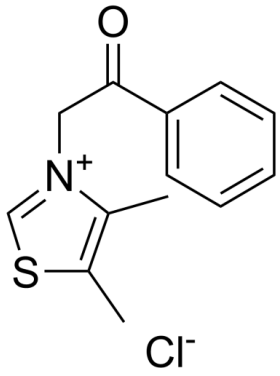
Data Sheet

Product Name:	Alagebrium chloride
Cat. No.:	CS-0024653
CAS No.:	341028-37-3
Molecular Formula:	$C_{13}H_{14}ClNOS$
Molecular Weight:	267.78
Target:	Endogenous Metabolite
Pathway:	Metabolic Enzyme/Protease
Solubility:	DMSO : ≥ 25 mg/mL;H_2O : 50 mg/mL (ultrasonic)



BIOLOGICAL ACTIVITY:

Alagebrium chloride (ALT711) is an **advanced glycation end product** (**AGE**) inhibitor. IC50 & Target:AGE[1] ***In Vitro:*** Alagebrium chloride is an **advanced glycation end product** (**AGE**) inhibitor. Endothelial cell (EC) proliferation is increased for all groups receiving Alagebrium (ALT-711), particularly when seeded on matrix from the AAo of obese (ZO) and diabetic (ZD) rats[2]. ***In Vivo:*** Blood pressure is not affected by treatment with Alagebrium. In diabetic RAGE apoE double-KO mice, treatment with Alagebrium is associated with a modest reduction in renal mass and reduces hyperfiltration compare with nontreated mice. Treatment with Alagebrium in diabetic RAGE apoE double-KO mice is associated with a further reduction in glomerular collagen IV levels, approaching levels observed in control mice[1]. Body weight, heart rate (HR), and mean blood pressure (BP) are similar in Zucker lean (ZL), obese (ZO), and diabetic (ZD) groups in the absence or presence of Alagebrium (ALT-711). Alagebrium increases blood flow (BF) in ZO rats but reduces distal vascular resistance in ZD rats. A decrease in neointimal hyperplasia (NH) intrastrut thickness as a function of local radius is found in all groups with Alagebrium treatment. A significant increase in TGF-β expression is also found in the AAo of ZL rats treated with Alagebrium[2].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [2]**Human aortic endothelial cells** (HAECs) are seeded on decellularized matrices derived from the abdominal aorta (AAo) of Zucker lean (ZL), obese (ZO), and diabetic (ZD) rats **with or without Alagebrium** (ALT-711) (**20 μg/mL** in Dulbecco's PBS with 1× antibiotic-antimycotic). Experiments are performed when **cells reach 80 to 90% confluence**. Flow chambers are sealed to the HAEC monolayers via a vacuum network. Flow is driven by a Masterflex L/S peristaltic pump in a humidified chamber heated to 37°C for 4 h. Leibovitz-15 medium, supplemented with 10% FBS, endothelial BulletKit, and 1× antibiotic-antimycotic solution, is used as the flow medium to maintain pH in the absence of CO_2[2]. **Animal Administration:** [1]***RAGE apoE*** **mice** are randomized to be treated with **Alagebrium** (**1 mg/kg/day by gavage**), or no treatment (n=20/group). After 20 weeks of diabetes, mice are placed into individual metabolic cages for 24 h and urine is collected. Body weight as well as fluid and food intake are recorded. Urinary albumin excretion is estimated in urine samples by a mouse albumin enzyme-linked immunosorbent assay (ELISA) kit according to the kit protocol. Urinary and serum creatinine concentrations are measured by high-performance liquid chromatography (HPLC). Systolic blood pressure is assessed by a noninvasive tail cuff method in conscious mice at the end of the study[1].

References:

[1]. Watson AM, et al. Alagebrium reduces glomerular fibrogenesis and inflammation beyond preventing RAGEactivation in diabetic apolipoprotein E

knockout mice. Diabetes. 2012 Aug;61(8):2105-13.

[2]. Wang H, et al. Alagebrium inhibits neointimal hyperplasia and restores distributions of wall shear stress by reducing downstream vascular resistance in obese and diabetic rats. Am J Physiol Heart Circ Physiol. 2015 Oct;309(7):H1130-40.

CAIndexNames:

Thiazolium, 4,5-dimethyl-3-(2-oxo-2-phenylethyl)-, chloride (1:1)

SMILES:

O=C(C1=CC=CC=C1)C[N+]2=CSC(C)=C2C.[Cl-]

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