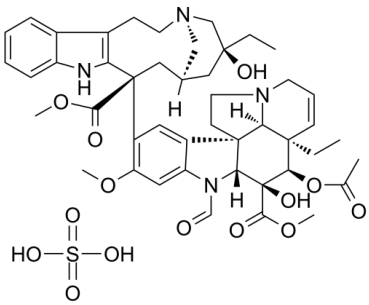
Data Sheet

Product Name:	Vincristine (sulfate)
Cat. No.:	CS-1778
CAS No.:	2068-78-2
Molecular Formula:	C46H58N4O14S
Molecular Weight:	923.04
Target:	Apoptosis; Microtubule/Tubulin
Pathway:	Apoptosis; Cell Cycle/DNA Damage; Cytoskeleton
Solubility:	DMSO : 82.5 mg/mL (89.38 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

Vincristine sulfate is an antitumor vinca alkaloid which inhibits **microtubule** formation in mitotic spindle, resulting in an arrest of dividing cells at the metaphase stage. It binds to **microtubule** with a K_i of 85 nM. **In Vitro:** Vincristine inhibits net addition of tubulin dimers at assembly ends of steady-state microtubules with K_i of 85 nM[1]. Vincristine stabilizes the spindle apparatus resulting in failure of the chromosomes to segregate leading to metaphase arrest and inhibition of mitosis at low concentrations. At higher concentrations, Vincristine may disrupt and induce total depolymerization of microtubules[2]. Vincristine induces apoptosis in tumor cells and inhibits SH-SY5Y cell proliferation with IC_{50} of 0.1 μM. Vincristine induces mitotic arrest and promots the expression of caspase-3 and -9 and cyclin B, while decreasing the expression of cyclin D[3]. Vincristine induced neurotoxicity is caused by interference with microtubule function, which results in blockage of axonal transport and thus in axonal degeneration[4]. **In Vivo:** Vincristine (3 mg/kg, i.p.) induces mean growth delay of > 120 and > 52 day, and repopulates fractions of 0.06% and 5%, administrated in mice bearing bilateral subcutaneous xenografts Rh12 or Rh18, respectively[5].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [1]Cells are plated in 2 mL of medium in 35 mm plates at a concentration of about 5×10^4 cells/mL and grow for 24 h at 37°C in an atmosphere of 5% CO_2 and 95% air. Then medium is replaced with fresh medium lacking or containing 4 nM drug and proliferation is continued for 3 days. Cell counts are done each day in a Coulter Counter after detaching the cells with trypsin and EDTA.

References:

[1]. Jordan, M.A., et al. Comparison of the effects of vinblastine, vincristine, vindesine, and vinepidine on microtubule dynamics and cell proliferation in vitro. Cancer Res, 1985. 45(6): p. 2741-7.

[2]. Gidding, C.E., et al, Vincristine revisited. Crit Rev Oncol Hematol, 1999. 29(3): p. 267-87.

[3]. Donoso, J.A., et al, Action of the vinca alkaloids vincristine, vinblastine, and desacetyl vinblastine amide on axonal fibrillar organelles in vitro. Cancer Res, 1977. 37(5): p. 1401-7.

[4]. Horton, J.K., et al. Relationships between tumor responsiveness, vincristine pharmacokinetics and arrest of mitosis in human tumor xenografts. Biochem Pharmacol, 1988. 37(20): p. 3995-4000.

[5]. Baguley, B.C., et al, Inhibition of growth of colon 38 adenocarcinoma by vinblastine and colchicine: evidence for a vascular mechanism. Eur J Cancer, 1991. 27(4): p. 482-7.

[6]. Zhang D, et al. Co-delivery nanoparticles with characteristics of intracellular precision release drugs for overcoming multidrug resistance. Int J Nanomedicine. 2017 Mar 16;12:2081-2108.

CAIndexNames:

Vincaleukoblastine, 22-oxo-, sulfate (1:1)

SMILES:

CC[C@@]1(C=CCN2CC3)[C@@]2([H])[C@@]3(C4=CC([C@](C5=C6C7=CC=CC=C7N5)(C[C@](C[C@](CC)(O)C8)([H])C[N@@]8CC6)C(OC)=O)=C(OC)C=C4N9C=O)[C@]9([H])[C@](C(OC)=O)(O)[C@@H]1OC(C)=O.O=S(O)(O)=O

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

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