

Product Data Sheet

Product Name: CK2-TN03

Cat. No.: GC79111

Chemical Properties

Cas. No. 313226-24-3

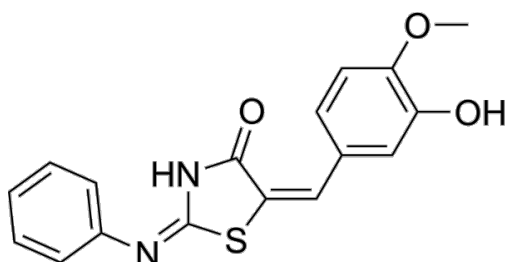
Formula $C_{17}H_{14}N_2O_3S$ M.Wt 326.37

Solubility DMSO: 83.33 mg/mL (255.32 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

CK2-TN03 is an ATP-competitive casein kinase 2 (CK2) inhibitor, with an IC_{50} of 165 nM and a K_i of 20 nM. CK2-TN03 inhibits CK2-mediated survivin activation and reduces CK2-dependent phosphorylation levels of BRD4/MYCN and AKT1. CK2-TN03 exerts anti-neuroblastoma effects by

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

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inhibiting survivin, leading to mitotic catastrophe and apoptosis of cancer cells. CK2-TN03 can be used in studies related to neuroblastoma [1].

In Vivo, CK2-TN03 (40 mg/kg; i.p.; once daily; 28 days) significantly reduces neuroblastoma xenograft tumor growth and improves mouse survival, with a subset of mice achieving long-term tumor control or remission[1].

In Vitro, CK2-TN03 (0.2-25.0 μ M; 48 h) induces dose-dependent cell death in the medulloblastoma DAOY cell line and multiple neuroblastoma cell lines, with EC50 values ranging from 0.31 to 1.95 μ M; it shows no activity in glioblastoma U87 cells (EC50 >25 μ M)[1]. CK2-TN03 (0.35-1.0 μ M; 48 h) activates caspase 3/7 in CHP-212 neuroblastoma cells in a time-dependent manner, and primarily induces caspase-dependent apoptosis[1]. CK2-TN03 (0.5-1.0 μ M; 24-48 h) induces G2/M cell cycle arrest and subsequent cell death in CHP-212 neuroblastoma cells[1]. CK2-TN03 (0.5 μ M; 24 h) arrests CHP-212 neuroblastoma cells in mitosis, blocks successful cell division, and induces mitotic catastrophe and cell death[1]. CK2-TN03 (0.5 μ M; 48 h) downregulates the activity and expression of survivin in CHP-212 neuroblastoma cells by directly inhibiting CK2-mediated phosphorylation of survivin and indirectly altering the AKT1/MDM2/p53 and BRD4/MYCN pathways, without changing the expression of CK2[1]. CK2-TN03 (0.5 μ M; 48 h) does not affect the viability of differentiated quiescent SH-SY5Y neuroblastoma cells, which exhibit low survivin expression levels[1]. CK2-TN03 (1-10 μ M; 72 h) induces cell death in a diverse panel of 160 cancer cell lines, with significant tumor entity selectivity, and exhibits the highest potency against melanoma, lymphoma, lung cancer, neuroblastoma, ovarian cancer, myeloma, soft tissue cancer and osteosarcoma cell lines[1]. CK2-TN03 (10 μ M; 1 h) exhibits excellent permeability in MDCKII-MDR1 cells, with negligible efflux and no P-gp substrate activity[1].

References:

[1]. Cazzanelli G, et al. Switching off CK2-mediated activation of survivin offers new therapeutic opportunities in neuroblastoma. Exp Mol Med. 2026;58(1):227-242.

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