CORPORATE OVERVIEW MAY 2019

APPLIED THERAPEUTICS

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Corporate Vision & Strategy

A New Way to Develop Drugs

Novel product candidates against previously validated and well known molecular targets, leveraging abbreviated regulatory pathways & recent technological advances to design improved drugs

Biomarkers	**Experienced Leaders Flexible Thinkers**	**Validated Molecular Targets**	**High Unmet Need Indications**	**New Regulatory Guidelines**

Expedited development, delivering drugs to patients more quickly

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Investment Highlights

Novel pipeline based on unlocking the potential of Aldose Reductase (AR) inhibition

- Broad applications for high unmet need in AR-mediated indications
- AT-001: Large Phase 1/2 SAD/MAD trial in diabetic patients demonstrated POC, no SAEs
- AT-003: Proof-of-concept in models of retinopathy
- AT-007: Proof-of-mechanism in Galactosemia (rare pediatric metabolic disease)

High operational efficiency

- Reduced cost and timeline for development expected based on abbreviated development regulatory framework
  - High unmet need indications
  - Potential to use biomarkers and other non-outcomes-based endpoints

Strong IP portfolio covering composition of matter and method of use in target indications, with coverage through 2030's for each patent family

Near term clinical milestones enhance value

- AT-001: Phase 2/3 registrational trial expected to start in 2019 with interim data expected in mid 2020
- AT-007: Phase 1 SAD/MAD data in adults expected Q3 2019
- AT-003: IND submission and Phase 1 data expected in H1 2020

Pipeline

Compound	Preclinical	Phase 1	Phase 2	Phase 3*	Dosing Route	Target Tissue	Anticipated Milestones
Aldose Reductase Franchise							
AT-001	Diabetic Cardiomyopathy				Oral	Systemic	Initiate Ph 2/3 2019
AT-001	Diabetic Peripheral Neuropathy				Oral	Peripheral Nerve	
AT-001	Acute Myocardial Infarction				SC**	Systemic / Peripheral Nerve	
AT-007	Galactosemia				Oral	CNS	Initiate Ph 1 in adults in H1 2019
AT-003	Diabetic Retinopathy				Oral	Retina	Preclinical data 2019; Initiate Ph1 2020
PI3 Kinase Franchise							
AT-104	PTCL, CTCL, TALL***				SC / Oral	Selective δ/γ inhibitor	Initiate Ph 1 2020

* We plan to initiate a pivotal Phase 2/3 clinical trial of this product candidate as the basis for applying for marketing approval with the FDA
** Subcutaneous
*** Peripheral T-cell lymphoma, cutaneous T-cell lymphoma and T-cell acute lymphoblastic leukemia

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Unlocking the Potential of Aldose Reductase Inhibition

Validated Target Resistant to Therapeutic Development	• AR known to play a key role in diabetic complications and heart disease • Past efforts failed to produce sufficiently potent, selective and tolerable drugs
Recent Advances Enable Improved ARI's	• New understanding of structural changes within the active site of AR following enzymatic activation – Utilized advanced crystallography techniques and in situ structural design • Increased potency and selectivity compared to prior compounds with none of the prior off-target safety issues to date
R&D and Regulatory Opportunities	• High unmet need in numerous AR-mediated diseases • Leverage prior ARI programs for streamlined, abbreviated development of our novel compounds • Potential to utilize regulatory pathways designed for accelerated drug development

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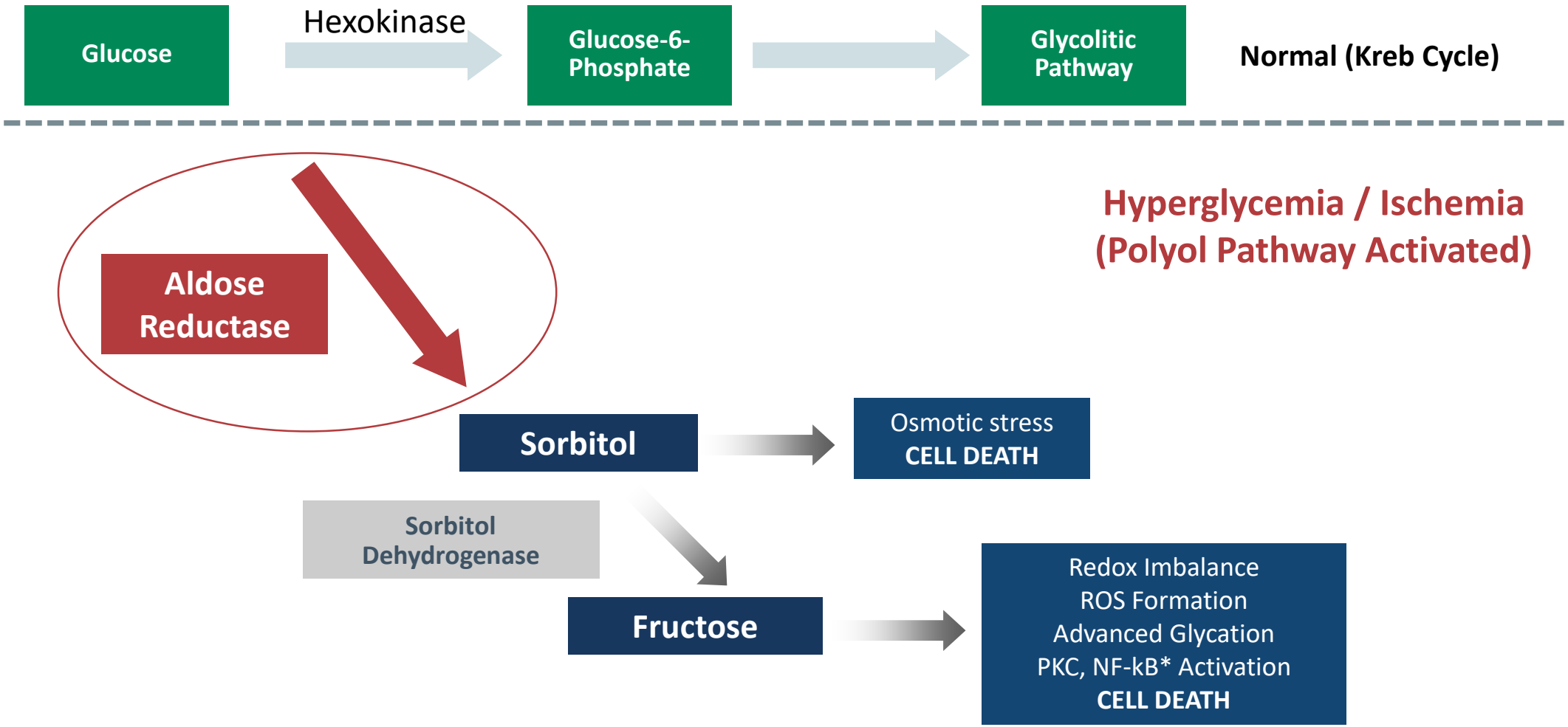
Aldose Reductase Causes Damage to Tissues Under Conditions of Oxidative Stress



*Nf-kB is a protein complex that controls transcription of DNA, cytokine production and cell survival

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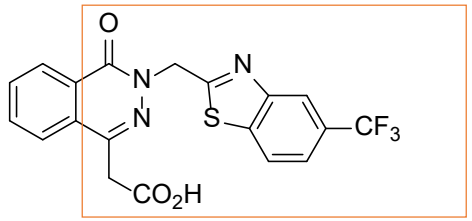
Novel Chemistry For Better Drugs

Backbone



zopolrestat

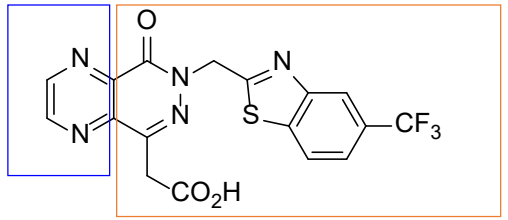
Similar backbone to zopolrestat (prior best in class efficacy, but liver tox issues)

Technological Advancements

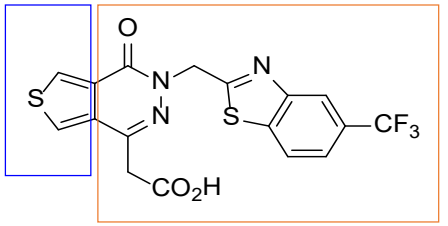
- Advanced crystallography provided novel understanding of structural changes within AR active site
- Many prior ARIs were unable to inhibit redox-activated AR

Impact of Modified Structure

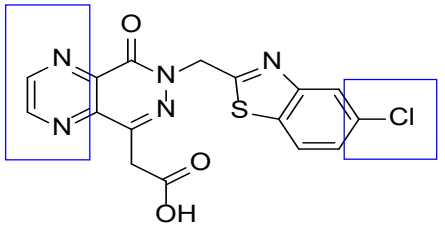
- Functional modifications improve compound's binding affinity and specificity
- Novel dimeric binding within the catalytic core
- Higher enzymatic inhibitory activity
- Increased selectivity leads to less off-target activity and potentially better safety



AT-001



AT-007



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Compound Differentiation

Compound	IC_{50}[1]	Maximum Tolerated Dose in Animals	LogD[2]	Tissue Penetration (in rats)		
				Systemic/ Nerve	Retina	CNS
AT-001	30pM	>2,000mg/kg	-1.00	✓	✓	X
AT-007	100pM	>1,000mg/kg	-0.09	✓	✓	✓
AT-003	54pM	>1,000mg/kg	-1.53	✓	✓	X
Zopolrestat (prior Pfizer compound)	10nM	100mg/kg	+0.06	✓	X	X

- High potency (>1,000x more potent than prior best-in-class ARI)
- Targeted penetration into specific tissues

(1) IC_{50} is the amount of a compound required to inhibit 50% of enzyme activity
(2) LogD is a log of partition of a chemical compound between the lipid and aqueous phases. LogD often predicts retinal permeability, with compounds with negative LogD passing through the back of the eye

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Addressing Large Indications in Areas of High Unmet Medical Need – Opportunities for Abbreviated Clinical Development

Indication	Prevalence	Market	Unmet Need	Development Strategy
Diabetic Cardiomyopathy	17-24% Diabetics	~77M patients worldwide	• No therapies approved • No known drugs in development • Entresto approved in stage 4 disease	Independent; Abbreviated Development
Retinopathy	35% Diabetics	~158M patients worldwide	• 2 therapies approved (intravitrial injection) • Anti-VEGFs only for late stage disease	Independent; Abbreviated Development
Diabetic Peripheral Neuropathy	50% Diabetics	~226M patients worldwide	• No disease-modifying therapies approved • Only symptomatic treatments available (Lyrica) • Epalrestat, an off-patent ARI, approved in Japan, China, India	Strategic Partner; Standard Development
Galactosemia	1/50k to 1/90k	~2,800 patients in the US	• No therapies approved; lactose dietary restriction not sufficient • No known drugs in development	Independent; Abbreviated Development (includes PRV)

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AT-001 for Diabetic Cardiomyopathy

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Diabetic Cardiomyopathy (DbCM)

Burden of Disease	Standard of Care
• Early disease asymptomatic; structural changes to the heart limit contractility and decrease plasticity (fibrosis) (1-2 yrs) • Decrease in heart function causes symptoms – shortness of breath and limitations in daily activities • Progresses to overt heart failure and death in many cases within 9 years	• Diagnosis by echocardiogram & exclusion of other types of heart disease • Counseling on diabetic glucose control to limit hyperglycemia (ineffective) • No treatments available; no drugs used off label to slow disease progression

	Stage 1	Stage 2	Stage 3	Stage 4
Symptoms	Asymptomatic; no limitations at rest or on exercise	Mild symptoms; shortness of breath on exercise and ordinary activities	Marked limitation in activity; comfortable at rest	Severe limitations; experience symptoms even at rest
Imaging/ functional features	Decreased tissue velocities			
	Increased LV mass	Increased LV mass & wall thickness		
	Diastolic dysfunction	Diastolic & systolic dysfunction		Severe dysfunction
	Normal EF	Normal EF	EF <50%	
	No cavity dilation	Mild cavity dilation	Marked dilatation	Severe dilation

Initially targeting patients in stage 2 and 3 (~50% of all DbCM patients likely to be most responsive to treatment) with incremental opportunity to target patients in stage 1 and 4

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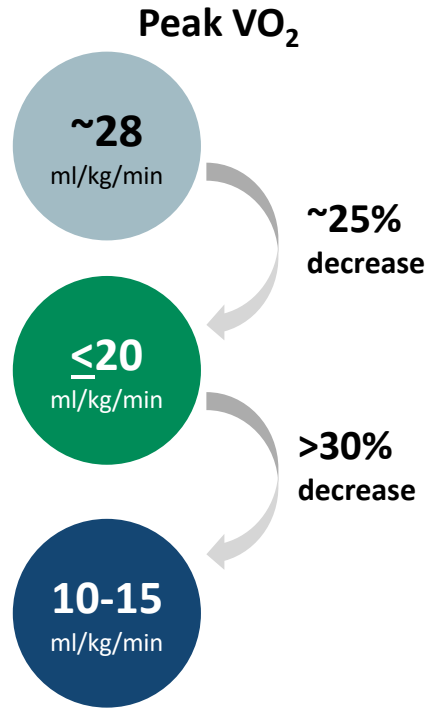
Understanding Diabetic Cardiomyopathy as a Form of Heart Failure

Diabetes **Stage A Heart Failure**	• Metabolic derangement of the myocardium due to diabetes
DbCM **Stage B Heart Failure**	• Cardiac structural abnormalities • Diastolic dysfunction; LVH • Early symptoms of DbCM; noticeable impact on activities • Decreased exercise capacity
Stage C Heart Failure	• Overt Heart Failure • HFpEF or HFrEF • Significant impact on daily activities
Stage D Heart Failure	• Refractory Heart Failure requiring specialized interventions (e.g. LV Assist Device) • Inability to complete daily activities



- ~24% of DbCM patients progress to overt heart failure or death within 1.5 years
- 37% within 5 years

References: Kosmala et al, JACC V O L . 6 5 , NO . 3 , 20 1 5; Swank et al. Circ HF 2012; Wang et al. JACC: Cardiovasc Imaging 2018; From et al. JACC 2010

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Strong Rationale for AT-001 Development in Diabetic Cardiomyopathy: First-in-Class Potential

Building on Prior Body of Evidence

- The role of AR in DbCM is well supported by preclinical and clinical evidence
- Proof of mechanism: Pfizer's zopolrestat achieved proof-of-concept on LVEF in Phase 2 Diabetic Cardiomyopathy trial
- Literature: Effects on heart function (LVEF) leads to effects on exercise tolerance (peak VO_2)

AT-001's Robust Pre-Clinical Profile

- 1,000X more potent than prior best-in-class ARI (zopolrestat), in vitro and in vivo
- Broad exposure: Cardiac and nerve tissue
- Highly favorable preclinical profile: MTD>2,000mg/kg

AT-001's Robust Clinical Profile (Ph 1/2 trial)

- Clinical proof-of-concept via sorbitol biomarker observed in T2D patients
- No drug related AEs observed at any dose; well tolerated
- Heart inflammatory biomarkers in 28 day arm in DbCM patients inform dose selection for pivotal study

AT-001 for Diabetic Cardiomyopathy: Path to Registration and Beyond

Current Phase 1/2 SAD/MAD Trial

- Phase 1/2 trial in ~120 Type 2 Diabetic patients (all on concomitant glucose control and SOC meds)
    - 30 with DbCM
- Well tolerated over 28 days; no drug-related AEs reported in study
- Proof of biological activity demonstrated - sorbitol normalization achieved
- Cardiac biomarker (NTpro-BNP) examined in 30 patients with early DbCM treated for 28 days

Future Path to Registration

- Potential for approval based on a single Phase 2/3 pivotal trial
- Primary endpoint: Peak V02 (CPET)
    - Stabilization or decrease in slope of decline (powered to detect ≥6% difference)
- 2 doses AT-001 vs. placebo; ~600 patients
- 1 year treatment duration; interim biomarker analysis at 6 months
- Symptomatic patients (Stage B Heart Failure)
- Extension at 18 and 24 months to examine hard HF outcomes (hospitalizations, CV death, progression to Stage C HF)

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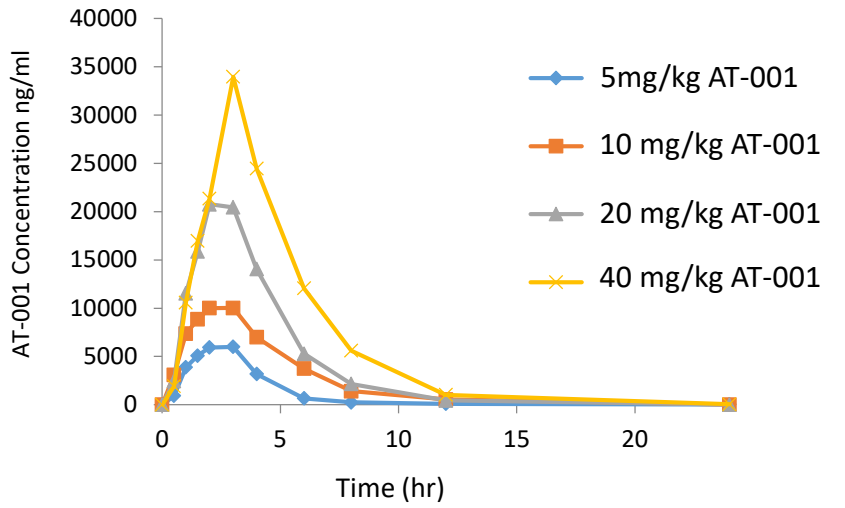
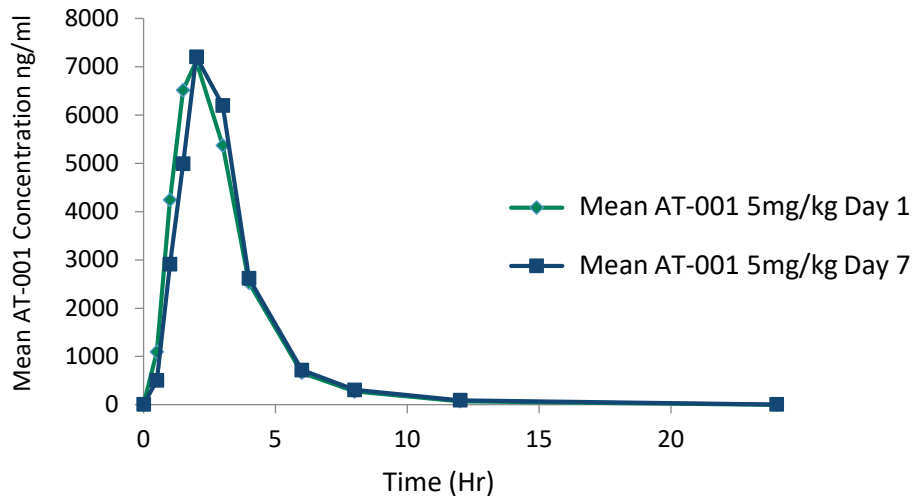
AT-001 Pharmacokinetics (in Rapid Release Capsule)

Mean PK Timeframe for Phase 1 SAD Cohorts (each curve represents mean of eight patients)



- Linear dose dependence on C_{max} and AUC confirms clean absorption in the gut and good bioavailability

Multiple Dose PK Profile – No First Pass Clearance or Drug Accumulation



- Half-life of the drug in rapid release capsule is 3-6 hours at higher doses
- Effects on enzyme inhibition for 10-12 hours per dose

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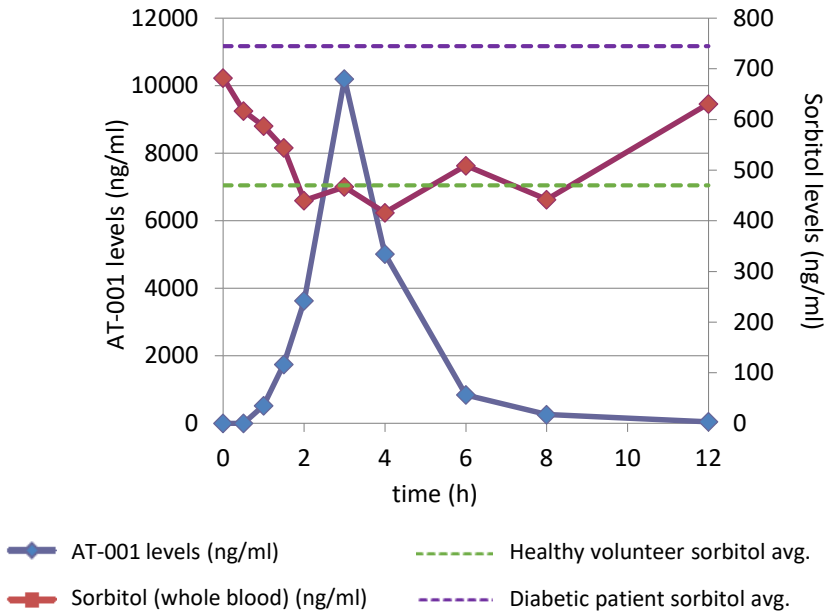
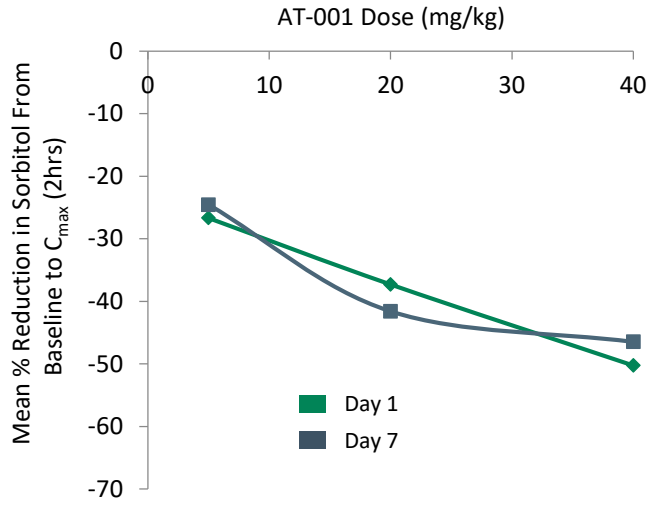
AT-001 Normalizes Sorbitol in Diabetic Patients

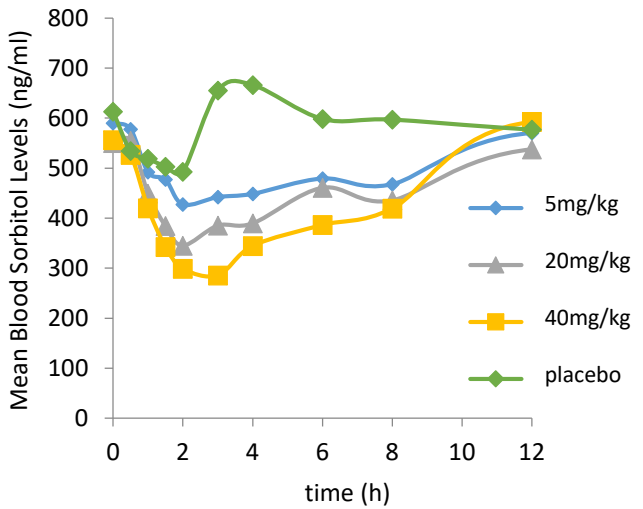
Proof of Biological Activity



Sorbitol Reduction by Dose



Sorbitol Normalization Over Time



- Drug treatment with AT-001 normalized sorbitol to healthy volunteer levels

- Mean reduction in sorbitol at Day 1 and Day 7: Results are persistent over 1 week of treatment
- At 40mg/kg patients were normalized to healthy volunteer sorbitol levels, demonstrating complete AR inhibition

- Rapid release capsule provides sorbitol normalization effects (PD biomarker) through 10-12hrs post-dose at >10mg/kg
- Includes protection from food-related sorbitol spikes during times of post-prandial hyperglycemia

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AT-001 Phase 1/2 Trial in Type 2 Diabetic Patients

Parts A & B

Design

- 80 Type 2 Diabetic Patients
- All patients remained on concomitant meds
- 40 patients in SAD – (5, 10, 20, 40mg/kg)
- 40 patients in MAD – (5, 20, 40mg/kg; 20mg/kg BID)
- 8 drug treated & 2 placebo in each cohort

Results

- No drug-related AEs in entire study (up to 7 days treatment)
- No abnormal labs
- Normalization of sorbitol (PD biomarker)

Part C

Design

- 30 DbCM patients
- 10 patients per cohort (8 drug treated, 2 placebo)
- 3,000mg/day
  - ER tablet once daily
  - 1,500mg BID (rapid release capsule)
  - 1,000mg TID (rapid release capsule)

Results

- No drug-related AEs in entire study (up to 28 days treatment)
- No drug-related lab abnormalities
- Effect on cardiac biomarker NTproBNP

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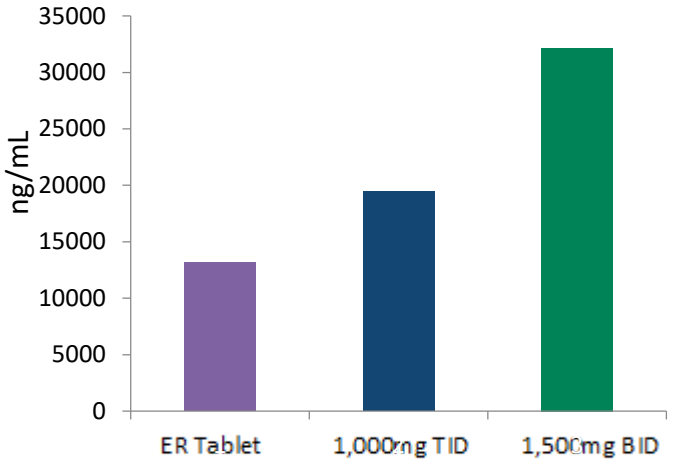
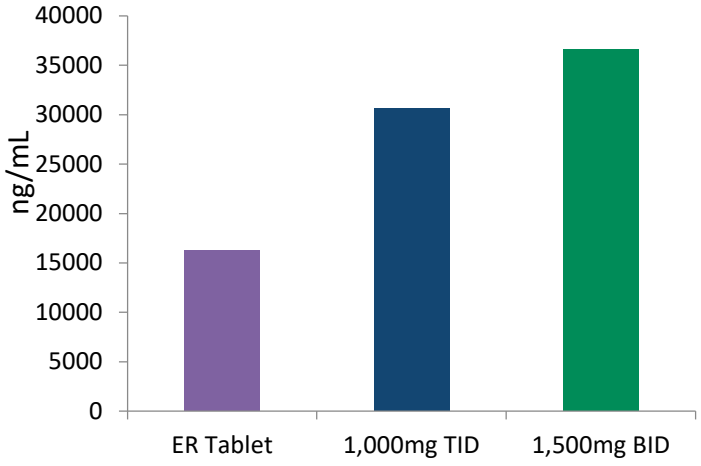
Part C Dosing Posology and Pharmacokinetics

- BID and TID rapid release capsule dosing provided sustained drug levels over 24 hours
  - ER tablet did not release drug as predicted, providing low overall exposure; defines minimally efficacious dose

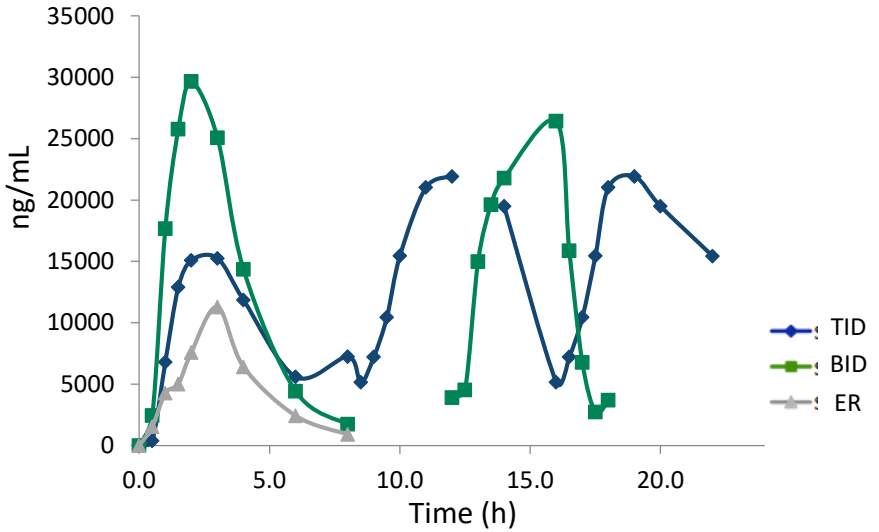
AT-001 C_{max} (mean)



AT-001 AUC (mean)



AT-001 PK profile



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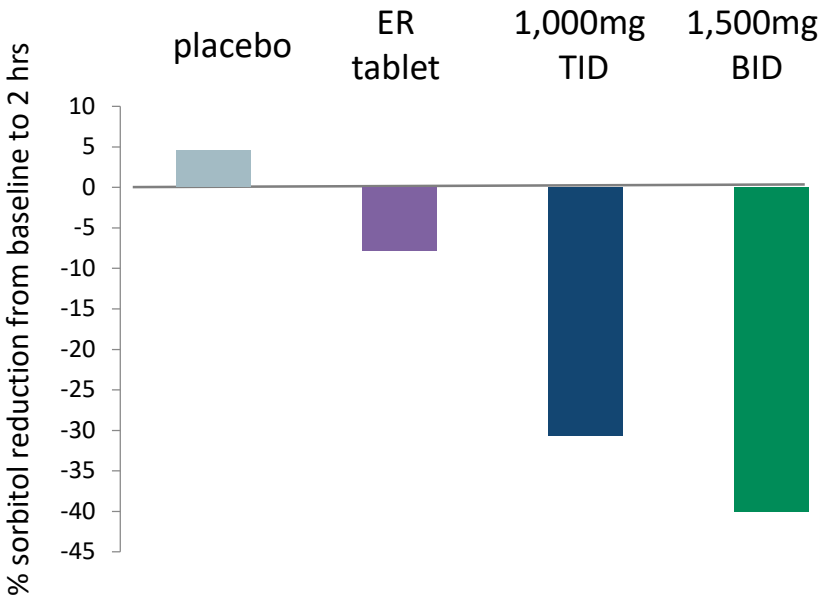
Sorbitol Reduction Correlates with AT-001 Drug Exposure: Rapid Release Capsule Superior to ER Tablet

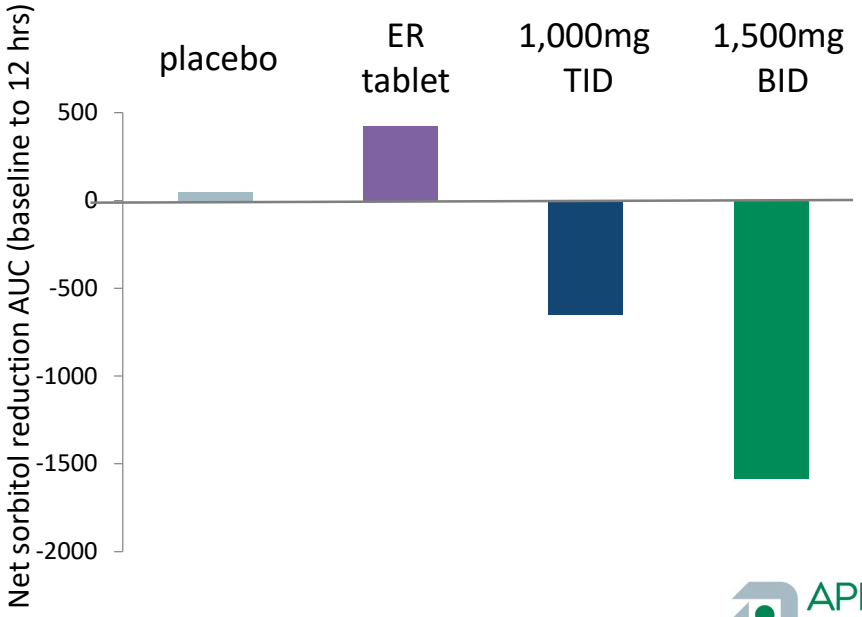
- Higher C_{max} at first dose achieved with 1500mg BID provided greater sorbitol reduction at 2 hours post-dose

- Sorbitol reduction over 12 hours as measured by AUC is greatest for 1000mg TID
- Significant AUC sorbitol reduction AUC achieved by both 1500mg BID and 1000mg TID AT-001

Sorbitol % Change at C_{max}



Sorbitol AUC 1-12h



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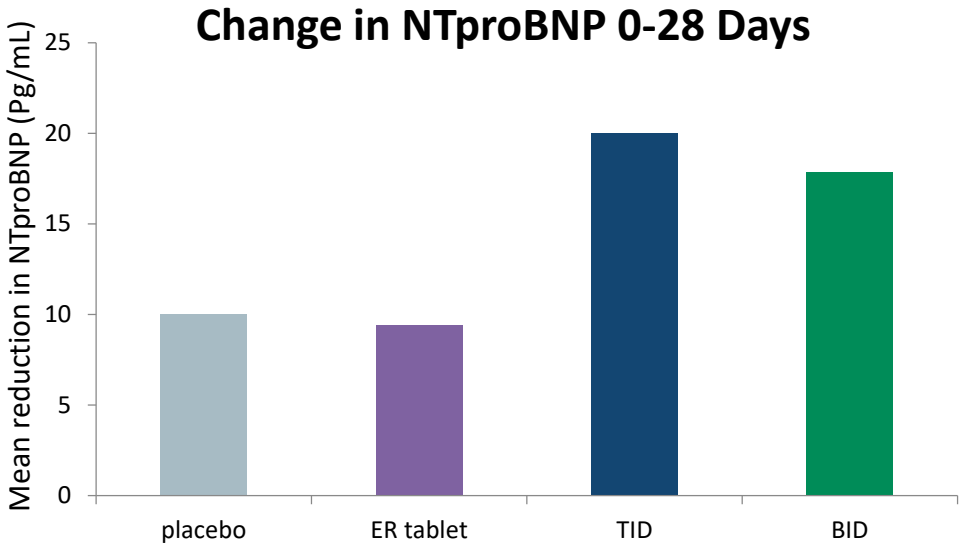
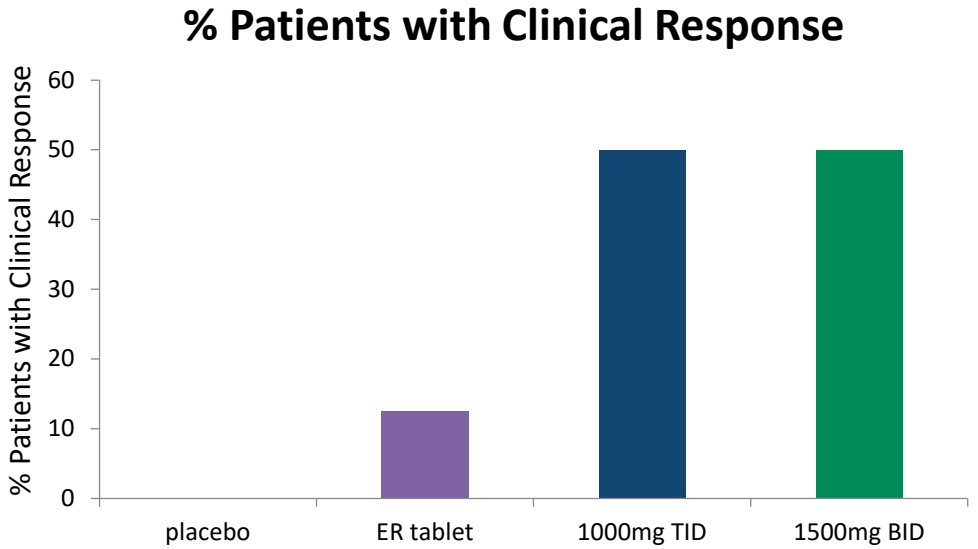
AT-001 Treatment for 28 Days Decreases NT proBNP in Early Stage DbCM Patients

- NTproBNP: Prohormone released in response to changes in left ventricular pressure
  - Used to diagnose overt heart failure and Acute MI
  - Higher levels of in diabetics correlates with higher risk of heart failure and worse outcomes
- Mean change in NTproBNP from baseline to 28 days was greater in patients that received AT-001 BID or TID vs. placebo or ER tablet

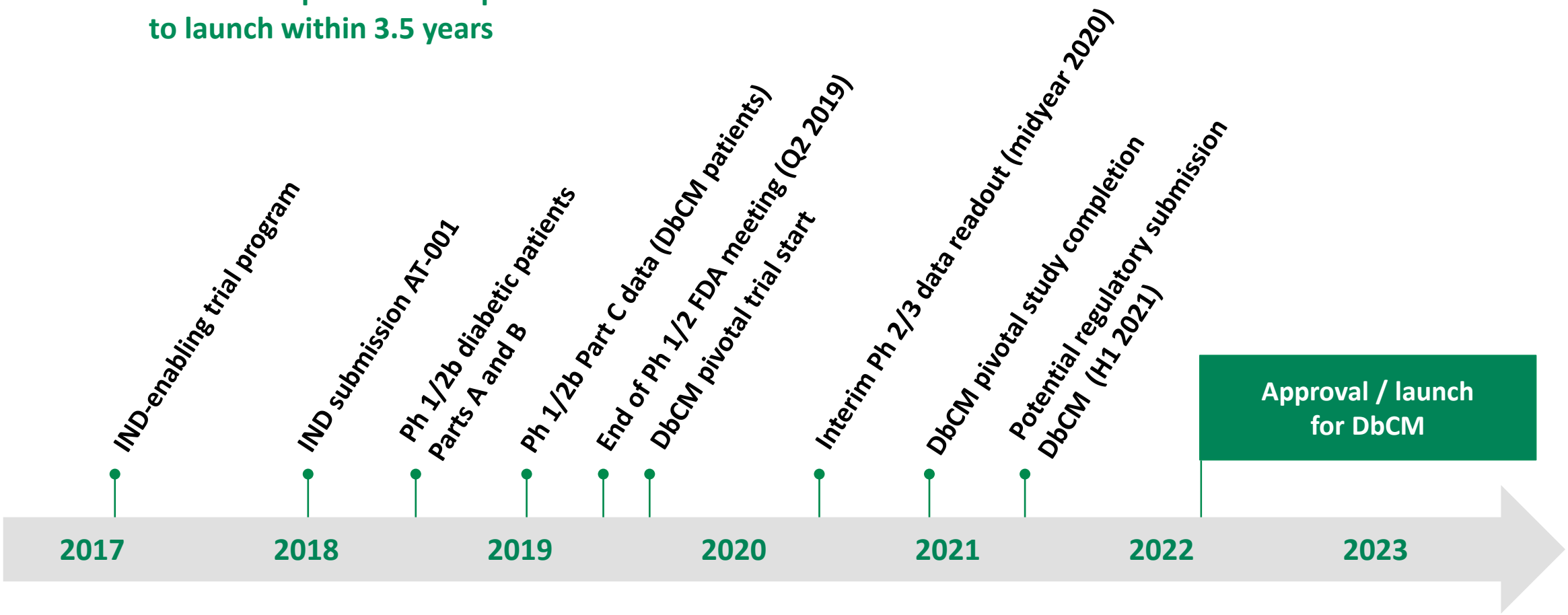
- 50% of BID/TID treated patients showed a clinically meaningful reduction in NTproBNP at 28 days
  - Defined as >25 pg/mL reduction from baseline
  - Baseline range was 30-235pg/ml
  - Mean at baseline was 65pg/ml





Anticipated Development Timeline
Diabetic Cardiomyopathy

Abbreviated development offers potential to launch within 3.5 years



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AT-001 for Diabetic Peripheral Neuropathy

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Diabetic Peripheral Neuropathy

Burden of Disease

- Aldose Reductase activity in neurons causes osmotic dysregulation and cell death/neuronal dysfunction
- Tingling/burning/stinging sensation and loss of feeling in peripheral tissues
- Significant impact on quality of life and pharmacoeconomic metrics (ability to work)

Standard of Care

- No disease modifying therapies approved
- Epalrestat (ARI) approved for 20+ years in Japan: dosed 3-5x/day; numerous side effects
- Standard of care outside of Japan/China is analgesic (pain) management, primarily Lyrica

Building on Prior Body of Evidence

- Epalrestat is understood to be safe and moderately effective, but unfavorable PK profile (5X daily dosing)
- Never approved in US/EU; now generic in Japan/China
- Phase 4 trials in Japan demonstrated statistical effects on MNCV and symptomatic pain (Hotta et al)

Current Phase 1 SAD/MAD Trial

- Current AT-001 Phase 1 results show favorable PK vs. Epalrestat
- DPN metrics (MNCV) will be captured in Phase 2/3 pivotal Diabetic Cardiomyopathy trial
- Demonstrate POC for AT-001 in DPN and inform on dose selection for registrational DPN trials

Future Path to Registration

- Will require “typical” path to registration
  - 2 large Phase 3 trials
- Design will follow Epalrestat Phase 4 trials– careful selection of patient population and performance of endpoint testing
- Likely to pursue strategic partnership with large pharmaceutical company

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AT-003 for Diabetic Retinopathy

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AT-003 for Diabetic Retinopathy

Burden of Disease

- One of the major causes of blindness worldwide
- Current therapies (anti-VEGFs) are high cost biologics that require intravitreal administration by an ophthalmologist
- Limited access for patients and high economic burden
- AR is an upstream target vs. VEGF – opportunity to blunt damage to the eye at the earliest stages

Standard of Care

- Current treatments (anti-VEGF therapies) target downstream consequences of diabetic complications in the eye
- Lucentis & Eylea are leading approved therapies for DME; limited to treating later stage / more severe stages of disease

Building on Prior Body of Evidence

- Clear proof of mechanism: AR activation / increased sorbitol as the initial pathogenesis of retinopathy is well supported
- Sorbitol build up in the lens causes osmotic dysregulation
- AR knock-out mice do not develop diabetic retinopathy; AR over-expressing mice develop retinopathy earlier than WT
- 2 prior ARIs met endpoints in Phase 2 trials, but were toxic

AT-003 in Preclinical Development

- Proof-of-concept in animal models of retinopathy
- AT-003 displays a similar PK to AT-001, but has greater retinal penetrance
- IND-enabling studies and manufacturing scale up are under way

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AT-007 for Galactosemia

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AT-007 for Galactosemia

Burden of Disease

- Rare genetic metabolic disease caused by inability to break down galactose
  - Metabolite of lactose
  - Produced de novo by cells
- Even with strict dietary restriction of external lactose, endogenous galactose is produced within the body, leading to toxic build-up of galactitol
- Long-term consequences of disease include: Frequent pre-senile cataracts, significant motor, speech, cognitive, and psychiatric impairments, and ovarian insufficiency

Standard of Care

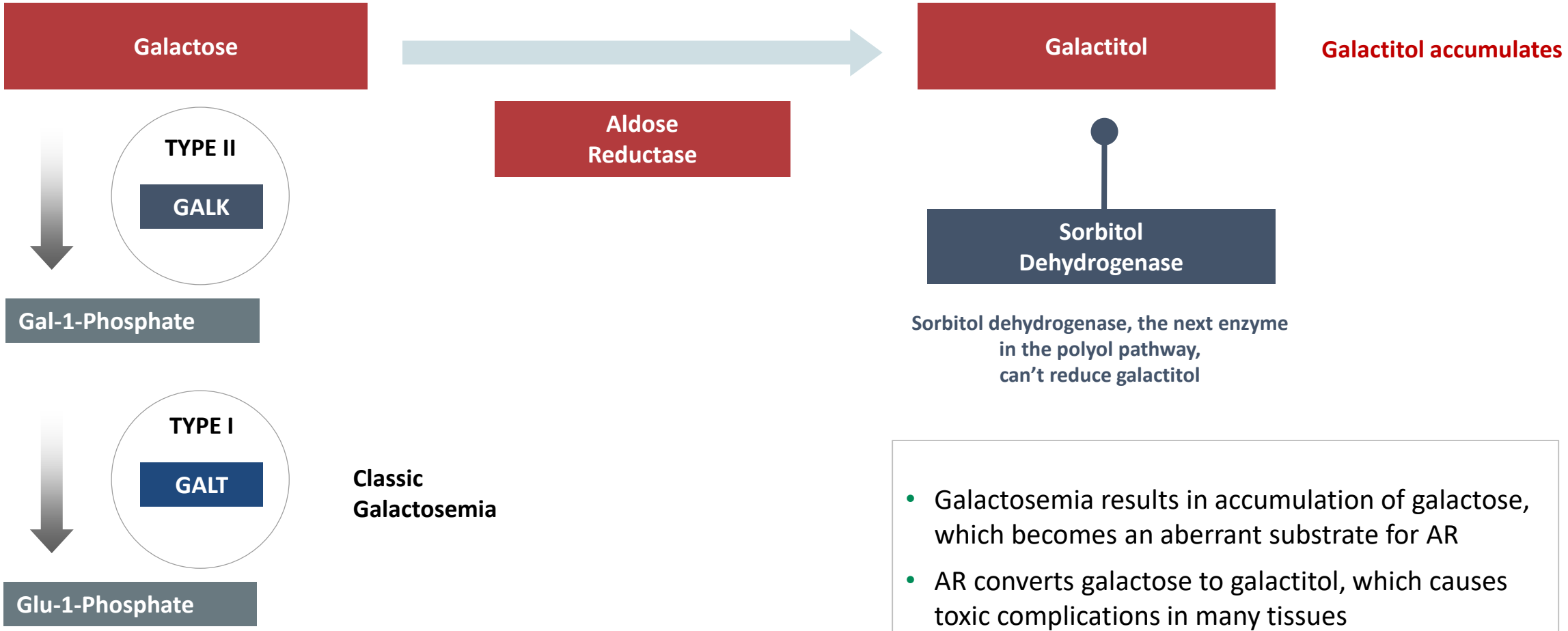
- Mandatory newborn screening in the US/EU; potentially fatal if undetected in first weeks of life and infant is exposed to lactose in breast milk or formula
- No approved therapies
- Standard of care is strict dietary restriction of lactose and galactose, which prevents fatalities, but **does not prevent long term consequences of disease**
- Greatly impacts children's development potential and quality of life (causes severe and permanent cognitive, intellectual and speech deficiencies)
- In adults, frequent cataracts due to galactitol build up in the eye; many develop persistent tremors

Galactosemia Market & Regulatory Environment

- Incidence **1:50,000-1:90,000**
- However, actual number of live patients is much lower than projected; prior to newborn screening, nearly all infants with Galactosemia died
- **~2,800** US patients
- Births per year are estimated at **~80** in the US
- Majority of patients are under the age of 40
- Is a “low prevalence” disease as defined by the FDA

Regulatory Guidelines: Because Galactosemia is a “slowly progressing” rare metabolic disease, under new FDA guidance, surrogate metabolic biomarkers may be acceptable for demonstration of therapeutic activity = low burden of clinical development

Aldose Reductase Activity Causes Toxic Accumulation of Galactitol in Galactosemia



- Galactosemia results in accumulation of galactose, which becomes an aberrant substrate for AR
- AR converts galactose to galactitol, which causes toxic complications in many tissues

APPLIED THERAPEUTICS

AT-007: Oral CNS Penetrant Aldose Reductase Inhibitor – Summary of Preclinical Data

Preclinical Profile	• Structurally distinct molecule with potent AR inhibition and unique PK profile • Exposure to all Galactosemia target tissues – CNS, nerve and retina penetrant • Oral once-daily dosing (half life 12-18 hrs)
Safety	• IND-enabling studies completed • No safety issues in newborn rat treatment studies, supporting eventual infant/pediatric use
Preclinical Disease Model Takeaways	• Prevented complications of disease in a newborn Galactosemia rat model • Prevented galactosemic cataract formation and prevented CNS abnormalities (rotarod) • Clear biochemical effects correlate with clinical endpoints • Reduced galactitol levels in serum and affected tissues • Did not increase galactose levels or levels of other galactose metabolites (Gal1P)

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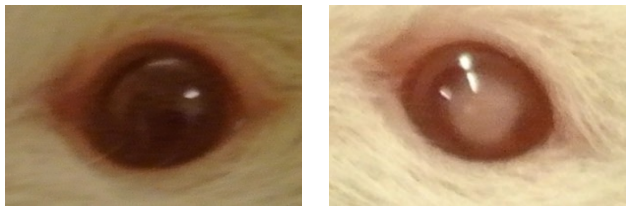
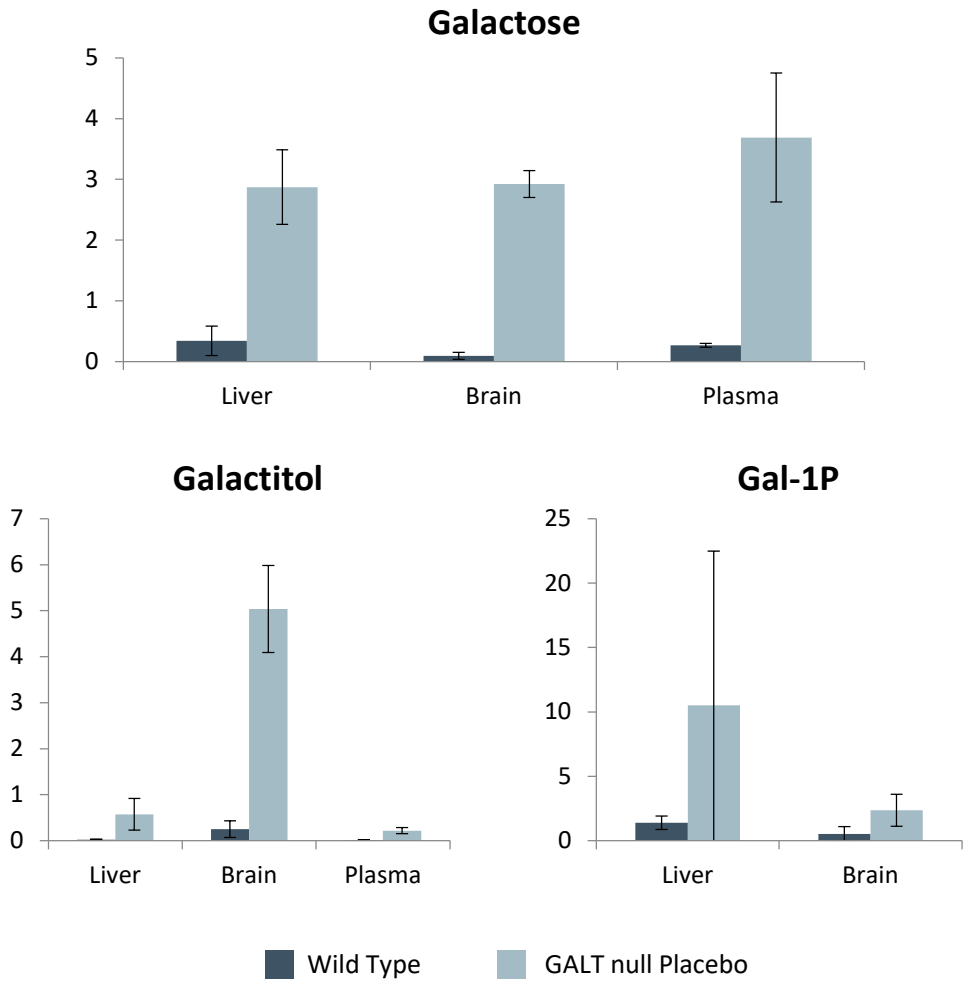
Galactosemia Animal Model:
GALT Deficiency (Classic Galactosemia) Rat

- GALT deficient rats closely mirror human disease:
  - Bilateral cataracts
  - Biochemical effects on galactitol, galactose and Gal1P similar to those seen in humans
  - CNS deficiencies indicative of cognitive, intellectual, memory and motor abnormalities
- To date, no evidence of tremor or ovarian insufficiency

WT GALT null





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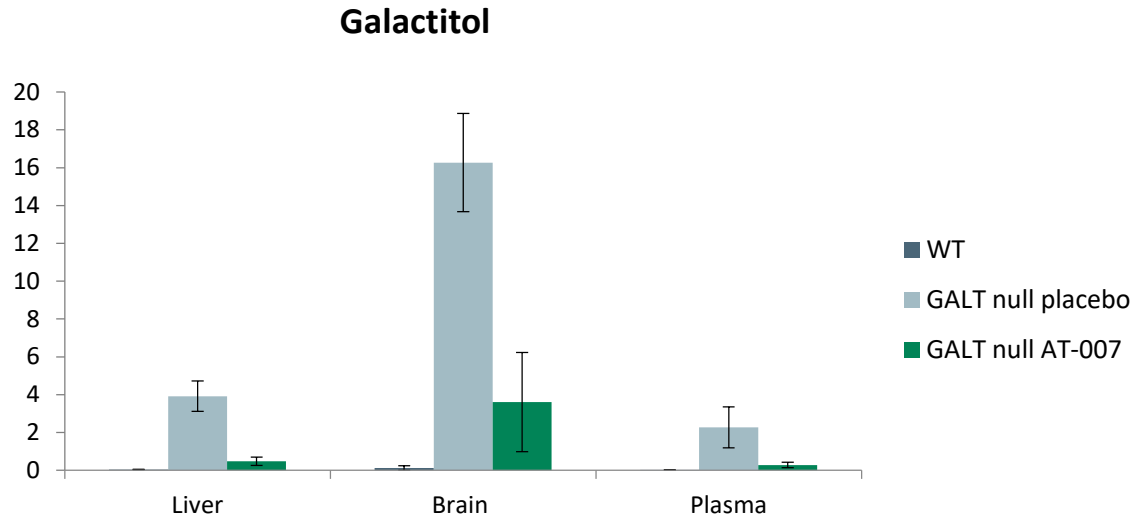
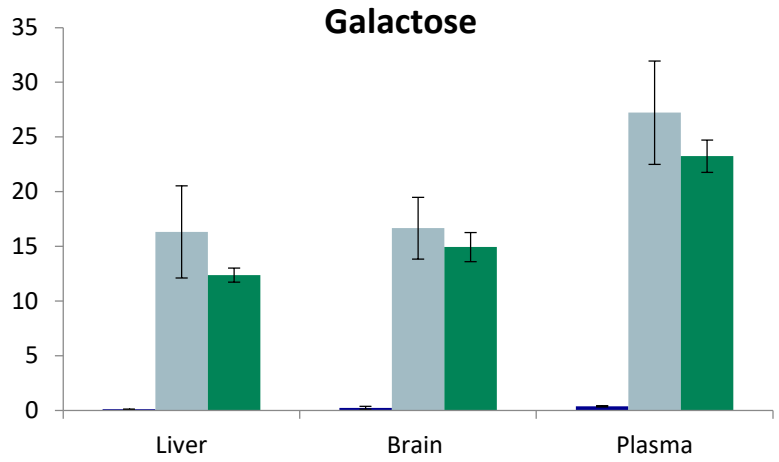
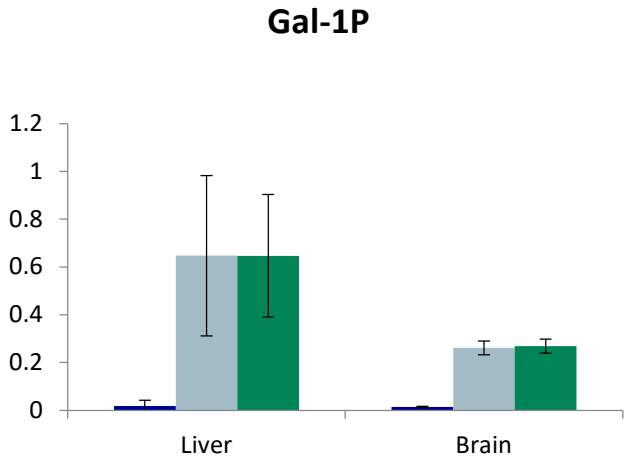
AT-007 Significantly Reduces Galactitol Levels in GALT Null Rats in all Target Tissues



- AT-007 treatment from neonatal Day 1 to Day 10 significantly reduced galactitol in liver, brain and plasma
- Treatment did not increase galactose or Gal1P levels; similar results seen at Day 22 and age 5 months





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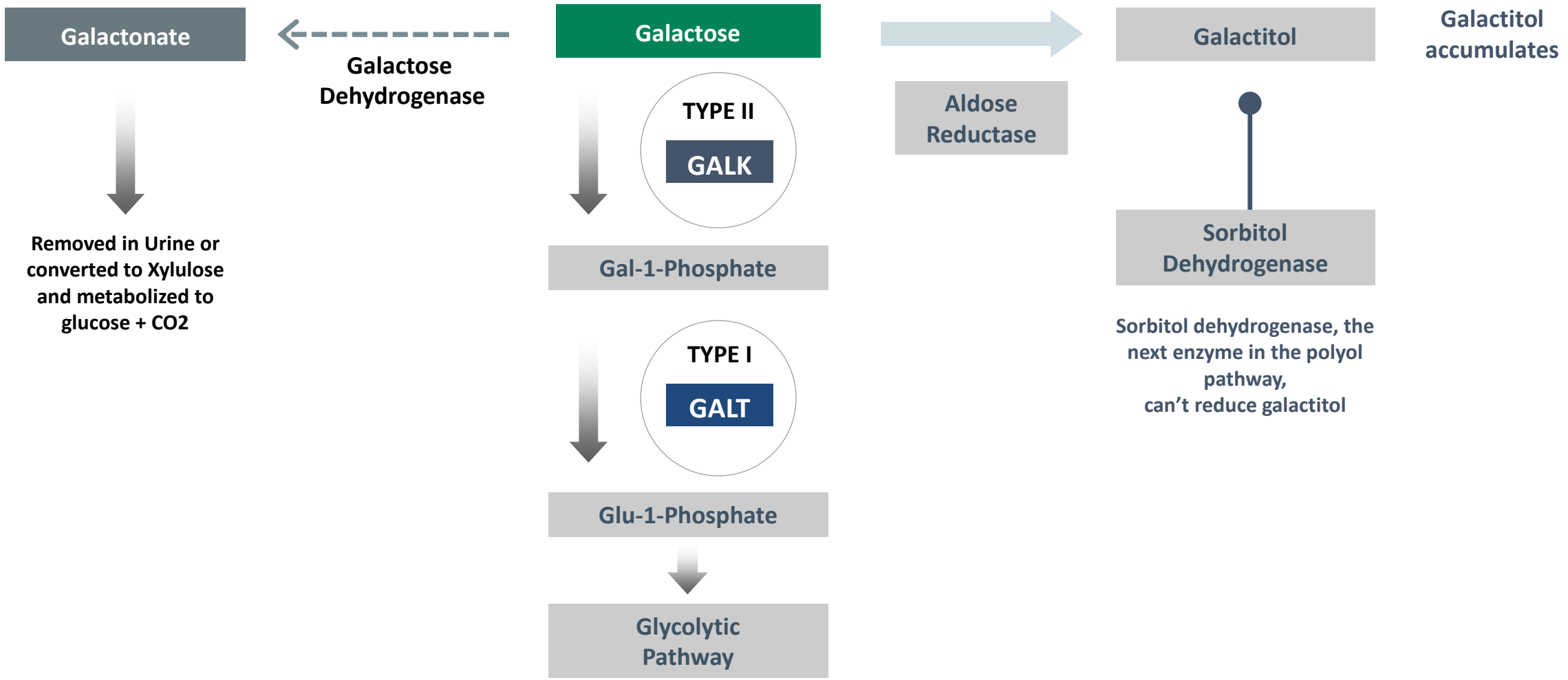
If Blocking AR Doesn't Increase Galactose or Gal-1P…..
Where Does the Extra Substrate Go?



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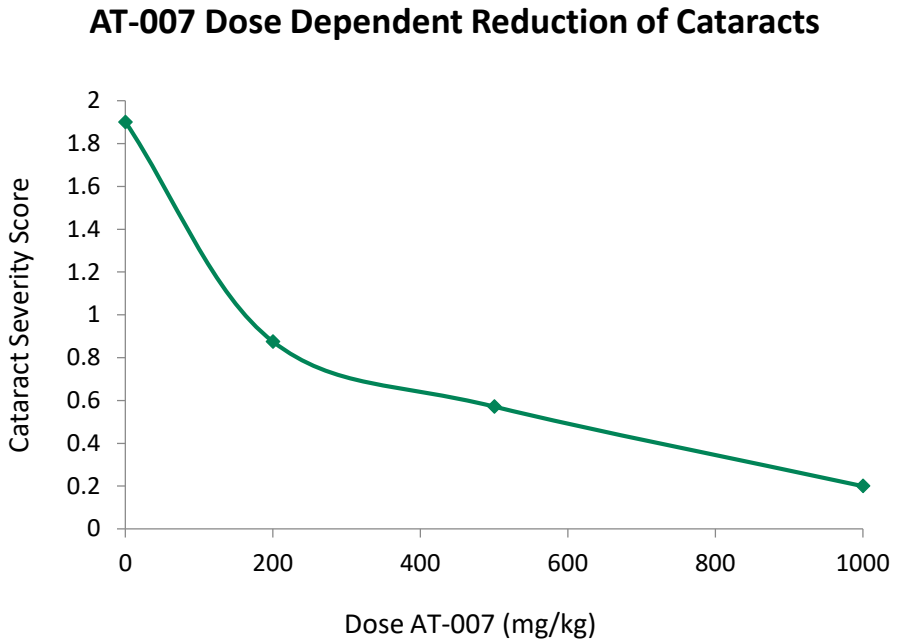
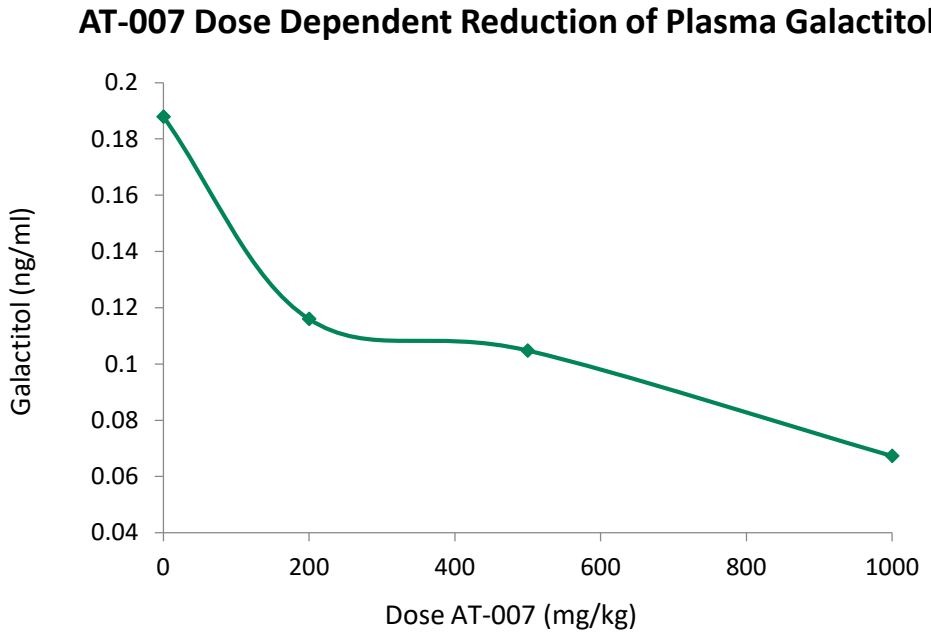
AT-007 Cataract Prevention Dose Response

Greater doses of AT-007 reduced galactitol levels and the severity of cataracts





No cataracts in WT or AT-007 treated GALT null rats, but visible cataracts in all GALT null placebo rats at Neonatal Day 22

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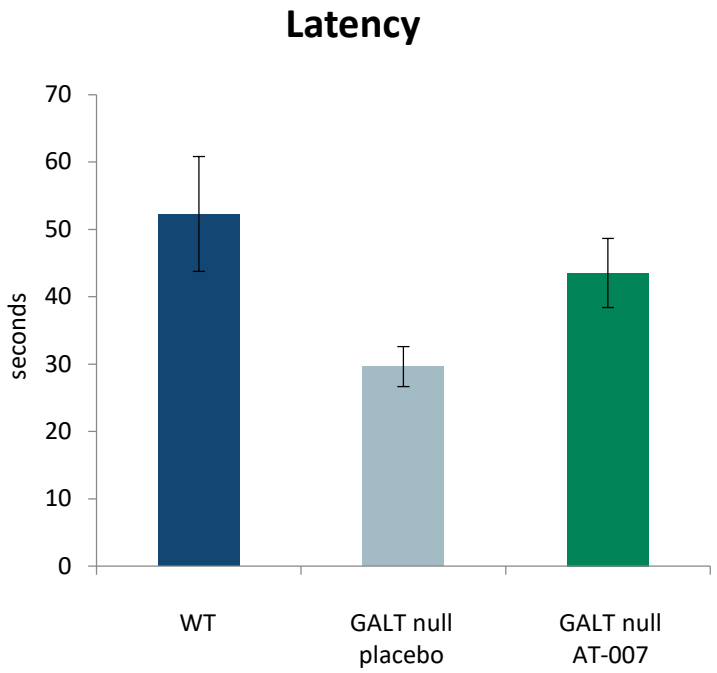
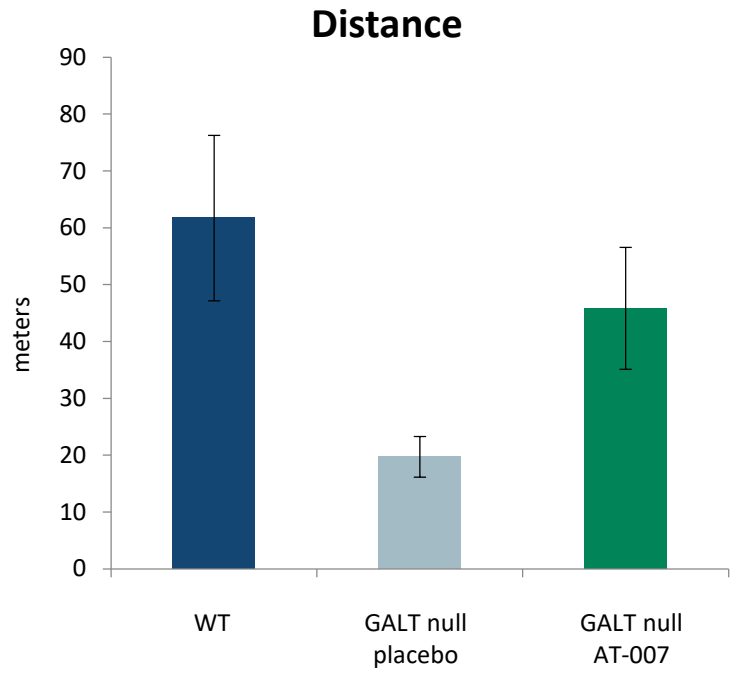
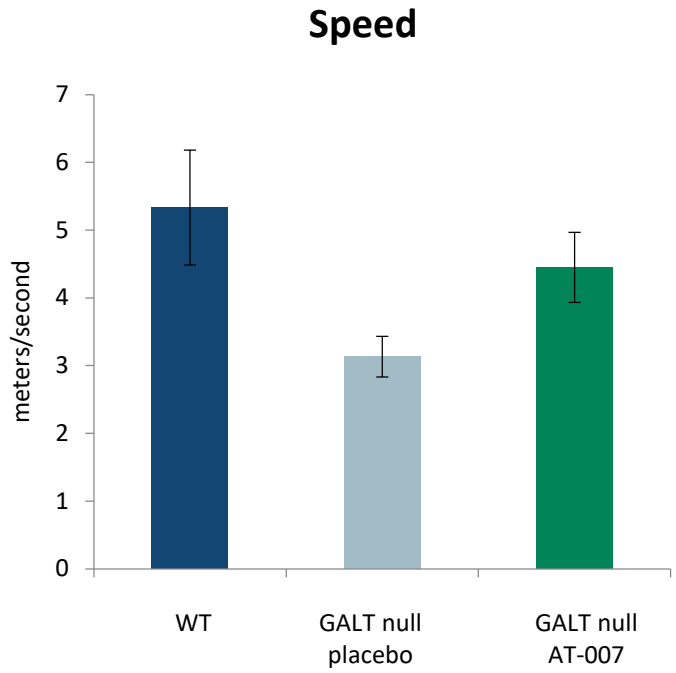
AT-007 Treatment Prevents CNS Deficits in Galactosemia Rat Model







While galactosemic rates showed deficits in learning and motor coordination versus WT rats, treatment with AT-007 was able to prevent these deficiencies and normalize cognitive and motor function

APPLIED THERAPEUTICS

AT-007 Adult Galactosemia Development Program

SAD/MAD in Healthy Volunteers

- 4 cohorts 6-8 subjects per cohort, including placebo controls (2)
- Dose escalation until MTD is reached
- MAD: 7 day treatment
- Primary endpoints: safety / tolerability & PK

SAD to MAD Transition in Adult Galactosemia Patients

- SAD to MAD adaptive design due to rare disease setting (limited patients)
- 4 cohorts 3-4 subjects per cohort
- Sentinel dose (single dose 4 days ahead) followed by 27 days consecutive dosing
- Primary endpoints: safety / tolerability & PK
- Examine galactitol levels in blood and urine as surrogate biomarker

3 Month Safety Extension

- If favorable effects are seen on galactitol biomarker at safe / tolerable doses in Part 2, a 3 month safety extension will be initiated
- Primary endpoints: safety / tolerability
- Examine biomarker outcomes

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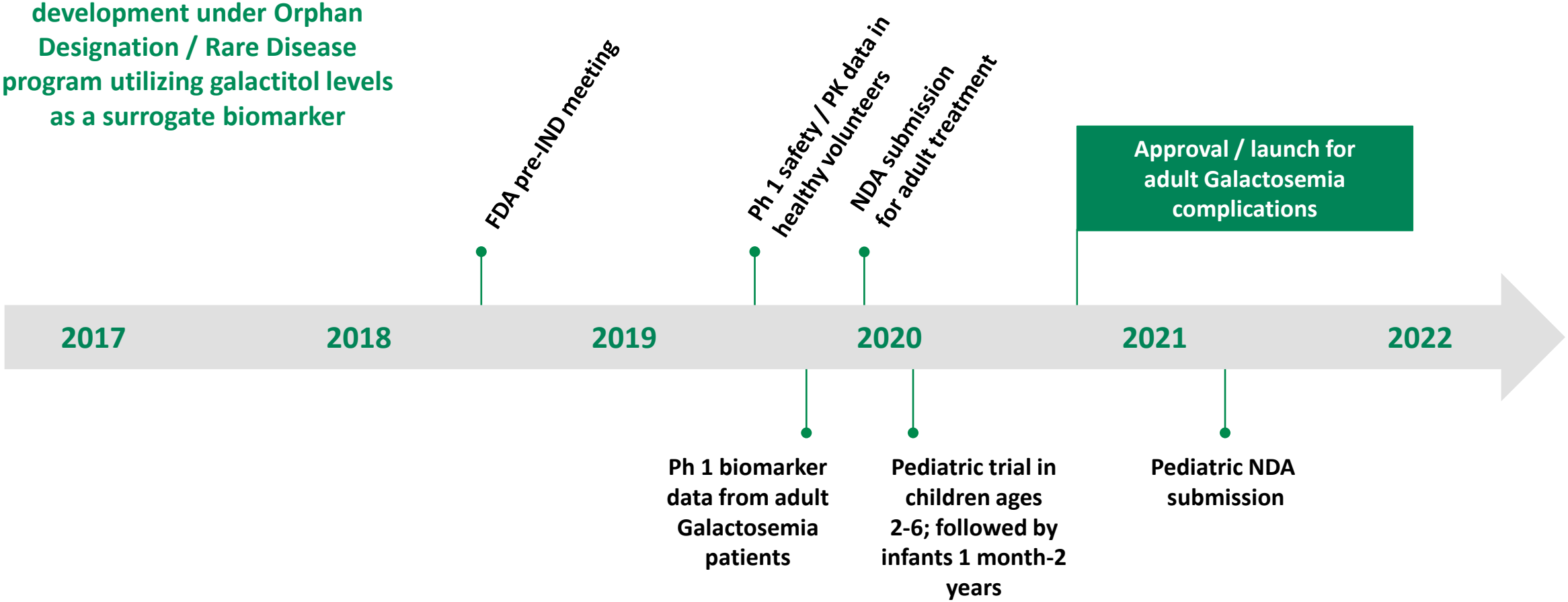
Anticipated Development Timeline: AT-007 for Galactosemia Complications

Potential abbreviated development under Orphan Designation / Rare Disease program utilizing galactitol levels as a surrogate biomarker



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Key Anticipated Milestones

Q2 2019	AT-001 End of Phase 1 FDA meeting; clarity on development path forward
Q2 2019	Start AT-007 healthy volunteer SAD/MAD portion of Phase 1 trial
Mid 2019	Start of AT-001 Diabetic Cardiomyopathy Phase 2/3 trial
Q3 2019	Readout of AT-007 Phase 1 SAD/MAD trial in adult Galactosemia patients; extension phase initiated
Q4 2019	AT-007 Galactosemia 3 month safety extension completion; initiate NDA submission
Q1 2020	IND submission in AT-003 Diabetic Retinopathy program/Phase 1 start
Q1 2020	AT-001 DbCM Phase 2/3 trial fully enrolled
Q1 2020	AT-007 Galactosemia pediatric trial start
Q1 2020	Additional pipeline programs move into Phase 1 (e.g. PI3k selective inhibitors)
Q2 2020	AT-003 Phase 1 readout
Mid 2020	Interim readout AT-001 Phase 2/3 DbCM trial

Intellectual Property Summary

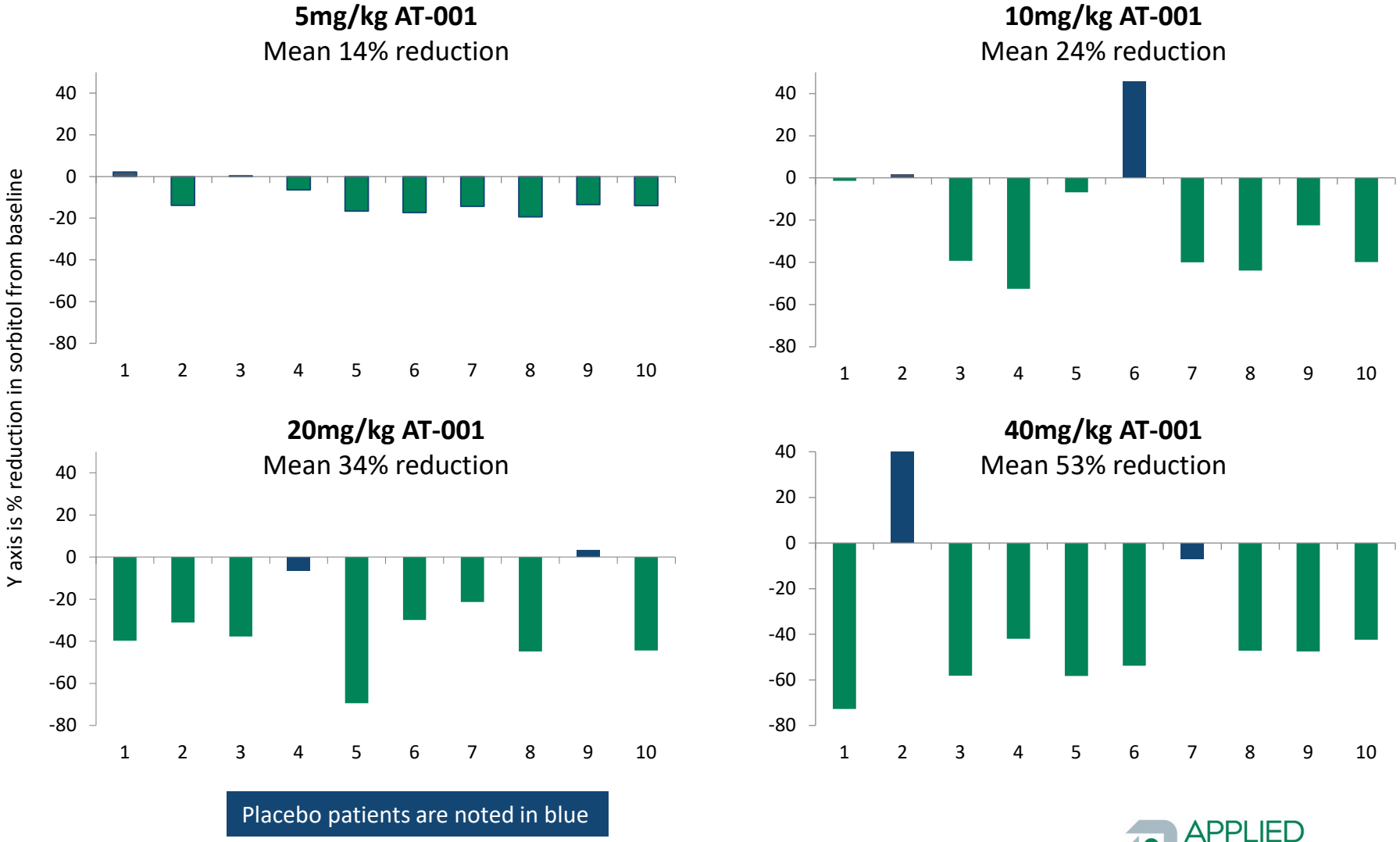
- Dominant IP and Freedom to Operate on all compounds & all target indications
- Expected IP runway of at least 10 years post-launch in key indications
- Composition of matter patents that cover AT-001 and related compounds obtained US, EP, JP, CA and AU
  - Patent protection through 2031, regulatory extension of term possible
  - Method claims obtained or currently being pursued
- Composition of matter patent that covers AT-007 and related compounds obtained in US
  - Pending on fast track in Europe, pending in other countries
- Company-owned international applications (PCT) cover methods for treating Galactosemia and additional compound derivatives

Appendix

APPLIED THERAPEUTICS

Summary PD Biomarker Data: Effects on Sorbitol

- Change in Whole Blood Sorbitol from Baseline to C_{max}
- Dose dependent effect of AT-001 on ARI activity based on sorbitol as a PD biomarker
- Magnitude and duration of sorbitol effects were stronger at higher doses



APPLIED THERAPEUTICS