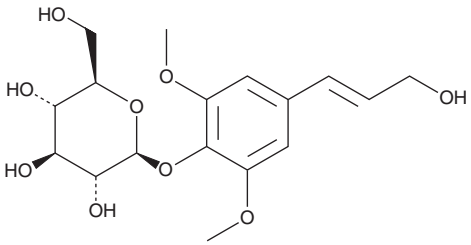
PRODUCT INFORMATION

Syringin

Item No. 22387

CAS Registry No.:	118-34-3
Formal Name:	4-[(1E)-3-hydroxy-1-propen-1-yl]-2,6-dimethoxyphenyl-β-D-glucopyranoside
Synonyms:	Eleutheroside B, NSC 287441
MF:	$C_{17}H_{24}O_9$
FW:	372.4
Purity:	≥98%
UV/Vis.:	λ_{max}: 221, 266 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



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

Laboratory Procedures

Syringin is supplied as a crystalline solid. A stock solution may be made by dissolving the syringin in the solvent of choice, which should be purged with an inert gas. Syringin is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of syringin in ethanol is approximately 1 mg/ml in DMSO and approximately 30 mg/ml in DMF.

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 syringin can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of syringin in PBS, pH 7.2, is approximately 1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Syringin is a phenylpropanoid glycoside first isolated from *A. senticosus* that enhances acetylcholine release in pancreatic cells leading to an increase in insulin release through the muscarinic M_3 receptor.[1,2] Syringin dose-dependently (50, 75, and 100 µg/kg, i.v.) decreases plasma glucose levels and increases insulin-like immunoreactivity and C-peptide in rats, and these effects last at least 60 minutes. In a rat model of type 1 diabetes, it decreases plasma glucose and increases β-endorphin release from the adrenal medulla.[3] Syringin increases autophagy through AMP-activated protein kinase α (AMPKα) activation concomitant with preventing the progression of cardiac hypertrophy in mice following aortic banding.[4] It also has immunomodulatory effects, likely due to its metabolite sinapyl alcohol.[5]

References

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2. Liu, K.Y., Wu, Y.-C., Liu, I.-M., *et al. Neurosci. Lett.* **434(2)**, 195-199 (2008).
3. Niu, H.-S., Hsu, F.-L., Liu, I.-M., *et al. Horm. Metab. Res.* **39(12)**, 894-898 (2007).
4. Li, F., Zhang, N., Wu, Q., *et al. Int. J. Mol. Med.* **39(1)**, 199-207 (2017).
5. Choi, J., Shin, K.-M., Park, H.-J., *et al. Planta Med.* **70(11)**, 1027-1032 (2004).

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