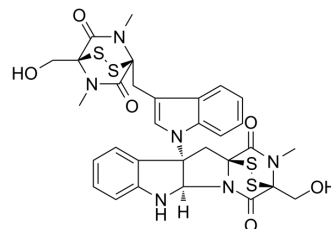


Chetomin

Cat. No.:	HY-107553
CAS No.:	1403-36-7
Molecular Formula:	C ₃₁ H ₃₀ N ₆ O ₆ S ₄
Molecular Weight:	710.87
Target:	HSP; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Chetomin, an active component of <i>Chaetomium globosum</i> , is a heat shock protein 90/hypoxia-inducible factor 1 alpha (Hsp90/HIF1 α) pathway inhibitor. Chetomin is a potent, nontoxic non-small cell lung cancer cancer stem cells (NSCLC CSC)-targeting molecule ^[1] .												
IC₅₀ & Target	HSP90												
In Vitro	<p>Chetomin (0~10 μM; 24 hours; H460 and H1299 cells) shows progressively lower expression of several survival-promoting proteins promoted by Hsp90/HIF1α activity, including insulin-like growth factor 1 (IGF1 R), epidermal growth factor receptor (EGFR), Src, mitogen-activated protein kinase kinase 1/2 (MEK1/2), activation of protein kinase B (Akt), and mammalian target of rapamycin (mTOR) ^[1].</p> <p>Chetomin (0~10 μM; 24 hours; H1299 cells) elicits cell cycle arrest in susceptible and chemoresistant NSCLC cell lines^[1].</p> <p>Chetomin (1 μM; 3 days; H460 and H1299 cells) pretreatment abolishes their sphere-forming capacity. Chetomin inhibits sphere-forming by NSCLC CSCs within a nanomolar range, and proliferation of susceptible and chemoresistant NSCLC non-CSCs within a micromolar range. Chetomin (24 h) decreases HIF-response element activity in H460 and H1299 monolayer cultures. Chetomin (0~10 μM) specifically inhibits the Hsp90-HIF1α binding interaction in HIF1α's N-terminus ^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Western Blot Analysis^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>H460 and H1299 cells</td> </tr> <tr> <td>Concentration:</td> <td>0~10 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>24 hours</td> </tr> <tr> <td>Result:</td> <td>Showed progressively lower expression of several survival-promoting proteins promoted by Hsp90/HIF1α activity, including insulin-like growth factor 1 (IGF1 R), epidermal growth factor receptor (EGFR), Src, mitogen-activated protein kinase kinase 1/2 (MEK1/2), activation of protein kinase B (Akt), and mammalian target of rapamycin (mTOR).</td> </tr> </table> <p>Cell Cycle Analysis^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>H1299 cells</td> </tr> <tr> <td>Concentration:</td> <td>0~10 μM</td> </tr> </table>	Cell Line:	H460 and H1299 cells	Concentration:	0~10 μ M	Incubation Time:	24 hours	Result:	Showed progressively lower expression of several survival-promoting proteins promoted by Hsp90/HIF1 α activity, including insulin-like growth factor 1 (IGF1 R), epidermal growth factor receptor (EGFR), Src, mitogen-activated protein kinase kinase 1/2 (MEK1/2), activation of protein kinase B (Akt), and mammalian target of rapamycin (mTOR).	Cell Line:	H1299 cells	Concentration:	0~10 μ M
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Cell Line:	H1299 cells												
Concentration:	0~10 μ M												

	Incubation Time:	24 hours
	Result:	Elicited cell cycle arrest in susceptible and chemoresistant NSCLC cell lines.
In Vivo	Chetomin (0~100 mg/kg; p.o.) inhibits lung tumorigenesis in NSCLC mouse models ^[1] . Chetomin markedly decreases tumor formation in several murine models of NSCLC ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.	
	Animal Model:	Mouse
	Dosage:	0~100 mg/kg
	Administration:	P.o.
	Result:	Inhibited lung tumorigenesis in NSCLC mouse models.

REFERENCES

[1]. Min S, et al. Chetomin, a Hsp90/HIF1 α pathway inhibitor, effectively targets lung cancer stem cells and non-stem cells. *Cancer Biol Ther.* 2020;21(8):698-708.

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

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