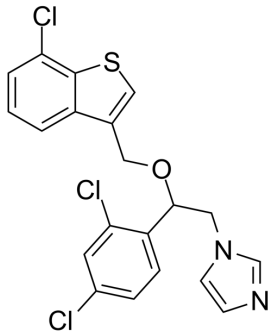
Sertaconazole

Cat. No.:	HY-B0736
CAS No.:	99592-32-2
Molecular Formula:	$C_{20}H_{15}Cl_3N_2OS$
Molecular Weight:	437.77
Target:	Fungal; Autophagy; Apoptosis; p38 MAPK; Microtubule/Tubulin
Pathway:	Anti-infection; Autophagy; Apoptosis; MAPK/ERK Pathway; Cell Cycle/DNA Damage; Cytoskeleton
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Sertaconazole (FI7056 free base) is a broad-spectrum topical antifungal agent, exhibits anti-inflammatory activity via activation of a p38-COX-2-PGE2 pathway. Sertaconazole is also a microtubule inhibitor, shows antiproliferative effect, induces apoptosis and autophagy, and can also inhibit the migration of cells[1][2][3][4].

In Vitro

Sertaconazole (0.03-40 μg/mL; 24 h) inhibits 150 strains of yeasts which includes six Candida species with arithmetic mean MIC of 0.77 μg/mL[1].
Sertaconazole (1 μg/mL; 5, 10, 30, 60 min) activates p38 MAP kinase in a time-dependent manner[2].
Sertaconazole (1, 2 μg/mL; 6, 8, or 24 h) increases a twofold release of PGE2 via COX-2 in keratinocytes, which is dependent on p38 activation[2].
Cetaconazole (10, 20, 30, 40 μM; 24 h) induces strong mitotic arrest by depolymerizing interphase and spindle microtubules, thereby inducing chromosome aggregation defects and causing anti-proliferation effect[3].
Sertaconazole (20, 40 μM; 24 h) induces apoptosis through p53 pathway in HeLa cells[3].
Sertaconazole (20, 30 μM; 24, 48, and 72 h) inhibits the migration of HeLa cells in a concentration-dependent manner[3].
Sertaconazole (15, 30 μM; 24 h) induces autophagy in A549, H460 cells[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	C. albicans, C. guilliermondii, C. krusei, C. parapsilosi, C. tropicalis, C. glabrata
Concentration:	0.03-40 μg/m
Incubation Time:	24 h
Result:	Againsted 150 strains of yeasts (six Candida species) which included C. albicans, C. guilliermondii, C. krusei, C. parapsilosi, C. tropicalis, C. glabrata species with arithmetic mean MIC values of 1.02, 0.51, 0.38, 0.31, 1.67 and 0.78 μg/mL, respectively.

Western Blot Analysis[2]

Cell Line:	HaCaT cells
Concentration:	1 μg/mL
Incubation Time:	5, 10, 30, 60 min

Result:	Showed activity of activating p38 MAP kinase and Hsp27 in a time-dependent manner.

Western Blot Analysis[2]

Cell Line:	HaCaT cells
Concentration:	1, 2 μg/mL
Incubation Time:	6 or 8 h
Result:	Induced 50% expression of COX-2 and resulted in a twofold increased in PGE2 release.

Western Blot Analysis[2]

Cell Line:	siRNA-transfected HaCaT cells (without p38 MAP kinase expression)
Concentration:	1 μg/mL
Incubation Time:	24 h
Result:	Mediated induction of PGE2 was dependent on p38 activation.

Cell Proliferation Assay[3]

Cell Line:	HeLa, HEK-293, MCF-7, A549 cells
Concentration:	0-100 μM
Incubation Time:	24 h
Result:	Showed antiproliferation activity with IC_{50}s of 38, 45.1, 41.5, and 40.8 μM for HeLa, HEK-293, A549, and MCF-7 cells, respectively. Exhibited mitotic block activity and induced cell death at concentration above 30 μM, but no significant increased in the number of mitotic cells. Depolymerized interphase and spindle microtubules inducing defect in chromosomal congression.

Apoptosis Analysis[3]

Cell Line:	HeLa cells
Concentration:	10, 20, 40 μM
Incubation Time:	24 h
Result:	Induced approximately 5%, 10%, and 21% cells apoptotic at concentrations of 10, 20 and 40 μM, respectively.

Western Blot Analysis[3]

Cell Line:	A549 cells
Concentration:	20, 40 μM
Incubation Time:	24 h
Result:	Induced apoptosis through p53 pathway that the expression of p53 from 30% to 50% and 95% and p21 from 11 to 39% and 40% respectively.

	Resulted in Noxa and Puma, two direct transcriptional targets of p53 to be overexpressed.

Cell Migration Assay [3]

Cell Line:	HeLa cells
Concentration:	20, 30 μM
Incubation Time:	24, 48, and 72 h
Result:	Inhibited the migration of HeLa cells at concentrations lesser than its IC_{50}, which in a concentration-dependent manner.

Cell Autophagy Assay[4]

Cell Line:	A549, H460 cells
Concentration:	15, 30 μM
Incubation Time:	24 h
Result:	Increased endogenous LC3 puncta and LC3 intensity, which indicated induction of autophagy in A549 and H460 cells.

In Vivo

Sertaconazole (1% (w/v); apply to the left ear, once) suppresses of TPA-induced ear edema CD-1 mice[2].

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Animal Model:	CD-1 mice (TPA-induced ear edema model)[2].
Dosage:	1% (w/v)
Administration:	Apply to the left ear, once.
Result:	Exhibited a significant reduction of inflammation in mice by mediating PGE2 release.

CUSTOMER VALIDATION

- MedComm. 16 December 2021.

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REFERENCES

[1]. Carrillo-Muñoz AJ, et al. In-vitro antifungal activity of sertaconazole, econazole, and bifonazole against Candida spp. J Antimicrob Chemother. 1995 Oct;36(4):713-6.

[2]. Sur R, et al. Anti-inflammatory activity of sertaconazole nitrate is mediated via activation of a p38-COX-2-PGE2 pathway. J Invest Dermatol. 2008 Feb;128(2):336-44.

[3]. Sebastian J, et al. Sertaconazole induced toxicity in HeLa cells through mitotic arrest and inhibition of microtubule assembly. Naunyn Schmiedebergs Arch Pharmacol. 2021 Jun;394(6):1231-1249.

[4]. Zhang W, et al. Sertaconazole provokes proapoptotic autophagy via stabilizing TRADD in nonsmall cell lung cancer cells. MedComm (2020). 2021 Dec 16;2(4):821-837.

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

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