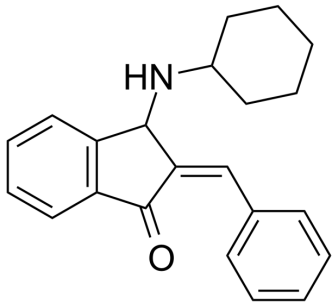
(E/Z)-BCI

Cat. No.:	HY-126390
CAS No.:	15982-84-0
Molecular Formula:	$C_{22}H_{23}NO$
Molecular Weight:	317.42
Target:	Phosphatase; Apoptosis
Pathway:	Metabolic Enzyme/Protease; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

(E/Z)-BCI (NSC 150117) is a dual-specificity phosphatase 6 (DUSP6) inhibitor with anti-inflammatory activities. (E/Z)-BCI attenuates LPS-induced inflammatory mediators and ROS production in macrophage cells via activating the Nrf2 signaling axis and inhibiting the NF-κB pathway[1].

IC_{50} & Target

DUSP6[1]

In Vitro

(E/Z)-BCI hydrochloride (2-10 μM; 72 hours) significantly decreases cell viability in a time and dose-dependent manner in gastric epithelial cell GES1, GC cell lines, and AGS cell lines[2].
(E/Z)-BCI hydrochloride (0.5-4 μM; 24 hours) significantly inhibits DUSP6 expression in LPS-activated macrophages[1].
(E/Z)-BCI hydrochloride (0.5-2 μM; 24 hours) treatment significantly inhibits the expression of IL-1β, TNF-α and IL-6 mRNA in LPS-activated macrophages[1].
(E/Z)-BCI hydrochloride decreases ROS production and activates the Nrf2 pathway in LPS-activated macrophages[1].(E/Z)-BCI hydrochloride inhibits cell proliferation, migration and invasion in a receptor-independent manner and enhances Cisplatin (CDDP) cytotoxicity (enhances CDDP-induced cell death and apoptosis) at pharmacological concentrations in the gastric cancer (GC) cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	Gastric epithelial cell GES1, GC cell lines (HGC27, SGC7901, MKN45, BGC823, MGC803, SNU216, NUGC4), AGS cell lines
Concentration:	2 μM, 4 μM, 6 μM, 8 μM, 10 μM
Incubation Time:	72 hours
Result:	Cell viability was significantly decreased in a time and dose-dependent manner.

Western Blot Analysis[1]

Cell Line:	RAW264.7 macrophage cells (by LPS-activated macrophages)
Concentration:	0.5 μM, 1 μM, 2 μM, 4 μM
Incubation Time:	24 hours

Result:	DUSP6 protein was significantly downregulated in LPS-activated macrophages.

RT-PCR[1]

Cell Line:	RAW264.7 macrophage cells (by LPS-activated macrophages)
Concentration:	0.5 μM, 1 μM, 2 μM
Incubation Time:	24 hours
Result:	The expression of IL-1β, TNF-α and IL-6 mRNA was significantly inhibited inLPS-activated macrophages.

In Vivo

(E/Z)-BCI hydrochloride (35 mg/kg; intraperitoneal injection; every 7 days; for four weeks; female BALB/c nude mice) treatment enhances cisplatin efficacy in PDX models[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Patient-derived xenograft (PDX) models (4-5-week-old female BALB/c nude mice)[2]
Dosage:	35 mg/kg
Administration:	Intraperitoneal injection; every 7 days; for four weeks
Result:	Tumor weights in the PDX models treated plus CDDP were significantly suppressed compared with tumors from PDX model mice treated with either agent alone.

CUSTOMER VALIDATION

- Cell Death Dis. 2021 Sep 2;12(9):825.

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REFERENCES

[1]. Zhang F, et al. DUSP6 Inhibitor (E/Z)-BCI Hydrochloride Attenuates Lipopolysaccharide-Induced Inflammatory Responses in Murine Macrophage Cells via Activating the Nrf2 Signaling Axis and Inhibiting the NF-κB Pathway. Inflammation. 2019 Apr;42(2):672-681.

[2]. Wu QN,et al. Pharmacological inhibition of DUSP6 suppresses gastric cancer growth and metastasis and overcomes cisplatin resistance. Cancer Lett. 2018 Jan 1;412:243-255.

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