Vogenx

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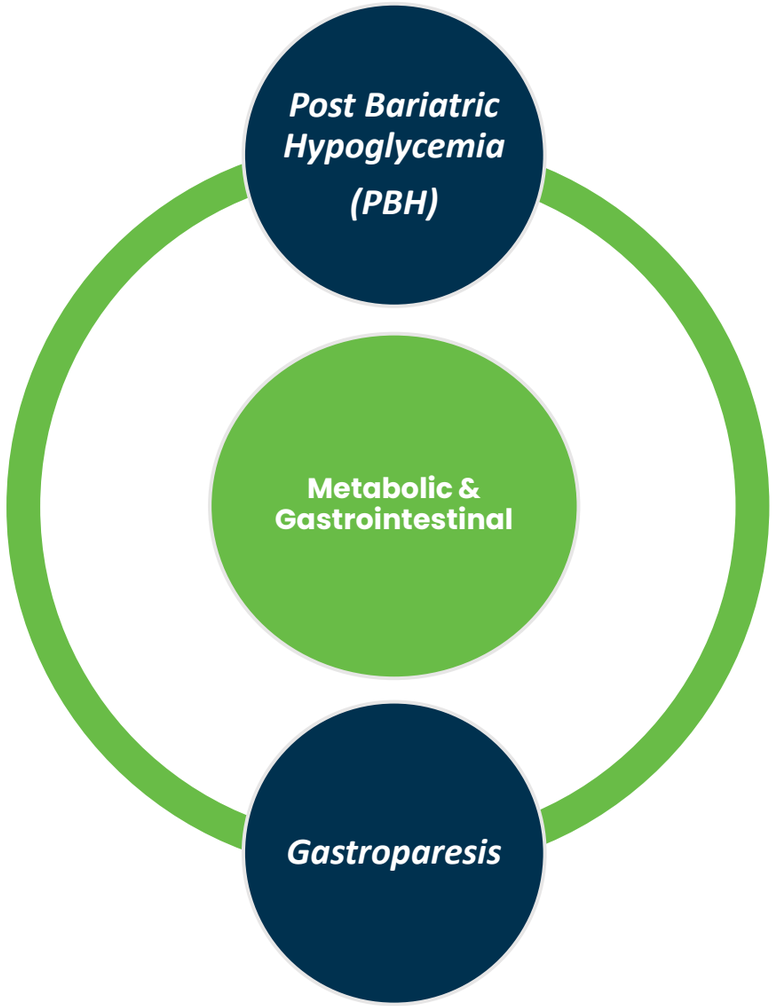
Corporate Overview



- Founded in 2021; Based in Raleigh, North Carolina
- Series A Round, Q1 2022
- Novel Phase 2 Oral Small Molecule Drug Candidate
- No drugs approved for PBH; Single approved option for gastroparesis
- Two successful Phase 2 readouts in patients diagnosed with PBH
- PBH program is Phase 2b ready; gastroparesis entering Phase 2
- Both indications with potential multi-billion-dollar addressable markets

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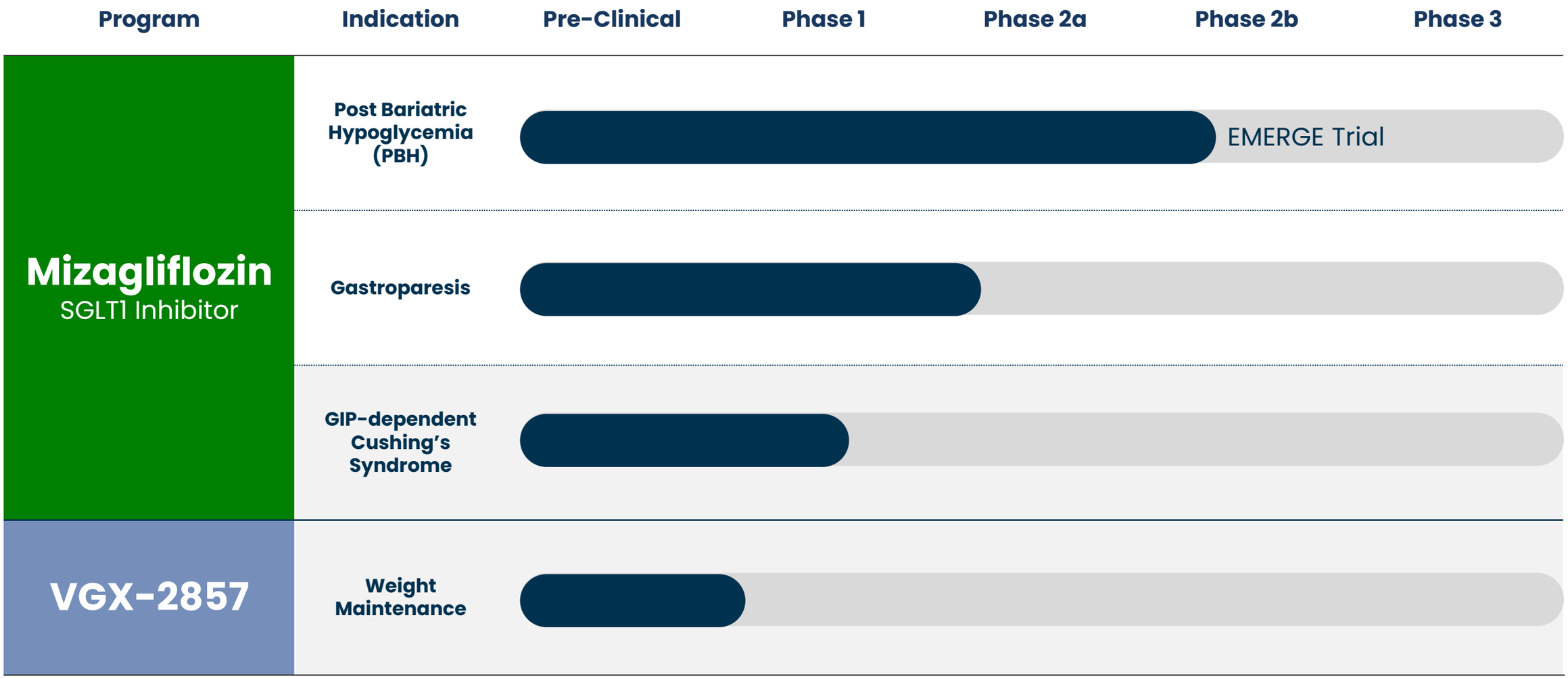

Program
Indication
Pre-Clinical
Phase 1
Phase 2a
Phase 2b
Phase 3
Mizagliflozin
SGLT1 Inhibitor
Post Bariatric Hypoglycemia (PBH)
EMERGE Trial
Gastroparesis
GIP-dependent Cushing's Syndrome
VGX-2857
Weight Maintenance

Mizagliflozin

Novel orally dosed small molecule inhibitor

Target and Site of Action:

SGLT1 in the intestinal lumen

Mechanism of Action:

Inhibition of glucose absorption and reductions in secretion of insulin and GIP

Mizagliflozin Overview

Development Status
- Phase 2b ready for PBH
- Phase 2a ready for Gastroparesis
- Pre-clinical package complete (including 2-year carcinogenicity in rats)

Safety
- Observed to be well tolerated in Phase 2 studies
- No drug-related significant adverse events

Efficacy
- Significant lowering of insulin, glucose and GIP (glucose-dependent insulinotropic polypeptide)
- Improvements in glucose nadir, Level 2 and Level 3 hypoglycemic events

CMC
- Optimized synthetic route
- API stable for 60 months at long-term conditions and for 6 months at accelerated conditions

Intellectual Property
- Composition of matter issued
- PBH method of use issued in the U.S. & pending in E.U. (2019 priority date)
- Gastroparesis method of use pending (2024 priority date)

Post Bariatric Hypoglycemia (PBH)

Indication Summary

- Chronic disorder related to bariatric surgery and characterized by reactive postprandial hyperinsulinemic hypoglycemia
- Spectrum disease with varying levels of severity and frequency of events
- Patients have above average rates of hospitalization & medical burden, and mental health issues associated with meal related anxiety
- No FDA approved therapeutics; standard of care is diet modification

Prevalence

- Recent studies suggest between approximately 25% and 75% of bariatric surgery patients wearing continuous glucose monitors exhibited PBH (U.S.: ~3 million living bariatric surgery patients; ~200,000 surgeries per year)
- Spectrum disease with estimated ~500,000 symptomatic PBH patients
  - ~200,000 require daily medical management
  - ~300,000 with less severe or lower frequency of events may benefit from daily or as-needed/PRN treatment
- >25% of bariatric surgery patients have an asymptomatic hypo-unaware form of PBH suggesting prevalence estimates substantially underestimated
- Potential multi-billion-dollar addressable market

Why Mizagliflozin – Competitive Advantage

Company	Product Candidate	Drug Substance	Target Mechanism	Target Distribution	Development Status	Administration	Reduce Glucose Absorption	Reduce GIP	Reduce Insulin Secretion
Vogenx	**Mizagliflozin**	**Small Molecule**	**SGLT1 Inhibitor**	**GI Only**	**Phase 2b**	**Oral**	✓	✓	✓
Amylyx	Avexitide	Peptide	GLP-1 Antagonist	Systemic	Phase 3	Injectable	✗	✗	✓
Recordati	Pasireotide	Peptide	Somatostatin Analog	Systemic	Phase 2	Injectable	✗	✓	✓
MBX	Imapextide	Peptide	GLP-1 Antagonist	Systemic	Phase 2	Injectable	✗	✗	✓

✓ **Oral Administration**

- Oral small molecule preferred by patients; lower supply chain costs
- TID oral reduces substantial anxiety associated with meals and allows for addressing large segment of PBH market

✓ **Mechanism: Acts locally in the GI tract**

- PBH hypo events correlate strongly to upstream glucose absorption (GLP-1 elevation is a downstream secondary effect), and bariatric surgery patients upregulate SGLT1 post-surgery — the primary transporter of prandial glucose
- Miza targets PBH events by inhibiting upstream glucose absorption via SGLT1; GLP-1 antagonists treat PBH via downstream postprandial GLP-1 secretion
- SGLT1 inhibition reduces postprandial glucose and GIP, with an effect on insulin that appears superior to GLP-1 antagonists
- Confounding real-world evidence: anecdotal reports of PBH patients benefiting from GLP-1 agonists (likely via slowed GI transit) and carb-restricted diets as first-line therapy

Why Mizagliflozin – Competitive Advantage

Safety & Tolerability

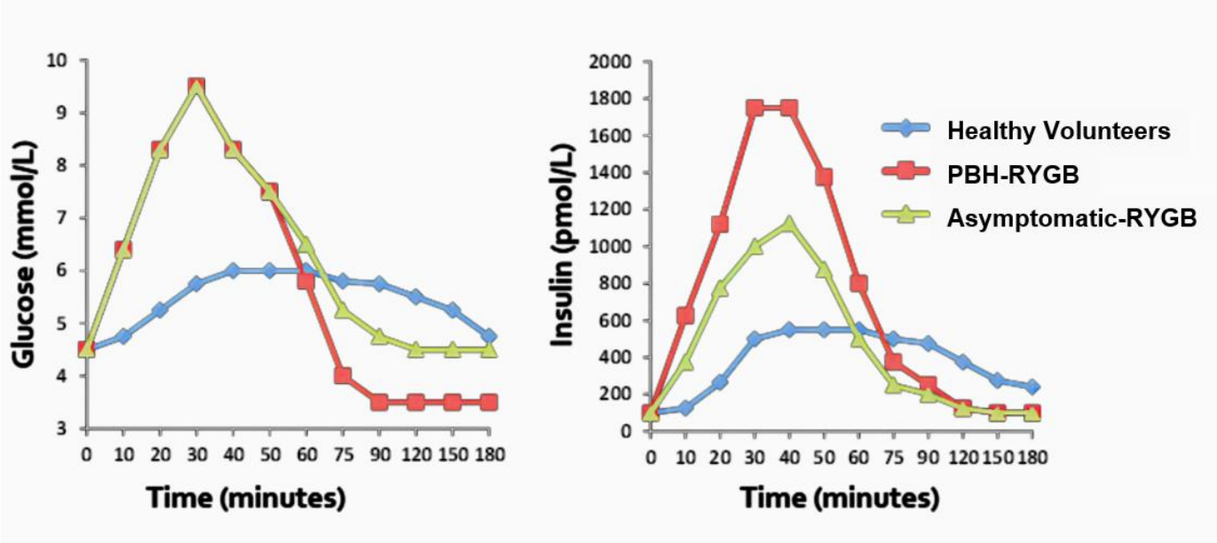
- Other drugs in development focus only on downstream effects that can lead to postprandial hyperglycemia (pathway to diabetes)
- Systemic injectable peptides with unknown long-term safety risks vs. mizagliflozin, a non-absorbable inhibitor
- A GLP-1 antagonist may have long term effects opposite of a GLP-1 agonist

Commercial

- Oral mizagliflozin expected to be suitable for the entire spectrum of PBH patients vs GLP-1 antagonist likely appropriate for subset of PBH market
- Large segment of PBH patients may benefit from oral PRN product
- Unlike mizagliflozin, SGLT2 and absorbable SGLT1 inhibitors inhibit glucose reabsorption in renal filtrate, which does not provide upstream relief to PBH patients

Post Bariatric Hypoglycemia – Glucose & Insulin

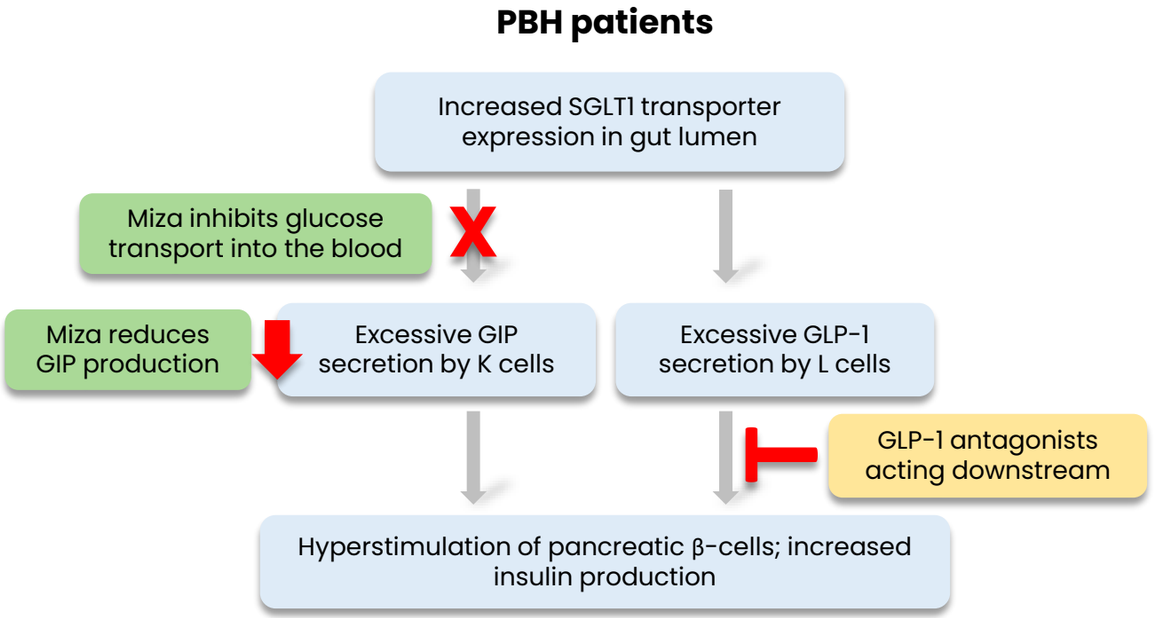
- The ideal PBH treatment would delay and reduce postprandial absorption of glucose and GIP secretion, thereby reducing the uncontrolled spike in insulin secretion[1]
- Therapeutics aimed only at reducing insulin secretion without a reduction in glucose absorption may lead to complications such as hyperglycemia or glucose toxicity[2]

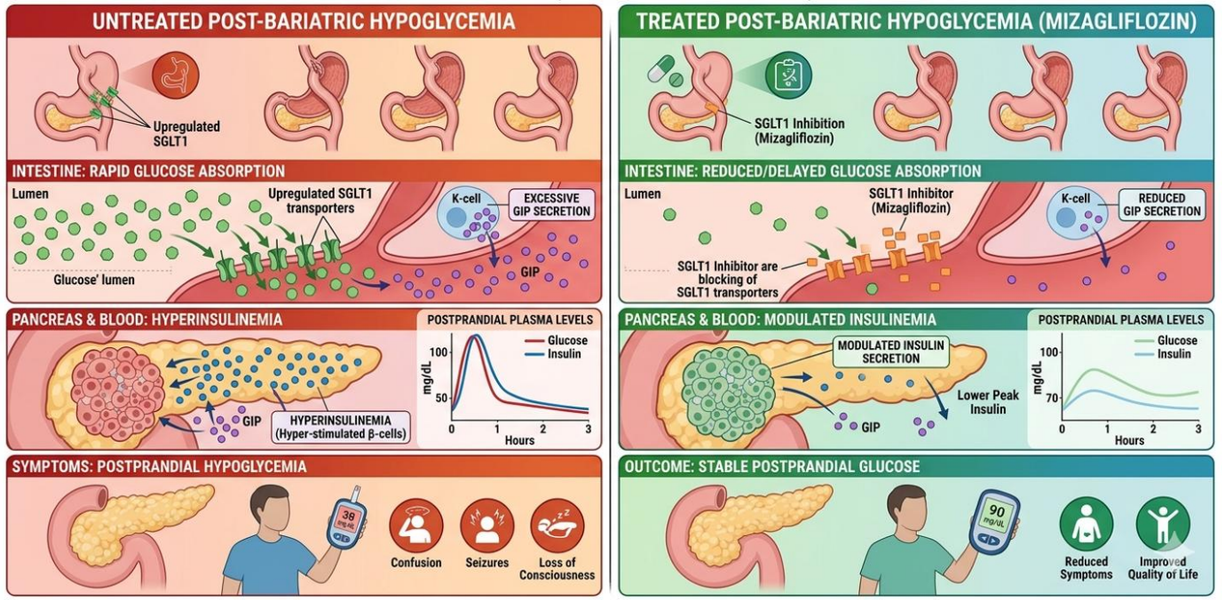


Adapted from Salehi et al, 2014

1 (Øhrstrøm CC et al 2019)
2 (Tan et al 2020)

Mizagliflozin: Upstream of Insulin Secretion





- SGLT1 is the primary transporter of glucose from the GI tract into the bloodstream and is overexpressed in bariatric surgery patients
- PBH patients have elevated SGLT1 expression which leads to abnormally high levels of absorbed glucose and subsequently increased GIP secretion[1]

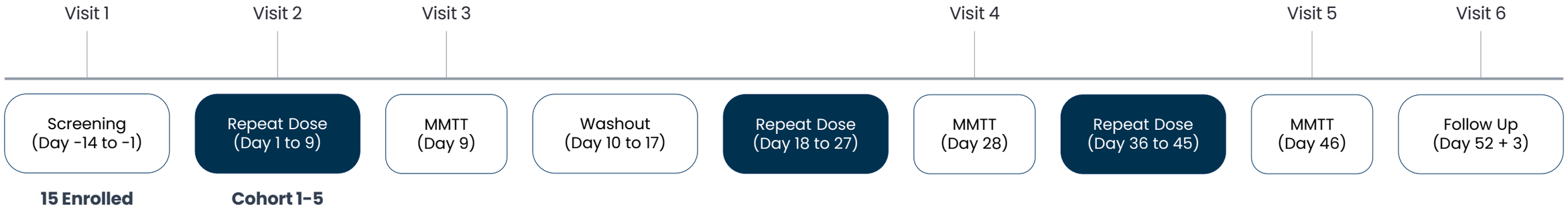
- Rapid glucose absorption leads to uncontrolled insulin secretion and postprandial hypoglycemia
- GIP may account for 60% - 80% postprandial insulin response[2]
- **Mizagliflozin reduces hypoglycemic events by:**
  - Upstream inhibition of glucose absorption
  - Reducing GIP and insulin secretion

1 Nguyen et al. Obesity, 2014
2 Wolfe et al. Endocrine Reviews, 2025

Mizagliflozin for PBH
Clinical Data

Phase 2 Repeat Dose Study 012 for PBH

Study Objective: Confirm doses and regimen for Phase 2b



Note: No run-in period

3 Subjects per Cohort

Three 7-day Treatment Periods with washout

Each subject randomized to placebo and 2 Doses of Miza

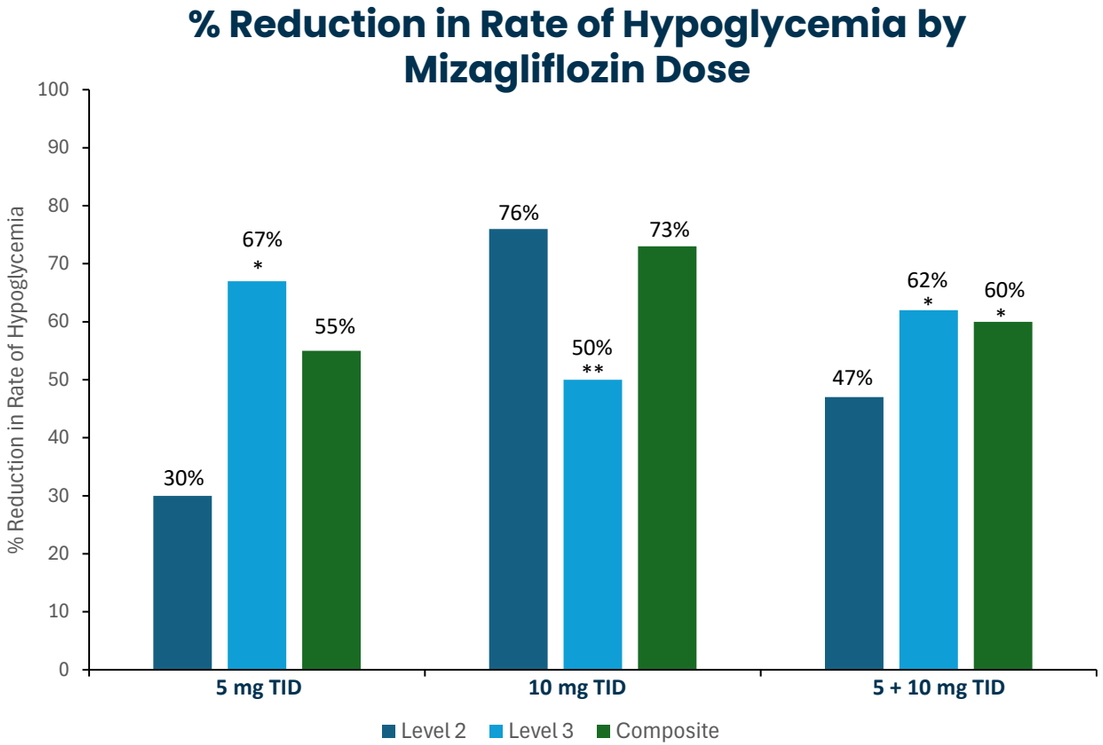
Dose escalation: 0.5 to 10 mg TID

Phase 2 Study Results

- Primary and Secondary endpoints met
- Statistically significant reductions in Level 3 Hypoglycemia Events
- 5 mg & 10 mg TID selected to bring forward into Phase 2b (6 subjects and 3 subjects dosed with 5mg & 10mg respectively)

Phase 2 Repeat Dose Study 012 for PBH

Exploratory Endpoint – Change in Hypoglycemic Events



Study 012: Cross over study with 15 subjects; Blinded, randomized, 7 day placebo period and 2 cross over mizagliflozin doses (both 7 days).

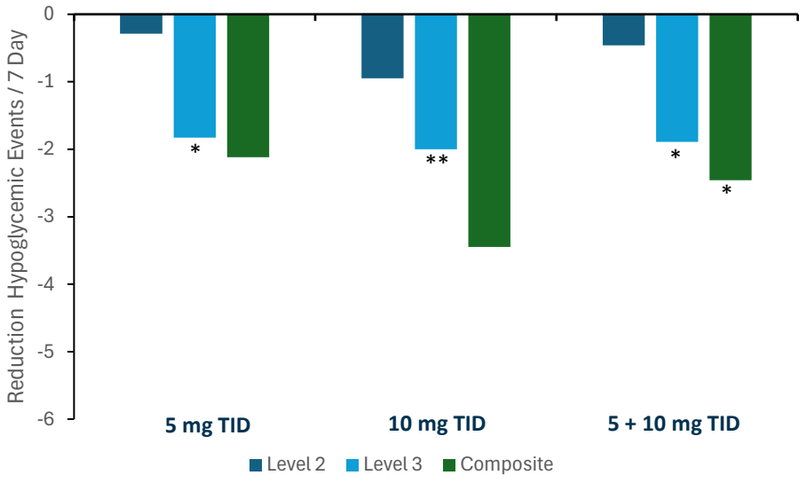
All Subjects dosed with either 5 mg (n=6) or 10 mg (n=3)
*p<0.05
**p<0.10

- Level 2 Hypo Event is defined when at least 2 to 3 consecutive CGM Sensor Glucose values encompassing a 15-minute interval are <54 mg/dL. The end of the event is when 3 subsequent consecutive CGM Sensor Glucose values encompassing a 15-minute interval are ≥ 54 mg/dL. Also defined as a single glucose monitor reading (i.e., fingerstick) at <54 mg/dL.
- Level 3 Hypo Event is defined as a severe event causing the subject to seek medical care or assistance from another person because of the hypoglycemic episode
- FDA guidance that acceptable registration endpoint is either reduction in incidence of Level 3 hypoglycemia events, or a composite of Level 3 and Level 2 events.

Phase 2 Repeat Dose Study 012 for PBH

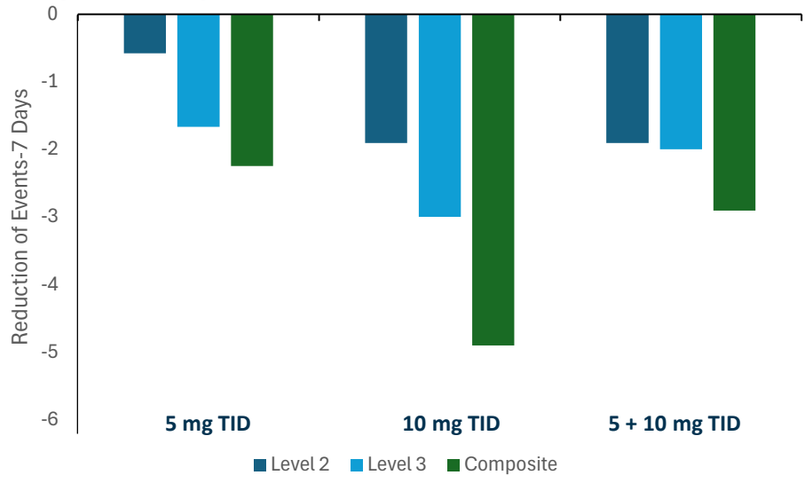
Exploratory Endpoint – Change in Hypoglycemic Events

Reduction of Events
All Subjects Dosed with 5 mg or 10 mg



*p<0.05
**p<0.10

Reduction of Events
5 mg & 10 mg Subjects with Hypo Events on Placebo



	Dose	Reduction of L2 (SE)	p-value	Reduction of L3 (SE)	p-value
Mizagliflozin (012)	5 mg TID	-0.35 (0.38)	0.61	-1.83 (0.60)	0.028
Mizagliflozin (012)	10 mg TID	-1.47 (1.44)	0.50	-2 (0.58)	0.07

- Level 2 Hypo Event is defined when at least 2 to 3 consecutive CGM Sensor Glucose values encompassing a 15-minute interval are <54 mg/dL. The end of the event is when 3 subsequent consecutive CGM Sensor Glucose values encompassing a 15-minute interval are ≥ 54 mg/dL. Also defined as a single glucose monitor reading (i.e., fingerstick) at <54 mg/dL.
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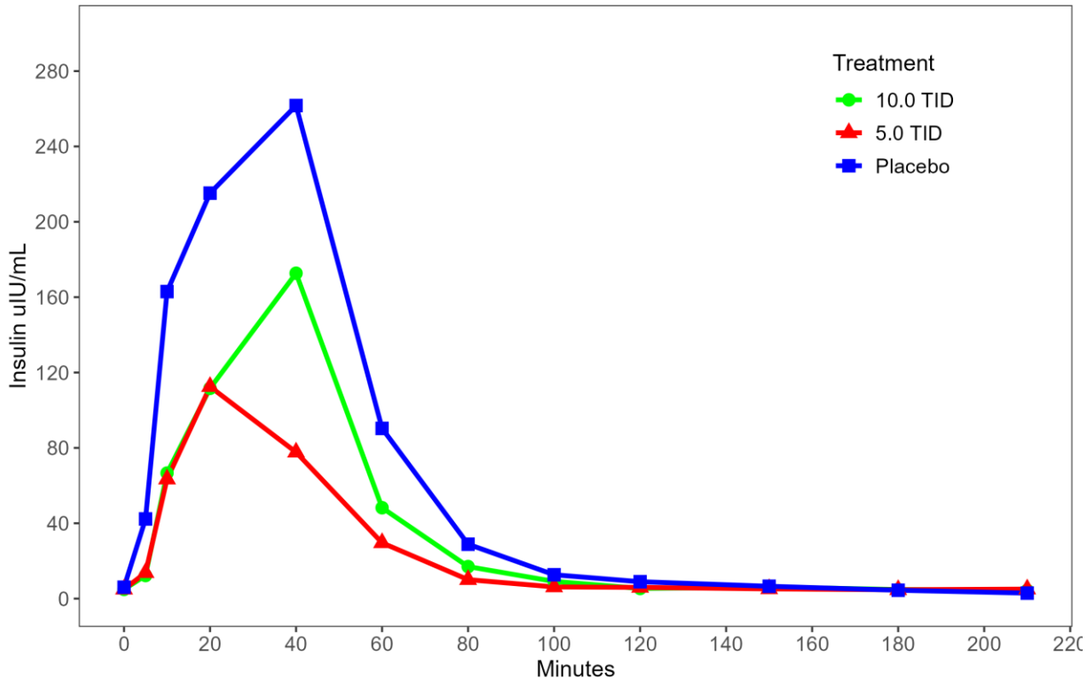
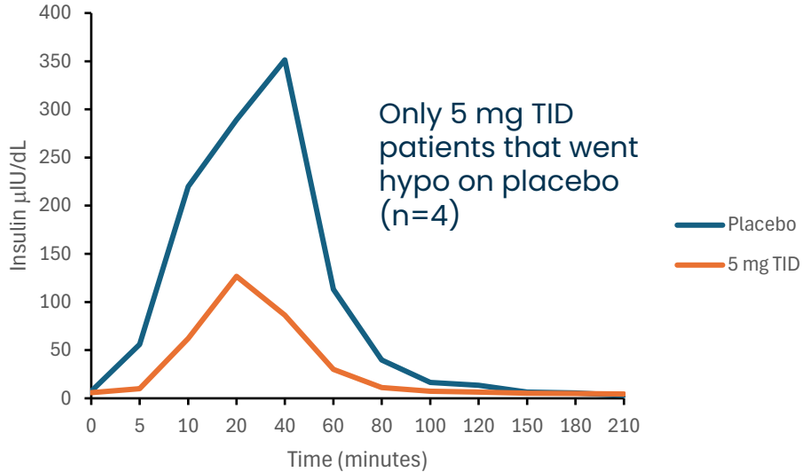
Phase 2 Repeat Dose Study 012 for PBH

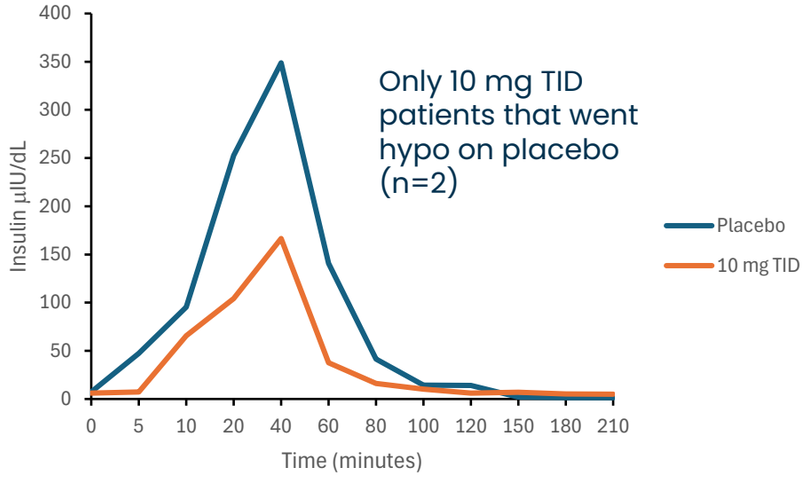
Secondary Endpoint – Change in Insulin vs. Placebo

Insulin Over Time During MMTT

All Subjects dosed with 5 mg TID or 10 mg TID (n=6). Placebo averaged from both 5 mg TID and 10 mg TID subjects



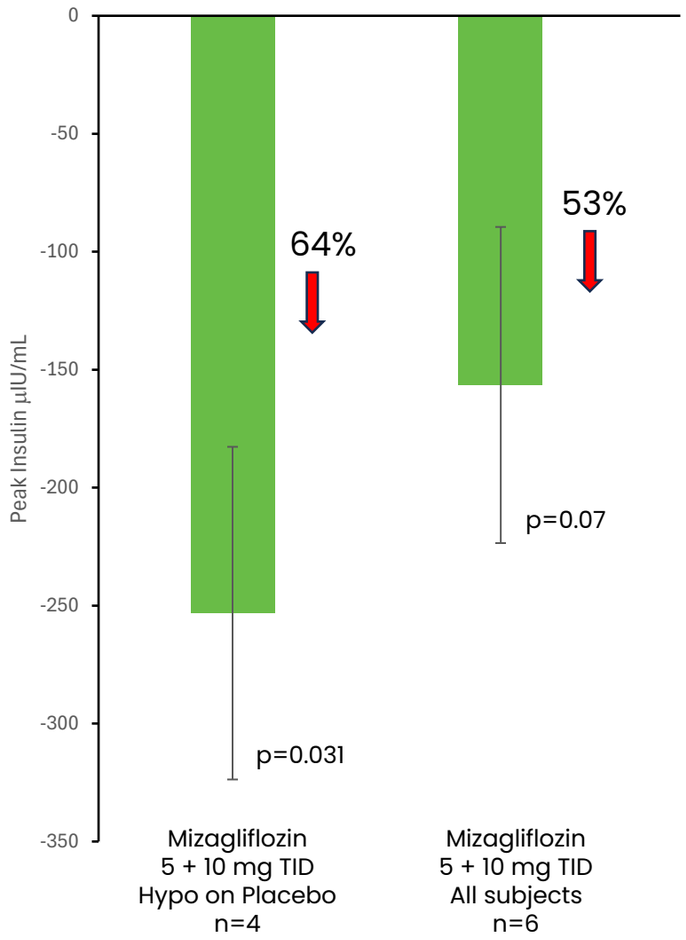




Phase 2 Repeat Dose Study 012 for PBH

Secondary Endpoint – Peak Insulin

Mizagliflozin Markedly Reduces Peak Insulin



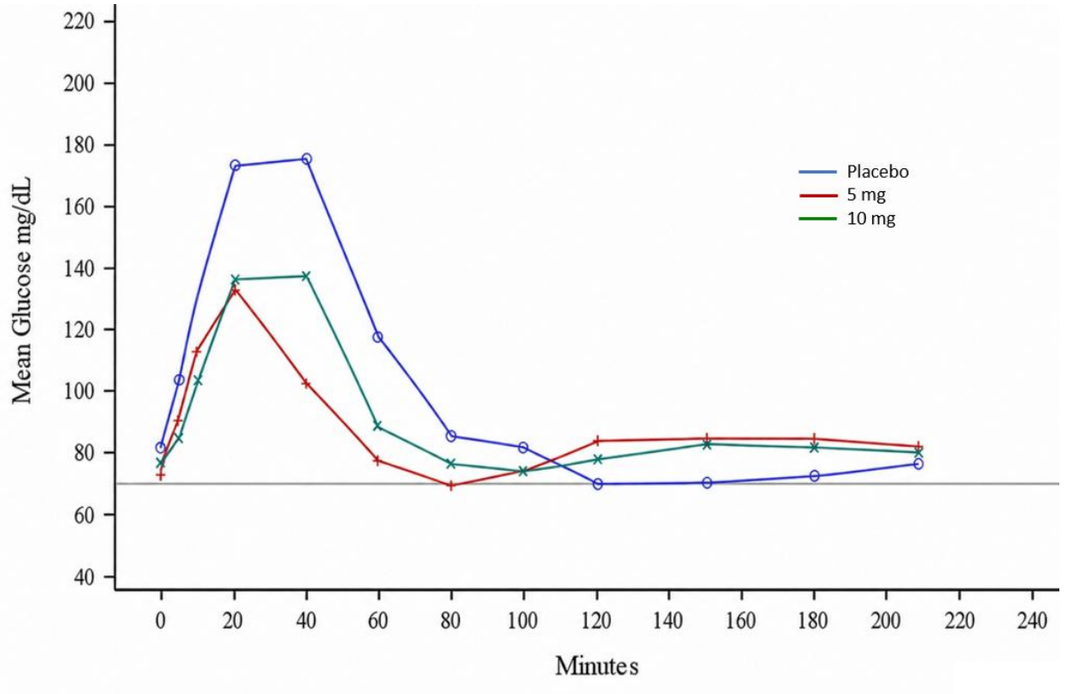
	Dose	Peak Insulin Mean Change from Placebo	Standard Error	p-value
Mizagliflozin (012)	5 + 10 mg TID (hypo on placebo only)	-253.2 (64%)	70.5	0.031
Mizagliflozin (012)	5 + 10 mg TID	-156.5 (53%)	67.0	0.070

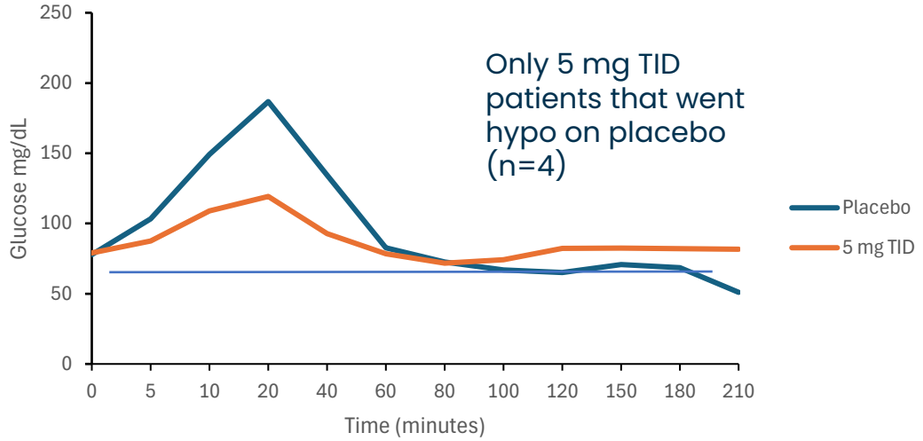
Phase 2 Repeat Dose Study 012 for PBH

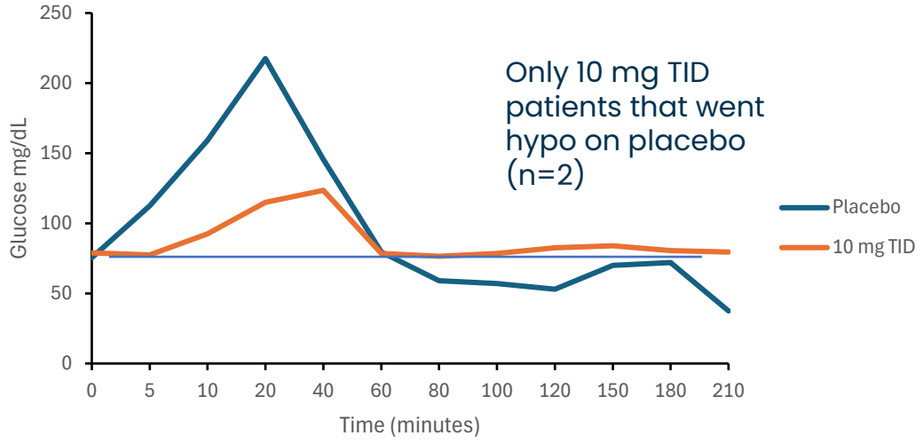
Secondary Endpoint – Peak Glucose: Change from Placebo

Glucose Over Time During MMTT

All Subjects dosed with either 5 mg TID or 10 mg TID (n=6).
Placebo averaged from both 5 mg TID and 10 mg TID subjects.



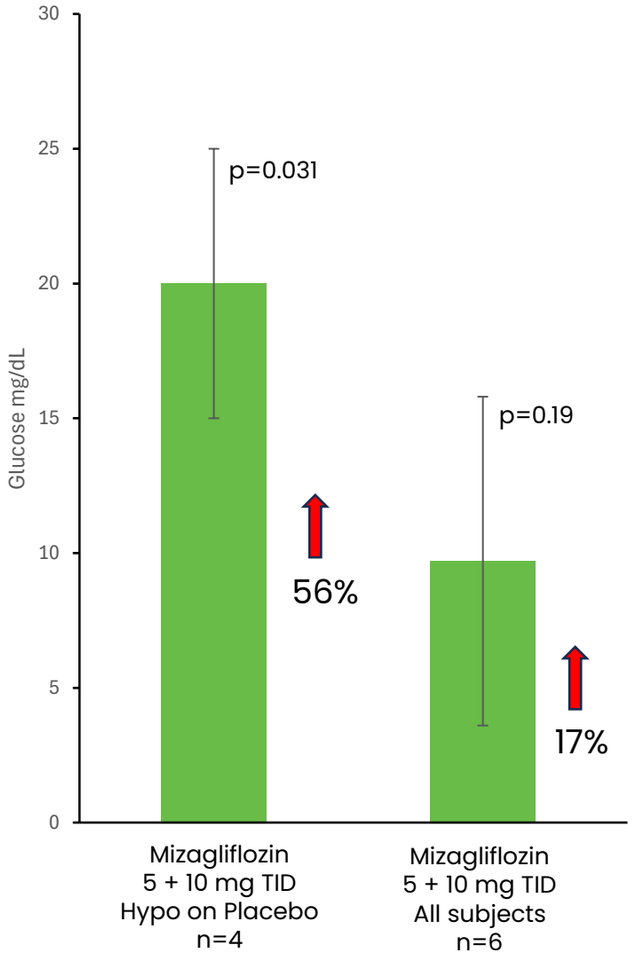




Phase 2 Repeat Dose Study 012 for PBH

Primary Endpoint – Change in Glucose Nadir vs. PBO

Mizagliflozin Increases Glucose Nadir

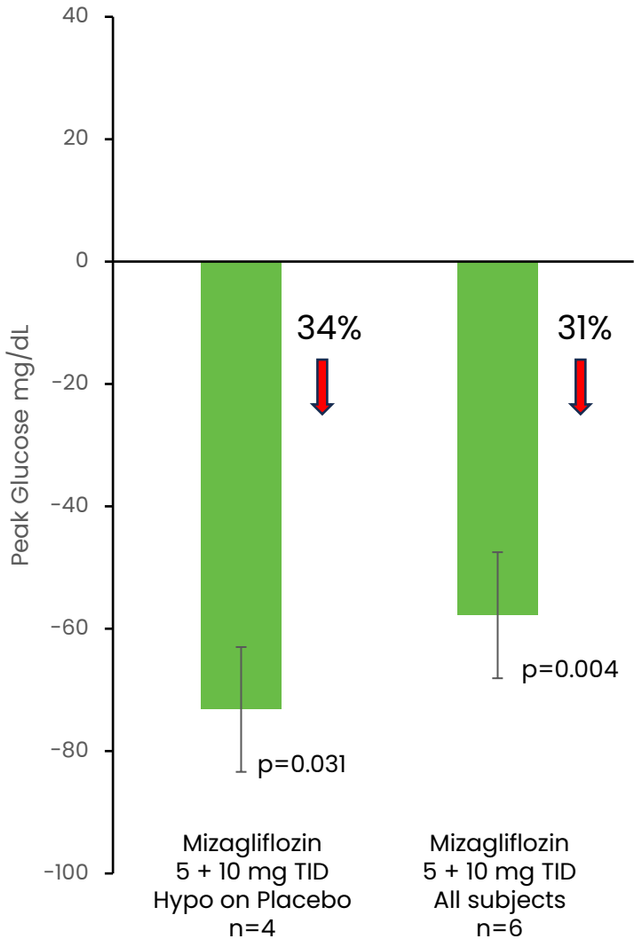


	Dose	Glucose Nadir Mean Change from Placebo	Standard Error	p-value
Mizagliflozin (012)	5 + 10 mg TID (hypo on placebo only)	20 (56%)	5.0	0.031
Mizagliflozin (012)	5 + 10 mg TID	9.7 (17%)	6.1	0.19

Phase 2 Repeat Dose Study 012 for PBH

Secondary Endpoint – Peak Glucose: Change from Placebo

Mizagliflozin Reduces Glucose Peak



	Dose	Glucose Peak Mean Change from Placebo	Standard Error	p-value
Mizagliflozin (012)	5 + 10 mg TID (hypo on placebo only)	-73.2 (34%)	10.2	0.031
Mizagliflozin (012)	5 + 10 mg TID	-57.6 (31%)	10.3	0.004

Reductions in Postprandial Circulating GIP

Reductions in GIP Across Patient Populations

Subject Population	Treatment	GIP Reduction as % of Control
Healthy Subjects	5 mg TID (14 day)	79.0%
Type 2 Diabetics	5 mg BID (6 day)	59.0%
PBH Patients	5 mg (single dose)	45.2%

- Postprandial GIP secretion in the gut stimulates insulin secretion
- GIP may account for 60% - 80% postprandial insulin response[1]
- Blocking or reducing GIP lowers insulin secretion
- Mizagliflozin reduces circulating GIP (45.2% decrease from baseline in PBH patients) providing a complementary effect to reductions in glucose absorption

1 (Wolfe et al. Endocrine Reviews, 2025)

Phase 2 Repeat Dose Study 012 for PBH

Adverse Events

	Placebo (N=15) n (%)	5 mg TID (N=6) n (%)	10 mg TID (N=3) n (%)	Total Active (N=15) n (%)
Subjects with any AEs	7 (46.7)	5 (83.3)	1 (33.3)	15 (100.0)
Subjects with any TEAEs	7 (46.7)	5 (83.3)	1 (33.3)	15 (100.0)
TEAEs by maximum intensity CTCAE grade				
Mild	4 (26.7)	2 (33.3)	0	10 (66.7)
Moderate	0	3 (50.0)	1 (33.3)	4 (26.7)
Severe	0	0	0	1 (6.7)
Life-threatening	0	0	0	0
Death	0	0	0	0
Related TEAEs	3 (20.0)	3 (50.0)	1 (33.3)	11 (73.3)
Subjects with a TEAE leading to study drug discontinuation	0	0	0	0
Subjects with a TEAE leading to early withdrawal from study	0	0	0	0
Subjects with a serious TEAE	0	0	0	1 (6.7)
Subjects with a TEAE with fatal outcome	0	0	0	0
Subjects with Drug-Related Treatment-Emergent Adverse Events by System Organ Class and Preferred Term				
Gastrointestinal disorders	3 (20.0)	3 (50.0)	1 (33.3)	11 (73.3)
Diarrhea	3 (20.0)	2 (33.3)	1 (33.3)	10 (66.7)
Abdominal distension	0	1 (16.7)	0	1 (6.7)
Abdominal pain	0	1 (16.7)	0	1 (6.7)
Gastrointestinal pain	0	1 (16.7)	0	1 (6.7)
Reproductive system and breast disorders	0	1 (16.7)	0	1 (6.7)
Dysmenorrhea	0	1 (16.7)	0	1 (6.7)

N = number of subjects, n = number of events
All AE were diarrhea except one subject (5 mg) with AE associated with menstruation
One SAE (bradycardia with severe hypotension-1 mg TID) was unrelated to drug

Mizagliflozin for PBH – Clinical Development Plan

Phase 2b – EMERGE Study

- Multicenter, randomized, double-blind, placebo-controlled, repeat dose parallel group study
- 21-day run in + 28-day treatment period
- Primary Endpoint: Decrease composite of Level 3 and Level 2 hypoglycemic events (patient reported & SMBG)
- Secondary Endpoints: Tolerability

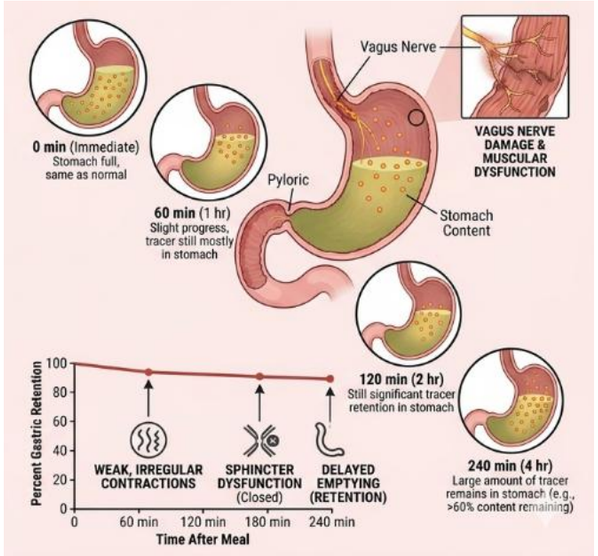
Phase 3

- Possibility for single Phase 3
- Randomized, double-blind, placebo-controlled six month treatment + six month open-label treatment
- Endpoints: Decrease Level 3 and Level 2 hypoglycemic events (patient reported and SMBG)
- Secondary Endpoints: Tolerability

Mizagliflozin for Gastroparesis

Gastroparesis - Overview

- Gastroparesis can be a chronic health problem with no effective long-term treatment
- The cause of gastroparesis is generally unknown, but most commonly is a complication related to diabetes, certain surgeries and some medications (GLP-1 agonists)
- Gastroparesis (aka delayed gastric emptying or stomach paralysis) is a disorder that slows or stops the digestive process and disrupts normal bowel movement activity
- Potential multi-billion-dollar addressable market

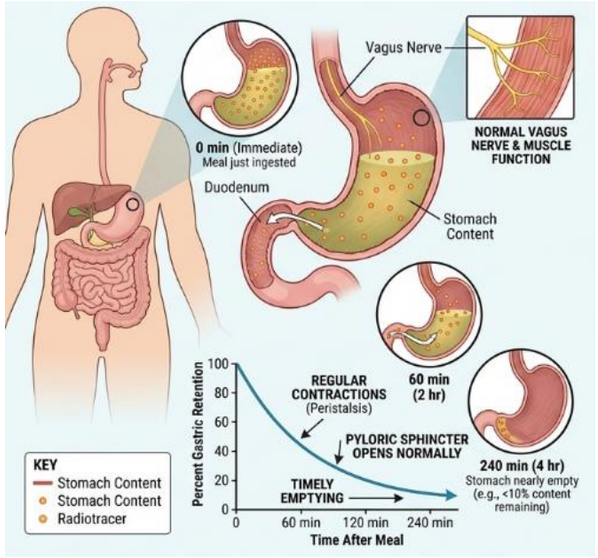


Gastroparesis (Delayed Gastric Emptying)

Symptoms Include:

- Nausea and Vomiting
- Abdominal pain
- Acid reflux
- Lack of appetite
- Overall impaired quality of life
- Bloating
- Feeling full after only a few bites of food
- Changes in blood sugar levels
- Weight loss and malnutrition

Normal Gastric Emptying



Mizagliflozin for Gastroparesis - Rationale

Gastroparesis is associated with

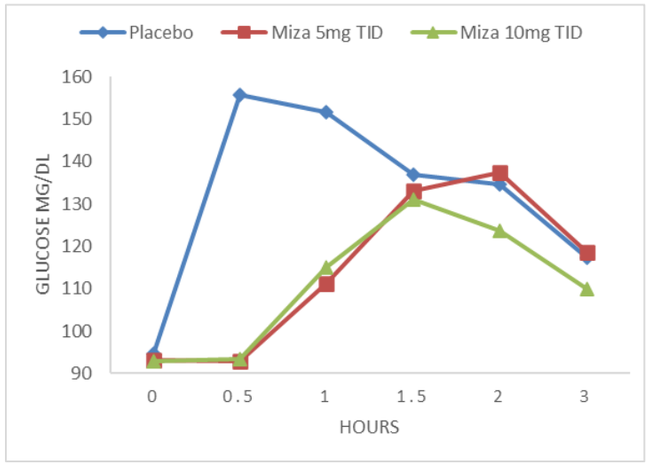
- Elevated fasting and post-prandial GIP concentrations[1]
- Acute postprandial hyperglycemia slows gastric emptying[2]
- Reduced GI transit time and higher rates of slow transit constipation[3]
- Mizagliflozin has been shown to reduce postprandial circulating GIP in healthy volunteers, patients with T2DM and patients diagnosed with PBH
- Mizagliflozin has been shown to reduce postprandial hyperglycemia in healthy volunteers, patients with T2DM and patients diagnosed with PBH
- Mizagliflozin increases hydration of the GI tract, increases GI transit, and is effective relieving chronic idiopathic constipation
- **We believe that all three of these actions will positively impact gastroparesis outcomes**

1 (Prevost et al. Neurogastroenterol 2013, 25(8):694)
2 (Marathe et al. Diabetes Care 2013, 30:1396)
3 (J Neurogastroenterol Motil. 2019 Apr; 25(2): 267–275)

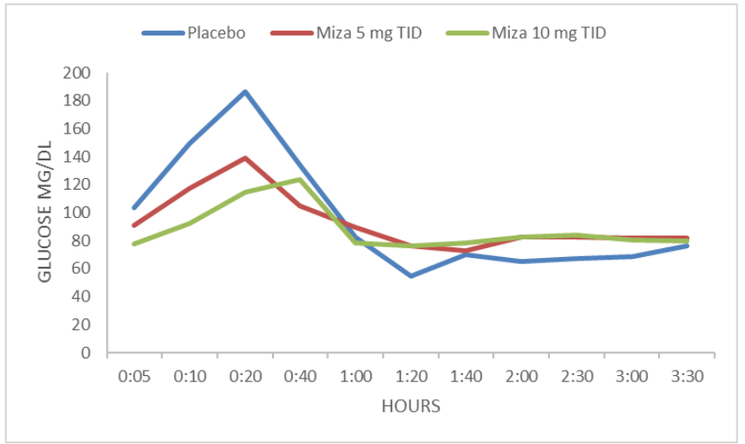
Mizagliflozin for Gastroparesis

Reductions in Postprandial Plasma Glucose & Effective in Chronic Constipation

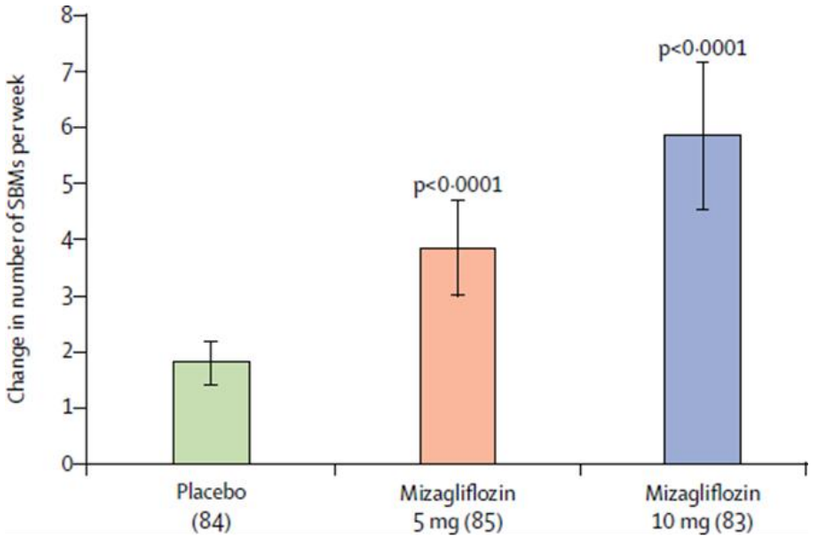
Healthy Human Subjects



PBH Patients



Mizagliflozin relieves constipation and hydrates the GI tract



Fukudo et al Lancet Gastroenterol Hepatol 2018; 3: 603–13

Mizagliflozin for Gastroparesis

Phase 2 POC Study Design

- Randomized, parallel group, double-blind, placebo-controlled study comparing different doses of mizagliflozin on gastric emptying time in patients with gastroparesis
- 28-day run-in + 28-day treatment period
- Primary endpoint: Change from baseline in gastric emptying rate as determined by Gastric Emptying Breath Test (GEBT)* after 28 day administration of mizagliflozin or placebo
- Secondary endpoint: Tolerability/PK, symptom relief (ANMS GSCI-DD**)

*https://www.uspharmacist.com/article/diagnostic-spotlight-gastric-emptying-breath-test
**User Manual for the ANMS GCSI-DD (The American Neurogastroenterology and Motility Society Gastroparesis Cardinal Symptom Index Daily Diary)

Gastroparesis – Competitive Landscape

Competitive Landscape

Single FDA approved product (generic)

- *Metoclopramide (Reglan)*
- Gastroparesis label limited to diabetic gastroparesis
- 10 mg orally/IV/IM four times daily
- Black box warning for tardive dyskinesis resulting in maximum permitted treatment of 12 weeks
- Metoclopramide appears to bind to dopamine D2 receptors with nanomolar affinity (Ki = 28.8 nM) where it is a receptor antagonist and is also a mixed 5-HT3 receptor antagonist/5-HT4 receptor agonist

Products used off-label with varying levels of success:

Company	Product	Approved Indication	Rationale	Mechanism
Abbvie	Linaclotide	Constipation	GI Tract Hydration	Oligo-peptide agonist
Takeda	Lubiproston	Constipation	GI Tract Hydration	CIC-2 chloride channel inhibitor
Takeda	Prucalopride	Constipation	Colonic motility stimulation	5-HT_4 receptor agonist
Generic	Erythromycin	Antibiotic	Colonic contraction stimulation	Motilin agonist

Competitor Products in development for Gastroparesis:

Company	Asset	Phase	Mechanism
Vanda	Tradipant	CRL	Antagonists of tachykinin receptor ($TACR_1$, also called NK1R)
Falk Pharma GmbH	Naronapride	Phase 2	Serotonin type 4 ($_5HT_4$) and dopamine-2 receptor (D_2)
CINRx Pharma	CIN-102	Phase 2	Dopamine D_2/D_3 receptor agonist
Neurogastrx	NG101	Phase 2	Dopamine D_2 receptor antagonist

Vogenx