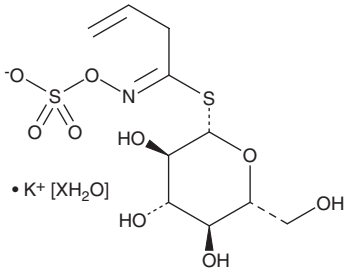
PRODUCT INFORMATION

Sinigrin (hydrate)

Item No. 26028

Formal Name:	1-[N-(sulfooxy)-3-butenimidate]1-thio-β-D-glucopyranose, monopotassium salt, hydrate
MF:	$C_{10}H_{16}NO_9S_2 \bullet K\ [XH_2O]$
FW:	397.5
Purity:	≥95%
UV/Vis.:	λ_{max}: 227 nm
Supplied as:	A solid
Storage:	-20°C
Stability:	≥2 years
Item Origin:	Plant/*Brassica juncea*



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

Laboratory Procedures

Sinigrin (hydrate) is supplied as a solid. A stock solution may be made by dissolving the sinigrin (hydrate) in the solvent of choice, which should be purged with an inert gas. Sinigrin (hydrate) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of sinigrin (hydrate) in these solvents is approximately 30 mg/ml.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of sinigrin (hydrate) can be prepared by directly dissolving the solid in aqueous buffers. The solubility of sinigrin (hydrate) in PBS (pH 7.2) is approximately 1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Sinigrin is a glucosinolate that has been found in *Brassica* and has diverse biological activities.[1-3] It inhibits NOD-like receptor protein 3 (NLRP3) inflammasome activation in ATP-stimulated, LPS-primed RAW 264.7 cells in a concentration-dependent manner.[1] Sinigrin (1-100 µg/ml) reduces LPS-induced production of nitric oxide (NO) and prostaglandin E_2 (PGE_2; Item No. 14010), as well as NF-κB activation, in RAW 264.7 cells. It induces cell cycle arrest at the G_0/G_1 phase in HepG2 hepatocellular carcinoma cells when used at concentrations of 0.1 and 0.5 mM.[2] Sinigrin (10 and 20 mg/kg) reduces increases in renal glomerular basement membrane thickness, as well as increases in systolic and diastolic blood pressure, in a rat model of hypertension induced by angiotensin II.[3]

References

1. Lee, H.-W., Lee, C.G., Rhee, D.-K., *et al.* Sinigrin inhibits production of inflammatory mediators by suppressing NF-κB/MAPK pathways or NLRP3 inflammasome activation in macrophages. *Int. Immunopharmacol.* **45**, 163-173 (2017).
2. Jie, M., Cheung, W.M., Yu, V., *et al.* Anti-proliferative activities of sinigrin on carcinogen-induced hepatotoxicity in rats. *PLoS One* **9(10)**, e110145 (2014).
3. Cong, C., Yuan, X., Hu, Y., *et al.* Sinigrin attenuates angiotensin II-induced kidney injury by inactivating nuclear factor-κB and extracellular signal-regulated kinase signaling *in vivo* and *in vitro*. *Int. J. Mol. Med.* **48(2)**, 161 (2021).

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