Vogenx

Investor Presentation

Phase 2b Clinical Stage Company
Post Bariatric Hypoglycemia & Gastroparesis

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Corporate Overview

METABOLIC & GASTROINTESTINAL

Two indications with potential multi-billion-dollar addressable markets

Post Bariatric Hypoglycemia (PBH)

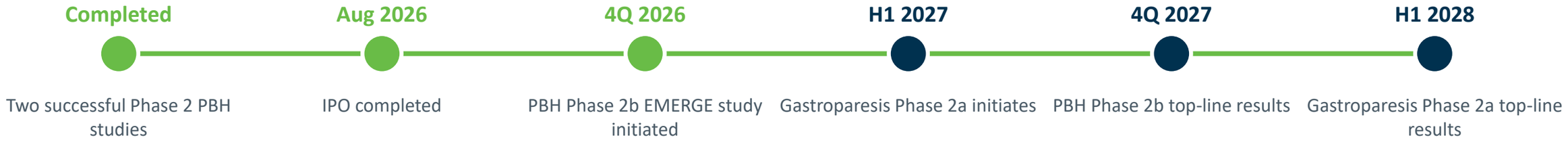
No FDA-approved therapeutics

Two successful Phase 2 studies completed; Phase 2b EMERGE study initiated Q4, 2026

Gastroparesis

One FDA-approved option exists; boxed warning

Phase 2a study planned to start H1 2027, with top-line results expected H1 2028.



2028

Cash runway through 2028

Multi-billion

Both indications represent large addressable markets

Raleigh, NC

Founded in 2021

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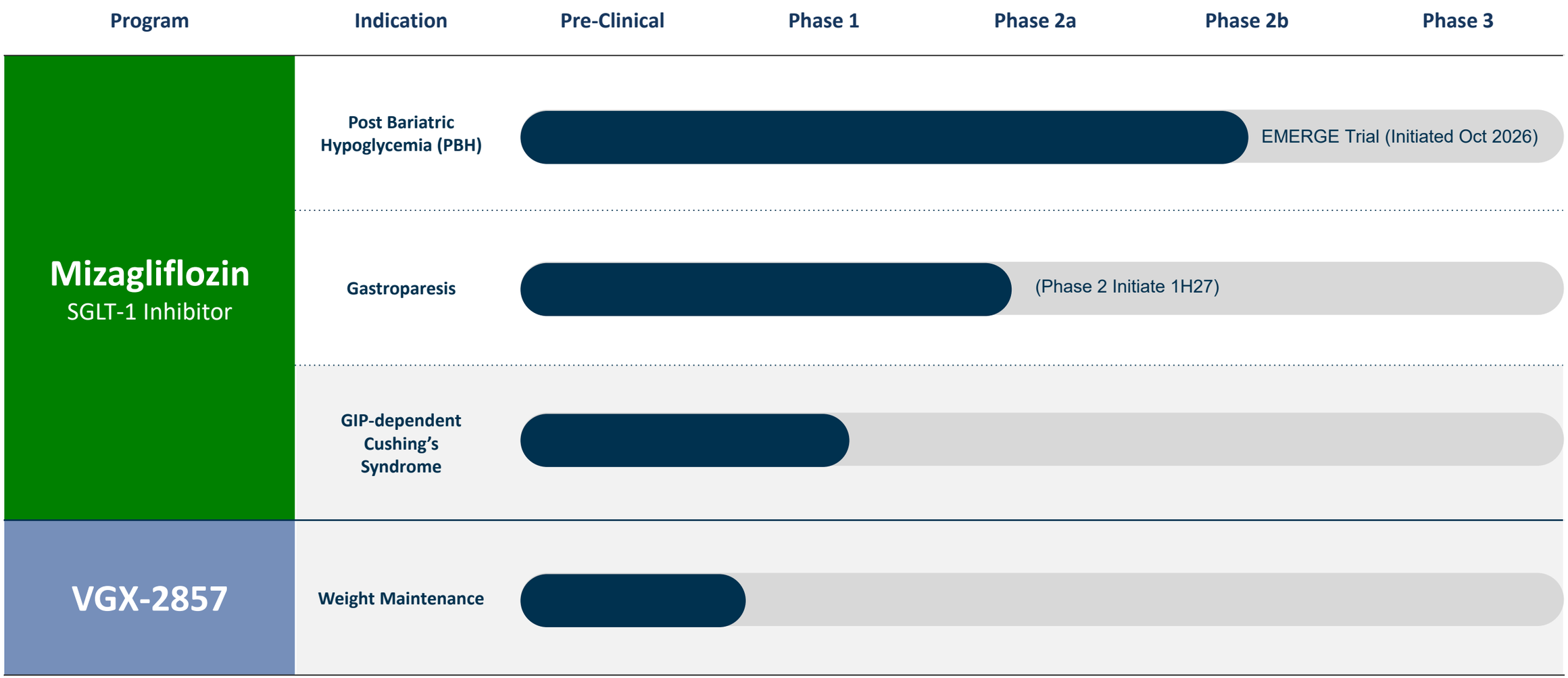
Pipeline


Program
Indication
Pre-Clinical
Phase 1
Phase 2a
Phase 2b
Phase 3
Mizagliflozin
SGLT-1 Inhibitor
Post Bariatric Hypoglycemia (PBH)
EMERGE Trial (Initiated Oct 2026)
Gastroparesis
(Phase 2 Initiate 1H27)
GIP-dependent Cushing's Syndrome
VGX-2857
Weight Maintenance

Mizagliflozin Overview

Novel Orally Dosed Small Molecule Inhibitor
- Target: SGLT-1 locally in small intestine (gut restricted)
- Mechanism of Action: Inhibition of glucose absorption and reductions in secretion of GIP and insulin

Safety
- Observed to be well tolerated in Phase 2 studies
- No drug-related significant adverse events
- Pre-clinical package complete (including 2-year carcinogenicity in rats)

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 Hypoglycemic (PBH)

Indication Summary

- ✓ Chronic disorder related to bariatric surgery, characterized by reactive postprandial hyperinsulinemic hypoglycemia
- ✓ Spectrum disease with varying severity and frequency of events
- ✓ Above-average rates of hospitalization, medical burden, and meal-related anxiety
- ✓ No FDA-approved therapeutics; standard of care is diet modification

Prevalence (U.S.)

~3 million
living bariatric surgery patients (+~200K surgeries/yr)

25–75%
exhibit PBH on continuous glucose monitoring

~500,000
symptomatic PBH patients

~200,000
require daily medical management

~300,000
less severe / frequent: daily or PRN treatment

HIDDEN SEGMENT

>25%

of bariatric surgery patients have asymptomatic, hypo-unaware PBH

Prevalence estimates likely understate a multi-billion-dollar addressable market

PBH = post-bariatric hypoglycemia; PRN = as needed. Sources: Øhrstrøm CC et al. 2019; Tan et al. 2020.

PBH – Competitive Landscape

Company	Product Candidate	Drug Substance	Target Mechanism	Target Distribution	Development Status	Administration	Reduce Glucose Absorption	Reduce GIP	Reduce Insulin Secretion
Vogenx	**Mizagliflozin**	**Small Molecule**	**SGLT-1 Inhibitor**	**Gut Restricted**	**Phase 2b**	**Oral**	✓	✓	✓
Amylyx	Avexitide	Peptide	GLP-1 Antagonist	Systemic	Pre-NDA	Injectable	✗	✗	✓
Recordati	Pasireotide	Peptide	Somatostatin Analog	Systemic	Phase 2	Injectable	✗	✓	✓
Rezolute	Ersodetug	biologic	Insulin receptor modulator	Systemic	Phase 2	Injectable	✗	✗	✓

✓ **Oral Administration**

- Oral small molecule preferred by patients; lower supply chain costs
- Meal adjacent oral regimen reduces substantial anxiety associated with meals and allows for addressing all segments of the PBH market
- Severe Patients: prior to each meal; Moderate to Mild Patients: PRN prior to stimuli that could reasonably lead to event

✓ **Mechanism: Upstream Glucose and GIP**

- PBH hypo events correlate strongly to upstream glucose absorption; bariatric surgery patients upregulate SGLT-1 post-surgery (primary transporter of prandial glucose)
- Miza targets PBH events by inhibiting upstream glucose absorption via SGLT-1; GLP-1 antagonists work downstream by disrupting binding at the GLP-1 receptor
- SGLT-1 inhibition reduces postprandial glucose and GIP, with an effect on insulin that appears superior to GLP-1 antagonism

PBH – Competitive Landscape

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

Broad Commercial Positioning

- ✓ 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, SGLT-2 and absorbable SGLT-1 inhibitors inhibit glucose reabsorption in renal filtrate, which does not provide upstream relief to PBH patients

POST BARIATRIC HYPOGLYCEMIA (PBH)

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]

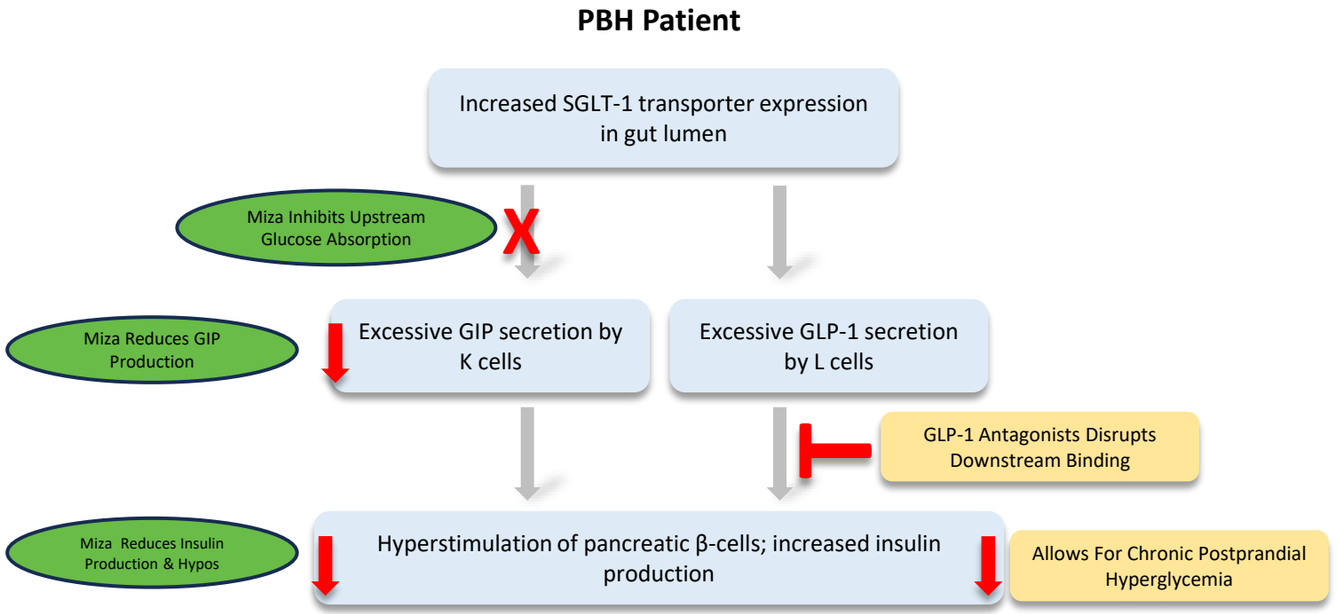
Excess Glucose and Resulting Hyperinsulinemia Leads to Hypoglycemia

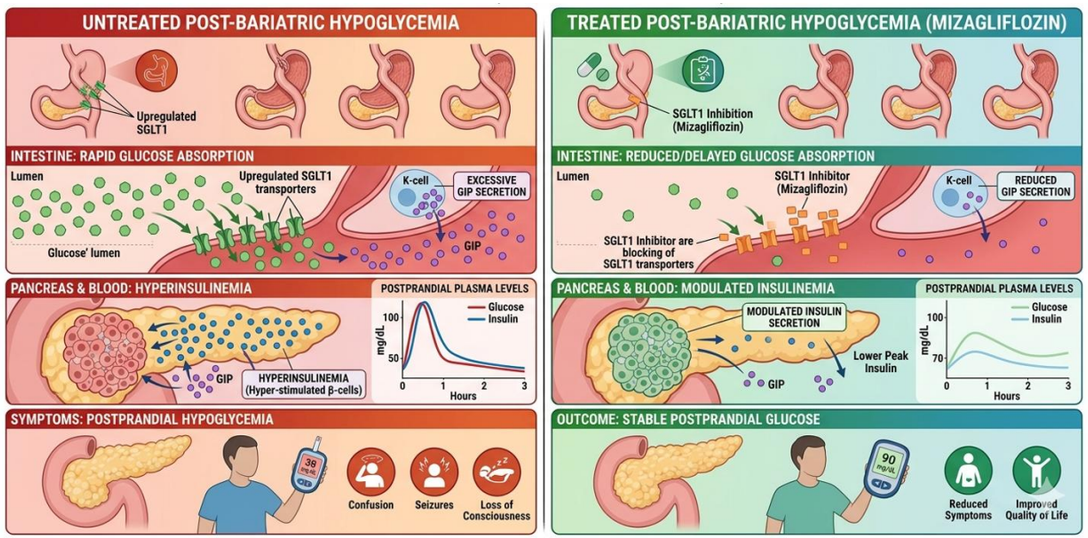
Rapid excess absorption of glucose via upregulated SGLT1.

Hyperinsulinemia caused by excess glucose absorption.

Charts adapted from Salehi et al, 2014 | Sources: [1] Øhrstrøm CC et al, 2019; [2] Tan et al, 2020

PBH Mechanism: Upstream vs. Downstream





WHY TARGET SGLT1

SGLT-1 is the primary transporter of glucose from the GI tract into the bloodstream and is overexpressed in bariatric surgery patients

PBH patients have elevated SGLT-1 expression which leads to abnormally high levels of absorbed glucose and subsequently increased GIP secretion[1]

MIZAGLIFLOZIN'S UPSTREAM EFFECT

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 inhibiting glucose absorption upstream and reducing GIP & insulin secretion

1 Nguyen et al. Obesity, 2014

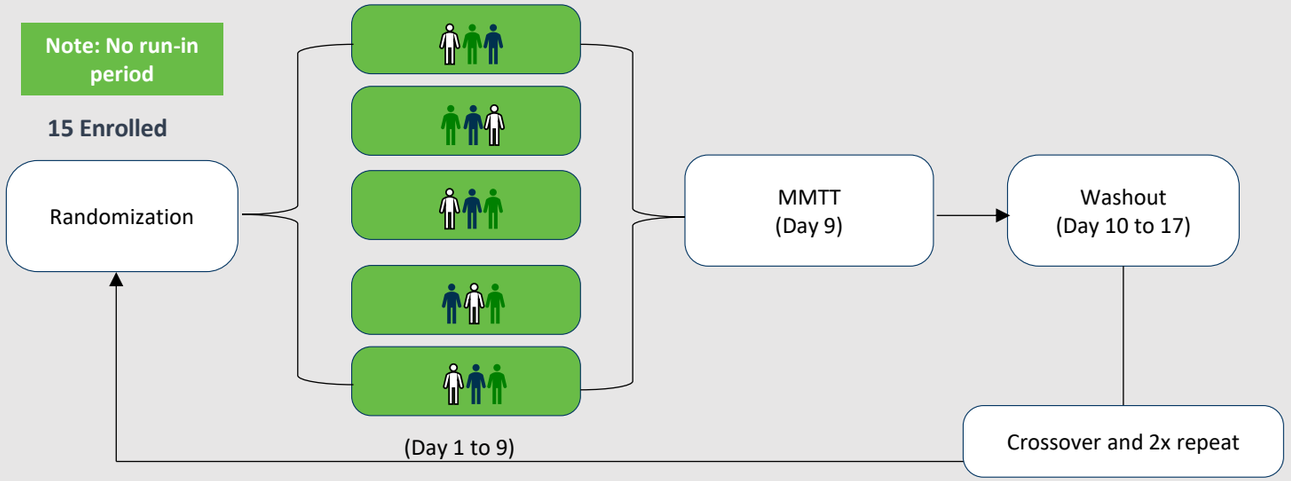
2 Wolfe et al. Endocrine Reviews, 2025

Mizagliflozin for PBH
Clinical Data

Phase 2 Repeat Dose Study 012 for PBH

Study Objective: Confirm doses and regimen for Phase 2b

Randomized, placebo-controlled, crossover, single-blind, repeat dose study



What was tested?

- 5 cohort X 3 subjects
- Each cohort has placebo, low dose and high dose
- 5 doses: 0.5 mg to 10 mg
- 3 regimens: QD, BID, TID

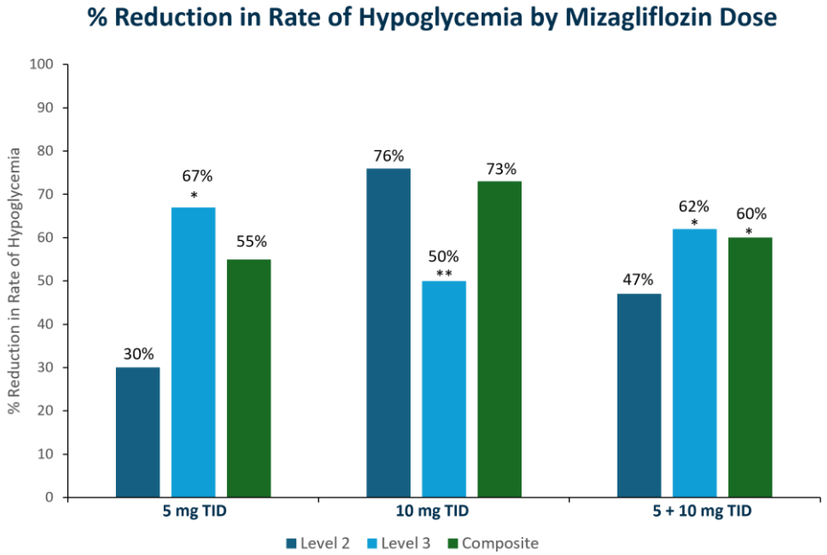
= placebo
= low dose
= high dose

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)

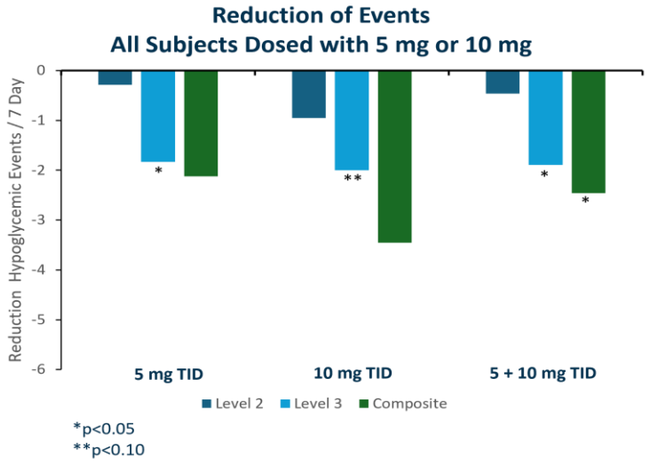
Phase 2 Repeat Dose Study 012 for PBH

Change in Hypoglycemic Events



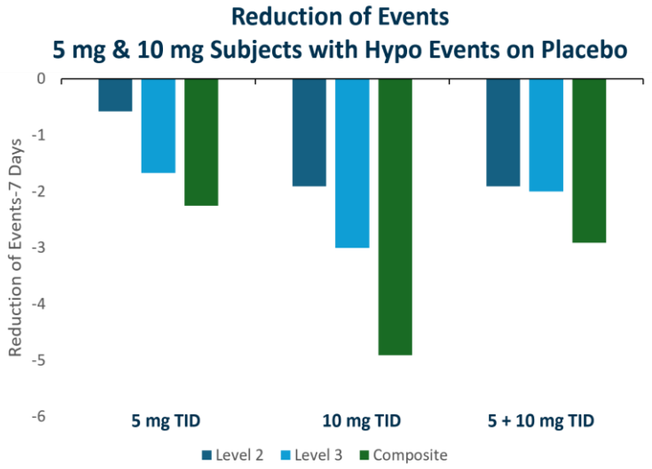
All Subjects dosed with either 5 mg (n=6) or 10 mg (n=3)
*p<0.05
**p<0.10

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



*p<0.05
**p<0.10

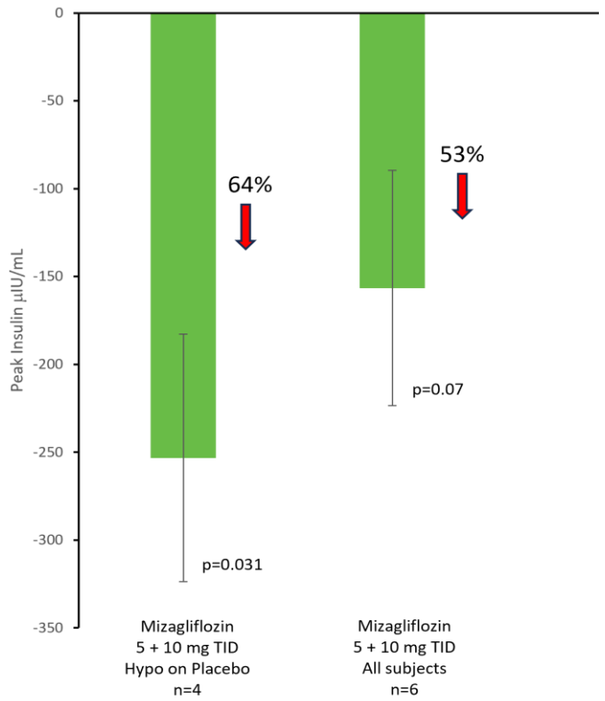
	Dose	Reduction of L2 (SE)	p-value	Reduction of L3 (SE)	p-value
Mizagliflozin (012)	5 mg TID	-0.35 (0.38)	0.61	-1.83 (0.60)	0.028
Mizagliflozin (012)	10 mg TID	-1.47 (1.44)	0.50	-2 (0.58)	0.07



Phase 2 Repeat Dose Study 012 for PBH

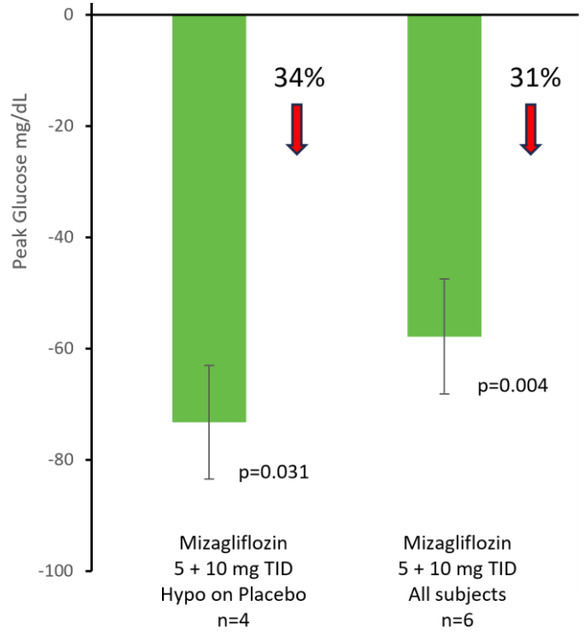
Pharmacodynamics

Reductions in Peak Insulin



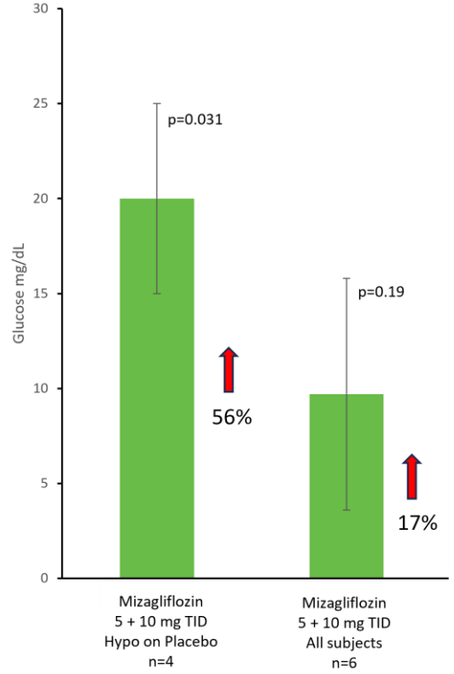
	Dose	Peak Insulin Mean Change from Placebo	Standard Error
Mizagliflozin (012)	5 + 10 mg TID (hypo on placebo only)	-253.2 (64%)	70.5
Mizagliflozin (012)	5 + 10 mg TID	-156.5 (53%)	67.0

Reductions in Peak Glucose



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

Improvement in Glucose Nadir



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

Reductions in Postprandial Circulating GIP

Reductions in GIP Across Patient Populations

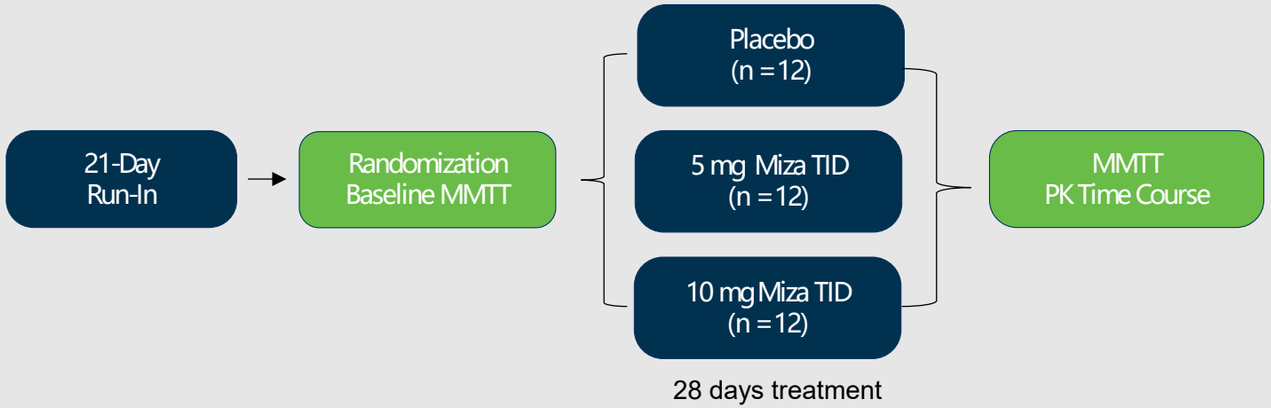
Subject Population	Treatment	GIP Reduction as % of Control
Healthy Subjects	5 mg TID (14 day)	79.0%
Type 2 Diabetics	5 mg BID (6 day)	59.0%
PBH Patients	5 mg (single dose)	45.2%

- 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)

Mizagliflozin for PBH – Clinical Development Plan

Phase 2b – EMERGE Study



Primary Endpoint:

Composite change Level 2 (SMBG) and Level 3 (adjudicated) hypoglycemic events

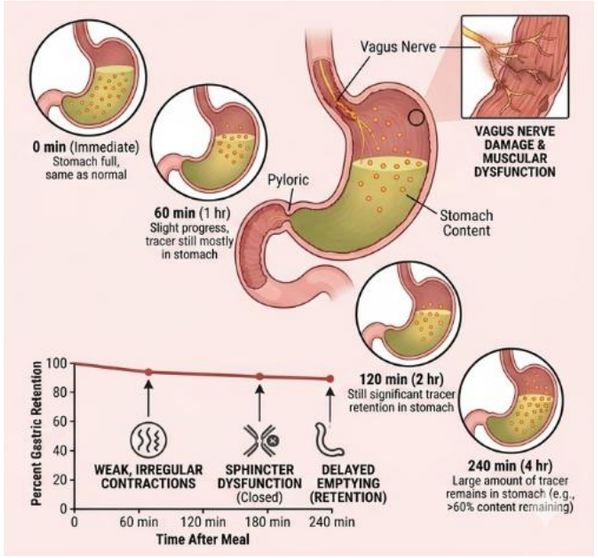
Phase 3

- Possibility for single Phase 3
- International, randomized, double-blind, placebo-controlled treatment + open-label treatment (12 months)
- Endpoint: Decrease Level 2 and Level 3 hypoglycemic events (SMBG & adjudicated)
- Secondary Endpoints: Tolerability

Mizagliflozin for Gastroparesis

Gastroparesis - Overview

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

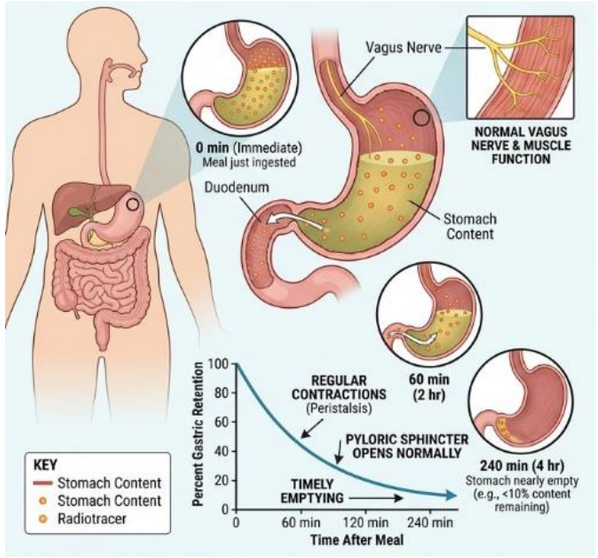


Gastroparesis (Delayed Gastric Emptying)

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

Each core feature of gastroparesis is matched by a demonstrated effect of mizagliflozin

		GASTROPARESIS IS ASSOCIATED WITH	MIZAGLIFLOZIN HAS BEEN SHOWN TO	EXPECTED IMPACT
1	**Incretin (GIP)**	Elevated fasting and postprandial GIP concentrations[1]	**Reduce postprandial circulating GIP** Blocks SGLT1-mediated glucose uptake that triggers GIP release	**Normalize excess GIP signaling**
2	**Glycemic**	Acute postprandial hyperglycemia slows gastric emptying[2]	**Reduce postprandial hyperglycemia** Slows intestinal glucose absorption, blunting the post-meal spike	**Remove a glycemic brake on gastric emptying**
3	**Motility**	Slowed GI transit and higher rates of slow-transit constipation[3]	**Increase GI hydration and transit** Unabsorbed glucose draws water into the lumen; effective in chronic idiopathic constipation	**Accelerate GI transit and ease constipation**

Effects demonstrated in healthy volunteers, patients with T2DM, and patients diagnosed with PBH

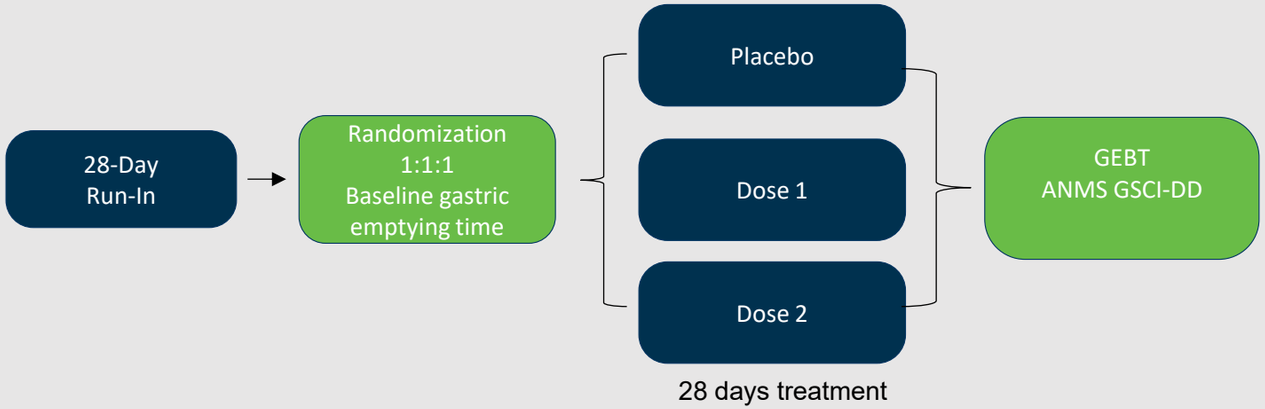
One oral agent, three complementary actions: we believe mizagliflozin will positively impact gastroparesis outcomes

1 Prevost et al. Neurogastroenterol Motil 2013, 25(8):694
2 Marathe et al. Diabetes Care 2013, 36:1396
3 J Neurogastroenterol Motil 2019 Apr; 25(2):267–275

Mizagliflozin for Gastroparesis

Phase 2 POC Study Design

Randomized, parallel group, double-blind, placebo-controlled study



Phase 2 POC Study

- Randomized, double-blind, placebo-controlled treatment for 28 days
- Primary Endpoint: Gastric emptying breath test
- Secondary Endpoints: Symptom relief (ANMS GSCI-DD**)

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

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