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- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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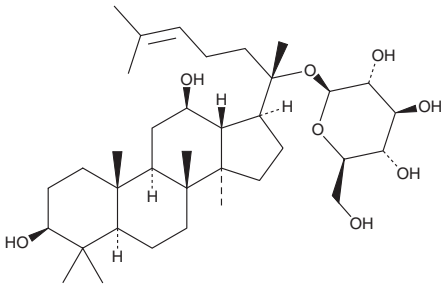
PRODUCT INFORMATION

Ginsenoside CK

Item No. 29926

CAS Registry No.:	39262-14-1
Formal Name:	(3β,12β)-3,12-dihydroxydammar-24-en-20-yl, β-D-glucopyranoside
Synonyms:	Ginsenoside Compound K, Ginsenoside IH901
MF:	$C_{36}H_{62}O_8$
FW:	622.9
Purity:	≥95%
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Ginsenoside CK is supplied as a crystalline solid. A stock solution may be made by dissolving the ginsenoside CK in the solvent of choice, which should be purged with an inert gas. Ginsenoside CK is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of ginsenoside CK in these solvents is approximately 10 mg/ml.

Ginsenoside CK is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, ginsenoside CK should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Ginsenoside CK has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Ginsenoside CK is a metabolite of the saponin ginsenoside Rb_1 (Item No. 15319) that has diverse biological activities.[1-4] Ginsenoside CK is formed from ginsenoside Rb_1 *via* intestinal bacterial *exo*-β-D-glucosidase.[1] It reduces protein levels of matrix metalloproteinase-2 (MMP-2) and pro-MMP-9 in, and inhibits growth, adhesion, and invasion of, MHCC97-H human hepatocellular carcinoma cells (HCCs) when used at concentrations of 50 and 75 µM.[2] It inhibits metastasis in an MHCC97-H mouse xenograft model but does not reduce tumor growth. Ginsenoside CK reduces the percentage of memory B cells in the spleen, the number of swollen joints, and lymph node hyperplasia in an adjuvant-induced model of rheumatoid arthritis.[3] It increases the activity of pSer9-glycogen synthase kinase and insulin degrading enzyme (IDE) and reduces the accumulation of amyloid-β (1-42) (Aβ42; Item No. 20574) in the rat hippocampal CA1 and CA3 regions in a model of vascular dementia induced by chronic cerebral hypofusion.[4] It also increases the percentage of time spent in the target quadrant of the Morris water maze when administered at doses of 100 and 200 mg/kg in the same model.

References

1. Jung, I.-H., Lee, J.H., Hyun, Y.-J., *et al. Biol. Pharm. Bull.* **35(4)**, 573-581 (2012).
2. Ming, Y., Cen, Z., Chen, L., *et al. Planta Med.* **77(5)**, 428-433 (2011).
3. Chen, J., Wang, Q., Wu, H., *et al. Pharm. Biol.* **54(7)**, 1280-1288 (2016).
4. Zong, W., Zeng, X., Chen, S., *et al. J. Pharmacol. Sci.* **139(3)**, 223-230 (2019).

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