

Product Data Sheet

Product Name: PD-1/PD-L1 inhibitor 1 (BMS-1)

Cat. No.: GC15142

Chemical Properties

Cas. No. 1675201-83-8

Chemical Name (S)-1-(2,6-dimethoxy-4-((2-methyl-[1,1'-biphenyl]-3-yl)methoxy)benzyl)piperidine-2-carboxylic acid

SMILES O=C([C@H]1N(CC2=C(OC)C=C(OCC3=CC=CC(C4=CC=CC=C4)=C3C)C=C2OC)CCC1)O

Formula $C_{29}H_{33}NO_5$

M.Wt 475.58

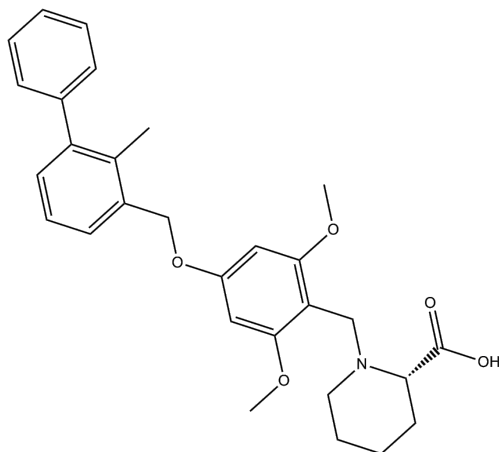
Solubility $\geq 5.98\text{mg/mL}$ in DMSO with ultrasonic and warming

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]:

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Cell lines	4T1 cells (mouse triple-negative breast cancer cell line); BMDM2 cells (mouse bone marrow-derived macrophages polarized to M2 phenotype)
Preparation Method	4T1 cells and BMDM2 cells were maintained in RPMI-1640 medium supplemented with 10% FBS at 37°C, 5% CO ₂ . Cells were pretreated with PD-1/PD-L1 inhibitor 1 at 2μM (with 10μg/mL CNH-PG) for 30min, then irradiated with 808nm laser (1.3W/cm ² or 1.5W/cm ²) for 10min, followed by 24h continued incubation. Cell viability was assayed by CCK-8; cell proliferation and apoptosis were assessed by CFSE decay and annexin V staining via flow cytometry; PD-L1, γH2AX, cleaved caspase 3, BAX, BCL2 and GAPDH were detected by Western blot and flow cytometry; surface PD-L1 was measured by immunofluorescent staining and flow cytometry. BMDM2 cells (single or co-cultured with 4T1) after same treatment were stained for F4/80, CD80, CD86, CD206 and assessed for iNOS, GBP5, ARG1 by Western blot and phagocytosis of fluorescent latex beads by flow cytometry.
Reaction Conditions	2μM; 30min pretreatment + 10min laser + 24h incubation
Applications	PD-1/PD-L1 inhibitor 1 combined with CNH-PG-mediated mild photothermia in 4T1 cells increased cytotoxicity, suppressed proliferation, increased annexin V positivity, upregulated γH2AX, cleaved caspase 3 and BAX, downregulated BCL2, and repressed CNH-PG-mPT-induced total and surface PD-L1 upregulation. In BMDM2 cells, PD-1/PD-L1 inhibitor 1 with CNH-PG showed no toxicity. PD-1/PD-L1 inhibitor 1 combined with CNH-PG-mPT in BMDM2 cells (single or co-culture with 4T1) repressed CNH-PG-mPT-induced PD-L1 upregulation, boosted M1-like phenotype, and these shifts were more pronounced in co-culture.
Animal experiment [2]:	
Animal models	8-week-old male C57BL/6 mice subcutaneously inoculated with 1×10 ⁶ B16.F10 melanoma cells (tumor volume ~100mm ³)

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Preparation Method	Mice were intraperitoneally injected with PD-1/PD-L1 inhibitor 1 at 10mg/kg every two days for 14 consecutive days (control groups received PBS or 250mg IgG i.p. every two days). Twenty-four hours after the final injection, mice were euthanized; body weight, food intake, heart weight/body weight ratio, tumor weight were recorded; echocardiography was performed before sacrifice to measure ejection fraction, fractional shortening, LVIDs and LVIDd; heart tissues were collected for H&E and Masson staining, TUNEL staining, RT-PCR (BNP, IL-6, TNF- α , CD31, VEGF), Western blot (SOCS3, p-JAK/JAK, STAT3, VEGF, GAPDH) and flow cytometry (CD45, PD-L1, F4/80, CD86, Ly6C, Ly6G on tumor-infiltrating macrophages).
Dosage form	10mg/kg; i.p.; every two days for 14 days
Applications	PD-1/PD-L1 inhibitor 1 administration reduced body weight in mice. PD-1/PD-L1 inhibitor 1 increased heart weight/body weight ratio. PD-1/PD-L1 inhibitor 1 reduced tumor weight. PD-1/PD-L1 inhibitor 1 decreased ejection fraction and fractional shortening, and increased LVIDs and LVIDd in mice. PD-1/PD-L1 inhibitor 1 increased myocardial BNP mRNA, IL-6 mRNA and TNF- α mRNA, and decreased CD31 and VEGF mRNA in myocardium. PD-1/PD-L1 inhibitor 1 decreased SOCS3, p-JAK/JAK and STAT3 protein levels in myocardium, while increasing VEGF protein. PD-1/PD-L1 inhibitor 1 increased myocardial fibrosis area, TUNEL-positive apoptotic area and inflammatory foci area in heart sections. PD-1/PD-L1 inhibitor 1 increased CD86+MHCII+ macrophage proportion in tumor tissues.

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References:

- [1] Wang C, Xu Y, Xu H, et al. PD-L1 blockade TAM-dependently potentiates mild photothermal therapy against triple-negative breast cancer. J Nanobiotechnol. 2023 Dec 11;21:476.
- [2] Fu J, Wang G, Zeng L, et al. PD-1/PD-L1 inhibitor treatment associated with cardiotoxicity regulated by macrophage polarization and SOCS3/JAK/STAT3 signaling pathway. Cent Eur J Immunol. 2025;50(1):24-37.

Background

PD-1/PD-L1 inhibitor 1 (BMS-1) is a potent inhibitor of PD-1/PD-L1 protein interaction ($IC_{50}=6-100nM$). PD-1/PD-L1 inhibitor 1 blocks PD-1 binding to PD-L1, relieves immune suppression exerted by tumor cells through PD-L1 on T cells and other immune effector cells, enhances anti-tumor immune responses, and produces synergistic effects when combined with chemotherapy, targeted therapy or HDAC inhibitors. PD-1/PD-L1 inhibitor 1 can be used in immune checkpoint blockade therapy and related mechanistic studies for various malignancies including melanoma, B-cell lymphoma, triple-negative breast cancer and hepatocellular carcinoma^[1-4].

In vitro, treatment with PD-1/PD-L1 inhibitor 1 (2 μ M) combined with mild photothermia for 24h in mouse 4T1 triple-negative breast cancer cells inhibited cell proliferation and induced apoptosis^[5]. Treatment with PD-1/PD-L1 inhibitor 1 (2 μ M) combined with ticagrelor (2 μ M) for 30min to 14 days in tumor-associated macrophages (P-TAMs, B-TAMs, H-TAMs) co-cultured with pancreatic cancer cells (PANC-1, BxPC-3, HPCCs) enhanced phagocytosis of pancreatic cancer cells by tumor-associated macrophages, and inhibited tumor-associated macrophage-induced colony formation, proliferation and migration of pancreatic cancer cells^[6]. Treatment with PD-1/PD-L1 inhibitor 1 (1 μ M) for 24h in M2-type tumor-associated macrophages co-cultured with A549 cells or Lewis cells upregulated CD80, CD86, MHC-II surface markers and GBP5 protein expression, inhibited tumor cell proliferation, and induced apoptosis^[7].

In vivo, male C57BL/6 mice subcutaneously inoculated with melanoma cells received intraperitoneal injections of PD-1/PD-L1 inhibitor 1 at 10mg/kg every two days for 14 days, PD-1/PD-L1 inhibitor 1 reduced tumor weight, decreased left ventricular ejection fraction and fractional shortening, increased

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left ventricular internal dimensions in diastole and systole, and increased myocardial fibrosis area and inflammatory foci area^[8]. Male C57BL/6J mice subcutaneously inoculated with B16/BL6 melanoma cells (Cflar knockdown) received intratumoral injections of PD-1/PD-L1 inhibitor 1 at 50µg daily for 12 days, PD-1/PD-L1 inhibitor 1 significantly increased phagocytic activity of tumor-associated macrophages and inhibited melanoma growth^[9]. C57BL/6 mice inoculated with B16F10-luciferase cells on the back received intraperitoneal injections of PD-1/PD-L1 inhibitor 1 at 5mg/kg or 10mg/kg every two days for 12 days, PD-1/PD-L1 inhibitor 1 reduced body weight and increased heart weight/body weight ratio, increased myocardial inflammatory infiltration, interstitial fibrosis and TUNEL-positive cardiomyocyte numbers, and reduced tumor weight^[10].

References:

- [1] Zhang R, Wan Y, Lv H, et al. DTX@VTX NPs synergy PD-L1 immune checkpoint nanoinhibitor to reshape immunosuppressive tumor microenvironment for enhancing chemo-immunotherapy. J Mater Chem B. 2021;9(38):7544-7556.
- [2] Zhao Y, Jia Y, Shi T, et al. Depression promotes hepatocellular carcinoma progression through a glucocorticoid-mediated upregulation of PD-1 expression in tumor-infiltrating NK cells. Carcinogenesis. 2019 Sep 18;40(9):1132-1141.
- [3] Liu Y, Zeng Y, Xia Y, et al. Programmed cell death-1/programmed cell death-ligand 1 inhibitors exert antiapoptosis and antiinflammatory activity in lipopolysaccharide stimulated murine alveolar macrophages. Int J Mol Med. 2021;47(4):382-396.
- [4] Li T, Xu D, Ruan Z, et al. Metabolism/Immunity Dual-Regulation Thermogels Potentiating Immunotherapy of Glioblastoma Through Lactate-Excretion Inhibition and PD-1/PD-L1 Blockade. Adv Sci (Weinh). 2024 May;11(18):e2310163.
- [5] Wang C, Xu YH, Xu HZ, et al. PD-L1 blockade TAM-dependently potentiates mild photothermal therapy against triple-negative breast cancer. J Nanobiotechnology. 2023 Dec 11;21:476.
- [6] Kang Y, Amofo EB, Entsie P, et al. Co-treatment of ticagrelor and anti-PD-1 immunotherapy in tumor-associated macrophages reduces pancreatic cancer cell growth and migration through the TGF-β1/Smad2 pathway. Biomed Pharmacother. 2025 Nov;192:118695.
- [7] Xu H, Li T, Wang C, et al. Synergy of nanodiamond-doxorubicin conjugates and PD-L1 blockade

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effectively turns tumor-associated macrophages against tumor cells. J Nanobiotechnol. 2021 Sep 6;19:268.

[8] Fu J, Wang G, Zeng L, et al. PD-1/PD-L1 inhibitor treatment associated with cardiotoxicity regulated by macrophage polarization and SOCS3/JAK/STAT3 signaling pathway. Cent Eur J Immunol. 2025;50(1):24-37.

[9] Tsuruta A, Shiiba Y, Matsunaga N, et al. Diurnal expression of PD-1 on tumor-associated macrophages underlies the dosing time-dependent antitumor effects of the PD-1/PD-L1 inhibitor BMS-1 in B16/BL6 melanoma-bearing mice. Mol Cancer Res. 2022 Jun;20(6):972-982.

[10] Chen Y, Liu Y, Wang Y, et al. Gut microbiota dysbiosis amplifies PD-1/PD-L1 inhibitor-related cardiotoxicity by upregulating proinflammatory factors TNF- α and IL-1 β in colonic macrophages. J Exp Clin Cancer Res. 2022 Jan 3;41(1):1.

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