

Product Datasheet

CC-122

Catalog No. **BP-02322** · CAS **1015474-32-4** · Form **free base** · Physical state **other** · Version **1.0** · Revision **2026-09-10**

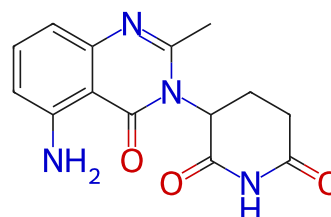
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Target E1/E2/E3 Enzyme; Apoptosis; Molecular Glues; NF-κB	Research area Inflammation/Immunology; Cancer
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Molecular formula **C₁₄H₁₄N₄O₃**

Molecular weight **286.2860**

STRUCTURE



DESCRIPTION

Avadomide (CC 122) is an orally active cereblon modulator. Avadomide modulates cereblon E3 ligase activity and induces apoptosis of diffuse large B-cell lymphoma (DLBCL) cell lines. Avadomide exhibits potent antitumor and immunomodulatory activities.

BIOLOGICAL ACTIVITY

In Vitro

Avadomide inhibits proliferation and induces apoptosis in ABC and GCB DLBCL. In DLBCL cell lines, Avadomide-induced degradation or short hairpin RNA-mediated knockdown of Aiolos and Ikaros correlates with increased transcription of IFN-stimulated genes independent of IFN-α, -β, and -γ production and/or secretion and results in apoptosis in both activated B-cell (ABC) and germinal center B-cell DLBCL.

In Vivo

Treatment of female CB-17 SCID mice with Avadomide (CC122) at 3 or 30 mg/kg once daily significantly decreased tumor growth in OCI-LY10 ABC-DLBCL (P = .028 and P < .001, respectively) and WSU-DLCL2 GCB-DLBCL derived xenograft models (P < .01) compared with the vehicle control. In a separate study, we assessed the ability of Avadomide (CC122) to promote degradation of Ikaros and Aiolos in vivo. In the 21-day efficacy study of WSU-DLCL2 xenograft transplanted mice, tumors were excised 1, 6, or 24 hours post final dosing. Aiolos and Ikaros expression was interrogated through immunohistochemistry (IHC) and was found to be decreased 64% and 30%, respectively, compared with vehicle within 1 hour of treatment, with a maximal reduction of 94% and 69%, respectively, observed at 6 hours. Aiolos and Ikaros levels partially recovered 24 hours postdosing with protein level within 20% and 34% of vehicle, respectively. The 24-hour postdose Aiolos and Ikaros expression represents the trough compound level following multiple doses of Avadomide (CC122). When the 1-hour time point is compared with the 24-hour postdose time point, there is a significant reduction in Aiolos but not Ikaros expression; however, at the 6-hour time point, both transcription factors are significantly different from the 24-hour time point. Taken together, these data reveal that Avadomide (CC122) inhibited DLBCL tumor growth in vivo and that this activity was

associated with the degradation of Aiolos and Ikaros in both ABC- and GCB-DLBCL xenograft models.

SOLUBILITY

Soluble in DMSO

REFERENCES

- [1]. Rasco DW, et al. A First-in-Human Study of Novel Cereblon Modulator Avadomide (CC-122) in Advanced Malignancies. Clin Cancer Res. 2019 Jan 1;25(1):90-98.
- [2]. Hagner, P.R.et al.CC-122, a pleiotropic pathway modifier, mimics an IFN response and has antitumor activity in DLBCL.Blood.Aug 6;126(6):779-89.
- [3]. Nishi H, et al., Anti-tumor effect of avadomide in gemcitabine-resistant pancreatic ductal adenocarcinoma. Cancer Chemother Pharmacol. 2023 Oct;92(4):303-314.
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Lot CoA and SDS are separate documents.