

Product Data Sheet

Product Name: Butyrolactone 3

Cat. No.: GC10226

Chemical Properties

Cas. No. 778649-18-6

Chemical Name *re*-tetrahydro-4-methylene-5-oxo-2R-propyl-3S-furancarboxylic acid

SMILES O=C1C([C@@H](C(O)=O)[C@H](CCC)O1)=C

Formula $C_9H_{12}O_4$

M.Wt 184.2

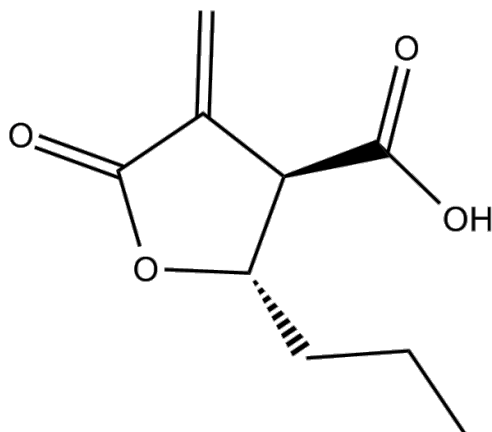
Solubility $\leq 14\text{mg/ml}$ in ethanol; 14mg/ml in DMSO; 13mg/ml in dimethyl formamide

Storage Store at -20°C , unstable in solution, ready to use.

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 C3H10T1/2 cells

Preparation Method C3H10T1/2 cells were cultured in DMEM supplemented with 10% fetal bovine serum (FBS) and transfected with the lentiviral vectors islet-1 or GFP (MOI = 20) at 5µg/mL polybrene. After 24 hours of incubation at 37°C and 5% CO₂, the medium was changed. The cells were divided into three groups: a blank control group (C3H10T1/2), a control group (lentiviral vector-transfected with GFP), and a group transfected with the lentiviral vector islet-1 (lentiviral vector-transfected with islet-1). The LV-islet-1 group was further divided into four subgroups based on the time of transfection: LV-islet-1-1w, LV-islet-1-2w, LV-islet-1-3w, and LV-islet-1-4w. 24h after LV-islet-1 or GFP transfection, Butyrolactone 3 (100µM) was added to the culture medium and treated for 4 weeks. The cells were defined as LV-islet-1 + Butyrolactone 3 group and control group + Butyrolactone 3 group.

Reaction Conditions 100µM; 4 weeks

Applications Butyrolactone 3 inhibits the expression of Gcn5 after islet-1 transfection.

Animal experiment [2]:

Animal models Ovarian cancer PDX mice model

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Preparation Method An ovarian cancer PDX model was established. Four-week-old female NCG (NOD/ShiLtJGpt-Prkdc em26Cd52 Il2rg em26Cd22 /Gpt) mice were purchased for use in PDX model development. Fresh tumor tissue was collected from individual patients, mechanically homogenized, and injected subcutaneously into the lower back or axilla of NCG mice. When the implanted tumors were stable for passage, equal lengths of tumor homogenate were implanted into new recipient NCG mice. Two weeks later, the mice were randomly divided into three groups: PBS + DMSO, Cisplatin (5mg/kg) + DMSO, and Cisplatin + Butyrolactone 3 (5mg/kg).

Dosage form 5mg/kg; ip; 30d

Applications Butyrolactone 3 can increase the sensitivity of certain ovarian cancer cases to Cisplatin and even reverse their resistance.

References:

- [1]. Xu H, Zhou Q, Yi Q, et al. Islet-1 synergizes with Gcn5 to promote MSC differentiation into cardiomyocytes[J]. Scientific Reports, 2020, 10(1): 1817.
- [2]. Sun C, Li X, Teng Q, et al. Targeting platinum-resistant ovarian cancer by disrupting histone and RAD51 lactylation[J]. Theranostics, 2025, 15(7): 3055.

Background

Butyrolactone 3 is a small molecule inhibitor of the histone acetyltransferase GCN5 ($IC_{50} = 100\mu M$) [1]. Butyrolactone 3 inhibits the catalytic activity of GCN5 to reduce histone acetylation levels and block downstream GCN5 signaling pathways, thereby disrupting tumor cell survival mechanisms [2-3]. Butyrolactone 3 is primarily used to treat lymphoma [4].

In C3H10T1/2 cells, Butyrolactone 3 (100 μM ; 4 weeks) inhibits the expression of Gcn5 after islet-1 transfection [5]. In H3K9ac cells, Butyrolactone 3 (0-200 μM ; 48h) inhibits GCN5 HAT activity and

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thus reduces the viability of burkitt lymphoma cells [6].

In ovarian cancer PDX mice model, Butyrolactone 3 (5mg/kg; ip; 30d) can increase the sensitivity of ovarian cancer cases to Cisplatin and even reverse their resistance [7]. In streptozotocin and a high fat diet rat model, Butyrolactone 3 (1mg/kg; nasal administrated; 8 weeks) attenuates increase in GCN5, H3K9 histone hyperacetylation on CDK5 promoter, and CDK5 upregulation [8].

References:

- [1]. Mai A, Rotili D, Tarantino D, et al. Small-molecule inhibitors of histone acetyltransferase activity: identification and biological properties[J]. Journal of medicinal chemistry, 2006, 49(23): 6897-6907.
- [2]. Haque M E, Jakaria M, Akther M, et al. The GCN5: its biological functions and therapeutic potentials[J]. Clinical Science, 2021, 135(1): 231-257.
- [3]. Luan J, Chu Z, Chandra J, et al. Small molecular inhibitors targeting chromatin regulating proteins for cancer[J]. Current Protein and Peptide Science, 2016, 17(5): 455-462.
- [4]. Gong Y, Fu W. Reversible role of MIR654/3P and MIR9/3P in pathogenesis of Epstein-Barr virus–negative, but not Epstein-Barr virus–positive, Burkitt lymphoma[J]. Journal of Leukocyte Biology, 2025, 117(3): qiae237.
- [5]. Xu H, Zhou Q, Yi Q, et al. Islet-1 synergizes with Gcn5 to promote MSC differentiation into cardiomyocytes[J]. Scientific Reports, 2020, 10(1): 1817.
- [6]. Farria A T, Mustachio L M, Akdemir Z H C, et al. GCN5 HAT inhibition reduces human Burkitt lymphoma cell survival through reduction of MYC target gene expression and impeding BCR signaling pathways[J]. Oncotarget, 2019, 10(56): 5847.
- [7]. Sun C, Li X, Teng Q, et al. Targeting platinum-resistant ovarian cancer by disrupting histone and RAD51 lactylation[J]. Theranostics, 2025, 15(7): 3055.
- [8]. Cai H B, Fan Z Z, Tian T, et al. Diabetes-induced H3K9 hyperacetylation promotes development of Alzheimer's disease through CDK5[J]. Journal of Alzheimer's Disease, 2020, 77(1): 75-84.

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