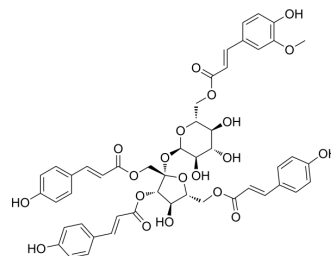


Vanicoside B

Cat. No.:	HY-N9561
CAS No.:	155179-21-8
Molecular Formula:	C ₄₉ H ₄₈ O ₂₀
Molecular Weight:	956.89
Target:	CDK; STAT
Pathway:	Cell Cycle/DNA Damage; JAK/STAT Signaling; Stem Cell/Wnt
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Vanicoside B is a phenylpropanoyl sucrose derivative, can be isolated from the herb <i>Persicaria dissitiflora</i> . Vanicoside B targets cyclin-dependent kinase 8 (CDK8) and exhibits anti-tumor activity. The potential mechanism is Vanicoside B blocks CDK8-mediated signaling pathways and decreases the expression of epithelial-mesenchymal transition proteins, so that it leads to cell cycle arrest and apoptosis ^{[1][2]} .															
IC₅₀ & Target	CDK3	STAT3														
In Vitro	<p>Vanicoside B (2.5-20 μM; 72 h) shows antiproliferative activity against a panel of cancer cell lines in triple-negative breast cancer (TNBC) MDA-MB-231 cells and HCC38 cells^[1].</p> <p>Vanicoside B (2.5-20 μM; 72 h, 14 d, and 72 h, respectively) inhibits cell viability, colony formation, and disturbs cell cycle distribution in TNBC cells^[1].</p> <p>Vanicoside B (2.5-10 μM; 48 h) decreased p-STAT1, p-STAT3, and p-S6 protein level, and induces apoptosis by regulating the Skp2-p27 axis in TNBC cells^[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" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 30%;">Cell Line:</td> <td>MDA-MB-231 cells and HCC38 cells</td> </tr> <tr> <td>Concentration:</td> <td>0, 2.5, 5, 10 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>48 hours</td> </tr> <tr> <td>Result:</td> <td>Increased cleaved PARP, and p27 protein expressions, but decreased Skp2 protein level. Suppressed CDK8 target genes and the expression of EMT-associated proteins. Suppressed the expression of the cell proliferation marker Ki-67 in tumor tissues, also significantly suppressed the expressions of p-STAT1 (S727) and AXL.</td> </tr> </table> <p>Cell Cycle Analysis^[1]</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 30%;">Cell Line:</td> <td>MDA-MB-231 cells and HCC38 cells</td> </tr> <tr> <td>Concentration:</td> <td>0, 2.5, 5, 10 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>72 hours</td> </tr> </table>		Cell Line:	MDA-MB-231 cells and HCC38 cells	Concentration:	0, 2.5, 5, 10 μM	Incubation Time:	48 hours	Result:	Increased cleaved PARP, and p27 protein expressions, but decreased Skp2 protein level. Suppressed CDK8 target genes and the expression of EMT-associated proteins. Suppressed the expression of the cell proliferation marker Ki-67 in tumor tissues, also significantly suppressed the expressions of p-STAT1 (S727) and AXL.	Cell Line:	MDA-MB-231 cells and HCC38 cells	Concentration:	0, 2.5, 5, 10 μM	Incubation Time:	72 hours
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Incubation Time:	72 hours															

	Result:	Inhibited cell cycle at sub-G1 phase.
In Vivo	Vanicoside B (5 mg/kg and 20 mg/kg; i.p.; 3 times per week for 4 weeks) inhibits tumor growth in xenografted mouse models with MDAMB-231 cells ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.	
	Animal Model:	MDA-MB-231 cell-implanted xenograft mouse model ^[1]
	Dosage:	5 mg/kg, 20 mg/kg
	Administration:	Intraperitoneal injection; 3 times per week over 4 weeks
	Result:	Significantly reduced tumor volumes at 5 mg/kg and 20 mg/kg by 53.85% and 65.72%, respectively.

REFERENCES

[1]. Kim D, et al. Antitumor Activity of Vanicoside B Isolated from *Pericaria dissitiflora* by Targeting CDK8 in Triple-Negative Breast Cancer Cells. *J Nat Prod.* 2019 Nov 22;82(11):3140-3149.

[2]. Takasaki M, et al. Cancer chemopreventive activity of phenylpropanoid esters of sucrose, vanicoside B and lapathoside A, from *Polygonum lapathifolium*. *Cancer Lett.* 2001 Nov 28;173(2):133-8.

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

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