

Product Data Sheet

Product Name: Avanbulin

Cat. No.: GC63663

Chemical Properties

Cas. No. 798577-91-0

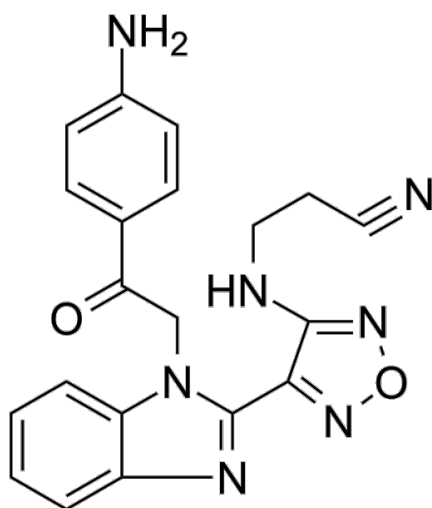
Formula $C_{20}H_{17}N_7O_2$ M.Wt 387.39

Solubility DMSO : 250 mg/mL (645.34 mM; Need ultrasonic) 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



Background

Avanbulin (BAL27862) is a potent, Colchicine site-binding, tubulin assembly inhibitor. Avanbulin

Caution: Product has not been fully validated for medical applications. For research use only.

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inhibits tubulin assembly at 37 °C with an IC₅₀ of 1.4 μM. Avanbulin binds to tubulin with an apparent K_d value of 244 nM. Avanbulin can be used for the research of cancer and cell division[1][2][3][4].

Avanbulin (0-4 μM) binds to tubulin with an apparent K_d value of 244 nM[1]. Avanbulin (0-20 μM) and Colchicine (0-10 μM) are competitive for binding to tubulin with an apparent K_i value of 1.8 μM[1]. Avanbulin binds to the same site as Colchicine at the intradimer interface[1]. Avanbulin (50 μM; 0, 10, 20, 30, 60 min) induces the proteolysis of tubulin[1]. Avanbulin (1-100 nM; 72 h; HeLa-tubGFP cells) has anti-proliferative effects[1]. Avanbulin (33 nM; 0, 10, 20, 30, 60 min; HeLa-tubGFP cells) collapses the mitotic spindle and forms the tiny tubulin aggregates[1]. Avanbulin does not induce the formation of tubulin oligomers[1]. Avanbulin (0.1 nM-1.0 μM; 96 hours) induces growth inhibition of cells with a median relative IC₅₀ of 13.8 nM[2]. Avanbulin (0-1,000 nM; 3 days) inhibits the growth of glioblastoma cancer stem-like cells, GBM6 (EC₅₀=20.8 nM) and GBM9 (EC₅₀=21.7 nM)[3]. Avanbulin (6 nM and 20 nM) inhibits the migration of GBM6 and GBM9 cells[3]. Avanbulin (6 nM and 20 nM; GBM6-shEB1 and GBM6-sh0 cells) triggers astrocytic differentiation of GBM6 in an EB1-dependent manner[3]. Avanbulin (12 nM; 4 h) reduces kinetochore-microtubule (KT-MT) occupancy of MG132(10 μM; 2h) treated hTert-RPE1 eGFP-α-tubulin cells[4]. Avanbulin (12 nM; 4 h) reduces average inter-KT distances of cells[4]. Avanbulin-treated (12 nM; 4 h) cells show intact spindle morphology and lack of obvious chromosome alignment defects[4].

[1]. Prota AE, et al. The novel microtubule-destabilizing drug avanbulin binds to the colchicine site of tubulin with distinct effects on microtubule organization. J Mol Biol. 2014 Apr 17;426(8):1848-60.

[2]. Kolb EA, et al. Initial testing (stage 1) of BAL101553, a novel tubulin binding agent, by the pediatric preclinical testing program. Pediatr Blood Cancer. 2015 Jun;62(6):1106-9.

[3]. Bergès R, et al. The Novel Tubulin-Binding Checkpoint Activator BAL101553 Inhibits EB1-Dependent Migration and Invasion and Promotes Differentiation of Glioblastoma Stem-like Cells. Mol Cancer Ther. 2016 Nov;15(11):2740-2749.

[4]. Dudka D, et al. Complete microtubule-kinetochore occupancy favours the segregation of merotelic attachments. Nat Commun. 2018;9(1):2042. Published 2018 May 23.

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