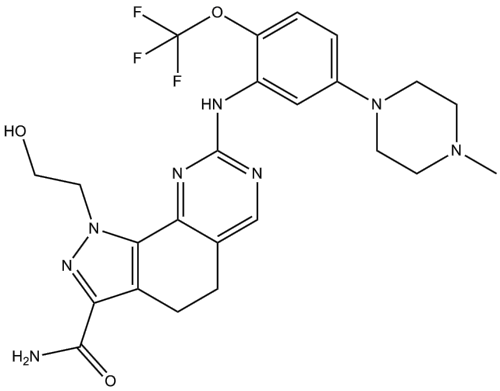
Product Data Sheet

Product Name: NMS-1286937
Cat. No.: GC12515

Chemical Properties

Cas. No.	1034616-18-6		
Formula	C24H27F3N8O3	M.Wt	532.52
Solubility	≥ 13.3mg/mL in DMSO	Storage	Store at -20°C
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 size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

Cell lines	Ovcar-3 and ES-2 cells (human ovarian carcinoma cell lines)
Preparation Method	Cells were cultured in RPMI-1640 or McCoy's 5A medium supplemented with 10% fetal bovine serum (FBS) and maintained at 37°C under 5% CO_2. Cells were treated with NMS-1286937 at concentrations of 10–50nM for 24–72 hours.

Product Data Sheet

Reaction Conditions	10-50μM; 24-72h
Applications	NMS-1286937 induced G_2/M phase cell cycle arrest, with a dose-dependent increase in G_2/M population.

Animal experiment [2]:

Animal models	NCr-nu/nu mice with patient-derived xenografts (PDXs) of platinum-resistant ovarian carcinomas
Preparation Method	Mice were orthotopically (intraperitoneally) or subcutaneously transplanted with tumor fragments/cells. NMS-1286937 was administered orally at 50mg/kg for 4 consecutive days per week for 3 weeks, combined with paclitaxel (15mg/kg intravenously once weekly for 3 weeks). Mice were monitored for tumor growth and survival.
Dosage form	50mg/kg; p.o.; 4 consecutive days per week for 3 weeks
Applications	The combination of NMS-1286937 and Paclitaxel significantly prolonged median survival in orthotopic PDX models. Tumor growth inhibition was accompanied by induction of mitotic block (pSer10H3) and DNA damage/apoptosis (γH2AX) in ex vivo tumor tissues.

Caution: Product has not been fully validated for medical applications. For research use only.
Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

References:
[1] Chiappa M, Decio A, Guarrera L, et al. Onvansertib treatment overcomes olaparib resistance in high-grade ovarian carcinomas. Cell Death Dis. 2024 Jul 22;15(7):521.
[2] Affatato R, Chiappa M, Guffanti F, et al. Onvansertib and paclitaxel combined in platinum-resistant ovarian carcinomas. Ther Adv Med Oncol. 2022 May 31;14:17588359221095064.

Background

NMS-1286937 is an orally active small-molecule compound. NMS-1286937 potently and selectively inhibits Polo-like kinase 1 (PLK1) activity with a half-maximal inhibitory concentration (IC_{50}) of 2nM[1]. By competitively binding to the ATP site, NMS-1286937 reversibly inhibits PLK1, leading to G2/M phase cell cycle arrest, chromosomal misalignment, and abnormal spindle formation in various tumor cells, ultimately resulting in apoptosis[2]. NMS-1286937 shows potential in the treatment of solid tumors and hematological malignancies[3-4].

In vitro, treatment of medulloblastoma cell lines (D425, D458) with NMS-1286937 (10–20nM) for 24–48 hours induced G2/M phase cell cycle arrest and significantly inhibited cell proliferation and stem cell self-renewal capacity[5]. In ovarian cancer cell lines (Ovcar-3, ES-2), treatment with NMS-1286937 (30–50nM) for 24–72 hours suppressed cell proliferation while activating apoptosis and DNA damage pathways[6].

In vivo, daily oral administration of NMS-1286937 (25mg/kg) for four weeks in LKB1 $^{fl/fl}$ p53$^{fl/fl}$ transgenic endometrial cancer model mice significantly inhibited tumor growth and reduced tumor weight[7]. In nude mice bearing patient-derived xenografts (PDX) of platinum-resistant ovarian cancer, once-weekly intravenous administration of NMS-1286937 (50mg/kg) in combination with Paclitaxel (15mg/kg) significantly extended median survival and induced mitotic arrest and DNA damage in tumor tissues[8].

References:
[1] Valsasina B, Beria I, Alli C, et al. NMS-P937, an orally available, specific small-molecule polo-like kinase 1 inhibitor with antitumor activity in solid and hematologic malignancies. Mol Cancer Ther. 2012 Apr;11(4):1006-16.

Caution: Product has not been fully validated for medical applications. For research use only.
Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

[2] Su S, Chhabra G, Singh CK, et al. PLK1 inhibition-based combination therapies for cancer management. Transl Oncol. 2022 Feb;16:101332.
[3] Ahn DH, Barzi A, Ridinger M, et al. Onvansertib in Combination with FOLFIRI and Bevacizumab in Second-Line Treatment of KRAS-Mutant Metastatic Colorectal Cancer: A Phase Ib Clinical Study. Clin Cancer Res. 2024 May 15;30(10):2039-2047.
[4] Nouri M, Varkaris A, Ridinger M, et al. AKT Inhibition Sensitizes to Polo-Like Kinase 1 Inhibitor Onvansertib in Prostate Cancer. Mol Cancer Ther. 2024 Oct 1;23(10):1404-1417.
[5] Wang D, Veo B, Pierce A, et al. A novel PLK1 inhibitor onvansertib effectively sensitizes MYC-driven medulloblastoma to radiotherapy. Neuro Oncol. 2022 Mar 12;24(3):414-426.
[6] Chiappa M, Decio A, Guarrera L, et al. Onvansertib treatment overcomes olaparib resistance in high-grade ovarian carcinomas. Cell Death Dis. 2024 Jul 22;15(7):521.
[7] Sinha N, Shen X, Haag J, et al. Onvansertib exhibits anti-proliferative and anti-invasive effects in endometrial cancer. Front Pharmacol. 2025 Mar 17;16:1545038.
[8] Affatato R, Chiappa M, Guffanti F, et al. Onvansertib and paclitaxel combined in platinum-resistant ovarian carcinomas. Ther Adv Med Oncol. 2022 May 31;14:17588359221095064.

Caution: Product has not been fully validated for medical applications. For research use only.
Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA