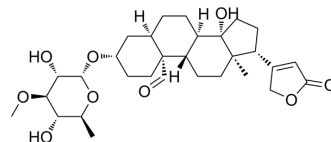


Peruvoside

Cat. No.:	HY-108016
CAS No.:	1182-87-2
Molecular Formula:	C ₃₀ H ₄₄ O ₉
Molecular Weight:	548.66
Target:	Src; PI3K; JNK; STAT; EGFR; Apoptosis; Autophagy
Pathway:	Protein Tyrosine Kinase/RTK; PI3K/Akt/mTOR; MAPK/ERK Pathway; JAK/STAT Signaling; Stem Cell/Wnt; Apoptosis; Autophagy
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Peruvoside is a potent inhibitor of Src, PI3K, JNK, STAT, and EGFR. Peruvoside induces apoptosis and autophagy and possesses a broad spectrum of anticancer activity in breast, lung, liver cancers and leukemia. Peruvoside is a broad-spectrum and potent antiviral activity against positive-sense RNA viruses. Peruvoside sensitizes Gefitinib (HY-50895)-resistant tumour cells (A549, PC9/gef and H1975) to Gefitinib ^{[1][2][3][4]} .														
In Vitro	<p>Peruvoside (50-1000 nM, 24 h) inhibits the viability and proliferation in PC9, PC9/gef, H3255, and H1975 cell lines^[1]. Peruvoside (0.005-0.5 μM, 72 h) sensitizes A549, PC9/gef and H1975 to Gefitinib when in combination with Gefitinib (0.01-0.5 μM)^[1]. Peruvoside (0-100 μM, 24 h) induces cell cycle arrest and apoptosis in MCF-7, HpG2, and A549 cells^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Cell Proliferation Assay^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>PC9, PC9/gef, H3255, and H1975 cell lines</td> </tr> <tr> <td>Concentration:</td> <td>0, 0.01, 0.05, 0.1, 0.5, 1 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>24, 48, 72, 96 h.</td> </tr> <tr> <td>Result:</td> <td>Inhibited the viabilities of TKI-sensitive and TKI-resistant cell lines at all tested time points. Inhibited the EGFR-mutant lung cancer cell viability and proliferation with 24 h IC₅₀s of 48 nM, 74 nM, 67 nM, 143 nM, 277 nM and 428 nM for A549, PC9, PC9/gef, H3255, H1975 and BEAS-2B cells, respectively. Significantly inhibited the proliferation of A549 (48, 72, and 96 h) and H3255 (24, 48, 72, and 96 h) lungcancer cells.</td> </tr> </table> <p>Cell Viability Assay^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>A549, PC9/gef and H1975 cell lines</td> </tr> <tr> <td>Concentration:</td> <td>0.005, 0.0075, 0.01, 0.025, 0.05, 0.5 μM (in combination with 0.01, 0.05, 0.1, 0.25, 0.5 μM Gefitinib)</td> </tr> <tr> <td>Incubation Time:</td> <td>72 h.</td> </tr> </table>	Cell Line:	PC9, PC9/gef, H3255, and H1975 cell lines	Concentration:	0, 0.01, 0.05, 0.1, 0.5, 1 μM	Incubation Time:	24, 48, 72, 96 h.	Result:	Inhibited the viabilities of TKI-sensitive and TKI-resistant cell lines at all tested time points. Inhibited the EGFR-mutant lung cancer cell viability and proliferation with 24 h IC ₅₀ s of 48 nM, 74 nM, 67 nM, 143 nM, 277 nM and 428 nM for A549, PC9, PC9/gef, H3255, H1975 and BEAS-2B cells, respectively. Significantly inhibited the proliferation of A549 (48, 72, and 96 h) and H3255 (24, 48, 72, and 96 h) lungcancer cells.	Cell Line:	A549, PC9/gef and H1975 cell lines	Concentration:	0.005, 0.0075, 0.01, 0.025, 0.05, 0.5 μM (in combination with 0.01, 0.05, 0.1, 0.25, 0.5 μM Gefitinib)	Incubation Time:	72 h.
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Concentration:	0.005, 0.0075, 0.01, 0.025, 0.05, 0.5 μM (in combination with 0.01, 0.05, 0.1, 0.25, 0.5 μM Gefitinib)														
Incubation Time:	72 h.														

	<p>Result: Had synergistic effects on A549 cells at the combination of 0.005, 0.075, or 0.01 μM and a low dose of gefitinib (0.01 or 0.05 μM). Increased the sensitivity of PC9/gef and H1975 cells to Gefitinib at 0.025 or 0.05 μM.</p>
	<p>Apoptosis Analysis^[2]</p>
	<p>Cell Line: MCF-7, A549 and HepG2 cell lines</p>
	<p>Concentration: 0-100 μM</p>
	<p>Incubation Time: 24 h</p>
	<p>Result: Induced cell cycle arrest and apoptosis with lethal concentrations of IC50 for (MCF-7 - 100 nM), (A549 - 100 nM) and (HepG2 - 100 nM), respectively. Arrested cell cycle at G0/G1 in MCF-7, A549 and HepG2 cells. Significantly decreased the transcription of Chk1, Chk2, CDK6 and Cyclin D1 cell cycle genes in MCF-7, A549, and HepG2 cells.</p>
In Vivo	<p>Peruvoside (0.1 mg/kg for i.p; once daily for 28 days) suppresses the tumour growth in lung cancer mice model^[1]. Peruvoside (0.59 mg/kg for i.p; once daily for 7 days) reduces mortality in EV-A71-infected mice model^[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
	<p>Animal Model: Lung cancer mouse model^[1]</p>
	<p>Dosage: 0.1 mg/kg</p>
	<p>Administration: Intraperitoneal injection (i.p.); Once daily for 28 days</p>
	<p>Result: Reduced the tumour size to 129.9 mm³, which was significantly smaller than the control group (348 mm³). Significantly decreased levels of phosphorylated Src Y419 in tumour tissues compared with control tissues.</p>
	<p>Animal Model: EV-A71-infected mice model^[4]</p>
	<p>Dosage: 0.59 mg/kg</p>
	<p>Administration: Intraperitoneal injection (i.p.); Once daily for 7 days</p>
	<p>Result: Substantially reduced clinical scores based on physical symptoms of body weight, activity, breathing, movement, and dehydration. Showed about 6 log reduction in viral titre with 99.9% efficacy in inhibiting virus.</p>

REFERENCES

- [1]. Lai Y, et al. Peruvoside is a novel Src inhibitor that suppresses NSCLC cell growth and motility by downregulating multiple Src-EGFR-related pathways. *Am J Cancer Res.* 2022 Jun 15;12(6):2576-2593.
- [2]. Reddy D, et al. Peruvoside targets apoptosis and autophagy through MAPK Wnt/ β -catenin and PI3K/AKT/mTOR signaling pathways in human cancers. *Life Sci.* 2020 Jan 15;241:117147. doi: 10.1016/j.lfs.2019.117147. Epub 2019 Dec 9. PMID: 31830480.
- [3]. Feng Q, et al. Peruvoside, a Cardiac Glycoside, Induces Primitive Myeloid Leukemia Cell Death. *Molecules.* 2016 Apr 22;21(4):534.

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

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