## Cholestyramine

MedChemExpress

Cat. No.:	HY-104081		
CAS No.:	11041-12-6		
Target:	Others		
Pathway:	Others		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month

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# SOLVENT & SOLUBILITY

In Vitro	1M HCl : < 1 mg/mL (insoluble) DMSO : < 1 mg/mL (insoluble or slightly soluble) H <sub>2</sub> O : < 0.1 mg/mL (insoluble)
In Vivo	1. Add each solvent one by one: 0.5% CMC-Na/saline water Solubility: 60 mg/mL (Infinity mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIV	
Description	Cholestyramine (Colestyramine) is a bile acid binding resin and can inhibit intestinal bile acid absorption which results in the increasing bile acid synthesis from cholesterol.
In Vitro	Cholestyramine (0.1-50 µg/mL) produced the most dramatic results after a 24-hour exposure; an efflux rate of 65% compared with control cells. Cholestyramine is an anion-exchange resin and is insoluble in water. alcohol, chloro-form, and ether. For the assay, cholestyramine is initially wetted with a small amount of DMSO further diluting with media. A blank sample prepared with dimethylsulfoxide DMSO without cholestyramine displayed no differences from the control samples [3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Cholestyramine is a bile acid binding resin and can inhibit intestinal bile acid absorption which results in the? increasing bile acid synthesis from cholesterol <sup>[1]</sup> . Results reveal that GSPE treatment alone, and co-administration with Cholestyramine, regulate BA, cholesterol and TG metabolism differently compare to Cholestyramine administration alone. Notably, GSPE decreases intestinal apical sodium-dependent bile acid transporter (Asbt) gene expression, while Cholestyramine significantly induces expression. Administration with GSPE or Cholestyramine robustly induces hepatic BA biosynthetic gene expression, especially cholesterol 7α-hydroxylase (Cyp7a1), compare to control, while co-administration further enhances expression. Treatment with Cholestyramine induces both intestinal and hepatic cholesterologenic gene expression, while co-administration with GSPE attenuates the Cholestyramine-inducing increase in the liver but not in the intestine. Cholestyramine also induces hepatic lipogenic gene expression, which is attenuated by co-administration with GSPE <sup>[2]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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### PROTOCOL

#### Animal Administration <sup>[2]</sup>

Mice are purchased at 7 weeks of age and allowed to acclimate for one week. At 8-weeks of age the mice are given either a control or a 2% Cholestyramine-supplementing diet for 4 weeks (n=18 per group). Body weight for each mouse is recorded weekly. After 4 weeks, the mice in each group are randomly assigned to one of two treatment groups and orally gavaged with either vehicle (water) or GSPE (250 mg/kg) and terminated 14 hours later (n=9 per experimental group). The four treatment groups are as follows: 1. CON: Control diet for 4 weeks following by oral gavage with vehicle (water) for 14 hrs; 2. GSPE: Control diet for 4 weeks following by oral gavage with 250 mg/kg GSPE for 14 hrs; 3.Cholestyramine 2% Cholestyramine-supplementing diet for 4 weeks following by oral gavage with vehicle for 14 hrs; and 4. Cholestyramine+GSPE: 2% cholestyramine-supplementing diet for 4 weeks following by oral gavage with vehicle for 14 hrs; and 4. Cholestyramine+GSPE: 2% cholestyramine-supplementing diet for 4 weeks following by oral gavage with vehicle for 14 hrs; and 4. Cholestyramine+GSPE: 2% cholestyramine-supplementing diet for 4 weeks following by oral gavage with vehicle for 14 hrs; and 4. Cholestyramine+GSPE: 2% cholestyramine-supplementing diet for 4 weeks following by oral gavage with vehicle for 14 hrs; and 4. Cholestyramine+GSPE: 2% cholestyramine-supplementing diet for 4 weeks following by oral gavage with vehicle for 14 hrs; and 4. Cholestyramine+GSPE: 2% cholestyramine-supplementing diet for 4 weeks following by oral gavage with 250 mg/kg GSPE for 14 hrs. Blood is collected from the orbital plexus under isoflurane anesthesia, and intestines and livers are snap-frozen in liquid nitrogen and stored at -80°C until use. At the start of the 14 hr experiment mice are placed into clean cages, and feces are manually collected at the end of the study<sup>[2]</sup>.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

#### **CUSTOMER VALIDATION**

- Microbiome. 2023 May 2;11(1):96.
- Cellulose. 27, 4019-4028 (2020).
- J Appl Toxicol. 2022 Nov 3.
- SSRN. 2023 Aug 28.

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#### REFERENCES

[1]. Maugeais C, et al. rHDL administration increases reverse cholesterol transport in mice, but is not additive on top of ezetimibe or cholestyramine treatment. Atherosclerosis. 2013 Jul;229(1):94-101.

[2]. Rebecca M. Heidker, et al. Grape Seed Procyanidins and Cholestyramine Differentially Alter Bile Acid and Cholesterol Homeostatic Gene Expression in Mouse Intestine and Liver. PLoS One. 2016; 11(4): e0154305.

[3]. J M Pruckler, et al. Use of a human microvascular endothelial cell line as a model system to evaluate cholesterol uptake. Pathobiology. 1993;61(5-6):283-7.

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

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