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Advancing Restorative Therapies to Treat Ischemic Disease

David J. Mazzo, PhD
President and Chief Executive Officer

Forward-looking statement

This Investor Presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Forward-looking statements reflect management's current expectations, as of the date of this presentation, and involve certain risks and uncertainties. All statements other than statements of historical fact contained in this Investor Presentation are forward-looking statements. The Company's actual results could differ materially from those anticipated in these forward-looking statements as a result of various factors. Factors that could cause future results to differ materially from the recent results or those projected in forward-looking statements include the "Risk Factors" described in the Company's Annual Report on Form 10-K filed with the Securities and Exchange Commission ("SEC") on March 14, 2019, as subsequently amended on March 19, 2019, and in the Company's other periodic filings with the SEC. The Company's further development is highly dependent on, among other things, future medical and research developments and market acceptance, which are outside of its control. You are cautioned not to place undue reliance on forward-looking statements, which speak only as of the date of this Investor Presentation. Caladrius does not intend, and disclaims any obligation, to update or revise any forward-looking information contained in this Investor Presentation or with respect to the matters described herein.

Presentation contents

- Investment case summary
- Management team introduction
- CD34+ cell therapy platform technology overview
- Pipeline description and individual program summaries
- Financial overview
- Milestone timeline
- Conclusion

Caladrius investment case summary

- CD34+ cell therapy platform company with an advanced clinical pipeline with two programs with cell therapy “breakthrough” designation
- Proprietary field-leading technology in multi-billion dollar global indications backed by a strong IP portfolio
- Multiple potential value creating events in the next 12 months based on development milestones across the pipeline
- Seasoned management team with noteworthy domain expertise along with big pharma and emerging biotech experience
- Strong balance sheet; $29.2 million in cash (September 30, 2019) with no debt and cash runway projected to 2Q 2021

Caladrius management team

David J. Mazzo, PhD

President and
Chief Executive Officer

RegadoBiosciences
CHUGAI
Schering-Plough
Hoechst
RHÔNE-POULENC RORER

Douglas Losordo, MD

EVP, Global Head of R&D and
Chief Medical Officer

Baxter
Northwestern Medicine
Feinberg School of Medicine
Tufts University School of Medicine

Joseph Talamo, CPA

Senior VP and
Chief Financial Officer

(OSI) pharmaceuticals
Bristol-Myers Squibb
KPMG

Todd Girolamo, JD

Senior VP, General Counsel
and Corporate Secretary

100 years CAHILL
Cahill Gordon & Reindel LLP
Thelen Reid & Priest LLP
Attorneys At Law
OPPENHEIMER
CIBC

John Menditto

Vice President, IR and
Corporate Communications

Argos Therapeutics
medco
NOVARTIS

Note: Select experience is shown above. For a comprehensive bio, please visit: www.caladrius.com

Esteemed cardiovascular disease scientific advisory board

C. Noel Bairey Merz, MD
Cedars-Sinai, Los Angeles

C. Michael Gibson, MD
Harvard Medical School

Timothy Henry, MD
The Christ Hospital, Cincinnati

Thomas Povsic, MD, PhD
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Scripps Clinic, San Diego

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Stanford Cardiovascular Institute

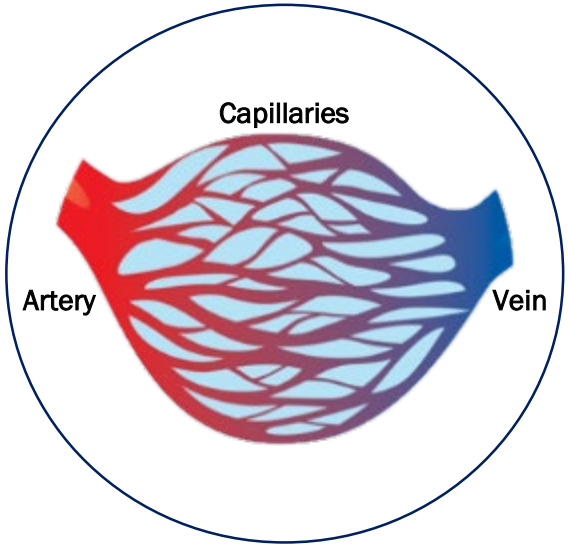
Andreas Zeiher, MD
Goethe University, Frankfurt

Zan Fleming, MD
Executive Chairman, Kinexum

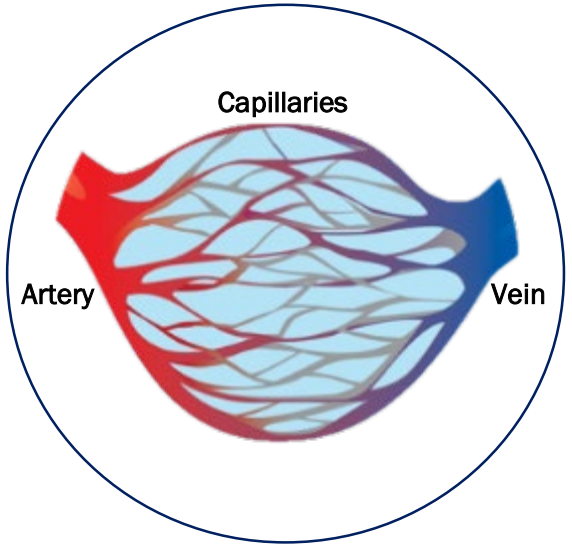
CD34+ cell therapy platform technology overview

CD34+ cells have a well characterized mechanism of action

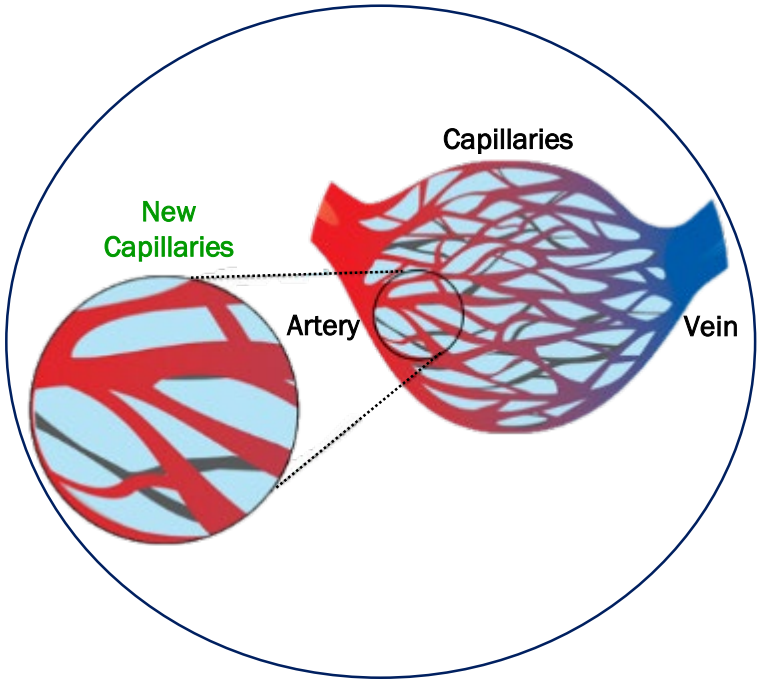
Normal microvasculature



Compromised microvasculature



Augmented microvasculature post-CD34+ cells treatment



- Naturally occurring vascular repair (endothelial progenitor) cell
- Provokes restorative angiogenesis of the microvasculature
- CD34+ cells reestablish blood flow to under-perfused tissues[1,2]

[1]Mackie, A.R. et al., Tex Heart Inst J 2011, 38(5), 474-485
[2]Kocher, A.A. et al., *Nat Med* 2001, 440-436

CD34+ cell therapy is extensively studied/clinically validated

- CD34+ cells were clinically studied in multiple ischemic disease indications by numerous investigators across many sites and countries
- Consistent and compelling results of rigorous clinical studies comprising >1,000 patients have been published in peer reviewed journals[1,2,3,4]
- Single treatment has elicited durable therapeutic effect
- No cell-related adverse events reported to date

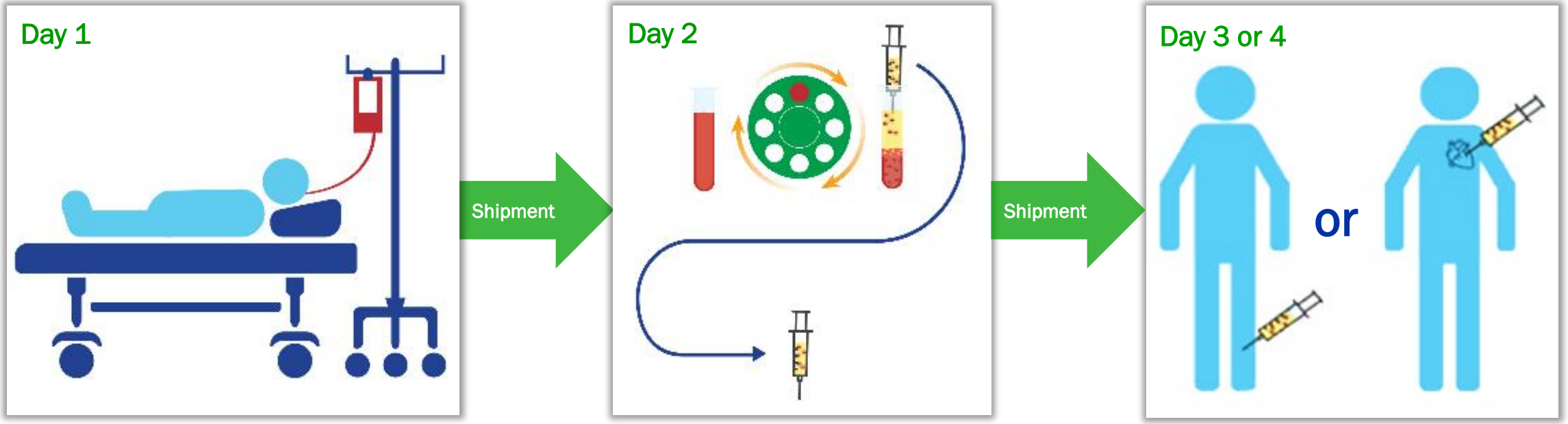
[1] Povsic, T. et al. *JACC Cardiovasc Interv*, 2016, 9 (15) 1576-1585
[2] Losordo, D.W. et al. *Circ Cardiovasc Interv,* 2012; 5:821–830
[3] Velagapudi P, et al, *Cardiovas Revasc Med*, 2018, 20(3):215-219
[4] Henry T.D., et al, *European Heart Jour* 2018, 2208–2216

Caladrius CD34 process is simple/fast/scalable/economical

Sample collection via apheresis after 5 days of GCSF mobilization

Isolation, concentration and formulation of CD34+/CXCR4+ cells

Cells returned to same patient by injection; site indication dependent



- GCSF mobilization eliminates need for surgical bone marrow aspiration
- No genetic manipulation or *ex vivo* expansion of cells
- Four days or less from donation to treatment
- Cost-of-goods an order of magnitude less expensive than CAR-T therapies

Caladrius CD34 technology has robust intellectual property

Fundamental protection to 2031+

9
U.S. patents granted

23
Foreign patents granted

Key Claims

- Pharmaceutical composition of non-expanded CD34+/CXCR4+ stem cells
- Therapeutic concentration range
- Stabilizing serum
- Repair of injury caused by vascular insufficiency

Caladrius' innovative CD34+ cell therapy pipeline*

Product	Indication	Development Stage	Commercialization Target
CLBS12	CLI	Registration eligible Phase 2 *(Japan; ongoing)*	2021
CLBS16	CMD	Proof-of-concept *(USA; ongoing)*	TBD
CLBS14	NORDA	Phase 3 confirmatory *(USA; initiation pending funding)*	TBD

CLI = Critical Limb Ischemia
CMD = Coronary Microvascular Dysfunction
NORDA = No Option Refractory Disabling Angina

*Products are distinct and not interchangeable

CLBS12

Critical Limb Ischemia

(Japan)

SAKIGAKE designated – Japan

Advanced Therapeutic Medicinal Product (ATMP) designated - EU

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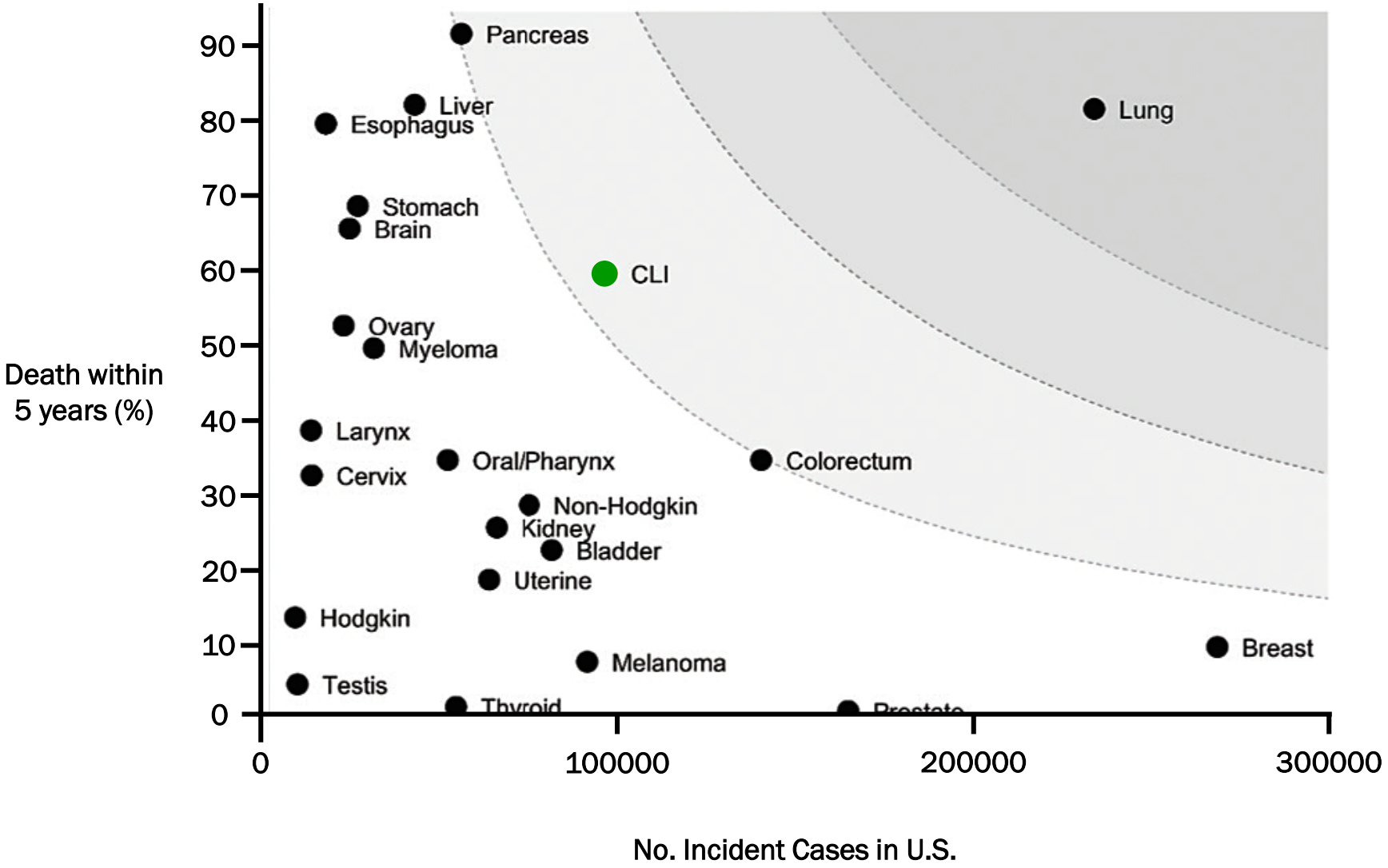
Indication: Critical Limb Ischemia (CLI)

- Severe arterial obstruction impeding blood flow in the lower extremities
  - Often found as a co-morbidity in diabetes patients
  - Includes severe rest pain and non-healing ulcers
- Buerger's disease (inflammation in small and medium arteries) also causes CLI; exacerbated by a history of heavy smoking
- Patients with no-option CLI have persistent symptoms even after bypass surgery, angioplasty, stenting and available pharmacotherapy
- CLI patients are at high risk of amputation and increased risk of death
- Multi-billion dollar global commercial opportunity

CLI: higher mortality rate than most cancers



Mustapha, J. A., Katzen, B. T., et al. (2019, May). Endovascular Today, 18(5), 80-82

CLI amputation rates increase with disease severity[1]

Rutherford ("R") scale
R 6: Functional foot no longer salvageable
R 5: Minor tissue loss non-healing ulcer; focal gangrene with diffuse pedal ischemia
R 4: Debilitating rest pain
R 1-3: CLI-free

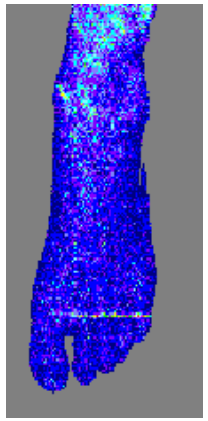
CLBS12 targets patients with **R4** or **R5** disease

[1] Reinecke H., European Heart Journal, 2015 Apr 14;36(15):932-8

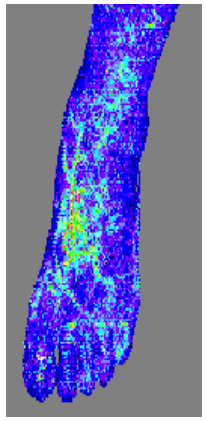
Single treatment of CD34+ cells reversed CLI

Actual CLI Patient Laser Doppler Image

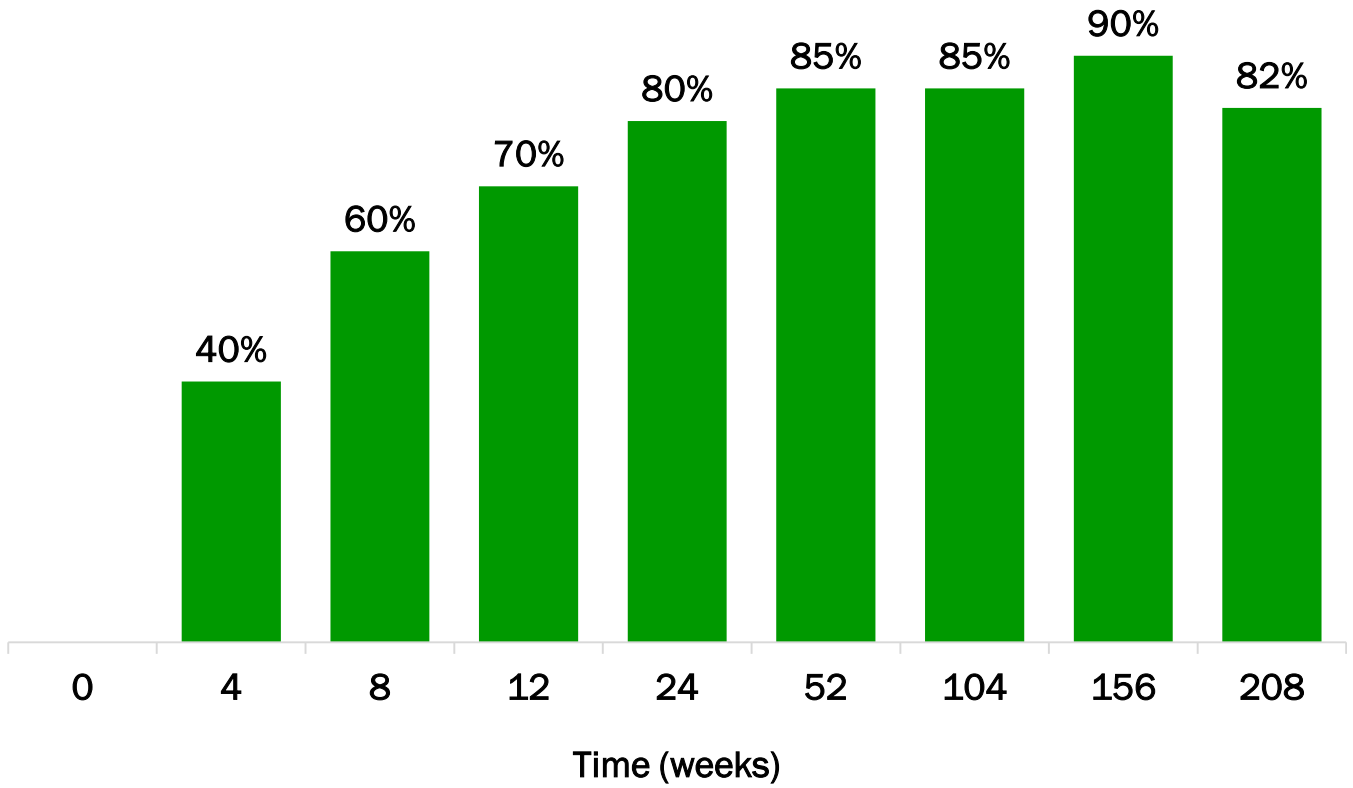
Pre-treatment



Post-treatment (week 12)



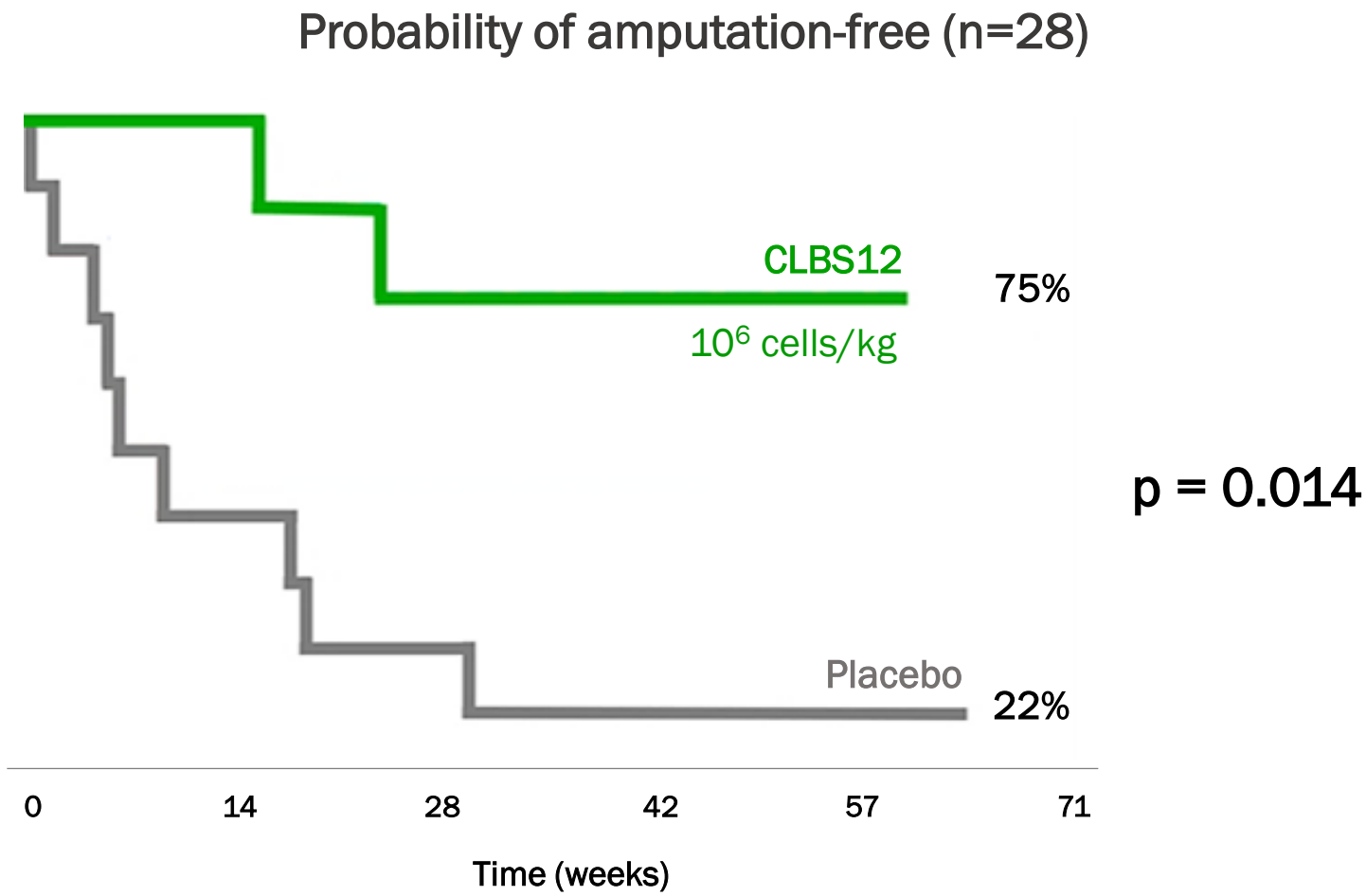
% of Patients (CLI + BD) Achieving CLI-free Status



~80% of patients achieved sustainable remission within 6 months of a single treatment; durable for at least 4 years

Kinoshita et al, Atherosclerosis 224 (2012) 440-445

Single treatment of CD34+ cells increased amputation-free survival

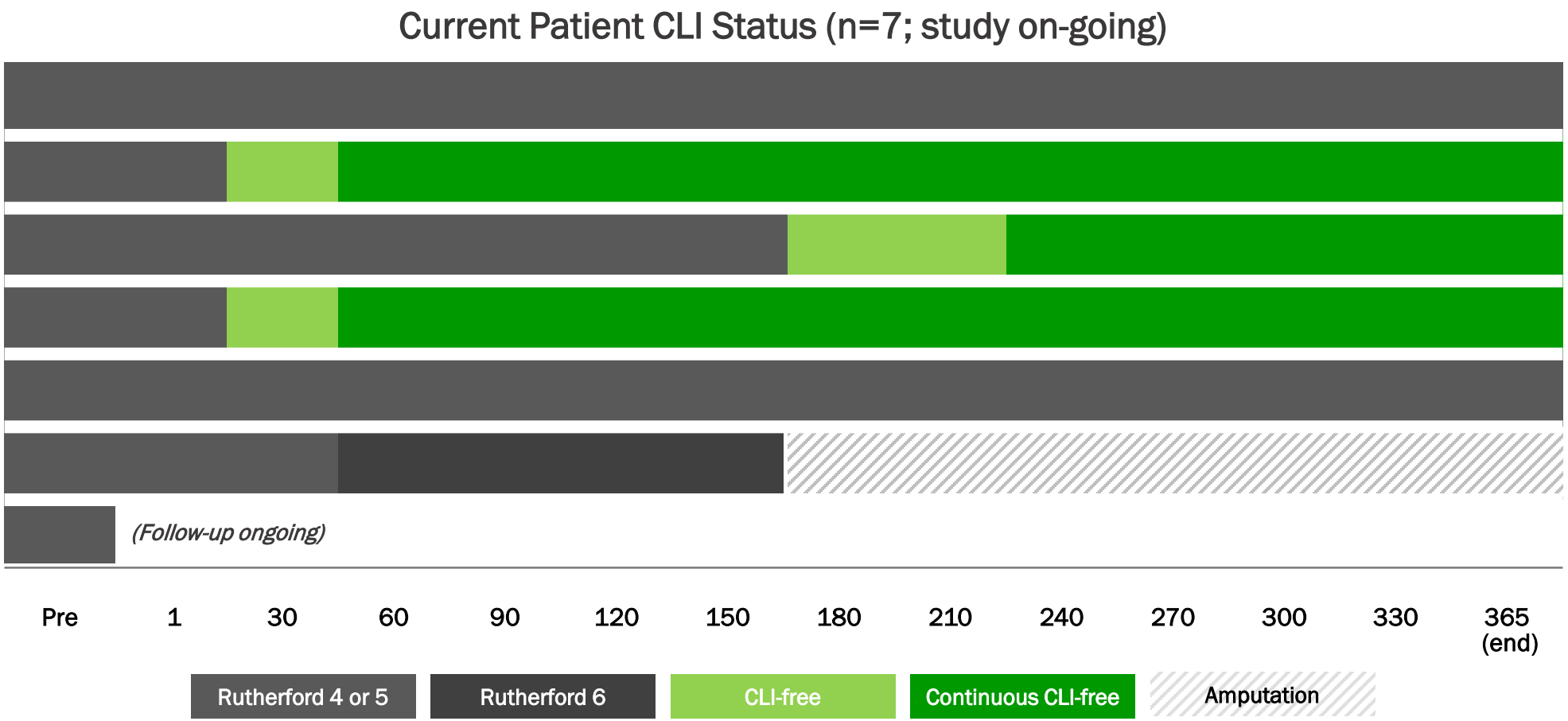


Losordo, D.W. et al, Circulation 2012; 5(6):821-830

CLBS12 registration-eligible study (Japan)

Primary Endpoint	▪ Continuous CLI-free (2 consecutive monthly visits, adjudicated independently)
Study Size	▪ 30 subjects with no-option CLI + 7 Buerger's Disease pts.; all R4 or R5; 12 centers in Japan
Dose	▪ 10^6 cells/kg (CLBS12) per affected limb (studied in previous trial)
Control/Comparator	▪ Standard of Care: wound care plus drugs approved in Japan • Including antimicrobials, antiplatelets, anticoagulants and vasodilators
Mode of administration	▪ Intramuscular, 20 injections in affected lower limb in a single treatment
Timing/Costs	▪ Results expected end 2020/early 2021 ▪ Earliest possible commercialization 2021 ▪ Study funded to completion in current budget projections

Extraordinary CLBS12 results in Buerger's Disease (Japan)



- Natural evolution of Buerger's Disease is continual deterioration for all patients
- Surgery is not viable and existing pharmacotherapies do not prevent amputation[1]
- To date, CLBS12 treatment has resulted in 50% of patients achieving a positive outcome

[1] Cacione DG, et al, Pharm. treatment of Buerger's Disease, Cochrane Database of Systematic Reviews, 2016, (3) CD011033

CLBS16

Coronary Microvascular Dysfunction

(USA)

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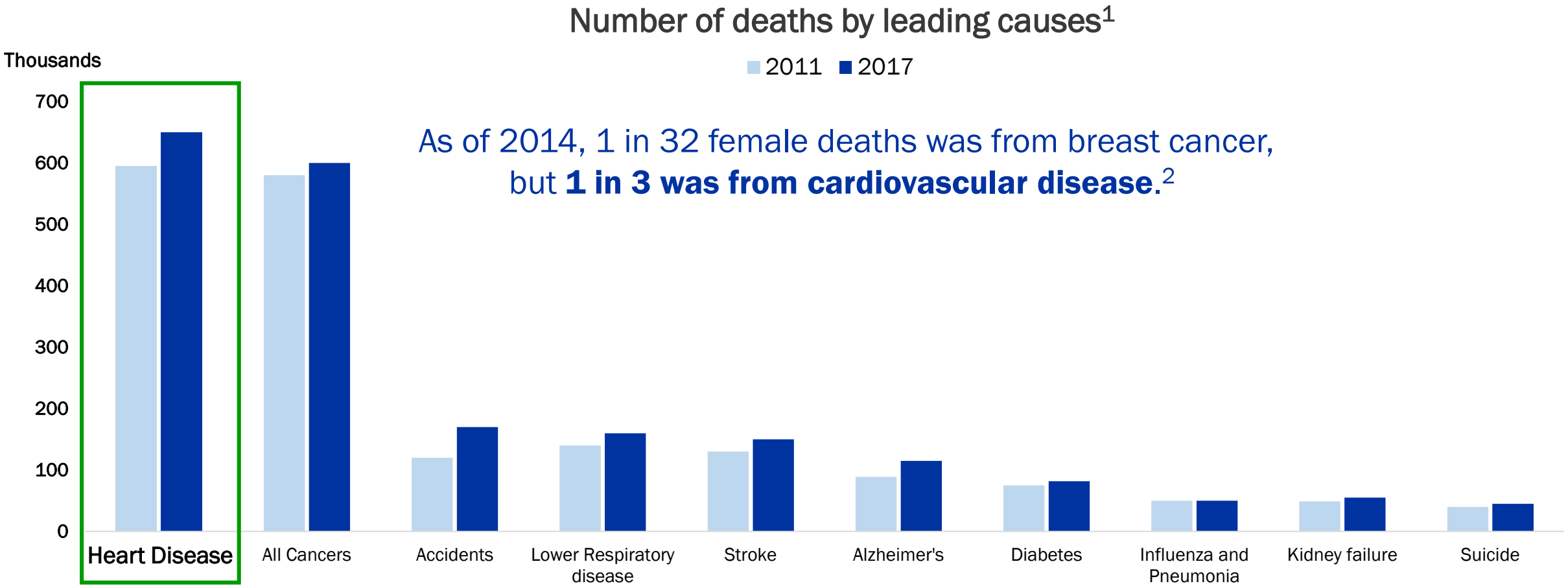
Heart disease: No. 1 cause of death in the U.S. and growing



As of 2014, 1 in 32 female deaths was from breast cancer, but **1 in 3 was from cardiovascular disease.**[2]

[1] Centers for Disease Control and Prevention as cited in McKay, Betsy. "Heart-Failure Deaths Rise, Contributing to Worsening Life Expectancy." The Wall Street Journal, 30 Oct. 2019, www.wsj.com/articles/heart-failure-deaths-rise-contributing-to-worsening-life-expectancy-11572411901.

[2] Kochanek, KD., et al. (2016). Deaths: final data for 2014. National vital statistics reports: from the Centers for Disease Control and Prevention, National Center for Health Statistics, National Vital Statistics System, 65(4), 1-122.

Indication: Coronary Microvascular Dysfunction (CMD)

- Deficient heart microvasculature *without* obstructive vessel disease
- Causes frequent, debilitating chest pain that is not treatable by stents or bypass; responds poorly or not at all to available medications
- Afflicts women more frequently, especially younger women[1,2]
- Results in poor prognosis for patients with the condition[3]
  - Significantly elevated risk of all-cause mortality in women[4]
- Quantitatively diagnosed using Coronary Flow Reserve (CFR)
  - CFR is the ratio of maximal to resting coronary blood flow[5]
- Multi-billion dollar global commercial opportunity

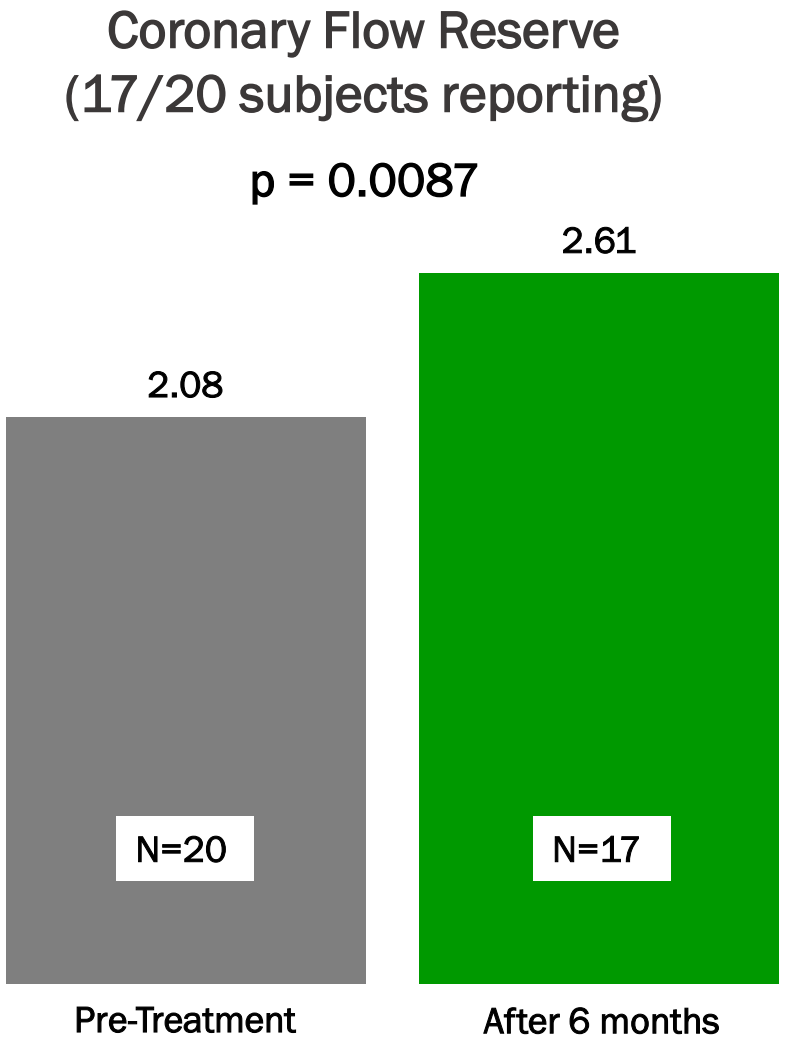
[1] Coronary Microvascular Disease. (2015, July 31). In American Heart Association
[2] R. David Anderson, John W. Petersen, Puja K. Mehta, et al., Journal of Interventional Cardiology, 2019: 8
[3] Loffler and Bourque, Curr Cardiol Rep. 2016 Jan; 18(1): 1
[4] Kenkre, T.S. et al., Circ: CV Qual & Outcomes 2017, 10(12) 1-9
[5] Collins, P., British heart journal (1993) 69(4), 279–281

ESCaPE-CMD: CLBS16 interventional, proof-of-concept trial

Endpoints	▪ Therapeutic effect and the evaluation of adverse events; including changes from baseline to 6 months for coronary flow reserve, endothelial-dependent microvascular function, time to angina; other CV metrics
Study Size	▪ 20 subjects (U.S. centers - Cedars Sinai, Los Angeles & Mayo Clinic, Rochester)
Dose	▪ Up to 300 x 10^6 CD34+ cells
Mode of administration	▪ Single intracoronary infusion
Timing/Cost	▪ Positive results reported at AHA on Nov. 16, 2019 (17/20 subjects) ▪ Full results expected by early 1Q 2020 ▪ Study funded to completion in current budget projections (including NIH grant)

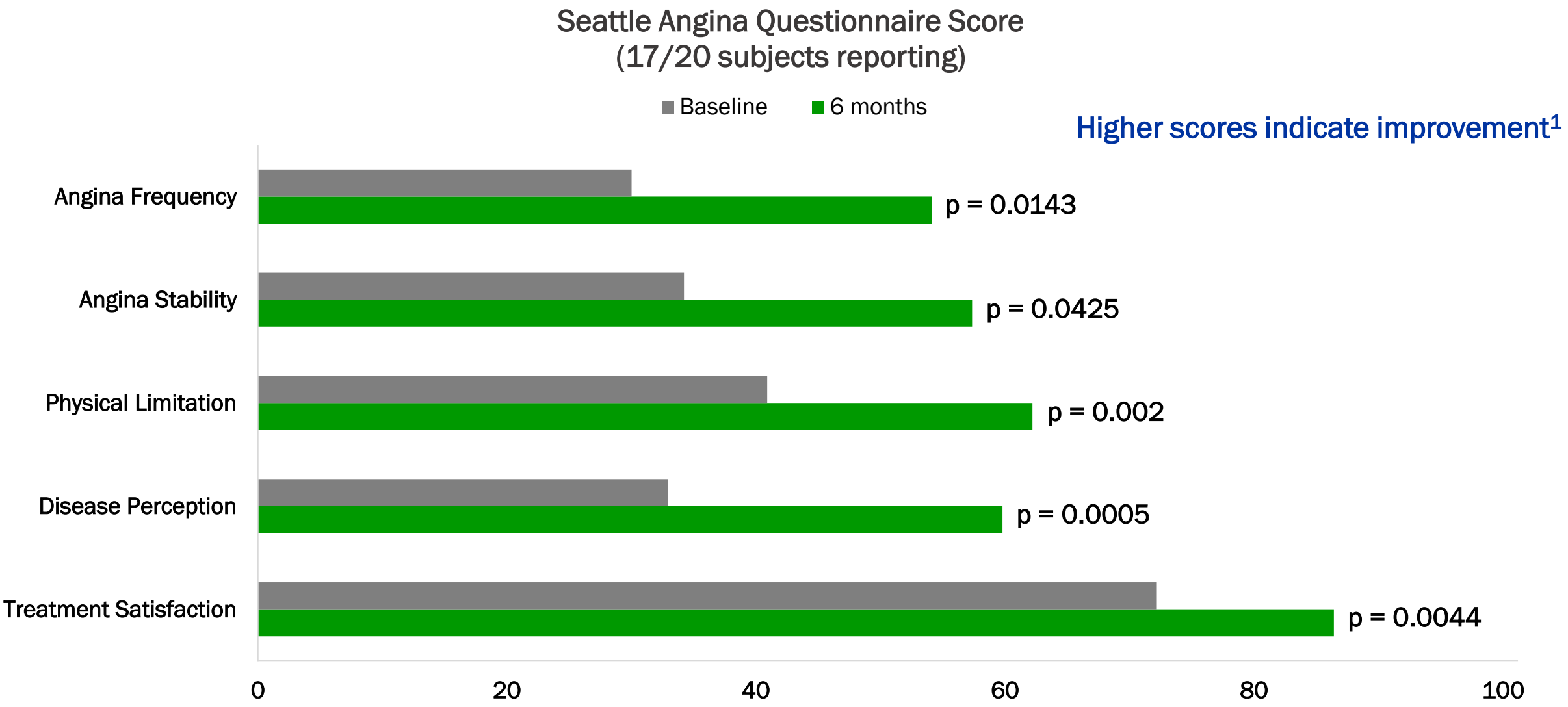
CLBS16 ESCaPE-CMD results are unique and compelling

- CFR≤2.5 indicates CMD
- CFR≥2.5 is in “normal” range
- Results after a single intracoronary administration of CLBS16



Bairey Merz, C. N., AHA 2019 Scientific Sessions

CLBS16 ESCaPE-CMD results are unique and compelling



[1]Spertus, J.A. et al, JACC Vol. 25, No. 2 February 1995: 333-341

ESCaPE-CMD CLBS16 reported results summary

- Statistically significant improvement in heart function and symptoms
- First therapy to show the ability to durably increase CFR and potentially reverse CMD after a single administration
- No evidence of cell related adverse events
- Expected to lead to a decreased risk of adverse cardiovascular outcomes, including CV-related death, associated with CMD
- Supports microvascular repair mechanism of CD34+ cells across all indications
- Represents a potential breakthrough for the treatment of CMD, a condition that affects millions in the U.S. and that disproportionately afflicts women

CLBS14

No Option Refractory Disabling Angina

(USA)

Regenerative Medicine Advanced Therapy (RMAT) designated - USA

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Indication: No Option Refractory Disabling Angina (NORDA)

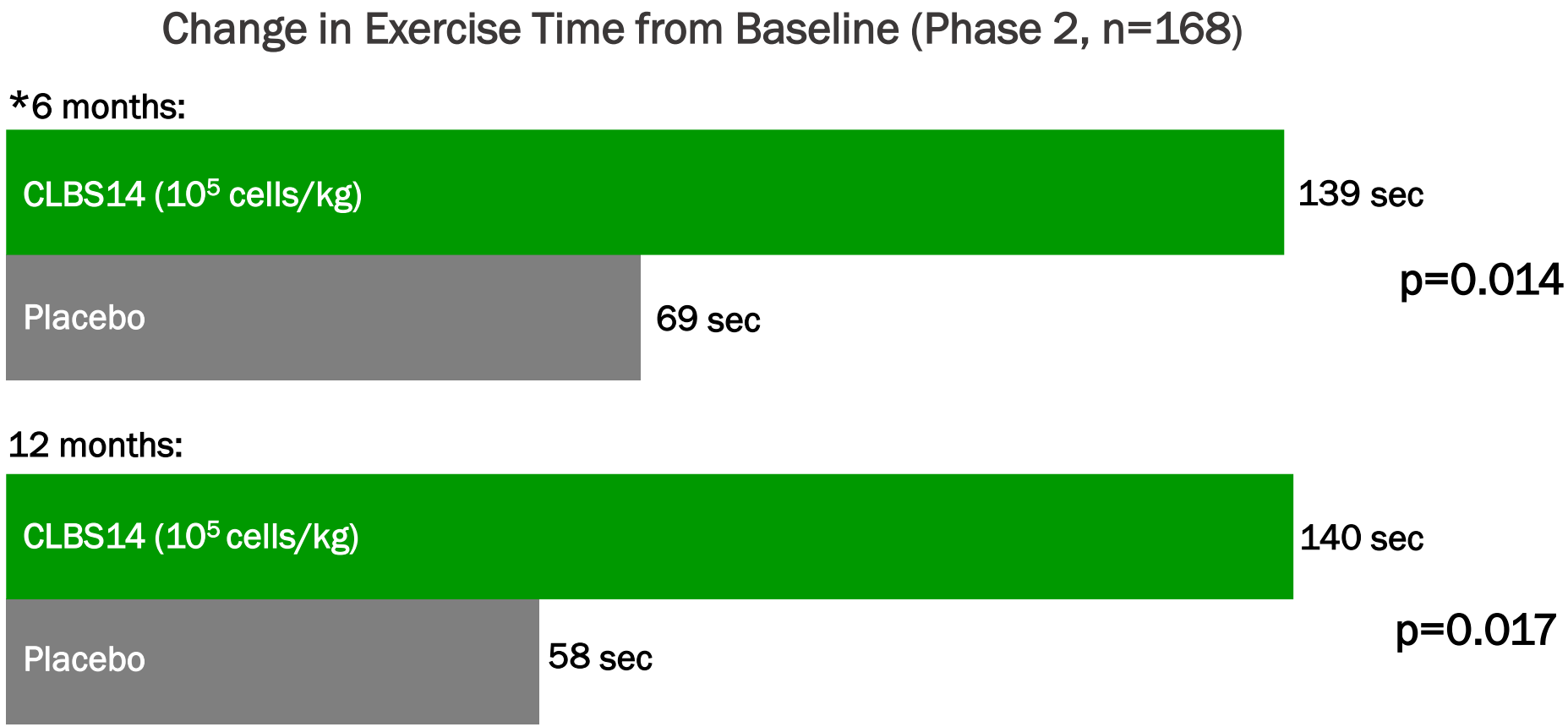
- Recurring angina results from chronically impaired cardiac blood supply
- The condition persists even after bypass surgery, angioplasty, stenting and available pharmacotherapy; no current treatment options
- NORDA patients experience very frequent disabling chest pain at rest or with minimal activity
- Cardiac microcirculation deficiency is the remaining treatment target
- Multi-billion dollar global commercial opportunity

Our solution: CLBS14

- Clinical data from double-blind, randomized, placebo-controlled clinical trials, including big pharma sponsored Phase 2 and partial Phase 3[1,2,3,4,5]
- Published results demonstrate:
  - Statistically significant improvement in exercise capacity
  - Statistically significant reduction in angina
  - Statistically significant reduction in mortality
  - Pristine cell safety profile

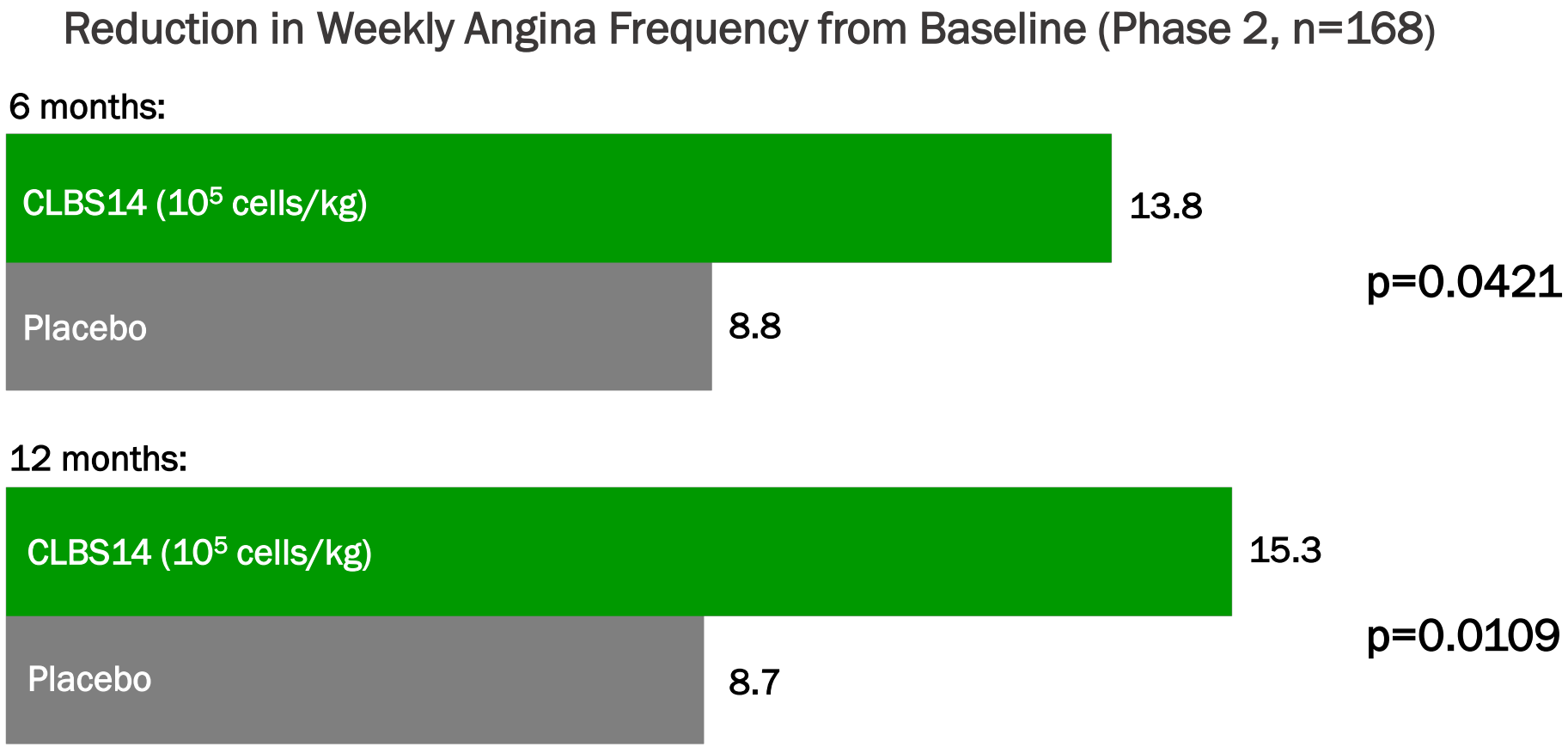
[1] Losordo, D.W., et al, Circulation 2007, 115(25): 3165-72.
[2] Losordo, D.W., et al, Circ Res 2011, 109(4): 428-36
[3] Povsic, T.J., et al, JACC Cardiovasc Interv, 2016 9(15): 1576-85
[4] Povsic, T. J. et al, European Heart Journal, 2018 39(23), 2208-2216
[5] Velagapudi P, et al, *Cardiovas Revasc Med*, 2018, 20(3):215-219

CLBS14 single treatment significantly improved exercise time



**Change in exercise time from baseline at 6 months will be the Phase 3 primary endpoint*

CLBS14 single treatment significantly reduced angina frequency

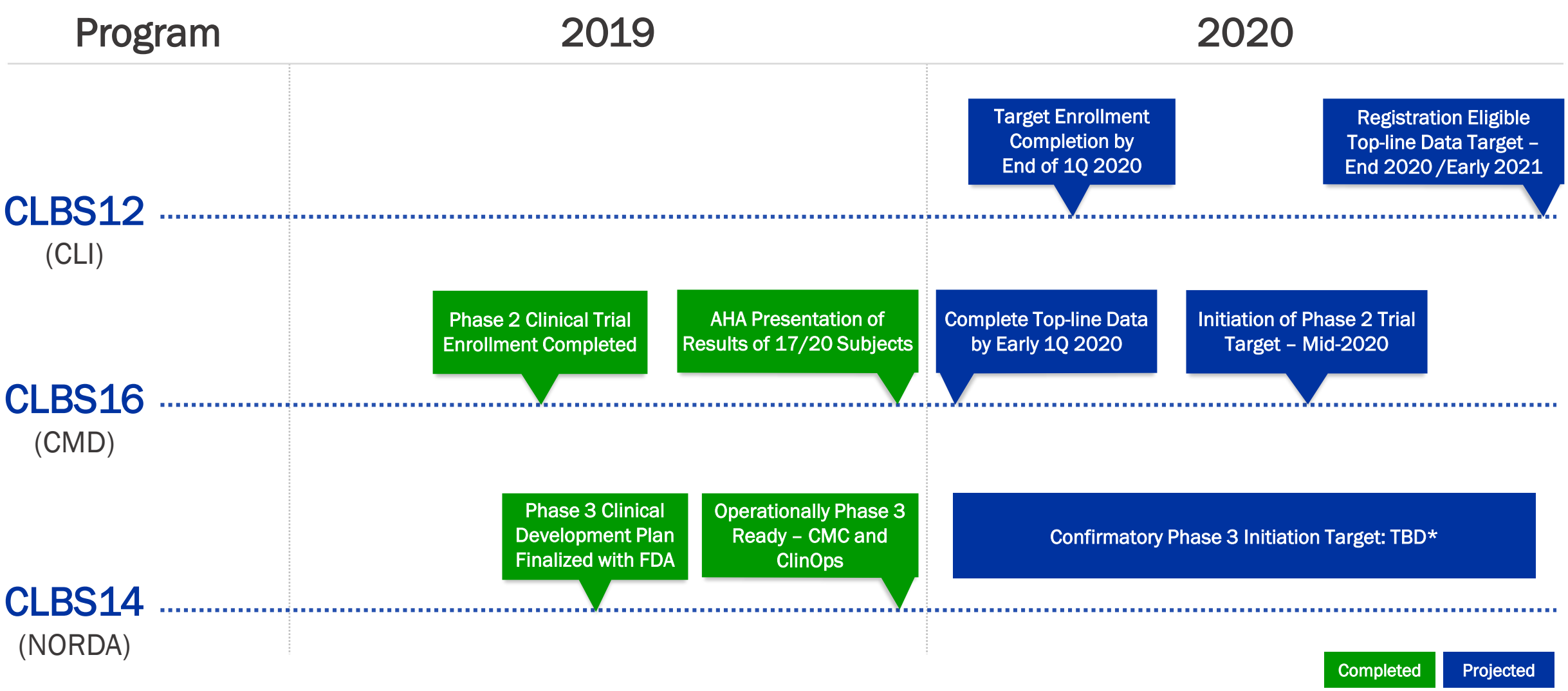


CLBS14 single treatment significantly improved survival

CLBS14 Phase 3 confirmatory registration study (U.S.)

Primary Endpoint	▪ Change in exercise time from baseline at month 6 (studied in Phase 2)
Timing	▪ 39 months from first-patient-in to top-line data; interim analysis after 50% of patients complete 6-month follow-up
Study Size	▪ ~400 subjects (~200 active, ~150 placebo, ~50 SOC with cross-over to open label treatment at 6 months)
Dose	▪ 10^5 cells/kg body weight (studied in Phase 2)
Control/Comparator	▪ Placebo control (blinded) ▪ Standard-of-care (unblinded)
Mode of administration	▪ Intramyocardial injection guided by mapping catheter (NOGA)
Timing/Costs	▪ External costs: ~$70 million over a 3-4 years period ▪ Target initiation: Upon acquisition of sufficient capital that provides confidence that the study could be funded through completion

Caladrius timeline of key development milestones



**Pending funding*

Caladrius key financial information

Cash & Investments as of September 30, 2019:	$29.2 million
Nine Months Ended September 30, 2019 Operating Cash Burn:	$14.7 million
Cash Runway Based on Current Plan:	Through 1Q 2021
Debt:	$0
Common Shares Outstanding as of September 30, 2019:	10.4 million shares
Options Outstanding as of September 30, 2019: Exercise Price < $5.00 = 647,000 shares Exercise Price > $5.00 = 448,000 shares	1.1 million shares

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Thank you!

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