

**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)

November 3, 2014

CAPRICOR THERAPEUTICS, INC.

(Exact name of Registrant as Specified in its Charter)

Delaware
(State or other jurisdiction
of incorporation)

001-34058
(Commission
File Number)

88-0363465
(I.R.S. Employer
Identification No.)

8840 Wilshire Blvd., 2nd Floor, Beverly Hills, CA
(Address of principal executive offices)

90211
(Zip Code)

(310) 358-3200
(Registrant's telephone number, including area code)

Not Applicable
(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:

- 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 8.01. Other Events.

On November 3, 2014, Capricor Therapeutics, Inc., a Delaware corporation (the “Company”), posted a Corporate Presentation to provide an update of the Company’s current business and products on the “Investors” section of the Company’s website at www.capricor.com. A copy of the Corporate Presentation is attached hereto as Exhibit 99.1 and is incorporated by reference into this Item 8.01 of this Current Report on Form 8-K.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

99.1 Capricor Therapeutics, Inc. Corporate Presentation, dated November 2014.

SIGNATURES

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

CAPRICOR THERAPEUTICS, INC.

Date: November 6, 2014

By: /s/ Linda Marbán, Ph.D.

Linda Marbán, Ph.D.

Chief Executive Officer



Capricor
Therapeutics TM

**Transformative Therapies
from Bench to Bedside**

Corporate Presentation
November 2014

**Clinical-stage biotechnology company developing novel
therapeutics for the treatment of cardiovascular diseases**

Ticker: CAPR

Non-confidential

Forward Looking Statements

This presentation contains forward-looking statements and information that are based on the beliefs of the management of Capricor Therapeutics, Inc. (Capricor) as well as assumptions made by and information currently available to Capricor. All statements other than statements of historical fact included in this presentation are forward-looking statements, including but not limited to statements identified by the words “anticipates,” “believes,” “estimates,” and “expects” and similar expressions. Such forward-looking statements also include any expectation of or dates for commencement of clinical trials, IND filings, similar plans or projections and other matters that do not relate strictly to historical facts. These statements reflect Capricor’s current views with respect to future events, based on what we believe are reasonable assumptions; however, the statements are subject to a number of risks, uncertainties and assumptions. There are a number of important factors that could cause actual results or events to differ materially from those indicated by such forward-looking statements. More information about these and other risks that may impact our business are set forth in our Annual Report on Form 10-K for the year ended December 31, 2013, as filed with the Securities and Exchange Commission on March 31, 2014, in our Amendment No. 1 to Registration Statement on Form S-1, as filed with the Securities and Exchange Commission on May 23, 2014, and in our Form 10-Q for the period ended June 30, 2014, as filed with the Securities and Exchange Commission on August 14, 2014. Should one or more of these risks or uncertainties materialize, or should underlying assumptions prove incorrect, actual results may vary materially from those in the forward-looking statements. Further, Capricor’s management does not intend to update these forward-looking statements and information after the date of this presentation.



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Capricor Overview

Capricor Investment Opportunity

- **Ongoing Phase II clinical trial with Cardiosphere-derived cells (CDCs)**
 - Phase II funded by \$19.8M loan award from CIRM
 - Janssen Biotech (J&J) collaboration
- **Operational leverage permits cost effective product development and evaluation**
- **Approximately 12-15 months cash on hand**
- **Multiple pipeline opportunities permit development of diversified product portfolio focused on cardiovascular indications**
 - Cell therapy technology
 - Natriuretic peptide technology
 - Micro-RNA technology
- **\$39.5M of non-dilutive capital since inception**

Senior Management & Board of Directors

Senior Management

- **Chief Executive Officer**
Linda Marbán, Ph.D.
- **EVP & General Counsel**
Karen Krasney, J.D.
- **VP of Research & Development**
Rachel Smith, Ph.D.
- **VP of Finance**
AJ Bergmann, M.B.A.
- **VP of Medical Affairs**
Andrew Hamer, M.D.
- **VP of Clinical Operations**
Shane Smith
- **VP of Quality**
Denise McDade

Board of Directors

- **Executive Chairman**
Frank Litvack, M.D.
- Linda Marbán, Ph.D.
- Dave Musket
- Earl M. (Duke) Collier, Jr.
- George W. Dunbar, Jr.
- Louis Manzo
- Louis J. Grasmick
- Joshua Kazam
- Gregory Schafer

Capricor: Key Metrics

Select Data (approximate)	As of 6.30.14
Cash, cash equivalents and marketable securities	\$11.9M
Publicly traded: OTCBB	Ticker: CAPR
52 week range	\$2.15-\$17.15
Shares outstanding	11.7M
Fully diluted shares outstanding	16.9M
Cash through	Q3 2015 – Q4 2015
Non-dilutive capital funding to date	\$39.5M
Exclusive Licenses	Johns Hopkins University, Cedars-Sinai Medical Center, Mayo Foundation for Medical Education and Research, and The University of Rome
Headquarters	Los Angeles, CA
Employees	26



Capricor
Therapeutics

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Capricor's Product Pipeline

	CARDIOSPHERE-DERIVED CELLS (CAP-1002)	CENDERITIDE (CD-NP)	EXOSOMES
Technology	CELL THERAPY	NATRIURETIC PEPTIDE	MICRO-RNA
Clinical	Currently enrolling Phase II – ALLSTAR trial	Planned Phase I 1H 2015	Targeting IND 2015-2016
Development	Strategic Collaboration with Janssen Biotech (J&J)	Delivery Collaboration: Insulet OmniPod® IP: Medtronic, Inc.	Clinical Development Plan Underway Data: AHA Nov. 2014
Indications	Ischemic Heart Disease Heart Failure Duchenne Muscular Dystrophy	Post-Acute Heart Failure	Ischemic Heart Disease Duchenne Muscular Dystrophy



Non-confidential

Janssen Deal Highlights

In December 2013, Capricor entered into Collaboration Agreement and Exclusive License Option with Janssen Biotech (J&J)

- Capricor received an upfront payment of \$12.5M
- Capricor and Janssen collaborating on elements of cell manufacturing
- Janssen has option to enter into exclusive license agreement for CAP-1002 up to 60 days after receipt of **six month ALLSTAR Phase II data**
- If exercises option, Capricor to receive an upfront license fee and additional milestone payments which may total up to \$325M
- Double-digit royalty to be paid to Capricor on commercial sales of licensed products
- If option is exercised, Janssen will pay all future clinical trial and development costs of CAP-1002

Significant U.S. Target Market

Cardiovascular Disease Remains America's #1 Killer

- **1.3M** Americans will have a new or recurrent myocardial infarction (MI) or heart attack
 - 15% will die (1 death every 39 seconds)
 - 36% will develop heart failure (HF)
 - Survival correlated with infarct size
- **8.5M** people in the US have had a heart attack
- **6.6M Americans are living with HF**
 - 50% 5-year mortality rate

Wu et al. *Heart*. 2007.

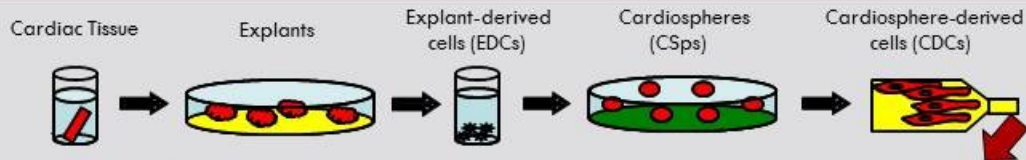
American Heart Association. 2010.

Cell Therapy Technology

Discovery of Cardiosphere Derived Cells (**CDCs**)

- Discovered and patented at Johns Hopkins University in 2004 by Eduardo Marbán, M.D., Ph.D.
- CDCs are isolated from cardiac tissue and possess unique characteristics and potentially regenerative capabilities
- CDCs act to stimulate cardiomyocyte proliferation, recruit endogenous stem cells, promote angiogenesis, and attenuate fibrosis
- Since their discovery, ~100 related publications have emerged in the last decade
- Capricor has exclusively licensed CDC Intellectual Property from Johns Hopkins University

Lead Product: CDCs (CAP-1002)

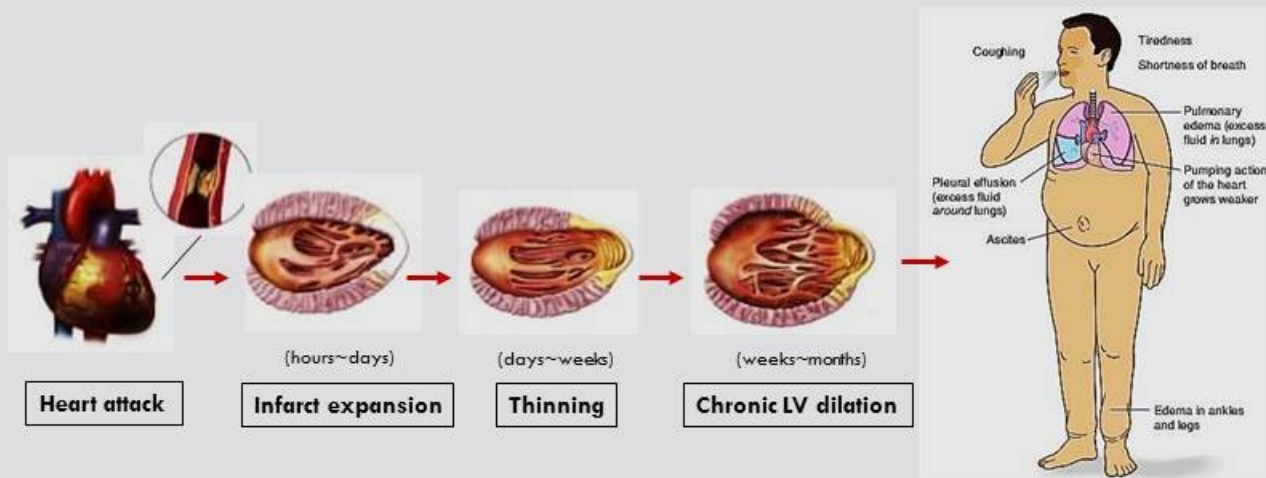


Features

Cardiosphere-Derived Cells (CDCs)

Cell source	Human Cardiac Tissue
Cell Type	Cardiac Derived Stem Cell
Dose Size	25M cells
Target Patient	Patients with heart muscle injury
Delivery Method	Intracoronary Delivery
Key Functions	Largely paracrine: <ul style="list-style-type: none">▪ Prevent cardiomyocyte apoptosis (programmed cell death)▪ Promote cardiomyocyte proliferation and angiogenesis (cell growth and blood vessel formation)▪ Attract endogenous stem cells▪ Anti-fibrotic (anti-scarring)

Development of Ischemic Heart Failure



CADUCEUS, Lancet: 2012

Intracoronary cardiosphere-derived cells for heart regeneration after myocardial infarction (CADUCEUS): a prospective, randomised phase 1 trial



Raj R Mulkar, Rachel R Smith, Ke Cheng, Konstantinos Malliaris, Louise E J Thomson, Daniel Bennett, Lawrence S C Czei, Linda Marbán, Adam Mendizabal, Peter V Johnston, Stuart D Russell, Karl H Schuler, Albert C Lardo, Guy Gerstenblith, Eduardo Marbán

Summary

Background Cardiosphere-derived cells (CDCs) reduce scarring after myocardial infarction, increase viable myocardium, and boost cardiac function in preclinical models. We aimed to assess safety of such an approach in patients with left ventricular dysfunction after myocardial infarction.

Methods In the prospective, randomised CARDiosphere-Derived aUtologous stem CELls to reverse ventricUlar dysfunction (CADUCEUS) trial, we enrolled patients 2–4 weeks after myocardial infarction (with left ventricular ejection fraction of 25–45%) at two medical centres in the USA. An independent data coordinating centre randomly allocated patients in a 2:1 ratio to receive CDCs or standard care. For patients assigned to receive CDCs, autologous cells grown from endomyocardial biopsy specimens were infused into the infarct-related artery 1–5–3 months after myocardial infarction. The primary endpoint was proportion of patients at 6 months who died due to ventricular tachycardia, ventricular fibrillation, or sudden unexpected death, or had myocardial infarction after cell infusion, new cardiac tumour formation on MRI, or a major adverse cardiac event (MACE; composite of death and hospital admission for heart failure or non-fatal recurrent myocardial infarction). We also assessed preliminary efficacy endpoints on MRI by 6 months. Data analysers were masked to group assignment. This study is registered with ClinicalTrials.gov, NCT00893360.

Findings Between May 5, 2009, and Dec 16, 2010, we randomly allocated 31 eligible participants of whom 25 were included in a per-protocol analysis (17 to CDC group and eight to standard of care). Mean baseline left ventricular ejection fraction (LVEF) was 39% (SD 12) and scar occupied 24% (10) of left ventricular mass. Biopsy samples yielded prescribed cell doses within 36 days (SD 6). No complications were reported within 24 h of CDC infusion. By 6 months, no patients had died, developed cardiac tumours, or MACE in either group. Four patients (24%) in the CDC group had serious adverse events compared with one control (13%; $p=1.00$). Compared with controls at 6 months, MRI analysis of patients treated with CDCs showed reductions in scar mass ($p=0.001$), increases in viable heart mass ($p=0.01$) and regional contractility ($p=0.02$), and regional systolic wall thickening ($p=0.015$). However, changes in end-diastolic volume, end-systolic volume, and LVEF did not differ between groups by 6 months.

Interpretation We show intracoronary infusion of autologous CDCs after myocardial infarction is safe, warranting the expansion of such therapy to phase 2 study. The unprecedented increases we noted in viable myocardium, which are consistent with therapeutic regeneration, merit further assessment of clinical outcomes.

Funding US National Heart, Lung and Blood Institute and Cedars-Sinai Board of Governors Heart Stem Cell Center.

Published Online

February 14, 2012

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6736(12)60295-0

See Online/Comment

DOI:10.1016/S0140-

6736(12)60296-0

Cedars-Sinai Heart Institute,

Los Angeles, CA, USA

(R R Mulkar MD, R R Smith PhD,

K Cheng PhD, K Malliaris MD,

L E Thomson MD,

Prof D Bennett MD,

L S Czei MD, L Marbán PhD,

Prof E Marbán MD); The EMMES

Corporation, Rockville, MD,

USA (A Mendizabal MD); and

The Johns Hopkins University,

Baltimore, MD, USA

(P V Johnston MD,

S D Russell MD, K H Schuler MD,

A C Lardo PhD,

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8700 Beverly Boulevard,

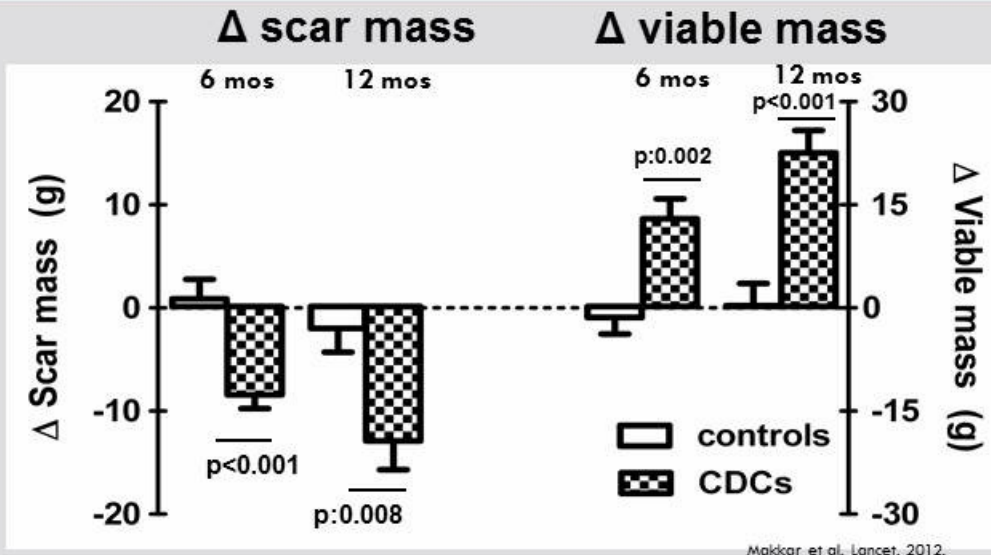
Los Angeles, CA 90048, USA

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CADUCEUS - Positive First-in-Man Data

- Autologous CDCs - 25M cells
- Patients with reduced Ejection Fraction % following MI
- Cedars-Sinai and Johns Hopkins
- Intracoronary delivery
- 25 patients
 - 17 CDCs
 - 8 Controls

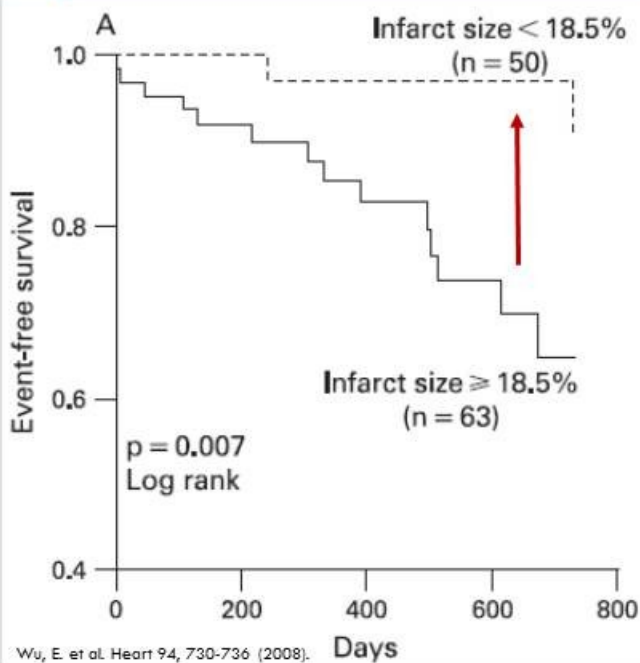
CDC Therapy Reduced Scar Size and Increased Healthy Heart Muscle in the **CADUCEUS** study



Makkar et al, Lancet, 2012.

**CDC Patients had a Significant Reduction in Infarct Size.
We hypothesize improvement in clinical outcomes**

Effect of Infarct Size on Survival



- Both groups (CDC and CTRL) started with Infarct Size of 24%
- Patients treated with CDCs saw a reduction of Infarct Size to 12.5%
- This moved patients from a **high** risk group to a **low** risk group
- CTRL patients saw no change in Infarct Size, which puts them at greater risk for adverse events

Wu, E. et al. Heart 94, 730-736 (2008).

Advantages of Allogeneic CDCs

- Donors pre-screened by organ procurement organizations
- Single donor permits manufacturing of thousands of doses
- Freezing permits off-the shelf product availability
- COGS reduced by 10X compared to autologous cells

Patient Dose of CAP-1002



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CDCs in Active Clinical Development

	ALLSTAR Clinical Trial	DYNAMIC Clinical Trial	Duchenne Muscular Dystrophy
Target Indication	Post Myocardial Infarction (30 days – 1 year after MI)	NYHA Class III or ambulatory Class IV heart failure	DMD-related cardiomyopathy
Trial Size	Phase I – 14 patients Phase II – 300 patients	Phase Ia – 14 patients Phase Ib – 28 patients	Planned Phase I (~10 patients)
Drug Delivery	Single vessel intracoronary	Multi-vessel intracoronary	Multi-vessel intracoronary
Data Readout	Estimated 2016 (Phase II)	Estimated late 2015 (Phase Ia)	Estimated 2016 (Phase I)
FDA Status	IND granted, Phase II currently enrolling	IND granted, Phase I plan to commence in 2014	Potential Orphan Designation by FDA

ALLSTAR: Capricor's Phase I/II Trial using CDCs

ALLogeneic heart **ST**em cells to **A**chieve myocardial **R**egeneration

- Ejection Fraction $\leq 45\%$ and Infarct Size $\geq 15\%$ (MRI)
- Primary Endpoint – Infarct Size by MRI at 1 year
- Secondary Endpoints – multiple; EF, volumes, quality of life, etc.
- Phase I: 14 patients – complete
- Phase II: Est. 300 patients – currently enrolling

ALLSTAR: Phase I Primary Endpoint Met

- 1 month post-infusion
 - **No** Acute Myocarditis attributable to CAP-1002
 - **No** clinically relevant immune events
 - **No** Death due to VT/VF
 - **No** Sudden Death
 - **No** Major Adverse Cardiac Events (MACE)
- NIH DSMB approved advancement to Phase II

ALLSTAR Phase I – 12 month MRI Analysis

- **ALLSTAR Phase I**
 - 14 treated patients - open label dose escalation
 - Met safety endpoint (1 month)
 - No control group
- **Preliminary 12 month MRI analysis on Phase II equivalent population (defined by tissue type compatibility)**
 - **Ejection fraction improved by 5.2%**
 - **Relative reduction in scar size of 20.7%**
 - **Measurements of viable mass and regional function also showed quantifiable improvements**

New Indication: Duchenne Muscular Dystrophy



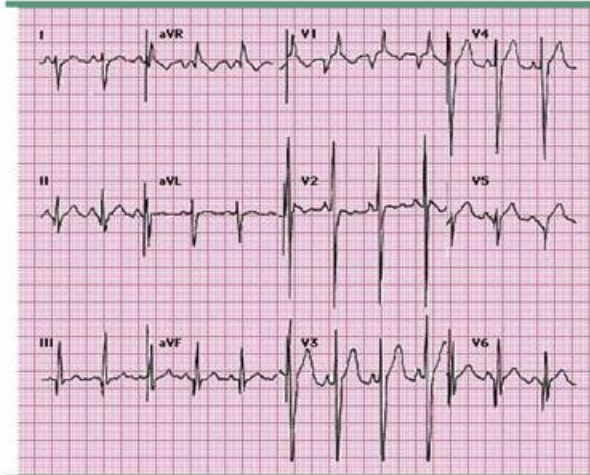
Duchenne Muscular Dystrophy

- Duchenne Muscular Dystrophy (DMD) – rare genetic disorder caused by a mutation of the dystrophin gene
 - Affects 1 in 3,500 male births worldwide
 - Approximately 20,000 male children affected in the US (275,000 worldwide)
 - Symptoms often appear in males before age six but may be visible in early infancy
 - The disease is often fatal
- Though characterized by progressive skeletal muscle weakness and respiratory complications, DMD also results in cardiac dysfunction in most patients.
 - **A majority of deaths occur due to cardiomyopathy**
 - Pre-clinical data to be presented at AHA in November 2014

CAP-1002 as a Potential Treatment for Cardiomyopathy Associated with DMD

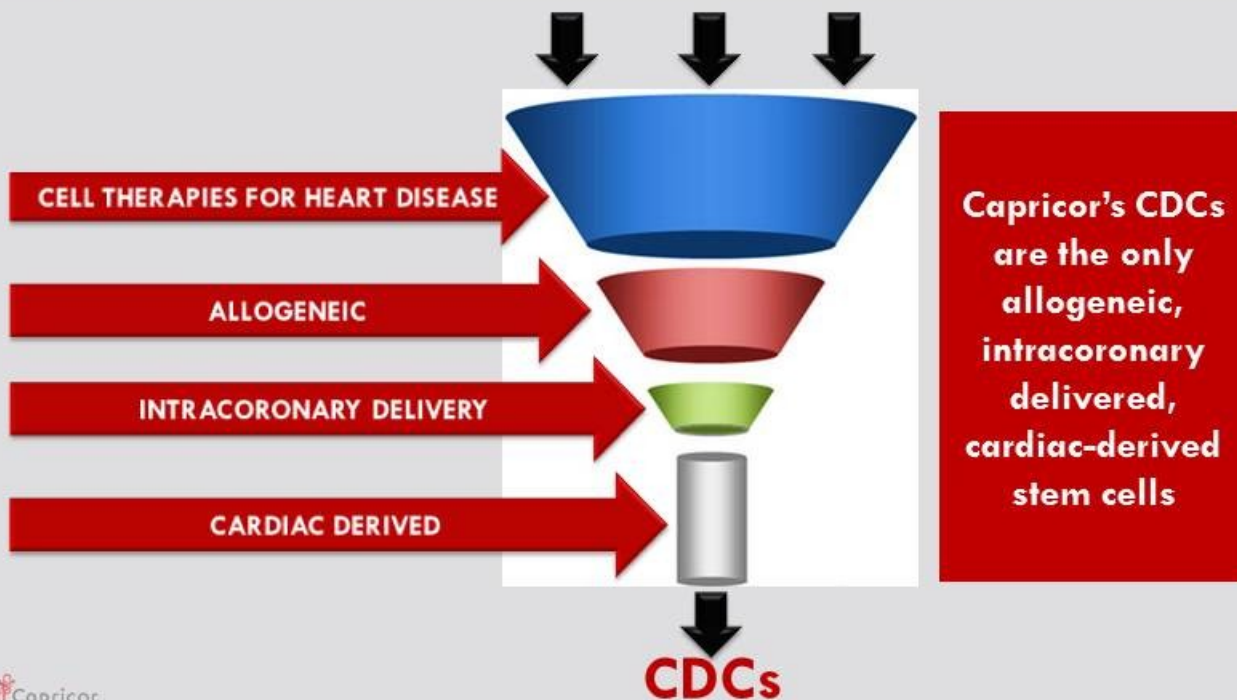
- **CAP-1002** has been shown to be safe and has been shown to reduce scar size in damaged hearts.
- Pre-clinical work on-going
- Targeting IND mid-2015
- Phase I planned for 2015
- Presentation: AHA Nov. 2014

Duchenne's muscular dystrophy



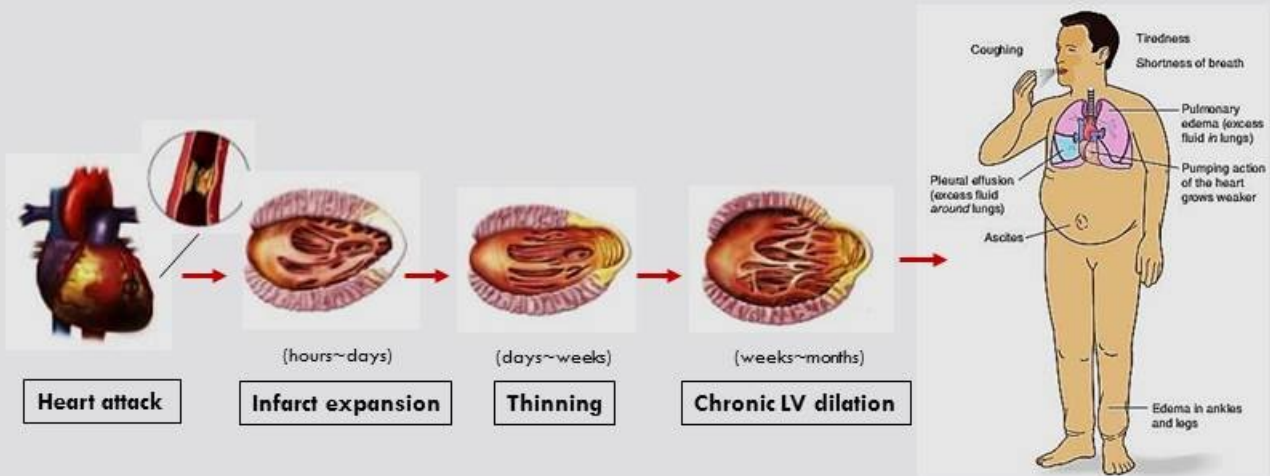
Classic findings in Duchenne's muscular dystrophy include resting sinus tachycardia, deep infero-lateral Q waves, and a tall R in lead V1 due to the underlying cardio-myopathy which is often associated with posterobasal fibrosis of the left ventricle. Left atrial enlargement, as indicated by the prominent negative P wave in lead V1, and marked right axis deviation are also present. Courtesy of Ary Goldberger, MD.

Capricor's CDCs Are Unique in Cardiac Cell Therapy

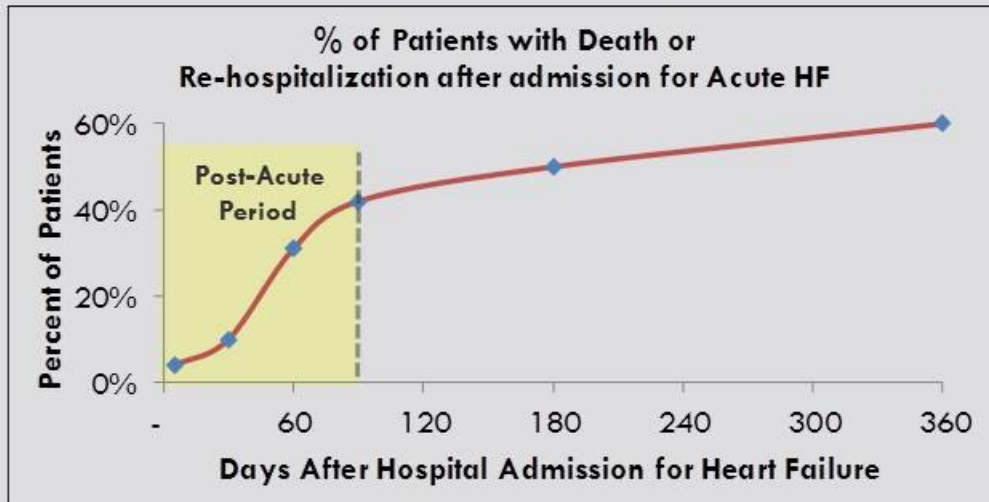


Natriuretic Peptide Technology

Cenderitide's treatment for Heart Failure



The Post-Acute Hospitalization Period (90 days): When the Rate of Re-hospitalization and Death are Highest



- As days in hospital have decreased, patient's physiology is unstable at discharge
- HF patients are frequently non-compliant with their chronic medications

Reference: estimate from analysis of DOSE, PROTECT, ASCEND, OPTIMIZE, & ADHERE

Post-Acute Heart Failure: New Indication

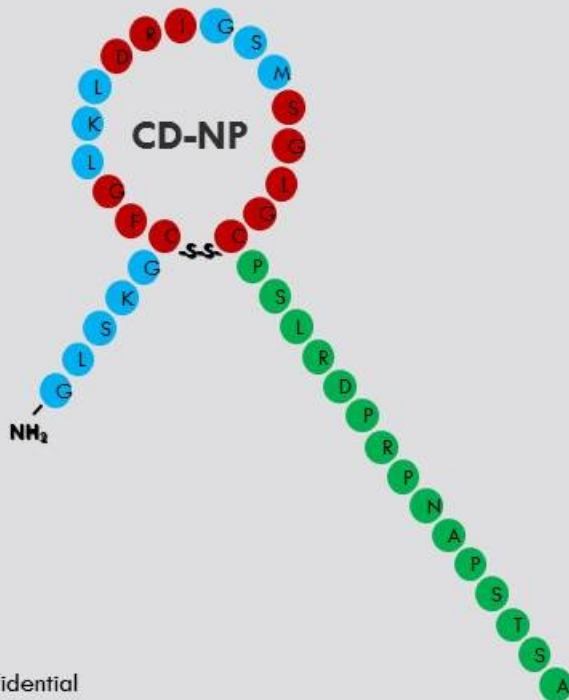
There are **no drugs** on the market targeting the post-acute period

	Acute	Post-Acute	Chronic
Duration	3-7 days	1 st 90 days after discharge	1-5 years
Location	In-Hospital	Home	Home
Mortality*	2-4%	8-12%	~20% (1 yr) / ~50% 5 yrs
Treatment Goals	<ul style="list-style-type: none"> • Relieve dyspnea • Off-load excess fluid 	<ul style="list-style-type: none"> • Survive • Stay out of hospital 	<ul style="list-style-type: none"> • Survive • Stay out of hospital
Treatment Options	<ul style="list-style-type: none"> • Lasix • IV Nitroglycerine • Inotropes • Natrecor 	<ul style="list-style-type: none"> • No treatment specifically targets stabilization and reducing the early re-hospitalization rate 	<ul style="list-style-type: none"> • Beta blockers • ACE inhibitors • ARB inhibitors • Aldactone • Cardio-Rhythm Device

* Includes all mortality up to that point (post-acute mortality = acute mortality + post-discharge mortality)

Cenderitide: Dual GC-A and GC-B Agonist

- Dual receptor activation means anti-fibrotic as well as natriuretic effects with less side effects
- Developed by scientists at the Mayo Clinic and derived from the venom of the green mamba snake
- **Cenderitide:**
 - Cardiac unloading
 - Renal function preserved
 - Aldosterone suppressing
 - Anti-fibrotic, apoptotic, and hypertrophic
- 270 patients with acute decompensated heart failure have been treated



Continuous Subcutaneous Infusion Using the Insulet Omnipod® Technology

- Current pump delivery systems are robust, simple, and well tolerated
 - In use by more than 300,000 patients worldwide
 - Pump use is simple for all types of patients, not just diabetics
- Continuous delivery is ideal for worry-free dosing throughout the day and night



Fill the Pod



Apply the Pod



Press Start

Sample shown: Omnipod®

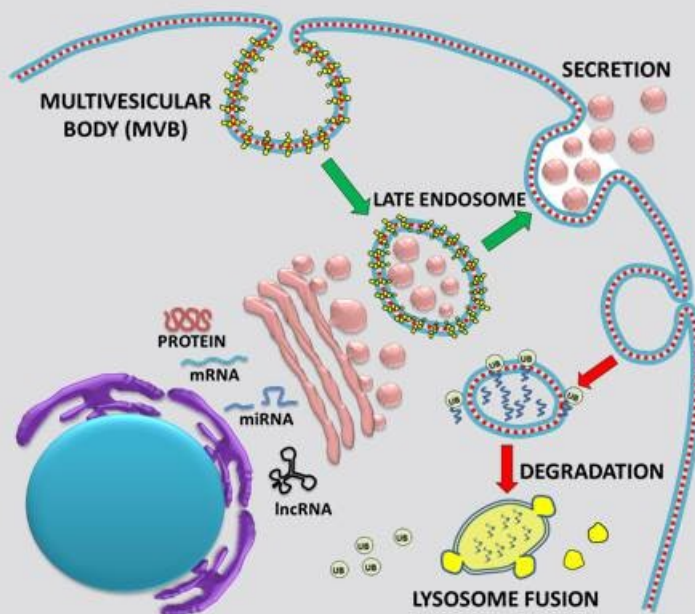
Cenderitide as a Potential Treatment for Post-Acute Heart Failure

Target Indication	Prevention of re-hospitalization in heart failure patients in the post-acute hospitalization period
Treatment Duration	90 days of out-patient treatment after hospital discharge for acute decompensated heart failure
Drug Delivery	Subcutaneous infusion using the Insulet Omnipod [®] validated technology
Clinical Progress	Planned Phase I 1H 2015
FDA Status	Fast Track designation granted March 2011

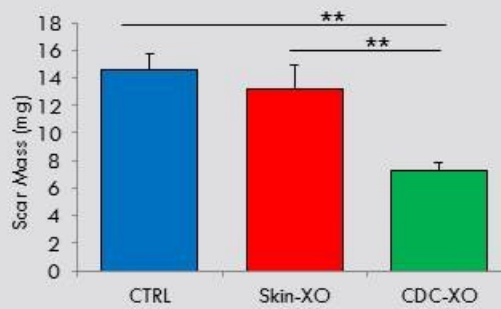
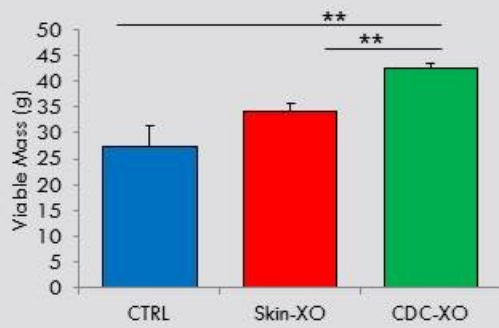
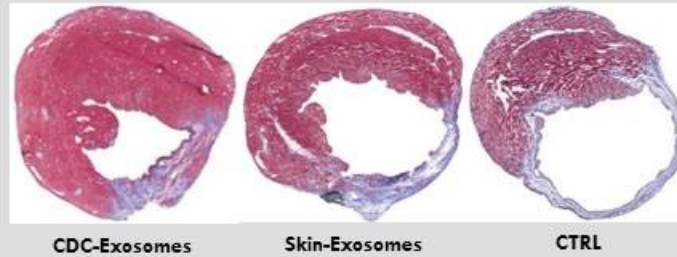
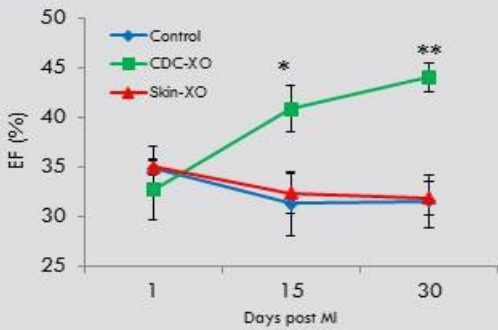
Micro-RNA Technology

Exosomes: Micro-RNA Platform Technology

- Nanometer-sized lipid-bilayer vesicles
- Produced via endosome fusion
- Transfer proteins and RNAs
- Rich in miRNAs
- Present in virtually all body fluids
- Released by nearly all cell types



CDC Exosomes: Cardioprotection



Exosomes as a potential new therapeutic

Product Description

Released by nearly all cell types and body fluids, these are nanometer sized lipid-bilayer vesicles rich in miRNAs

Product Function

Promotes similar paracrine effects of CDCs; prevent apoptosis, proliferation, angiogenesis, anti-fibrotic

Product Mechanism

Intercellular signaling and regulation

Target Indications

Pre-clinical evaluation in cardiac and non-cardiac diseases

Exosomes: Development Plan

- Ongoing pre-clinical work
- Developing manufacturing and pre-commercial scale-up
- Exclusively licensed IP portfolio from Cedars-Sinai Medical Center
- Targeting IND 2015-2016