VTV Therapeutics

Corporate Presentation

First-in-class molecules for organ protection in metabolic diseases

December 2019

Forward looking statements

The statements made in this presentation may include forward-looking statements regarding the type 1 diabetes, Alzheimer's disease, cystic fibrosis related diabetes, and non-alcoholic steatohepatitis markets, the development and attributes of investigational and marketed products to treat these diseases and other conditions, and the future operations, opportunities or financial performance of vTv Therapeutics Inc. These forward-looking statements are only estimations based upon the information available to vTv Therapeutics Inc. as of the date of this presentation. Except as required by law, we expressly disclaim any responsibility to publicly update or revise our forward-looking statements, whether as a result of new information, future events or otherwise. Thus, the forward-looking statements herein involve known and unknown risks and uncertainties and other important factors such that actual future operations, opportunities or financial performance may differ materially from these forward-looking statements.

For a more detailed discussion of our risks, see the Risk Factors section in our prospectus filed with the SEC and our other filings with the SEC, including our most recent 2018 Annual Report on Form 10-K.

Undue reliance should not be placed on forward-looking statements, which speak only as of the date hereof. All forward-looking statements contained herein are qualified in their entirety by the foregoing cautionary statements.

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Our Focus

We are focused on treating **metabolic diseases** to minimize their long-term **complications** through **end-organ protection**

Our innovative pipeline of **first-in-class small molecules, emphasis on clinical trial execution,** and **long-term sponsor support** are the keys to our success.

Our People

Jeff Kindler, JD
Chairman of the Board

CEO, Centrexion Therapeutics

Fmr. Chairman and CEO, Pfizer

Fmr. EVP, General Counsel for McDonald's Corporation

Fmr. Partner of William & Connelly

Steve Holcombe, B Sc
President, CEO

35 years experience growing start-up companies

18 years at vTv; founding team member

Negotiated 10 vTv partnerships

Raised $200 million equity capital

Focused on operational excellence: Assembled teams that moved projects forward on time and on budget

Carmen Valcarce, PhD
Executive Vice President, CSO

30+ years of R&D experience focused on diabetes and metabolic disease

Managed 12+ INDs

Part of the vTv IPO team

Involved in over 50 due diligence and partnership deals

Ran multiple positive clinical studies

+20 patents

7 years at Novo Nordisk

Trained biochemist and molecular biologist focused on mitochondrial metabolism

Rudy Howard, BA CPA
Executive Vice President, CFO

20+ years as CFO of 5 publicly held companies, ranging from early stage to $1B in revenues, and up to 7,000 employees

As CFO, led three companies through IPOs

Raised over $500M in public markets

Significant role in over 30 M&A transactions

Former partner with PWC

Aaron Burstein, PharmD
Senior Vice President, Clinical Development

24+ years clinical research and drug development experience across academia, federal government, large pharma and small biotech companies.

Supported 60+ clinical studies across Phases 1-4

48 peer reviewed scientific publications

Fellowship training in Clinical Neuropharmacology including PK/PD data analysis techniques

Pipeline

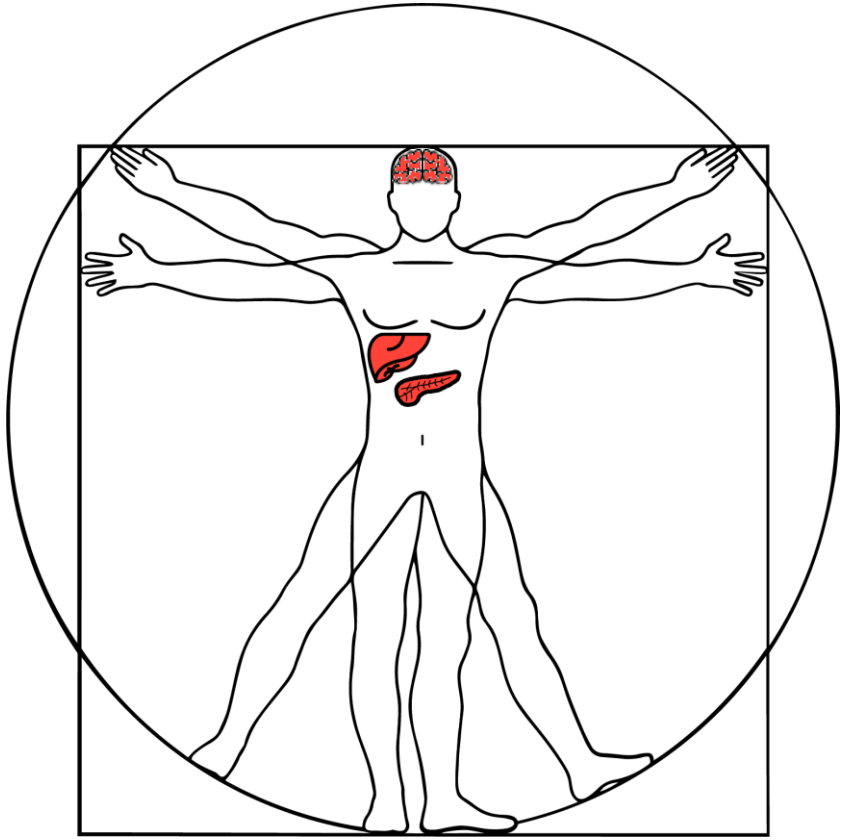
Indication	Preclinical	Phase I	Phase II	Phase III	Biological Rational
Type 1 Diabetes (T1D)	TTP399 (GKA)				Liver-selective GKA; no disruption of GK regulatory protein
Dementia with Diabetes	Azeliragon (RAGE)				Small molecule antagonist of RAGE
Cystic Fibrosis Related Diabetes (CFRD)	TTP273 (GLP1-R)				Small molecule oral GLP1-r agonist
Nonalcoholic Steatohepatitis (NASH)	HPP3033 Nrf2/Bach1				Non-electrophilic activator of Nrf2 pathway

Our Strategy

VVT Therapeutics

Addressing the Impact of Metabolic Diseases on End-organs

Our focused collection of assets are designed to provide protective solutions for the Brain, Pancreas and Liver



RAGE in Dementia with Diabetes

- Protect against cognitive decline with diabetes

GKA in Diabetes

- Improve glucose control
- Preserve beta cell function
- Reduce long term diabetic complications (i.e. kidney, heart)

Oral GLP-1r in Cystic Fibrosis Related Diabetes

- Improve glucose control
- Increase beta cell mass, proliferation and function
- Improve lung function[1]

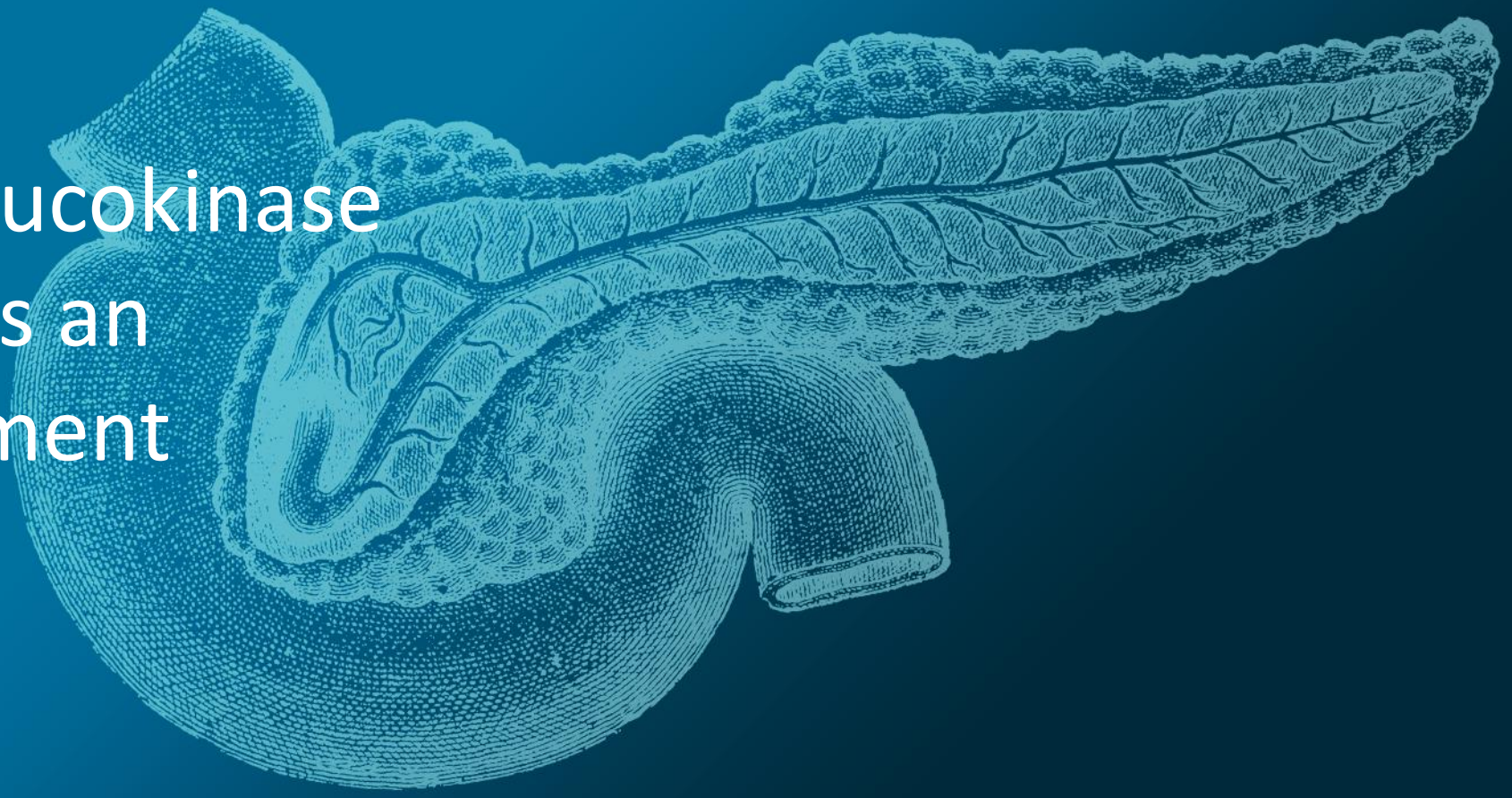
Nrf2/ Bach1 in NASH

- Prevent hepatocyte ballooning and inflammation in NASH

1. Raquel Barrio European Endocrinology (2015) 172, R131-R141

Diabetes

TTP399
Liver-Selective Glucokinase Activator (GKA) as an Adjunctive Treatment to Insulin in T1D



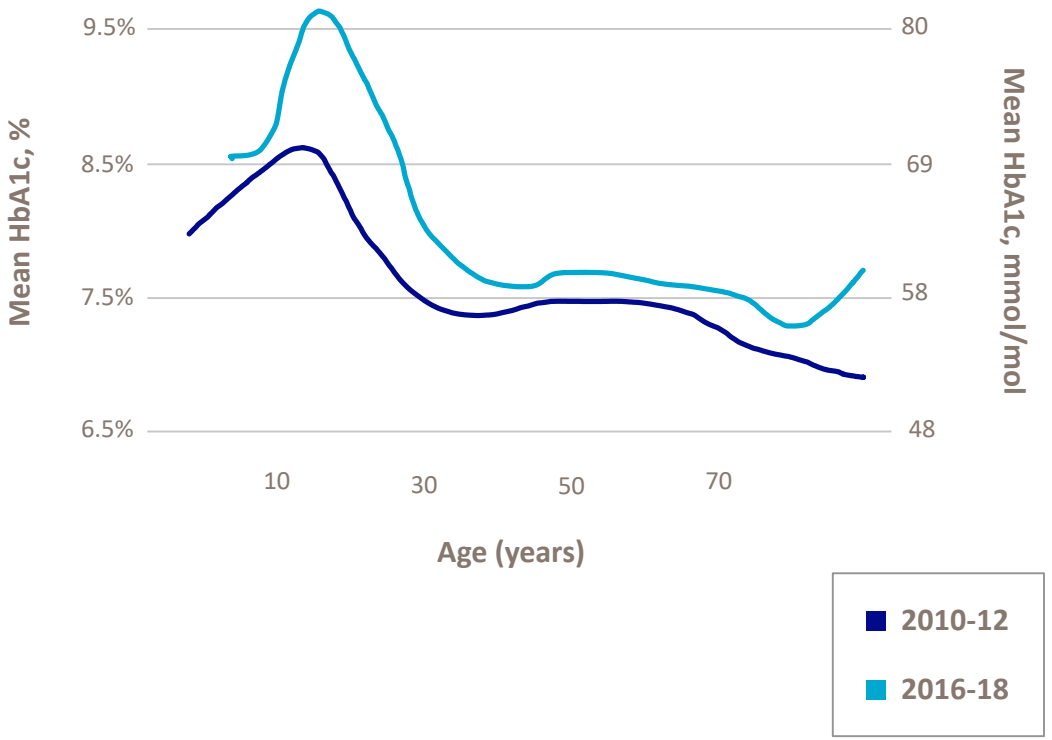
Insulin Alone is Not Enough

Nearly **80%** of people with type 1 diabetes **fail to achieve** ADA target A1c levels[1]

Despite improved and more widely adopted diabetes technology, clinical outcomes continue to decline.[2]

6% of people with type 1 diabetes reported having a **seizure** or **loss of consciousness** and 3% reported at least one event of **Diabetic Ketoacidosis ("DKA")** over the previous 3-month period.[3]

Patient Mean HbA1c Levels Throughout Time



1. Diabetes Technol Ther. 2019 Feb;21(2):66-72. doi: 10.1089/dia.2018.0384. Epub 2019 Jan 18.
2. (Foster et al. Diabetes Technology and Therapeutics (2019) 21:66-72; DOI: 10.1089/dia.2018.0384)
3. Miller KM, et al. Diabetes Care 2015;38:971–978 | DOI: 10.2337/dc15-0078

Limited Treatment Options for a Significant Patient Population

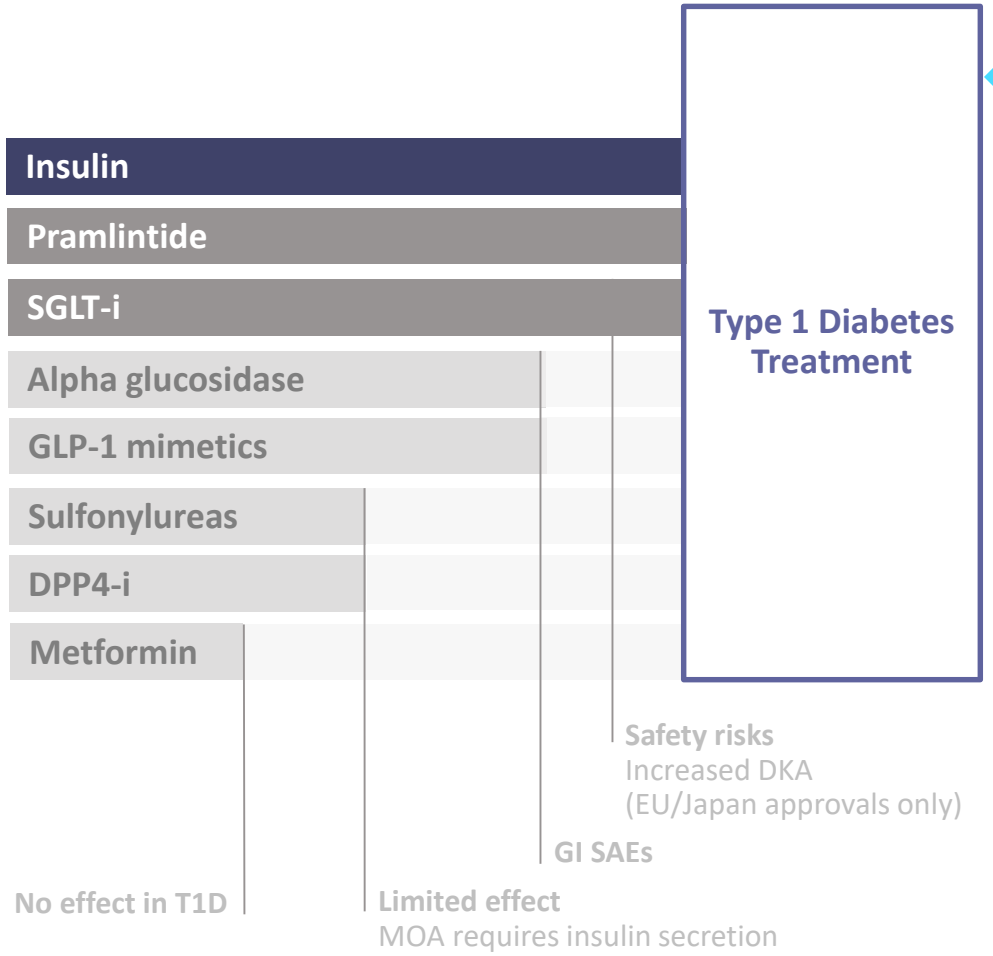
30 million people suffer from T1D globally[1]

1.5 M in the US[2]

Insulin together with Glucose monitoring **is standard of care**

Many oral adjunct treatments have been tried for type 1, including SGLTi, but all have shortcomings and **only two have been approved and only outside the US**

Available type 2 treatments have limited cross-over potential[3]



TTP399 GKA

Product attributes:

- Oral treatment
- Improve time-in-range
- Reduce insulin dose

Without:

- Hypoglycemia
- Diabetic ketoacidosis (“DKA”)
- Weight gain

1. IDF DIABETES ATLAS 8th edition 2017
2. Global Data, 2019
3. Diabetes Care 2019 Jan; 42(Supplement 1): S90-S92. https://doi.org/10.2337/dc19-S009

TTP399 a Potential Blockbuster Drug in Type 1 Diabetes

TTP399 Potentially the First Oral T1D Drug to Market in the US as an Adjunct to Insulin for Adults (>18 years)

Unmet Need

Nearly 80% of people with type 1 diabetes fail to achieve ADA target A1c levels[1]

Target Product Profile

Statistically and clinically significant, durable reduction in HbA1c (≥ 0.5%)

Improved glycemic control with lower risk of hypoglycemia than insulin alone

Improvement in one or more of the secondary outcomes:

- Increase the % time in range (70-180 mg/dL)
- Reduce % time in hyperglycemia without increasing % time in hypoglycemia
- Maintain glycemic control while reducing insulin dose (~10-20%)

Market Opportunity

Potential multi-billion dollar market for oral adjunctive treatments to insulin in T1D

(!) Diabetes Technol Ther. 2019 Feb;21(2):66-72. doi: 10.1089/dia.2018.0384. Epub 2019 Jan 18.

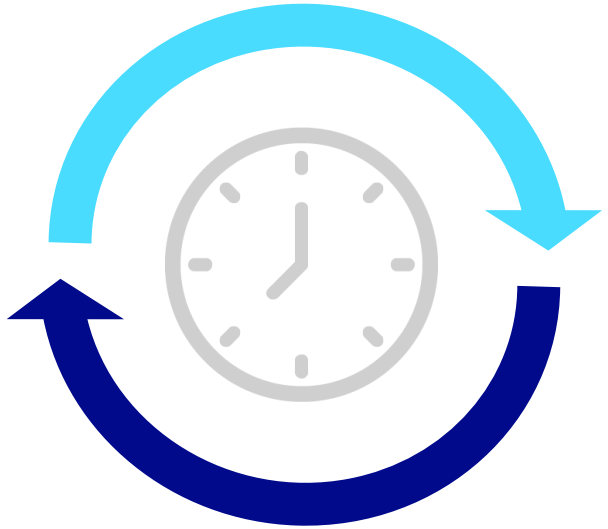
T1D is a Burdensome Disease

People with T1D never get a day off from managing it

It requires constant monitoring of blood glucose levels

People with T1D must wear a pump or use injections to dose insulin

Risk of day time hypoglycemia



Risk of night time hypoglycemia and seizures

It requires constant management, 24 hours a day

Must count the carbs and account for everything they eat

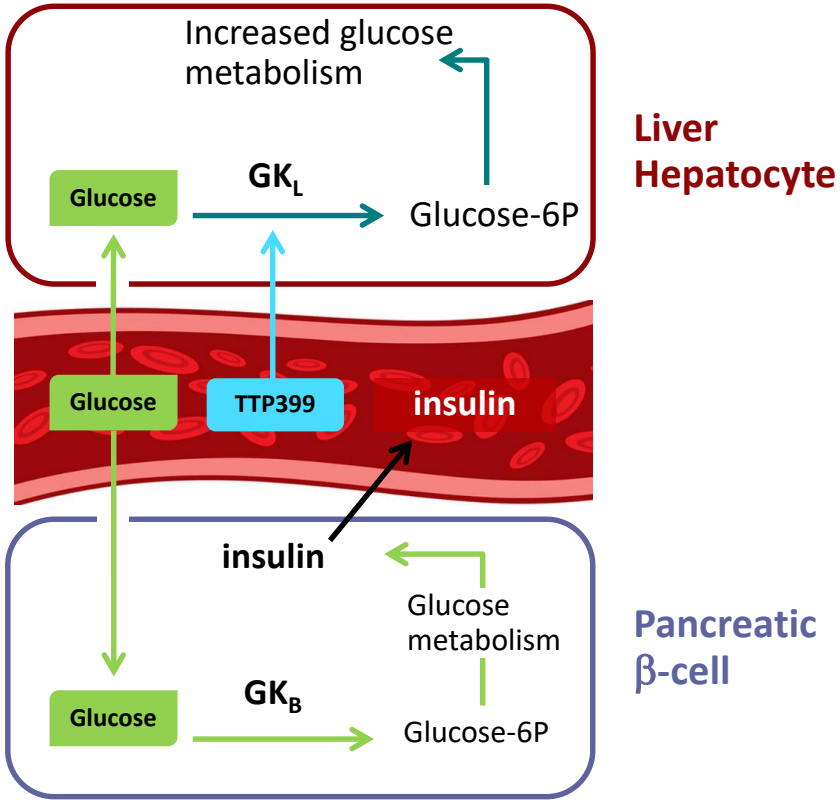
It is exhausting and has long-term dangerous complications

GKA, a Unique Biological Strategy to Support T1D Patients

Glucokinase is a critical step in sugar metabolism

Glucokinase is the glucose sensor of the body

Key role in glucose homeostasis supported by strong genetic evidence



TTP399: A liver selective Glucokinase Activator[1]

TTP399 activates GK in the liver

TTP399 does not activate GK in the pancreas and preserves beta cell function

TTP399 does not interrupt the interaction between GK and its regulatory protein

1 Vella A, Freeman J, Dunn I, Keller K, Buse J, Valcarce C. Targeting hepatic glucokinase to treat diabetes with TTP399, a hepatoselective glucokinase activator. Science Translational Medicine 16 Jan 2019

Simplici-T_1 — Adaptive Phase 1b/2 Study Trial Design

2018 | 2019 | 2020

MARCH 2018

Phase 1

(Sentinel)

TTP399 was well tolerated

JUNE 2019

Phase 2-Part 1

(Learning Phase) - 12wk, 19 patients

Positive Results Reported June 2019

Q1 2020

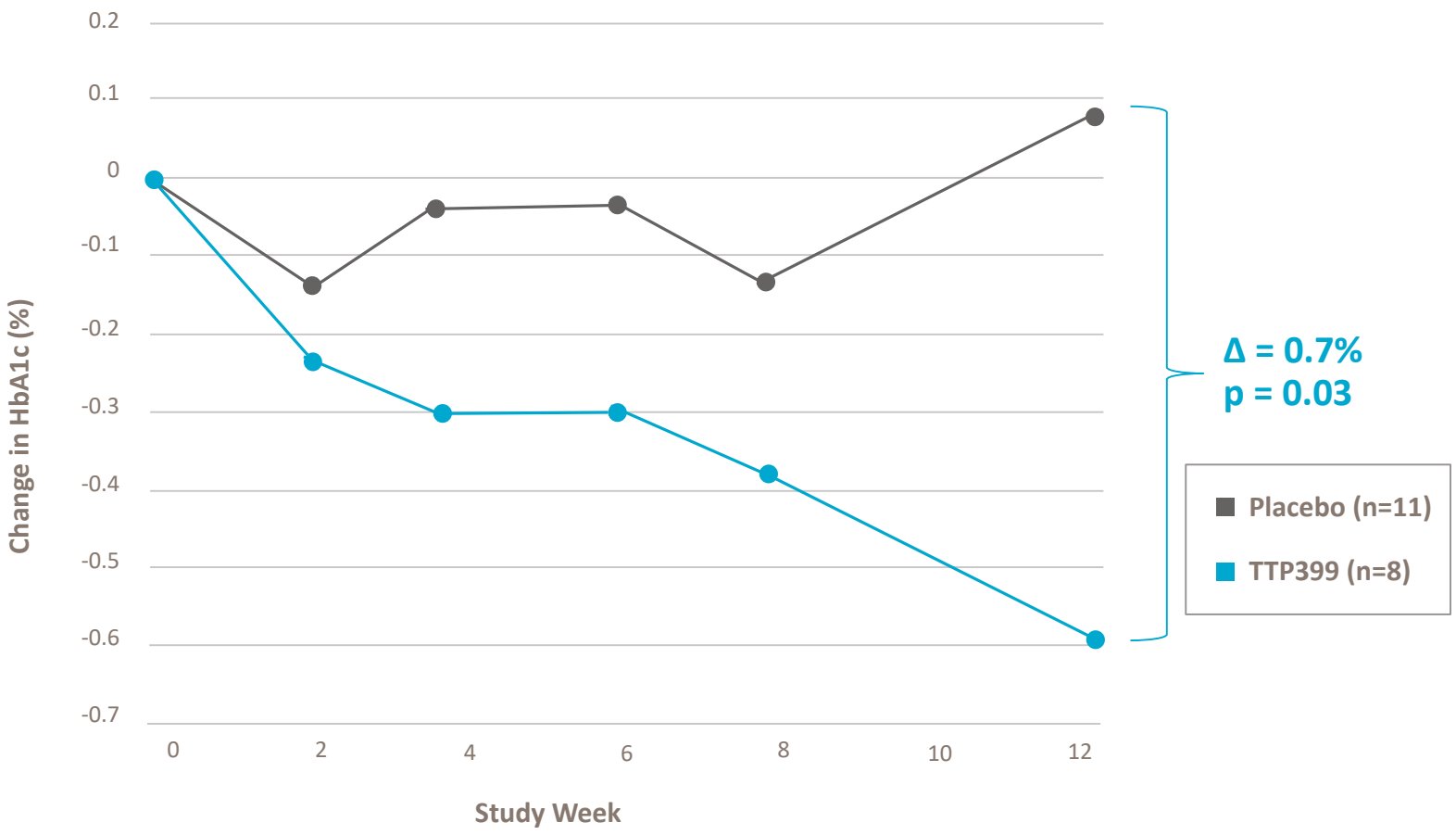
Phase 2-Part 2

(Confirming Phase) – 12wk, 85 patients

Results expected in Q1 2020

Reduced HbA1c Without Increases in Ketones or Hypoglycemia

Statistically Significant HbA1C Reduction

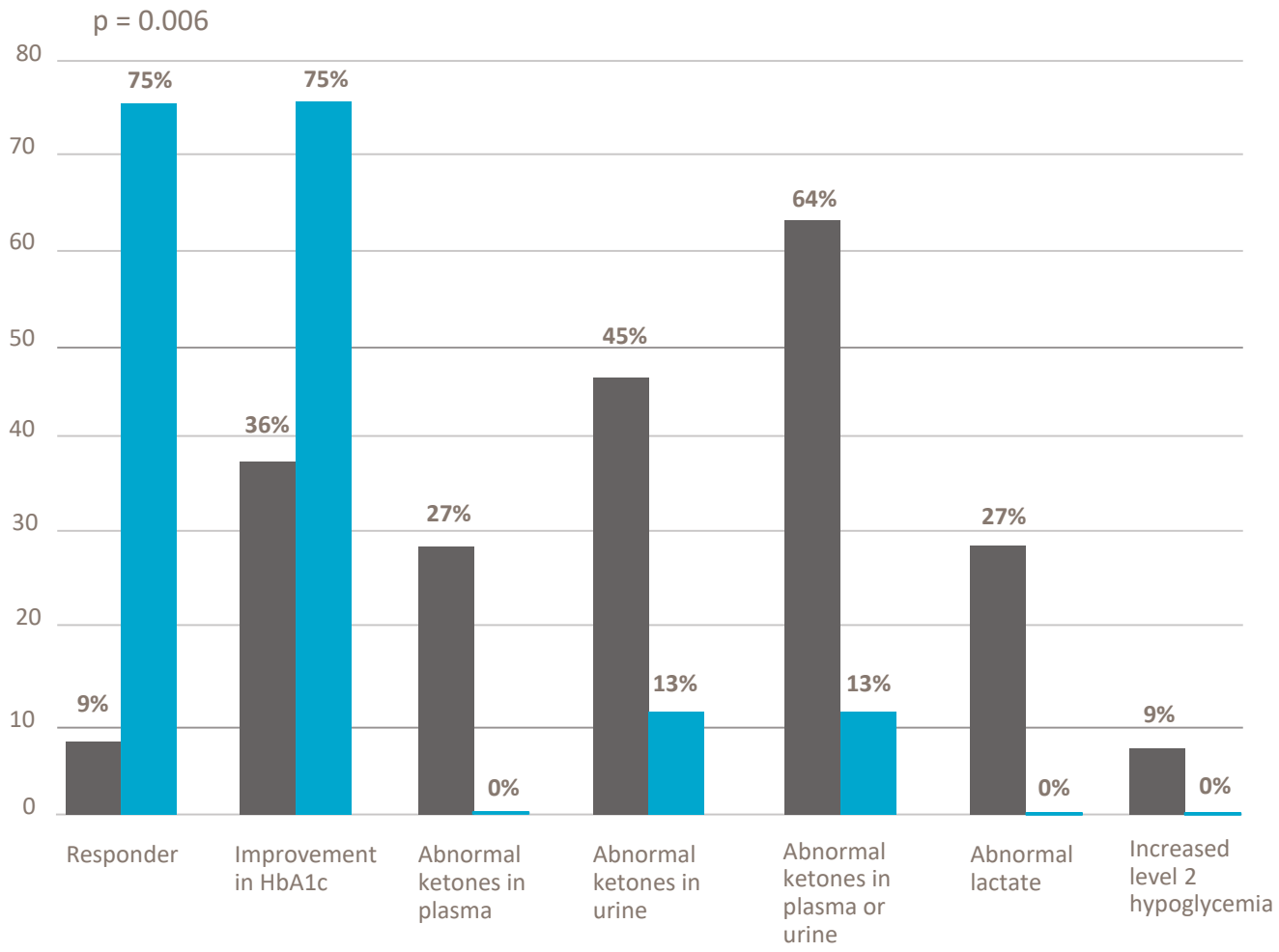


Safety:

- No SAEs
- **No reported hypoglycemia**
- **No Diabetic Ketoacidosis (DKA)**
- Similar profiles for reported TEAEs between TTP399 and placebo

Reduced HbA1c Without Increases in Ketones or Hypoglycemia

Responder Analysis and Individual Criteria



Placebo (n=11)

TTP399 (n=8)

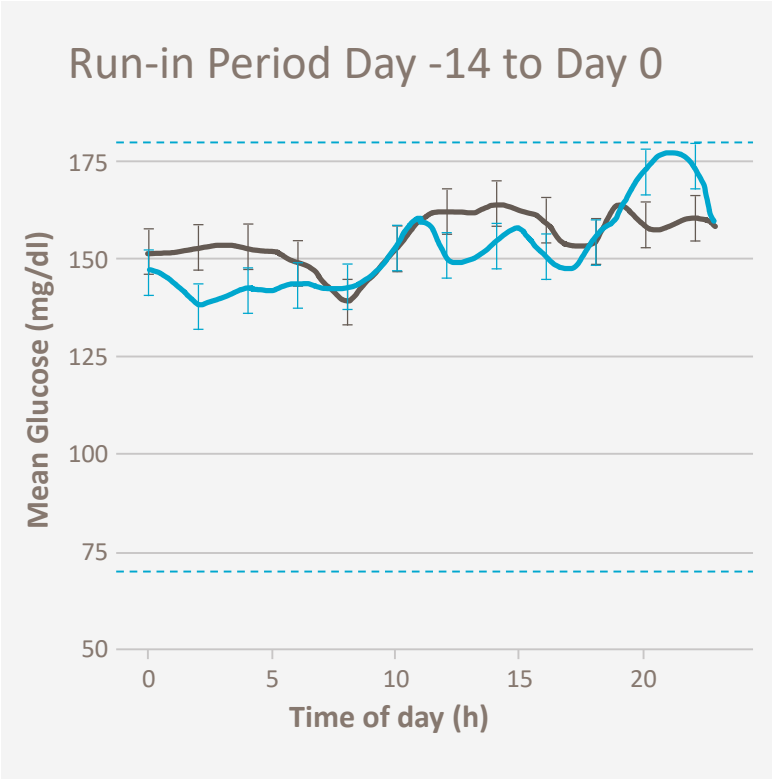
Responder definition:

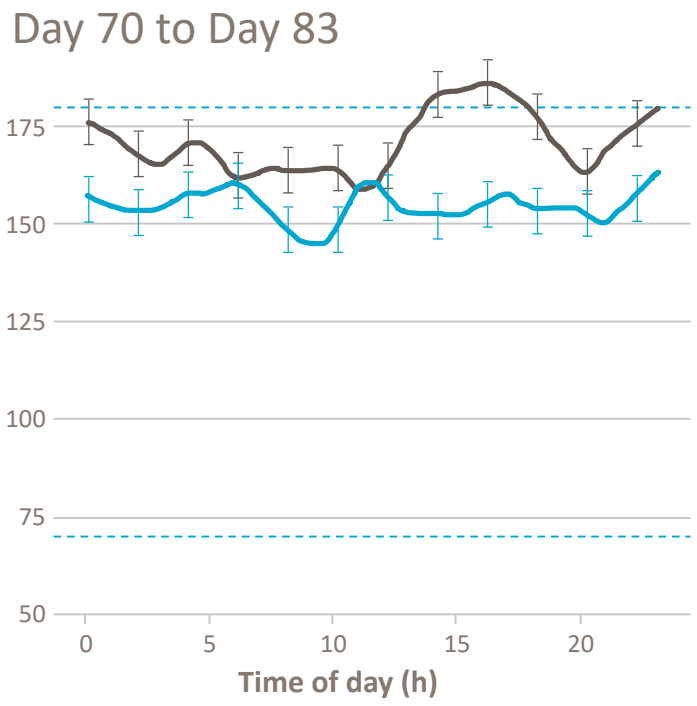
Proportions of subjects with improvement in HbA1c without the following predefined risks:

- Abnormal ketones in urine or plasma
- Abnormal lactate in plasma
- Increase in time in level 2 hypoglycemia (glucose <54 mg/dl)

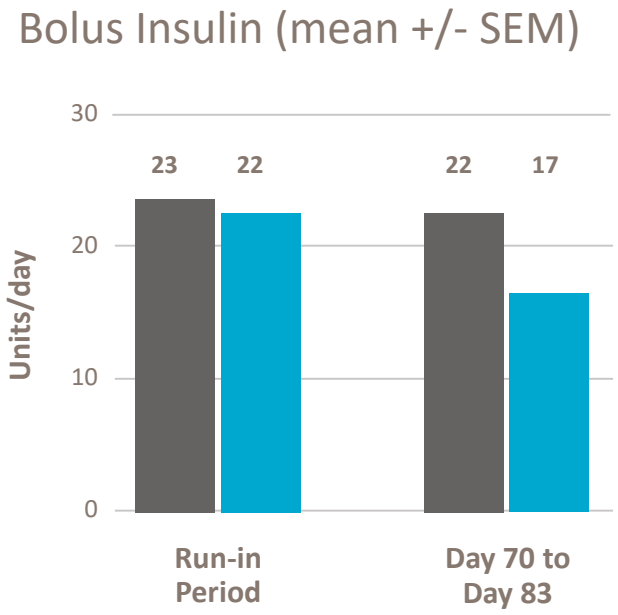
Better Glycemic Control was Achieved with Less Bolus “mealtime” Insulin

Treatment with TTP399 improved average daily glucose (ADG)





Reduction in Daily Bolus Insulin

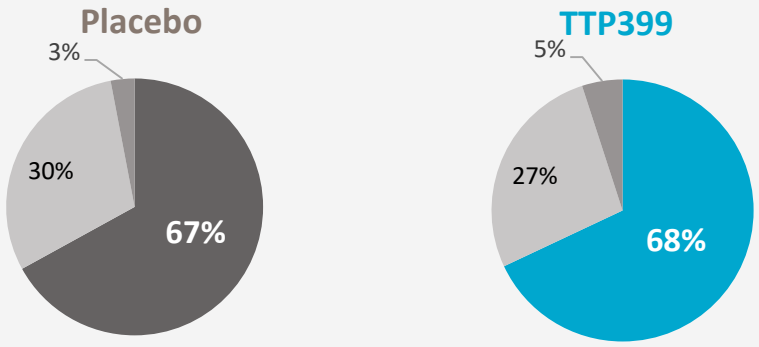


No change in Basal Insulin

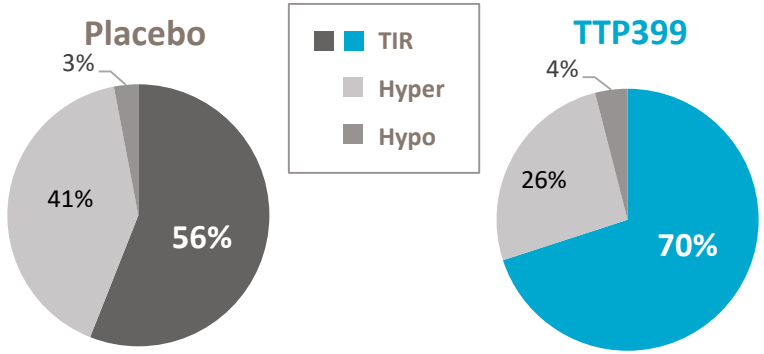
Increased Time-in-Range Without Increasing Time in Hypoglycemia

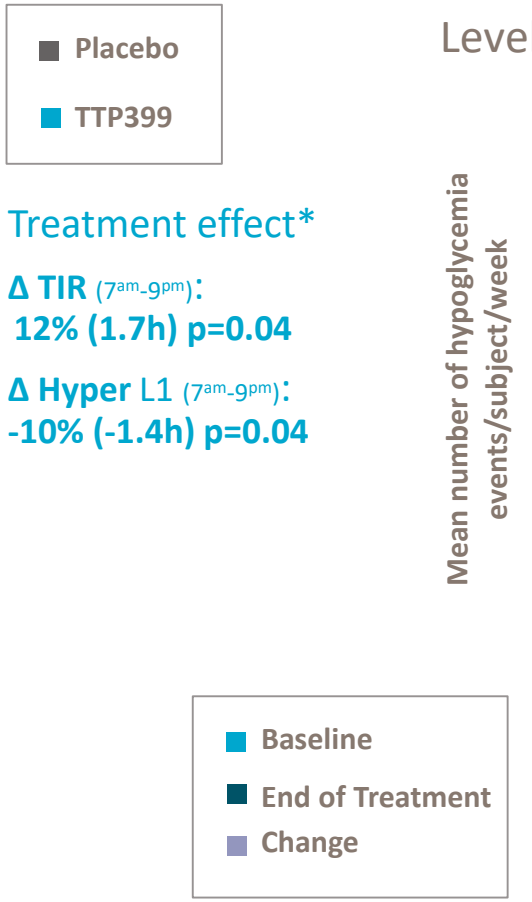
Statistically Significant Improvement in Time-in-Range (TIR) without Increasing Time in Hypoglycemia

Run-in Period Day -14 to Day 0 (7^{am}-9^{pm})



Day 70 to Day 83 (7^{am}-9^{pm})



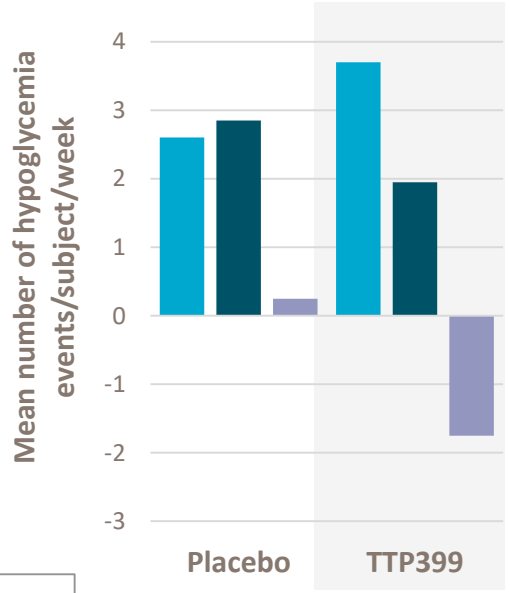


Treatment effect*

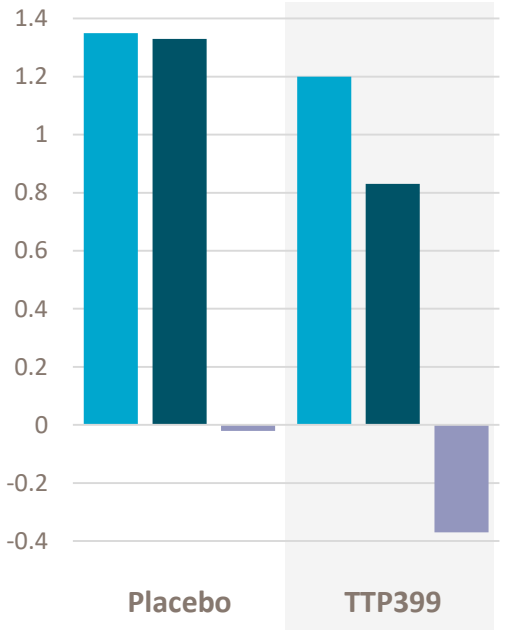
Δ TIR (7^{am}-9^{pm}): 12% (1.7h) p=0.04

Δ Hyper L1 (7^{am}-9^{pm}): -10% (-1.4h) p=0.04

Level 1 Hypoglycemia



Level 2 Hypoglycemia



No severe hypoglycemic events in either group during the study

Development Plan *

Q1 2020
Simplici-T1 study to be completed

Q2 2020
End of Ph2 meeting

YE 2020
Registration trial/trials (24wk + 24wk OLE) to be initiated

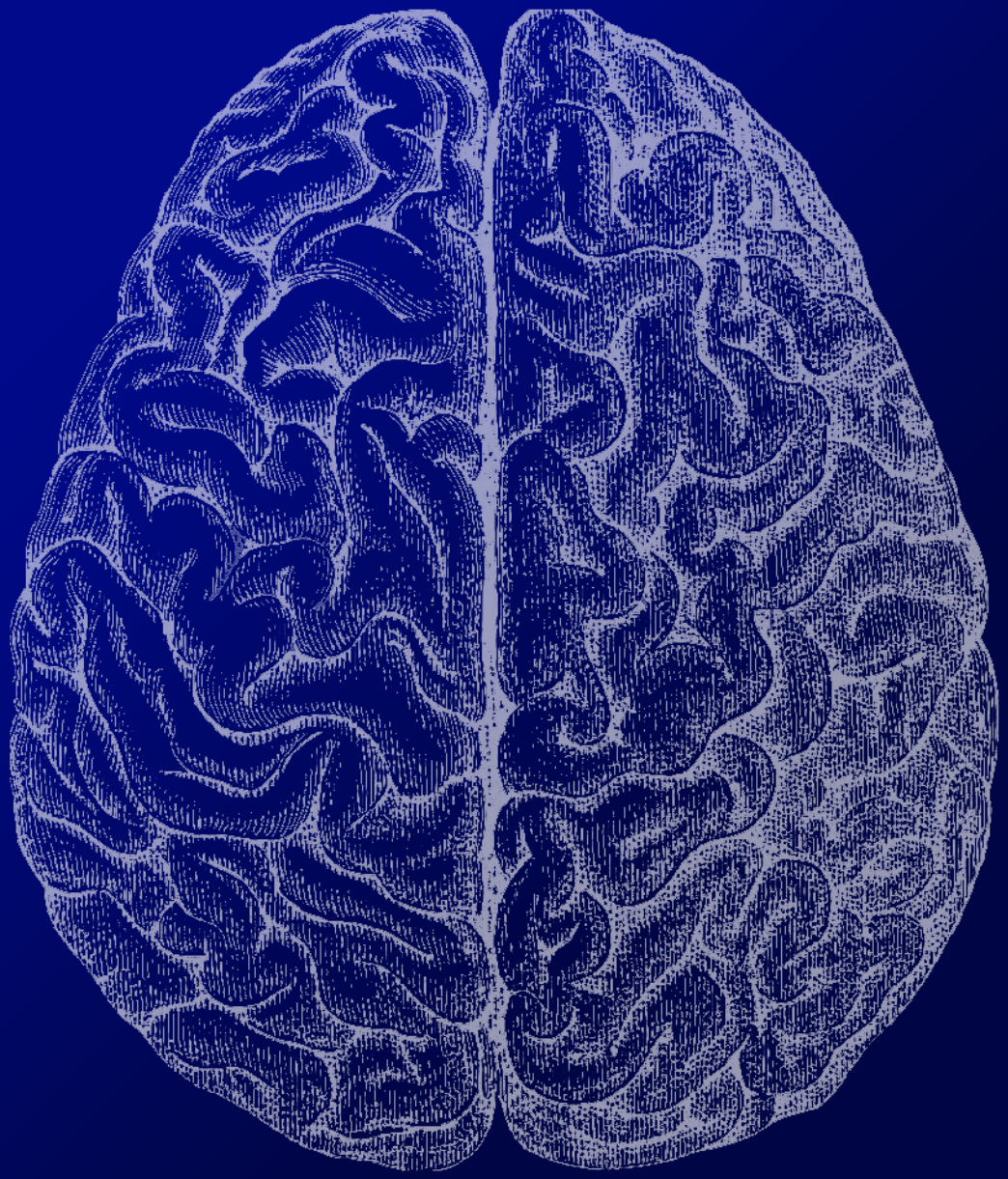
YE 2020
Activities to support NDA package to be initiated

2023
Registration trial/trials to read out

* Development plan may change based on discussions with regulatory authorities

Dementia

Azeliragon
RAGE antagonist
for Dementia with
Diabetes



The Significance of Dementia with Diabetes

Dementia[1,2]

In 2015

50 M people worldwide with Dementia

Expected to rise to **82M by 2030**

And further to **150M by 2050**

Diabetes[3]

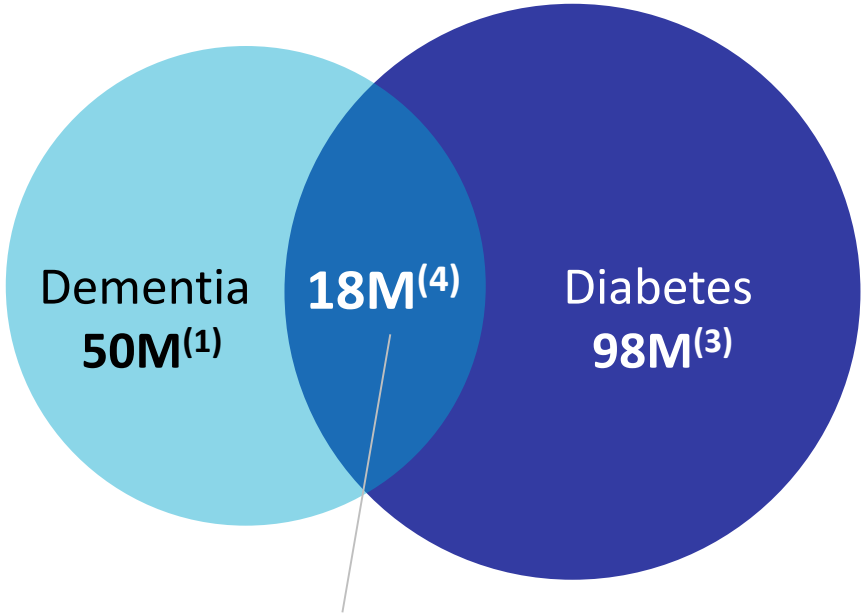
In 2017

425 million people worldwide have diabetes

- **98 million** of these ≥ 65 years of age

Prevalence of diabetes expected to rise to **629 million by 2045**

Dementia with Diabetes

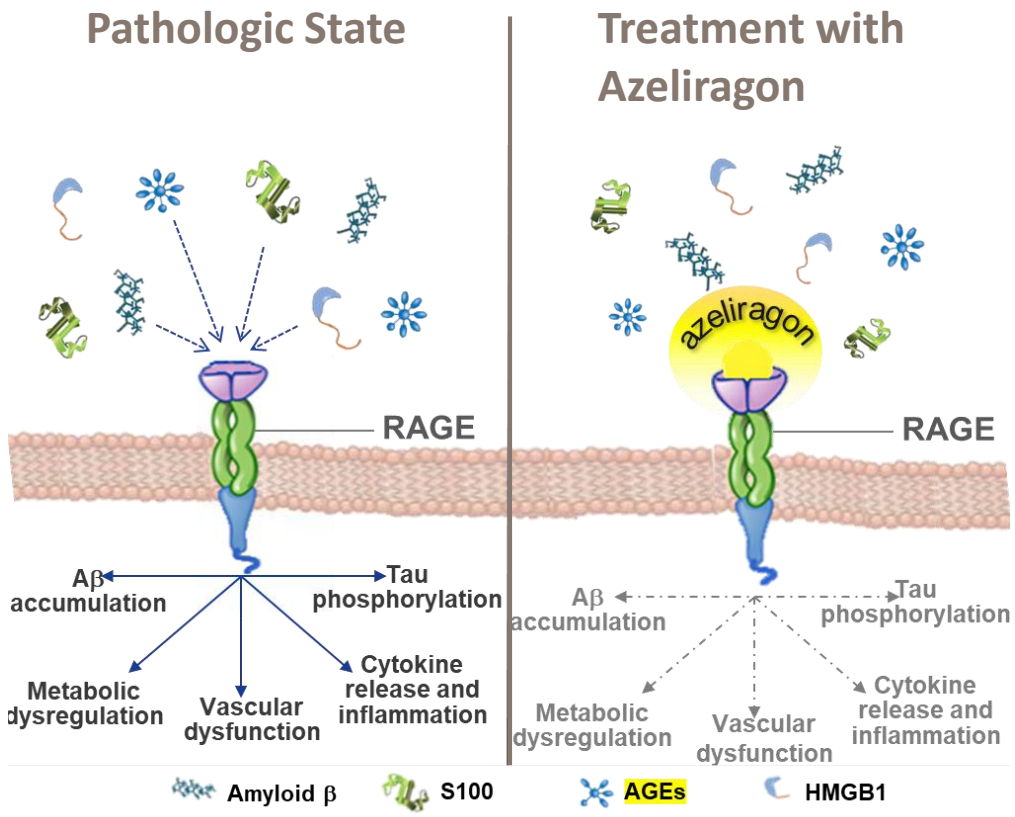


Studies have reported a correlation between T2D and dementia[5]

1. *Globally, number of people with dementia in 2017 (source: Dementia, key facts, WHO, 2017)*
2. *2019 Alzheimer's Disease Facts and Figures (source: Alzheimer's Association)*
3. *Globally, number of people with diabetes 65-79 years of age (source: IDF Diabetes Atlas 8th Edition)*
4. *Estimate based on 37%* of Medicare beneficiaries in United States age 65 and older with dementia who also have diabetes *(Alzheimer's Association. 2018 Alzheimer's Disease Facts and Figures)*
5. *Type 2 Diabetes and Dementia (2018) https://doi.org/10.1016/B978-0-12-809454-9.00001-9*

Targeting RAGE for Treatment of Dementia with Diabetes

Azeliragon antagonizes the Receptor for Advanced Glycation Endproducts (RAGE), blocking ligands from binding to the receptor and blunting resultant downstream pathologic events



Well established associations between AGEs / RAGE and diabetic complications

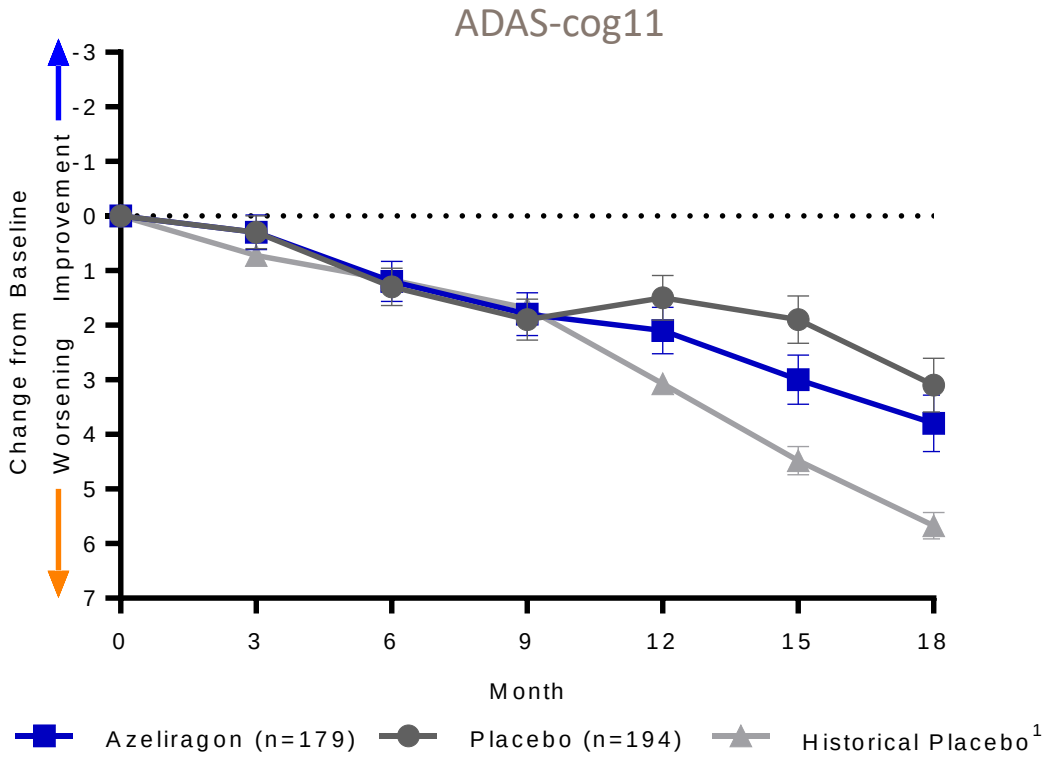
- Advanced glycation endproduct (AGE) accumulation is increased in patients with diabetes and parallels the development of cognitive impairment and dementia
- Increases in AGEs:
  - promote increased expression of RAGE
  - are linked to development of end-organ complications such as retinopathy, neuropathy and nephropathy

Potential benefits of RAGE antagonism for dementia in diabetes

- Blockade of, and reduction in, microglia activation
- Less brain atrophy
- Less dysregulation of brain glucose metabolism
- Reduction in inflammation
- Preservation of cognition and functional activities

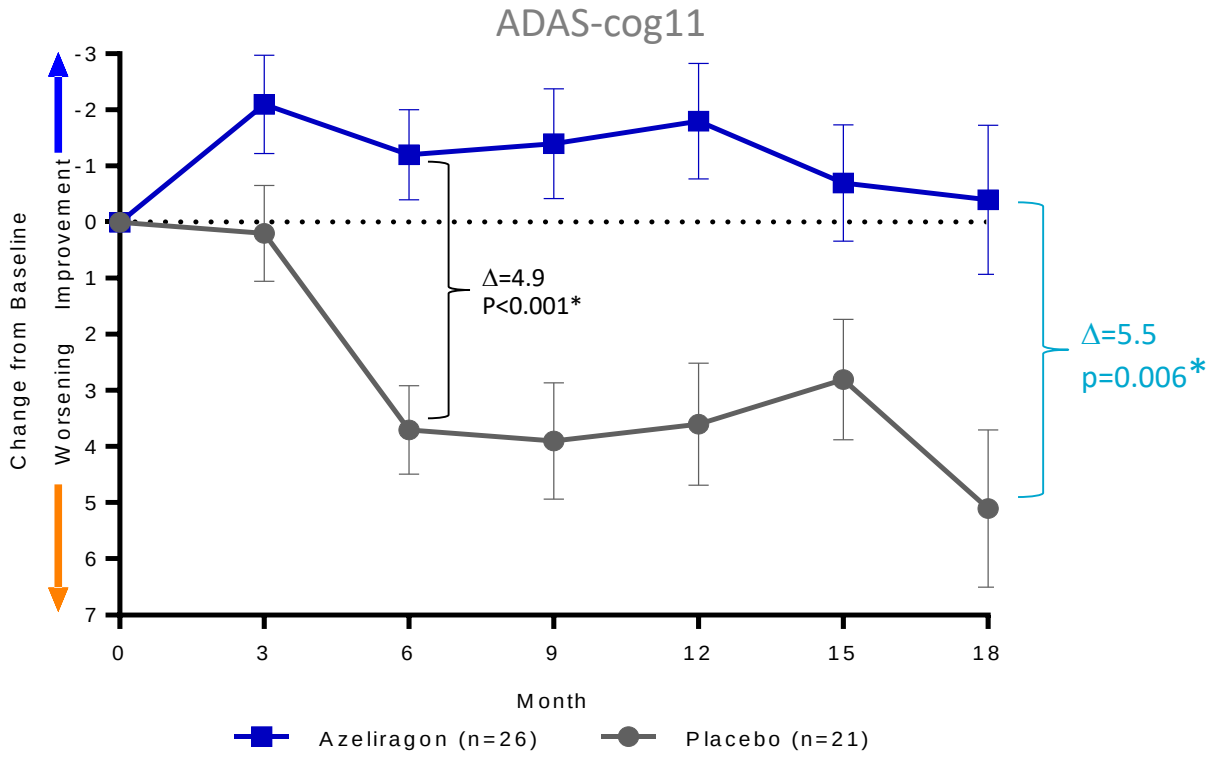
Potential Beneficial Effect on Cognition in Patients with Elevated HbA1c

STEADFAST A-Study All Subjects (FAS)



Placebo decline in A-Study FAS markedly less than expected from historical controls. No treatment differences noted.

STEADFAST A-Study Type 2 Diabetes Subgroup (FAS)



Cognitive benefits in ADA-T2D (HbA1c ≥s 6.5%) subgroup are nominally significant and clinically relevant as early as 6 months and through 18 months

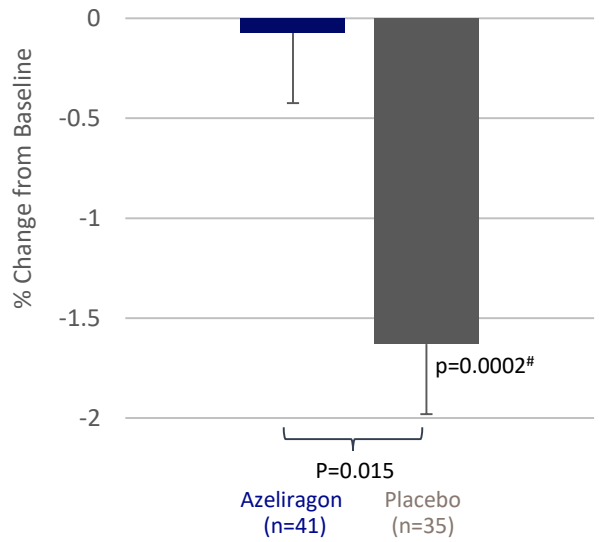
Type 2 Diabetes: Patients with diabetes (HbA1c ≥ 6.5% at anytime during the study)
Results are LSMeans ± SE based on MMRM model.
*All p values are nominal. FAS =Full Analysis Set

Data presented on March 30, 2019 at the 14th International Conference on Alzheimer's & Parkinson's Diseases held in Lisbon, Portugal
[1]*Thomas et al. Alzheimer's & Dementia 2016:12;598-603.*

Brain MRI, FDG-PET and Plasma Inflammatory Biomarker Results Support Biological Effect in Dementia with Diabetes

Less Brain Atrophy

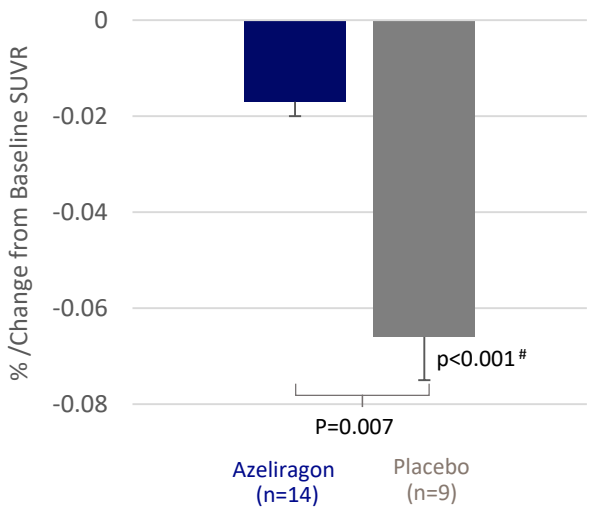
Whole Brain



Change in ventricular enlargement (%) and total hippocampal volume (%) also trending to favor azeliragon

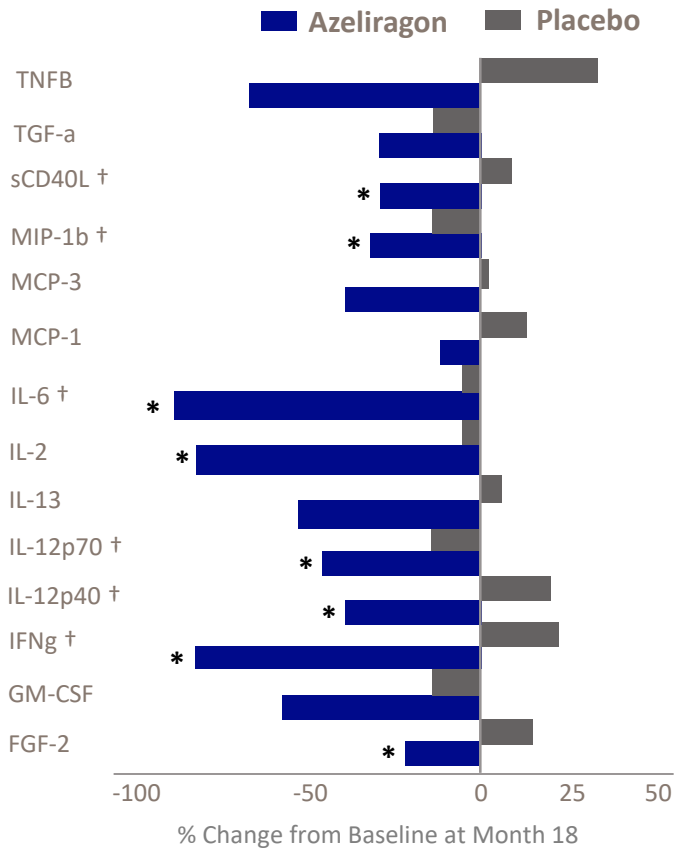
Less Reduction in Brain Glucose Utilization*

Month 18



Effect also evident at Month 12

Reduction in Plasma Inflammatory Biomarkers



* Nominal p<0.05 Wilcoxon test
† Biomarkers related to RAGE
Results are Means

*FDG-PET SUVR
Presented at 14th International Conference on Alzheimer's & Parkinson's Diseases. March 30, 2019. Lisbon, Portugal

Elevage Study: Two Studies Conducted Under a Single Protocol

2021 | 2022

STARTED JUNE 2019

Phase 2

(Part 1 / Proof of Concept) – 6 months, 100 patients

Proof of concept study to confirm the cognitive benefits evidenced in the diabetes subgroup of the STEADFAST study

Readout expected 1H 2021

Q1 2020

Phase 3

(Design pending)

Demonstrate safety and efficacy with co-primary endpoints of cognition and function to support possible registration

Study start expected 2H 2021

elevage

ClinicalTrials.gov Identifier: NCT03980730

Development Plan

NDA enabling studies are **complete** positioning azeliragon for an NDA submission upon successful, positive clinical trials

1H 2021
Complete Elevage Part 1

Seek FDA guidance Prior to Initiating Part 2 with Registration Intent

2H 2021
Initiate Part 2 of the Elevage Study

Diabetes

TTP273

Oral GLP1-R Agonist for Cystic Fibrosis Related Diabetes (CFRD)

Cystic Fibrosis Related Diabetes

~30,000 patients US; **~70,000** patients worldwide suffer from Cystic Fibrosis (CF)

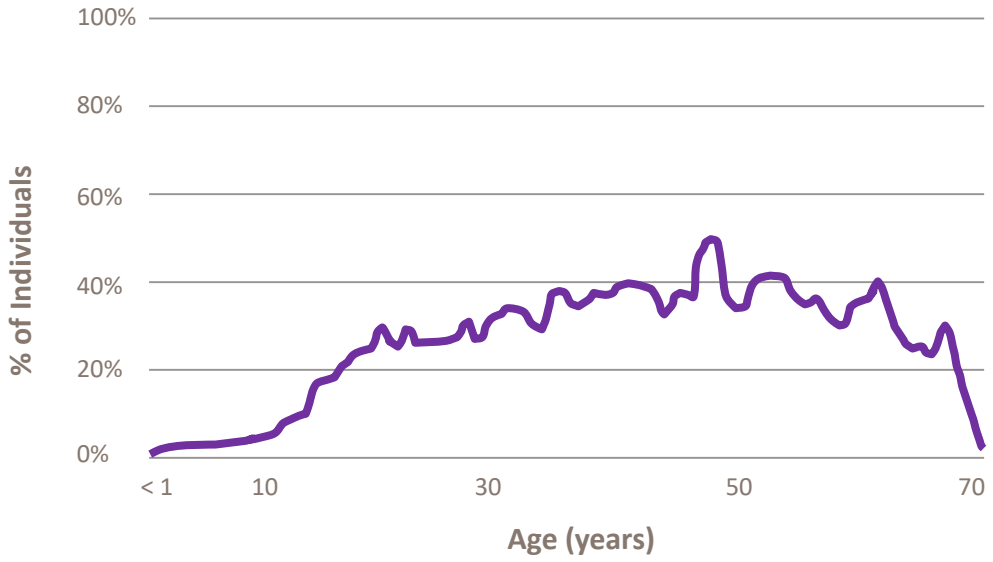
Cystic Fibrosis Related Diabetes (CFRD) is the most common CF co-morbidity[2]:

- **~40%** of adults
- **~20%** of teens

CFRD is associated with:

- Weight loss
- Lung function decline
- Increased mortality[1]

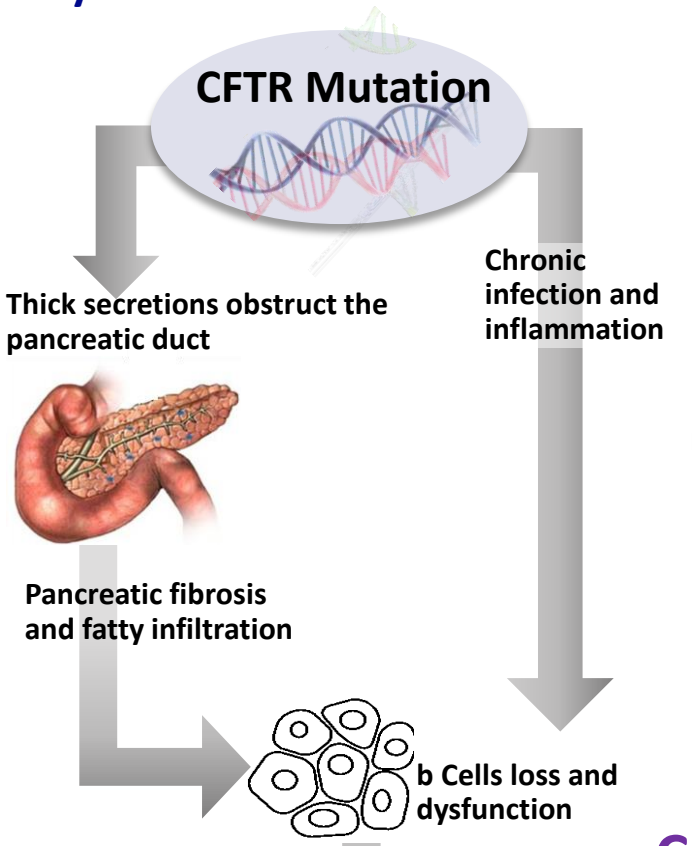
Cystic Fibrosis Related Diabetes (CFRD)



1. 2017 Cystic Fibrosis Foundation Patient Registry Highlights
2. https://www.ncbi.nlm.nih.gov/pubmed/20202149

GLP-1 Therapy Proposed as Treatment for CF and CFRD

Cystic Fibrosis Related Diabetes



Cystic Fibrosis Related Diabetes

GLP-1 and Cystic Fibrosis Related Diabetes

Increases:
Pulmonary protection
Insulin Secretion
b-cell neogenesis
b-cell proliferation

Decreases:
b-cell glucose sensitivity
Hepatic glucose production

TTP273 Addressing Unmet Need in CFRD

Benefits of TTP273:

Small molecule, oral GLP-1r agonist

Does not cause nausea and vomiting

Weight loss only in overweight patients

Potential for combination with other oral therapies

Limitations of Marketed GLP-1 Therapies:

Peptides dosed by injection

Nausea and vomiting side effects

Cause weight loss

Development Plan

Q4 2019
Seek orphan designation

Q1 2020
Apply for Cystic Fibrosis Foundation Clinical Research financial support

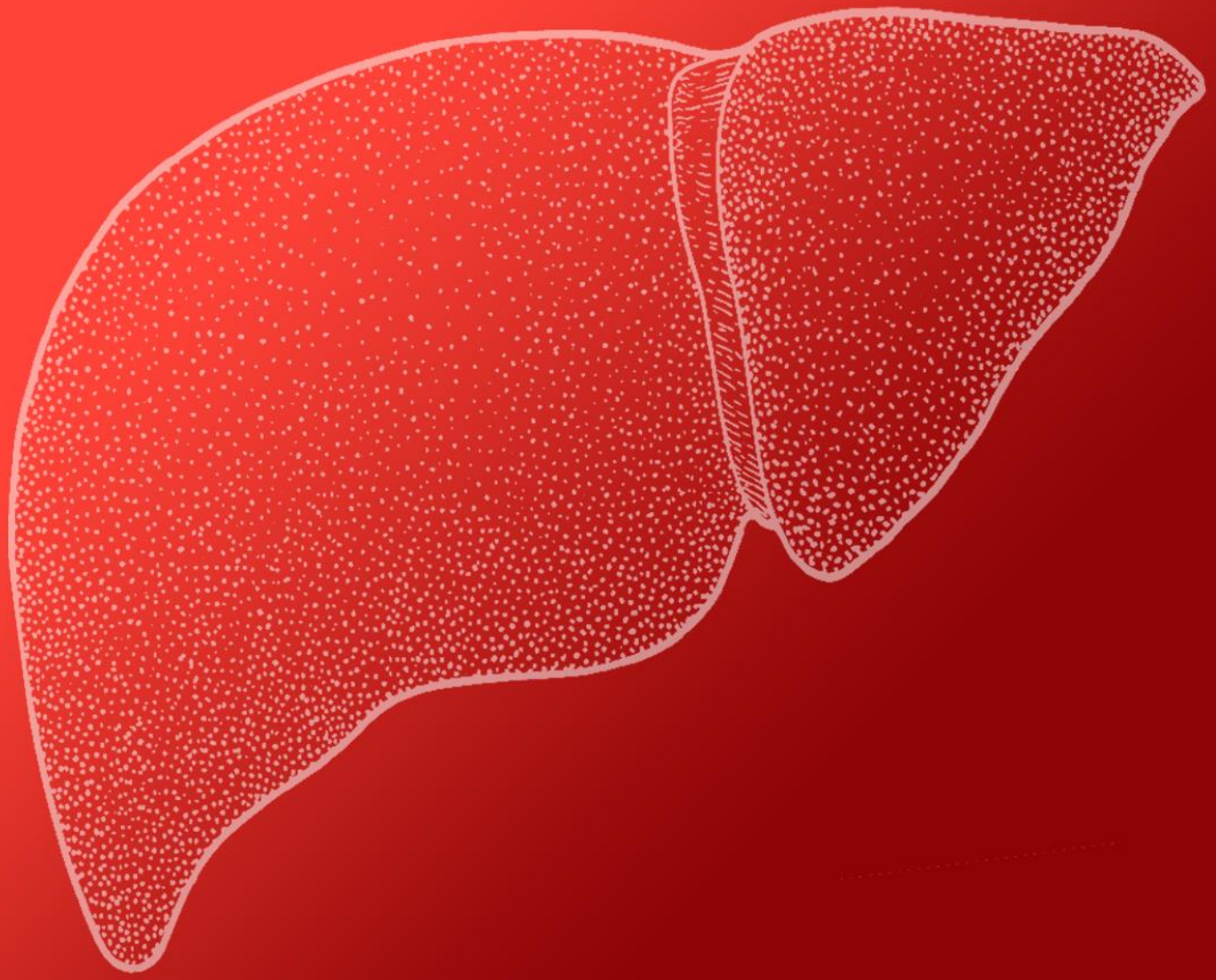
YE 2020
Initiate Part 1 of adaptive phase 1 study in patients with CFRD or impaired glucose tolerance

Q2 2021
Initiate Part 2 of adaptive phase 2 study in patients with CFRD or impaired glucose tolerance

NASH

HPP3033

Nrf2/Bach1 modulator for Non-Alcoholic Steatohepatitis (NASH)



NASH / HPP3033

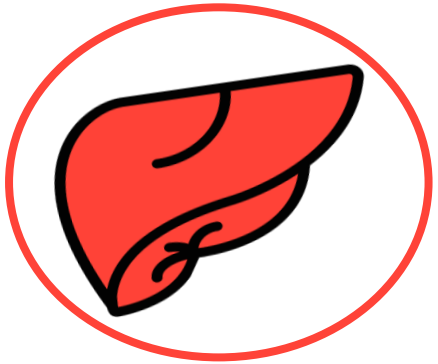
Nrf2 Activation – Promising Approach for Organ Protection

First-in-class, potentially best-in-class approach to targeting Nrf2 / Bach 1 pathway

vTv's Nrf2/Bach1 modulators, distinct MoA:

- Non-electrophilic molecules
- Keap1 Cys-151 independent Nrf2 activation
- Nrf2 stabilization
- Induction of Nrf2 nuclear import and Bach1 nuclear export
- Effects are not mediated by other transcription factors

vTv compounds demonstrated evidence of target engagement and efficacy in disease-relevant animal models and patient cells (e.g. kidney, heart, lung, liver, brain, eye, bone and blood)



Nrf2 benefits and impacts in the liver[1]

Suppressing lipogenesis

Supporting mitochondrial function

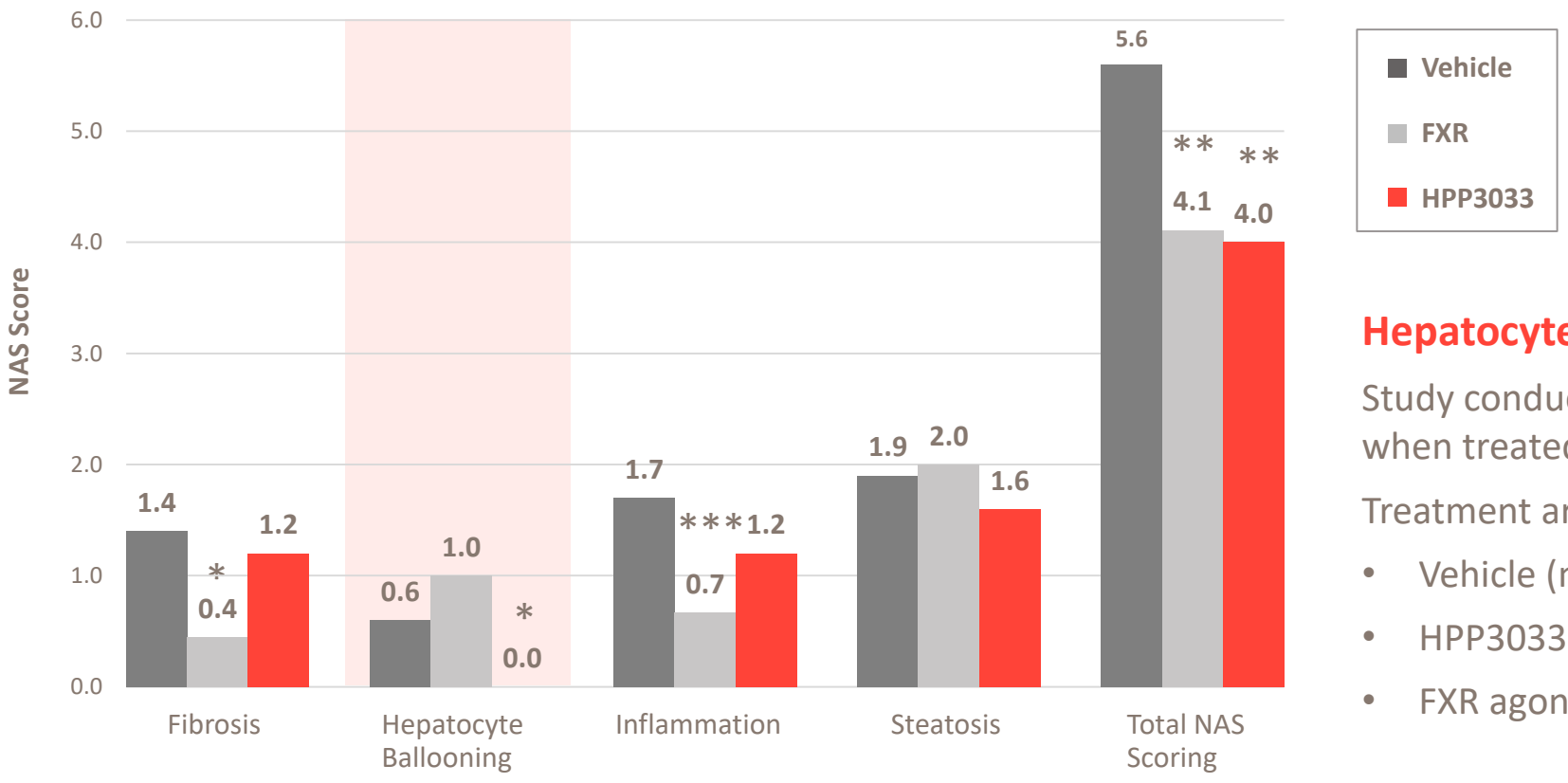
Increasing unfolded protein response

Reducing oxidative stress and inflammation

1. P Meakin Mol Cel Biol. 2014 Sep;34(17):3305-3320, H Sugimoto Am J Physiol Gastro&Liver Physiol 2010 Jan;298 (2) 283-294

HPP3033 Prevented Hepatocyte Ballooning in NASH Model

MCD NASH Model



*p<0.05, **p<0.01, ***p<0.001 vs. vehicle

Hepatocyte Ballooning Score of Zero

Study conducted in mice with diet induced NASH when treated once a day for 3 weeks

Treatment arms:

- Vehicle (n=10)
- HPP3033 30 mg/kg (n=22)
- FXR agonist WAY-362450 30 mg/kg (n=10)

Development Plan

Q2 2020
Conduct NASH Animal Model

YE 2020
Submit IND

Q1 2021
Initiate Phase 1

Partnered Development Programs

VTV Therapeutics

Creating Value Through Partnerships

Asset	Partner	Territory	Target Indications	Economics for vTv
HPP737 (PDE4i)	NEWSOARA 恒翼生物医药	China and other Pacific Rim Countries (excl. Japan)	COPD	Milestones and royalties Utilization of data to advance development in ROW
PPAR-δ Agonist Program	Reneo	Worldwide	Rare mitochondrial diseases, Fatty Acid Oxidation Disorder	Equity interest in Reneo Milestones and Royalties
TTP273 (Oral GLP-1r)	华东医药 HUADONG MEDICINE	China and other Pacific Rim Countries (excl. Japan)	Type 2 Diabetes	Milestones and Royalties Utilization of data to advance development in ROW

Thank you

VVT Therapeutics