Therapeutics

Corporate Presentation

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

August 2020

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 other 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.

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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 2019 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

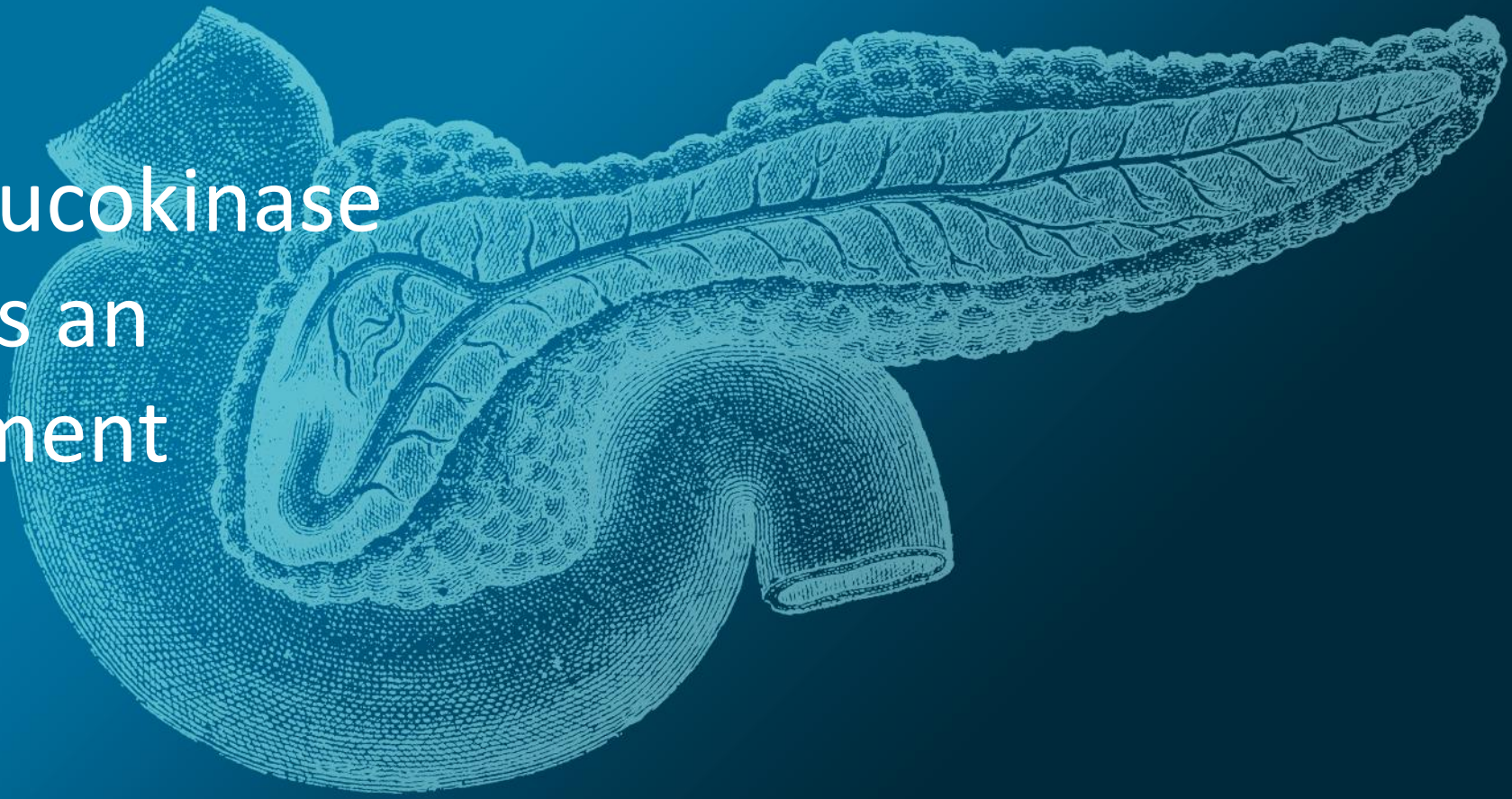
Indication	Preclinical	Phase I	Phase II	Phase III	Biological Rational
Type 1 Diabetes (T1D)	TTP399 (GKA)				Liver-selective GKA; no disruption of GK regulatory protein
Dementia with Diabetes	Azeliragon (RAGE)				Small molecule antagonist of RAGE
Cystic Fibrosis Related Diabetes (CFRD)	TTP273 (Oral GLP1-R)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

Partnered Programs

	Preclinical	Phase I	Phase II	Phase III	Partner / Territory
Type 2 Diabetes (T2D)	TTP273 (Oral GLP1-R)				华东医药 HUADONG MEDICINE — China and other Pacific Rim Countries (excl. Japan)
Rare Mitochondrial Disease	HPP593 (PPAR-d)				Reneo — Worldwide
COPD	HPP737 (PDE4)				NEWSOARA 恒翼生物医药 — China and other Pacific Rim Countries (excl. Japan)

Diabetes

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

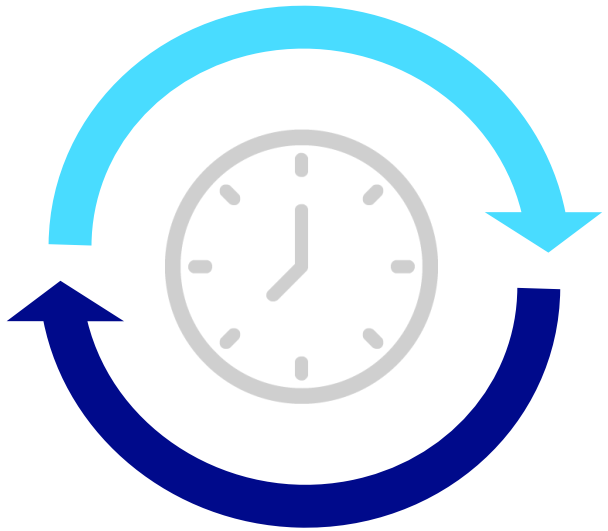


T1D is a Burdensome Disease

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



It requires constant management, 24 hours a day

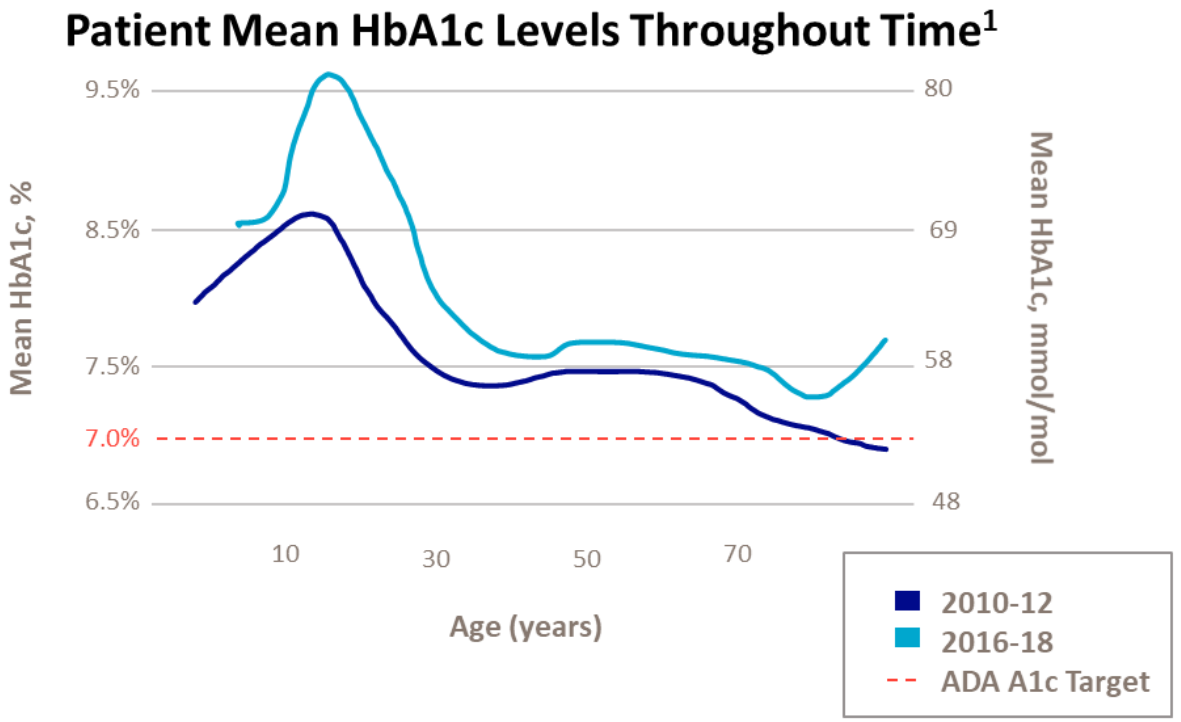
Must count the carbs and account for everything they eat

It is exhausting and has long-term dangerous complications

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]



Life-threatening, short-term complications of poor glycemic control

Severe Hypoglycemia:

- **6% of T1D patients** reported having a **seizure** or **loss of consciousness** (symptoms of severe hypoglycemia) over the previous 3-month period[3]
- **5% of T1D patients are hospitalized** or visit the ER at least once in the last year due to severe hypoglycemia[4]

Diabetic Ketoacidosis (DKA):

- DKA accounts **for 14% of all hospital admissions** of patients with diabetes and **16% of all diabetes-related fatalities[5]**

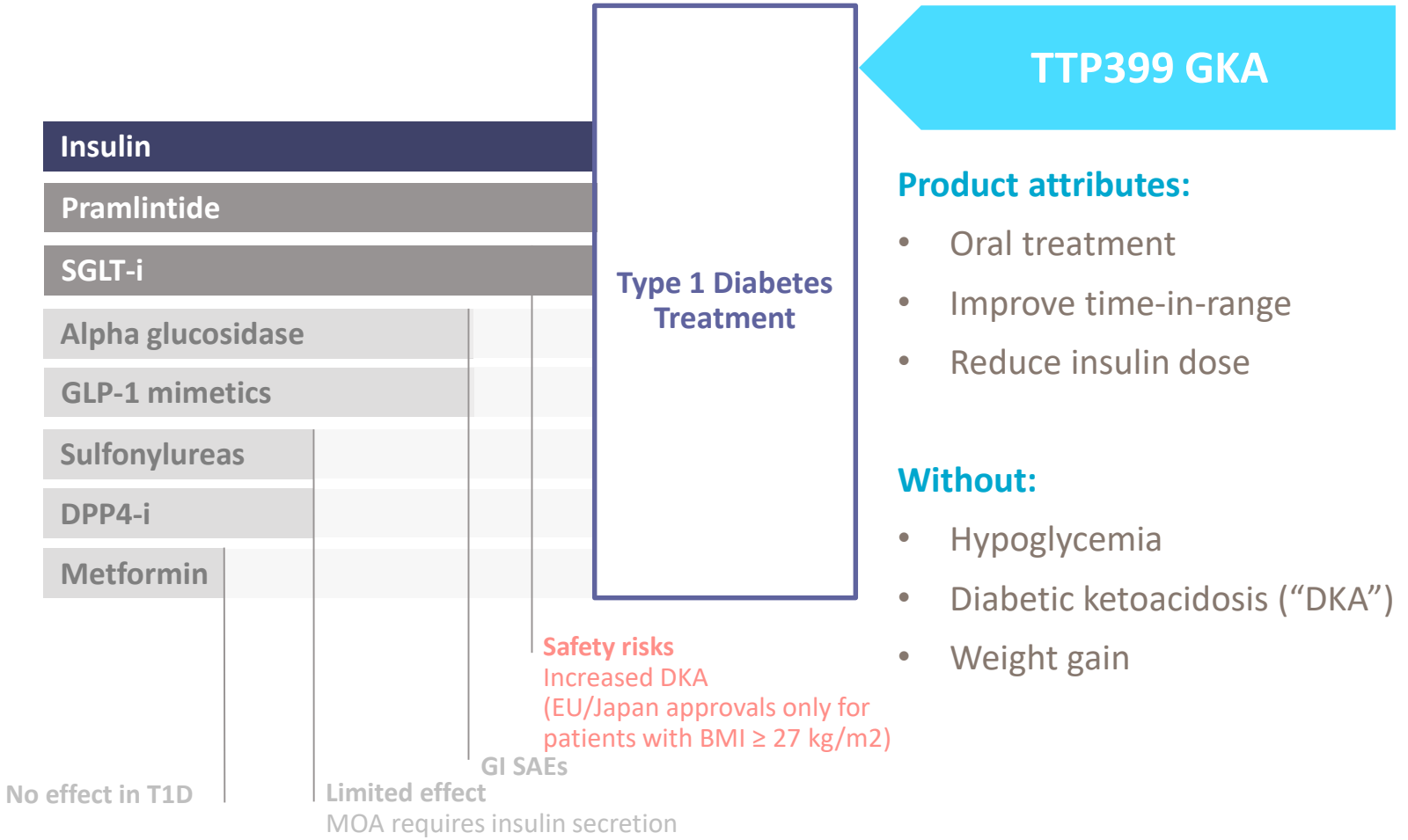
1. Diabetes Technol Ther. 2019 Feb;21(2):66-72. doi: 10.1089/dia.2018.0384. Epub 2019 Jan 18.
2. Foster et al. Diabetes Technology and Therapeutics (2019) 21:66-72; DOI: 10.1089/dia.2018.0384
3. Miller KM, et al. Diabetes Care 2015;38:971–978 | DOI: 10.2337/dc15-0078
4. Pettus J, et al. Diabetes Care 2019;42:2220-2227 I https://doi.org/10.2337/dc19-0830
5. Osama Hamdy, et al. Medscape May 31, 2019, Diabetic Ketoacidosis (DKA)

Limited Treatment Options for a Significant Patient Population

Large commercial opportunity with significant unmet need

- **30 million** people suffer from T1D globally(1)
- **In the US, 1.5 million** adult and pediatric T1D patients(2) ; ~77k new T1D adults diagnosed annually
- **Nearly 80%** of people with T1D **fail to achieve ADA target A1c levels**(3)
- Limited historical innovation for current standard of care
  - Requires constant management and monitoring
- **No oral adjunct therapies approved** in the US
- Potential >$1 billion market for oral adjunctive treatments to insulin in T1D

No approved oral therapies for T1D in the US, and available T2D treatments have limited potential in T1D(4)



TTP399 GKA

Product attributes:

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

Without:

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

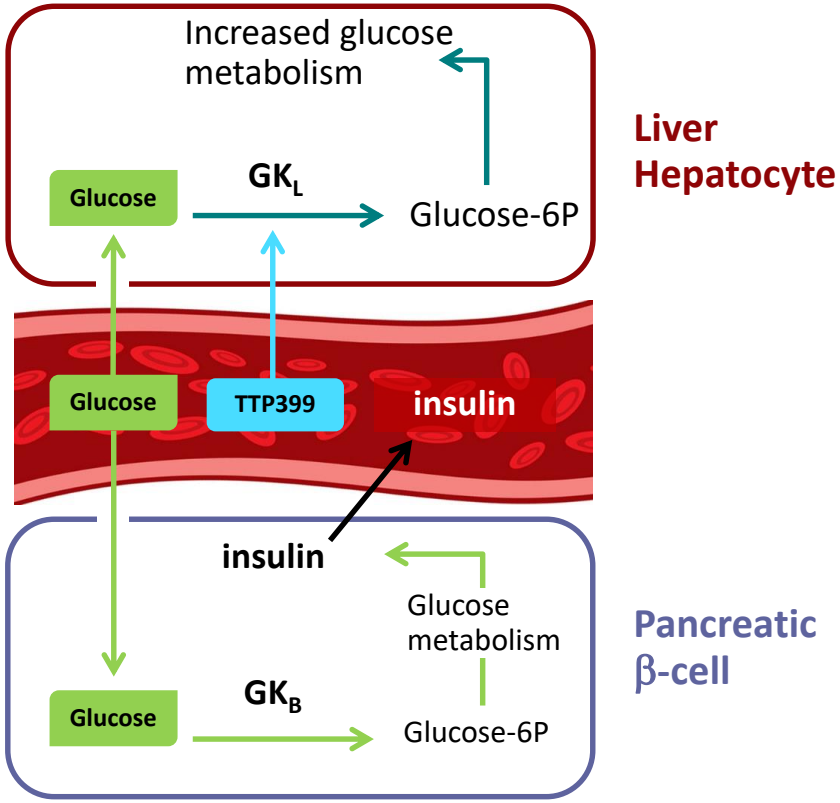
(1) IDF Diabetes ATLAS 8th edition.
(2) Global data, 2019.
(3) Diabetes Technol Ther. 2019 Feb; 21(2):66-72. doi: 10.1089. Epub 2019 Jan 18.
(4) American Diabetes Association: Diabetes Care 2019; 42 (Supplement 1):S90-S92, https://doi.org/10.2337/dc19-S009.

GKA, a Unique Biological Strategy to Support T1D Patients

Glucokinase facilitates 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

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

Study Details:

- Double-blind Placebo controlled
- The Simplici-T1 trial was designed to explore the **safety and efficacy** of the liver-selective GKA, TTP399, as an oral adjunctive therapy for T1D
- **12 weeks dosing** with 800mg QD and placebo
- The **treat-to-target**(2) design of the study allowed changes in insulin dose after the insulin-optimization period

Phase 2-Part 1
(Learning Phase)

- **4 sites**
- **19 adult subjects** with T1D on CSII and CGM(1)
- Primary Endpoint: Δ in HbA1c
- Basal insulin optimized prior to commencement of the study (baseline HbA1c 7.3%)

Positive Results Reported June 2019

Phase 2-Part 2
(Confirming Phase) –*Designed to confirm positive Phase 2-part 1 results*

- **13 sites**
- **85 adult subjects** with T1D (all comers)
- Primary Endpoint: Δ in HbA1c
- Basal insulin optimized prior to commencement of the study (baseline HbA1c of 7.6%)

Positive Results Reported February 2020

Note: ClinicalTrials.gov Identifier: NCT03335371.
(1) Subjects with Continuous Subcutaneous Insulin Infusion (CSII) and Continuous Glucose Monitoring (CGM).
(2) Treat-to-target: FPG: ~80-130mg/dL; post meal glucose: <180-200 mg/dL

JDRF IMPROVING LIVES. CURING TYPE 1 DIABETES.

Simplici-T1 Study - Key Results

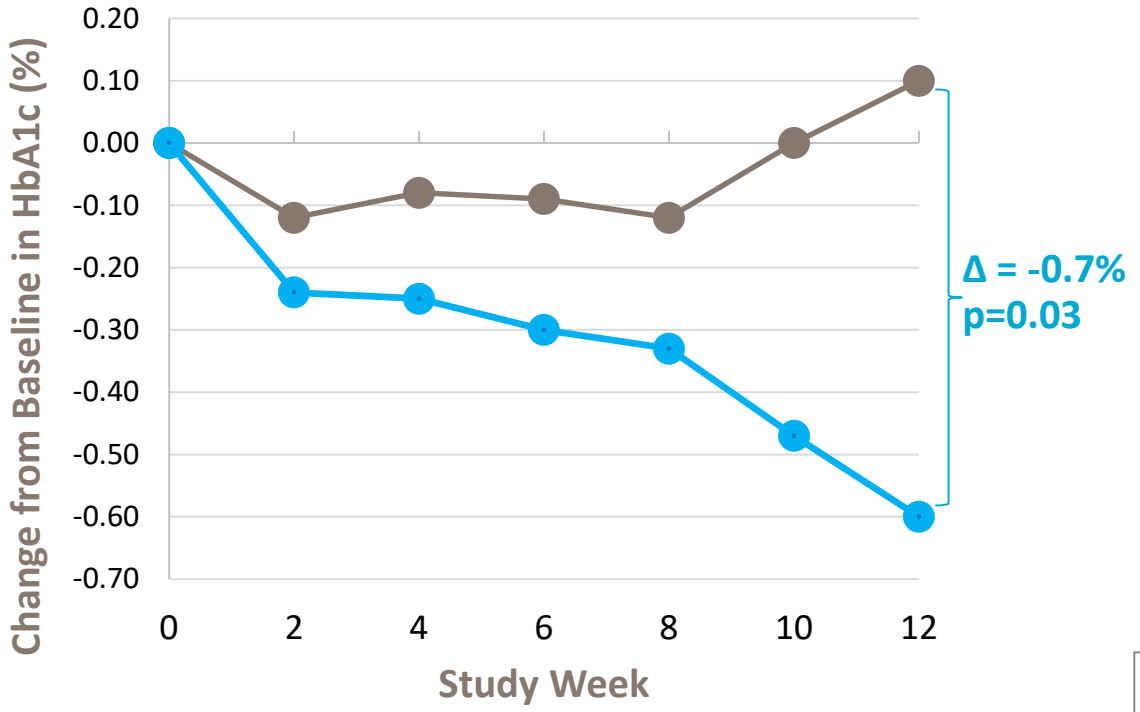
- **Statistically significant reduction in HbA1c** under a treat-to-target design (i.e. compared to intensive insulin treatment)
- **Reduced total daily mealtime bolus insulin** relative to baseline
- **No report of diabetic ketoacidosis,** trends towards reduction in ketone events were observed in the TTP399 treated group compared to placebo
- **Fewer hypoglycemic episodes** in TTP399 vs. placebo
- **Increase in time in range** relative to placebo

Phase 2 – Study (Parts 1 and 2) Met Primary Endpoint, Reduced HbA1c

Statistically Significant HbA1c Reductions Without Increases in Ketones or Hypoglycemia

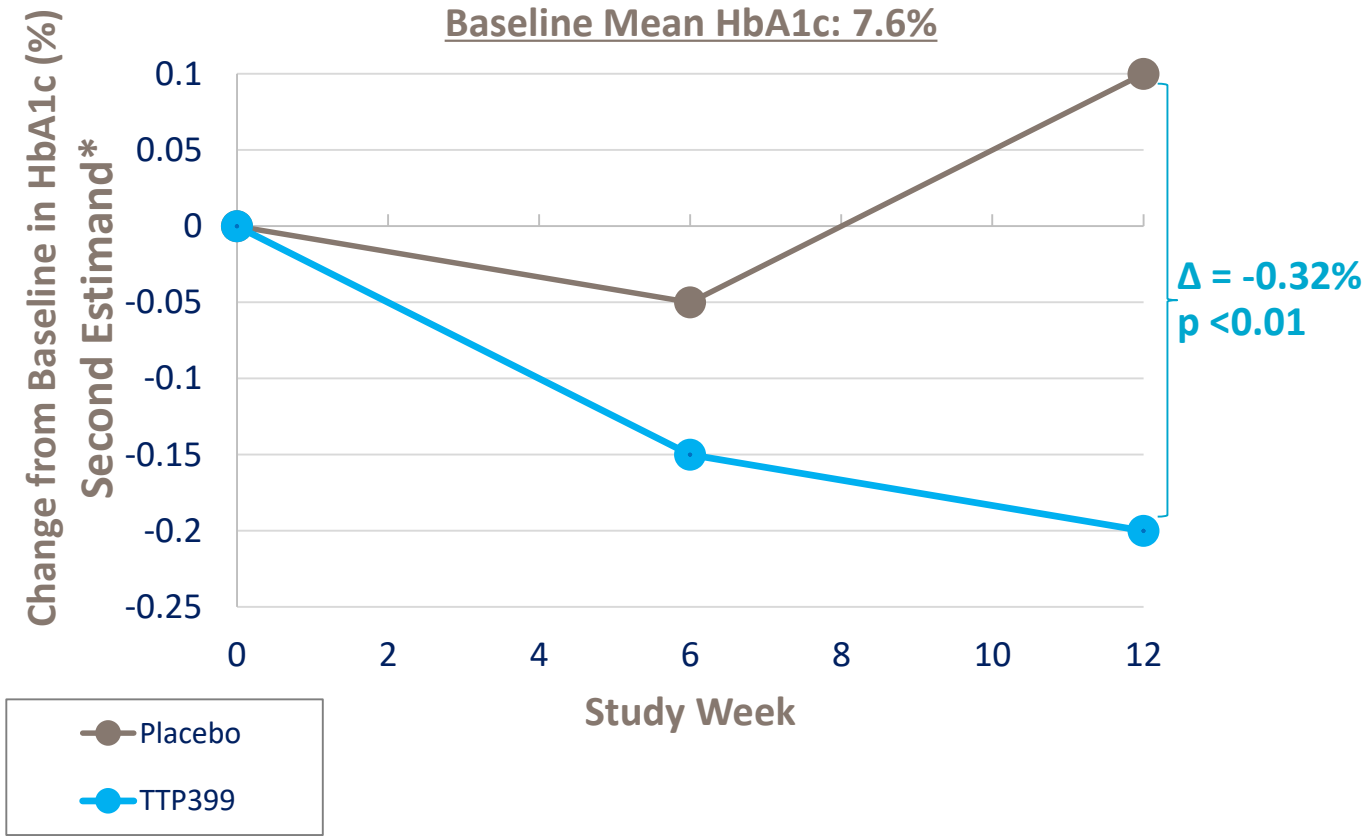
Part 1

Baseline Mean HbA1c: 7.3%



Part 2

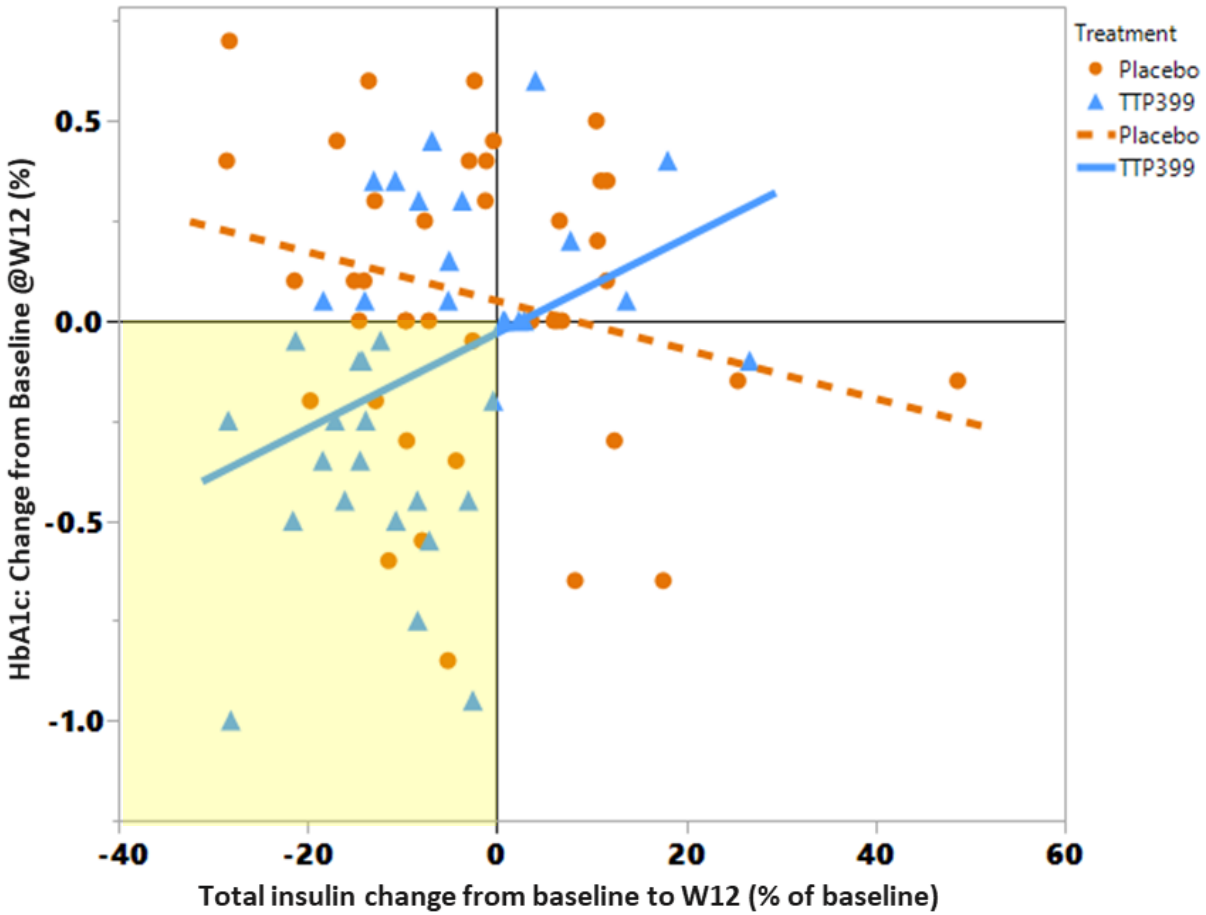
Baseline Mean HbA1c: 7.6%



*The pre-specified second estimand analysis evaluated the effect on HbA1c for patients without evidence of noncompliance with prescribed treatment who did not administer notable increases of bolus insulin of three or more units. This second estimand analysis was conducted consistent with current regulatory guidance.

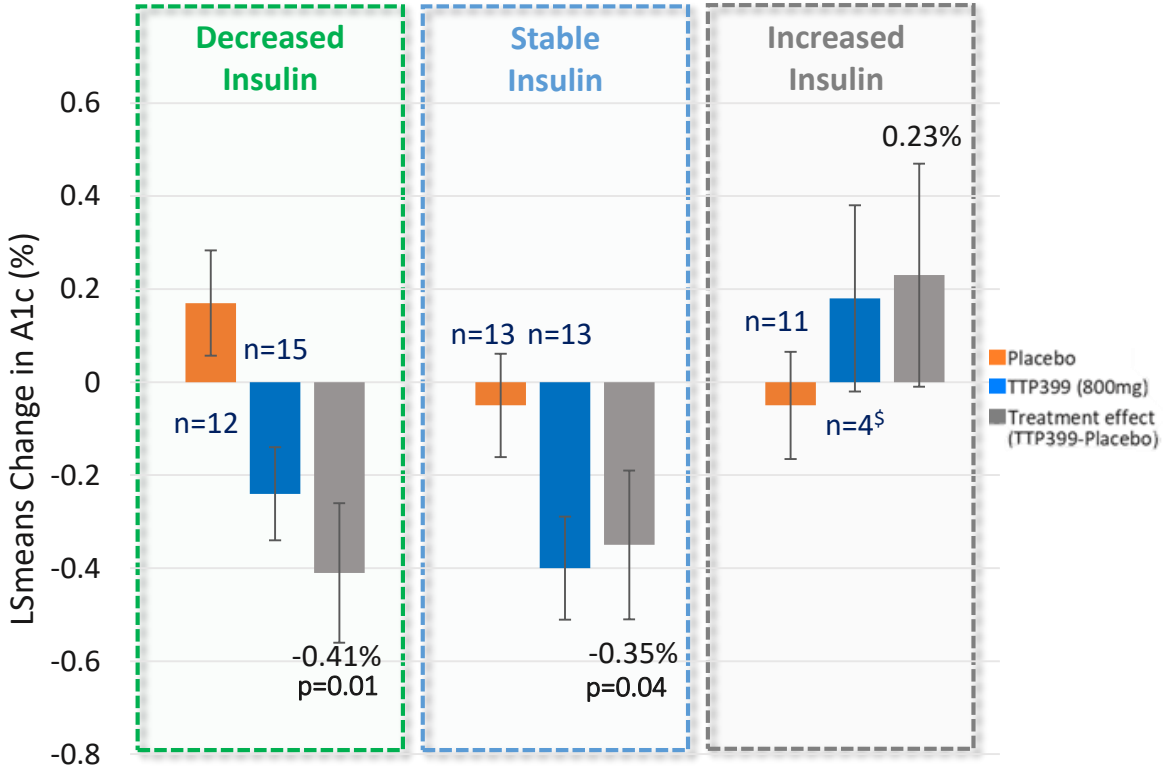
Phase 2 - Part 2: TTP399 Treated Subjects Achieved Better Glycemic Control while Reducing Insulin Dose

Δ HbA1c vs ΔTOTAL Insulin



*

Change in HbA1c @ W12 by Subgroup



The criteria used to define the subgroups were based on change from baseline in Total Insulin (U/kg/day):

Decreased insulin: Δ<= -0.06 U/Kg/day

Stable insulin: Δ =-0.06 - 0.03 U/Kg/day

Increased insulin: Δ >=0.03 U/Kg/day

$: TTP399 levels undetectable in two of the subjects that increased insulin dose during study

Error bars are SE

Phase 2 - Part 2: Compelling Safety Profile with No Diabetic Ketoacidosis and No Incidence of Severe Hypoglycemia in the TTP399 Group

Phase 2 – Part 2 Safety:

- No diabetic ketoacidosis
- No incidence of severe hypoglycemia in TTP399 group (1 incident in placebo group)
- Fewer subjects reported symptomatic hypoglycemic episodes in TTP399 group compared to placebo
  - 12% TTP399 vs 20% PBO
- Similar profiles for reported TEAEs between TTP399 and placebo

No detrimental safety signals across multiple parameters in TTP399 treated group when compared to placebo, unlike other oral MOAs investigated for T1D:

- BOHB in serum and ketones in urine
- LFTs
- Triglycerides
- Lipids

Development Plan*

Ongoing	**Requested and received Type C Meeting feedback from FDA** – dialogue continuing
End of 2020	**Initiate 6 month pivotal trial** followed by 6 mo Open Label Extension ➢Exploring primary endpoints of reduced HbA1c and/or reduced hypoglycemia or DKA
End of 2021	**Second 6 month pivotal trial** to start 9-12 months after first pivotal study

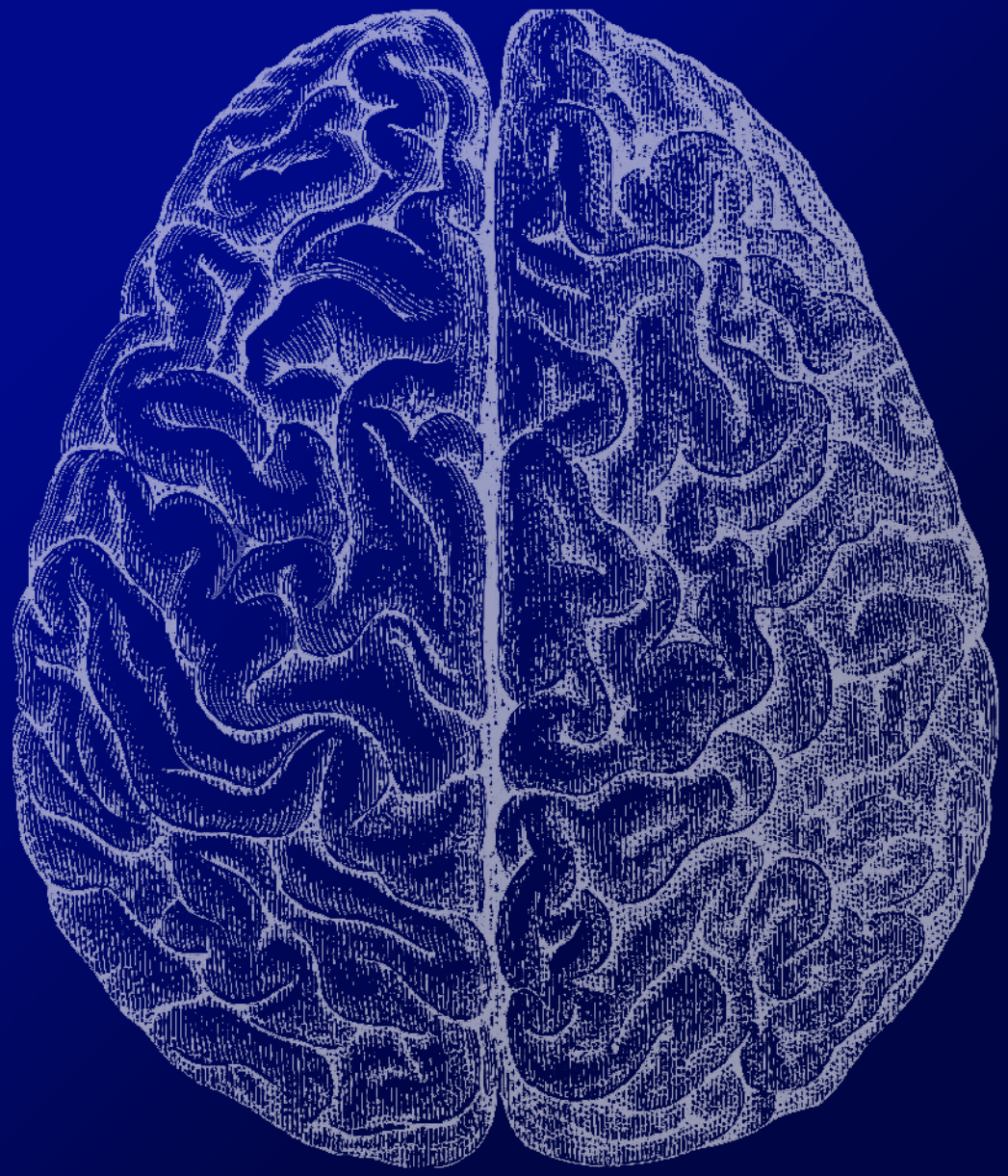
Estimated cost for entire development plan $75M-$90M

(including pivotal trials, clin pharm, API and drug product manufacturing)

*Current development plan may change based on continued dialogue with FDA and other stakeholders

Dementia

Azeliragon
RAGE antagonist
for Dementia with
Diabetes

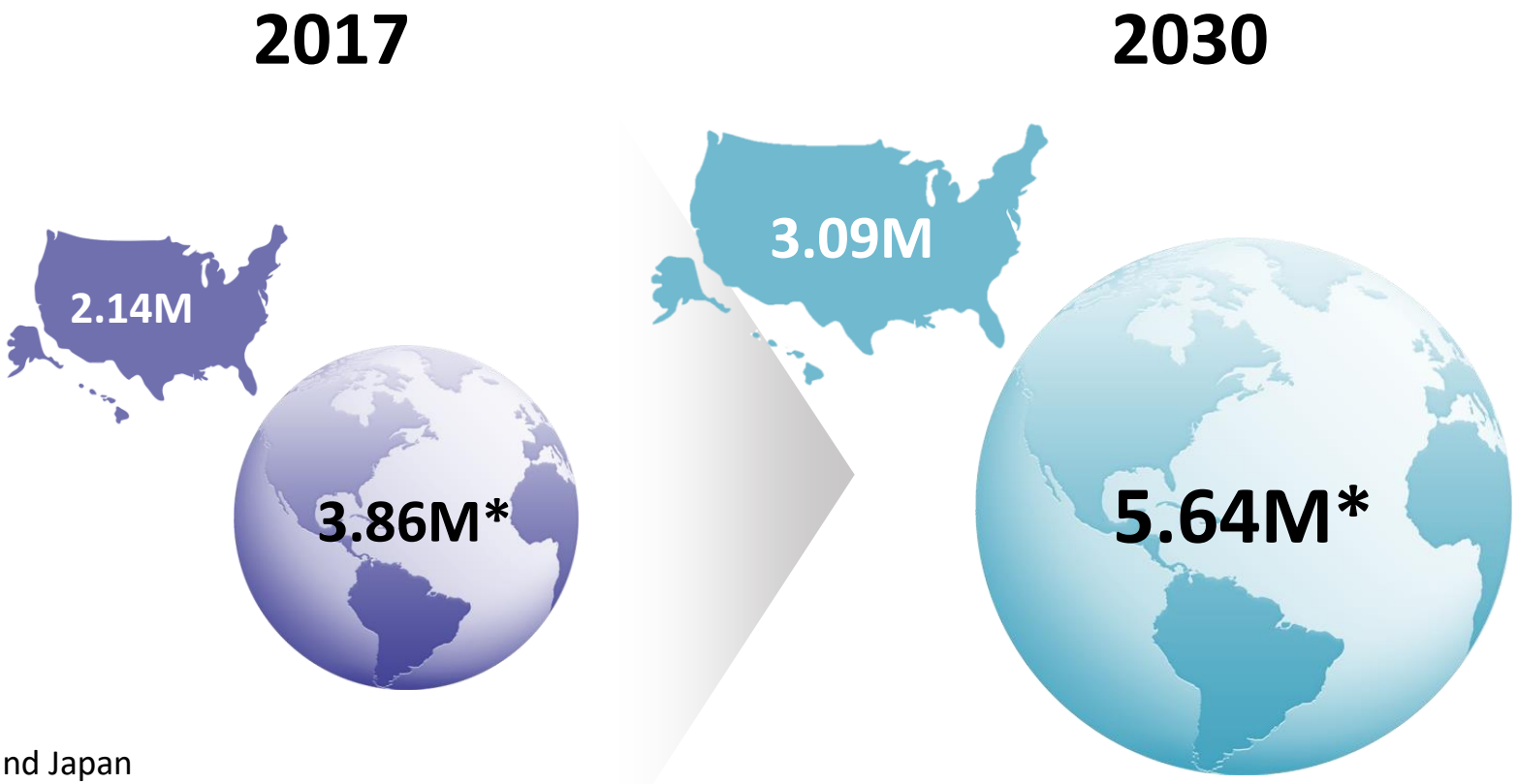


The Significance of Dementia with Diabetes

Market Size

Market for Dementia with diabetes expected to reach **$2.04B by 2030** in 7MM* from $1.18B in 2017

Prevalence of Dementia (Alzheimer's) with Diabetes

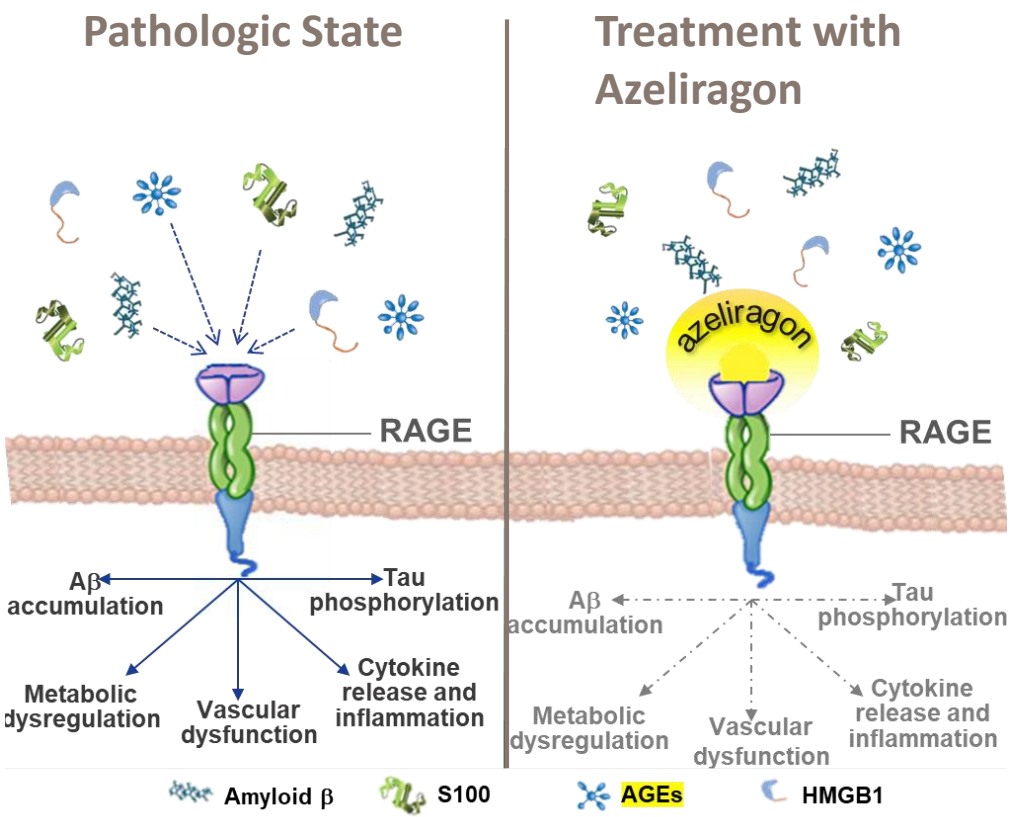


*7 Major Markets: US, Germany, France, Italy, Spain, UK and Japan

Source: Delveinsight, Dementia with Diabetes, Market Insights, Epidemiology and Market Forecast – 2030 , March 2020

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

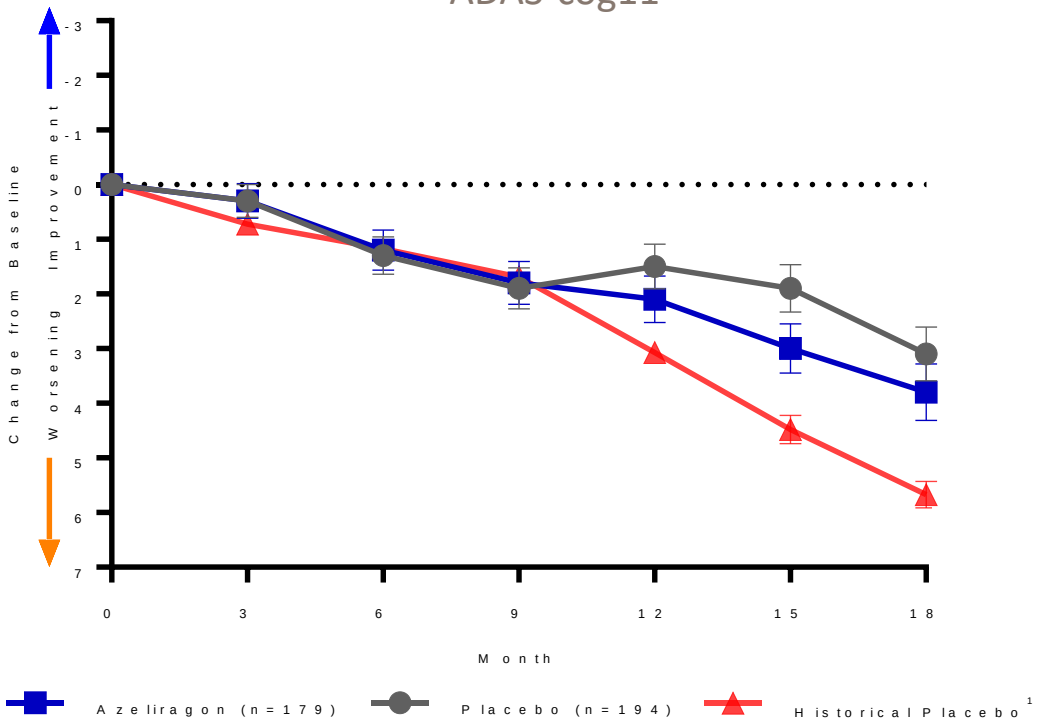
Potential benefits of RAGE antagonism for dementia in diabetes

- 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)

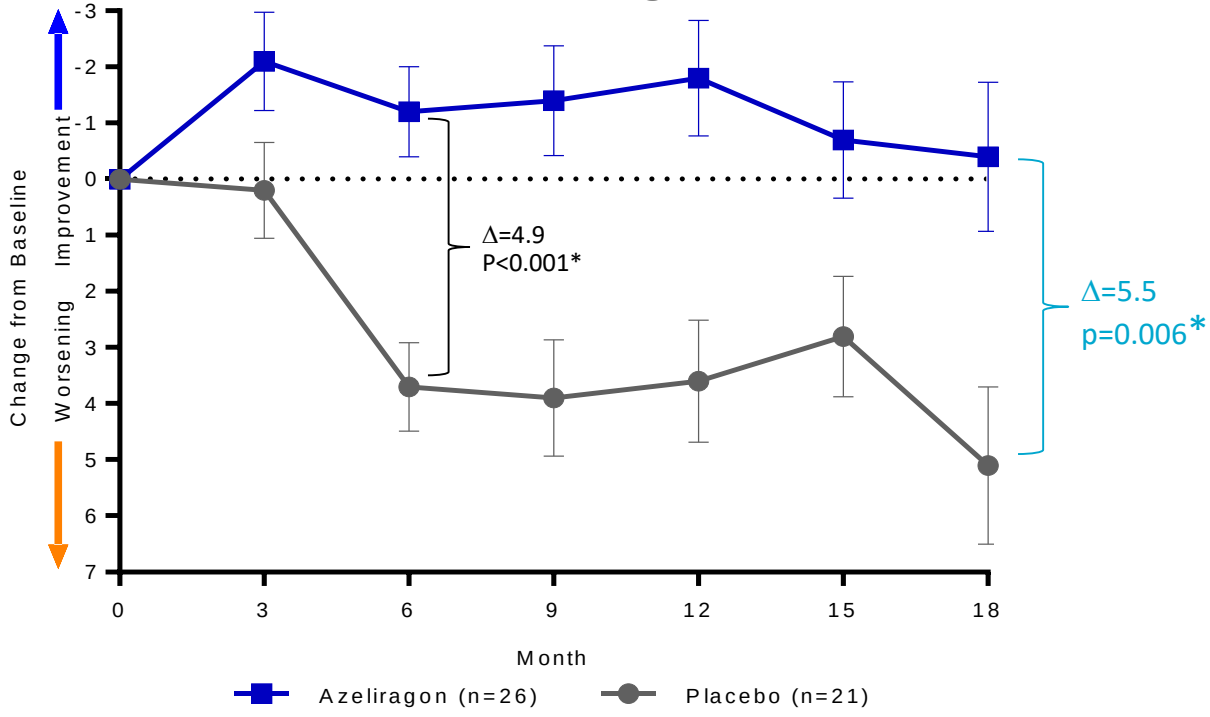
ADAS-cog11



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)

ADAS-cog11



Cognitive benefits in ADA-T2D (HbA1c ≥ 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.*

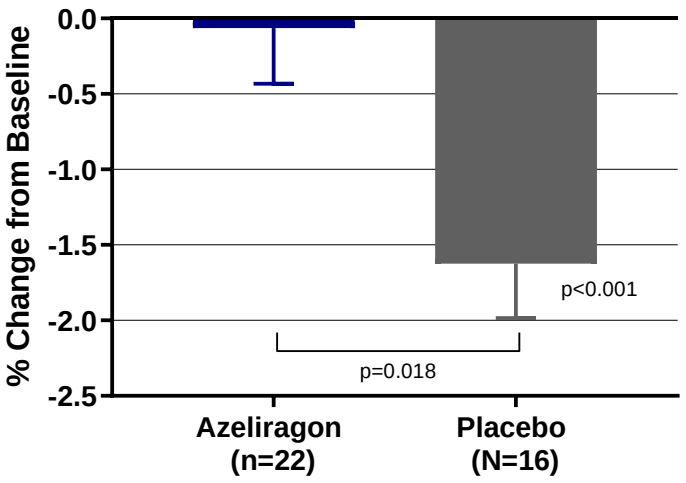
Brain MRI, FDG-PET and Plasma Inflammatory Biomarker Results Support Biological Effect in Dementia with Diabetes

STEADFAST A-Study Type 2 Diabetes Subgroup (FAS)

Less Brain Atrophy

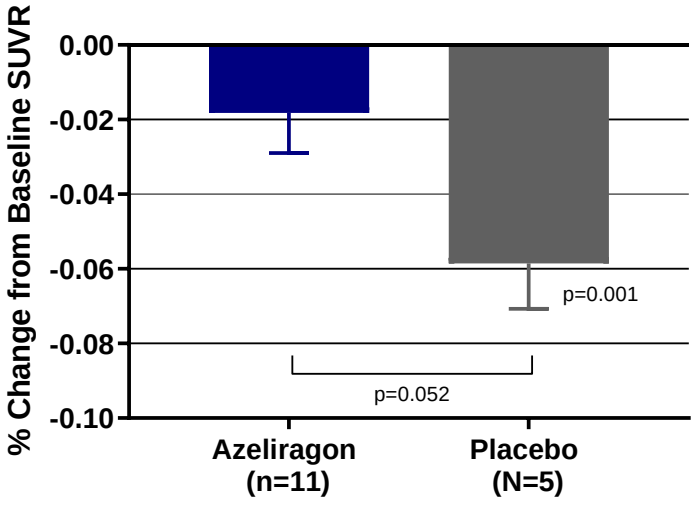
Whole Brain

Month 18



Less Reduction in Brain Glucose Utilization*

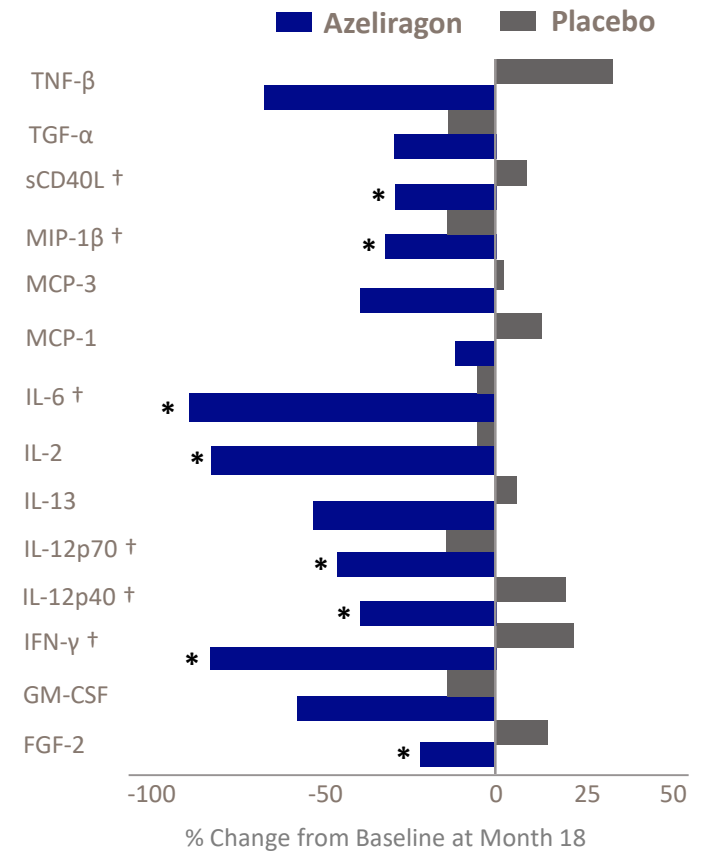
Month 18



Change in ventricular enlargement (%) and total hippocampal volume (%) also directionally consistent (not significant) to favoring azeliragon

Reduction in Plasma Inflammatory Biomarkers

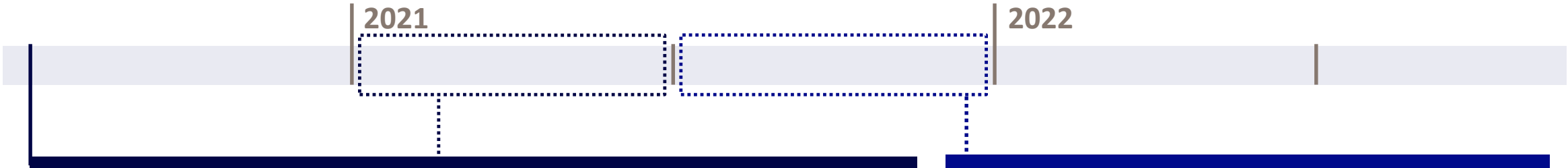
Month 18



Results are Medians
* Nominal p<0.05 Wilcoxon test
† Biomarkers with direct relationship to RAGE

*FDG-PET SUVR composite (unweighted combination of frontal, anterior/posterior cingulate, lateral parietal, lateral temporal, and hippocampus)

Elevage Study: Two Studies Conducted Under a Single Protocol



STARTED JUNE 2019

Phase 2

(Part 1 / Proof of Concept) – 6 months, targeting ~50 patients to be enrolled by end of Q3 2020

Proof of concept study to confirm the cognitive benefits evidenced in the diabetes subgroup (n=47) of the STEADFAST study

Powered to demonstrate treatment difference between azeliragon and placebo on the ADAS-cog

Readout Q2 2021

2H 2021

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

ClinicalTrials.gov Identifier: NCT03980730

elevage
Alzheimer's Disease study

Development Plan

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

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	Primary Mitochondrial Myopathy, Fatty Acid Oxidation Disorder, McArdle Disease	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