Product Data Sheet

Product Name: Colestyramine (Cholestyramine resin)
Cat. No.: GC32441

Chemical Properties

Cas. No.	11041-12-6		
SMILES	C[N+](C)(C)CC1=CC=C(C(CC(C)C2=CC=CC=C2)CC)C=C1.CC(CC)C.[Cl-].[n]		
Formula	$C_{27}H_{47}N$	M.Wt	385.67
Solubility	DMSO : < 1 mg/mL (insoluble or slightly soluble);Water : < 0.1 mg/mL (insoluble)	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure			

Protocol

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Animal experiment:

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 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[2].

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.

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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.

Background

Colestyramine (Cholestyramine) is a bile acid binding resin and can inhibit intestinal bile acid absorption which results in the increasing bile acid synthesis from cholesterol.

Colestyramine (Cholestyramine) is a bile acid binding resin and can inhibit intestinal bile acid absorption which results in the increasing bile acid synthesis from cholesterol[1]. Results reveal that GSPE treatment alone, and co-administration with Colestyramine (CHY), regulate BA, cholesterol and TG metabolism differently compare to Colestyramine (CHY) administration alone. Notably, GSPE decreases intestinal apical sodium-dependent bile acid transporter (Asbt) gene expression, while Colestyramine (CHY) significantly induces expression. Administration with GSPE or Colestyramine (CHY) robustly induces hepatic BA biosynthetic gene expression, especially cholesterol 7α-hydroxylase

(Cyp7a1), compare to control, while co-administration further enhances expression. Treatment with Colestyramine (CHY) induces both intestinal and hepatic cholesterologenic gene expression, while co-administration with GSPE attenuates the Colestyramine (CHY)-inducing increase in the liver but not in the intestine. Colestyramine (CHY) also induces hepatic lipogenic gene expression, which is attenuated by co-administration with GSPE[2].

[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.

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