

**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) February 16, 2012

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.

Dr. Claire Kruger, Chief Executive Officer, and Dr. Robert Lodder, President, will be presenting to investors as part of the LHA, Inc. Life Sciences & Medical Technologies Virtual Conference on Thursday, February 16, 2012, at 12:30 p.m. Eastern time.. 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, February 16, 2012

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: February 16, 2012

Exhibit 99.1: Spherix Incorporated Overview Presentation, February 16, 2012



Spherix Incorporated (NASDAQ: SPEX)
February 16, 2012

Forward-Looking Statements

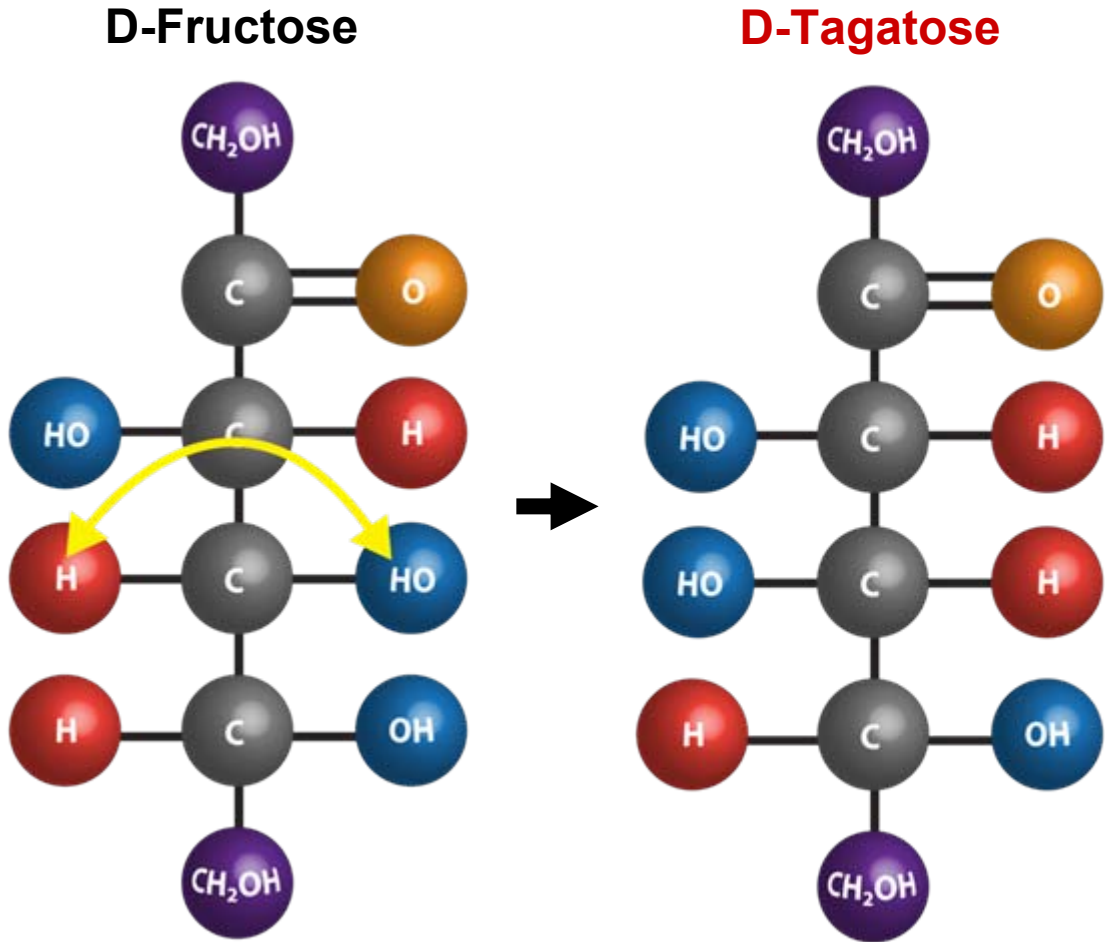
This presentation contains forward-looking statements made pursuant to provisions of Section 21E of the Securities Exchange Act of 1934. Investors are cautioned that such statements, including statements relating to planned clinical study design, regulatory and business strategies, plans and objectives of management and growth opportunities for existing or proposed products, constitute forward-looking statements which involve risks and uncertainties that could cause actual results to differ materially from those anticipated by the forward-looking statements. The risks and uncertainties include, without limitation, risks that product candidates may fail in the clinic or may not be successfully marketed or manufactured, we may lack financial resources to complete development of our products, the FDA may interpret the results of studies differently than us, competing products may be more successful, demand for new pharmaceutical products may decrease, the biopharmaceutical industry may experience negative market trends, our continuing efforts to develop products may be unsuccessful, our common stock could be delisted from the Nasdaq Capital Market, and other risks and challenges detailed in our filings with the U.S. Securities and Exchange Commission. You are cautioned not to place undue reliance on any forward-looking statements that speak only as of the date of this presentation. We undertake no obligation to publicly release the results of any revisions to these forward-looking statements that may be made to reflect events or circumstances that occur after the date of this presentation or to reflect the occurrence of unanticipated events.

- **Core expertise in scientific and technical aspects of drug and food development**
- **Operates two subsidiaries**
 - **Biospherics: Pharmaceutical development of D-tagatose and pipeline products**
 - **Dyslipidemias**
 - **Atherosclerosis**
 - **Metabolic Syndrome and Diabetes**
 - **Spherix Consulting: Scientific consulting on food and drug approvals for clients worldwide**

- **D-tagatose and SPX-106 show promise in multiple clinical applications**
 - **Hypertriglyceridemia (\$26 billion treatment market)**
 - **Diabetes (\$175 billion treatment market)**
- **Preclinical and Phase 2 data show significant lowering of triglycerides**
 - **Much faster and less expensive development pathway vs. diabetes**
 - **Clinical development program initiated**
- **Cash position sufficient to fund initial triglyceride studies**
- **Phase 3 study demonstrated statistically significant reductions in HbA1c in patients with mild Type 2 diabetes**
 - **Pursuing partnership(s) for further clinical development**

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
- Blocks lipid formation from carbohydrates
- Does not stimulate insulin production



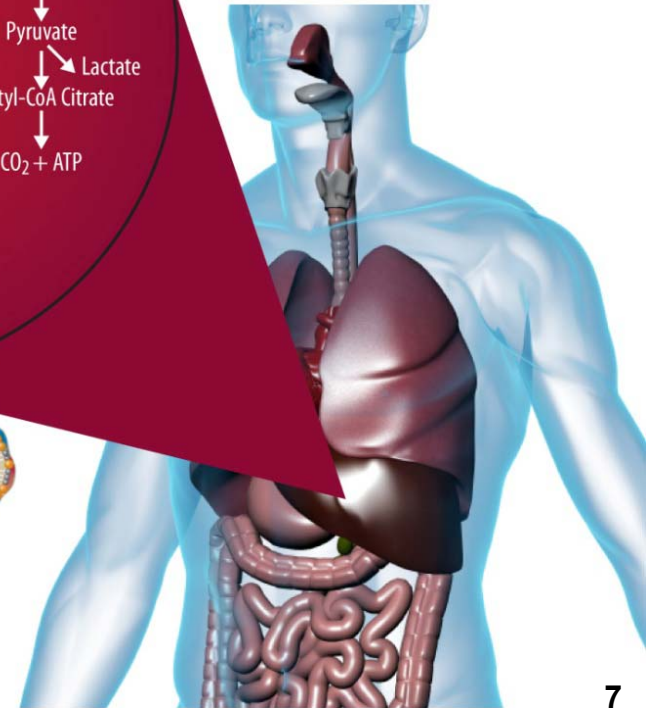
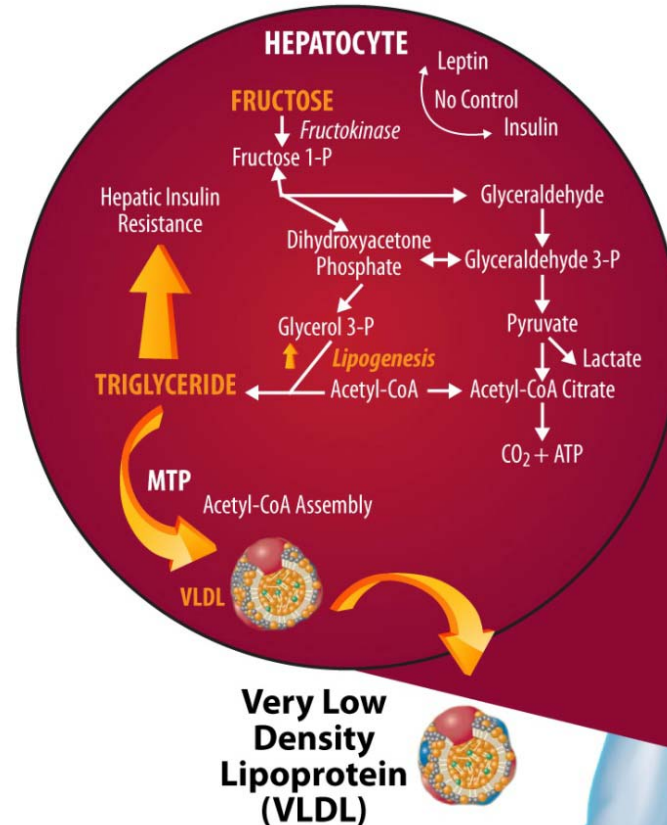
The D-Tagatose Difference

- **May provide a safety advantage over current agents**
 - **Approved as GRAS by FDA; approved as Novel Food in EU; ADI “not specified” by JECFA**
- **Provides lipoprotein and glycemic control through a mechanism of action unlike any agent currently marketed in the U.S.**
 - **“Sugar blocker” with potential fat, liver and gut mechanisms that may modify blood lipid and post-prandial glucose levels**

Proposed Mechanism of Action in Lowering VLDL and Triglycerides

- **D-Tagatose lowers blood lipoprotein and glucose levels**
 - Triglycerides reduced
 - VLDL reduced
 - LDL reduced
 - HDL essentially unchanged
 - TC reduced
- **D-Tagatose does not cause myopathy / rhabdomyolysis**
- **D-Tagatose does not stimulate insulin secretion**

Tagatose follows fructose pathway in the liver



Pharmaceutical Pipeline Compounds Formulated with D-Tagatose

| | Preclinical | Phase 1 | Phase 2 | Phase 3 | Marketed |
|----------------------------------------------------------------------|-------------|--------------------------|---------|--------------------------|----------|
| NEET Study (D-Tagatose)¹ <i>Type-2 diabetes</i> | | | | <i>Phase 3 Completed</i> | |
| Dose Ranging Study (D-Tagatose) <i>Type-2 diabetes</i> | | <i>Phase 2 Completed</i> | | | |
| SPX 7233801 <i>atherosclerosis</i> | | | | | |
| SPX 8522876 <i>dyslipidemias</i> | | | | | |
| SPX 10624258 “SPX-106” <i>metabolic syndrome</i> | | | | | |
| SPX 8818309 <i>obesity</i> | | | | | |
| SPX 8818440 <i>diabetes</i> | | | | | |

1. Proceeded directly from phase 1 to 3 due to FDA GRAS approval

- **Biospherics' goal to diversify pipeline by licensing in new drug candidates**
- **The Company has preclinical cardiovascular and metabolic drug candidates licensed from UKRF**
- **The Company is seeking and examining:**
 - **Later, clinical-stage compounds:**
 - **Phase 1 and Phase 2**
 - **Orphan drugs:**
 - **Developed specifically to treat a rare medical condition**
 - **7 years of data exclusivity in addition to patent life in US**
 - **In the US and EU, may be easier to gain marketing approval**

Metabolic syndrome is a combination of medical disorders that increase the risk of developing cardiovascular disease and diabetes. It affects one in five people, and prevalence increases with age.

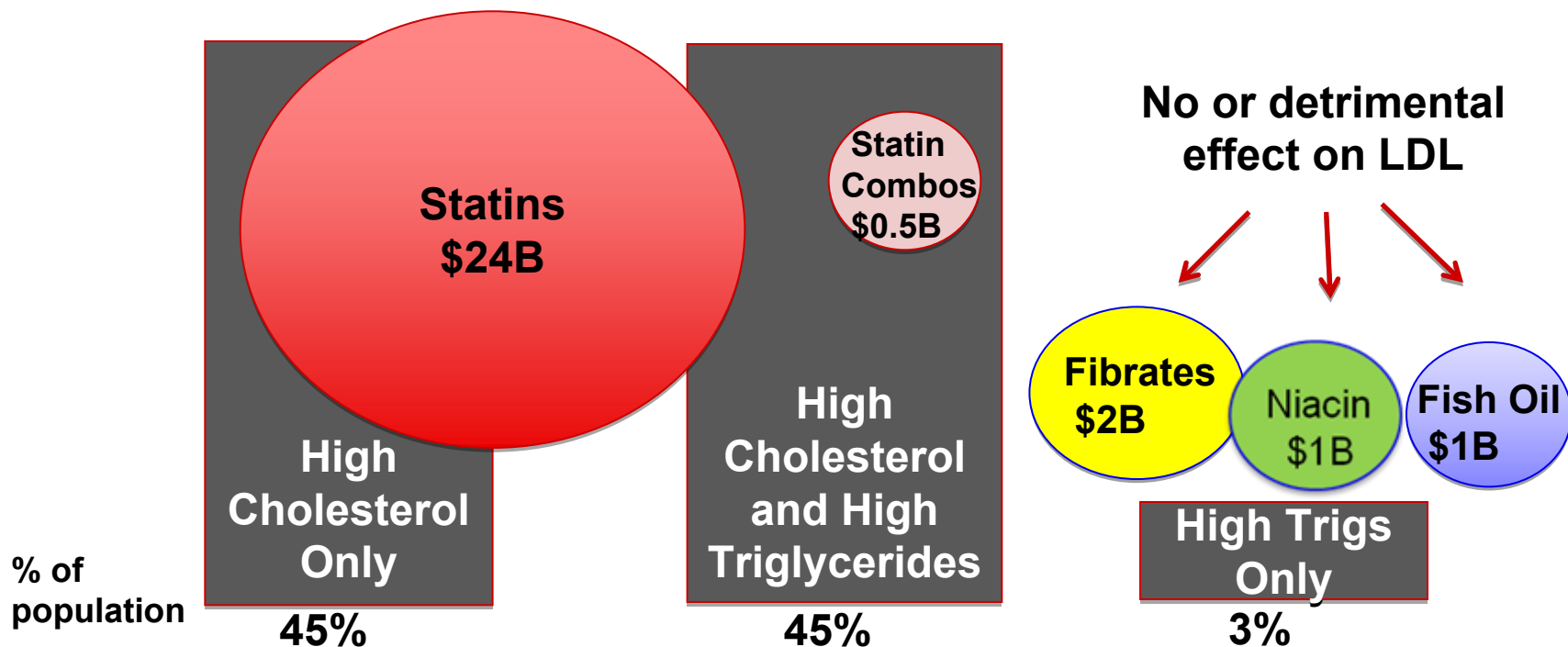
US National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP) (2001) requires at least three of the following:

- ✓ **central obesity: waist circumference \geq 102 cm or 40 inches (male), \geq 88 cm or 36 inches (female)**
- ✓ **dyslipidemia: TG \geq 1.7 mmol/L (150 mg/dl)**
- ✓ **dyslipidemia: HDL-C $<$ 40 mg/dL (male), $<$ 50 mg/dL (female)**
- ✓ **blood pressure \geq 130/85 mmHg**
- ✓ **fasting plasma glucose \geq 6.1 mmol/L (110 mg/dl)**

- **In the U.S. alone, more than 100 million people have elevated triglycerides, defined as more than 150 mg/dl**
- **Approximately 10 million are poorly served by current drug regimens**
- **The U.S. market for triglyceride-lowering drugs is in excess of \$4 billion**
- **The path to commercialization is shorter than for an oral antidiabetic drug**

Large and Growing Triglyceride Market Opportunity

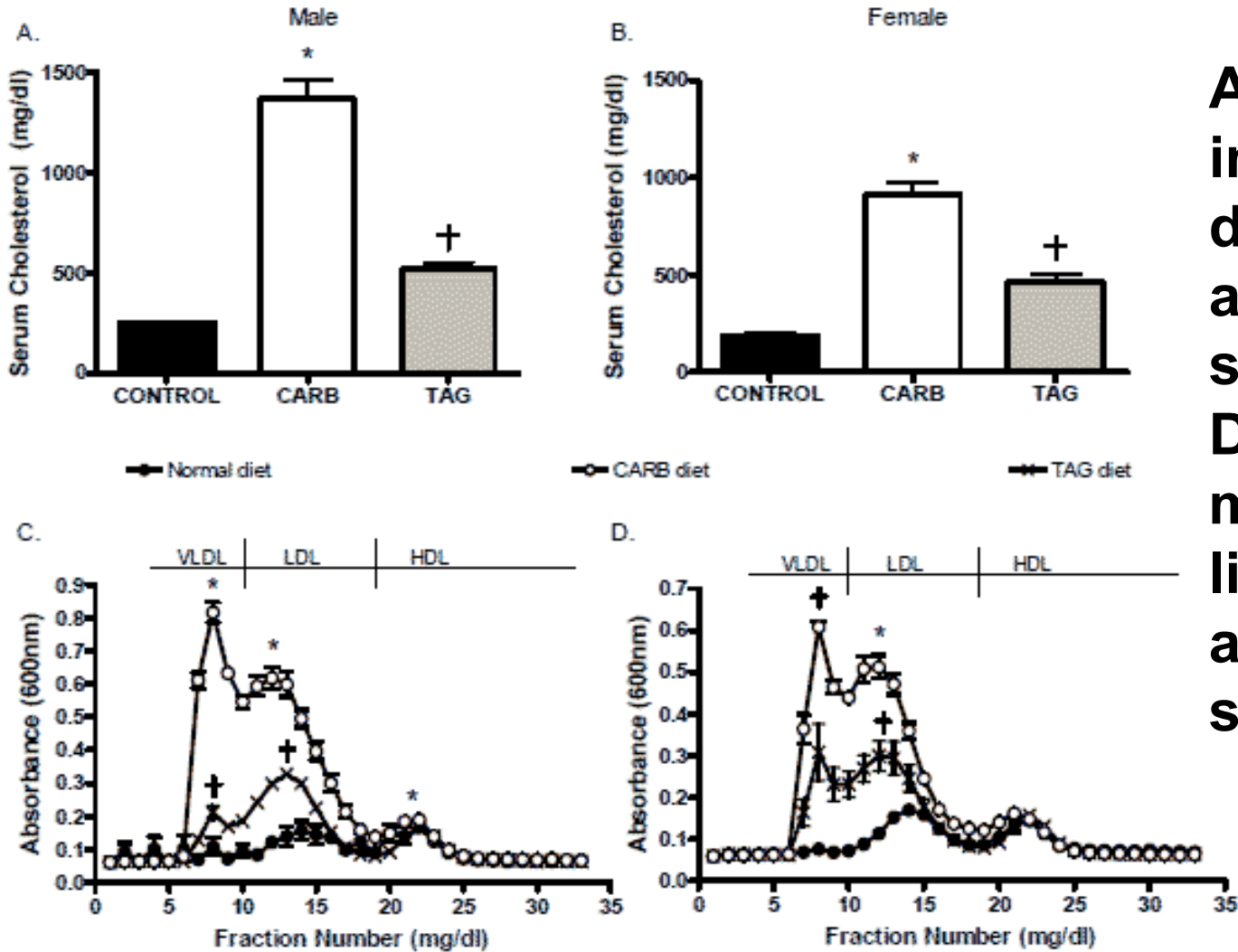
- A growing epidemic of metabolic syndrome and dyslipidemia supports blockbuster products in an area with limited competition
- Unmet needs still exist due to: adverse events with niacin, lack of robust cardiovascular benefit with fibrates, and unwanted lipid effects of raising the bad LDL cholesterol (fish oils) or decreasing the good HDL cholesterol (fibrates)
- Need new safe agents for use in patients with both high cholesterol and high triglycerides



The Metabolic Diseases Crisis- Hypertriglyceridemia

- **In the US alone, >100 million people have elevated triglycerides (> 150 mg/dl)**
- **Associated with other lipid abnormalities and the metabolic syndrome**
 - **abdominal obesity, insulin resistance, low high-density lipoprotein (HDL), and hypertension, which are linked to coronary artery disease**
- **Preclinical work and interim Phase 2 D-tagatose data support pursuit of this indication for development**
- **Good commercial opportunity! Large, but poorly-served market due to side effects of current products, with few competitors and low promotional intensity**

Tagatose Effects on Triglycerides LDLr Knockout Mice



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

Tagatose Effects on Triglycerides LDLr Mice (2)

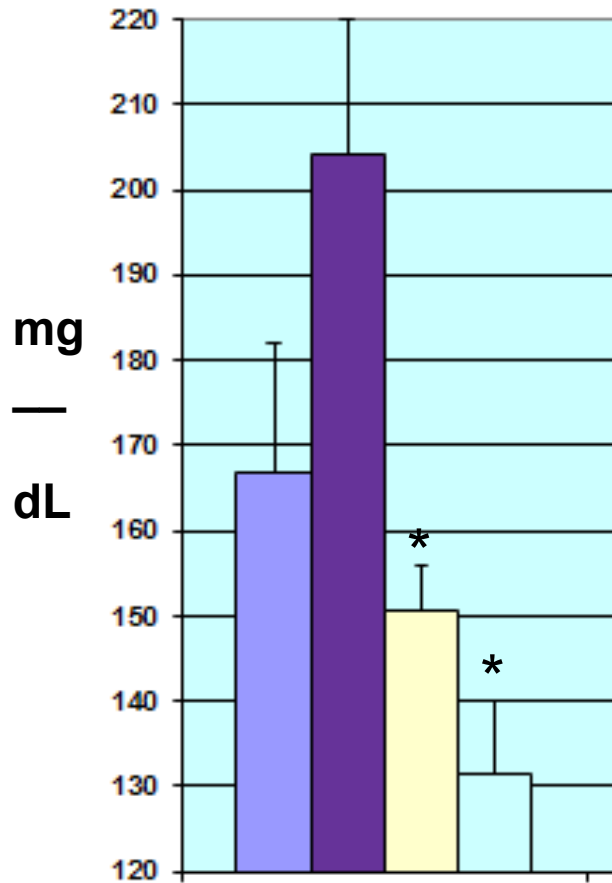
Tagatose administration reduced triglycerides, adiposity and body mass.

| 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).

LDLr -/- Mouse Treated with Sugar and Drug Mixtures for Three Weeks

Triglycerides



- Water
- Glu/Fruc
- Glu/Fruc/Tagatose
- Glu/Fruc/Tagatose / SPX-106

TRIGLYCERIDES

$p=0.03$ for tagatose vs. water
 $p=0.008$ for tagatose vs. glucose / fructose
 $p=0.001$ for tag/SPX106 vs. glucose / fructose

CHOLESTEROL

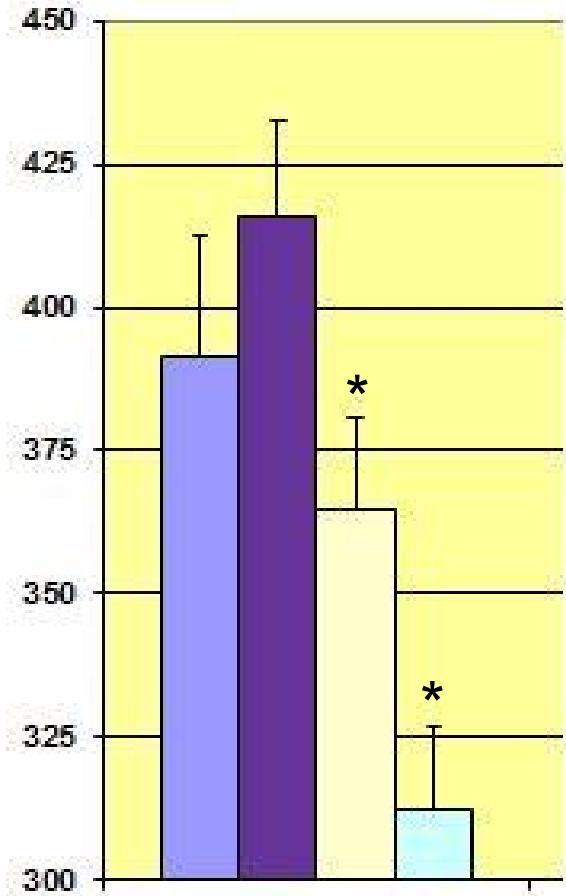
$p=0.05$ for gluc/fruc vs. tagatose
 $p=0.01$ for gluc/fruc vs. tagatose / SPX106

n=10 per group

Day 36

(SPX-106T = SPX-106 in D-tagatose)

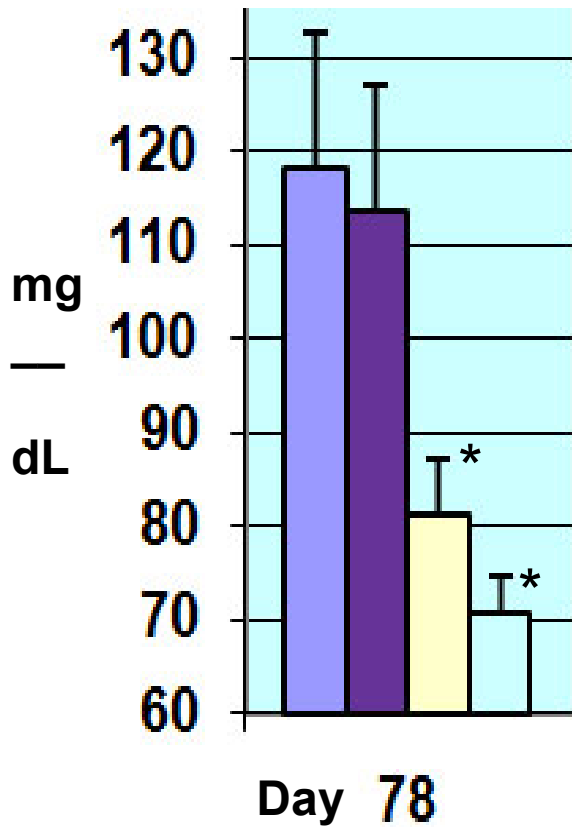
Total Cholesterol



Day 36

LDLr -/- Mouse Treated with Sugar and Drug Mixtures for Nine Weeks

Triglycerides



- Water
- Glu/Fruc
- Glu/Fruc/Tagatose
- Glu/Fruc / SPX-106T

TRIGLYCERIDES

- $p=0.035$ for tagatose vs. water
- $p=0.046$ for tagatose vs. glucose / fructose
- $p=0.009$ for SPX-106T vs. water
- $p=0.011$ for SPX-106T vs. glucose / fructose

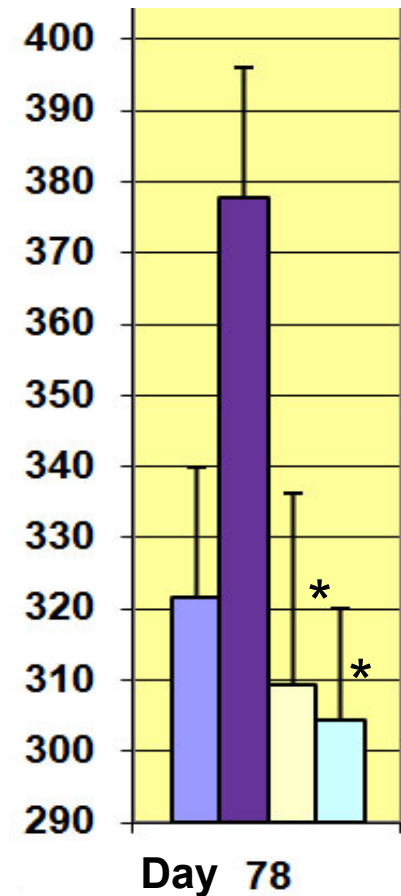
CHOLESTEROL

- $p=0.05$ for gluc/fruc vs. tagatose
- $p=0.01$ for gluc/fruc vs. SPX-106T

n=10 per group

(SPX-106T = SPX-106 in D-tagatose)

Total Cholesterol

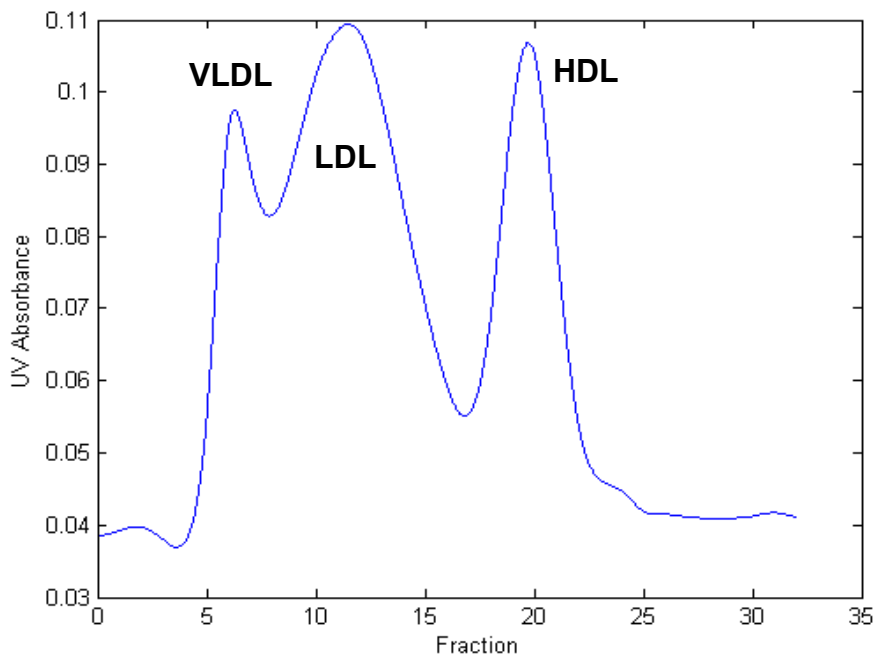


Peak Factor Analysis Results for the LDLr $-/-$ Mice

- The SPX-106T combination is the most effective of the treatments for lowering *triglycerides*.
 - The SPX-106 dose is responsible for lowering most of the triglycerides in the blood, followed by tagatose.
 - D-tagatose lowers triglycerides by -3.1 mg/dl per g/kg/dose of the sugar, while the SPX-106 lowers triglycerides by -372 mg/dl per g/kg/dose of the drug. Both glucose and fructose raise triglycerides.
- The SPX-106/tagatose combination is the most effective of the treatments for lowering *cholesterol*.
 - The SPX-106 concentration is responsible for lowering most of the cholesterol in the mice blood, followed by tagatose.
 - D-tagatose lowers cholesterol by -3.9 mg/dl per g/kg/dose of the sugar, while the SPX-106 lowers cholesterol by -629 mg/dl per g/kg/dose of the drug. Both glucose and fructose raise cholesterol.

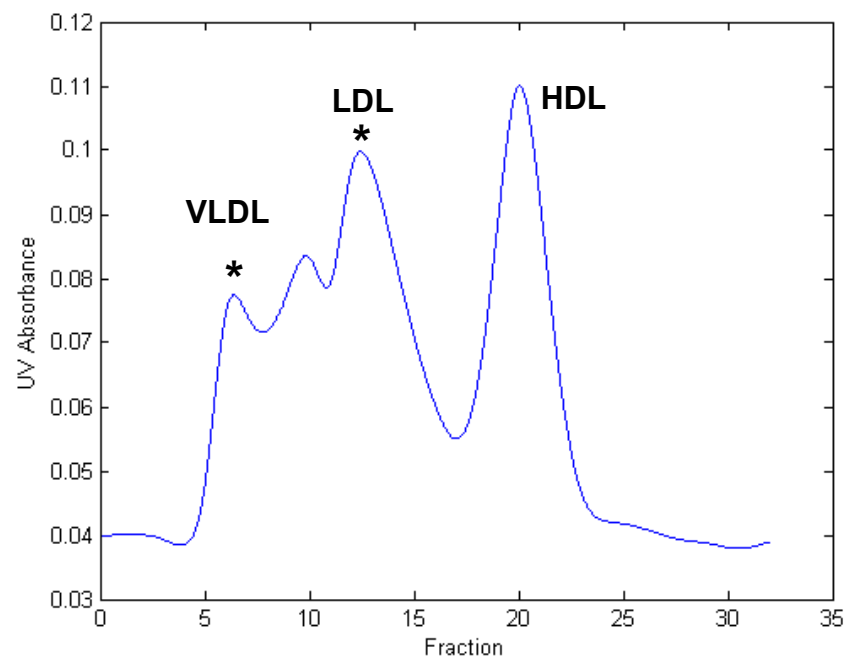
VLDL, LDL and HDL at 9 Weeks by FPLC and Enzymatic Assay in LDLr -/- Mice

Glucose & Fructose

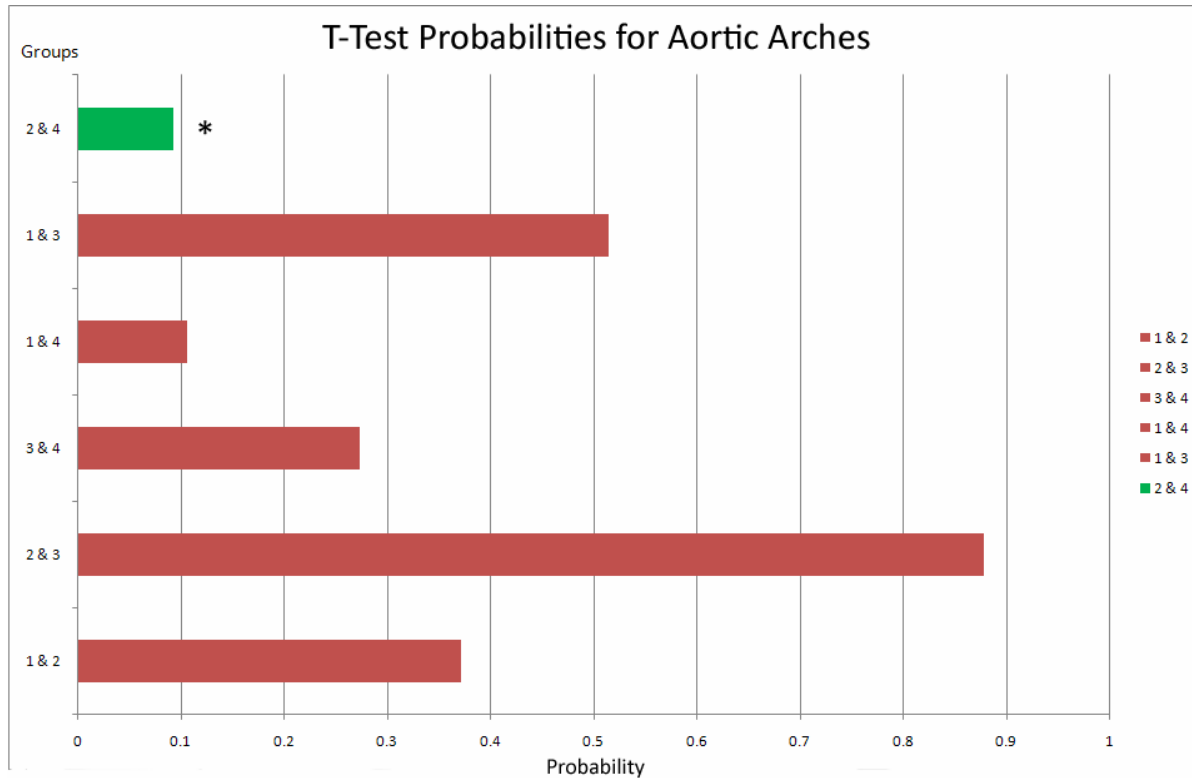


Mean VLDL=127, LDL=141, HDL=110 mg/dl
Mean TC=378 mg/dl
n=10

Glucose & Fructose & SPX-106T



Mean VLDL=82*, LDL=116*, HDL=107 mg/dl
Mean TC=305* mg/dl
p<0.05, n=10



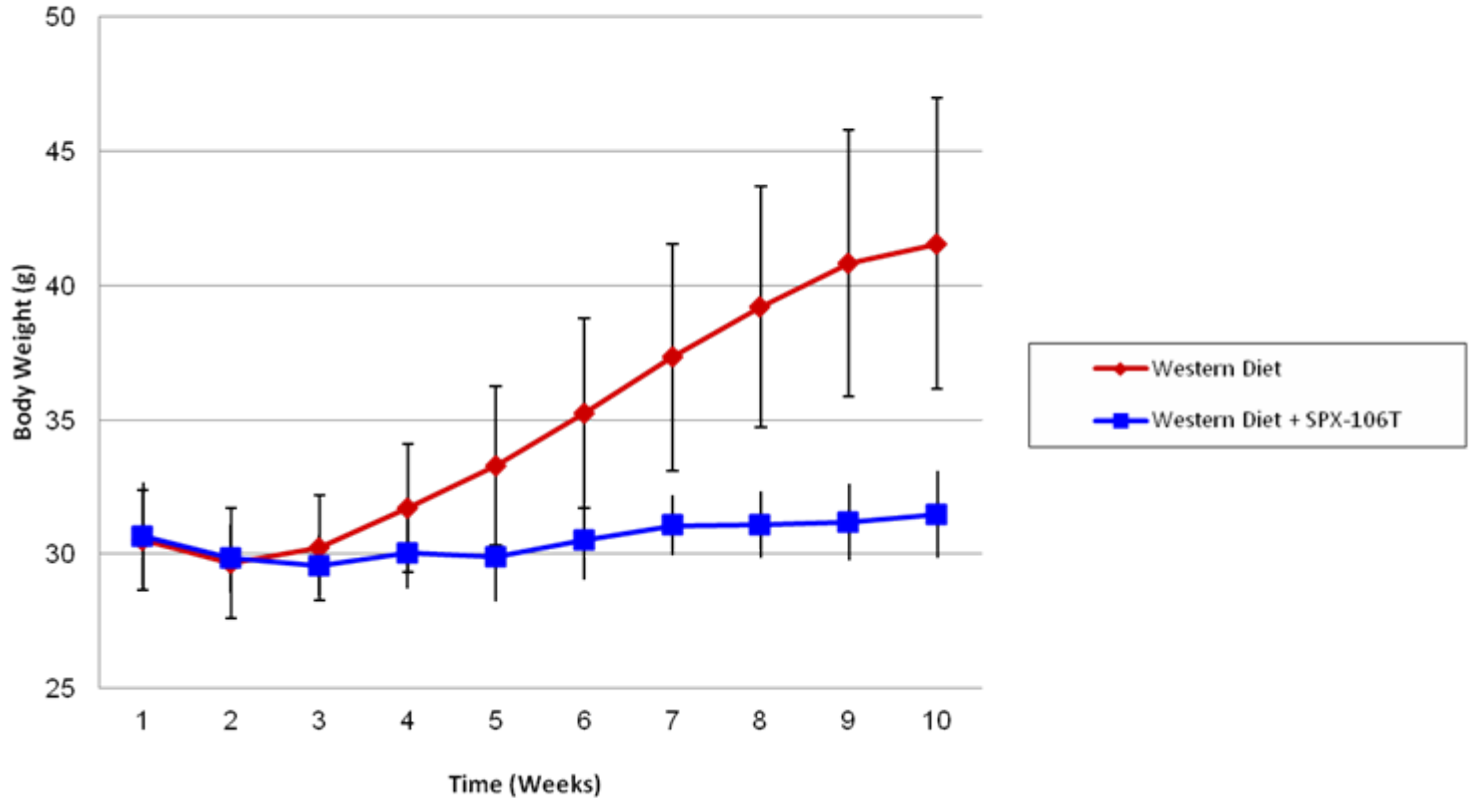
Group 1: Water (- control)
Group 2: Glucose+Fructose (+ control)

Group 3 : Glucose+Fructose+Tagatose
Group 4*: Glucose+Fructose+SPX106T
p < 0.10

- **Serum triglycerides (TG) were cut almost in half**
- **Reduction on VLDL cholesterol was the next largest, followed by the reduction in LDL cholesterol**
- **Reduction in TG, VLDL and LDL may explain the reduction in atherosclerotic lesion area in the aortic arch**

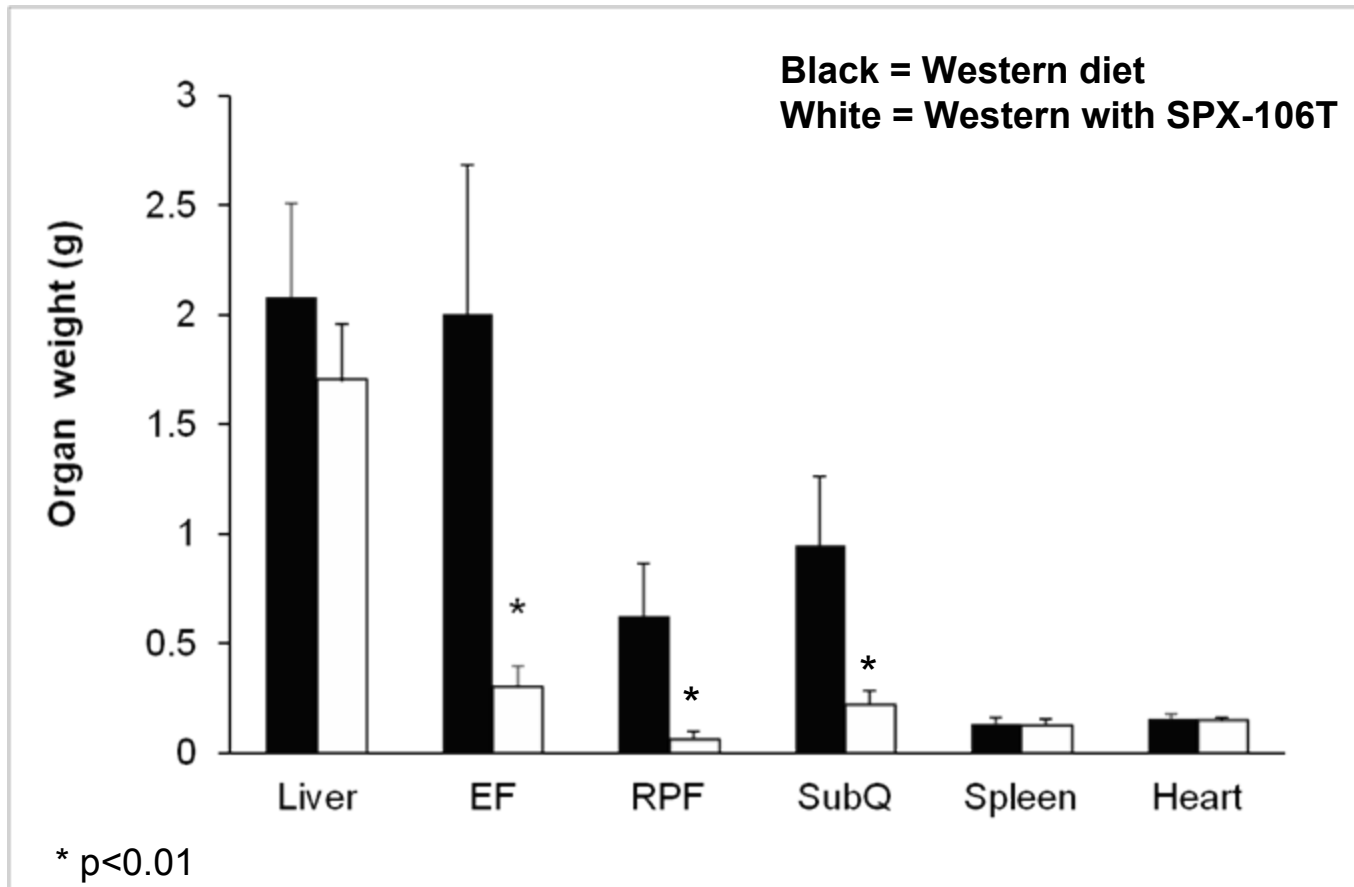
ApoE -/- Total Body Weight

ApoE -/- Mice on Western Diet



| Group | Kcal/g Diet | Total Feed Consumption (g) | Days of study | Number of Mice | Caloric Intake (Kcal/Mouse/Day) |
|----------|-------------|----------------------------|---------------|----------------|---------------------------------|
| Sucrose | 4.5 | 3985 | 70 | 13 | 19.71 |
| SPX-106T | 3.9 | 4591 | 70 | 13 | 19.68 |

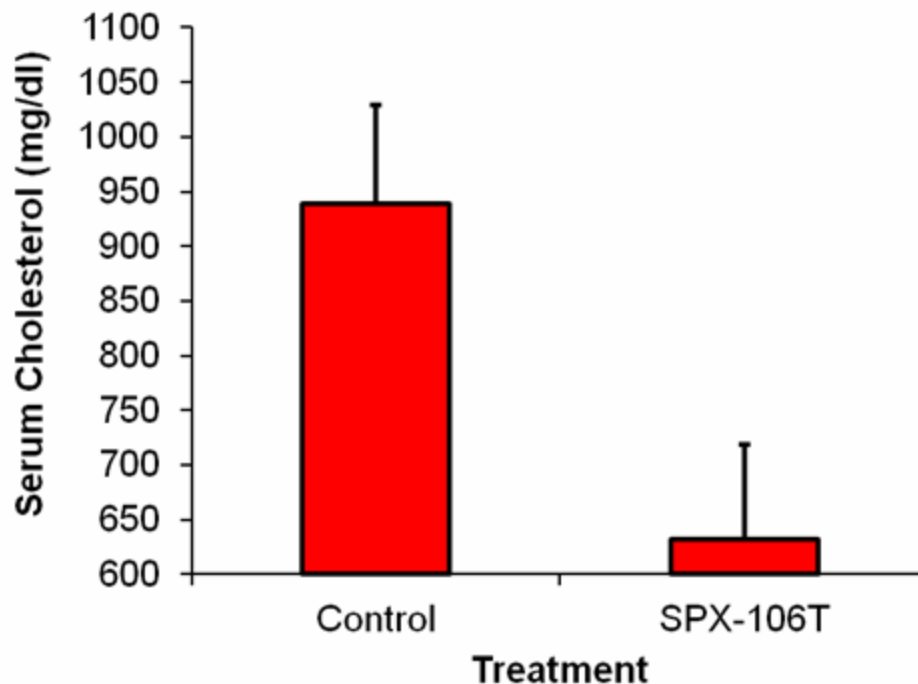
ApoE -/- Mouse Organ Weights



Mass reduction comes from fat pads in the body

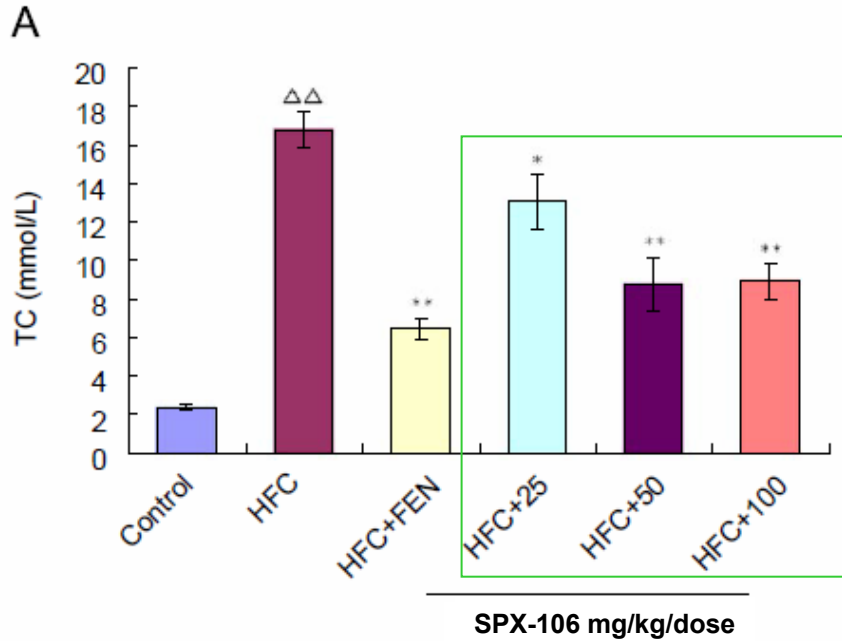
ApoE -/- Total Cholesterol

SPX-106T reduced diet-induced hypercholesterolemia in apo E-deficient mice

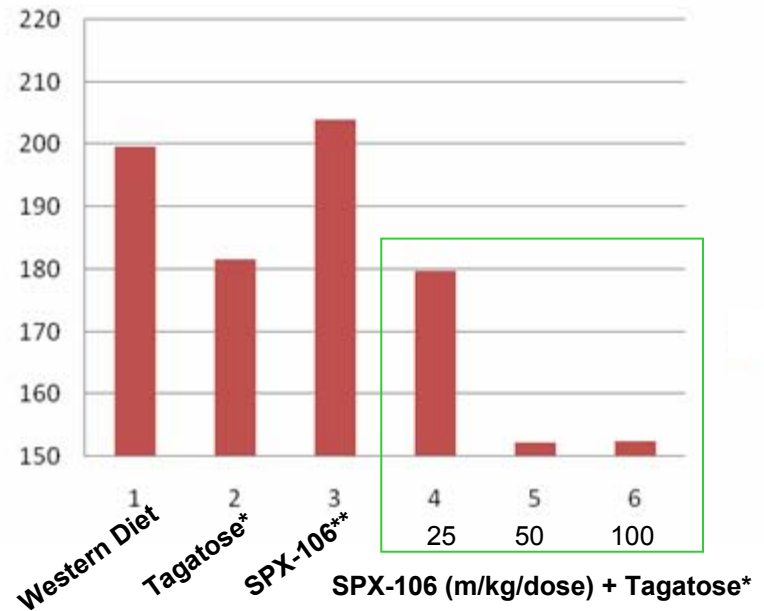


Both groups received a high fat, high carbohydrate diet. In the SPX-106T group, the sucrose in the diet was replaced with tagatose, and the diet was 0.1% SPX-106 by weight.

High Fat Chow (HFC) plus Treatment



Western Diet Plus Treatment



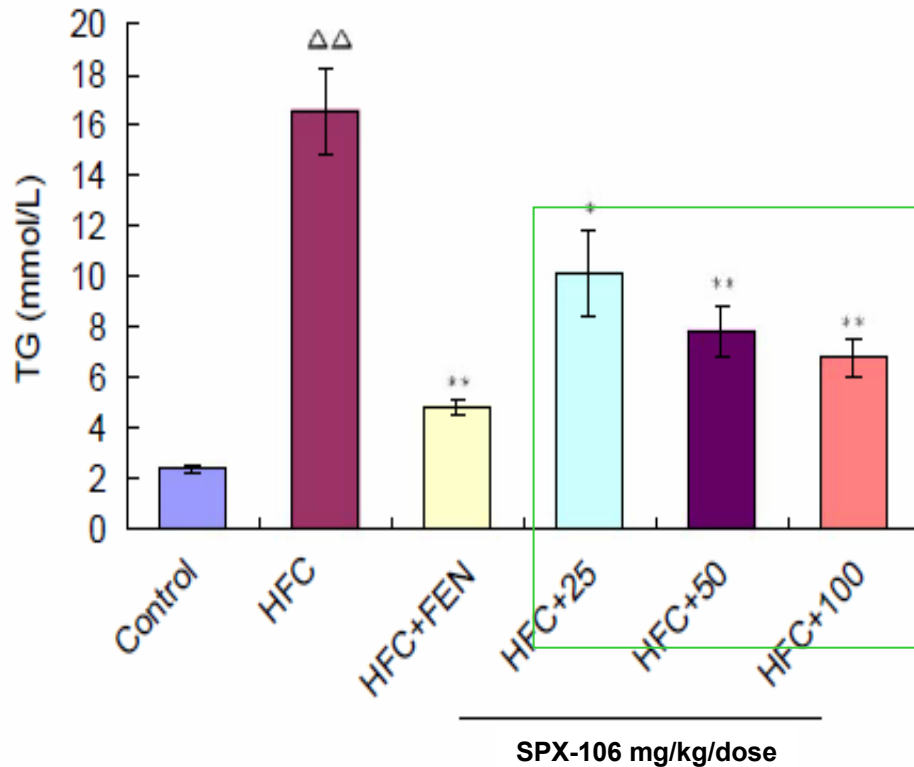
Measurements following 3 weeks of treatment in the hamster

* 6 g/kg/dose

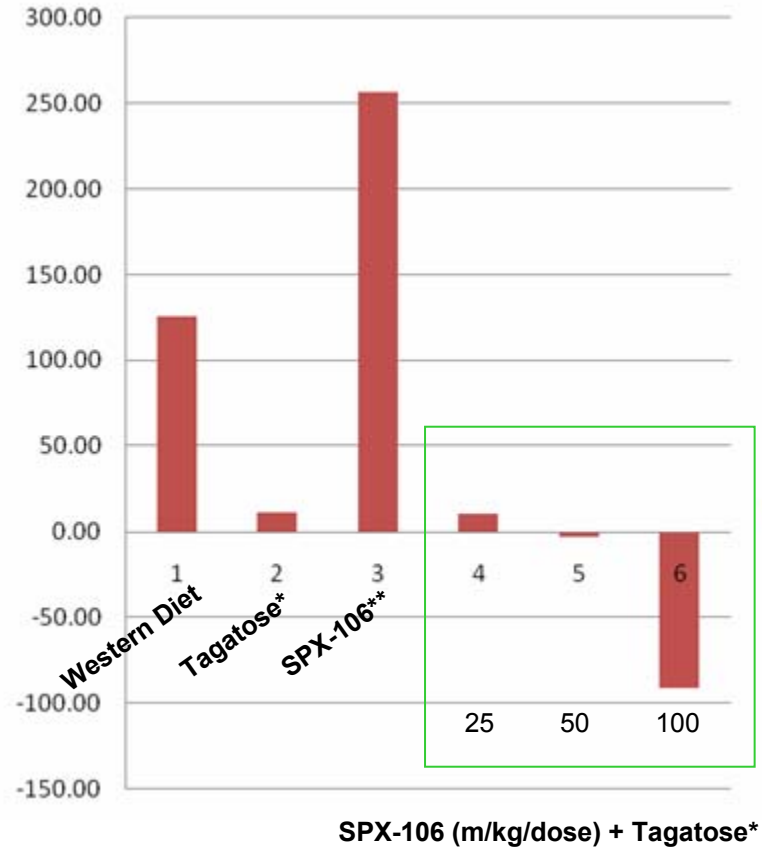
** 100 mg/kg/dose

FEN = fenofibrate

High Fat Chow (HFC) plus Treatment



Western Diet Plus Treatment



* 6 g/kg/dose
** 100 mg/kg/dose
FEN = fenofibrate

Measurements following 3 weeks of treatment in the hamster

- **Objective**
 - Evaluate dose-response effect of minimal doses of D-tagatose (2.5, 5.0 or 7.5 grams dosed 3-times daily) on glycemic control in subjects with Type 2 diabetes not well controlled by diet and exercise
 - 2.5 grams was comparator
- **Design**
 - Multi-center, single-blind, randomized, parallel group clinical study; 6 months duration
 - 34 patients in each of the 3 groups for a total of 102 evaluable patients
- **Clinical Endpoints**
 - Primary: HbA1c ($\Delta 7.5\text{g}-2.5\text{g} = -0.2\%$)
 - Secondary: LDL, HDL, TG, glucose, insulin, body weight
 - 7.5 g reduced triglycerides by -42 mg/dl vs. the 2.5 gram dose (EE) and -31 mg/dl ($p=0.03$) (ITT) at 3 months which remained -29 mg/dl at 6 months
 - Mean triglyceride level was 180 at start of trial

Phase 2 Dose Response Triglyceride Results

- **Unlike some other drugs, D-tagatose lowered triglycerides without elevating VLDL**
- **D-tagatose in the 7.5 g dose reduced LDL vs. the 2.5 g dose by -11 mg/dl by the third month of treatment**
- **The reduction essentially held steady at the six-month end-of-study visit (-10 mg/dl). HDL was unchanged, increasing only between 0.3 and 1.4 mg/dl over the entire course of the study vs. comparator**

HbA1c and Body Mass Index

| | HbA1c | BMI | TG |
|--------------|--------------|------------|-----------|
| HbA1c | 1 | 0.722 | 0.801 |
| BMI | 0.722 | 1 | 0.718 |
| TG | 0.801 | 0.718 | 1 |

Values are Pearson product-moment correlation coefficients, $p \leq 0.10$

Levels of glycated hemoglobin (HbA1c) are directly correlated to levels of triglycerides (TG) and body mass index (BMI). When one of these measures is elevated, the others are likely to be elevated as well. As a result, hyperphagia could, in effect, lead to “fat cell burn-out”.

- **Objective**
 - Evaluate 15 grams of D-tagatose dosed 3-times daily on glycemic control in subjects with Type 2 diabetes not well controlled by diet and exercise
- **Design**
 - Multi-center, double-blind, placebo-controlled study
 - 494 treatment-naïve patients randomized
 - 34 sites in the U.S., 23 sites in India
 - 102 patients enrolled in the U.S., 254 patients enrolled in India
 - Entry HbA1c between 6.6% and 9.0% (avg. 7.5%)
 - $\leq 6\%$ considered normal, $\geq 8\%$ considered high, ADA recommends $\leq 7\%$
- **Clinical Endpoints**
 - Primary: HbA1c
 - Secondary: TG, LDL, HDL, body weight, glucose, insulin,

NEET Study (Protocol 70971-004)

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

- **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**
- **The study was not powered for significance in secondary endpoints (e.g., triglycerides)**

- **Patents include:**
 - **Ten national filings for SPX-106T based on PCT publication # 2010/054001 as anti-metabolic, atherosclerosis, obesity & diabetes methods and composition**
 - **Filing for a tagatose-metformin formulation for diabetes**
- **New Chemical Entity Exclusivity (Hatch-Waxman) – U.S.**
 - **5-year exclusivity (no aNDA's accepted) usually granted to new drug products containing chemical entities never previously approved by FDA either alone or in combination**
 - **Essentially ~6 years exclusivity (base case)**
- **Pediatric Exclusivity – U.S.**
 - **6 months exclusivity for conducting studies in pediatric population**
 - **Added to end of all existing marketing exclusivity and patent periods**

Near Term Milestones

| Milestone | Initiating |
|--------------------------------------------------------------------------|------------|
| • Perform additional animal studies on hypertriglyceridemia for SPX-106T | 1Q'12 |
| • File IND for SPX-106T at FDA | 2Q'12 |
| • Begin human study with SPX-106T in hypertriglyceridemia | 2012 |
| • Engage partner for D-tagatose in diabetes to continue development | Ongoing |

| | |
|----------------------------------------------|------------------------------|
| Cash (9/30/11) | \$5.0 million |
| Working Capital (9/30/11) | \$4.7 million |
| Revenue (9 months ended 9/30/11) | \$0.7 million |
| Estimated Cash Burn (10/1/11-9/30/12) | approx. \$4-6 million |
| Shares Outstanding (11/30/2011) | 3.1 million |
| Warrants Outstanding @ \$32.5 | 110,435 |
| Warrants Outstanding @ \$15.0 | 210,000 |
| Warrants Outstanding @ \$15.625 | 12,600 |
| Warrants Outstanding @ \$8.125 | 12,807 |
| Warrants Outstanding @ \$8.00 | 213,450 |
| Warrants Outstanding @ \$2.24 | 532,559 |
| Warrants Outstanding @ \$2.95625 | 15,977 |

*Unaudited

- **Claire L. Kruger, Ph.D., Chief Executive Officer**
Toxicologist with 25 years of consulting experience; primary area of expertise is in pharmaceuticals, consumer products and foods, where she provides scientific, regulatory, and strategic support to clients in both the US and international regulatory arenas
- **Robert A. Lodder, Ph.D., President**
Founder of InfraReDx, Inc., Prescient Medical, Inc., and Escent Technologies, Inc. Professor of Pharmaceutical Sciences at the College of Pharmacy, University of Kentucky Medical Center. Joint appointments in Electrical & Computer Engineering and in Chemistry. Inventor with over 100 publications and 17 patents.
- **Robert L. Clayton, Chief Financial Officer**
16 years of experience in finance and accounting, including 5 years in public accounting; previously served as Director of Finance and Controller for Spherix
- **Katherine M. Brailer, Corporate Secretary and Director of Administrative Services**
25 years of experience in all aspects of administrative services, including 10 years as an Officer of Spherix

Authoritative Scientific Investigative Team

- **Lisa A. Cassis, Ph.D. – adipocytes and aneurysm**
Chair of the Department of Nutritional Sciences in the College of Medicine. A primary research interest is the study of production of components of the renin-angiotensin system by adipocytes, and the impact of an adipose renin-angiotensin system on the development of obesity and obesity-associated diseases (diabetes, hypertension, atherosclerosis).
- **Alan Daugherty, Ph.D. – lipids and atherosclerosis**
Editor-in-Chief of the AHA Journal *Arteriosclerosis, Thrombosis, and Vascular Biology* (ATVB). College of Medicine Senior Associate Dean for Research, Director, Saha Cardiovascular Research. Studies macrophages, endothelial cells and smooth muscle cells in the development of dyslipidemias, atherosclerosis, and abdominal aortic aneurysm.
- **Phillip Kern, M.D. – metabolic syndrome**
Professor, Division of Endocrinology and Molecular Medicine, Director, Barnstable Brown Kentucky Diabetes and Obesity Center, Director, Center for Clinical and Translational Sciences. Basic and clinical research related to obesity, metabolic syndrome, and diabetes. Clinical studies have examined adipose tissue and muscle gene expression in relation to insulin resistance, tissue lipotoxicity and inflammation in obesity related insulin resistance. Other research involves the gene expression of lipoprotein lipase (LPL), an important enzyme in adipose tissue that is involved in triglyceride metabolism, diabetes, and obesity.