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 15, 2017

CAPRICOR THERAPEUTICS, INC.

(Exact name of Registrant as Specified in its Charter)

Delaware	**001-34058**	**88-0363465**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

8840 Wilshire Blvd., 2nd Floor, Beverly Hills, CA	**90211**
(Address of principal executive offices)	**(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))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (17 CFR §230.405) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On November 15, 2017, Capricor Therapeutics, Inc., a Delaware corporation (the "Company"), issued a press release announcing positive twelve-month results from the randomized Phase I/II HOPE clinical trial in Duchenne muscular dystrophy. A copy of the press release is attached hereto as Exhibit 99.1 and is incorporated by reference into this Item 7.01 of this Current Report on Form 8-K.

On November 15, 2017, information related to the Company's HOPE and ALLSTAR clinical trials was presented at the American Heart Association's (the "AHA") Scientific Sessions 2017. Attached hereto as Exhibit 99.2 to this Current Report on Form 8-K is a presentation presented at the AHA's Scientific Sessions 2017 relating to the Company's HOPE clinical trial. Attached hereto as Exhibit 99.3 to this Current Report on Form 8-K is a presentation presented at the AHA's Scientific Sessions 2017 relating to the Company's ALLSTAR clinical trial.

The information under Item 7.01 of this Current Report on Form 8-K and Exhibits 99.1, 99.2, and 99.3 attached hereto are being furnished and shall not be deemed to be filed for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall they be incorporated by reference into any of the Company's filings under the Securities Act of 1933, as amended, or the Exchange Act, unless expressly set forth as being incorporated by reference into such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

99.1	Press Release, titled "Significant Improvements Reported in Duchenne Muscular Dystrophy Patients Treated with Capricor's Investigational Cell Therapy", dated November 15, 2017.
99.2	HOPE-Duchenne AHA Presentation, dated November 15, 2017.
99.3	ALLSTAR AHA Presentation, dated November 15, 2017.

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 15, 2017

By: /s/ Linda Marbán, Ph.D.
Linda Marbán, Ph.D.
Chief Executive Officer

Exhibit 99.1

Significant Improvements Reported in Duchenne Muscular Dystrophy Patients Treated with Capricor's Investigational Cell Therapy

Dr. Ronald G. Victor Presents 12-Month Results of HOPE Clinical Trial at AHA Late-Breaking Session

Company to Host Conference Call and Webcast Today at 4:30 p.m. ET

ANAHEIM, CA – Nov. 15, 2017 – Boys and young men in advanced stages of Duchenne muscular dystrophy experienced significant and sustained improvements in cardiac structure and function, as well as skeletal muscle function, following treatment with CAP-1002, the lead investigational therapy under development at Capricor Therapeutics (NASDAQ: CAPR). These findings were reported today by Ronald G. Victor, M.D. at a Late-Breaking Science session of the American Heart Association Scientific Sessions 2017.

Dr. Victor, who is the associate director for clinical research at the Cedars-Sinai Heart Institute and a lead investigator for the HOPE trial, reported that those treated with CAP-1002 had improvement in cardiac muscle function and reduction in cardiac scarring that were statistically-significant in comparison to the control group, according to a pre-specified analysis. In addition, in a post-hoc analysis, 89% of the CAP-1002 treated patients who were more severely impaired demonstrated sustained or improved skeletal muscle function at 12 months, as compared to none of the participants in the control group.

"Because Duchenne muscular dystrophy is a devastating, muscle-wasting disease that causes physical debilitation and eventually heart failure, the improvements in heart and skeletal muscle in those treated with a single dose of CAP-1002 are very promising and show that a subsequent trial is warranted," said Dr. Victor. "These early results provide hope for the Duchenne community, which is in urgent need of a major therapeutic breakthrough."

Dr. Victor, who has worked in the Duchenne muscular dystrophy field for 20 years, presented the 12-month results of the randomized, open-label, early stage Phase I/II HOPE clinical trial. The trial was designed to evaluate safety and explore efficacy. It enrolled 25 boys and young men in advanced stages of Duchenne muscular dystrophy and was conducted at three U.S. centers. All participants had significant cardiac scarring and approximately two-thirds were wheelchair-dependent at the time they began the trial.

During the 12-month course of the trial, all patients received standard-of-care for Duchenne muscular dystrophy, including oral steroids, and 13 also received one dose of intracoronary CAP-1002 upon randomization. CAP-1002 consists of allogeneic cardiosphere-derived cells which have been reported to improve muscle function and increase new muscle cell generation in preclinical models of Duchenne muscular dystrophy.

To assess skeletal muscle function, investigators used the Performance of the Upper Limb test (PUL). The PUL tests manual tasks that relate to activities of daily living that are very important for quality of life. PUL has been validated for the assessment of upper limb motor function in individuals with Duchenne muscular dystrophy. The functional tasks of the PUL are subdivided into three subscales reflecting disease progression from proximal to distal (from the middle of the body outward): (1) High-level: shoulder dimension; (2) Mid-level: elbow dimension; and (3) Distal-level: wrist and finger dimension.

As shoulder function had already been lost in most of the HOPE participants, investigators used the combined Mid-Distal PUL subscales to assess changes in skeletal muscle function and found significant improvement in those treated with CAP-1002 (defined post-hoc). Among the lower-functioning patients (baseline Mid-Distal PUL < 55 out of 58), investigators reported sustained or improved motor function in 8/9 (89%) of the CAP-1002 treated patients as compared to 0/4 (0%) of the usual care participants, at 12 months (p=0.007).

To assess cardiac structure and function, investigators used magnetic resonance imaging (MRI). They found significant improvements in cardiac muscle function among those treated with CAP-1002, according to measures of systolic thickening of the left ventricular wall. Systolic thickening is thought to be a principal mechanism of cardiac output generation in people with Duchenne muscular dystrophy.

In the inferior wall, they recorded a mean (SD) 31.2% (47.0%) increase in thickening six months after treatment and a mean 25.8% (46.7%) increase in thickening 12 months after treatment. In comparison, the usual care group showed a mean 8.8% (27.7%) decrease at six months and a mean 1.6% (37.9%) increase at 12 months in the systolic thickening of the inferior wall. The difference between the groups at six months achieved statistical significance (p=0.04; p=0.09 at 12 months).

Investigators also found that scarring of the heart muscle among those treated with CAP-1002 decreased relative to the control group. Progressive cardiac scarring eventually impairs the heart's pumping ability and is currently the leading cause of death in Duchenne muscular dystrophy. At the 12-month follow-up, those treated with CAP-1002 had a mean 7.1% (10.3%) reduction in scar size, in contrast to a mean 4.8% (22.3%) increase in scar size in the usual care group, a difference that achieved statistical significance (p=0.03).

CAP-1002 was generally safe and well-tolerated in the HOPE trial. There was no significant difference in the incidence of treatment-emergent adverse events in either group. There were no early study discontinuations due to adverse events.

"These 12-month results extend our prior findings with CAP-1002 and further support its potential to serve those with Duchenne muscular dystrophy," Linda Marbán, Ph.D., Capricor president and CEO, said. "Pending regulatory clearance, we plan to initiate the randomized, double-blind, placebo-controlled HOPE-2 clinical trial in the first quarter of 2018. HOPE-2 will evaluate multiple doses of CAP-1002 given intravenously, and the primary efficacy analysis will be driven by changes in skeletal muscle function as assessed by the PUL."

Drs. Victor and Marbán are scheduled to participate in a conference call and webcast at 4:30 p.m. ET today to review the results presented at the AHA meeting. To participate, please dial (866) 939-3921 (domestic) or (678) 302-3550 (international) and reference the access code 45894703. Slides to accompany the call may be viewed via the webcast link at http://wsw.com/webcast/cc/capr5. Access to the live webcast as well as the link to the replay of the call can be found at http://capricor.com/news/events/. The webcast will be archived for approximately 30 days.

The HOPE trial was funded in part by the California Institute for Regenerative Medicine.

About Duchenne Muscular Dystrophy

Duchenne muscular dystrophy is a devastating genetic disorder that causes muscle degeneration and leads to death, generally before the age of 30, most commonly from heart failure. It occurs in one in every 3,600 live male births across all races, cultures and countries. Duchenne muscular dystrophy afflicts approximately 200,000 boys and young men around the world. Treatment options are limited, and there is no cure.

About CAP-1002

CAP-1002 consists of allogeneic cardiosphere-derived cells, or CDCs, a unique population of cells that contains cardiac progenitor cells. CAP-1002 has been shown to exert potent immunomodulatory activity and stimulate cellular regeneration. CDCs have been the subject of over 100 peer-reviewed scientific publications and have been administered to approximately 140 human subjects across several clinical trials.

About Capricor Therapeutics

Capricor Therapeutics, Inc. (NASDAQ: CAPR) is a clinical-stage biotechnology company focused on the discovery, development and commercialization of first-in-class biological therapeutics for the treatment of rare disorders. Capricor's lead candidate, CAP-1002, is an allogeneic cell therapy that is currently in clinical development for the treatment of Duchenne muscular dystrophy. Capricor has also established itself as one of the leading companies investigating the field of extracellular vesicles and is exploring the potential of CAP-2003, a cell-free, exosome-based candidate, to treat a variety of disorders. For more information, visit www.capricor.com.

Cautionary Note Regarding Forward-Looking Statements

Statements in this press release regarding the efficacy, safety, and intended utilization of Capricor's product candidates; the initiation, conduct, size, timing and results of discovery efforts and clinical trials; the pace of enrollment of clinical trials; plans regarding regulatory filings, future research and clinical trials; regulatory developments involving products, including the ability to obtain regulatory approvals or otherwise bring products to market; plans regarding current and future collaborative activities and the ownership of commercial rights; scope, duration, validity and enforceability of intellectual property rights; future royalty streams, expectations with respect to the expected use of proceeds from the recently completed offerings and the anticipated effects of the offerings, and any other statements about Capricor's management team's future expectations, beliefs, goals, plans or prospects constitute forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Any statements that are not statements of historical fact (including statements containing the words "believes," "plans," "could," "anticipates," "expects," "estimates," "should," "target," "will," "would" and similar expressions) should also be considered to be forward-looking statements. 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 Capricor's business is set forth in Capricor's Annual Report on Form 10-K for the year ended December 31, 2016 as filed with the Securities and Exchange Commission on March 16, 2017, in its Registration Statement on Form S-3, as filed with the Securities and Exchange Commission on September 28, 2015, together with the prospectus included therein and prospectus supplements thereto, and in its Quarterly Report on Form 10-Q for the quarter ended September 30, 2017, as filed with the Securities and Exchange Commission on November 14, 2017. All forward-looking statements in this press release are based on information available to Capricor as of the date hereof, and Capricor assumes no obligation to update these forward-looking statements.

CAP-1002 is an Investigational New Drug and is not approved for any indications. Capricor's exosomes technology, including CAP-2003, has not yet been approved for clinical investigation.

For more information, please contact:

AJ Bergmann, Vice President of Finance
+1-310-358-3200
abergmann@capricor.com

Exhibit 99.2

Cardiosphere-Derived Cells for the Treatment of Duchenne Cardiomyopathy: Results of the Halt cardiOmyopathy ProgrEssion [HOPE]-Duchenne Trial

Ronald Victor, John Jefferies, Michael Taylor, Joao Lima, Rachel Smith, Konstantinos Malliaras, Brian Fedor, Jeff Rudy, Janice Pogoda, Linda Marban, Deborah Ascheim, Eduardo Marban

CEDARS-SINAI
HEART INSTITUTE

Disclosures

HOPE-Duchenne Trial sponsored by Capricor, Inc.

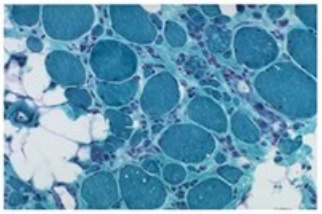
Ronald Victor, MD

- **Capricor:** Steering Committee, Site PI
- **Catabasis Pharmaceuticals:** Site PI
- **Coalition Duchenne:** Research Grant PI
- **Eli Lilly:** Steering Committee Chair, Global PI, Site PI

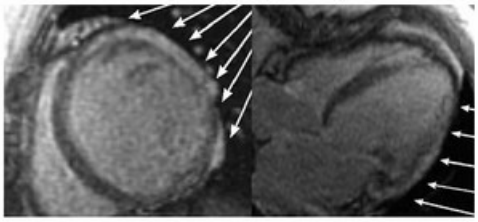
Duchenne Muscular Dystrophy

- *Dystrophin* mutations
- X-linked recessive
- Muscle wasting disease
- Patchy progressive fibrosis

Skeletal myopathy

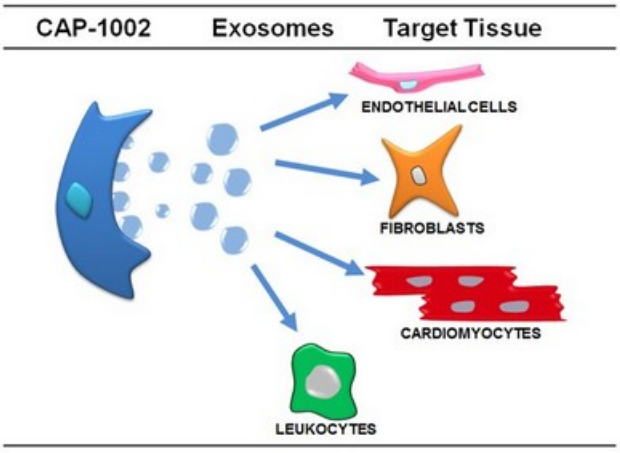


Cardiomyopathy

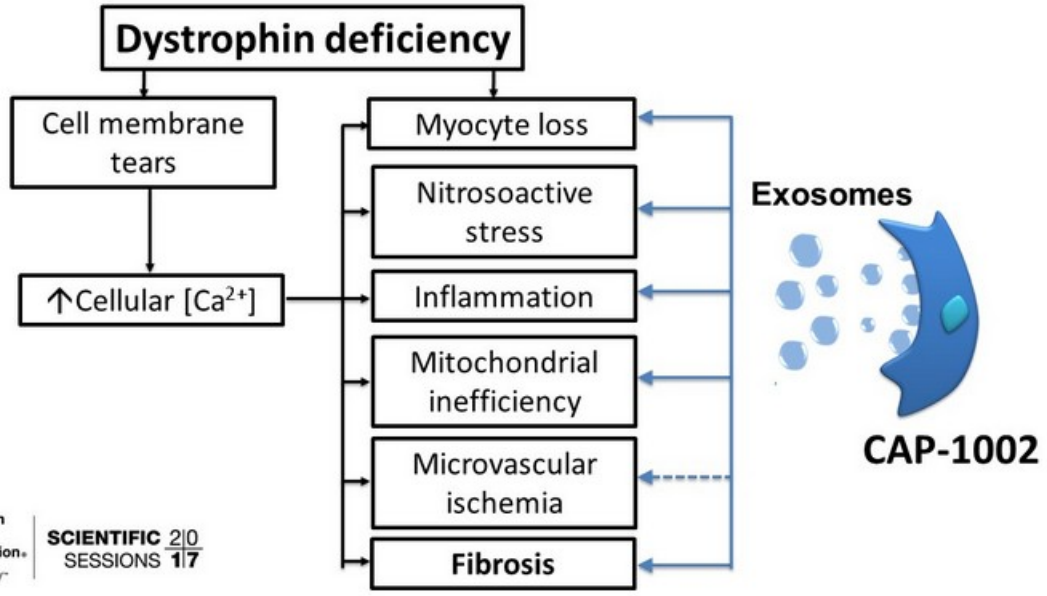


CAP-1002: Background

- Allogeneic cardiosphere-derived cells (CDCs) from donated heart muscle
- >100 peer-reviewed papers since 2007
- Clinically investigated (>100 patients)
- Does **not** engraft into host tissue



CAP-1002 to Target Multiple Pathways in DMD



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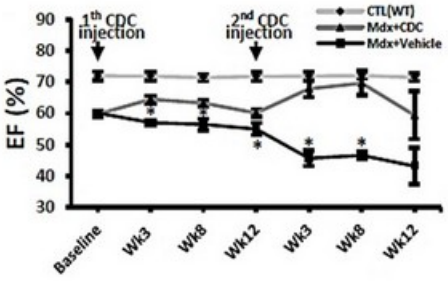
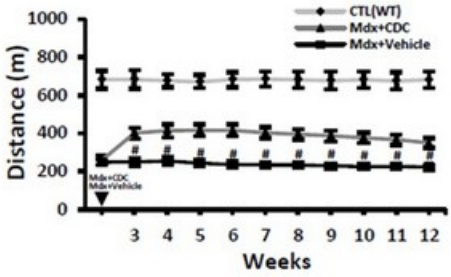
SCIENTIFIC 2|0 SESSIONS 1|7

Intracardiac CDCs in *mdx* Mouse Model of DMD

Improved cardiac function



Improved exercise capacity

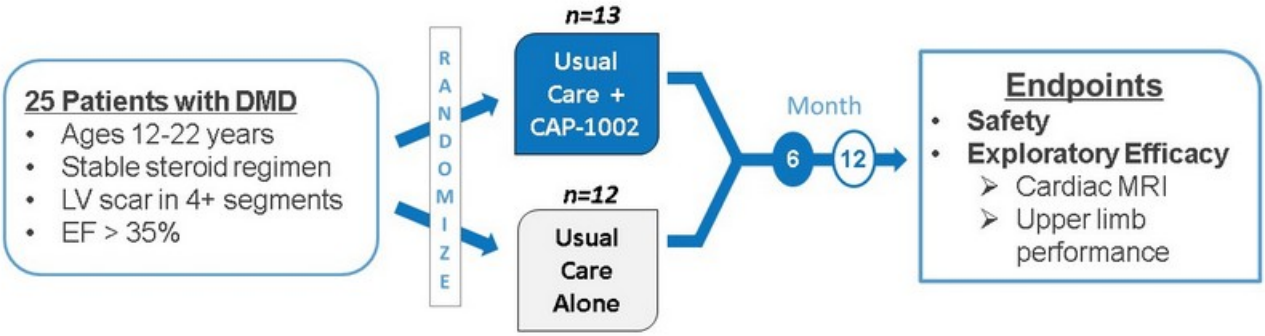


Aminzadeh et al., 2017. Preprint: http://biorxiv.org/content/early/2017/04/20/128900

The challenge: ***clinical translation…***

HOPE-Duchenne Clinical Trial Design

- Phase 1/2A Randomized Open Label Trial
- One-time, multi-vessel, intracoronary delivery of 75 M cells
- Safety trial with multiple exploratory efficacy endpoints
- Sites: Cedars-Sinai, Cincinnati Children's, U. of Florida



Baseline Characteristics

	Usual Care (n=12)	CAP-1002 (n=13)
Age, years	16.9 (2.75)	18.7 (3.5)
Non-Ambulatory	7 (58.3%)	10 (76.9%)
Cardiac Scar (SD)	21.39% (10.75)	17.55% (6.79)
LVEF (SD)	48.39% (7.49)	49.58% (6.69)
IC Dose (M cells)	n/a	73.7 (3.56)

Data are mean (SD)

Safety Outcomes

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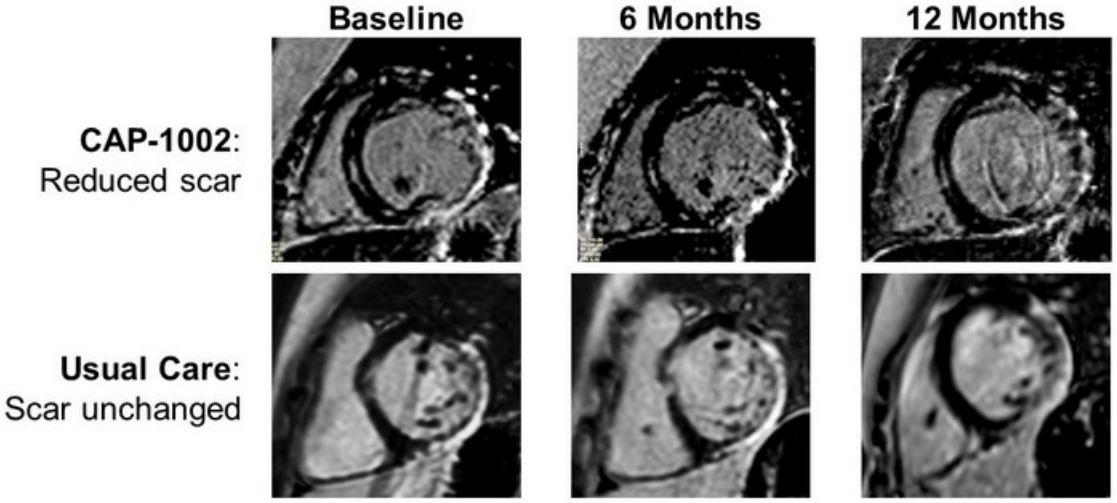
SCIENTIFIC SESSIONS 2017

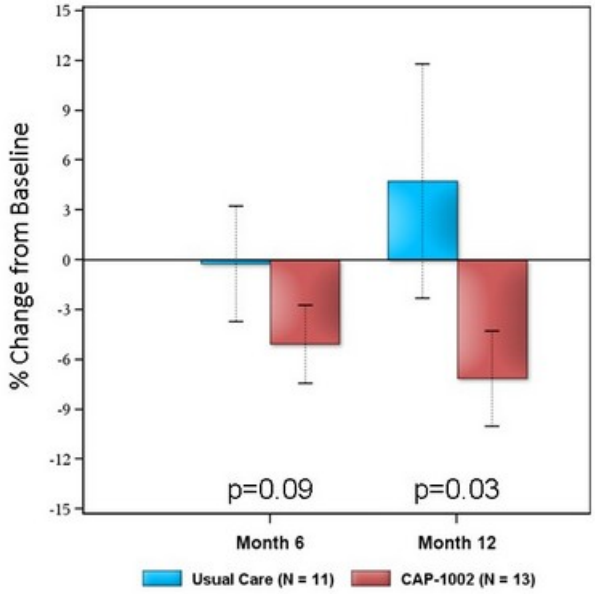
- CAP-1002 was generally safe and well-tolerated
- SAEs observed in both groups
  - Usual Care: Femur Fracture
  - CAP-1002: UTI, Fever & Confusion, Ventricular Fibrillation (pre-infusion)
- AE's consistent with an intracoronary infusion procedure
  - Transient atrial fibrillation in 5/13 in CAP-1002 group
  - Peri-procedural cardiac troponin (cTn) elevation*
    - 13/13 in CAP-1002 group (vs. 2/12 in Usual Care group)
    - Observed elevations at baseline c/w underlying disease

*cTn elevation defined as > 5x composite 99th percentile (0.03 pg/mL) or ≥ 20% of elevated baseline

Efficacy Endpoints: Reduced Cardiac Scar Size (LGE)



Reduced Myocardial Scar by Cardiac MRI



- Blinded analysis by core lab
- By Month 12, scar increased in the Usual Care group but decreased in the CAP-1002 group
  - ➢ 11.9% group difference in change score (p=0.03)
- Decreased scar is counter to the natural history of DMD.

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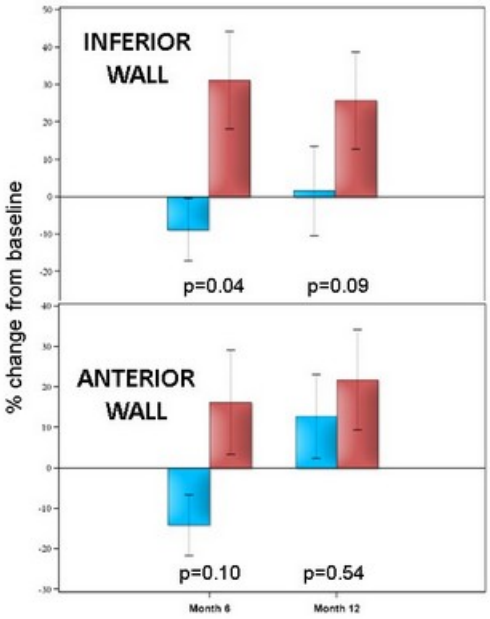
SCIENTIFIC SESSIONS 2017

Improved Regional Systolic Wall Thickening



- No effect detected in overall EF
- Most evidence of improvement in **inferior wall**
- Similar trend in anterior wall
- Lesser trends in lateral and septal walls

➢ Consistent with natural history of scar progression in DMD

Inferior → Anterior → Lateral → Septal

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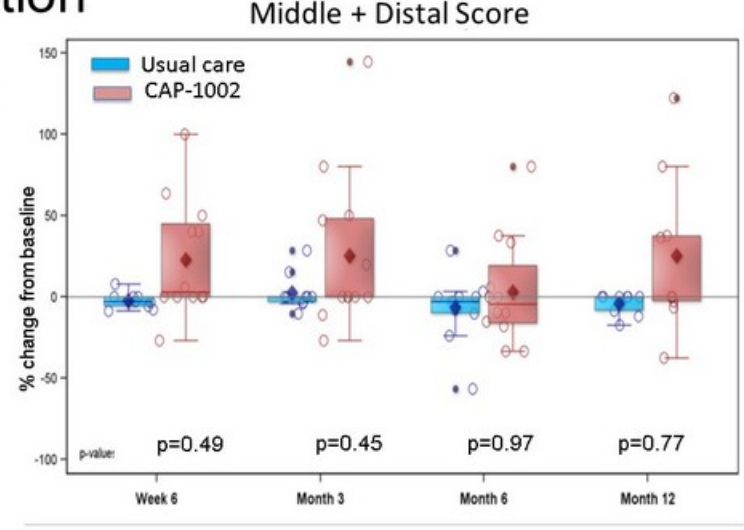
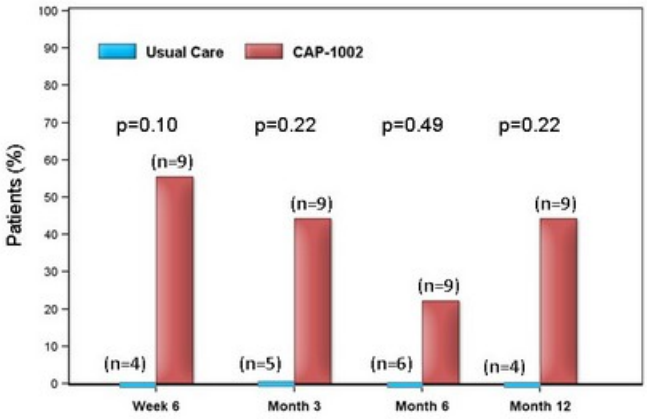
Performance of the Upper Limb (PUL): Skeletal Muscle Function

PUL – Clinically meaningful, activities of daily living, important to patients

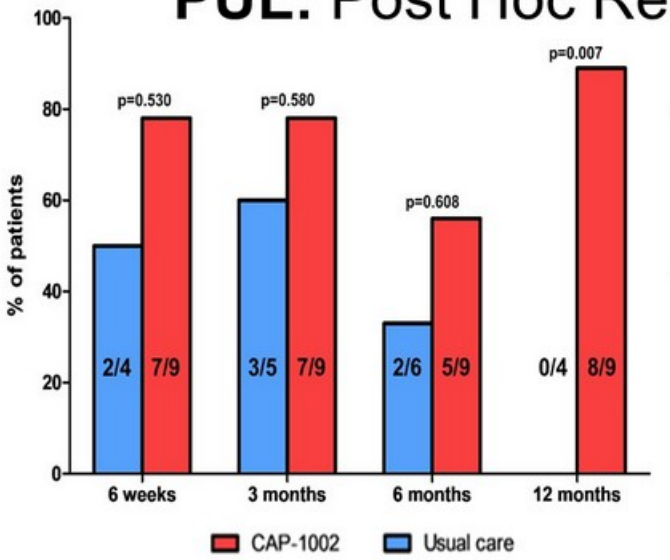


PUL: Post Hoc Responder Analysis

- Middle + Distal PUL
- Subgroup of patients with baseline middle + distal PUL score < 55 (maximum = 58)
- Responder defined as ≥ 10% from baseline or max possible improvement



PUL: Post Hoc Responder Analysis



- Subset of patients with baseline middle + distal PUL score < 55 (more advanced disease)
- At 12 months, mid-distal PUL score sustained or improved in 89% of CAP-treated patients vs. none in Usual Care group (p=0.007)

Conclusions

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SCIENTIFIC SESSIONS 2017

- CAP-1002 delivered via IC infusion was generally safe and well-tolerated
- These early clinical data are consistent with preclinical studies showing CAP-1002 benefits both cardiac and skeletal muscle in DMD.
- Exploratory efficacy analyses signal a potential benefit of CAP-1002 for patients with advanced DMD.

*Planned "**HOPE-2**" trial:*

- *Similar patient population*
- *Intravenous delivery Q3 months*
- *Evaluate skeletal and cardiac muscle function*
- *Enrollment to begin in 1Q 2018*

Acknowledgements

- Funded in part by the California Institute for Regenerative Medicine (CIRM)
- Coalition Duchenne
- CureDuchenne
- Parent Project Muscular Dystrophy

- Site Principal Investigators
  - John Jefferies
  - Barry Byrne
- Site Interventional Cardiologists
  - Raj Makkar
  - Bryan Goldstein
  - James Fudge

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Exhibit 99.3

Intracoronary ALLogeneic Heart STem Cells to Achieve Myocardial Regeneration (ALLSTAR): A Randomized, placebo-controlled, double-blind trial

Timothy D. Henry, Dean J. Kereiakes, Glenn Kowalchuk, Frank Aguirre, Konstantinos Malliaris, Anthony DeMaria, Gary Francis, Thomas J. Povsic, Richard Schatz, Jay H. Traverse, Tarun Chakravarty, Janice Pogoda, Paula Williams, Jeff Rudy, Rachel D. Smith, Linda Marbán, Deborah D. Ascheim, Eduardo Marbán, Raj R. Makkar

Cedars-Sinai Heart Institute

Disclosures

Trial Sponsors: Capricor, Inc.; NIH/NHLBI (Phase I - 1RC3HL103356 ARRA grant)

Funded in part by California Institute for Regenerative Medicine (CIRM; Phase 2)

Co-PIs Timothy D. Henry & Raj Makkar

Steering Committee

Anthony DeMaria, MD – Chair
Gary S. Francis, MD
Frank Aguirre, MD
Thomas Povsic, MD, PhD
Richard Schatz, MD
Eduardo Marbán, MD, PhD – Advisor

Introduction

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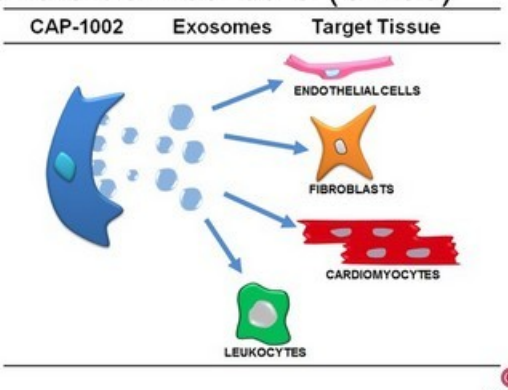
- Previous trials have demonstrated potential benefit of stem cell therapy in patients with recent MI
- Phase 1 data with autologous cardiosphere-derived cells (CDCs) demonstrated scar size reduction and evidence for myocardial regeneration
- Allogenic CDCs (CAP-1002) are equivalent to autologous CDCs in preclinical studies
- Over 100 peer reviewed papers regarding CAP-1002 since 2007



CS Cedars-Sinai Heart Institute

Rationale for Using CDCs to Treat Post MI Cardiomyopathy

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- Diverse effects of CDCs support their potential to retard or reverse the multiple pathological processes that contribute to post-MI cardiomyopathy

Pathophysiology		CDCs
Oxidative/nitrosative stress	⬅	Anti-oxidative
Inflammation	⬅	Anti-inflammatory
Apoptosis	⬅	Anti-apoptotic
Remodeling	⬅	Anti-remodeling
Loss of cardiomyocytes	⬅	Regenerative

Cedars-Sinai Heart Institute

CADUCEUS: Phase I Autologous CDCs

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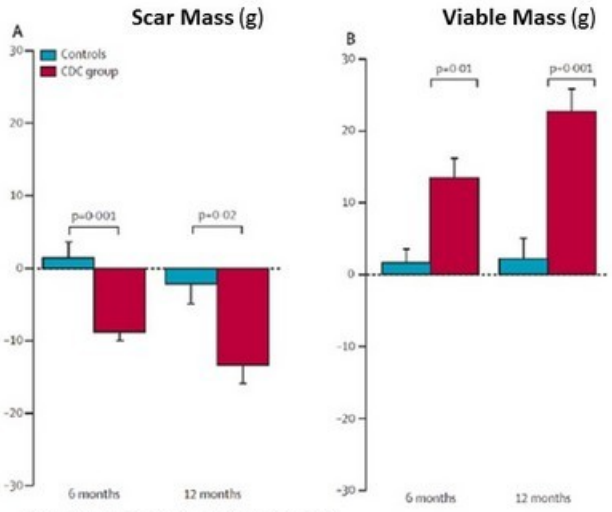
- Cedars-Sinai & Johns Hopkins
- Recent MI
- LVEF 25-45% following successful PCI
- 2:1 allocation to autologous CDCs or routine care
- Dose escalation 12.5-25M
- cMRI at baseline, 6, 12 months



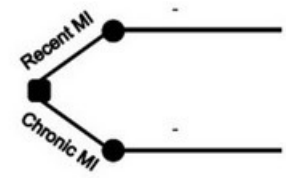
Makkar RR, et al., Lancet. 2012
Malliaras K, et al., J Am Coll Cardiol. 2014

Cedars-Sinai Heart Institute

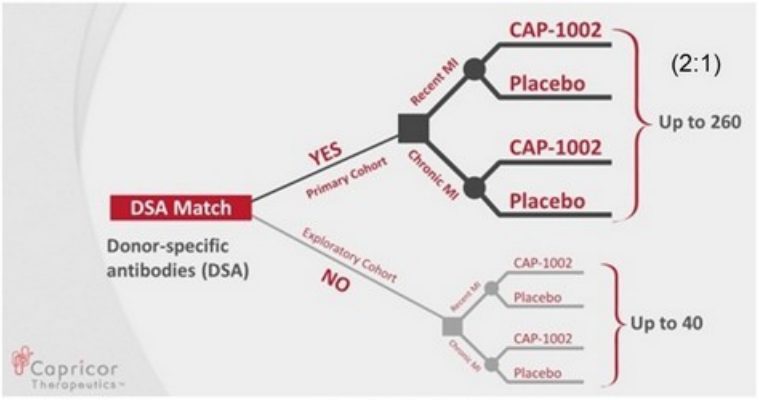
ALLSTAR Trial Design

American Heart Association. life is why™ | SCIENTIFIC SESSIONS 2|0 1|7

Phase 1	DSMB Review	Phase 2



Open Label Safety Cohort (N=14)



* Funded by NIH ARRA RC3

* Funded by CIRM

Cedars-Sinai Heart Institute

Key ALLSTAR Eligibility Criteria

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Inclusion Criteria

- History of STEMI or NSTEMI w/in 12 mo
- Successful PCI (TIMI flow = 3) in infarct related artery
- LVEF ≤ 45%
- LV infarct size ≥ 15% of LV mass in qualifying infarct-related region (by MRI core lab)
- No further revascularization needed
- Age ≥ 18 years

Exclusion Criteria

- Prior CABG
- Hx ACS within 4 wks prior to infusion
- Hx previous stem cell therapy
- Prior ICD or pacemaker at site not certified to conduct cMRI with device
- Estimated GFR < 30 mL/min
- Participation in another clinical trial w/in the last 30 days
- Current alcohol or drug abuse

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ALLSTAR Open Label Phase I Efficacy

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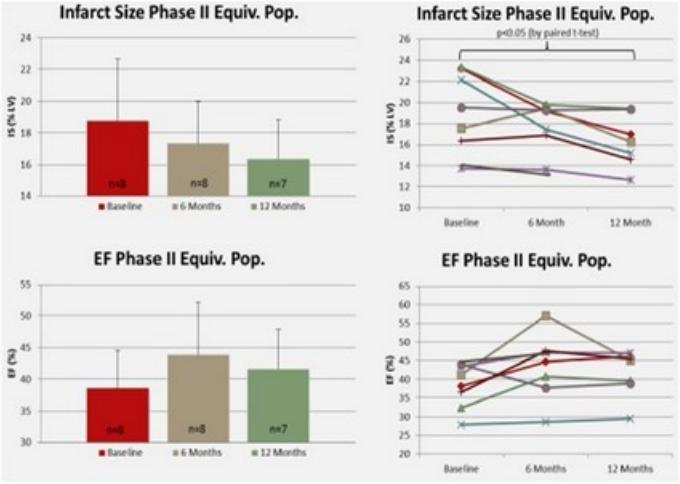
15% reduction in scar size at one year

4% improvement in EF at one year



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ALLSTAR Phase II Efficacy Endpoints

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- **Primary efficacy endpoint**: % change from baseline in infarct size (cMRI as a % of LV mass) at 12 months

- **Secondary efficacy endpoint**: absolute and % change from baseline in LV structure and function, clinical function, and cardiac biomarkers at 6 and 12 months post-infusion

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ALLSTAR Phase II Enrollment & Baseline Characteristics

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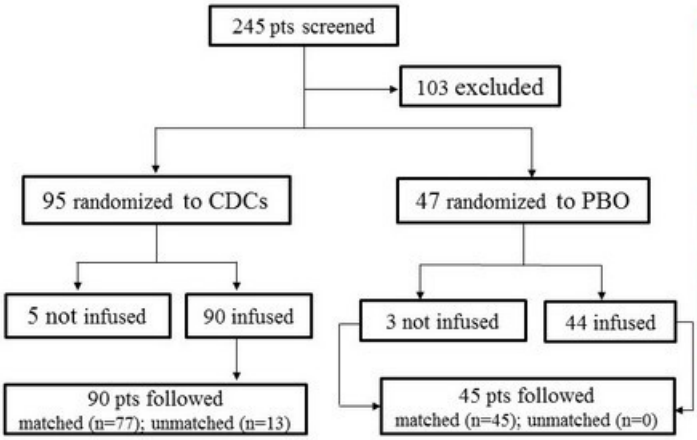

	CAP-1002 (N = 90)	Placebo (N = 44)
Sex, male	84.4%	86.4%
Age [mean (SD)]	54.8 (11.25)	53.8 (10.23)
LVEF [mean (SD)]	39.9% (6.62)	38.7% (8.12)
LV Scar [mean (SD)]	21.9% (5.15)	23.0% (5.19)
LVESV [mL (SD)]	129.6 (39.4)	140.2 (46.2)
LVEDV [mL (SD)]	213.1 (47.4)	225.5 (52.4)
NTproBNP [pg/mL (SD)]	883.8 (1122.8)	736.2 (700.9)
MI other than index	15.6%	15.9%

12 mos of on-study observation followed by 4 years of LT F/U

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Interim Analysis

- Pre-specified interim analysis – after all subjects observed ≥ 6 months
  - All data for 134 treated subjects (Primary Cohort, n=121; Exploratory Cohort, n=13)
- Given low probability that treatment effect would be observed in primary 12 month efficacy analysis, all subjects were transitioned to annual follow-up

Change in Scar Size by MRI

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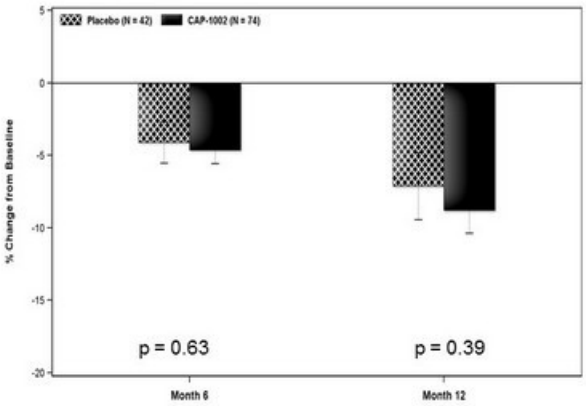
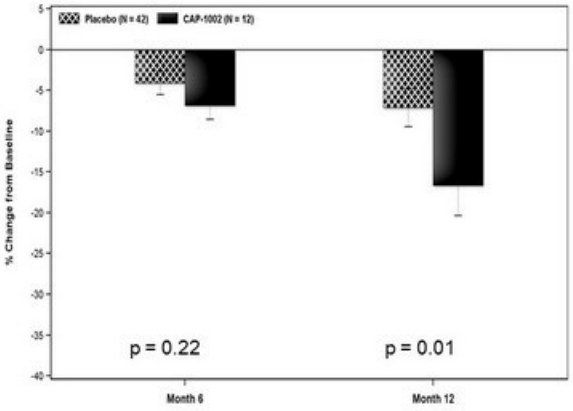
Matched

Primary mITT Population



Unmatched

Exploratory CAP-1002 & Placebo mITTs



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LV Volumes

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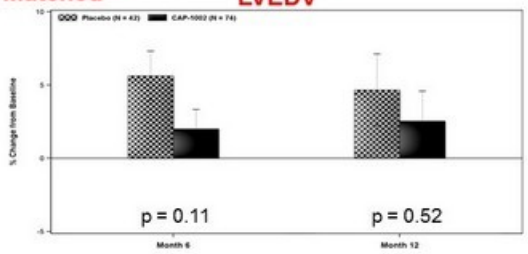
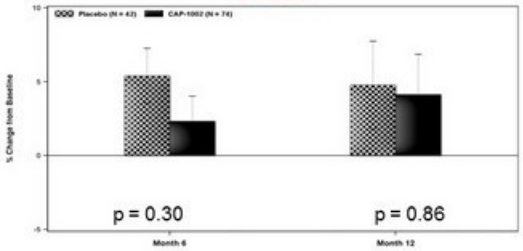
"Matched"

LVEDV



LVESV



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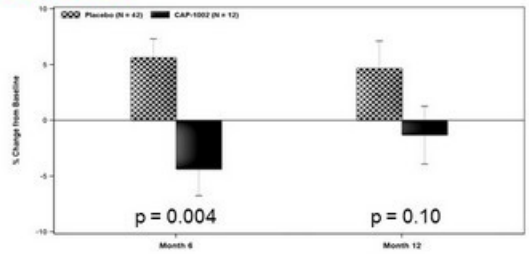

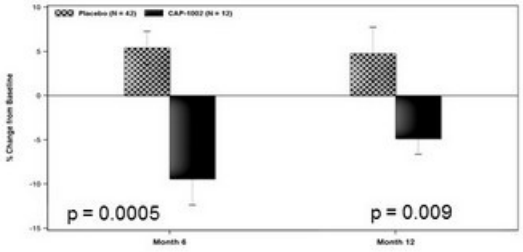

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NT-proBNP

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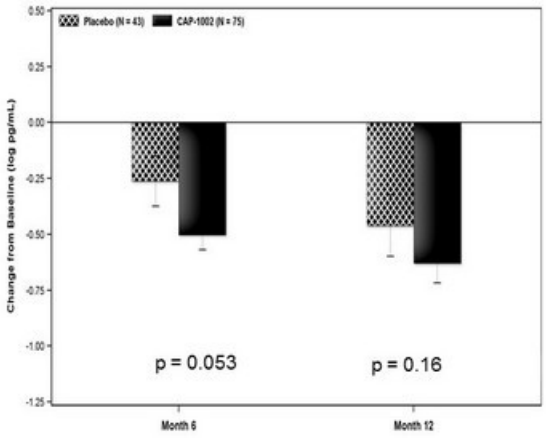
"Matched"



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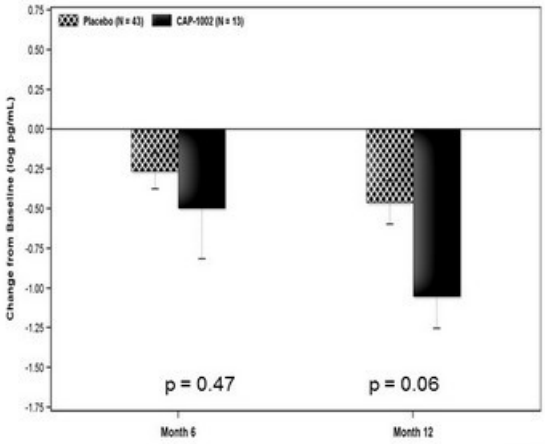

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Overall Outcomes
(all CAP-1002 vs placebo, 6 months)

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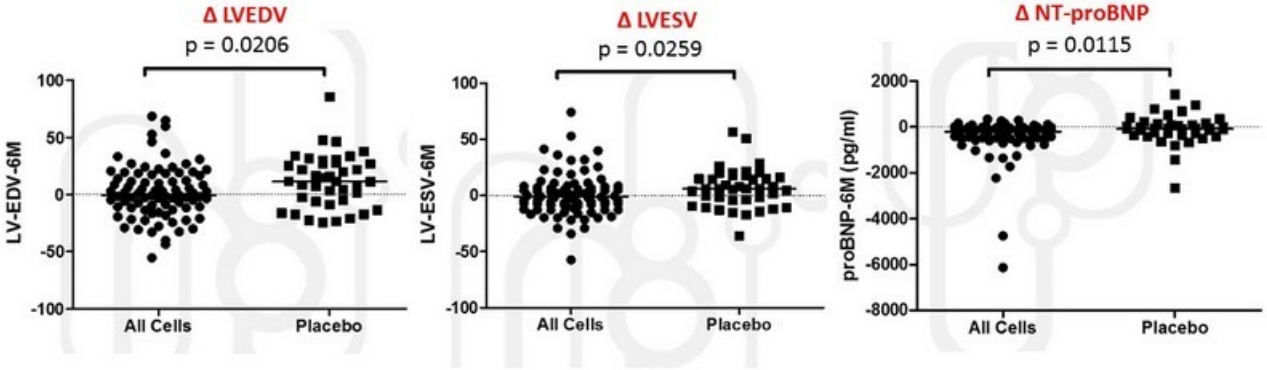

Deltas from Baseline to 6 months. Post hoc analysis, Mann-Whitney tests, 2-tailed

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Safety and Clinical Events

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Safety

- No primary safety endpoint events were observed during the study
- No significant treatment group difference in SAE rates

Clinical Events (adjudicated)

Clinical Events [n (%)]	CAP-1002 (N = 90)	Placebo (N = 44)	p-value
Death	0(0.0)	0(0.0)	--
Non-fatal MI	3 (3.3)	4 (9.1)	0.22
HF Hosp	4 (4.4)	3 (6.8)	0.68
Stroke	0 (0.0)	1 (2.3)	0.33
MACE	7 (7.8)	5 (11.4)	0.49

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Conclusions

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- No significant difference in scar size based on 6 or 12 month MRI
- Signs of improvement in LV volumes and BNP
- Very low clinical events → favoring the treatment group
- Challenges
  - Recruitment of patients with large anterior MI
  - MRI endpoint dropout and variability
- To be investigated: Influence of matched vs unmatched cells

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