [abstract]. In: Proceedings of the 106th Annual Meeting of the American Association for Cancer Research; 2015 Apr 18-22; Philadelphia, PA. Philadelphia (PA): AACR; Cancer Res 2015;75(15 Suppl):Abstract nr 4380. doi:10.1158/1538-7445.AM2015-4380.

[2]. Jennifer R Molina, et al. An inhibitor of oxidative phosphorylation exploits cancer vulnerability. Nat Med. 2018 Jul;24(7):1036-1046.

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Lot CoA and SDS are separate documents.