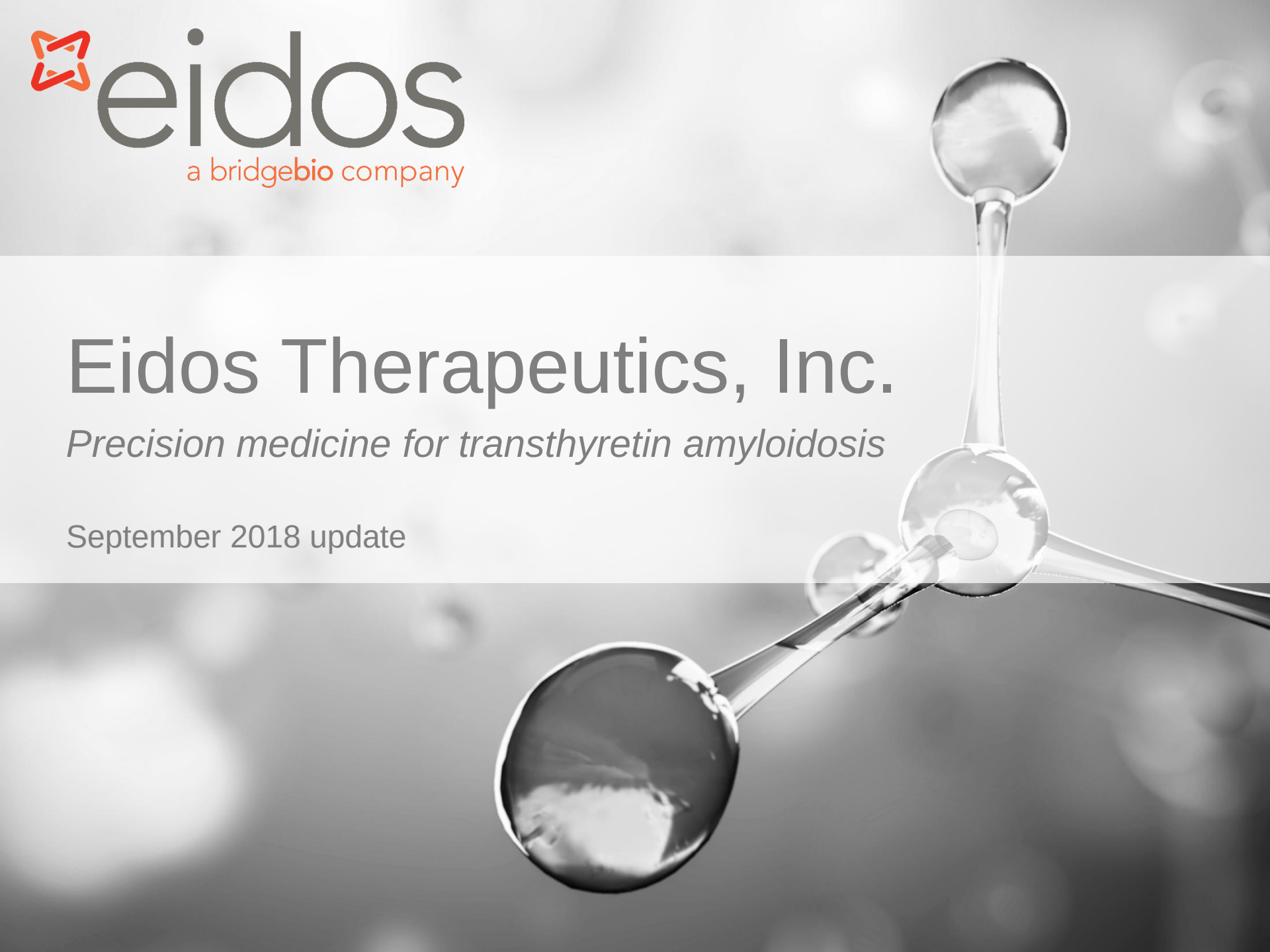
eidos
a bridgebio company

Eidos Therapeutics, Inc.

Precision medicine for transthyretin amyloidosis

September 2018 update



Eidos forward-looking statements

This presentation contains forward-looking statements about Eidos Therapeutics, Inc. (“we,” "Eidos" or the “Company”). All statements other than statements of historical facts contained in this presentation are forward-looking statements, including statements about our financial position, strategy, our expectations regarding the timing and achievement of our product candidate development activities, our ongoing and planned clinical trials, including the design of these trials and the availability of data from them, and plans and expectations for future operations. These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including, but not limited to: our limited operating history, net losses, our expectation that we will incur net losses for the foreseeable future, and that we may never be profitable; our need for additional funding and related risks for our business, product development programs and future commercialization activities; the timing and success of pre-clinical and clinical trials we conduct; the ability to obtain and maintain regulatory approval of our product candidates; the ability to commercialize our product candidates; our ability to compete in the marketplace; risks regarding our license agreements; our ability to obtain and maintain intellectual property protection for our product candidates; and our ability to manage our growth. In light of these and other risks, uncertainties and assumptions, the forward-looking events and circumstances discussed in this presentation may not occur and actual results could differ materially and adversely from those anticipated or implied in the forward-looking statements.

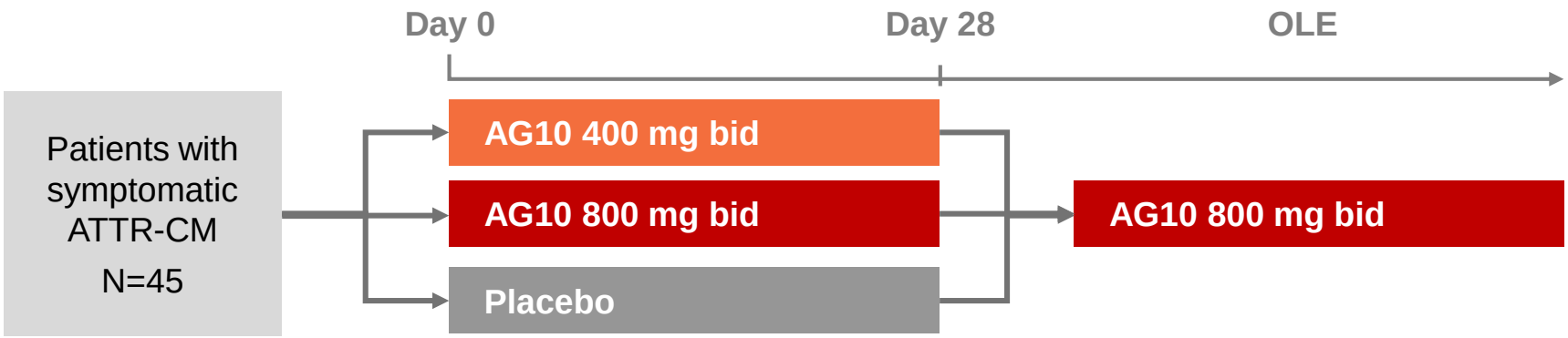
These and other potential risks and uncertainties that could cause actual results to differ from the results predicted are more fully detailed in the final prospectus for our initial public offering, filed with the U.S. Securities and Exchange Commission (the “SEC”) on June 21, 2018 and our Quarterly Report on Form 10-Q for the quarter ended June 30, 2018 filed with the SEC. It is not possible for our management to predict all risks, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statements we may make. Except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this presentation to conform these statements to actual results or to changes in our expectations.

Recent accomplishments and upcoming milestones

- Phase 2 study of AG10 in symptomatic ATTR cardiomyopathy (ATTR-CM) patients ongoing, top-line results to be announced by the end of 2018
- TTR stabilization mechanism validated by Pfizer's ATTR-ACT results reported at European Society of Cardiology meeting, published in *New England Journal of Medicine*
- AG10 molecular design manuscript published in the *Journal of Medicinal Chemistry*
- Complete data from Phase 1 study of AG10 in healthy volunteers to be presented in poster presentation at Heart Failure Society of America 22nd Annual Scientific Meeting (September 15-18)
- Phase 3 trial initiation planned for early 2019; discussions with clinical advisers and regulatory agencies ongoing

AG10 Phase 2 ATTR-CM study

Randomized, double-blind, placebo controlled, multi-center study of AG10 in ATTR-CM patients



- **Inclusion criteria:** At least one prior hospitalization for heart failure or clinical evidence of heart failure, confirmed ATTR by scan or biopsy (mutant or wild type)
- **Primary endpoint:** safety and tolerability
- **Secondary endpoints:** PK, TTR stabilization as measured by FPE and Western blot, TTR concentration
- **OLE:** safety and tolerability, cardiac biomarkers including NT-proBNP and troponin I, echocardiographic measurements including LV wall thickness, global longitudinal strain

- **Top-line results from randomized portion expected by the end of 2018**
- **Ongoing results from OLE may provide exploratory evidence of efficacy**

Key takeaways from Pfizer's ATTR-ACT results

Detail to follow

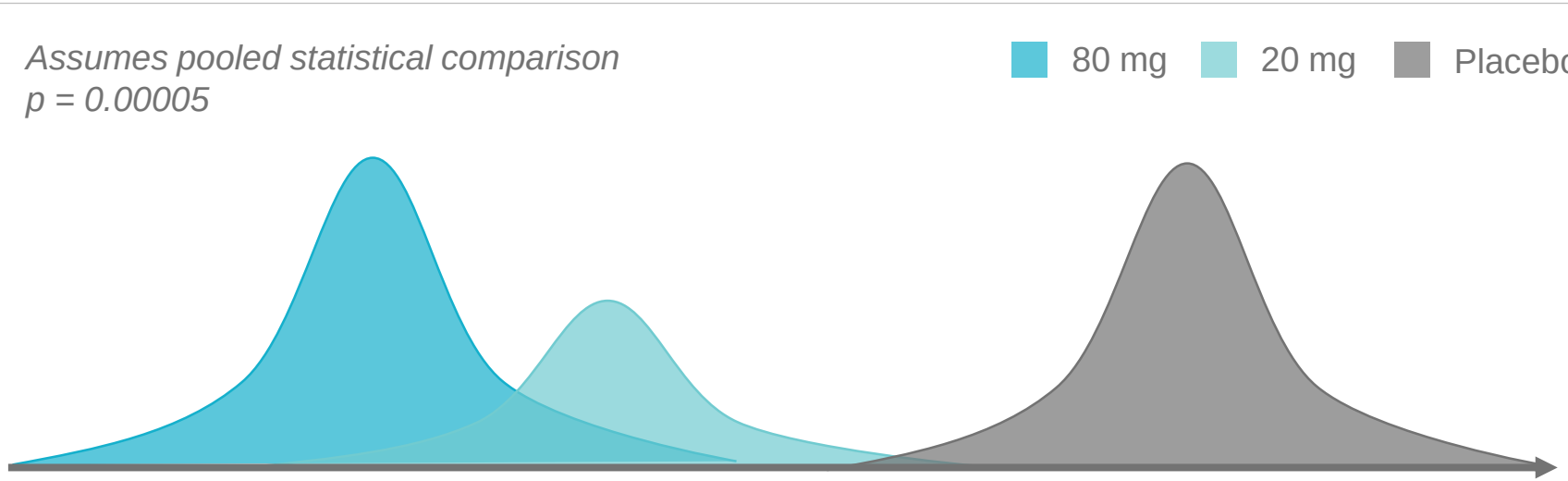
- **TTR stabilization mechanism validated**
  - Significant reduction in both all-cause mortality (30%) and CV hospitalizations (32%) as compared to placebo
  - Tafamidis well tolerated with no important differences between treatment group and placebo
- **Significant unmet need remains:**
  - 30 month mortality in treated patients was 29.5%, far above healthy age-matched population and above 25% rate modeled in placebo arm
  - Potentially limited benefit in Class III patients
- **Treatment effect lags:** treatment benefit on all-cause mortality delayed, benefit on functional endpoints and quality of life occurs earlier
- **No significant difference detected between 20 mg and 80 mg**

Source: Maurer, M. et al. NEJM 2018, DOI: 10.1056/NEJMoa1805689

RECALL: ATTR-ACT trial not powered to detect difference between 20 mg and 80 mg groups

Illustrative distributions of anticipated clinical response between ATTR-ACT groups[1]

Assumes pooled statistical comparison
p = 0.00005



16%

Power to detect significant dose response

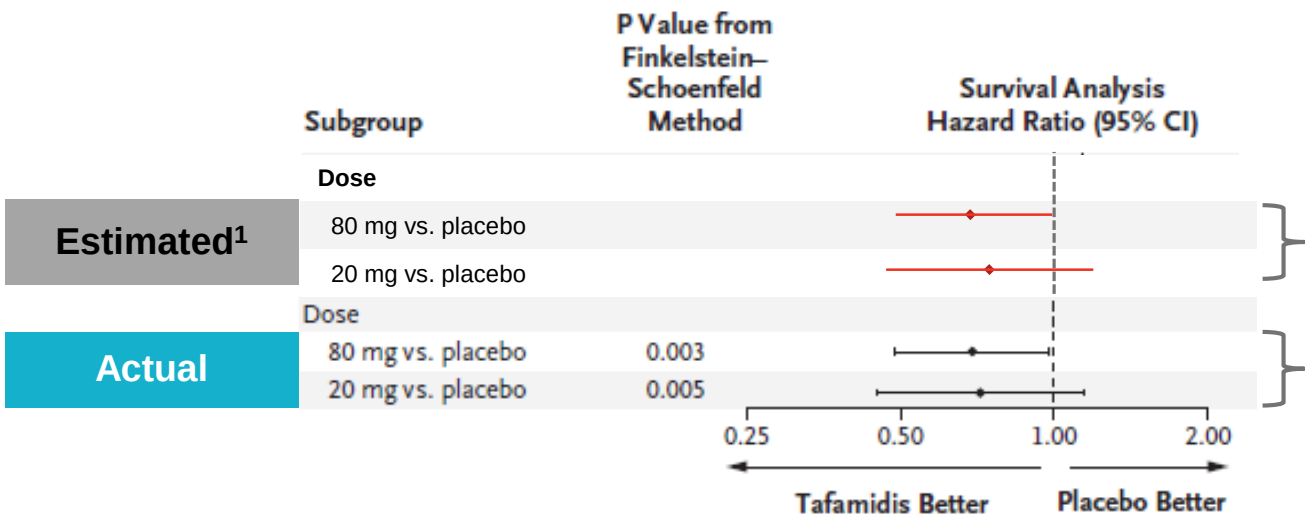
15-30%

Probability of 20 mg appearing numerically superior to 80 mg

[1] Assumes placebo mortality rate = 25%, placebo hospitalization rate = 50%, pooled treatment group mortality rate = 12.5%, pooled treatment group hospitalization rate = 35%, 10-33% larger response in 80 mg group than 20 mg group based on extent of TTR stabilization, population variability as observed in previous TTR stabilization trials

Minimal difference between estimated dose response and actual response

Comparison of Eidos' estimated result and actual result



30% greater response in 80 mg group than 20 mg group

~9% greater response in 80 mg group than 20 mg group

Shifting 1-2 deaths from the 80 mg group to the 20 mg group would be sufficient to equate actual result with estimated result

[1] Maintains pooled benefit as observed in ATTR-ACT, assumes 30% larger response in 80 mg group than 20 mg group based on 45% stabilization by 20 mg tafamidis and 60% stabilization by 80 mg tafamidis
Adapted from: Maurer, M. et al. NEJM 2018, DOI: 10.1056/NEJMoa1805689.

Lack of observable dose response could be readily explained by small trial biases

Potential explanations for limited dose response in ATTR-ACT trial

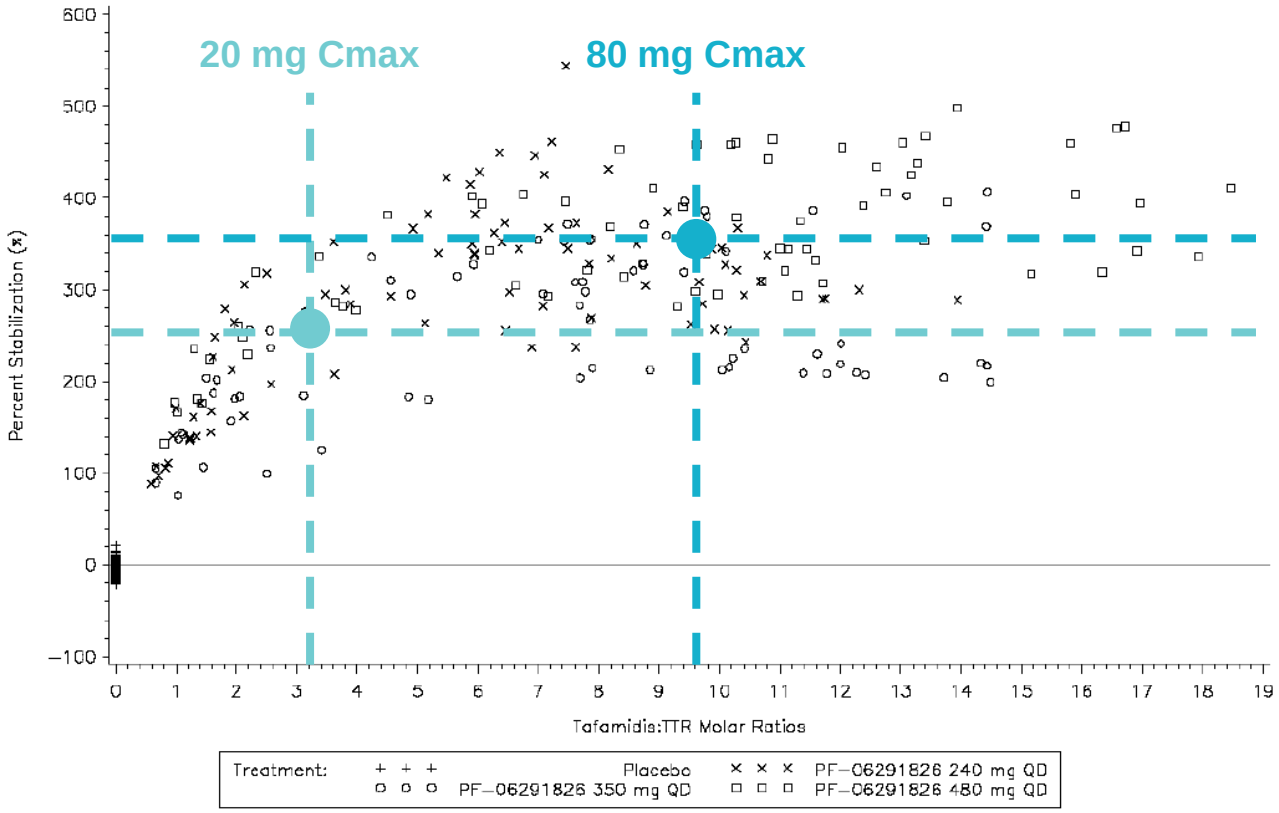
1. **Limited difference in stabilization between 20 mg and 80 mg doses[1]**
2. **Imbalance in trial discontinuations between 20 mg and 80 mg groups**
3. **Imbalance in Class 3 patients between 20 mg and 80 mg groups**

- **Power to detect a difference between 20 mg and 80 mg group further limited by half-sized 20 mg group (~90 patients).**
- **Dose response data only provided on primary endpoint. Difference on functional endpoints or quality of life may have been larger.**

1 Including lower stabilization in patients reduced to 40 mg dose from 80 mg dose

1 We believe Pfizer data suggests a limited difference in stabilization between 20 mg and 80 mg doses

Figure 1. Scatter Plot of TTR Percent Stabilization vs Tafamidis:TTR Molar Ratio by Treatment in Study B3461040



Remaining opportunity

~60% protein stabilization[1]

~45% protein stabilization[1]

1 "Percent Stabilization" as calculated by Pfizer represents level of tetrameric TTR stabilized relative to unstabilized TTR (~14% of initial protein (Bulawa 2012)). 200% stabilization therefore represents ~42% protein stabilized, 400% = ~70% protein stabilized, etc.. See Coelho, 2016 (DOI 10.1007/s40120-016-0040-x) for calculation method

Adapted from: Maurer, M. et al. NEJM 2018, DOI: 10.1056/NEJMoa1805689 (Protocol); Source: Ando, Y. et al. Journal of the Neurological Sciences 2016; 362:266-271

2 Discontinued patients represent a potential source of confounding

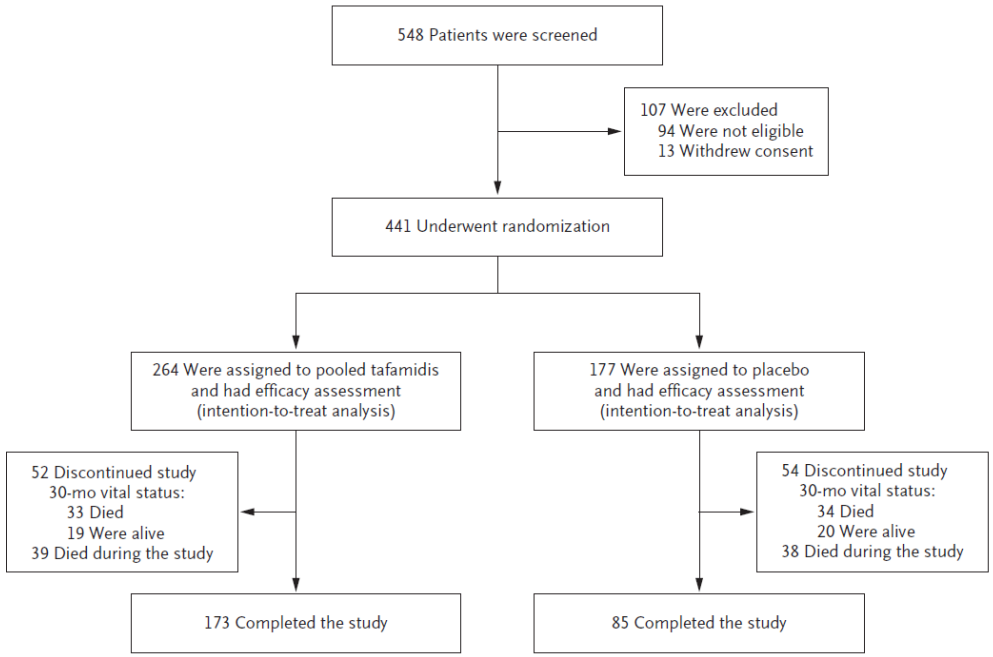
ATTR-ACT randomization, evaluation, and outcomes

Placebo

- 63.0% mortality in discontinued patients
- 30.8% mortality in completing patients

Pooled tafamidis

- 63.5% mortality in discontinued patients
- 18.4% mortality in completing patients

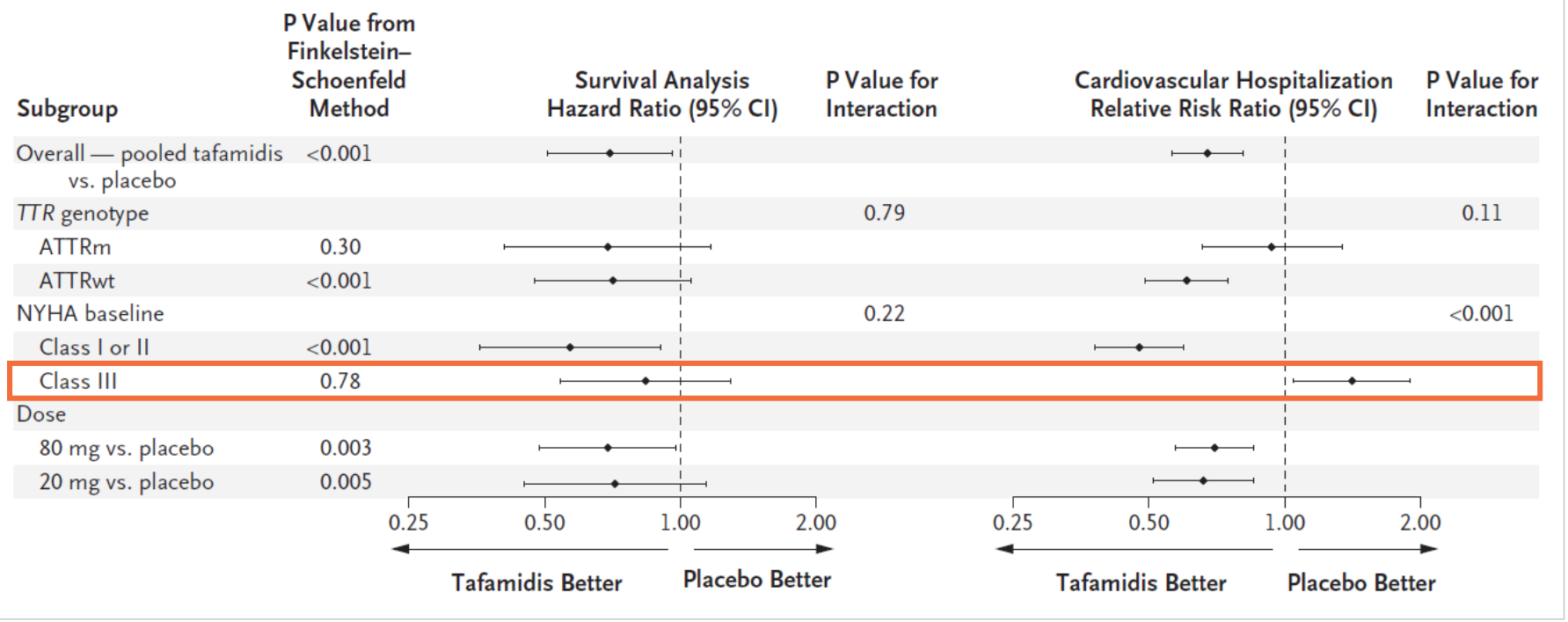


- Large difference in mortality between patients who completed and discontinued study
- Disproportionate discontinuation or death rate between 20 mg and 80 mg arms could meaningfully shift outcome

Adapted from: Maurer, M. et al. NEJM 2018, DOI: 10.1056/NEJMoa1805689

3 Class III patients represent a potential source of confounding

Overall and subgroup results from ATTR-ACT trial



Given limited treatment effect in Class III patients, a disproportionate allocation to 80 mg vs. 20 mg groups could mask differential effect between doses

Adapted from: Maurer, M. et al. NEJM 2018, DOI: 10.1056/NEJMoa1805689

We believe that an even more effective TTR stabilizer may further improve clinical outcome

Evidence supporting relationship between stabilization and clinical benefit

Human genetics

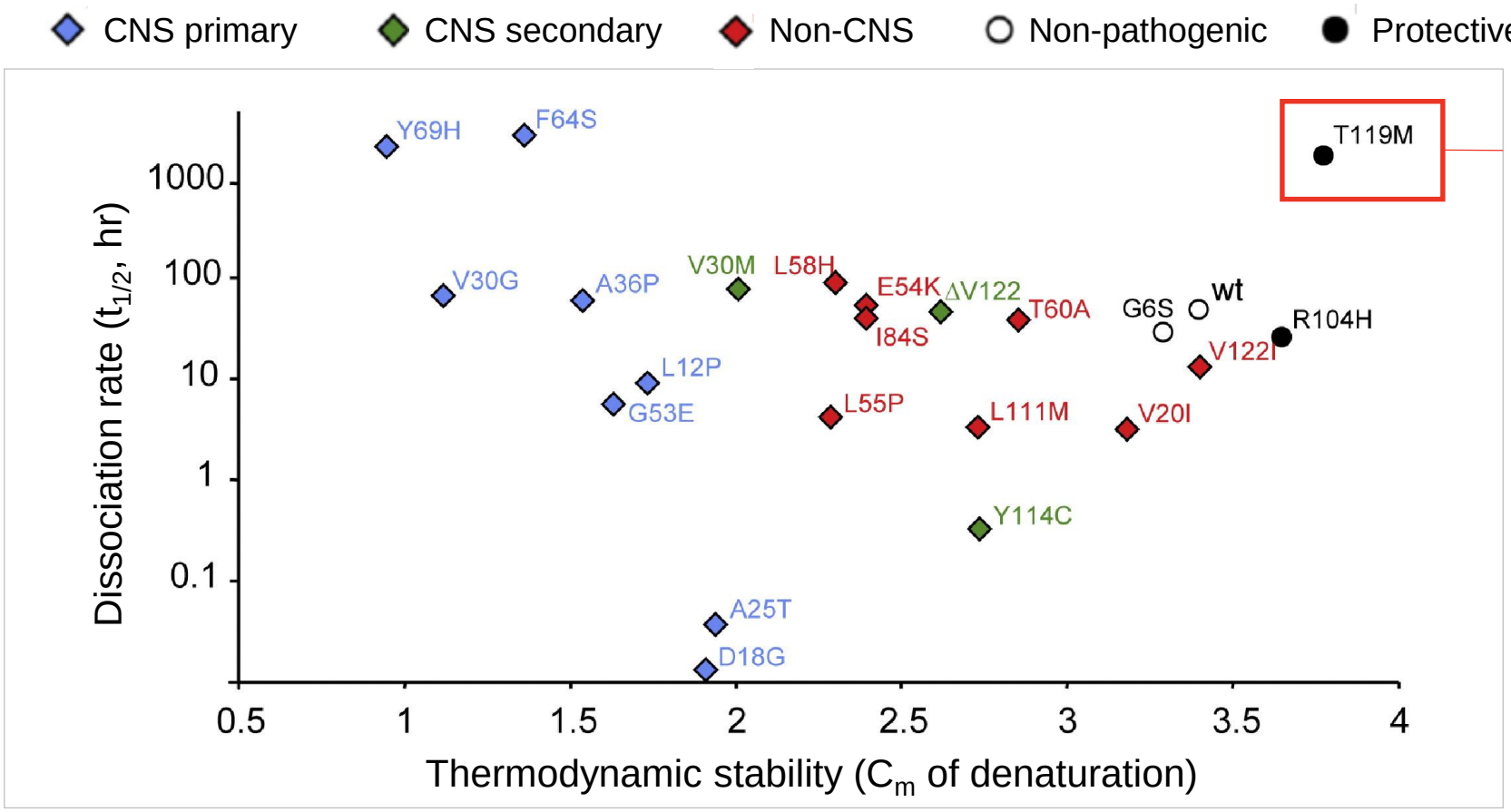
- ATTR-causing mutations destabilize TTR tetramer; greater destabilization leads to increased disease severity
- ATTR-protective mutations stabilize TTR tetramer and can increase median life expectancy as compared to non-carriers

Previous clinical trials

- Four trials in ATTR polyneuropathy (ATTR-PN) demonstrate that increasing levels of stabilization/knockdown lead to improved benefit
- Within knockdown studies, increased TTR knockdown leads to improved benefit

Genetic and clinical evidence suggests that enhanced TTR stabilization can maximize long-term patient outcomes

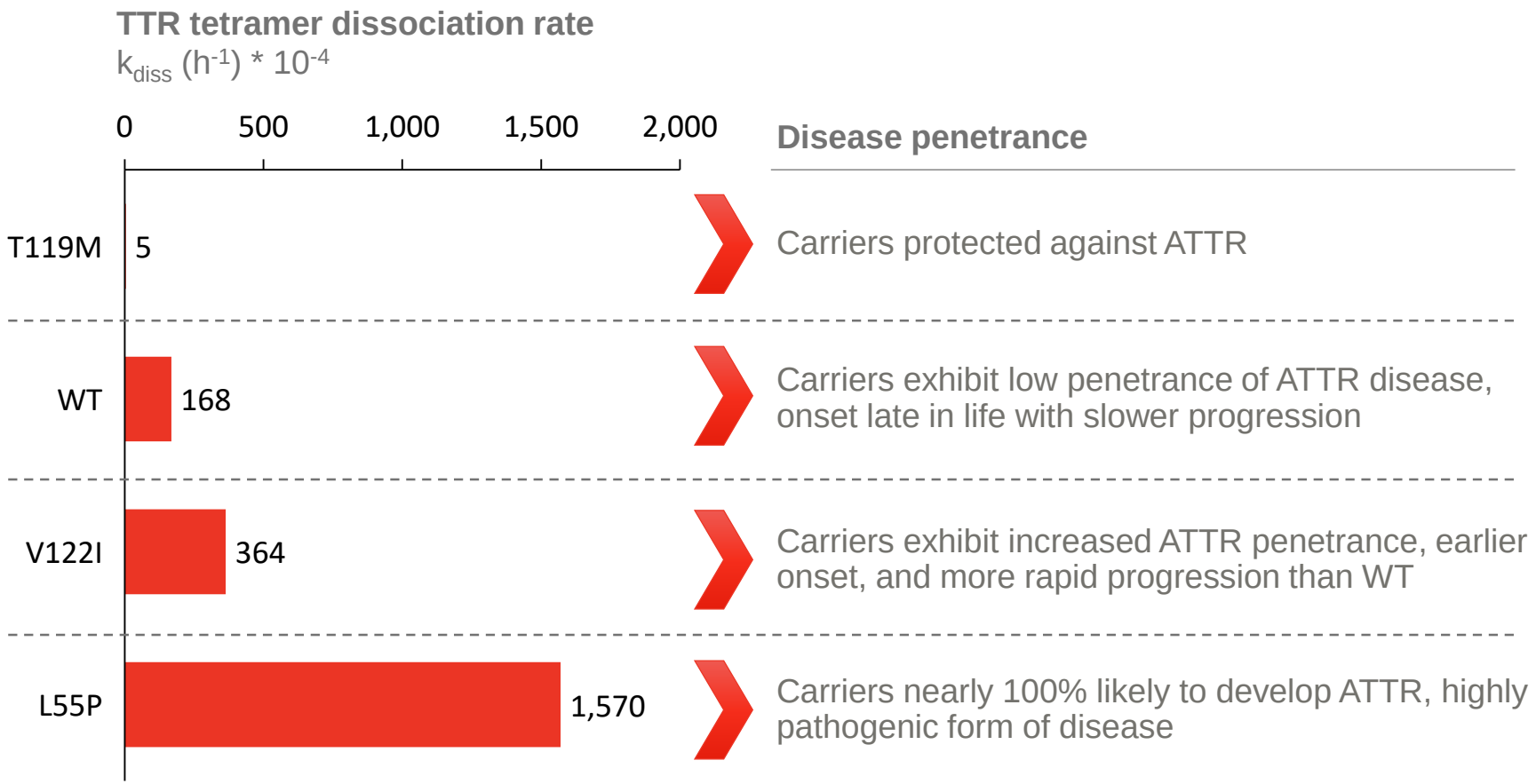
Human genetics suggests TTR stability is associated with disease severity



"These studies demonstrate that kinetic and thermodynamic data, considered together, nicely rationalize why certain mutations lead to severe pathology, whereas others protect against disease or lead to mild pathology."

Source: Sekijima, Y. et al. Cell, Vol. 121, 73–85, April 8, 2005; Hammarstrom, P. et al. PNAS 2002, 99:16427-16432

Human genetics suggests TTR stability is associated with disease severity



Source: Hammarstrom, P. et al. PNAS 2002, 99:16427-16432

ATTR-PN trials demonstrate improved clinical benefit with increased reduction in TTR monomers

Remaining Stabilized Knockdown

% monomer reduction at peak[1]		Clinical outcome in ATTR-PN Phase 3 trial
Tafamidis (20 mg qd)	~45%	▪ **Non-significant improvement on primary endpoint** ▪ **Treatment group progression ~ +0.2 NIS-LL points/month**
Diflunisal (250 mg bid)	~75%	▪ **Significant improvement on primary endpoint** ▪ **Treatment group progression ~ +0.4 NIS+7 points/month**
Inotersen (1wk SC)	~75%	▪ **Significant improvement on primary endpoint** ▪ **Treatment group progression ~ +0.3 mNIS+7 points/month**
Patisiran (3wk IV)	~85%	▪ **Significant improvement on primary endpoint** ▪ **Treatment group progression ~ -0.3 mNIS+7 points/month**

[1] Stabilization values averaged between Western blot and FPE assays (except Diflunisal, which includes only Western blot); tafamidis either purchased commercially or synthesized for research use

Source: Coelho, T. et al. Neurology 2012; 79:785–792; Berk, J.L. et al JAMA. 2013; 310:2658-2667; Ando, Y. et al. J Neurological Sci 2016; 362:266-271; Vyndaqel® SmPC; Diflunisal USPI; Alnylam and Ionis corporate presentations

Our Phase 3 trial design leverages expertise from leading TTR physicians and cardiovascular drug developers

Phase 2 investigators

John Berk, MD *Boston University*	**Dan Jacoby, MD** *Yale University*	**Jignesh Patel, MD, PhD** *Cedars-Sinai Medical Center*
Rodney Falk, MD *Brigham and Women's Hospital*	**Dan Judge, MD** *Medical University of South Carolina*	**Van Selby, MD** *University of California San Francisco*
Martha Grogan, MD *Mayo Clinic*	**Mat Maurer, MD** *Columbia University*	**Sanjiv Shah, MD** *Northwestern University*
Mazen Hanna, MD *Cleveland Clinic*	**Jose Nativi-Nicolau, MD** *University of Utah*	**Ronald Witteles, MD** *Stanford University*
Stephen Heitner, MD *Oregon Health & Science University*		

Eidos senior scientific leadership and advisors

Neil Kumar, PhD, CEO	MyoKardia	THIRD ROCK VENTURES	McKinsey&Company
Jonathan Fox, MD, PhD, President & CMO	MyoKardia	AstraZeneca	MERCK Be well
Uma Sinha, PhD, CSO	globalblood THERAPEUTICS	PORTOLA PHARMACEUTICALS	COR
Charles Homcy, MD, Senior Advisor	THIRD ROCK VENTURES	PORTOLA PHARMACEUTICALS	COR

Final Phase 3 design details to be provided following consultation with additional cardiovascular trial design experts and regulatory agencies

Financial summary: Nasdaq EIDX

Recent financings

$64M Series B
(Q1 2018)

$122M IPO[1]
(Q2 2018)

Cash balance

$176.7M
(June 30, 2018)

No debt

Expenses

Q2 YTD 2018:
$13.4M R&D
$4.0M G&A

2017:
$9.3M R&D
$2.7M G&A

1 Before deducting underwriting discounts and other offering expenses

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Thank You