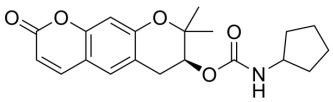
CGK012

Cat. No.:	HY-163409
CAS No.:	2044497-76-7
Molecular Formula:	$C_{20}H_{23}NO_5$
Molecular Weight:	357.4
Target:	β-catenin
Pathway:	Stem Cell/Wnt
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

CKG012 is an inhibitor for Wnt/βcatenin signaling pathway. CGK012 inhibits release of HMGB1 and transcription of β-catenin, exhibits attenuating activities against cecal ligation and puncture (CLP)-induced sepsis and multiple myeloma cancer[1][2].

In Vitro

CGK012 (0-20 μM) inhibits HMGB1-release by reducing LPS-induced HMGB1 acetylation and SIRT1 expression and suppresses therefore the excessive vascular permeability in HUVECs, decreases the expression of pathogen-associated molecules like TLR2/4 without affecting cellular viability of HUVECs.[1].

CGK012 (0-20 μM) exhibits ameliorates inflammatory response through decreases adhesion and migration of inflammatory immune cells, production of proinflammatory cytokines like IL-6, TNF-α, β, and transcription factors NF-kB and ERK1/2[1].

CGK012 (0-20 μM) promotes β-catenin phosphorylation/degradation and repressed the expression β-catenin-dependent genes, thereby inhibiting the proliferation of multiple myeloma cells RPMI-8226 with an IC_{50} of 5.08 μM[2].

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

Cell Viability Assay[1]

Cell Line:	HUVEC
Concentration:	5-100 μM
Incubation Time:	48 h
Result:	Revealed no effects on cell viability.

Western Blot Analysis[1]

Cell Line:	HUVECs, HEK293-FL reporter, RPMI-8226
Concentration:	0-20 μM
Incubation Time:	12 h
Result:	Reduced levels of SIRT1 and acetylation of HMGB1, inhibited vascular permeability in HUVECs.Reduced levels of β-catenin in HEK293-FL reporter and RPMI-8226.

Cell Migration Assay [1]

Cell Line:	HUVEC
Concentration:	0-20 μM
Incubation Time:	6 h
Result:	Inhibited migration of neutrophils through monolayers of HUVECs.

In Vivo

CGK012 (0.05-0.53 mg/kg, i.v., double doses) inhibits HMGB1 release and immune cell migration, improves vascular cell stability and survival rates of C57BL/6 mice with CLP-induced sepsis[1].

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

Animal Model:	CLP- induced sepsis in C57BL/6 mice[1]
Dosage:	0.26-0.53 mg/kg
Administration:	double doses, 12 or 50 h after surgery.
Result:	Reduced expression of HMGB1 and improved the survival rate.

REFERENCES

[1]. Park YJ, et al., Antiseptic Functions of CGK012 against HMGB1-Mediated Septic Responses. Int J Mol Sci. 2024 Mar 4;25(5):2976.

[2]. Choi PJ,et al., Anti-proliferative activity of CGK012 against multiple myeloma cells via Wnt/β-catenin signaling attenuation. Leuk Res. 2017 Sep;60:103-108.

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