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Produktinformation

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- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

www.szabo-scandic.com

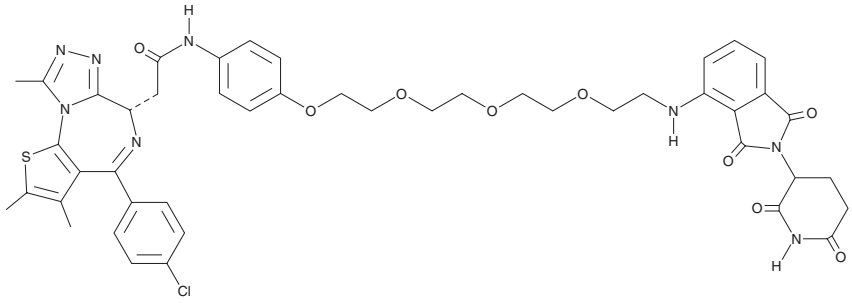
linkedin.com/company/szaboscandic

PRODUCT INFORMATION

ARV-825

Item No. 21109

CAS Registry No.: 1818885-28-7
Formal Name: 2-((S)-4-(4-chlorophenyl)-2,3,9-trimethyl-6H-thieno[3,2-f][1,2,4]triazolo[4,3-a][1,4]diazepin-6-yl)-N-(4-(2-(2-(2-((2-(2,6-dioxopiperidin-3-yl)-1,3-dioxoisoindolin-4-yl)amino)ethoxy)ethoxy)ethoxy)phenyl)acetamide
MF: $C_{46}H_{47}ClN_8O_9S$
FW: 923.4
Purity: ≥98%
UV/Vis.: λ_{max}: 247, 409 nm
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥2 years



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

Laboratory Procedures

ARV-825 is supplied as a crystalline solid. A stock solution may be made by dissolving the ARV-825 in the solvent of choice. ARV-825 is soluble in organic solvents such as DMSO and dimethyl formamide, which should be purged with an inert gas. The solubility of ARV-825 in these solvents is approximately 1 and 15 mg/ml, respectively.

Description

ARV-825 is a hetero-bifunctional molecule that is composed of a BRD4 binding moiety joined to an E3 ligase cereblon binding moiety using proteolysis targeting chimera (PROTAC) technology.[1] ARV-825 actively recruits BRD4 to cereblon, resulting in the rapid and efficient degradation of BRD4 by the proteasome (50% maximum degradation at < 1 nM).[1] In Burkitt's lymphoma cells, ARV-825 reduces c-Myc levels, blocks cell proliferation, and induces apoptosis.[1] The degradation of BRD4 in cells treated with ARV-825 can be blocked with lenalidomide (Item No. 14643), a competitor for binding to cereblon.[2]

References

1. Lu, J., Qian, Y., Altieri, M., *et al.* Hijacking the E3 ubiquitin ligase cereblon to efficiently target BRD4. *Chem. Biol.* **22(6)**, 755-763 (2015).
2. Abruzzese, M.P., Bilotta, M.T., Fionda, C. *et al.* Inhibition of bromodomain and extra-terminal (BET) proteins increases NKG2D ligand MICA expression and sensitivity to NK cell-mediated cytotoxicity in multiple myeloma cells: role of cMYC-IRF4-miR-125b interplay. *J. Hematol. Oncol.* **9(1)**, 134 (2016).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM