

**UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549**

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 OR 15(d) of The Securities Exchange Act of 1934

Date of Report (Date of earliest event reported) October 18, 2010

Spherix Incorporated

(Exact name of registrant as specified in its charter)

(State or other jurisdiction of incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)
Delaware	0-5576	52-0849320
(Address of principal executive offices)		(Zip Code)
6430 Rockledge Drive, Suite 503, Bethesda, Maryland		20817
Registrant's telephone number, including area code		<u>301-897-2540</u>

(Former name or former address, if changed since last report.)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

- Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
- Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
- Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
- Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 7.01 Regulation FD Disclosure.

On November 3, 2010, Dr. Robert A. Lodder, President, and Ms. Leisa Dennehy, Commercial and Corporate Development Advisor, of Spherix Incorporated (the "Company"), will deliver a company presentation to various prospective investors at the Windhover's Therapeutic Area Partnerships Conference in Boston, MA. The presentation is available on the Company's website at www.spherix.com, is attached hereto as Exhibit 99.1, and is incorporated herein by reference.

Section 9 – Financial Statements and Exhibits

Item 9.01 Financial Statements and Exhibits

Exhibits

99.1 Spherix Incorporated Overview Presentation, October 18, 2010

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Spherix Incorporated
(Registrant)

By:

/s/ Robert L. Clayton
Robert L. Clayton
CFO

Date: October 18, 2010



TAGATOSE NON-CONFIDENTIAL OVERVIEW

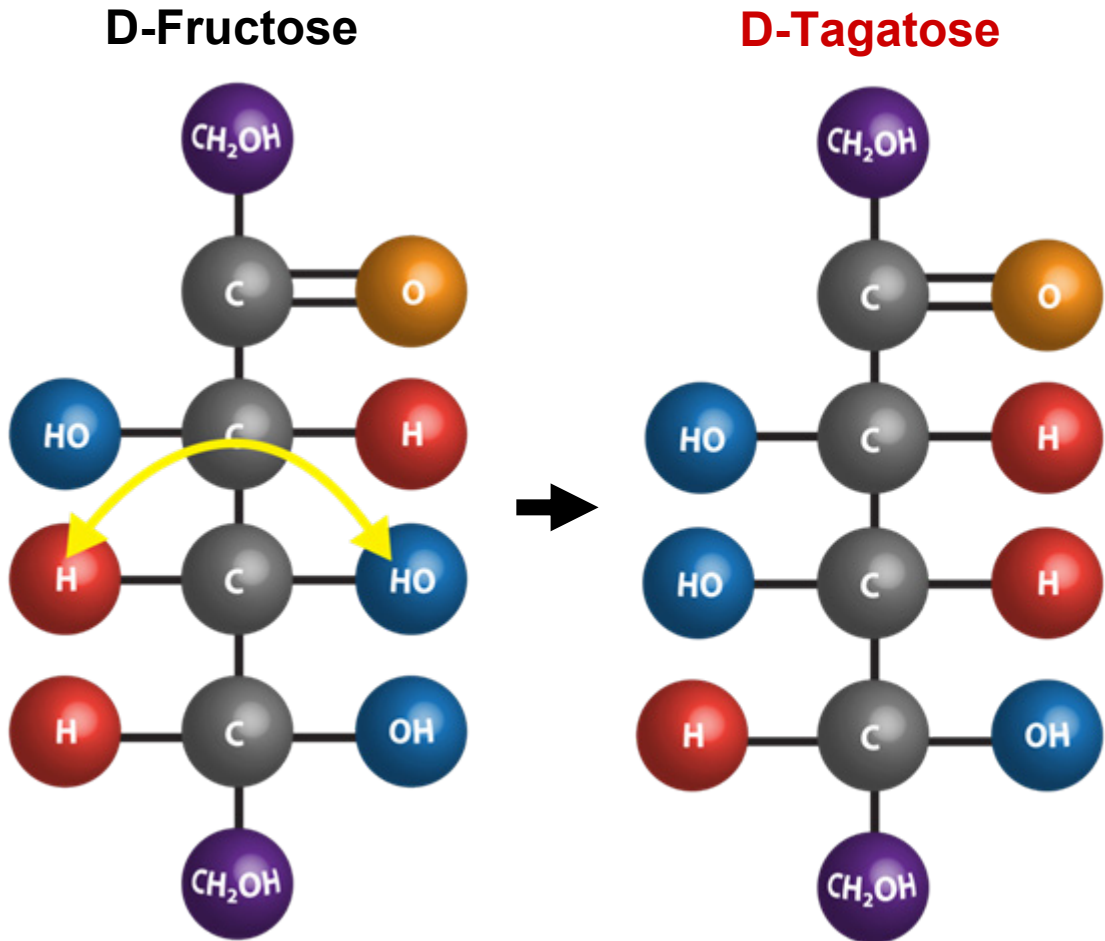
October 18, 2010

This presentation contains forward-looking statements that involve risks and uncertainties, including those described in the Company's Annual Report for the year ended December 31, 2009 filed on Form 10-K.

- **Spherix (NASDAQ: SPEX) has two subsidiaries:**
 - **Spherix Consulting: Scientific consulting on food and drug uses for US and overseas clients**
 - **Biospherics: Pharmaceutical development division**
- **Biospherics is focused on development of tagatose for the treatment of diabetes and metabolic disorders**
 - **Phase 3 trial as monotherapy in ‘mild’ Type 2 Diabetes reported Oct 2010**
 - **Phase 2 dose ranging study in Type 2 Diabetes to complete Dec10**
 - **Phase 2 dose ranging study in Hypertriglyceridemia expected to start in 2011**

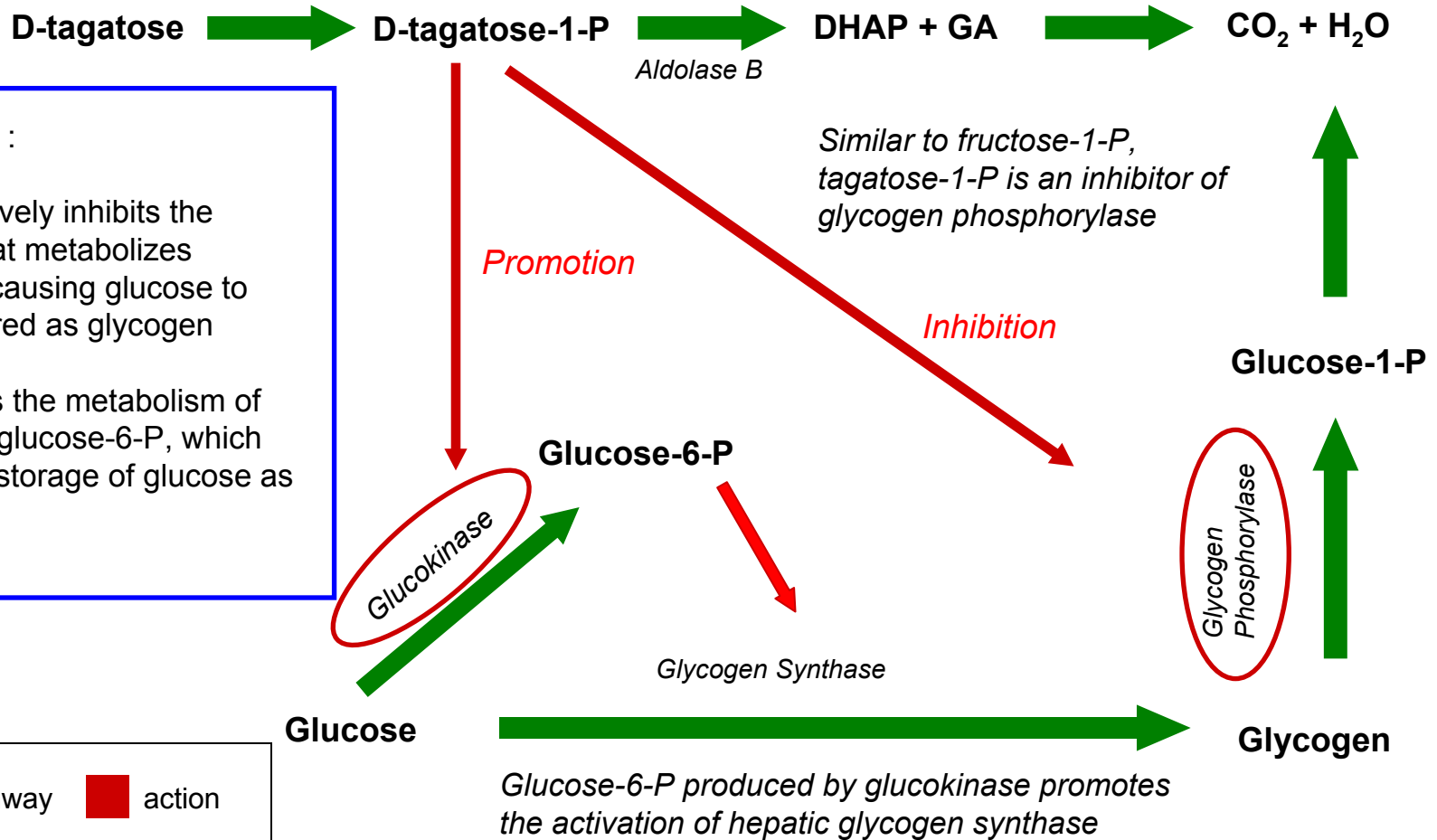
What is D-Tagatose?

- Naturally occurring L-epimer of D-fructose with an inversion at C4
- Spherix used chiral carbohydrate research to create L-sugars that are not metabolized, but retain sweetness
- Does not stimulate insulin production



Tagatose Glycemic Control MOA in the Liver

Metabolism of D-tagatose in liver is identical to that of fructose, but the cleavage of tagatose-1-P occurs at only about half the rate of that of fructose-1-P



Tagatose

- **May provide a safety advantage over current agents**
 - **Approved as GRAS substance by the FDA and WHO**
- **Provides glycemic control through a mechanism of action unlike any agent currently-marketed in the US**
 - **“Sugar blocker” may modify post-prandial glucose level**
- **Does not cause stimulation of beta cells or insulin secretion**
- **May have prebiotic benefits and other metabolic benefits**

Limitations of Current Oral Diabetes Therapies

Class	A1C Reduction	Fasting vs PPG	Hypo-glycemia	Weight Change	Dosing (times/day)	Other safety issues
Metformin	1.5	Fasting	No	Neutral	2	GI, lactic acidosis
Insulin, long acting	1.5 - 2.5	Fasting	Yes	Gain	1, Injected	
Insulin, rapid acting	1.5 - 2.5	PPG	Yes	Gain	1-4, Injected	
Sulfonylureas	1.5	Fasting	Yes	Gain	1	Allergies, secondary failure
Thiazolidinediones	0.5 - 1.4	Fasting	No	Gain	1	Edema, CHF, bone fractures
GLP-1 agonists (short)	0.5 - 1.0	PPG	No	Loss	2, Injected	GI, ?pancreatitis, ARF
Repaglinide	1 - 1.5	Both	Yes	Gain	3	
Nateglinide	0.5 - 0.8	PPG	Rare	Gain	3	
α -Glucosidase inhibitor	0.5 - 0.8	PPG	No	Neutral	3	GI
Amylin mimetics	0.5 - 1.0	PPG	No	Loss	3, Injected	GI
DPP-4 inhibitors	0.6 - 0.8	Both	No	Neutral	1	?pancreatitis
Bile acid sequestrant	0.5	Fasting	No	Neutral	1-2	GI
Bromocriptine	0.7	PPG	No	Neutral	1	GI
Long-acting GLP-1 agonist	~1.5	Both	No	Loss	1 or less, Injected	GI, ?pancreatitis, ?MTC, ?ARF

- **Diabetes affects >24 million people in the U.S. and ~285 million adults worldwide, and growing significantly^{1,2}**
 - **90-95% of those affected have Type 2 diabetes**
- **5th leading cause of death by disease in the U.S.**
- **\$175 billion annually in direct & indirect medical expenses³**
- **Poorly controlled even with aggressive intervention**
 - **~60% of diabetics don't achieve target blood sugar levels with their current treatment⁴**
- **Multiple co-morbidities**
 - **85% obesity, cardiovascular problems, renal disease, ophthalmic complications, etc.**
- **Up to 57 million Americans have “pre-diabetes”**

¹ International Diabetes Federation Diabetes Atlas. <http://www.diabetesatlas.org/content/some-285-million-people-worldwide-will-live-diabetes-2010>

² Diabetes Statistics. American Diabetes Association. <http://www.diabetes.org/diabetes-basics/diabetes-statistics/>

³ Direct and Indirect Costs of Diabetes in the United States. American Diabetes Association. <http://www.diabetes.org/how-to-help/action/resources/cost-of-diabetes.html>

⁴ Saydah SH, Fradkin J and Cowie CC. Poor control of risk factors for vascular disease among adults with previously diagnosed diabetes. *JAMA*. 2004;291:335-42.

Phase 3 Clinical Trial, Tagatose NEET Study (Protocol 70971-004)

- **Objective**
 - Evaluate effect of 15-gram, *t.i.d* dose of D-tagatose on glycemic control in subjects with Type 2 diabetes not controlled by diet & exercise (randomization HbA1c= 6.6 – 9.0)
- **Design**
 - Multi-center, double blind, randomized, parallel group
 - 494 patients randomized
 - 34 sites in US; 23 sites in India
 - Powered to detect 0.5 percentage point change in HbA1c
- **Clinical Endpoints**
 - Primary: HbA1c
 - Secondary: glucose, insulin, lipid profiles, body weight

Statistically significant reduction of HbA1c in US ITT and PP at all time points

Reduction in HbA1c Over Time

Patient population	2 months	6 months	10 months
U.S. PP	-0.4* (n=51)	-0.6* (n=29)	-1.1* (n=20)
U.S. ITT LOCF	-0.3* (n=100)	-0.3* (n=101)	-0.4* (n=101)
India PP	-0.1 (n=150)	0.0 (n=117)	-0.2 (n=72)
India ITT LOCF	-0.2 (n=253)	-0.1 (n=254)	-0.2* (n=254)
Global PP	-0.2 (n=201)	-0.2* (n=146)	-0.4* (n=92)
Global ITT LOCF	-0.2* (n=353)	-0.2* (n=355)	-0.2* (n=355)
Global ITT (7.5<HbA1c<9.0)	-0.3 (n=175)	0.1 (n=134)	-0.5* (n=92)

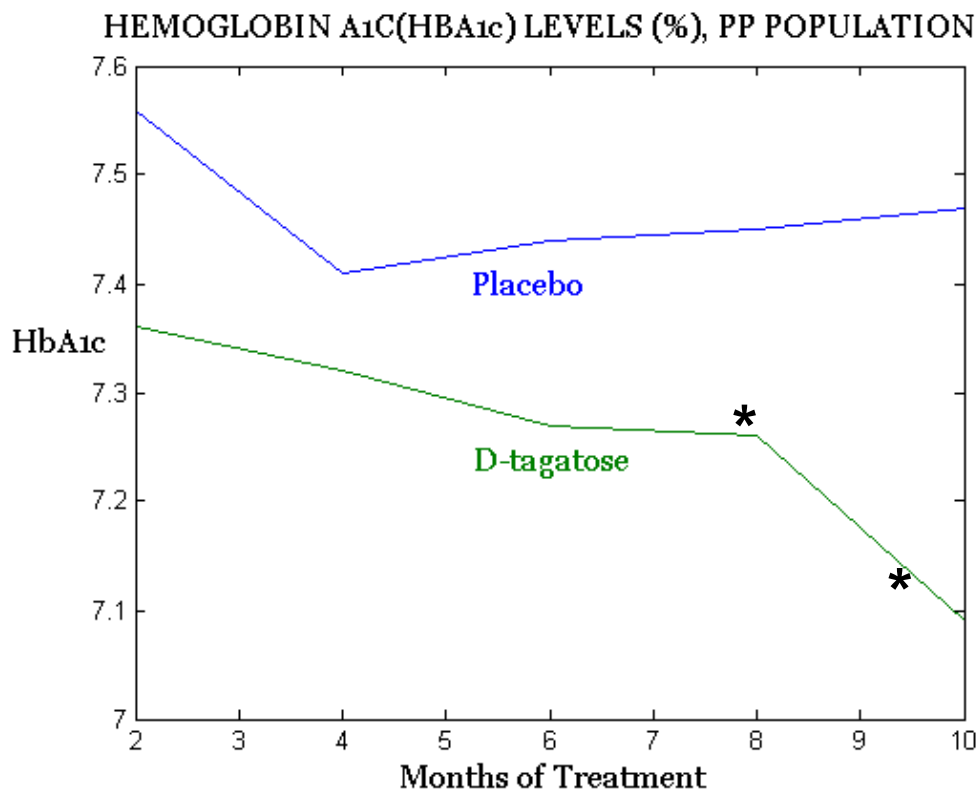
PP = Per-Protocol; ITT = Intent-to-Treat; LOCF = Last Observation Carried Forward

* p<0.05; all other figures do not have statistical significance

Effect on HbA1c: Global Per Protocol vs. Placebo

- **Statistically significant reduction vs placebo at 6 and 10 months**
 - Diet and exercise may be attributed to initial drop in the placebo group
- **Decreases in HbA1c in Type 2 diabetics are dependent on baseline HbA1c***

* Bloomgarden et al., *Diabetes Care*, Volume 29 Number 9 September 2006
- **Patients with HbA1c levels between 8.0% and 9.0% globally showed 0.7% reduction at 10 months of therapy**
 - Per protocol, n=30, p=0.09



* p<0.05

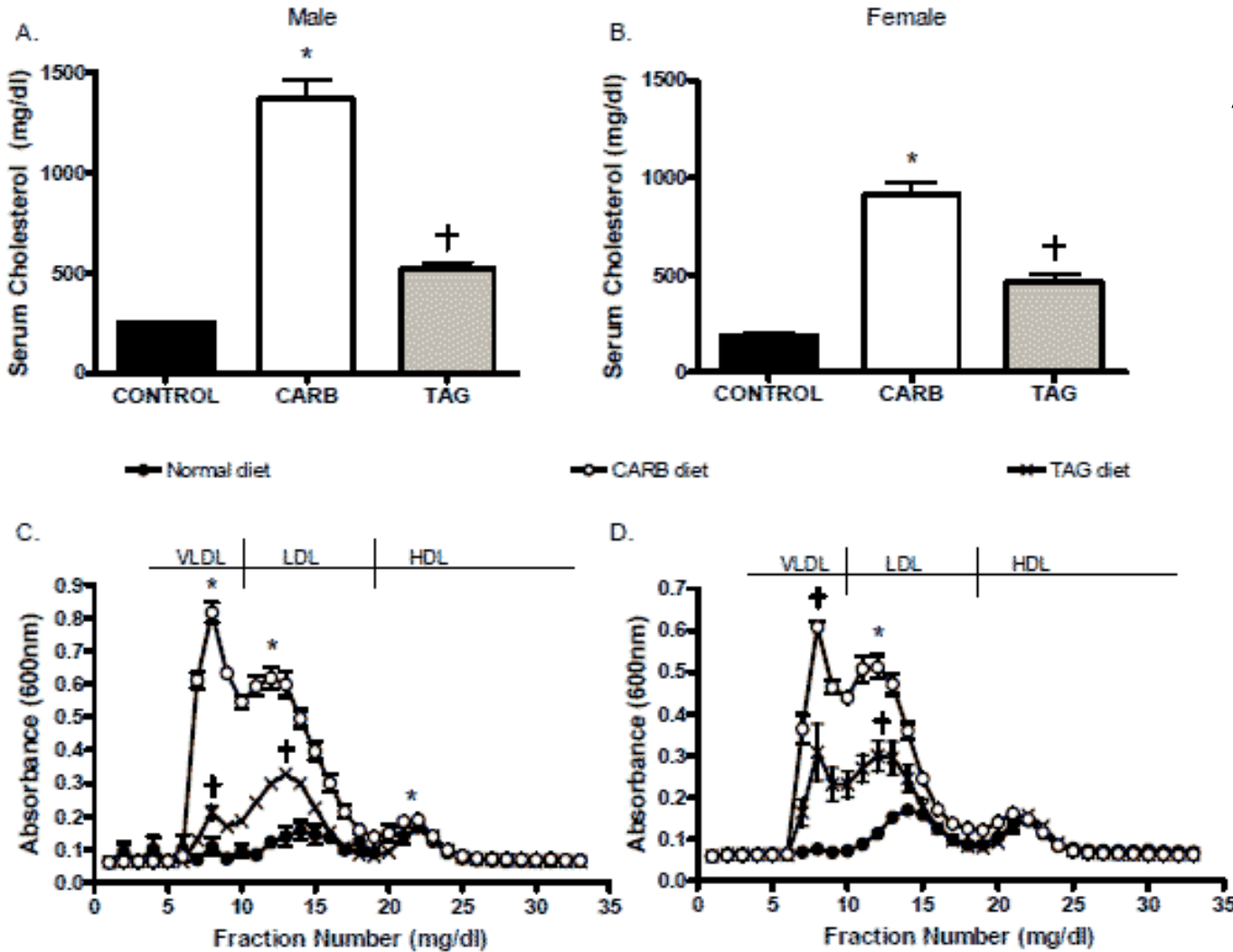
- Responder analysis demonstrated a high percentage of patients were able to reduce their HbA1c level to below 7.0 during treatment
 - Statistically significantly different from placebo
 - ADA recommends treatment when HbA1c is 7.0 or higher
- Difficult to gain additional lowering of HbA1c at such a low level

Final HbA1c Level	HbA1c < 6.5 %		HbA1c < 7.0%	
	Tagatose	Placebo	Tagatose	Placebo
Percent of Patients	24 %	11 %	58 %	26%
	<i>ns</i>		<i>p= 0.03</i>	

Interpret as: 60% of the patients achieve an HbA1c below 7% during treatment

- **Patients with ≥ 1 treatment-emergent adverse events in the active group (163) was comparable to the placebo group (166)**
 - **No serious adverse event deemed treatment related**
 - **No episodes of hypoglycemia or pancreatitis were reported among any trial subjects**
- **Limited size of patient population with abnormal triglycerides and BMI were not powered for significance in secondary endpoints**

Tagatose Effects on Triglycerides LDLr -/- Knockout Mice



Animal studies in a model of diet-induced atherosclerosis is suggested D-tagatose might reduce lipoproteins and atherosclerosis.

Tagatose Effects on Triglycerides LDLr -/- Mice (2)

Tagatose administration reduced triglycerides, adiposity and body mass.

Gender	Diet	Body weight (g)	Rate of body weight gain (g/week)	Tibialis anterior (mg)	Total triglycerides (mg/dl)	Adiposity index (%)
M	NORMAL	30.18 ± 0.36	0.28 ± 0.03	57.80 ± 3.50	110.3 ± 19.9	3.35 ± 0.27
	CARB	40.30 ± 1.78 *	0.97 ± 0.06	58.80 ± 2.73	822.4 ± 147.5 *	6.70 ± 0.77 *
	TAG	27.30 ± 0.42	0.17 ± 0.03	57.83 ± 2.20	162.3 ± 29.2	1.72 ± 0.13
F	NORMAL	24.43 ± 1.10	0.30 ± 0.05	35.25 ± 7.41	79.1 ± 15.5	2.71 ± 0.57
	CARB	30.56 ± 0.98 *	0.68 ± 0.05	43.14 ± 2.06	326.2 ± 37.3 *	5.39 ± 0.41 *
	TAG	24.06 ± 0.73	0.19 ± 0.02	42.40 ± 2.50	54.1 ± 8.0	1.60 ± 0.32

Data are mean ± SEM from N = 5/6 mice/group).

Near-Term Milestones

- **Report Phase 2 dose-finding data on diabetes, hypertriglyceridemia, body mass index** **4Q10**
- **Engage partner for D-tagatose in diabetes to continue development** ***Ongoing***
- **Perform additional animal studies on hypertriglyceridemia mechanism of action** **1Q11**
- **Begin Phase 2 study with D-Tagatose in hypertriglyceridemia** **1Q11**

- **Tagatose medical use focused on large and growing Type 2 diabetes and dyslipidemic markets**
- **Proof of concept demonstrated in human trials (diabetes) and animal models (triglycerides)**
- **Market position based on expected safety advantages, good tolerability and modest efficacy**
- **Interested in out-licensing commercial rights, outright sale of asset or entering a development agreement**

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