Innovating GPCR-Targeted Therapies to Reach Large Untapped Market Opportunities

FEBRUARY 2026

TECTONIC Therapeutic

DISCLAIMER

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These forward-looking statements reflect our current beliefs and expectations. Many factors may cause differences between current expectations and actual results, including the early stage of our development efforts; success in preclinical testing and earlier clinical trials does not ensure that later clinical trials will generate the same results or otherwise provide adequate data to demonstrate the efficacy and safety of a product candidates; clinical site activation rates or clinical trial enrollment rates that are lower than expected; changes in expected or existing competition; changes in the regulatory environment; the uncertainties and timing of the regulatory approval process; the impact of macroeconomic conditions, including the conflict in Ukraine and the conflict in the Middle East, heightened inflation and uncertain credit and financial markets, on our business, clinical trials and financial position; and unexpected litigation or other disputes. These and other risks are described more fully in our filings with the Securities and Exchange Commission (“SEC”), including the risks detailed in our Annual Report on Form 10-K filed with the SEC on February 26, 2026, and other documents we subsequently filed with or furnished to the SEC. All forward-looking statements contained in this presentation speak only as of the date on which they were made. Except as required by law, we assume no obligation to update any forward-looking statements contained herein to reflect any change in expectations, even as new information becomes available.

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Tectonic Tx: GPCR-Targeted Therapies for High-Value Opportunities

Clinical-Stage Biotech	**TECX focused on discovery & development of GPCR-target biologics with significant unmet need** • Founded in 2019 by Tim Springer and Andrew Kruse
Tenured Team	**Executive team with numerous accomplishments, resulting in 20 "first" approvals**
TX45	**Long-acting relaxin in Phase 2 trial, Phase 1 results support best-in-class potential** • Initial indication targeting Group 2 Pulmonary Hypertension (PH) associated with Heart Failure with Preserved Ejection Fraction (HFpEF), or PH-HFpEF, with Phase 2 trial enriched for CpcPH (combined pre- and post-capillary PH) • Potential to expand into PH-HFrEF, addressable patient population of ~1.1M in the U.S. with ~300K CpcPH (PVR≥3)
Relaxin Potentially Ideal for PH-HFpEF	**Relaxin physiologic and hemodynamic effects demonstrated preclinically and in Phase 1b study** • Positive Phase 1b trial results achieved or exceeded all hemodynamic targets, supporting Phase 2 study
PH-HFpEF Significant Market Potential	**~1.4M+ Group 2 PH-HFpEF patients in the U.S. with no approved therapy*; high 5-year mortality** • Potential peak multi-billion-dollar* revenue potential for Group 2 PH-HFpEF patients with EF ≥ 40% • AstraZeneca's oral relaxin program targeting chronic heart disease patients is ongoing
Potential to Expand TX45 to PH-ILD	**TX45 PH-ILD (PH associated with Interstitial Lung Disease) Phase 2 trial is open for screening** • Positive PH-ILD (WHO Group 3) Phase 2 results could potentially expand TX45 into a new $1B+ indication
TX2100	**Potential first-in-class APJ antagonist designed to treat Hereditary Hemorrhagic Telangiectasia (HHT)** • HHT is a rare bleeding disorder of dysregulated angiogenesis leading to bleeding, anemia arteriovenous malformations • Significant market potential, no approved therapies for HHT, estimated ~75K patients in the U.S. alone (15-20% severe) • TX2100 Phase 1a healthy volunteer clinical trial randomized first subject in Feb '26, expect topline results Q4'26
Well-Capitalized	**Cash runway into Q4'28 with $253.8 million in cash and cash equivalents as of 12/31/25**

** Estimates based on company sponsored market analysis conducted by Health Advances*

This Accomplished Team Has Delivered for Patients and Investors

Alise Reicin, M.D.
CEO, Director

Celgene · MERCK · Columbia University · KEYTRUDA® (pembrolizumab) Injection 100 mg

Daniel Lochner
CFO

VIATRIS · OYSTER POINT · Goldman Sachs · Columbia University

Peter McNamara, Ph.D.
CSO

NOVARTIS · GNF · Penn · BRAFTOVI® (encorafenib) 75 mg capsules

Anthony Muslin, M.D.
CDO

NOVARTIS · SANOFI · Washington University in St. Louis School of Medicine · Entresto® (sacubitril/valsartan) tablets 24/26mg · 49/51mg · 97/103mg

Marcella Ruddy, M.D.
CMO

REGENERON · MERCK · Washington University in St. Louis School of Medicine · DUPIXENT® (dupilumab) Injection 200mg · 300mg

Marc Schwabish, Ph.D.
CBO

Fusion · BAYER · Eisai · LEERINK SWANN · RBS

Timothy Springer, Ph.D.
Co-Founder

FOUNDED MULTIPLE SUCCESSFUL COMPANIES

LeukoSite · moderna · MORPHIC THERAPEUTIC · SEISMIC THERAPEUTIC · Scholar Rock

2022 Lasker Award

Andrew Kruse, Ph.D.
Co-Founder

GPCR EXPERT, FORBES "30 under 30"

HARVARD MEDICAL SCHOOL · SEISMIC THERAPEUTIC

Multiple Awards and Fellowships
(Biomedical Research, NIH, Amgen, Sloan Research)

Biologics Offer Advantages Over Small Molecules in Targeting GPCRs, Hold Potential to Transform Therapeutic Landscape

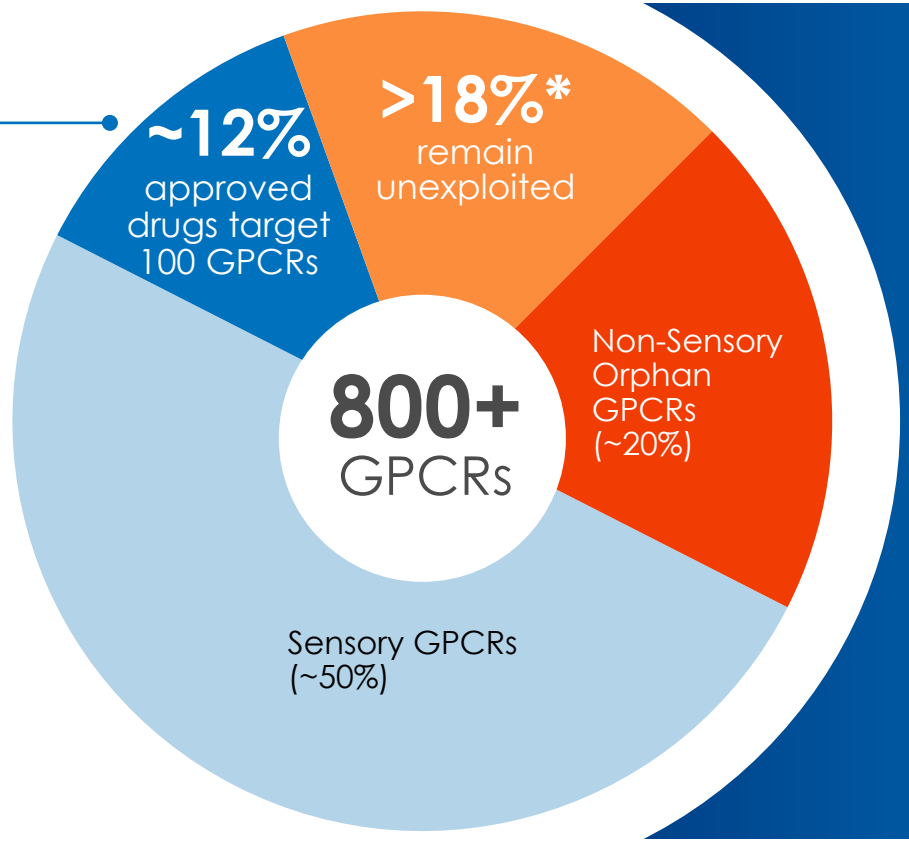
Of the approved drugs that target GPCRs only three are antibodies

>470
approved drugs
(~33% of all)

>$180B
In annual sales

Predominantly small molecules

Address broad range of therapeutic areas



- When difficult to target with small molecule, biologic captures complexity of ligand/receptor engagement
- Biologic minimizes off-target binding to improve safety/tolerability
- Engineer biologic to target or exclude tissue or compartment
- Bispecific approach enables dual target engagement when multi-modal action is required

(*) Hauser, A.S. et al., Cell. 2018 Jan 11; 172(1-2): 41–54.e19.
** 18% = 100% - 12% (approved drug targets) – 50% (sensory) – 20% (non-sensory, orphan)*

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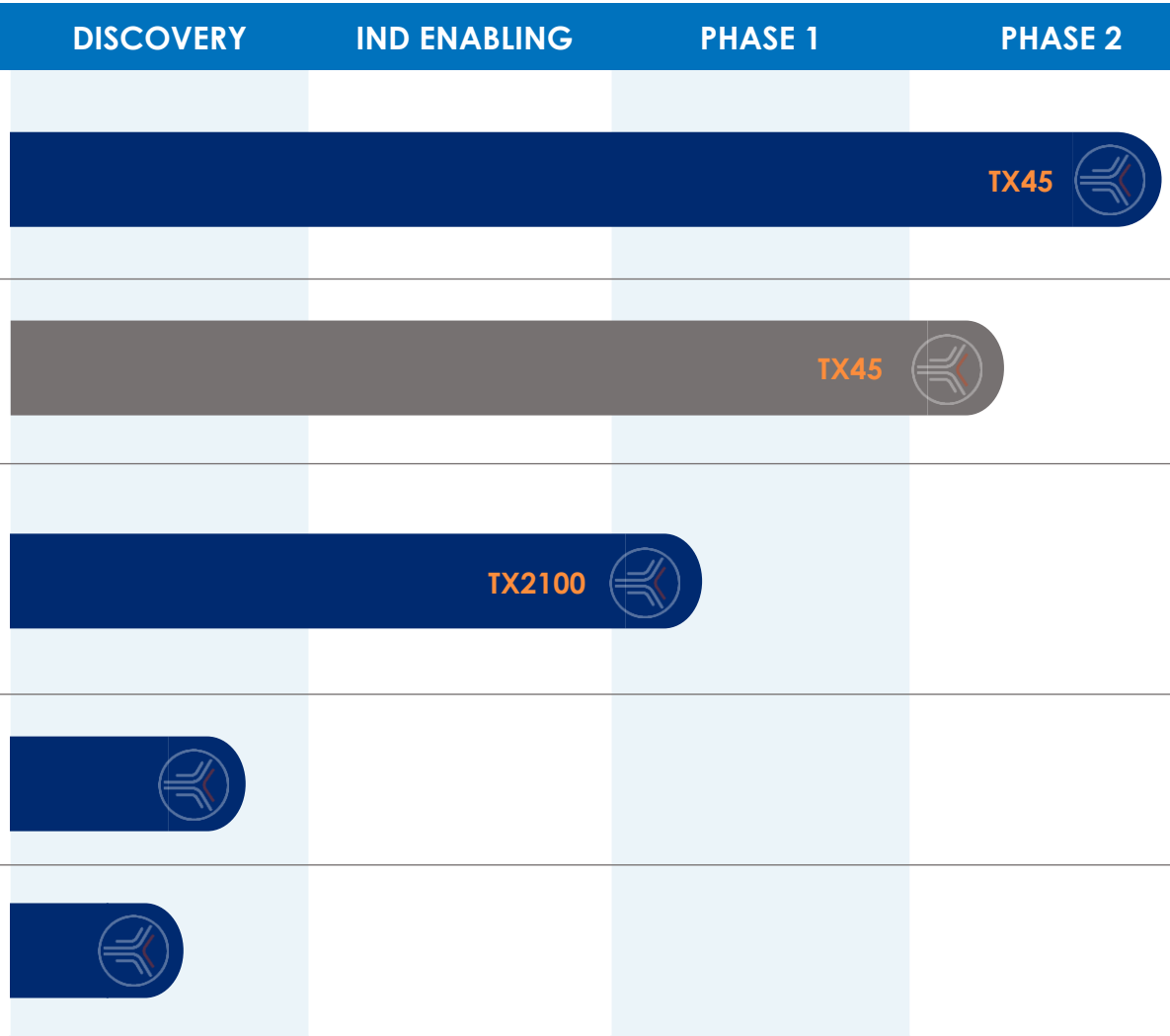
Unique Pipeline of GPCR-Targeted Biologics



ANTICIPATED MILESTONES

- Ongoing APEX Phase 2 PH-HFpEF topline results expected in 2026
- Phase 2 PH-ILD trial opened for screening in Feb 2026
- Phase 1a HHT trial randomized first subject in Feb 2026, expect topline results in Q4'2026
- Phase 1b HHT trial planned*
- Phase 2 HHT trial planned early 2027*
- Bispecific / anti-fibrotic and other GPCR modulators ongoing

** Subject to positive Phase 1 data*

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TX45: Long-acting relaxin to address large, unmet need in Group 2 PH

RXFP1 agonist with differentiated profile

TX45: Potential Best-in-class Treatment for Group 2 PH-HFpEF

High unmet need	• Group 2 PH-HFpEF has no approved therapy • >1M+ patients in US* and high 5-year mortality
Mechanism appears ideal to address disease pathology	• Pulmonary and systemic vasodilator; improves cardiac relaxation during diastole • Reversal of fibrosis in pulmonary vasculature and heart • Anti-inflammatory
Relaxin with optimized PK	• Protein engineering has extended pharmacologic half-life to support monthly dosing • Rigorous Phase 1 PK/PD model enabled robust Phase 2 dose selection
Supporting clinical and pre-clinical data	• Phase 1b Part A hemodynamic data in PH-HFpEF demonstrated improvement in left heart function and pulmonary hemodynamics • Clear benefit observed with TX45 in rodent PH and congestive heart failure models
Streamlined and differentiated clinical strategy	• Enrichment strategy for CpcPH patients where there is the greatest unmet need • Expected 6 min walk test for Phase 3 endpoint, no outcome study needed for approval • Potential early launch and premium pricing relative to broad heart failure indication
Potential to expand opportunity	• Group 2 PH-HFrEF, PH-ILD, Other PH Groups

** US prevalence numbers for Class 2 and 3, estimates based on company sponsored market analysis conducted by Health Advances*

TX45 Initial Indication: Group 2 Pulmonary Hypertension (PH)

Pulmonary hypertension consists of 5 distinct diseases, or groups

Group 1 PAH	• Idiopathic, hereditary or drug-induced • Connective tissue disease-associated • Congenital heart disease-associated
Group 2 PH	• **Due to left heart failure (HFpEF, HFrEF*) or valvular heart disease** • **CAD, HTN, T2DM**, high cholesterol are risk factors** • **Two Subtypes: CpcPH & IpcPH**
Group 3 PH	• Due to lung disease or hypoxia • PH due to interstitial lung disease (ILD), COPD, obstructive sleep apnea, etc.
Group 4 CTEPH	• Chronic thrombo-embolic pulmonary hypertension – i.e., as a consequence of blood clots
Group 5 Misc.	• Miscellaneous group including sickle cell, polycythemia vera, and sarcoidosis

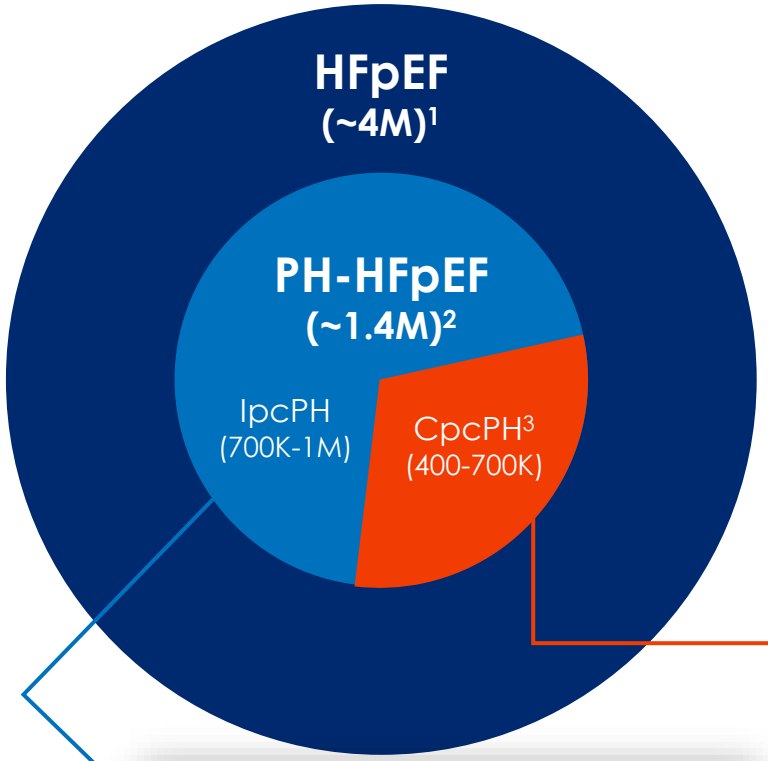
Group 2 PH is chronic, progressive and the largest category of Pulmonary Hypertension

- Elevated blood pressure in the pulmonary arteries
- Chronically elevated pulmonary arterial pressures taxes the right side of the heart
- Pulmonary artery narrowing and muscularization
- Over time, the disease can lead to right heart failure and death
- No approved therapies

** Heart Failure with reduced Ejection Fraction*
*** CAD: Coronary Artery Disease, HTN: Hypertension, T2DM: Type 2 Diabetes Mellitus*

Initial Focus on Group 2 PH due to Heart Failure with Preserved EF (PH-HFpEF), Enriched for CpcPH Patients

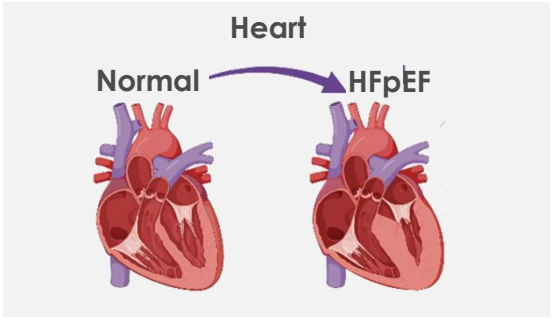
Clinical program designed to enable evaluation of efficacy in overall population and CpcPH



Group 2 PH-HFrEF could add ~1.1M[2] on top of the ~1.4M for PH-HFpEF

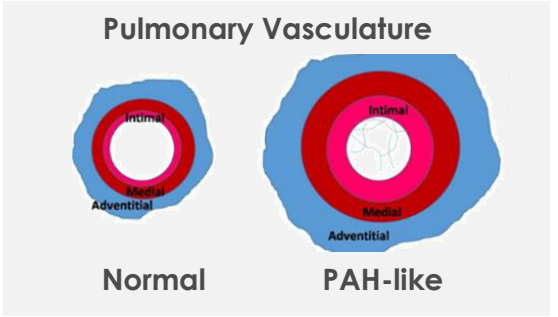
IpcPH (**I**solated, **p**ost **c**apillary **PH**)

Increased Left Ventricle Filling Pressures
↓
Increased Pulmonary Venous Pressures
↓
Passive Pressure Backflow
↓
Pulmonary Hypertension



CpcPH (**C**ombined, **p**re- and post **c**apillary **PH**)

Chronic PH, HF, and/or Other Drivers
↓
Permanent Vascular Changes, e.g. Pulmonary Artery Remodeling
↓
Increased Vascular Resistance
↓
Right Heart Failure



1. *US prevalence estimates based on company sponsored market analysis conducted by Health Advances*
2. *Numbers for only Class 2 and 3 PH-HFpEF based on company sponsored market analysis conducted by Health Advances*
3. *400K CpCPH and 1M IpcPH assumes diagnosis based on PVR≥3; 700K CpcPH and 700K IpcPH assumes diagnosis based on PVR≥2.*

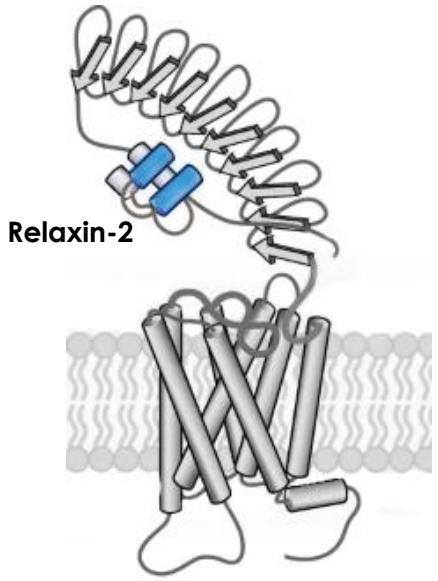
Hemodynamic and Anti-fibrotic Properties of Relaxin Demonstrated by its Role in Pregnancy

Pharmacology

AGONIST
Natural ligand of RXFP1 receptor

No RXFP1 internalization from relaxin agonism → no desensitization with chronic therapy

Relaxin upregulated in pregnancy



RXFP1 active state

Facilitates Gestation

PULMONARY AND SYSTEMIC VASODILATOR

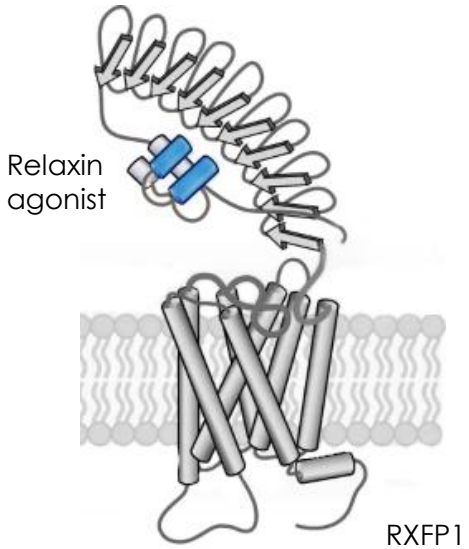
Increases cardiac output to accommodate the increased demand from developing fetus

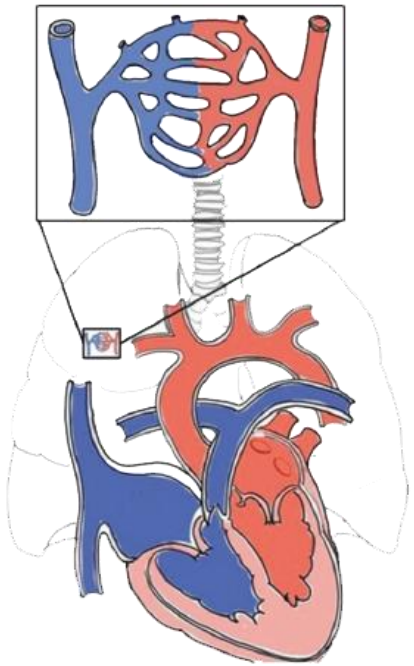
ANTIFIBROTIC

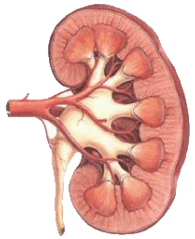
Prepares musculoskeletal tissues for pregnancy and childbirth

Relaxin Addresses Multiple Organ System Pathologies in PH-HFpEF

Phase 1b data was anticipated to capture the acute impact of vasodilation and LV relaxation







↑ Vasodilation
Pulmonary & Systemic
↑ NO ↓ Endothelin-1

↓ RV / LV*
Afterload

↑ LV Relaxation
↑ SERCA2 function

Improved LV Diastolic Function

↓ Fibrosis
Anti-Inflammatory
↑ MMPs ↓ TGFβ

RV / LV and Pulmonary Artery Reverse Remodeling

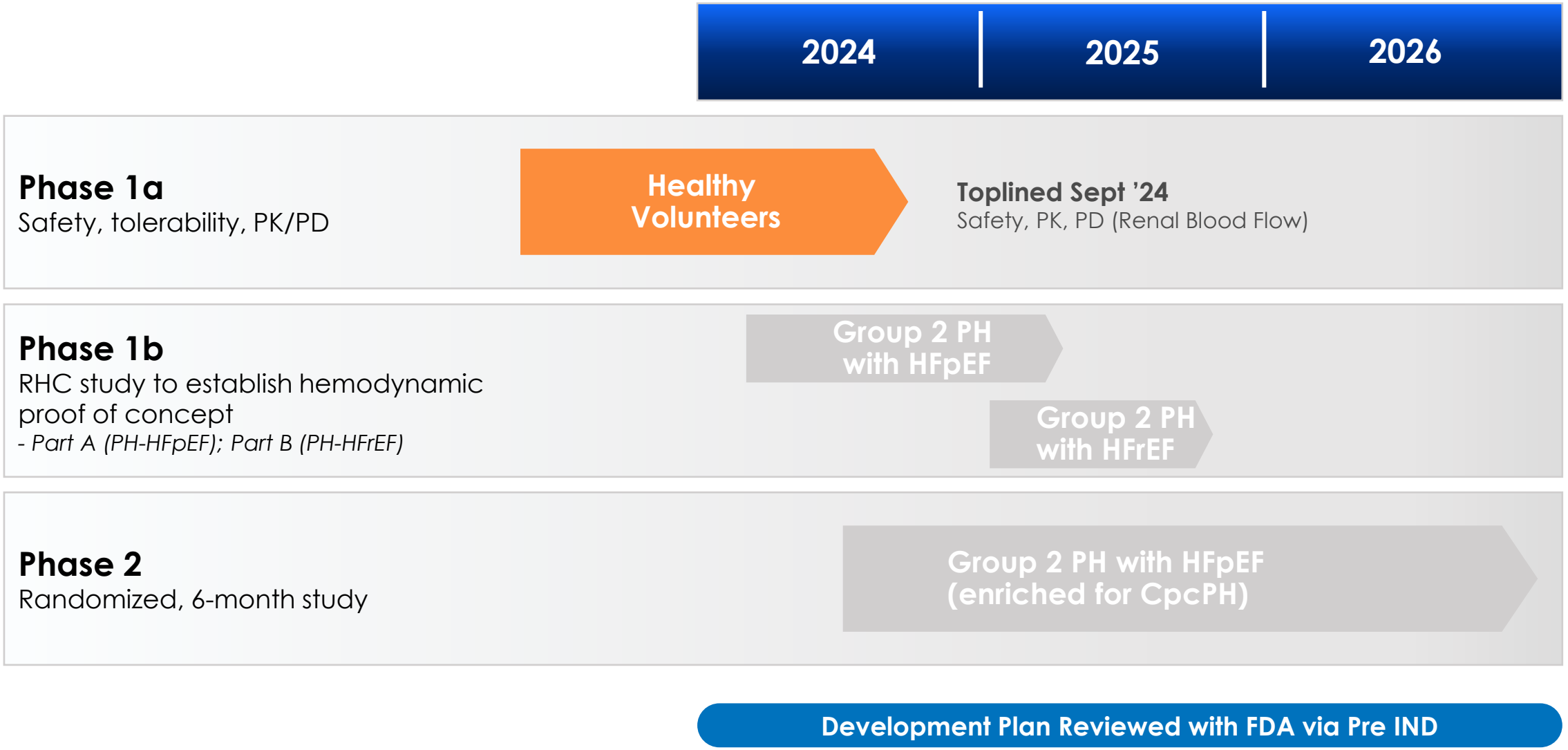
↑ Renal Blood Flow

↑ Na^+ Excretion

↓ RV / LV Preload

** RV: right ventricle; LV: left ventricle*

TX45 Group 2 PH Development Program Overview



RHC: *Right Heart Catheter*

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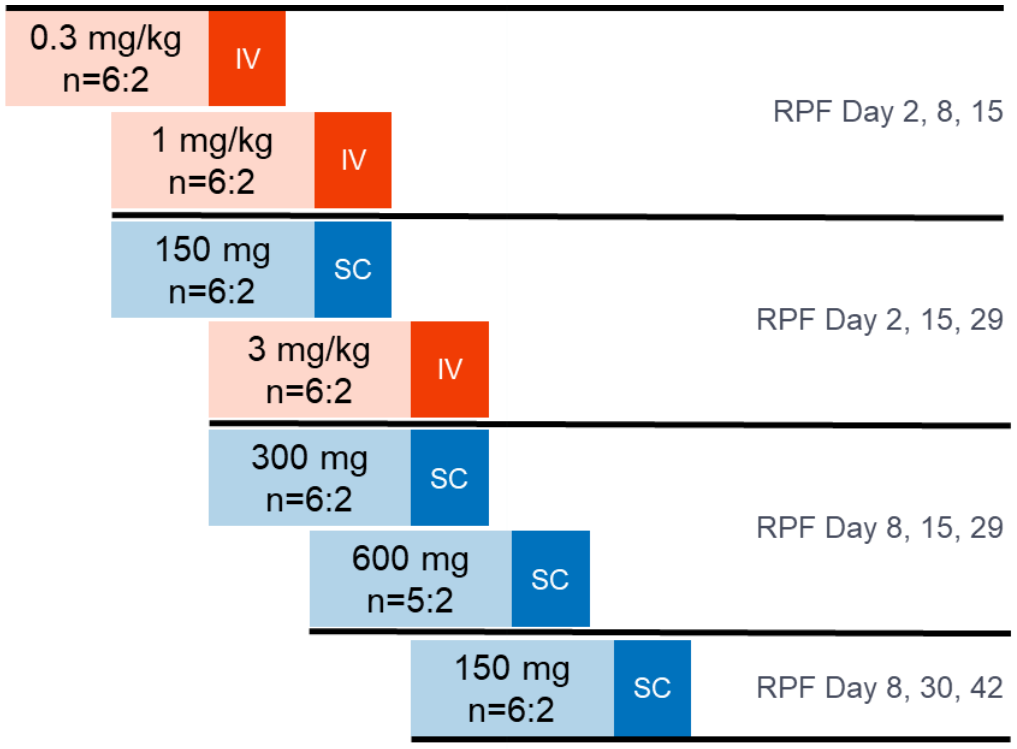
Phase 1a Clinical Study

Robust Design of TX45 SAD Study



Day 1 = Dosing Day

RPF = Renal Plasma Flow

Benefits of the Study Design

- Exposure-response model developed with over 200 data points
- Overcomes impact of outlier values on mean values based on 6 patients per dose cohort
- Enables more robust dataset with which to choose doses for Phase 2

Phase 1a Conclusions

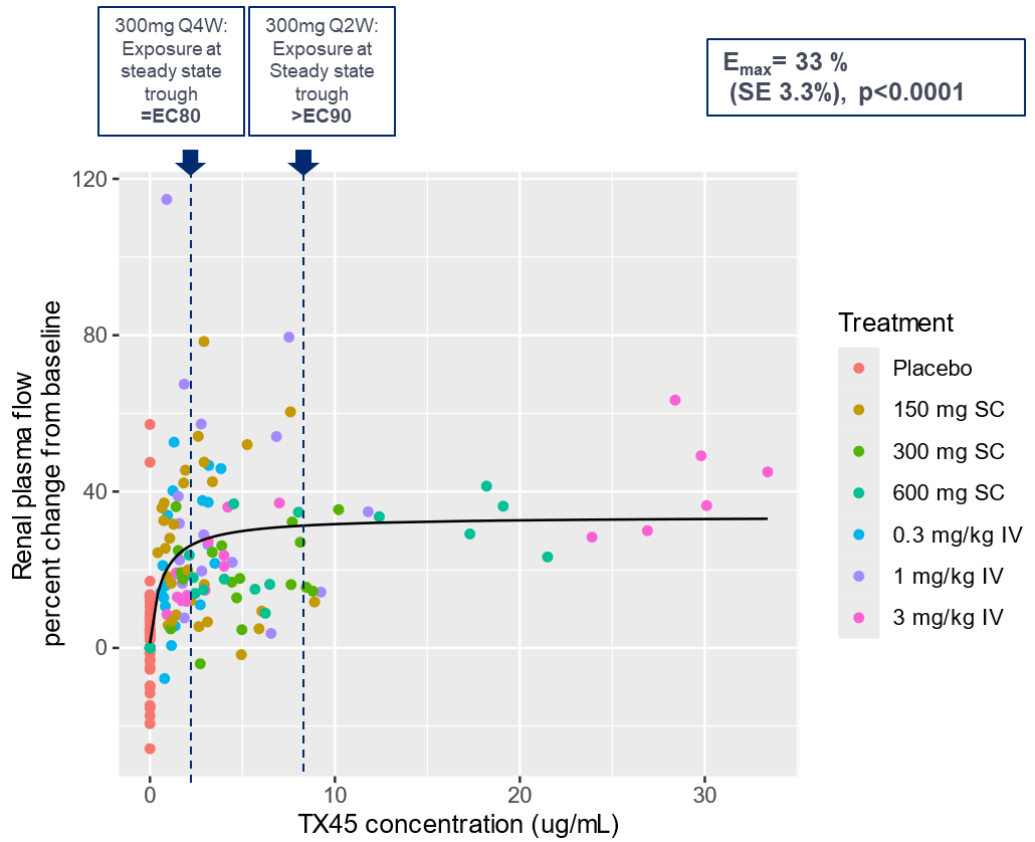
- Safe and well-tolerated
- PK was dose proportional with subcutaneous bioavailability of ~50%
- Potential best-in-class terminal elimination half life of 14-20 days
- PK/PD model developed using renal plasma flow enabled Phase 2 dose selection

Robust Human-Exposure Model Allows for Phase 2 Dose Selection

Phase 2 Dose Selection

- Used preclinical models to extrapolate human dose:
  - Rat renal blood flow model (PD marker), rat PH MCT* model (dose for maximal efficacy), and differences in rat vs. human potency
  - The trough levels required for maximal efficacy in the MCT* model, provide ~ EC_{70} response in the RBF model and predict a trough exposure of 2 ug/ml in humans
- Conclusion was to test two doses:
  - 300 mg SC monthly: Steady state trough of 2.6 ug/ml (EC_{80}) slightly higher than preclinical predicted exposures associated with maximal efficacy
  - 300 mg SC every 2 weeks: Steady state trough of 8.7 ug/ml (>EC_{90}), to evaluate whether increased exposure translates to greater efficacy

TX45-RPF E_{max} model



*MCT = monocrotaline

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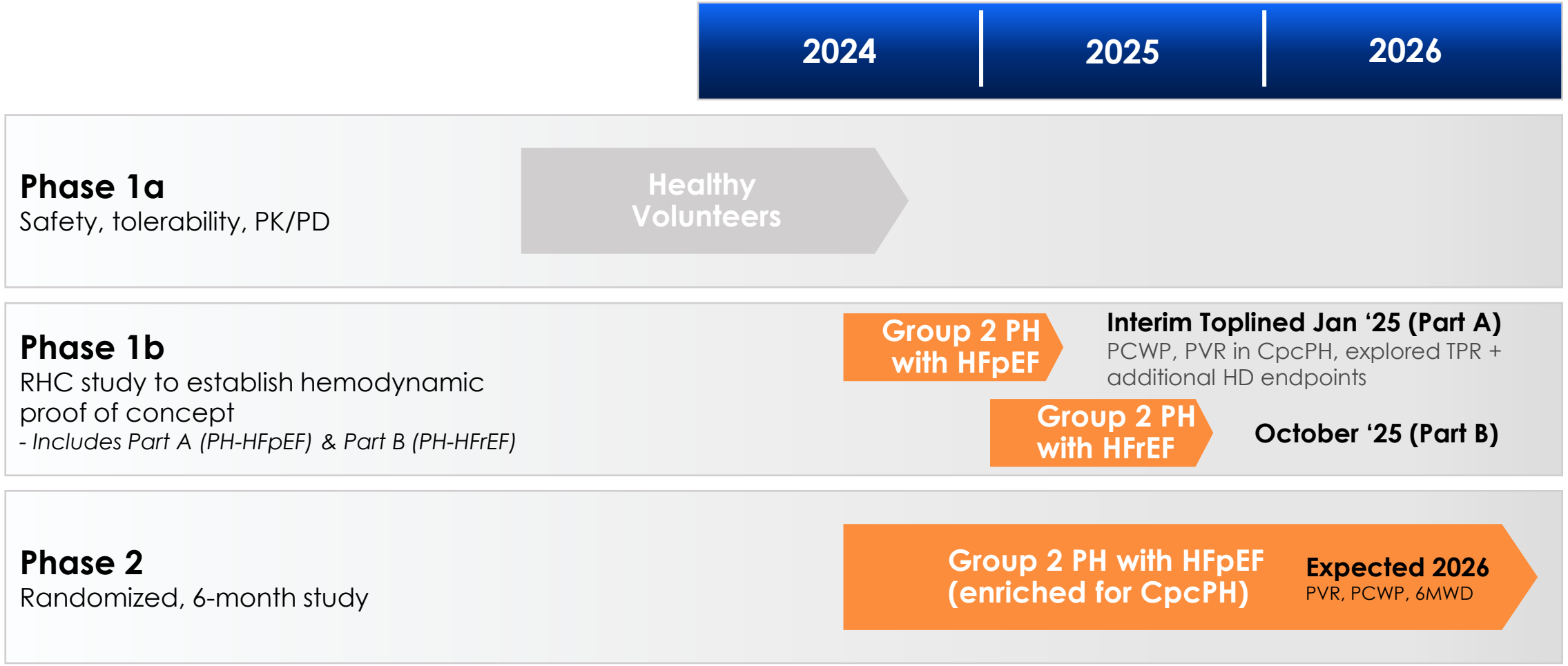
TX45 Group 2 PH Development Program Overview



RHC: *Right Heart Catheter*
PVR: *Pulmonary Vascular Resistance*
PCWP: *Pulmonary Capillary Wedge Pressure*
6MWD: *6-Minute Walk Distance*

Development Plan Reviewed with FDA via Pre IND

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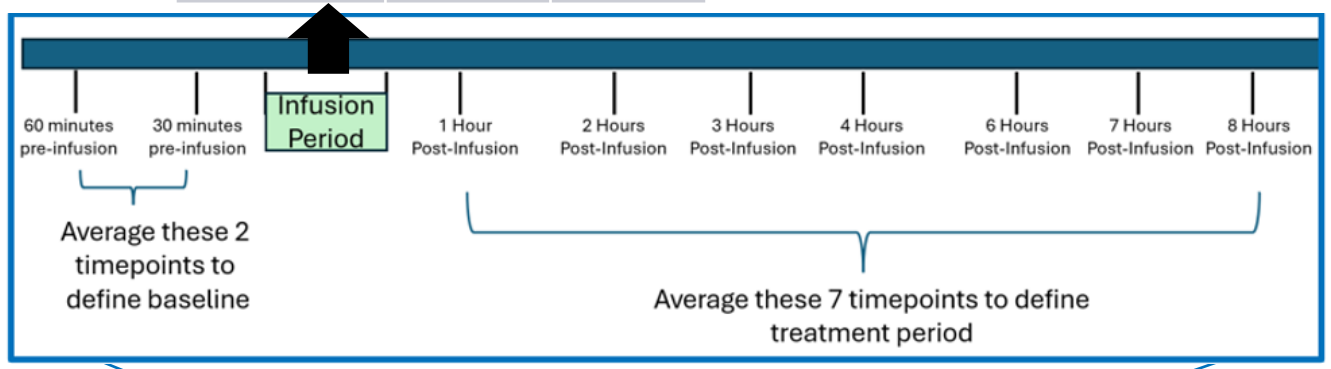
Phase 1b Clinical Trial Design: A Single Dose, Open-Label, Acute Hemodynamic Trial in PH-HFpEF (Part A, n=19) and in PH-HFrEF (Part B, n=14)

TX45 IV Dose	PH-HFpEF	PH-HFrEF
0.3 mg/kg	n = 3	n = 1
1 mg/kg	n = 7	n = 1
3 mg/kg	n = 9	n = 12



Screening Day -28 to -2

Admit to Unit

Right Heart Catheter (RHC) is inserted and hemodynamic measurements are captured at the timepoints listed above

Days 2, 15, 29: ECHO
Days 8, 43: Safety Follow-ups

Day -1 **Day 1**

- Hemodynamic data was prespecified to be pooled across all doses. After IV administration, all dose levels result in exposures which are in the predicted efficacious range during the 8-hour assessment period (i.e., above trough exposure of 2 ug/ml)
- Pure intention to treat (ITT) analysis performed: all data points included in the analysis

Key Hemodynamic Measures

Goal: Treatment for PH due to heart failure needs to **both** increase LV function and improve pulmonary vascular and right ventricular component of the disease

Hemodynamic	Definition	Significance
PCWP *(Pulmonary Capillary Wedge Pressure)*	• Measure of left atrial pressure	• Key marker of left ventricular (LV) function
PVR *(Pulmonary Vascular Resistance)*	• Measure of resistance to blood flow in pulmonary vessels • PVR = (mPAP – PCWP)/CO	• Health of the pulmonary vessels
SVR *(Systemic Vascular Resistance)*	• Measure of resistance to blood flow through the entire systemic circulation • SVR = (mAP – CVP)/CO	• Critical indicator of cardiovascular heath
TPR *(Total Pulmonary Resistance)*	• Measure of right ventricular afterload • TPR = mPAP/CO	• Key marker of resistance, how hard must the right ventricle (RV) work
CO *(Cardiac Output)*	• Amount of blood heart pumps (volume/time) • CO = heart rate x stroke volume	• How well is the heart working (both RV and LV)
SV *(Stroke Volume)*	• Amount of blood ejected from ventricle per beat	• Effectiveness of the heart at pumping blood (both RV and LV)

Note: mPAP = mean Pulmonary Artery Pressure = average pressure required to pump blood through the lungs, mAP = mean Arterial Pressure, CO = Cardiac Output, CVP = central venous pressure

Phase 1b Part A & B: Baseline Characteristics and Concomitant Medications Consistent with Target Population; Enriched for CpcPH

Baseline Characteristics	PH-HFpEF (Part A)	PH-HFrEF (Part B)
LVEF (%); [mean (SD)]*	49.0 (7.2)	34.1 (6.6)
NT-proBNP (pg/mL); [mean (SD)]	1347 (1146)	3188 (2217)
Creatinine (uMol/L); [mean (SD)]**	82.7 (18.9)	98.6 (23.6)
NYHA Class [n (%)]:		
Class II	12 (63.2%)	6 (42.9%)
Class III	7 (36.8%)	8 (57.1%)

Key Concomitant Medications	PH-HFpEF (Part A)	PH-HFrEF (Part B)
ACEi/ARB/ARNi [n (%)]	10 (52.6%)	14 (100%)
MRA [n (%)]	16 (84.2%)	10 (71.4%)
SGLT2i [n (%)]	8 (42.1%)	9 (64.3%)
Loop Diuretic [n (%)]	13 (68.4%)	12 (85.7%)
Beta-blocker [n (%)]	15 (78.9%)	11 (78.6%)
Digoxin [n (%)]	6 (31.6%)	6 (42.9%)

PVR Classification	PH-HFpEF (Part A)	PH-HFrEF (Part B)
PVR < 2WU	n = 10	n = 2
2 WU ≤ PVR < 3WU	n = 4	n = 5
PVR ≥3 WU	n = 5	n = 7

** Left Ventricular Ejection Fraction*
*** Creatinine normal range (uMol/L): Males: 61.9-114.9 / Females: 53.0 to 97.2*

Phase 1b Part A & B: Baseline Hemodynamics Consistent with Group 2 PH

Parameter	Generally Accepted Normal Ranges (At Rest)	PH-HFpEF (Part A) Baseline Mean (SD)	PH-HFrEF (Part B) Baseline Mean (SD)
Pulmonary Capillary Wedge Pressure (mm Hg)	4-12	17.2 (3.6)	21.1 (5.2)
Pulmonary Vascular Resistance (Wood Units)	<2	2.33 (1.06)	3.26 (1.46)
Cardiac Output (L/min)	4-8	4.48 (1.06)	4.23 (1.53)
Stroke Volume (mL)	60-100	66.8 (19.3)	62.9 (23.9)
Total Pulmonary Resistance (Wood Units)	<3	6.4 (1.7)	8.94 (3.3)
Mean Pulmonary Artery Pressure (mm Hg)	12-16	27.0 (4.4)	34.1 (6.8)
Systemic Vascular Resistance (Wood Units)	10-15	20.3 (6.0)	22.4 (6.5)
Right Atrial Pressure (mm Hg)	2-8	11.7 (4.6)	10.3 (3.6)

TX45 Was Well-Tolerated After Single Dose in Patients in Part A & B

- No serious or severe adverse events, discontinuations, infusion related reactions or drug related adverse events
- Transient asymptomatic drop of sBP (5-11 mm Hg) on day 1
- No signs or symptoms of congestion

PH-HFpEF (Part A)				
Preferred Term	**Cohort A 0.3 mg/kg (n=3)**	**Cohort B 1 mg/kg (n=7)**	**Cohort C 3 mg/kg (n=9)**	**Total (n=19)**
Fatigue	0	0	4	4 (21.1%)
Back pain	0	1	1	2 (10.5%)
Nasopharyngitis	0	0	1	1 (5.3%)
Gout (worsening)	0	1	0	1 (5.3%)
Viral infection	0	0	1	1 (5.3%)
Procedural pain	0	0	1	1 (5.3%)

PH-HFrEF (Part B)				
Preferred Term	**0.3 mg/kg (n=1)**	**1 mg/kg (n=1)**	**3 mg/kg (n=12)**	**Total (n=14)**
Procedural back pain*	1	--	5	6 (42.9%)

** TEAE of procedural back pain due to Right Heart Catheterization*

TEAE of fatigue: All occurred in the evening of D1 (<3 hr duration), investigator reported as "non-drug related", and no fatigue after D1 despite high drug levels

PH-HFpEF (Part A): TX45 Improved Left Heart Function and Pulmonary Hemodynamics in PH-HFpEF Patients

Hemodynamic Endpoints	CFB* Mean [95% CI]	Average % CFB* Mean [95% CI]
Key Hemodynamic Endpoints (n = 19)		
Δ PCWP in all participants	-3.2 [-4.3 to -2.1] mm Hg	**-19.0% [-26.1% to -11.9%]**
Δ PVR in CpcPH (PVR ≥ 2 WU) (n= 9)	-1.06 [-1.34 to -0.78] WU	**-32.0% [-35.9% to -28.1%]**
Δ PVR in CpcPH (PVR ≥ 3 WU) (n= 5)	-1.35 [-1.55 to -1.15] WU	**-35.5% [-38.6% to -32.5%]**
Other Hemodynamic Effects (n=19)		
Δ CO (cardiac output)	+0.73 [0.39 to 1.08] L/min	**+18.5% [10.2% to 26.9%]**
Δ SV (stroke volume)	+7.4 [2.9 to 11.9] mL	**+14.3% [6.0% to 22.7%]**
Δ TPR (total pulmonary resistance)	-1.89 [-2.42 to -1.36] WU	**-28.7% [-34.1% to -22.1%]**
Δ mPAP (mean pulmonary artery pressure)	-4.63 [-5.77 to -3.48] mmHg	**-16.8% [-20.8% to -12.8%]**
Δ SVR (systemic vascular resistance)	-3.95 [-5.82 to -2.08] mmHg	**-16.6% [-24.4% to -8.8%]**
Δ RAP (right atrial pressure)	-3.57 [-5.40 to -1.74] mmHg	**-25.8% [-41.6% to -10.0%]**

WU = Wood Unit
Green *CFB endpoints signify 95% confidence interval does not cross zero*
** CFB = Change from Baseline = (Average of Hours 1-8) – Baseline*

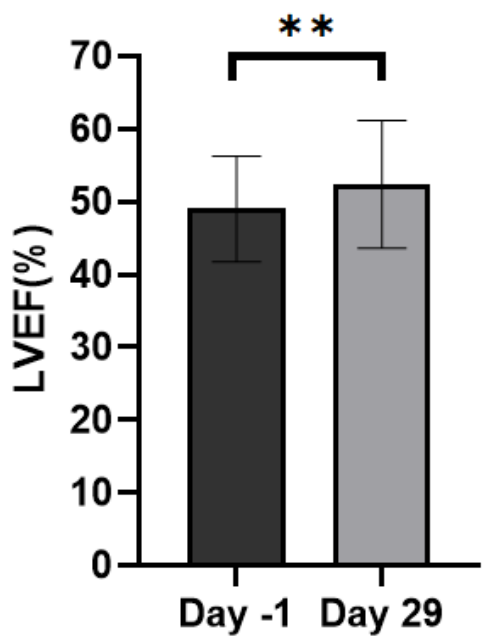
PH-HFrEF (Part B): TX45 Improved Left Heart Function and Pulmonary Hemodynamics in PH-HFrEF Patients

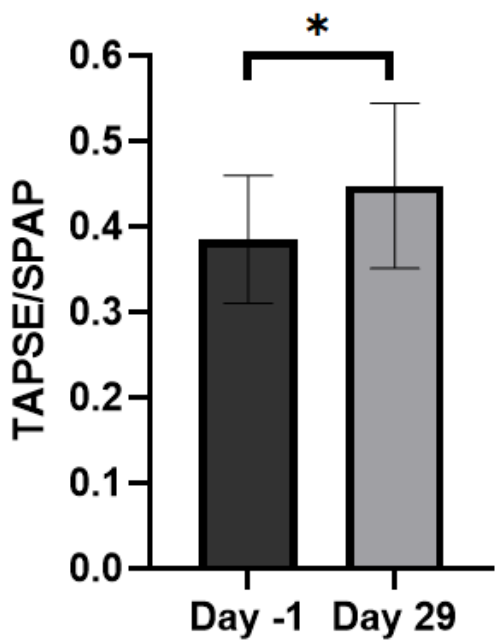
Hemodynamic Endpoints	CFB* Mean [95% CI]	% CFB* Mean [95% CI]
Key Hemodynamic Endpoints (n = 14)		
Δ PCWP in all participants	**-6.4 [-8.6 to -4.2] mm Hg**	**-29.2% [-36.0% to -22.4%]**
Δ PVR in CpcPH (PVR ≥ 3 WU) (n = 7)	-1.10 [-2.79 to +0.59] WU	-19.7% [-45.2% to +5.8%]
Δ PVR in CpcPH (PVR ≥ 2 WU) (n = 12)	-0.61 [-1.72 to +0.50] WU	-10.3% [-36.6% to +15.9%]**
Other Hemodynamic Effects (n = 14)		
Δ CO (cardiac output)	**+0.65 [+0.25 to +1.05] L/min**	**+17.3% [+5.2% to +29.3%]**
Δ SV (stroke volume)	**+6.8 [+0.6 to +13.0] mL**	**+13.4% [+0.9% to +25.9%]**
Δ TPR (total pulmonary resistance)	**-2.82 [-4.00 to -1.64] WU**	**-29.2% [-37.1% to -21.3%]**
Δ mPAP (mean pulmonary artery pressure)	**-6.5 [-8.7 to -4.2] mm Hg**	**-19.3% [-24.8% to -13.8%]**
Δ SVR (systemic vascular resistance)	**-3.2 [-5.7 to -0.7] WU**	**-12.9% [-20.8% to -5.0%]**
Δ RAP (right atrial pressure)	**-3.1 [-4.3 to -1.9] mm Hg**	**-29.2% [-39.1% to -19.4%]**

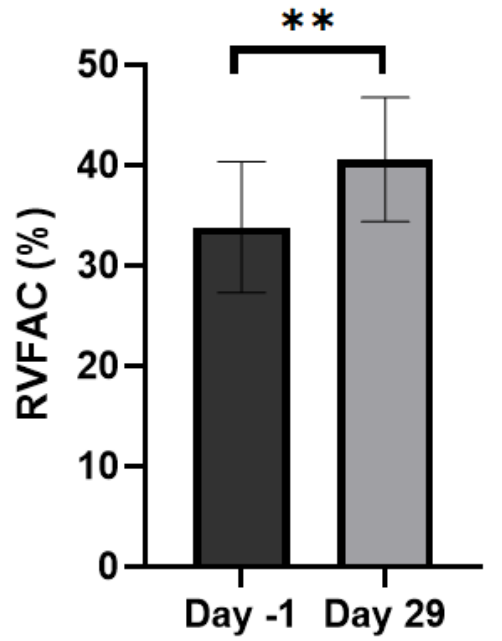
WU = Wood Unit
***Green** CFB endpoints signify 95% confidence interval does not cross zero*
** CFB = Change from Baseline = (Average of Hours 1-8) – Baseline*
*** In the PVR ≥2 WU group, there was one outlier that drove a difference between the mean (-10.3%) and the median (-18.3%); mean and median values were similar for other hemodynamic assessments*

PH-HFpEF (Part A): Echo Results Demonstrated Sustained Improvement in Markers of LV and RV Function, and Pulmonary Hemodynamics

Data Presented as Mean ± SD for All Subjects (n=19 overall)







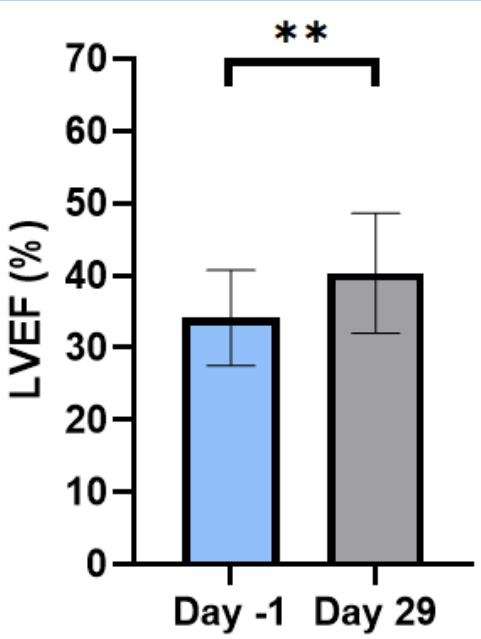
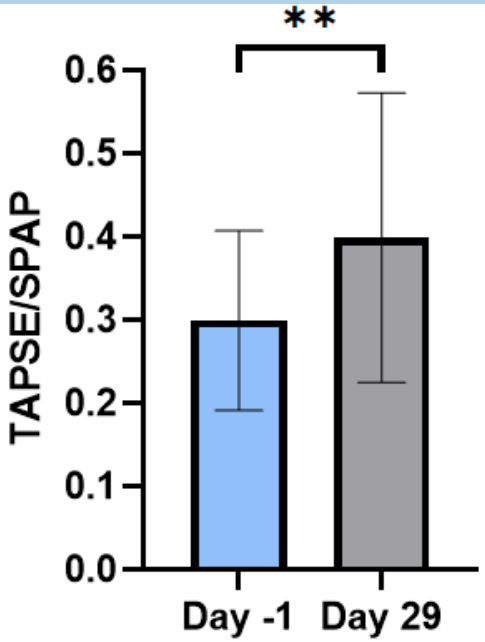
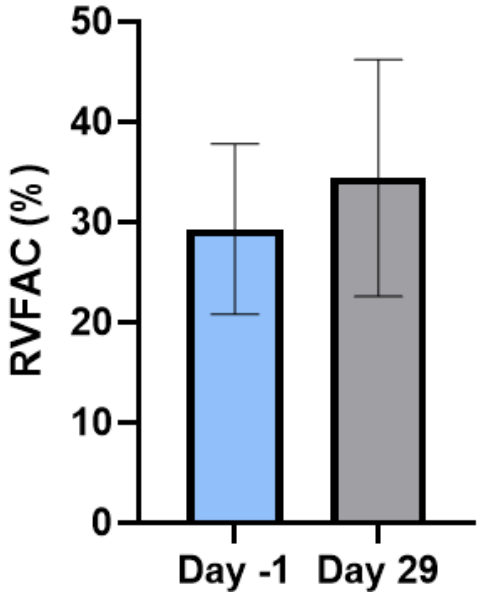
	LVEF (n=19)	TAPSE/SPAP (n=16)	RVFAC (n=18)
CFB Mean [95% CI]	+3.4 [+1.2, +5.6]	+0.06 [+0.01, +0.11]	+6.7 [+3.3, +10.2]
% CFB Mean [95% CI]	+7.0% [+2.5%, +11.5%]	+17.9% [+4.4%, +31.4%]	+23.3% [+9.7%, +36.9%]

CFB = change from baseline, CI = confidence interval
Green *CFB endpoints signify 95% confidence interval does not cross zero*
**Nominal P<0.05, **Nominal P<0.01 for treated (Day 29) versus baseline (Day -1)*

***LV / RV** = left ventricle / right ventricle*
***LVEF** = left ventricular ejection fraction*
***TAPSE/SPAP** = tricuspid annular plane systolic excursion/systolic pulmonary arterial pressure, an inversely correlated surrogate for PVR*
***RVFAC** = right ventricular fractional area of change; a measure of right heart function*

PH-HFrEF (Part B): Echo Results Demonstrated Sustained Improvement in Markers of LV and RV Function, and Pulmonary Hemodynamics

Data Presented as Mean ± SD for All Subjects (n = 14 overall)

	LVEF (n=14)	TAPSE/SPAP (n=14)	RVFAC (n=14)
			
CFB Mean [95% CI]	+6.2 [+2.7 to +9.7]	+0.10 [+0.03 to +0.17]	+5.1 [-0.11 to +10.3]
% CFB Mean [95% CI]	+19.4% [+9.0% to +29.8%]	+36.3% [+11.3% to +61.2%]	+20.3% [+3.2% to +37.3%]

***CFB** = change from baseline, CI = confidence interval*
***Green** CFB endpoints signify 95% confidence interval does not cross zero*
**Nominal P<0.05, **Nominal P<0.01 for treated (Day 29) versus baseline (Day -1)*
Note: p-value for RVFAC Part B was 0.055

***LV / RV** = left ventricle / right ventricle*
***LVEF** = left ventricular ejection fraction*
***TAPSE/SPAP** = tricuspid annular plane systolic excursion/systolic pulmonary arterial pressure, an inversely correlated surrogate for PVR*
***RVFAC** = right ventricular fractional area of change; a measure of right heart function*

Summary and Implications: TX45 Improves Cardiac and Pulmonary Hemodynamics in both PH-HFrEF and PH-HFpEF Patients

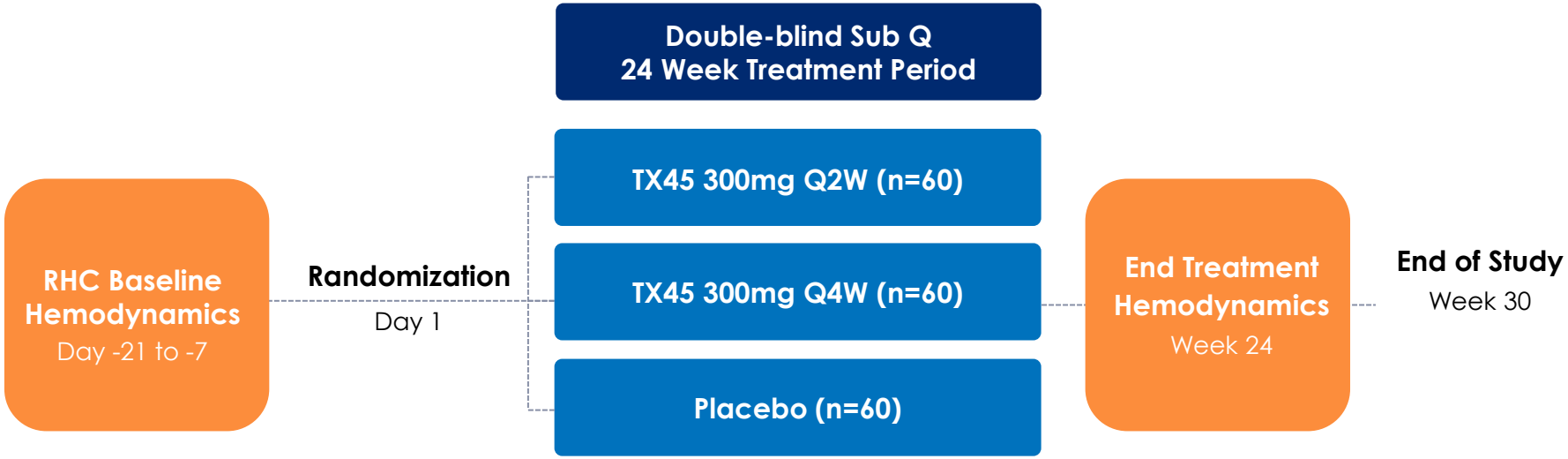
Well-Tolerated:	• TX45 was well-tolerated in both patient populations
Hemodynamic Improvements in LH Function, Pulmonary Vasculature & RV Afterload:	• TX45 administration associated with hemodynamic improvements in PCWP, CO, PVR, TPR, and mPAP • Echocardiographic improvements at Day 29 in LVEF, TAPSE/SPAP, and RVFAC • TPR improved (reduced RV afterload has been associated with improved mortality and outcomes in cohort studies of patients with Group 2 PH[4])
Correlation Between Hemodynamics & 6MWD:	• The demonstrated magnitude of reduction in PCWP and PVR in Phase 1b (HFpEF and HFrEF cohorts) have been associated with meaningful changes in 6MWD in previous studies[1-3]
Data Supports Phase 2 Design & Future Expansion Potential:	• Enrichment strategy in APEX Phase 2 trial for CpcPH subjects where the benefit could be the greatest • Future expansion of TX45's addressable Group 2 PH patient population to PH-HFrEF, pending results of ongoing APEX

1. *Lewis GD et al. Circ. Heart Failure 2023*
2. *Lewis GD et al. Circulation 2007*
3. *Zhang H et al. JACC Cardiovasc. Interv. 2019*
4. *Tampakakis,, Circ Heart Failure, 2018*

APEX Phase 2 Efficacy Clinical Trial Design for TX45

Clinical trial in subjects with PH-HFpEF enriched for subjects with CpcPH subgroup

- Global multicenter, double-blind, randomized, placebo-controlled proof-of-concept clinical trial to evaluate the efficacy of TX45



- **Primary Endpoint:**
  Change from baseline in PVR in the PVR>3 subpopulation (70% of patients will have PVR>3)
- **Secondary Endpoints:**
  Change from baseline in PCWP, 6MWD, KCCQ

RHC: *Right Heart Catheter*
PVR: *Pulmonary Vascular Resistance*
PCWP: *Pulmonary Capillary Wedge Pressure*
6MWD: *6-Minute Walk Distance*
KCCQ: *Kansas City cardiomyopathy questionnaire*

Pharma Has Interest In The Relaxin MoA and Group 2 PH Patients

Tectonic has potential best-in-class Relaxin molecule

Company	Format	Formulation	Half Life in NHV	Dosing*	Patient Population	Phase 2 Endpoints***	Est. Data Read-Out
TECTONIC Therapeutic	Fc-Relaxin Fusion (TX45)	Sub Q	14-20 days	Q4 Weeks	Group 2 PH / HFpEF (enriched for CpcPH)	ΔPVR (in PVR>3 subpopulation)	Ph 2 - 2026
AstraZeneca	Small Molecule Relaxin (AZD5462)	Oral	3-6 hours	QD*	CHF	ΔEcho Parameters	2H'26
MERCK	ActRIIA-Fc (ACE-011)	Sub Q	n/a	Q3 Weeks	Group 2 PH (CpcPH) / HFpEF	ΔPVR	Q4'25 (Primary endpoint PVR met -> Phase 3)
TENAX THERAPEUTICS	Levosimendan (TNX-103)	Oral	n/a	BID/TID	Group 2 PH / HFpEF	Δ6MWD	2H'26

** Expected dosing frequency, AZN based on dosing frequency in Phase 2 studies listed in clinical trials database*
*** Half life of 13-14 days reported in patients with CHF based on sparse PK profiling; no head-to-head comparison trials were conducted*
**** Tenax TNX-103 is in a Phase 3 clinical trial*
Δ = Change in

TX45 PH-ILD Program

Potential differentiated treatment of Pulmonary Hypertension associated with Interstitial Lung Disease (PH-ILD)

TX45: Overview and Rationale in PH-ILD (WHO Group 3 PH)

TX45 Phase 2 PH-ILD Trial Open for Screening

PH-ILD Overview

- PH-ILD is a subgroup of Group 3 Pulmonary Hypertension (PH)
- PH leads to worsening of both exercise capacity and oxygenation with exertion
- Diagnosed by right heart catheterization in the setting of Interstitial Lung Disease (ILD)*
  - Increased PVR and mPAP with reduced CO

High Unmet Medical Need

- 60K+ PH-ILD patients in U.S.**
- High mortality: 3-year mortality rate of 60% to 77%*** (5x higher than ILD without PH)
- Only inhaled treprostinil therapies approved; both w/AEs of cough / bronchospasm

TX45 Preclinical and Clinical Data Support PH-ILD Indication

- TX45 anti-inflammatory, anti-fibrotic and vascular remodeling data in preclinical PH models support evaluation of TX45 in PH-ILD patients
- TX45 hemodynamic data in Group 2 PH supports rationale for PH-ILD
  - Phase 1b PH-HFpEF subjects showed decrease in PVR and mPAP, increase in CO

Commercial Opportunity

- Multi-billion-dollar market potential given unmet need, patient population and orphan drug pricing (Tyvaso WAC price ~$300K/year)

PH-ILD = Pulmonary Hypertension and Interstitial Lung Disease, PVR = Pulmonary Vascular Resistance, mPAP = mean Pulmonary Artery Pressure, CO = Cardiac Output, AE = Adverse Event

** ILD is a collection of rare parenchymal lung diseases*
*** Company Estimates*
**** Lettieri, 2006, Chest, 129, 746-752*

Strong Rationale for TX45 in PH-ILD

-Pulmonary Vasodilation, Anti-inflammatory and Anti-Fibrotic Effects of Relaxin:
-Potential for Increased Exercise Capacity and Disease Modification

CHARACTERISTICS OF PH-ILD	ANTICIPATED RELAXIN EFFECTS
Pulmonary vascular constriction due to underlying lung disease occurs throughout the lung, not just in areas of fibrotic disease • Leads to increase in mPAP and PVR	**Pulmonary vasodilation via activation of nitric oxide (NO) pathway and antagonizing endothelin-1 (ET-1) pathway** • Leads to improvement in mPAP and PVR and exercise capacity
Pulmonary vessels develop histology similar to pulmonary arterial hypertension (PAH) with muscularization and narrowing of vessels in the lung • Leads to additional increases in mPAP and PVR and eventually right ventricular dysfunction	**Anti-inflammatory effects and inhibition of TGFβ pathway should heal abnormal histologic changes (remodeling) and result in improvement of pulmonary hemodynamics and right ventricular function** • Leads to additional improvement in exercise capacity, quality of life, and outcomes
Parenchymal fibrosis of the lung • Key driver of abnormal lung function	**Anti-inflammatory and anti-fibrotic effects via inhibition of TGFβ pathway may also attenuate underlying lung inflammation and fibrosis** • Leads to preservation in pulmonary function

PH-ILD = Pulmonary Hypertension and Interstitial Lung Disease, PVR= Pulmonary Vascular Resistance, mPAP= mean Pulmonary Artery Pressure, CO= Cardiac Output

TX45 Demonstrates Clinical Hemodynamic Changes and Preclinical Effects on Histopathology Important for PH-ILD

TX45 improves abnormal hemodynamics in PH-HFpEF patients also seen in PH-ILD patients

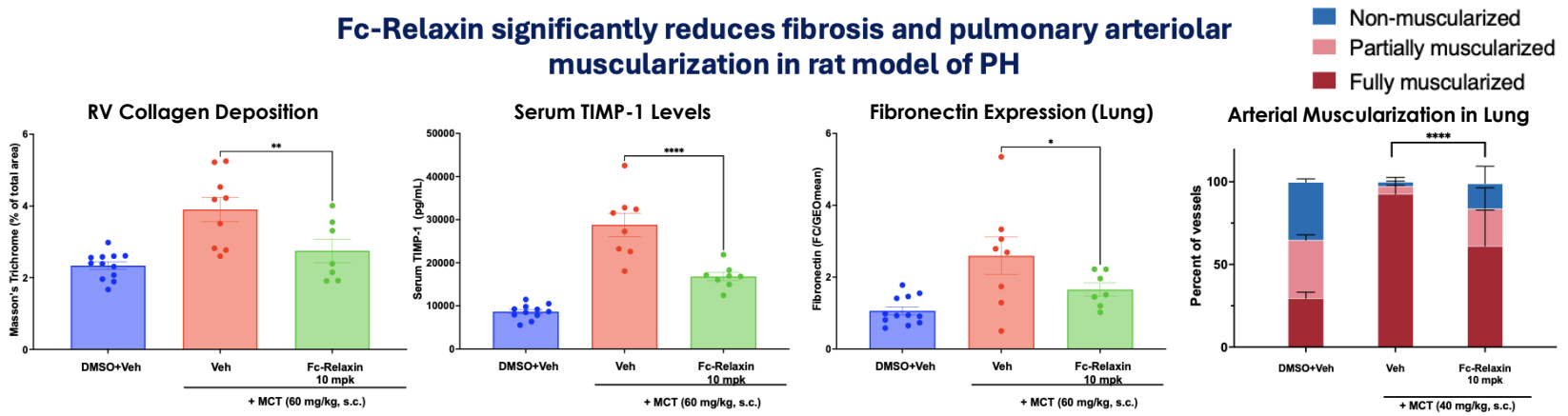
TX45 Effect on Hemodynamics in PH-HFpEF is Relevant in PH-ILD

Hemodynamic Endpoints	CFB* Mean [95% CI]	Average % CFB* Mean [95% CI]
Hemodynamics (N = 9)		
Mean Δ PVR in CpcPH (PVR ≥ 2 WU) (n= 9)	-1.06 [-1.34 to -0.78] WU	**-32.0% [-35.9% to -28.1%]**
Mean Δ PVR in CpcPH (PVR ≥ 3 WU) (n= 5)	-1.35 [-1.55 to -1.15] WU	**-35.5% [-38.6% to -32.5%]**
Other Hemodynamic Effects (N=19)		
Mean Δ Cardiac Output in all participants	+0.73 [0.39 to 1.08] L/min	**+18.5% [10.2% to 26.9%]**
Mean Δ mPAP in all participants	-4.63 [-5.77 to -3.48] mmHg	**-16.8% [-20.8% to -12.8%]**
Mean Δ SVR in all participants	-3.95 [-5.82 to -2.08] mmHg	**-16.6% [-24.4% to -8.8%]**

** CFB = Change from Baseline = (Average of Hours 1-8) - Baseline*

TX45* has remodeling effects in animal models of PH relevant to PH-ILD

Fc-Relaxin significantly reduces fibrosis and pulmonary arteriolar muscularization in rat model of PH



**Mouse Fc-fusion with TX45 relaxin sequence*

PH-ILD = Pulmonary Hypertension and Interstitial Lung Disease, PVR = Pulmonary Vascular Resistance, mPAP = mean Pulmonary Artery Pressure, CO = Cardiac Output, HFpEF = heart failure with preserved ejection fraction, CpcPH = combined post-capillary and pre-capillary pulmonary hypertension, SVR = systemic vascular resistance

TECTONIC Therapeutic

Why Have Inhaled Therapies Succeeded in PH-ILD And Systemic Pulmonary Vasodilatory Compounds Have Failed?

- **Success of inhaled treprostinil treatments: Targeted pulmonary hemodynamic effects while avoiding prostacyclin-specific systemic side effects**
  - Clinical trials in focused population of confirmed PH-ILD with 6MWD and QoL as endpoints
  - Inhaled approach resulted in: 1) lowering of PVR and mPAP without potent systemic BP effects, 2) avoided ventilation/perfusion (VQ) mismatch and resultant worsening oxygenation seen with systemic prostacyclin*
- **Multi-factorial reasons for failure of oral pulmonary arterial hypertension (PAH) therapies include selected patient population, toxicity of specific mechanisms and study design – not due to worsening hypoxemia**
- **Promising data in PDE5 inhibitors*** – Sildenafil, which acts to enhance the nitric oxide (NO) pathway, demonstrated promising clinical data in PH-ILD subjects with positive trends in 6MWD, oxygenation and QoL. Larger trials evaluated ILD subjects and failed

TX45 has the potential to improve pulmonary hemodynamics without systemic hypotension or worsening hypoxemia

PH-ILD = Pulmonary Hypertension and Interstitial Lung Disease, PVR = Pulmonary Vascular Resistance, mPAP = mean Pulmonary Artery Pressure, CO = Cardiac Output, 6MWD = Six Minute Walk Distance

** Sholbin 2024, Kattih, 2025*

TX45 Phase 2 PH-ILD Study: Overview of Design and Rationale

TX45 Open Label, Repeat Dose, 16 Week, Phase 2 Trial

- **Size:**
  - Up to 25 patients with PH-ILD
  - Baseline right heart catheterization (RHC) to diagnose PH
- **Dosing:**
  - Initiating at 300mg Q4 weeks subcutaneous
- **Endpoints: Δ from baseline in the following:**
  - **Safety**: oxygenation and systolic blood pressure (sBP)
  - **Primary Efficacy**: PVR
  - **Secondary and Exploratory**: mPAP and CO, 6MWD, quality of life (QoL)

Rationale for Approach

- **Potential improvement in efficacy endpoints over 16 weeks in open-label trial should replicate in placebo-controlled trial**
  - PVR and 6MWD have not improved over 16 weeks in placebo arms of randomized controlled trials in PH-ILD*
  - Adequate treatment time to assess improvement in fibrotic/remodeled vessels and to address maintenance of effect

PH-ILD = Pulmonary Hypertension and Interstitial Lung Disease, PVR = Pulmonary Vascular Resistance, mPAP = mean Pulmonary Artery Pressure, CO = Cardiac Output, 6MWD = Six Minute Walk Distance

** Corte 2014, Vachiery 2018, Waxman , 2021*

TX45: A Differentiated Therapy for PH-ILD Offering Potential Efficacy, Safety and Convenience

Company	Format	Administration	Dosing	MOA	Clinical Stage	Primary Endpoint
TECTONIC Therapeutic	Fc-Relaxin Fusion (TX45)	Sub-Q	4 Weeks	Relaxin	Planned Phase 2 in 2026	ΔPVR at Week 16
United Therapeutics	Tyvaso (Treprostinil)	**Inhaled** (nebulizer / dry powder)	4x Daily (9-12 breaths/session)	Prostacyclin	**Approved**	Δ6MWD at Week 16
Liquidia	Yutrepia (Treprostinil)	**Inhaled** (dry powder)	3-5x Daily (2 breaths/session)	Prostacyclin	**Approved**	Δ6MWD at Week 16
insmed	Treprostinil Palmitil Inhalation Powder (TPIP)	**Inhaled** (dry powder)	1x Daily	Prostacyclin	Phase 3	Δ6MWD at Week 24
pulmovant	Mosliciguat (BAY 1237592)	**Inhaled** (dry powder)	1x Daily	sGC Activator	Phase 2	ΔPVR at Week 16
Halo Biosciences	Hymecromone (HB-1614)	Oral	2x Daily	Hyaluronan Inhibitor	Phase 2a	ΔPVR at Week 24
Foresee Pharmaceuticals	Mirivadelgat (FP-045)	Oral	1x Daily	ALDH2 Activator	Phase 2	ΔPVR at Week 12

Δ = change in, 6MWD = Six Minute Walk Distance, PVR = Pulmonary Vascular Resistance
head-to-head comparison trials have not been conducted

Summary: Rationale for TX45 to Bring a Differentiated Treatment Approach to Address Unmet Needs in PH-ILD

Disease Burden

PH-ILD is a disease with high morbidity/mortality and insufficient therapeutic options

Relevant Preclinical / Clinical Data

Preclinical PH data and clinical hemodynamic data in PH-HFpEF suggest that TX45 is well suited as a treatment for PH-ILD

Expand TX45 Potential

TX45 offers a potential systemic relaxin therapy for treatment of PH-ILD, enabling expansion of the TX45 program into another large market opportunity

Phase 2

Phase 2 study open for screening to explore safety and efficacy of TX45 treatment in subjects with PH-ILD over 16 weeks

TX2100: APJ Antagonist

Potential first-in-class and indication opportunity for Hereditary Hemorrhagic Telangiectasia (HHT), the 2nd most common genetic bleeding disorder

TX2100 for Hereditary Hemorrhagic Telangiectasia (HHT)

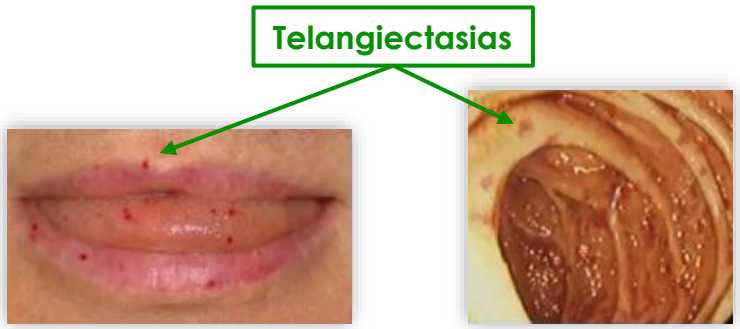
Blockbuster Potential	• HHT is a genetic disorder of dysregulated angiogenesis leading to recurrent bleeding, anemia, arteriovenous malformations (AVMs) and reduced life expectancy with no approved therapies
Orphan Indication	• Estimated ~75K HHT patients in the US; anti-angiogenic drugs (e.g., bevacizumab, pomalidomide) reduce bleeding but chronic use limited by toxicity
APJ: The GPCR Target for the Hormone Apelin	• Highly selective/specific anti-angiogenic target. APJ expressed mainly in endothelial cells, Apelin/APJ pathway is usually quiescent and upregulated during pathologic angiogenesis for greater selectivity vs. other anti-angiogenic agents – Potential to expand into a broader group of bleeding disorders caused by dysregulated angiogenesis
TX2100	• A potential first-in-class APJ antagonist with subcutaneous administration designed to treat HHT with anticipated benefit of anti-angiogenic therapy with improved safety
Preclinical to Clinical Translation	• Anti-angiogenic agents demonstrate activity both in HHT preclinical models and in patients • Efficacy of TX2100 shown in two HHT preclinical models, increasing probability of success
TX2100 Phase 1 Study Initiated	• Phase 1a healthy volunteer clinical trial first subject randomized in Feb 2026

HHT: Multisystem Hereditary Bleeding Disorder with Numerous Morbid and Potentially Fatal Manifestations

Second most common inherited bleeding disorder

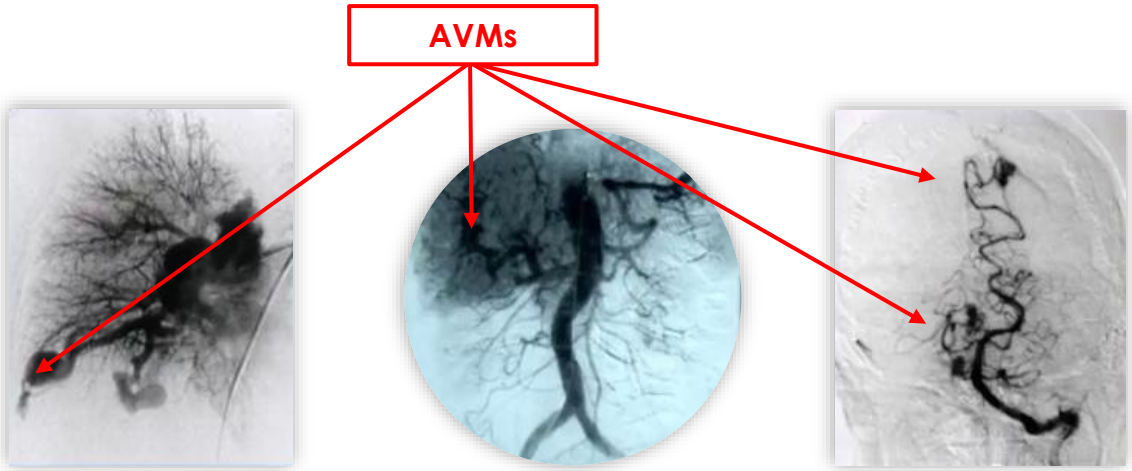
- Caused by mutations in the BMP9/ALK1 pathway
- High degree of phenotypic variability (15-20% severe)
- Increased mortality risk

Nosebleeds



Skin

GI tract



Lung

Liver

Brain

FREQUENCY OF ABNORMAL HHT VESSELS

>95% Nose (epistaxis)
>90% Skin (telangiectasia)
50% Lungs (pulmonary AVMs*)
50% Liver (hepatic AVMs)
20% Gastrointestinal tract
10% Brain (cerebral AVMs)

INCREASED FREQUENCY OF THE FOLLOWING

- Iron and transfusion dependent anemia (10-30% of patients)
- High output CHF 2nd to Liver AVM → liver transplant
- Stroke
- Brain abscesses and other deep tissue abscesses
- Venous thromboembolism (VTE)
- Pulmonary Hypertension
- Migraines

*AVM= arterial venous malformation

TECTONIC Therapeutic

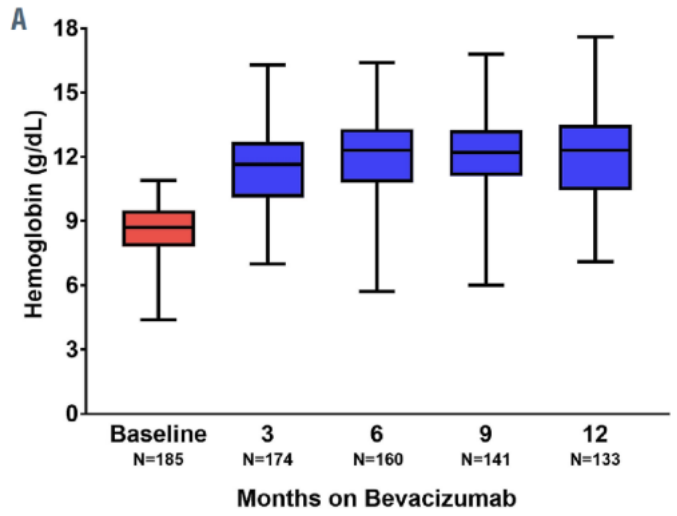
Anti-Angiogenic Therapies are Effective in Translational Models and in the Clinic, but On-Target Toxicities Limit Long-Term Use

Response to drug in mouse models predict clinical efficacy

Mouse models of HHT have a phenotype similar to human disease with GI bleeding and AVMs in numerous locations

Drug efficacy in mouse models predicts human response (bevacizumab[1], pazopanib[2], and thalidomide[3] have efficacy in mouse models and in humans with HHT)

Anti-VEGF improves hemoglobin in severe HHT anemia[4]



Limited Tolerability of Currently Used Anti-Angiogenic Drugs[5]

Bevacizumab

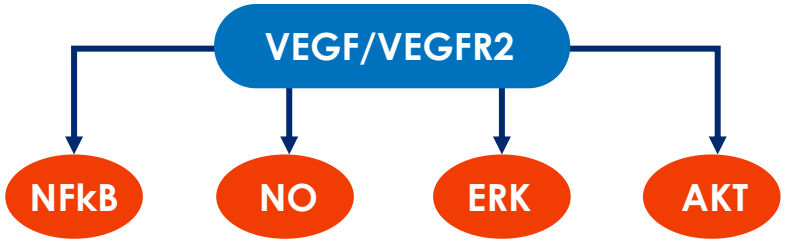
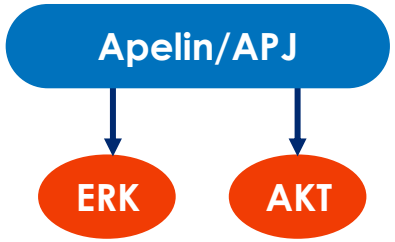
- Side effects: hypertension, proteinuria, thromboembolism risk
- Waning efficacy

Pomalidomide

- Side effects: neutropenia, rash, neurologic side effects, constipation, thromboembolism risk
- Waning efficacy

[1]Walker EJ et al. Stroke. 2012; [2]Kim YH et al. J Thromb Haemost. 2017;
[3]Lebrin F et al. Nat Med 2010; [4] Al-Samkari H et al. Haematologica. 2021; [5]Al-Samkari H. Blood 2024

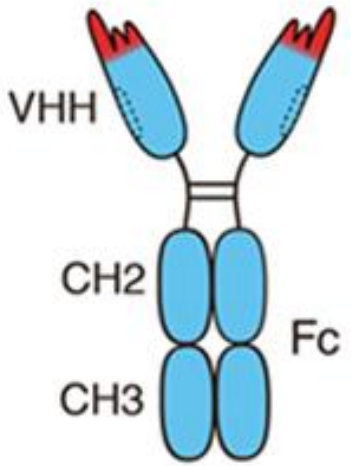
APJ Antagonist: A More Selective & Tolerable Anti-Angiogenic Agent

	VEGFR antagonism: Proven efficacy but poor long-term safety	**APJ antagonism:** Potential for durable efficacy without VEGFR toxicity
Selectivity	VEGFR and AKT signaling broadly required across adult tissues and vascular beds	APJ is endothelial cell enriched and pathway is most active in pathological sprouting angiogenesis
Normal biological function	Central to vascular homeostasis, renal microvascular integrity and repair biology	Low baseline activity in quiescent adult vasculature
Signaling pathways activated		

TX2100 is a Highly Potent and Selective Human APJ Antagonist

Low-nanomolar potency at human APJ with >1,000-fold selectivity vs. related GPCRs

TX2100

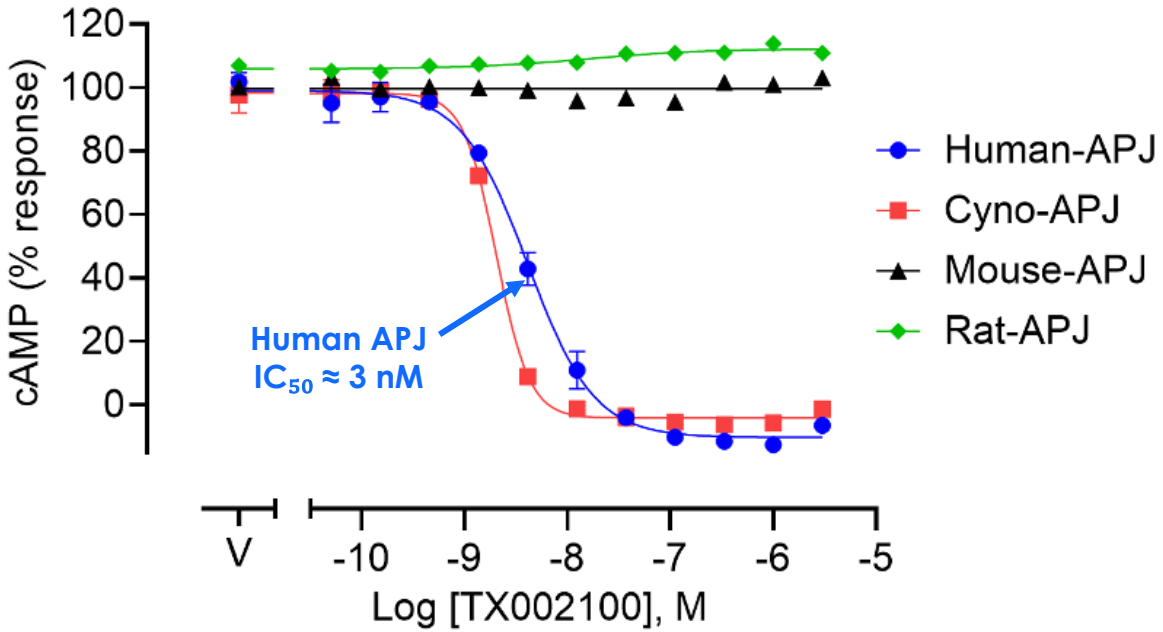


VHH-Fc fusion

- Highly specific, limits off-target toxicities
- Long half-life, less frequent dosing

Receptor	Pathway	IC_{50} (nM)
Human-APJ	cAMP	**3.1**
	β-arrestin	5.6
Most closely related GPCR	β-arrestin	**>1,000**
Mouse-APJ	cAMP	>1,000

TX2100 blocks cAMP signaling with nanomolar potency



cAMP measured in vitro in HEK293 cells

TECTONIC Therapeutic

APJ Antagonism[1] Shows Robust and Durable Preclinical Activity in Two Complementary HHT Models

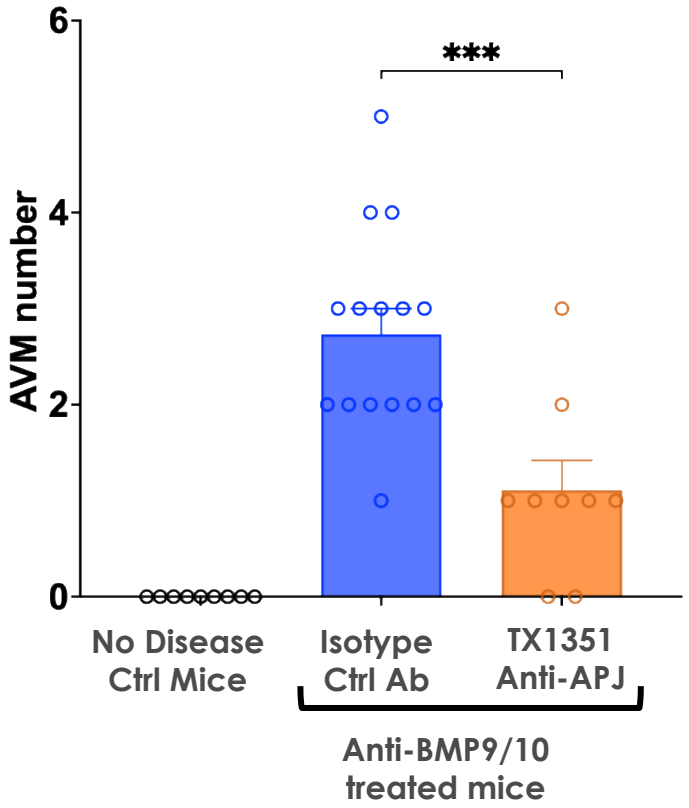
	Preclinical result of APJ antagonism
Neonatal anti-BMP9/10 Translational model of HHT generated by injection of anti-BMP9/10 antibodies into neonatal mice	• Reduced AVMs • Increased hemoglobin • Improved bleeding
Severe adult inducible ALK1-KO Most severe, clinically relevant model where disease is generated in a mature vascular system by tamoxifen-induced knockout of ALK1 in adult mice	• Durably increased hemoglobin (compared to anti-VEGF that waned over time) • Improved bleeding • Improved vascular architecture (reduced hypervascularization, abnormal dilation and AV shunts)

[1]*TX1351 (surrogate anti-mAPJ VHH-Fc; potency matched to TX2100 against APJ) enables translatable in vivo testing*

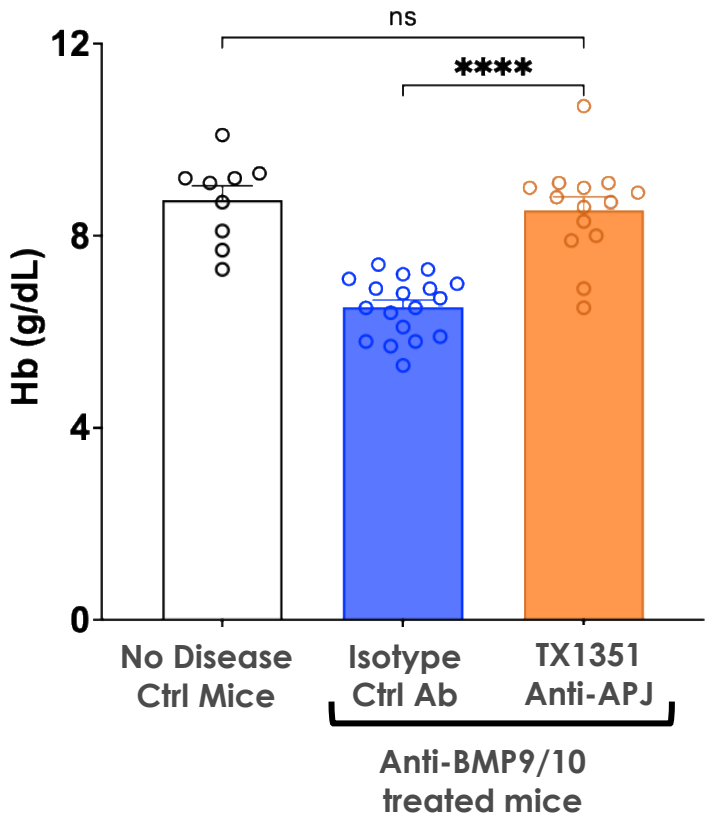
** AVMs = Arteriovenous Malformations*

TX1351[1] Delivers Robust Disease-Modifying Phenotype in the Anti-BMP9/10 Model

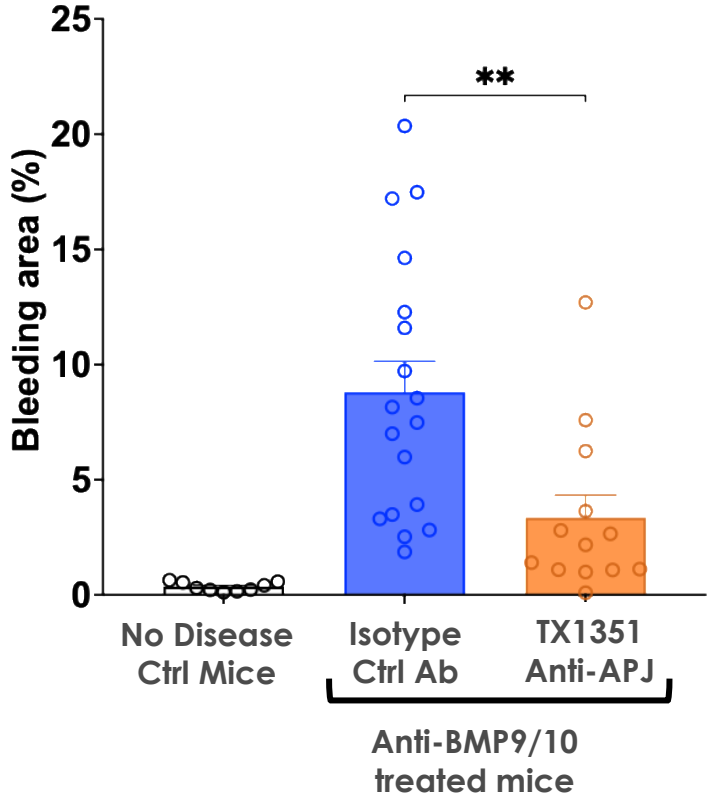
Decreases AVM formation



Increases hemoglobin



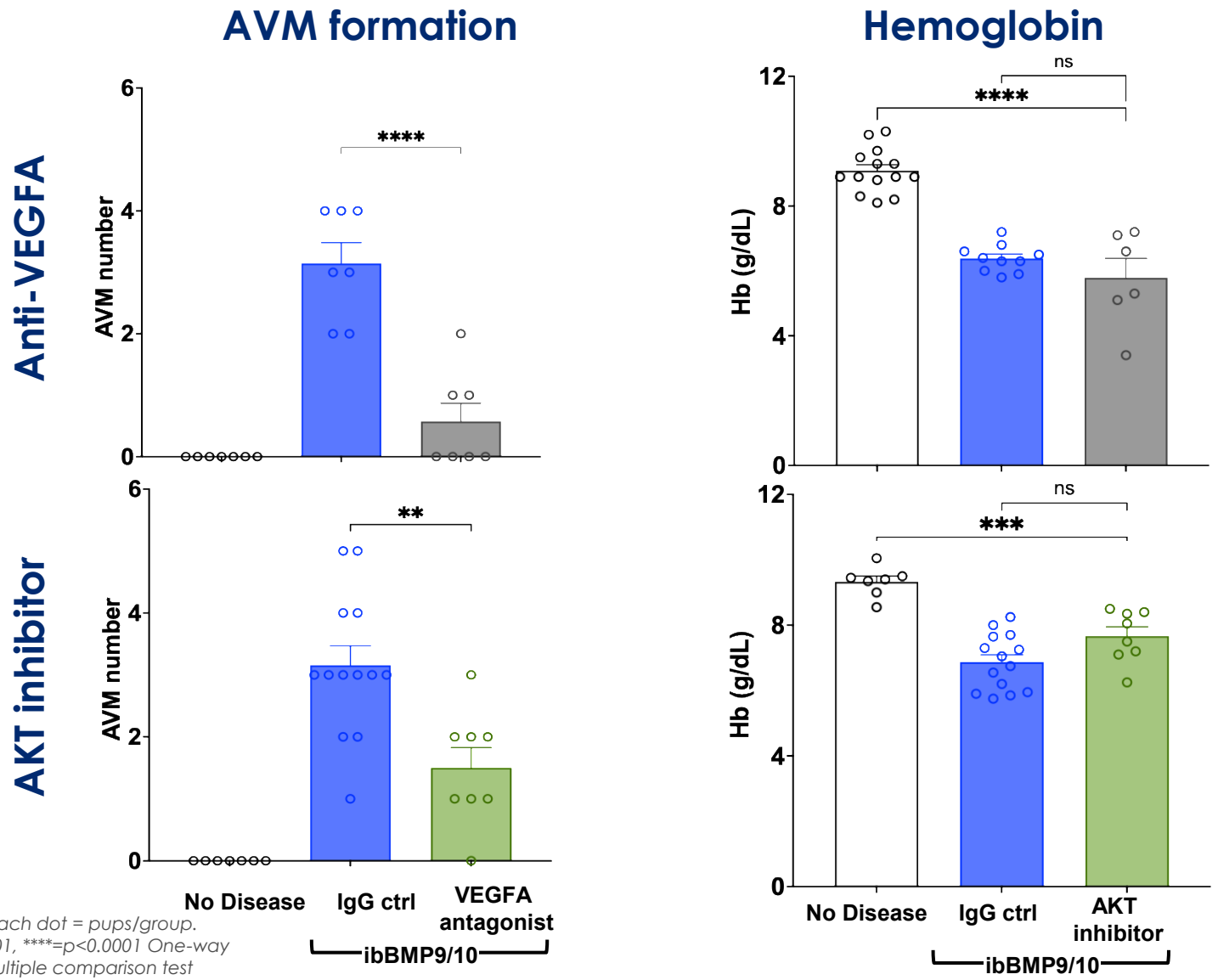
Reduces bleeding (retinal)



[1]*TX1351 = TX2100 surrogate, anti-mouse VHH-Fc; Isotype Ctrl Ab = non-targeting VHH-Fc control*
**=p<0.05, **=p<0.01, ***=p<0.001, ****=p<0.0001 One-way ANOVA followed by Tukey's multiple comparison test*

TECTONIC Therapeutic

Anti-VEGFA, AKT Inhibition Provide Less Robust Disease Modification in the Anti-BMP9/10 Model



*Data represent mean ± SEM; each dot = pups/group. *=p<0.05, **=p<0.01, ***=p<0.001, ****=p<0.0001 One-way ANOVA followed by Tukey's multiple comparison test*

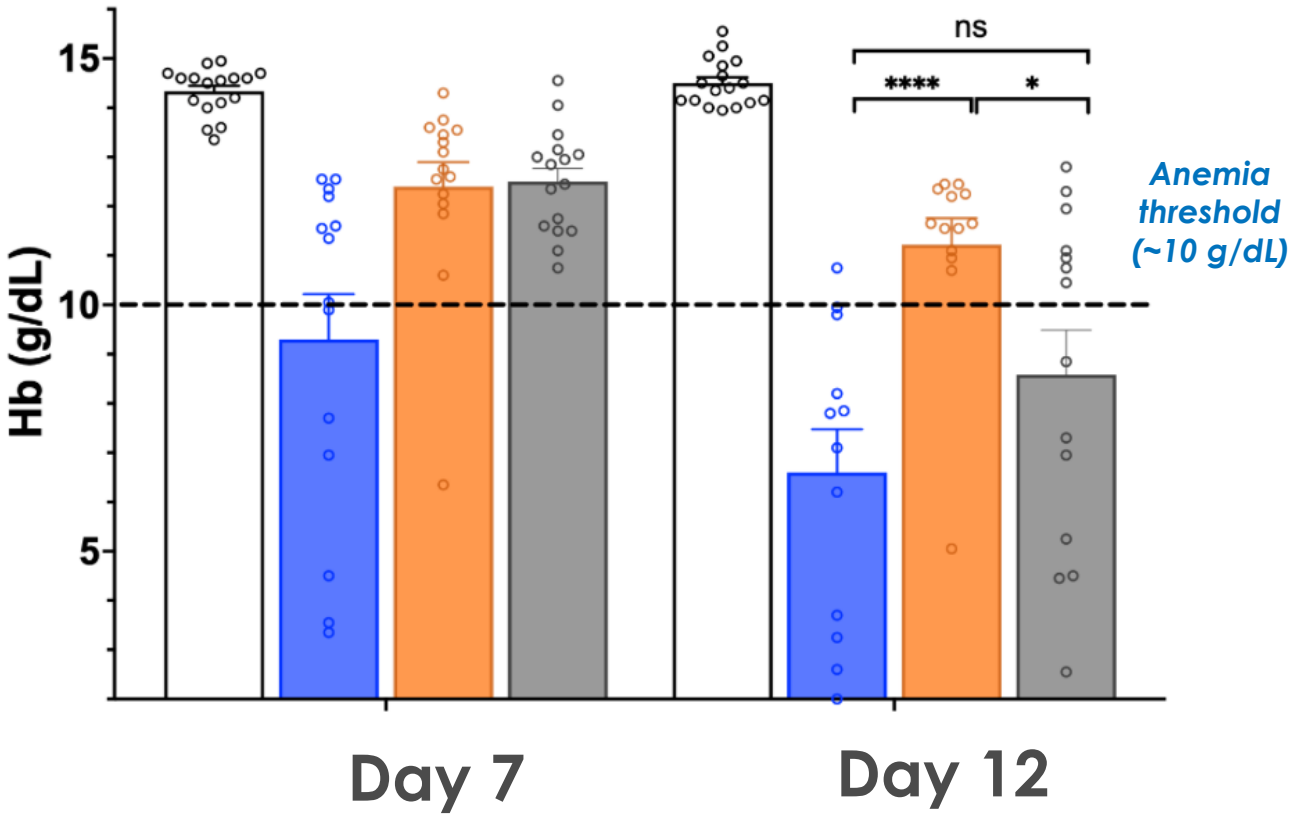
Both mechanisms decrease AVM formation

Neither improved hemoglobin levels

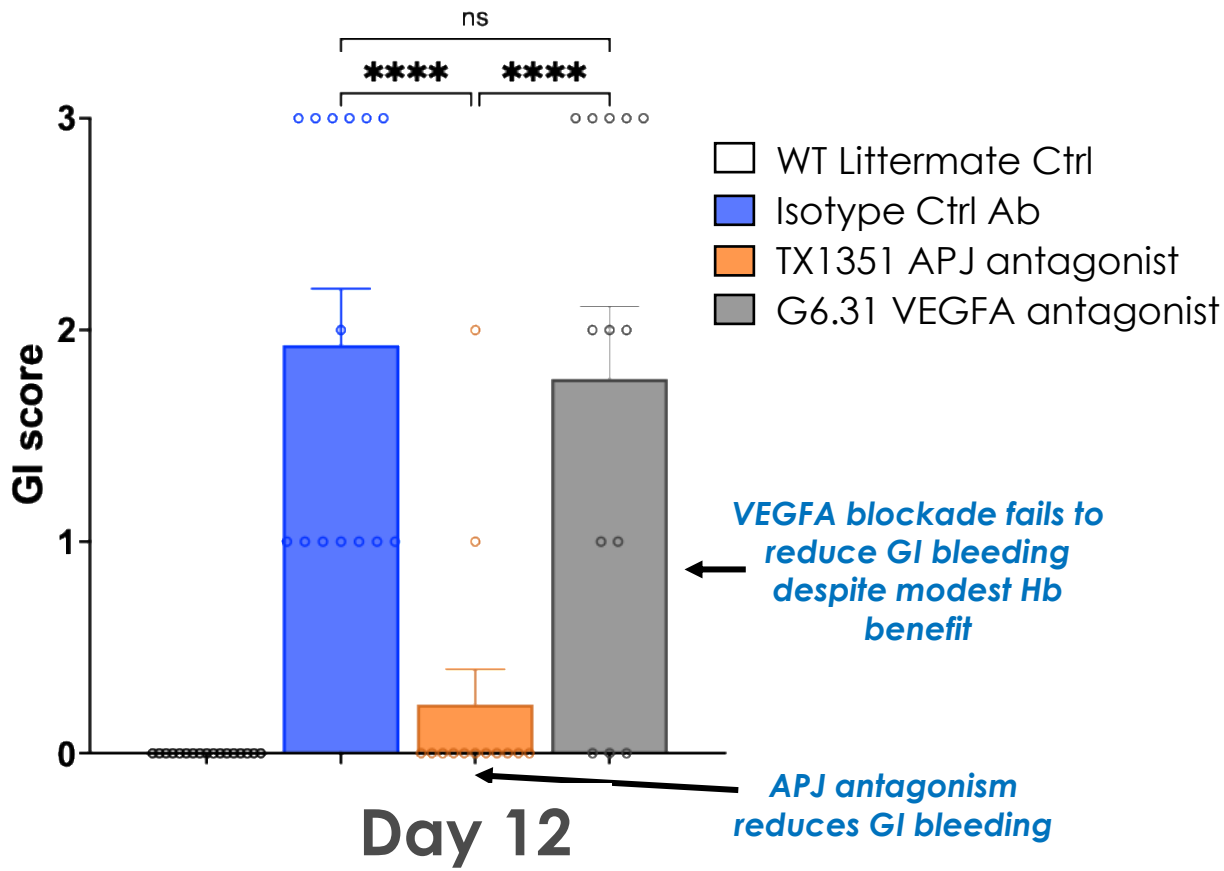
TX1351[1] Reduces Anemia & Bleeding in the iALK1-KO Model

APJ antagonism maintains durable benefits while VEGFA antagonism effects diminish over time

Hemoglobin Levels



GI Bleeding

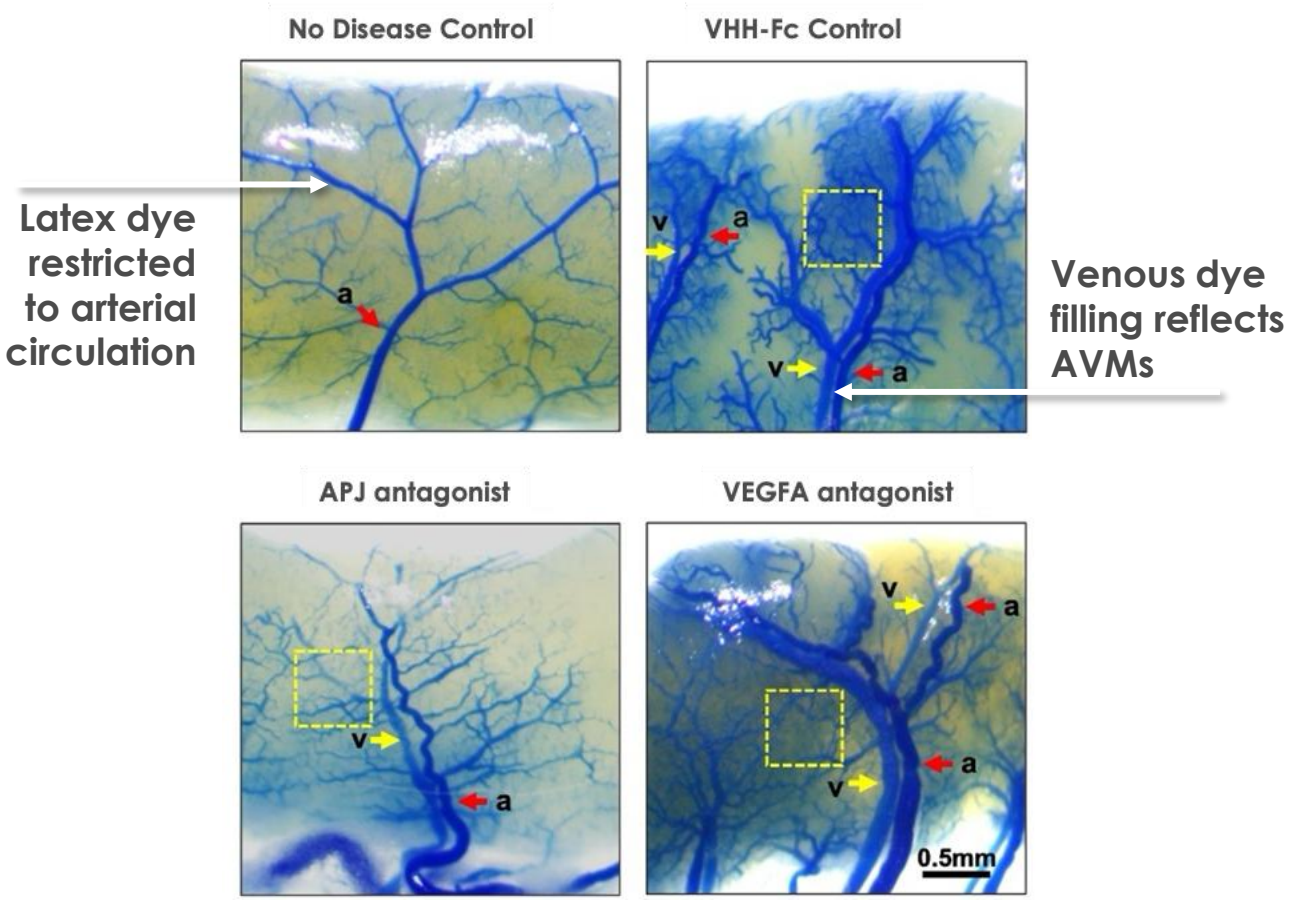


[1]TX1351 = TX2100 surrogate, anti-mouse VHH-Fc; GI bleeding score measured on day 12
*=p<0.05, **=p<0.01, ***=p<0.001, ****=p<0.0001 One-way ANOVA followed by Tukey's multiple comparison test

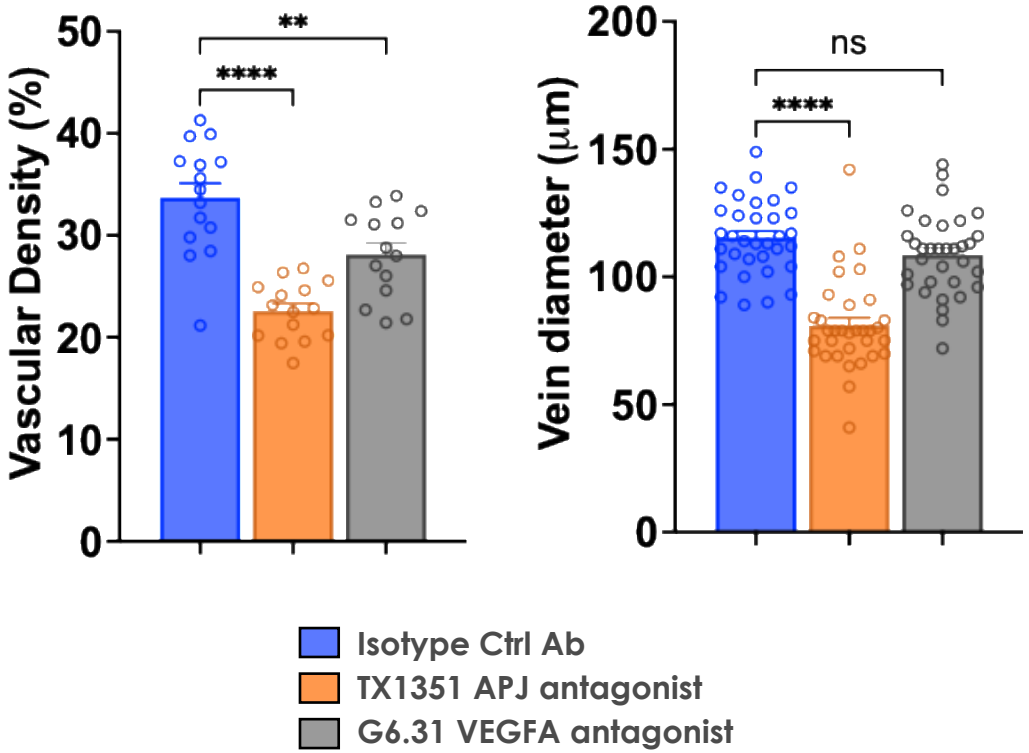
TECTONIC Therapeutic

TX1351[1] Significantly Reduces GI Hypervascularization, Hemorrhage, and Vein Dilation in iALK1-KO Mice

APJ antagonism provides more complete vascular rescue than VEGFA antagonism



TX1351 restores vascular architecture toward normal in a severe HHT model



[1]*TX1351 = TX2100 surrogate, anti-mouse VHH-Fc; Isotype Ctrl Ab = non-targeting VHH-Fc control*
**=p<0.05, **=p<0.01, ***=p<0.001, ****=p<0.0001 One-way ANOVA followed by Tukey's multiple comparison test*

Preclinical Program Did Not Show TX2100 or Target-Related Safety Signals

- **Apelin/APJ pathway has been studied mostly in the context of agonist pharmacology**
- **Clinical agonist programs did not show meaningful benefit**
  - Discontinued for lack of efficacy
  - Were generally safe and well tolerated, without major on-target liabilities

Previously reported physiological effects of apelin[1] and APJ antagonism were not reproduced in multiple in-house preclinical studies

- Blood pressure
- Renal function
- Platelets and bleeding time
- Glucose homeostasis
- Inflammation

Completed 13-week GLP toxicology study in non-human primates, showed no safety findings

- No CV, renal, muscle, or hematology findings
- No changes in glucose
- No BP or fluid balance issues
- NOAEL = 100 mg/kg/week (highest dose tested)

[1]*Szokodi I Circ Res 2002, Coqueral D Am J Physiol 2021, Dray C Cell Metab 2008, Hus-Citharel A Endo 2014, Tatemoto K Reg Peptide 2001*

Overview of TX2100 Clinical Development Plans

Ongoing Phase 1a first-in-human clinical trial in healthy volunteers

- Assess safety, tolerability and PK of single doses of TX2100
- Phase 1a first subject randomized in Feb 2026, expect topline results in Q4'26

Phase 1b clinical trial in patients with severe HHT

- Open label, multiple dose TX2100 study to assess safety and tolerability in patients
- Explore efficacy endpoints of epistaxis, anemia, and hematologic support

Phase 2 proof-of-concept clinical trial in moderate to severe HHT patients

- Randomized double blind placebo-controlled dose ranging study
- Assess safety and efficacy of TX2100
  - Improvement in epistaxis, anemia, hematologic support, and other HHT endpoints

TX2100: A Potential First-in-Class APJ Antagonist to Treat HHT

Validated approach

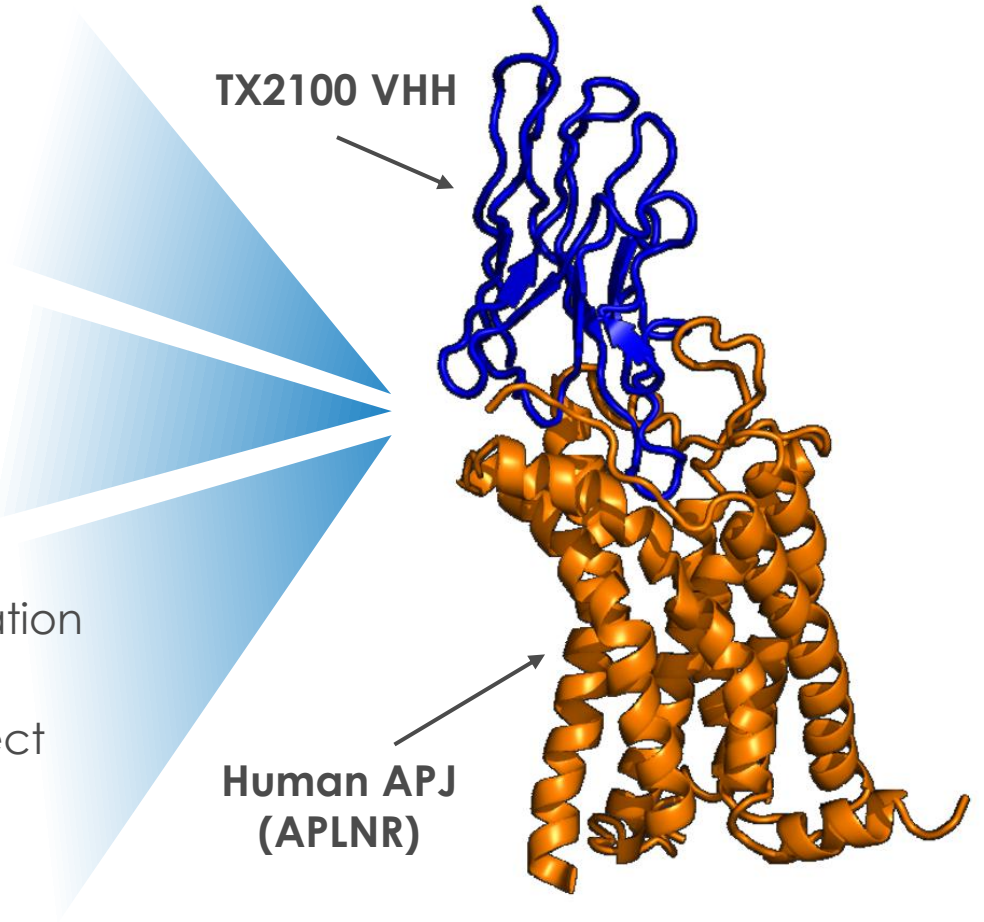
- Anti-angiogenesis improves bleeding/anemia in HHT
- Oncology agents can't be used chronically

Differentiated target / design

- APJ is endothelial-enriched + pathology-biased
- Built to capture anti-angiogenic benefit with improved safety

De-risked translation + path to value

- Preclinical activity in two validated HHT models + vascular normalization imaging
- Clean NHP GLP tox + durable PK → Phase 1a ongoing with first subject randomized in Feb 2026; Phase 1b and Phase 2 PoC planned



TX2100: A Differentiated Therapy for HHT

Company	Program	Administration	MOA	Clinical Stage
TECTONIC Therapeutic	TX2100	Sub-Q	APJ/apelin GPCR antagonist	Phase 1a initiated Q1'26
diagonal Therapeutics	DAIG723	Sub-Q	Clustering antibody agonist restoring ALK1	Initiate clinical trials in 1H'26
Vaderis THERAPEUTICS	VAD044 (Engasertib)	Oral	Allosteric AKT inhibitor	Initiate Phase 3 trial in 1H'26
ATAVISTIK	ATV-1601	Oral	Allosteric AKT1-selective inhibitor	Initiate clinical trials in 1H'26
TERREMOTO	TER-1754	Oral	AKT1-selective inhibitor	Phase 1a/b
Alnylam	ALN-6400	Sub-Q	RNAi reducing plasminogen	Phase 1/2

Platform

Proprietary platform enables reproducible discovery and optimization of GPCR targeted biologics

Solving Key Challenges in GPCR Targeted Biologics Discovery

Challenges

RETAIN
endogenous GPCR structure to enable screening against relevant form of receptor

PURIFY
target in sufficient quantities to power screening campaign

INDUCE
immune response to human GPCR in animals if immunization strategy is pursued

STABILIZE
receptor in active conformation to enable agonist discovery

GEODe™ Platform Features Designed for Success

1.
Receptor Engineering, and Purification Technology
Delivers abundant receptor reagent in native conformation

2.
***In-vitro* Yeast Display Antibody Discovery**
Optimized high-diversity Fab and VHH libraries
Selection protocols optimized for membrane embedded GPCRs

3.
Protein Engineering
Optimize protein pharmacology
Engineer antigen formats to enable screening for agonists or antagonists as needed

Tectonic Tx: Positioned to Deliver on Value-Creating Milestones

Two clinical candidates addressing untapped markets with significant market potential

- TX45, a long-acting relaxin in Phase 2 supported by Phase 1b clinical trial results
  - TX45 has best-in-class potential for >1M+ patients with Group 2 Pulmonary Hypertension (PH) with HFpEF; potential to expand to Group 2 PH-HFrEF and Group 3 PH-ILD (Phase 2 open for screening)
- TX2100, an APJ antagonist in ongoing Phase 1a healthy volunteer clinical trial for HHT
  - Potential First-in-Class & Indication therapy for HHT and other bleeding disorders due to dysregulated angiogenesis

Expected 2026 Data Readouts

- TX2100 Phase 1a healthy volunteer clinical trial topline results expected Q4'26
- TX45 APEX Phase 2 trial topline results in PH-HFpEF expected in 2026

Proven leadership team well-positioned to execute with strong balance sheet

- Executive team with proven track record
- $253.8 million in cash and cash equivalents as of 12/31/25, expected to provide a cash runway into Q4'28

Thank You

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