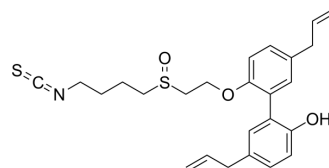


## CT1-3

Cat. No.:	HY-150596
CAS No.:	2460738-32-1
Molecular Formula:	C <sub>25</sub> H <sub>29</sub> NO <sub>3</sub> S <sub>2</sub>
Molecular Weight:	455.63
Target:	Apoptosis; Bcl-2 Family; JNK
Pathway:	Apoptosis; MAPK/ERK Pathway
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



## BIOLOGICAL ACTIVITY

Description	CT1-3 is a potent anticancer agent. CT1-3 induces mitochondria-mediated apoptosis by regulating JNK/Bcl-2/Bax/XIAP pathway. CT1-3 suppresses the epithelial mesenchymal transition (EMT) potential of human cancer cells (HCCs) via regulating the E-cadherin/Snail axis, thus inhibits tumorigenesis. CT1-3 has a strong antitumor effect in mice model and exhibits no significant hepatic and renal toxicity <sup>[1]</sup> .								
In Vitro	<p>CT1-3 has excellent inhibitory activity against multiple cancer cells with IC<sub>50</sub> range of 5.10~14.06 μM in LOVO, A549, HepG2, MDA-MB-231 and HONE1, et al<sup>[1]</sup>.</p> <p>CT1-3 (10 μM; 24 h) notably increases ROS production in HCCs, significantly decreases mitochondrial membrane potential, and reduces Bcl-2 and XIAP levels and increases phospho-JNK and Bax levels<sup>[1]</sup>.</p> <p>CT1-3 (7.5 μM; 24 h) reduces the capacity of migration and invasion of HCCs, significantly promotes the expression level of E-cadherin (E-cad), and markedly decreases the pro-metastatic and pro-invasive protein Snail<sup>[1]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Western Blot Analysis<sup>[1]</sup></p> <table border="1"> <tr> <td>Cell Line:</td> <td>A549, H460 and LOVO</td> </tr> <tr> <td>Concentration:</td> <td>10 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>24 h</td> </tr> <tr> <td>Result:</td> <td>Notably increased ROS production, significantly decreased mitochondrial membrane potential, and reduced Bcl-2 and XIAP levels and increased phospho-JNK and Bax levels.</td> </tr> </table>	Cell Line:	A549, H460 and LOVO	Concentration:	10 μM	Incubation Time:	24 h	Result:	Notably increased ROS production, significantly decreased mitochondrial membrane potential, and reduced Bcl-2 and XIAP levels and increased phospho-JNK and Bax levels.
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In Vivo	<p>CT1-3 (20 mg/kg; IP, for 28 days) significantly suppresses tumor growth and exhibits no hepatic and renal toxicity in MDA-MB-231 xenografts model<sup>[1]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1"> <tr> <td>Animal Model:</td> <td>Male BALB/c-nu/nu mice (injected with A549, OVCAR3 and MDA-MB-231)<sup>[1]</sup></td> </tr> <tr> <td>Dosage:</td> <td>20 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>IP, for 28 days</td> </tr> </table>	Animal Model:	Male BALB/c-nu/nu mice (injected with A549, OVCAR3 and MDA-MB-231) <sup>[1]</sup>	Dosage:	20 mg/kg	Administration:	IP, for 28 days		
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Result:	Significantly suppresses tumor growth and exhibited no hepatic and renal toxicity.
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## REFERENCES

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[1]. Tao C, et al. CT1-3, a novel magnolol-sulforaphane hybrid suppresses tumorigenesis through inducing mitochondria-mediated apoptosis and inhibiting epithelial mesenchymal transition. Eur J Med Chem. 2020 Aug 1;199:112441.

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

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