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): **September 18, 2014**

AMICUS THERAPEUTICS, INC.
(Exact Name of Registrant as Specified in Its Charter)

Delaware	
(State or Other Jurisdiction of Incorporation)	
001-33497	**71-0869350**
(Commission File Number)	(IRS Employer Identification No.)
1 Cedar Brook Drive, Cranbury, NJ	**08512**
(Address of Principal Executive Offices)	(Zip Code)

Registrant's telephone number, including area code: **(609) 662-2000**

(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:

o Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

o Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

o Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

o Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 8.01 — Other Events.

The senior management of Amicus Therapeutics, Inc. (the "Company") is using the presentation attached as Exhibit 99.1 to this Current Report in its current meetings with investors and analysts.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits: The Exhibit Index annexed hereto is incorporated herein by reference.

Exhibit Number	Description
99.1	Presentation Materials

SIGNATURES

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

Amicus Therapeutics, Inc.

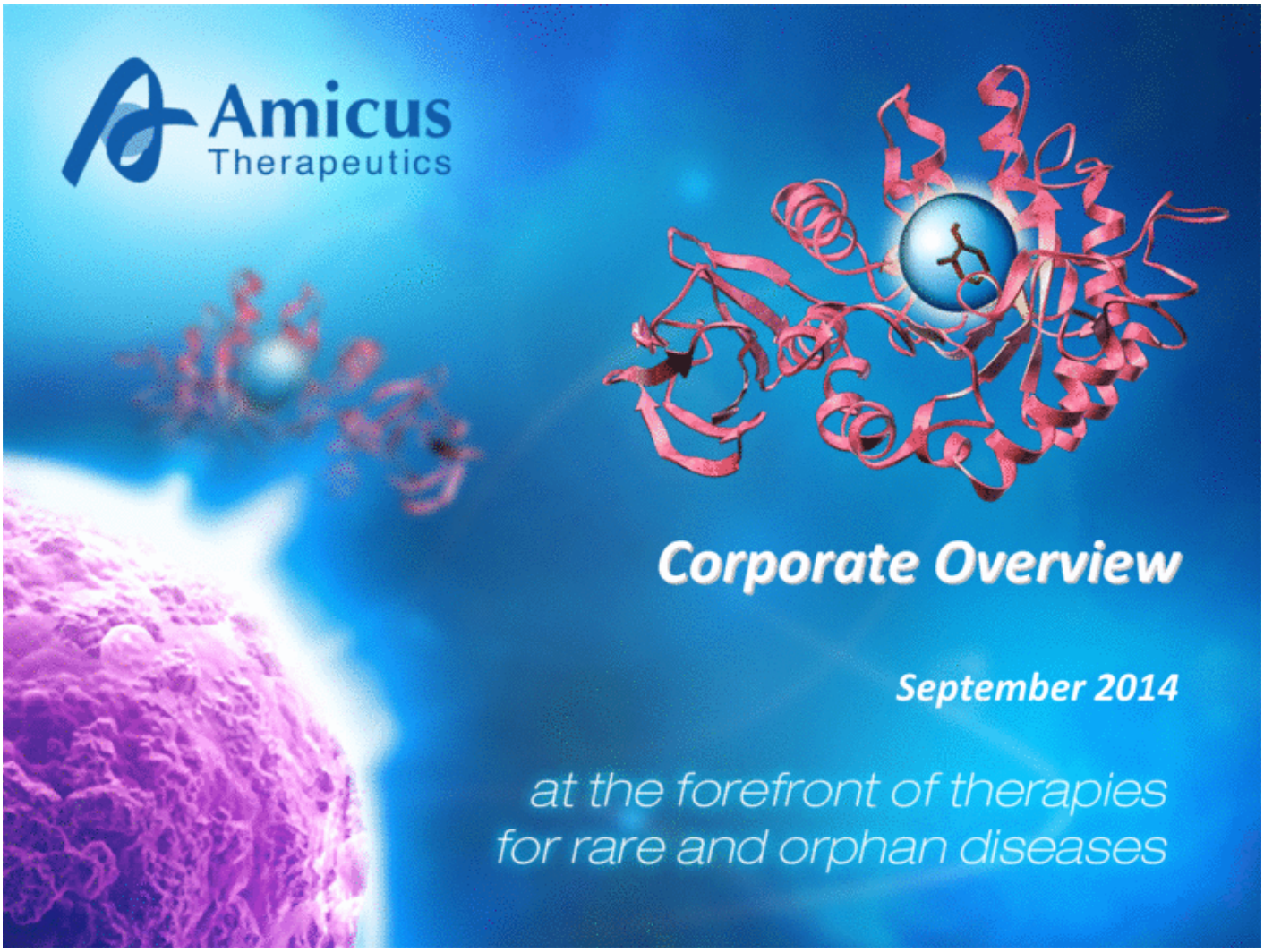
Date: September 18, 2014

By: /s/ William D. Baird III
William D. Baird III
Chief Financial Officer

EXHIBIT INDEX

Exhibit Number	Description
99.1	Presentation Materials

Exhibit 99.1

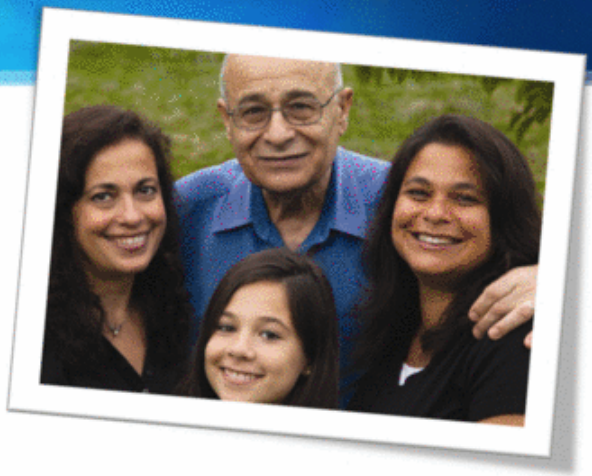

Amicus
Therapeutics
Corporate Overview
September 2014
at the forefront of therapies
for rare and orphan diseases

Safe Harbor

This presentation contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995 relating to business, operations and financial conditions of Amicus including but not limited to preclinical and clinical development of Amicus' candidate drug products, cash runway, ongoing collaborations and the timing and reporting of results from clinical trials evaluating Amicus' candidate drug products. Words such as, but not limited to, "look forward to," "believe," "expect," "anticipate," "estimate," "intend," "plan," "would," "should" and "could," and similar expressions or words, identify forward-looking statements. Although Amicus believes the expectations reflected in such forward-looking statements are based upon reasonable assumptions, there can be no assurance that its expectations will be realized. Actual results could differ materially from those projected in Amicus' forward-looking statements due to numerous known and unknown risks and uncertainties, including the "Risk Factors" described in our Annual Report on Form 10-K for the year ended December 31, 2013. All forward-looking statements are qualified in their entirety by this cautionary statement, and Amicus undertakes no obligation to revise or update this presentation to reflect events or circumstances after the date hereof.

Amicus Therapeutics

Company Mission



Amicus Therapeutics is a biopharmaceutical company at the forefront of developing next-generation medicines to treat a range of rare and orphan diseases, with a focus on improved therapies for Lysosomal Storage Disorders

Amicus Therapeutics

Investment Highlights

Strength of Clinical Programs and Breadth of Technology Platforms With Potential to Create Significant Value for Shareholders and Patients Living with LSDs

Migalastat Monotherapy

- First oral therapy for Fabry patients with amenable mutations
  - Two positive Phase 3 studies
  - WW rights
  - EMA pre-submission meeting 4Q14

3 in 3 Strategy

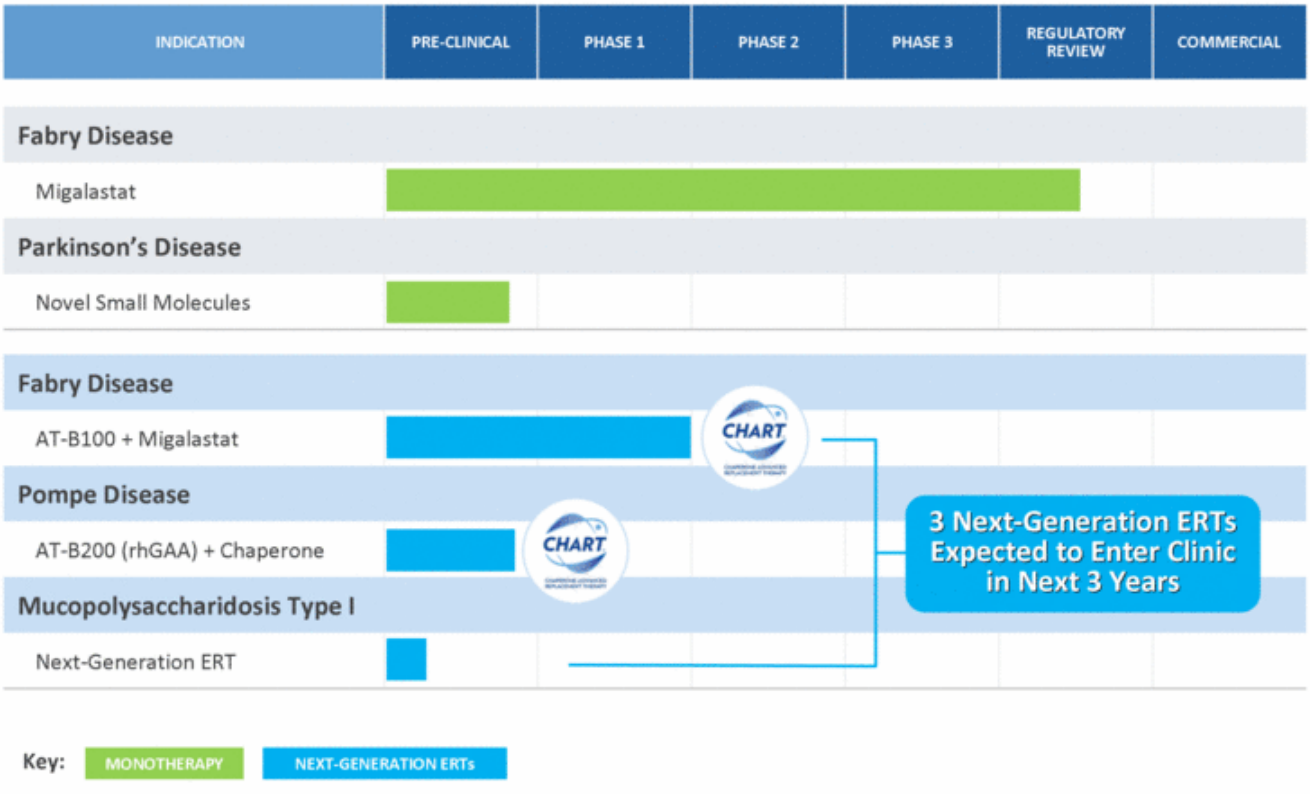
- Advancing three next-gen ERTs into clinic in next three years
  - Fabry
  - Pompe
  - MPS I

Proprietary Technologies

- Addressing common limitations of ERTs
  - CHART™
  - Optimized carbohydrates
  - vIGF2 tagging

Amicus Therapeutics

Advanced Product Pipeline



Amicus Therapeutics

Chaperone Technology
Proposed Mechanism of Action

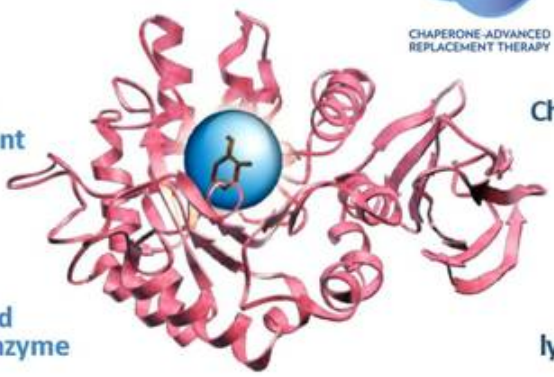
Pharmacological chaperones are designed to stabilize a patient's own enzyme *or* an infused ERT

Monotherapy

Chaperone binds to and stabilizes endogenous mutant enzyme in ER

Increases trafficking and lysosomal levels of active enzyme

CHART
CHAPERONE-ADVANCED REPLACEMENT THERAPY



Chaperone binds to and stabilizes exogenous enzyme (ERT) in circulation

Increases tissue uptake and lysosomal levels of active enzyme

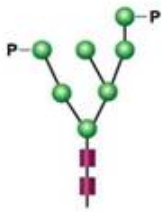
LOWERED SUBSTRATE

Amicus Therapeutics

ERT Targeting Technology
Proposed Mechanism of Action

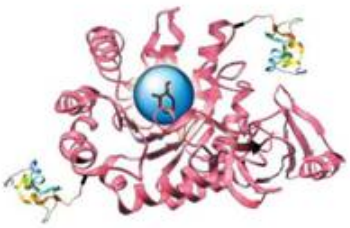
Amicus' has multiple targeting technologies to address the common challenges of ERT and increase the amount of ERT taken up into cells

Optimized Carbohydrates



Optimizes M6P-containing glycans for increased phosphorylation

vIGF-2 Tagging



Conjugation of vIGF2 peptide tag enhances drug targeting and uptake

Increases tissue uptake and lysosomal levels of active enzyme

LOWERED SUBSTRATE

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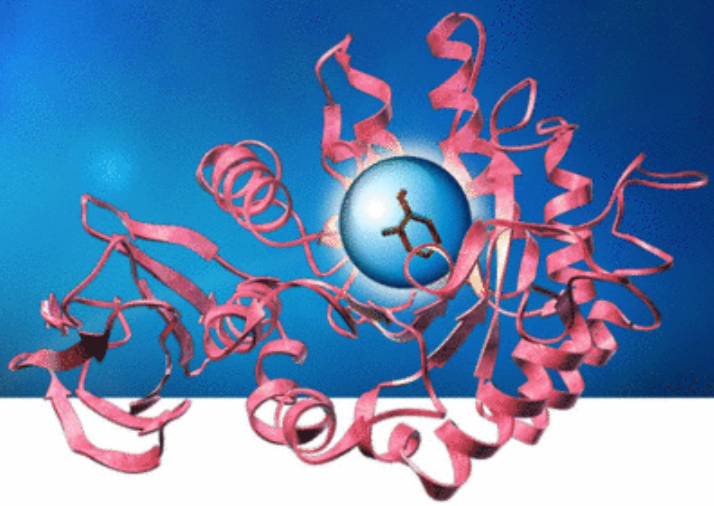
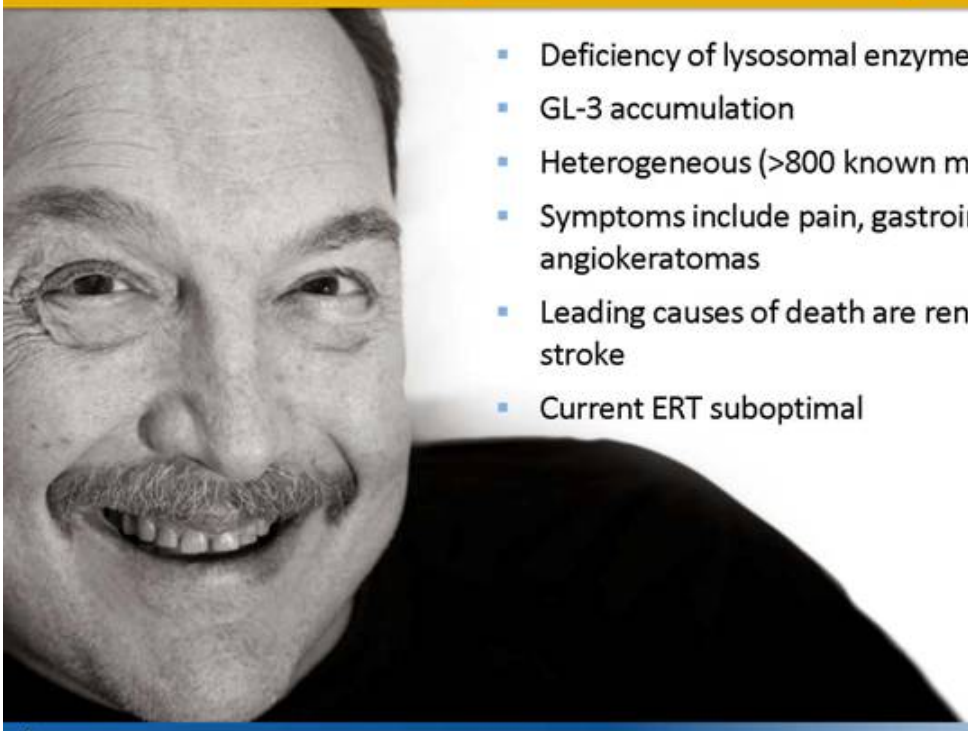
Amicus Therapeutics



Migalastat Monotherapy for Fabry Disease

Fabry Disease Overview

Fatal Lysosomal Storage Disease with Significant Unmet Needs Despite Available Treatment Options



- Deficiency of lysosomal enzyme α-Galactosidase A (α-Gal A)
- GL-3 accumulation
- Heterogeneous (>800 known mutations)
- Symptoms include pain, gastrointestinal problems, angiokeratomas
- Leading causes of death are renal failure, cardiac failure, stroke
- Current ERT suboptimal

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Fabry Unmet Needs

Currently Approved ERTs Do Not Fully Address Fabry Disease

> **Over 40 years of working with patients with Fabry disease…I believe there remains an unmet medical need among these patients.**

Robert Desnick, M.D.
Dean for Genetics and Genomic Medicine, Professor and Chairman Emeritus, Genetics and Genomic Sciences at Icahn School of Medicine at Mount Sinai

> **Given the choice, I would use migalastat over ERT for the treatment of Fabry patients with amenable mutations.**

Raphael Schiffmann, M.D., M.H.Sc.
Investigator, Institute of Metabolic Disease, Baylor Research Institute

Current Treatment Limitations

- Long term ERT does not prevent disease progression[1]
- Burden of intravenous infusions
- Additional costs for hospital administration
- 50%-55% of patients in Fabrazyme clinical studies experienced at least 1 infusion-related reaction[2]
- IgG positive patients might have worse clinical outcome than IgG negative patients[3]

[1]Rombach, et al. 2013 [2]Fabrazyme PI [3]Deegan, et al. 2012

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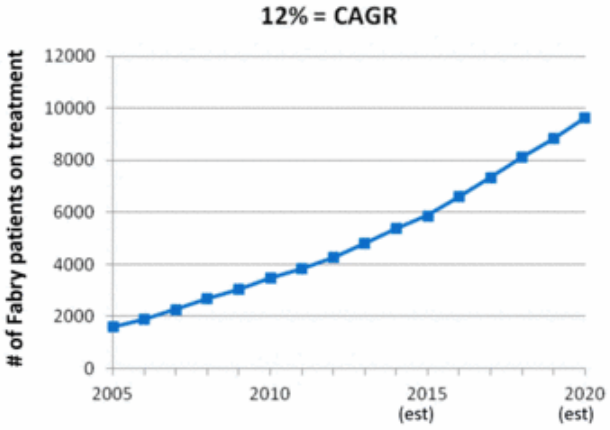
Fabry Commercial Opportunity

Market Size, Growth Rate and Limitations of Current Therapies Make Fabry a Compelling Market Opportunity

Global Fabry Market ($993M in 2013)



Sources: Analyst Reports, Company 10Ks, Market Research

- $993M in FY13 global ERT sales (Fabrazyme and Replagal)
- 5-10K diagnosed WW (51% female/49% male[1])
- <50% of diagnosed patients are currently treated with ERT
- 30-50% of Fabry patients with amenable mutations

[1]Fabry Registry 2011;

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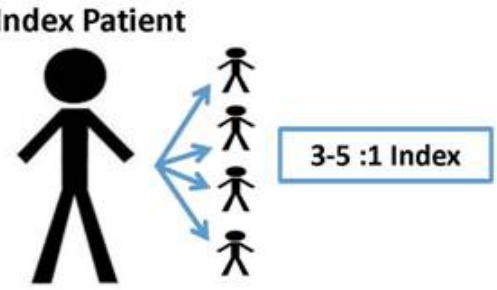
Significant Underdiagnosis: *Long-Term Growth Potential*

Newborn screening supports significant underdiagnosis of Fabry disease with the majority of patients identified as having amenable mutations

Newborn Screening Study	# Newborns Screened	# Confirmed Fabry Mutations	% Amenable
Burton, 2012, US	8,012	7 [1: ~1100]	TBD
Mechtler, 2011, Austria	34,736	9 [1: ~3,800]	100%
Hwu, 2009, Taiwan	171,977	75 [1: ~2300]	75%
Spada, 2006, Italy	37,104	12 [1: ~3100]	86%
Historic published incidence		1:40,000 to 1:60,000	



Burton, **LDN WORLD Symposium,** 2012 Feb. Hwu *et al.*, **Hum Mutation,** 2009 Jun
Mechtler *et al.*, **The Lancet,** 2011 Dec. Spada *et al.*, **Am J Human Genet.,** 2006 Jul

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Global Registration Studies

Assembling Robust Dataset to Maximize Chances for Global Approvals of Migalastat Monotherapy for Fabry Patients with Amenable Mutations

Study 011
(FACETS)

- 67 patients naïve to ERT
- Placebo-controlled (6 months)
- Primary endpoint: reduction in substrate (kidney GL-3) at 6 and 12 months
- Secondary endpoint: kidney function (eGFR and mGFR) and 12- and 24-months

Study 012
(ATTRACT)

- 60 ERT experienced patients
- Switch study – 36 patients switched to migalastat, 24 remained on ERT
- Primary endpoint: comparability to ERT based on eGFR and mGFR over 18 months

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Phase 3 FACETS Study (Study 011)

Migalastat
150 mg QOD

67 patients
1:1 Randomization
Stratified by gender

Placebo QOD

Open-Label Migalastat 150 mg QOD

Stage 1*
Month 0-6 Double-Blind Treatment Period

Stage 2*
Month 7-12 Open-Label Follow-Up Period

Optional Extension**
Month 13-24

6-month primary endpoint: kidney interstitial capillary GL-3

- 12-month biopsy and 24-month clinical data (NEW DATA)
- Pre-Specified GLP HEK Amenable Subgroup Analysis

*GL-3 Substrate Measured by Histology in Kidney Biopsies **Clinical Outcomes Assessed, Including eGFR and Proteinuria

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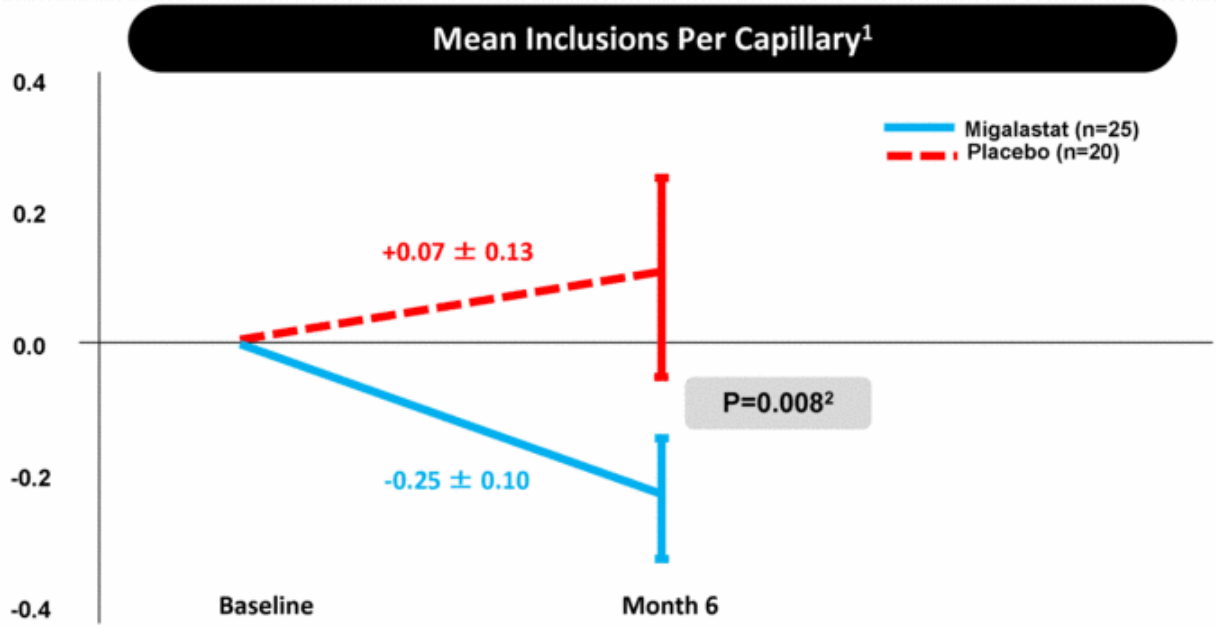
6-Month Post-Hoc Analysis
(Reported February 2014)

Statistically Significant Mean Change in Kidney Interstitial Capillary GL-3 Compared to Placebo (GLP HEK Amenable)*



*All patients with evaluable paired biopsies and amenable GLA mutations in GLP-validated HEK assay – post hoc at month 6 and pre-specified at month 12 [1]Data points are baseline corrected; represent mean ± standard error (SEM) change from baseline in the mean number of GL-3 inclusions per capillary after 6 months of treatment with migalastat or placebo. [2]Analysis of covariance (ANCOVA) model with covariate adjustment for baseline value and factors for treatment group and treatment by baseline interaction. P-value corresponding to least-square mean difference between migalastat and placebo is displayed.

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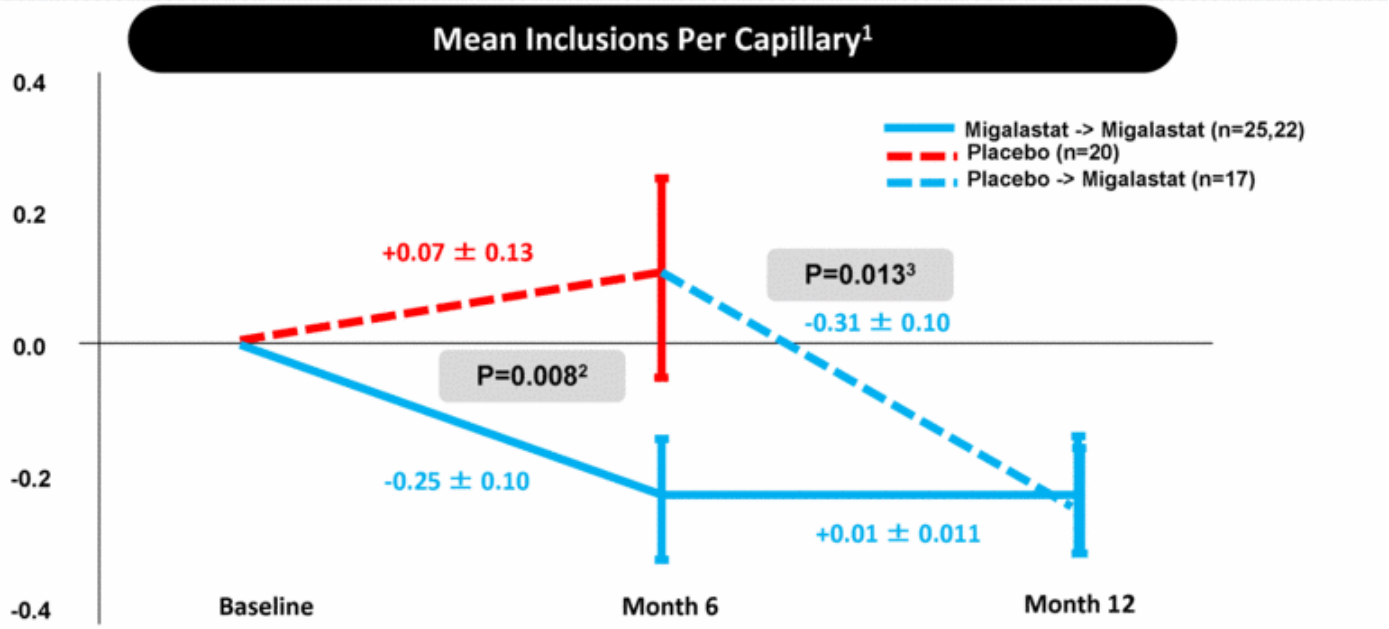
12-Month Pre-Specified Primary Analysis

Statistically Significant Mean Change in Kidney Interstitial Capillary GL-3 in Patients Switching from Placebo to Migalastat HCl (GLP HEK Amenable)*



*All patients with evaluable paired biopsies and amenable GLA mutations in GLP-validated HEK assay – post hoc at month 6 and pre-specified at month 12 [1]Data points are baseline corrected; represent mean ± standard error (SEM) change from baseline in the mean number of GL-3 inclusions per capillary after 6 months of treatment with migalastat or placebo. [2]Analysis of covariance (ANCOVA) model with covariate adjustment for baseline value and factors for treatment group and treatment by baseline interaction. P-value corresponding to least-square mean difference between migalastat and placebo is displayed. [3]MMRM Pbo change M6 to M12.

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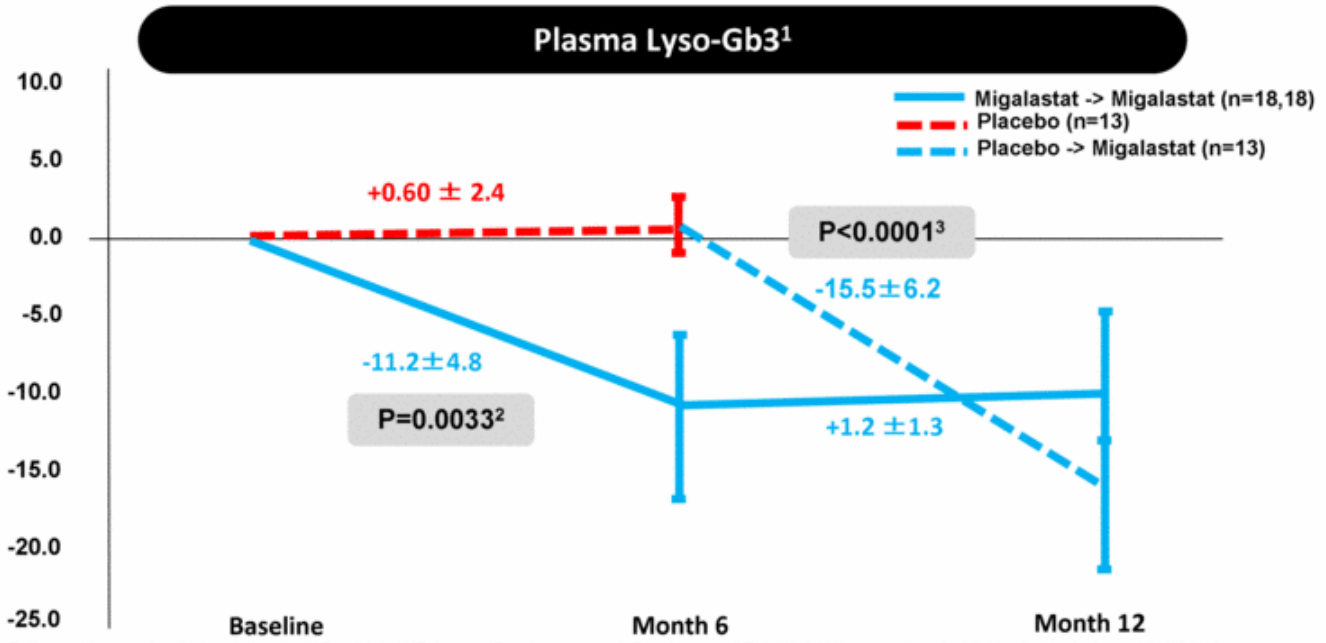
Disease Substrate in Plasma (Plasma Lyso-Gb3)

Statistically Significant Reduction in Plasma Lyso-Gb3 at Month 6 and Month 12 Following Treatment with Migalastat (GLP HEK Amenable)*



*Patients with amenable GLA mutations in GLP-validated HEK assay [1]Baseline corrected. Error bars are SEM [2]ANCOVA comparing migalstat to placebo in Stage 1 [3]ANCOVA comparing change from month 6 to month 12 in subjects switching from placebo to migalastat

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Kidney Function: Annualized Glomerular Filtration Rate (GFR)

GFR Remained Stable Over 18-24 Months (GLP HEK Amenable)*

Annualized GFR (ml/min/1.73m²/yr) at Month 18 or 24[1]

GFR Measure	N*	Mean	(SEM)
eGFR (CKD-EPI)	41	-0.30	(0.66)
eGFR (MDRD)	41	0.79	(1.03)
mGFR (iohexol)	37	-1.51	(1.33)

*Patients with amenable GLA mutations in GLP-validated HEK assay
[1]24 Months of Data in Subjects Treated with Migalastat from Baseline, 18 Months of Data in Subjects Switched from Placebo to Migalastat After 6 Months

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Safety Summary – Study 011

Migalastat Generally Safe and Well Tolerated

Most Common Treatment Emergent Adverse Events (≥ 10% of Subjects)

Adverse event	Baseline to Month 6		Months 7-12		Open-Label Extension (Months 13-24)	
	Placebo* (n=33)	Migalastat (n=34)	Placebo-Migalastat* (n=30)	Migalastat (n=33)	Placebo-Migalastat* (n=28)	Migalastat (n=29)
Any Event	91%	91%	80%	79%	86%	83%
Headache	21%	35%			11%	10%
Fatigue	12%	12%				
Nausea	9%	12%				
Nasopharyngitis	6%	15%				
Paresthesia	12%	9%				
Procedural Pain			10%	12%		
Proteinuria					18%	14%
Bronchitis					11%	10%

*Subjects Received Placebo from Baseline to Month 6, Switched to Migalastat After Month 6

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Phase 3 ATTRACT Study (Study 012)

Patients Randomized to Switch to Migalastat or Remain on ERT, with Option for All Patients to Receive Migalastat in Open-Label Extension

Migalastat
150 mg QOD

60 patients
Open-label
1.5:1 Randomization (Switch to Migalastat or Remain on ERT)
Stratified by Gender, Proteinuria

ERT QOW (Labeled Dose)

18-Month Primary Treatment Period

Open-Label Migalastat 150 mg QOD

Optional 12-Month Extension

- Descriptive assessment of comparability for migalastat and ERT in eGFR and mGFR
  - Overlap of 95% CI >50%
  - Means within 2.2 mL/min/1.73 m2/yr

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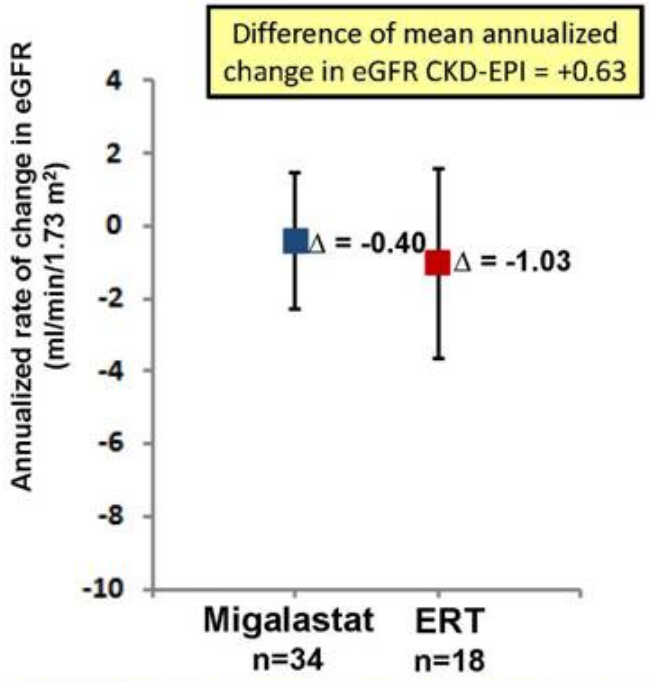
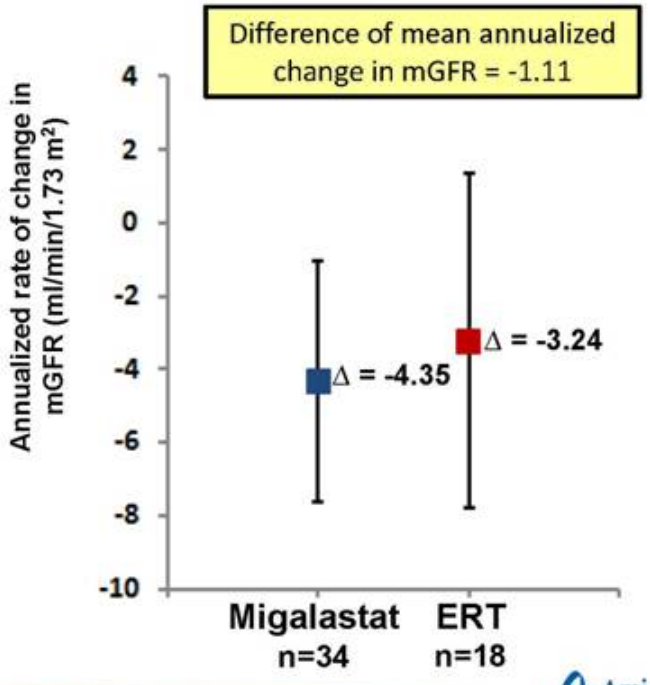
Study 012 Kidney Function:
Annualized GFR at Month 18[1]

100% Overlap of Migalastat Confidence Intervals with ERT Confidence Intervals





[1] ANCOVA model [mITT]. Data represent LS means and 95% confidence intervals

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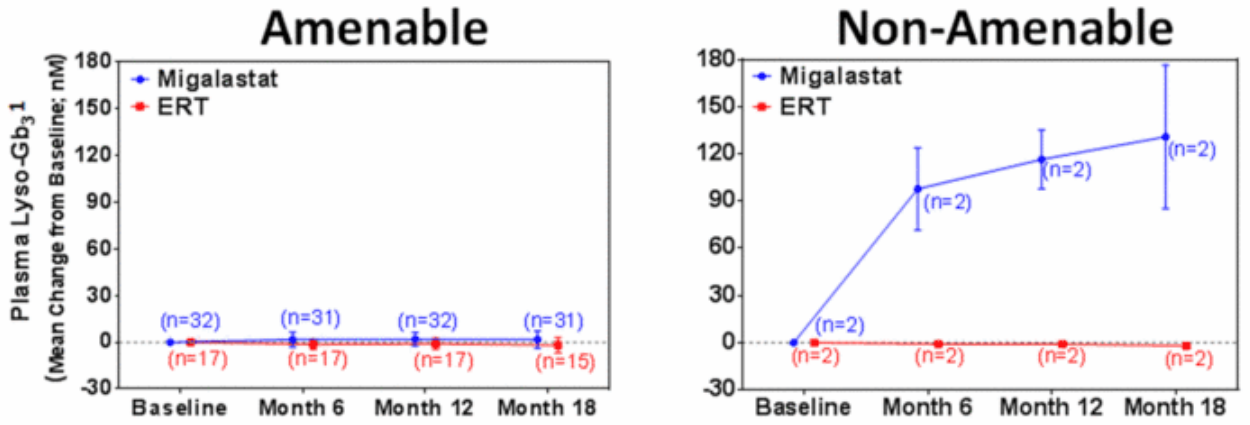
Disease Substrate in Plasma (Plasma Lyso-Gb3)

No Change in Plasma Lyso-Gb3 over 18 months Following Switch From ERT to Migalastat in Subjects with Amenable Mutations



- In subjects with amenable mutations the plasma lyso-Gb3 levels were comparable for migalastat and ERT
- In two male subjects with non-amenable mutations plasma lyso-Gb3 increased following switch from ERT as compared to two (1M, 1F) who remained on ERT

Data points represent the mean, Error bars are SD; Based on subjects with available samples for this analysis

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Safety Summary – Study 012
Common AEs (≥10%)

Migalastat Was Generally Safe and Well-Tolerated

	Migalastat	ERT
N subjects	36	21
n subjects with TEAEs (%)	34 (94%)	20 (95%)
Nasopharyngitis	33%	33%
Headache	25%	24%
Dizziness	17%	10%
Influenza	14%	19%
Abdominal Pain	14%	10%
Diarrhea	14%	10%
Nausea	14%	10%
Back Pain	11%	14%
Upper Respiratory Tract Infection	11%	5%
Urinary Tract Infection	11%	5%
Cough	8%	24%
Vomiting	8%	14%
Sinusitis	8%	14%
Arthralgia	8%	10%
Bronchitis	6%	14%
Edema Peripheral	6%	10%
Vertigo	3%	10%
Dry Mouth	3%	10%
Gastritis	3%	10%
Pain In Extremity	3%	10%
Dyspnea	3%	10%
Procedural Pain	-	10%

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Migalastat Monotherapy Experience for Fabry

97 Patients Today Take Migalastat HCl as Only Therapy for Fabry Disease[1]

Total patients who have ever taken migalastat:
143

Patients taking migalastat today as only therapy:
97

Total patient years of therapy:
377
(no drug-related SAEs)

Average retention rate into next study:
96%*

Maximum years on therapy:
8.6

Information as of August 2014. All patients are receiving investigational drug, migalastat HCl, as part of ongoing clinical trials
*Retention defined as # of patients who completed a study and chose to enter extension, e.g., Study 011 12-mo into 24-mo extension

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Global Regulatory Strategy

Pursuing Fastest Path to Approval for Migalastat

- Totality of clinical data
- 8+ years of data in extension studies
- Complete data set from Phase 3 studies (011 and 012)

- Centralized Procedure underway
- EMA Pre-Submission Meeting planned for 4Q 14
- Clear regulatory pathway
- Non-inferiority to ERT (Study 012)

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Key Milestones

Timing	Milestone	
2Q14	12-month Study 011 data (kidney biopsies)	✓
2Q14	24-month Study 011 data (clinical outcomes)	✓
3Q14	18-month Study 012 data (kidney function)	✓
4Q14	**Additional 011, 012, and Phase 2 extension data**	
4Q14	**EMA regulatory interaction**	
1Q15	**FDA regulatory interaction**	

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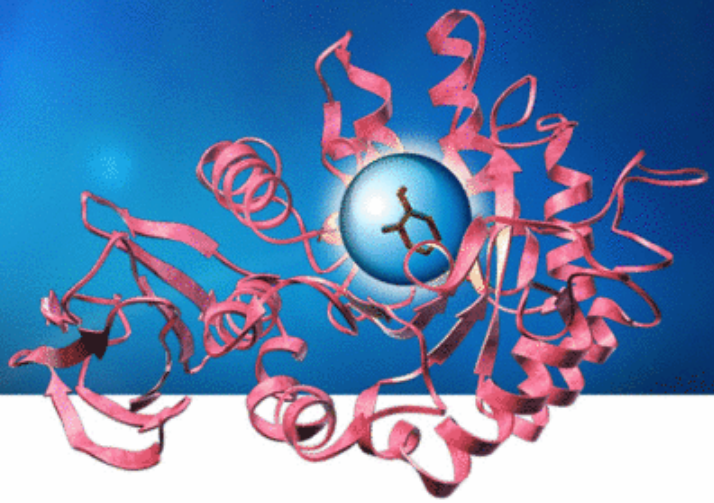
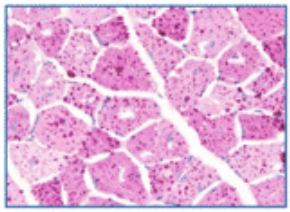


Next-Generation ERT for Pompe Disease

Pompe Disease Overview

Severe, progressive, fatal neuromuscular disease

- Deficiency of lysosomal enzyme GAA
- Age of onset ranges from infancy to adulthood
- Glycogen accumulation in muscle tissue
- Incidence 1:28,000[1]
- Current ERT suboptimal



Elevated Glycogen in Muscle

[1]Evidence Report – Newborn Screening for Pompe Disease – June 2013 – HRSA.gov

Three Challenges with Pompe ERT

Activity/ Stability	Uptake/ Targeting	Tolerability / Immunogenicity
Rapid denaturation of ERT in pH of blood[1]	Low M6P receptor uptake into skeletal muscle[2] Majority of rhGAA is not delivered to lysosomes[2]	Infusion-associated reactions in ~50% of late-onset patients[3] High antibody titers shown to affect treatment outcomes[4,5]

[1]Khanna *et al.*, **PLoS ONE**, 2012; [2]Zhu et al., Amer. Soc. Gene Therapy, 2009 June; [3]Banati *et al.*, **Muscle Nerve**, 2011 Dec. ; [4]Banugaria *et al.*, **Gen. Med.**, 2011 Aug.; [5]de Vries *et al.*, **Mol Genet Metab.**, 2010 Dec.

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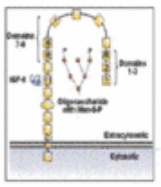
Pompe Development Strategy

Leveraging complementary technologies to address ERT challenges in Pompe disease

CHART
CHAPERONE-ADVANCED REPLACEMENT THERAPY

- Binds to and stabilizes rhGAA
- Increases uptake of active enzyme into tissues
- Potential to improve tolerability and mitigate immunogenicity

Targeting Technology



- Enzyme uniquely engineered with high M6P content and optimized carbohydrate structures
- Peptide tag (variant of IGF-2, or vIGF-2) further enhances drug targeting and uptake

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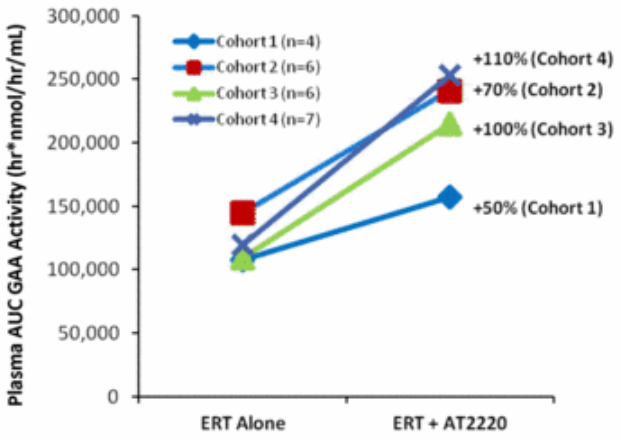
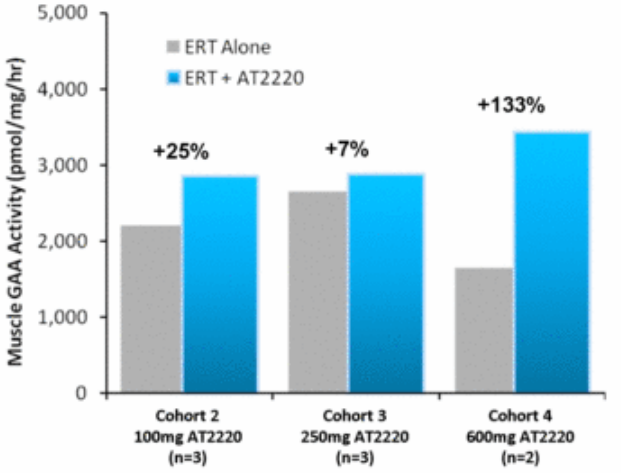
CHART Human Proof-of-Concept: Phase 2 Pompe Co-Administration Study

Co-Administration Consistently Increases Plasma Enzyme Levels and Tissue Uptake Compared to Myozyme/Lumizyme Alone[1]

Plasma AUC GAA Activity



Muscle GAA Activity (Day 3)*



[1]Kishnani, et al., A Phase 2a Study to Investigate Drug-Drug Interactions between Escalating Doses of AT2220 (Duvoglustat Hydrochloride) and Acid Alfa-Glucosidase in Subjects with Pompe Disease, LDN WORLD 2013
*Cohort 1 (AT2220 50 mg) muscle GAA activity not shown; 50 mg dose did not demonstrate meaningful change in tissue uptake (muscle)

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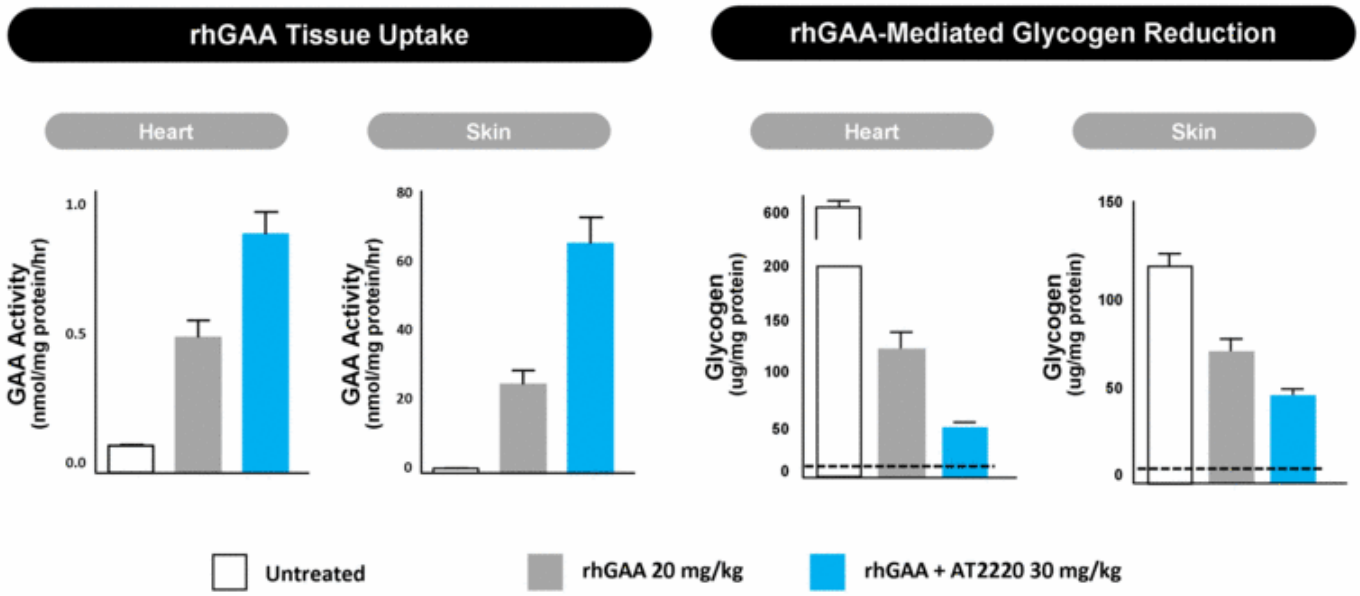
CHART Preclinical Proof-of-Concept: AT2220 + Myozyme/Lumizyme (rhGAA)[1]

Co-Formulation Results in Significantly Greater Tissue Uptake and Further Substrate Reduction Compared to Myozyme/Lumizyme Alone*



*Repeat-dose IV administration in GAA KO Mice

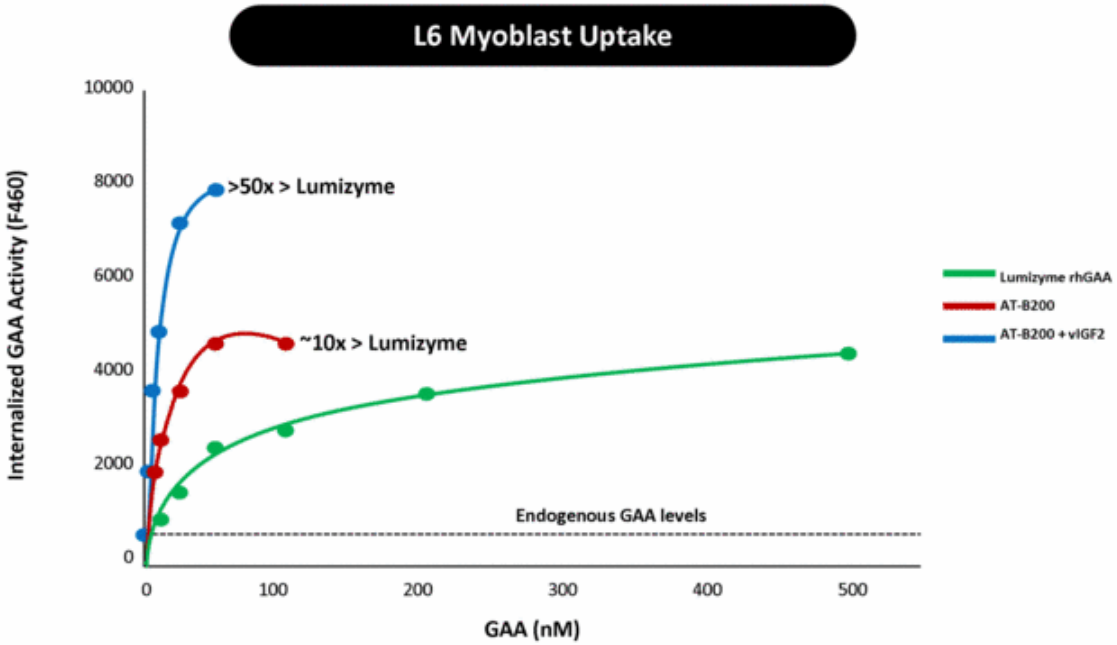
[1]Khanna, *et al.*, Exploring the Use of a Co-formulated Pharmacological Chaperone AT2220 with Recombinant Human Acid Alpha-Glucosidase for Pompe Disease, **LDN WORLD 2013**

Amicus Therapeutics

(Preliminary Results)

AT-B200: Next-Generation Pompe ERT (rhGAA)

AT-B200 Has Demonstrated Significant Advantages in Preclinical Studies that May Be Further Improved By Co-Formulating with a Chaperone



 *Do *et. al*, LDN WORLD 2014

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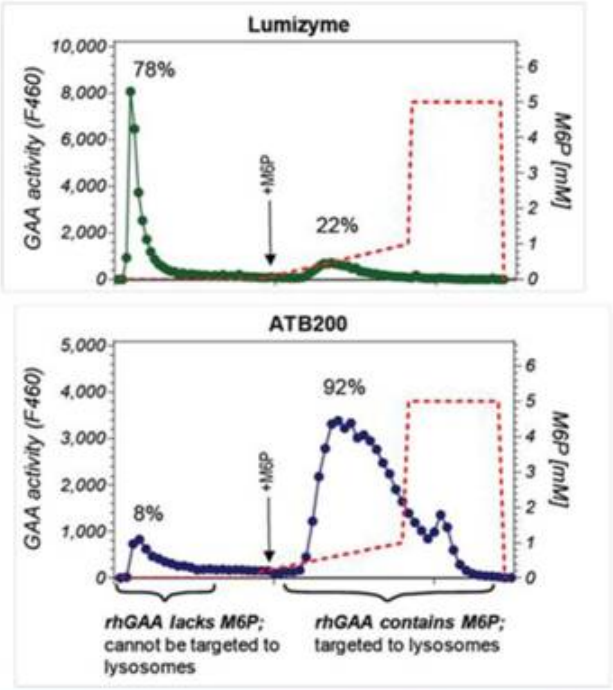
ATB200 rhGAA Contains Higher M6P and Binds M6P Receptor Better Than Myozyme/Lumizyme

Amicus Expertise and Capabilities Enabled Development of Proprietary rhGAA ERT (ATB200) with Optimal Glycosylation for Improved Drug Targeting



- Developed proprietary cell line for producing rhGAA (designated as ATB200)
- ATB200 has significantly higher M6P content than existing rhGAA ERTs
- ATB200 binds intended M6P receptor substantially better than standard of care ERT

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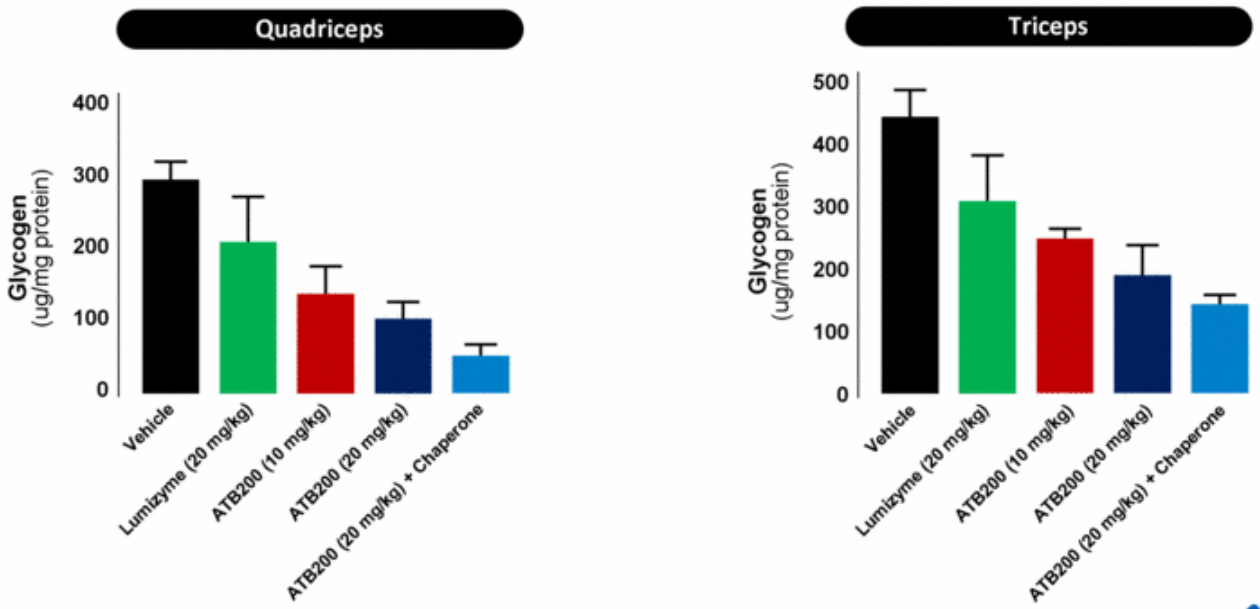
AT-B200: Next-Generation Pompe ERT (rhGAA) Updated Preclinical Proof-of-Concept

AT-B200 Led to Further Glycogen Reduction Compared to Lumizyme in Preclinical Studies in GAA Knock-Out Mice

Residual Muscle Glycogen After ERT



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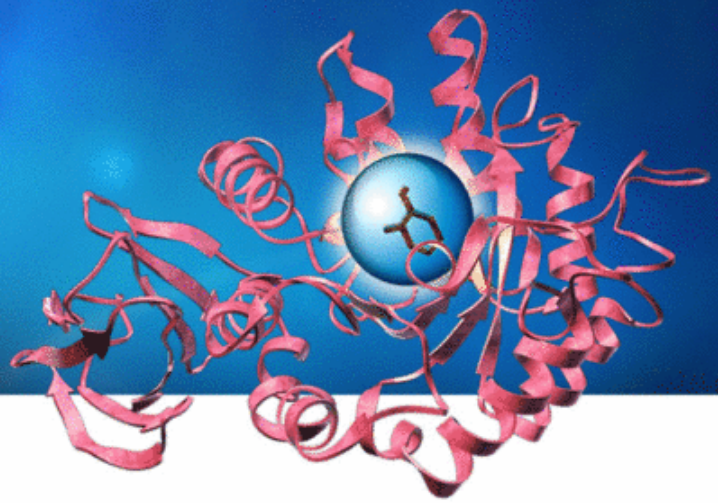
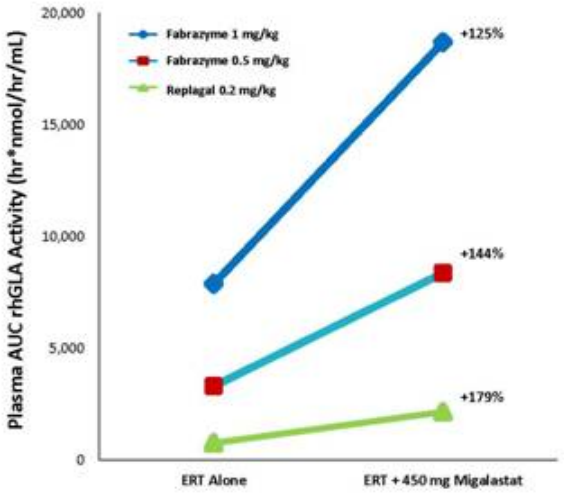


Next-Generation ERT for Fabry Disease

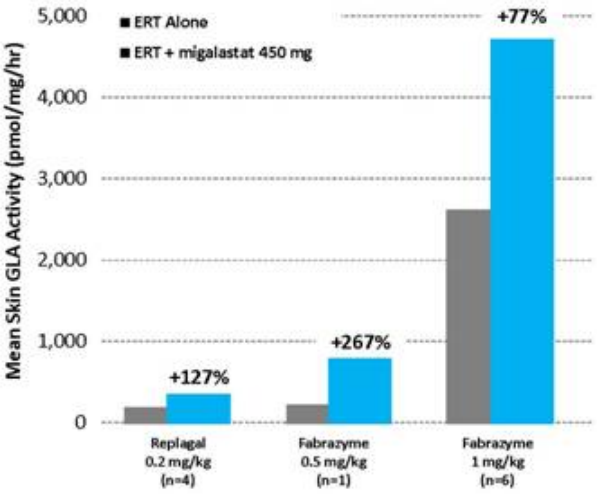
CHART Human Proof-of-Concept: Phase 2 Fabry Co-Administration Study

Co-Administration with Fabrazyme or Replagal Leads to Consistent Increases in Active Plasma Enzyme Levels and Tissue Uptake[1]

Plasma rhGLA Activity (Area Under Curve)



Mean Skin GLA Activity (Day 2)



[1] Bichet, et al., A Phase 2a Study to Investigate the Effect of a Single Dose of Migalastat HCl, a Pharmacological Chaperone, on Agalsidase Activity in Subjects with Fabry Disease, LDN WORLD 2013.

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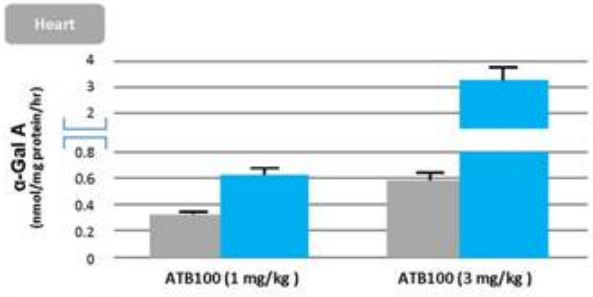
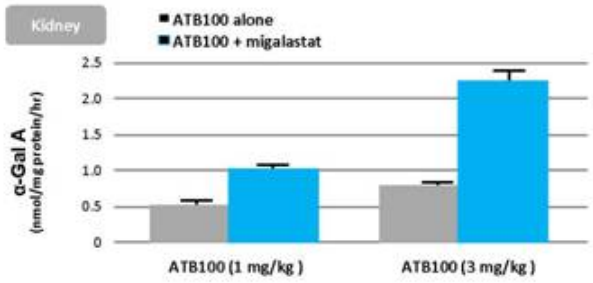
CHART Preclinical Proof-of-Concept: Next-Generation Fabry ERT

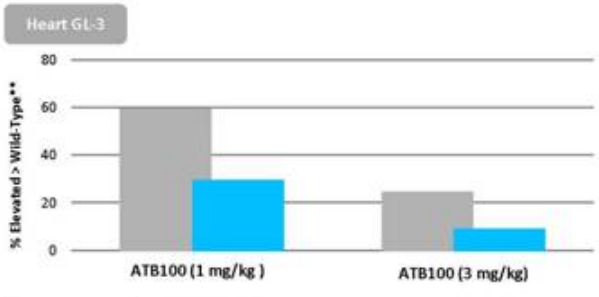
Co-Formulation (ATB100 + Migalastat) Results in Significantly Greater Tissue Uptake and Further Substrate Reduction*

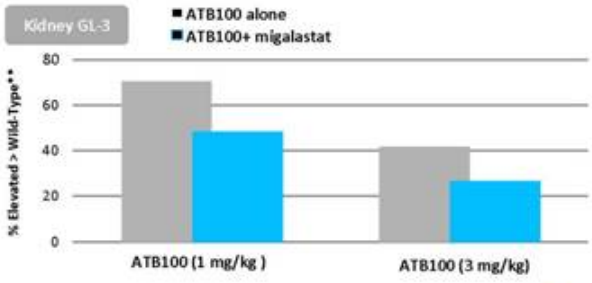
A-Gal A Tissue Uptake





GL-3 Substrate Reduction





*ATB100 +/- Migalastat HCl in GLA Knock-Out Mice (Repeat-Dose IV Administration) *ATB100 designed to be biosimilar to Fabrazyme; **0 = wild-type, 100 = untreated KO mouse

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3-in-3 Strategy: Pathway to Clinic

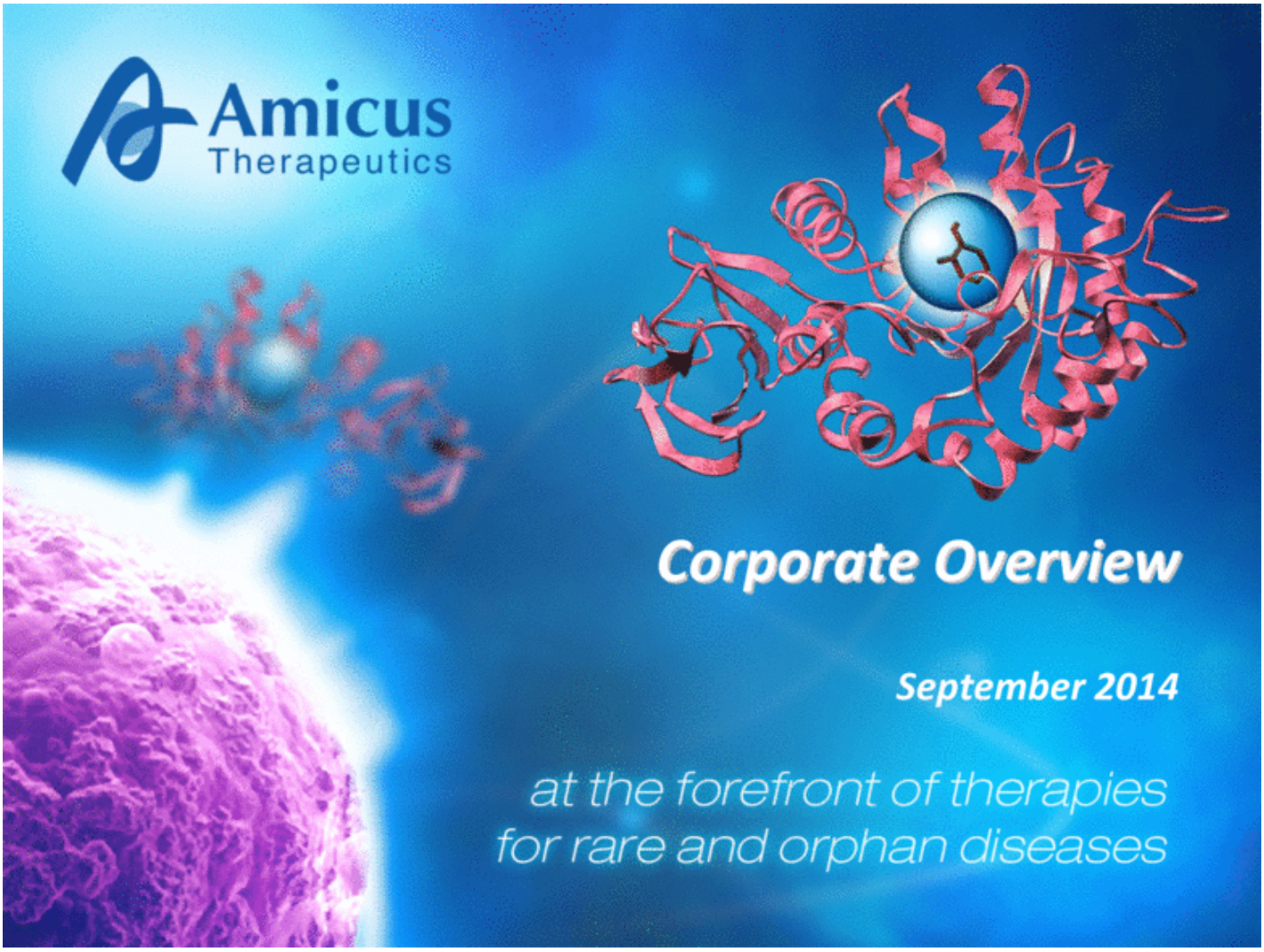
Executing Strategy to Advance 3 Next-Generation ERTs into Clinic in Next 3 Years with Lead Programs in Fabry, Pompe and MPS I

Milestones	Fabry Next-Generation ERT	
1H14	Phase 1 study initiation of IV migalastat in healthy volunteers	✓
4Q14-1Q15	**Phase 1/2 study initiation**	
Milestones	**Pompe Next-Generation ERT**	
1Q14	Initial preclinical proof-of-concept presented at LDN WORLD	✓
Ongoing	Longer-term preclinical proof-of-concept studies to optimize product for clinic with better tissue uptake and enzyme stability	✓
Ongoing	Manufacturing scale-up activities	✓
2H14	**Selection of final drug candidate and begin IND-enabling studies**	
2015	**Phase 1/2 study initiation**	

Current Financial Picture

Successful Execution Under ATM Equity Financing Strengthens Balance Sheet and Provides Runway Under Current Operating Plan Into 2016

Financial Position	June 30, 2014	July 2, 2014
Current Cash:	$78.0M	$98.4M
2014 net cash spend:	$54-59M	
Cash runway:	Into 2016	
Capitalization		
Shares outstanding:	72,869,861	78,685,241


Amicus
Therapeutics
Corporate Overview
September 2014
at the forefront of therapies
for rare and orphan diseases