UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): September 8, 2026

INHIBIKASE THERAPEUTICS, INC.
(Exact Name of Registrant as Specified in its Charter)

Delaware	**001-39676**	**26-3407249**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

1000 N. West Street, Suite 1200 Wilmington, DE	**19801**
(Address of Principal Executive Offices)	**(Zip Code)**

Registrant's Telephone Number, Including Area Code: (302) 295-3800

N/A
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (*see* General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, $0.001 par value	IKT	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01. Regulation FD Disclosure

On September 8, 2026, Inhibikase Therapeutics, Inc. updated its corporate presentation for use in meetings with investors, analysts, and others. A copy of the updated corporate presentation is furnished as Exhibit 99.1 to this Current Report on Form 8-K.

The information in Item 7.01 of this Current Report on Form 8-K (including Exhibit 99.1 attached hereto) is intended to be furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act") or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933 or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits.

99.1	Corporate Presentation issued by Inhibikase Therapeutics, Inc., dated September 2026, furnished herewith.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: September 8, 2026

INHIBIKASE THERAPEUTICS, INC.

By: /s/ Mark Iwicki
Mark Iwicki
Chief Executive Officer

Exhibit 99.1

Disclaimer

This presentation contains information that may constitute "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Inhibikase Therapeutics, Inc. (the "Company" or "we") intends for the forward-looking statements to be covered by the safe harbor provisions for forward-looking statements in those sections. Generally, we have identified such forward-looking statements by using the words "believe," "expect," "intend," "estimate," "anticipate," "project," "target," "forecast," "aim," "should," "will," "may", "continue", "assume", "contemplate", "could", "design", "due", "goal", "hope", "might", "plan", "opportunity", "predict", "possible", "potential", "seek", "strategy", "would" and similar expressions that are predictions or indicate future events and future trends, or the negative of these terms or other comparable terminology. Such statements are subject to a number of assumptions, risks and uncertainties which may cause actual results, performance or achievements to be materially different from those anticipated in these forward-looking statements. You should read statements that contain these words carefully because they discuss future expectations and plans which contain projections of future clinical studies or trials, regulatory approvals, product candidate development, results of operations or financial condition, anticipated cash runway or capital requirements, or market opportunity or state other forward-looking information. However, the absence of these words or similar expressions does not mean that a statement is not forward-looking. Forward-looking statements are not historical facts, but instead represent only the Company's beliefs regarding future events, many of which, by their nature, are inherently uncertain and outside of the Company's control. It is possible that the Company's actual results and financial condition may differ, possibly materially, from the anticipated results and financial condition indicated in these forward-looking statements. Management believes that these forward-looking statements are reasonable as of the time made. However, caution should be taken not to place undue reliance on any such forward-looking statements because such statements speak only as of the date when made. The Company undertakes no obligation to publicly update or revise any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by law. In addition, forward-looking statements are subject to certain risks and uncertainties that could cause actual results to differ materially from the Company's historical experience and our present expectations or projections. This presentation should be read in conjunction with the Company's filings with the Securities and Exchange Commission, including its annual report on Form 10-K, its quarterly Form 10-Q and any subsequent filings with the SEC (collectively, the "SEC Filings"). Important factors that could cause actual results to differ materially from those in the forward-looking statements include, but are not limited to, our ability to execute clinical trials, including to evaluate IKT-001 as a treatment for pulmonary arterial hypertension, as well as such other factors that are set forth in the Company's SEC filings, including under the caption "Risk Factors".

Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and our own internal estimates and research. While we believe these third-party studies, publications, surveys and other data to be reliable as of the date of this presentation, we have not independently verified, and make no representation as to the adequacy, fairness, accuracy or completeness of any information obtained from third-party sources. In addition, no independent source has evaluated the reasonableness or accuracy of our internal estimates or research and no reliance should be made on any information or statements made in this presentation related to or based on such internal estimates and research.

We do not intend our use or display of other entities' names, trade names, trademarks or service marks to imply a relationship with, or endorsement or sponsorship of us by, any other entity.

Inhibikase & IKT-001: Pulmonary Arterial Hypertension (PAH)

~30%

5-year mortality[1]

HIGH UNMET NEED

- **PAH is rare, progressive and life-threatening**, with poor quality of life and high economic burden
- **$8.3 billion[2] market** with antiproliferatives expected to drive rapid growth

45 meters

placebo-adjusted 6MWD gain[3]

STRONG EFFICACY

- Imatinib is an antiproliferative TKI with **potential best-in-class improvements in PVR and 6MWD** (45 meters[3]) based on Phase 3 IMPRES and Phase 2 studies
- Imatinib is approved for Oncology indications but likely not approved for PAH because of a combination of adverse events and **high discontinuation rate**, but hit primary endpoint

1st

oral once-daily anti-proliferative

INFORMED DESIGN

- IKT-001 is a novel **prodrug of imatinib designed to retain imatinib's efficacy** potential but with **improved GI tolerability**
- **Titration-based Phase 3 IMPROVE-PAH study** plus 2 decades of imatinib experience incorporated into study protocol
- Target: Improved study design with more patients "managed" to the highest tolerable dose **increases probability of success**

Late Stage

Phase 3 program currently enrolling

POSITIONED TO EXECUTE

- **Single pivotal Phase 3 enrolling** now across 26 countries and 46 sites (and growing)
- **Orphan drug designation, IP runway to 2044 & 505(b)(2) path**
- Executive team with deep PAH / CV experience
- **Cash runway to Phase 3** topline data readout[4]

PVR = pulmonary vascular resistance; 6MWD = 6-minute walk distance; GI = gastrointestinal | TKI = tyrosine kinase inhibitor
(1)Hoeper M, et al. Eur Respir J. 2017 (2) 2025 annual reports from Janssen, United Therapeutics, Liquidia, Bayer, and Merck (3 Placebo-adjusted 6MWD Improvement of 45m at Week 24 in patients at 400 mg for >50% of Treatment (4) Assuming the full and timely exercise of the outstanding Series A and B Warrants

Experienced Leadership with Deep Expertise in PAH

MARK IWICKI
Chief Executive Officer

CHRIS CABELL, MD MHS FACC
Head of R&D, Chief Medical Officer

DAVID McINTYRE BEC CPA LLB MBA
Chief Financial Officer

JOHN ADAMS, PHD
Chief Scientific Officer

TIM PIGOT
Chief Commercial Officer

JEFF KAGY
Chief Human Resource Officer

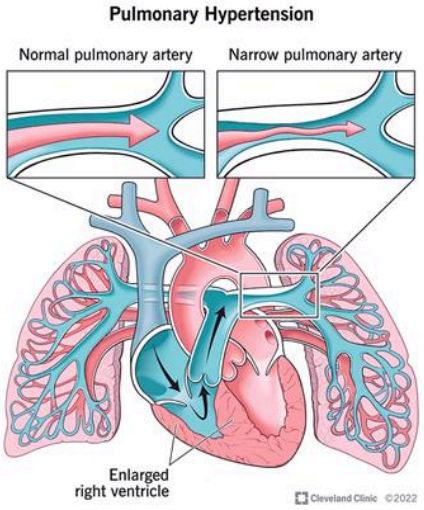
NOVARTIS GILEAD ARENA PHARMACEUTICALS Duke UNIVERSITY MERCK ANTHOS Pfizer

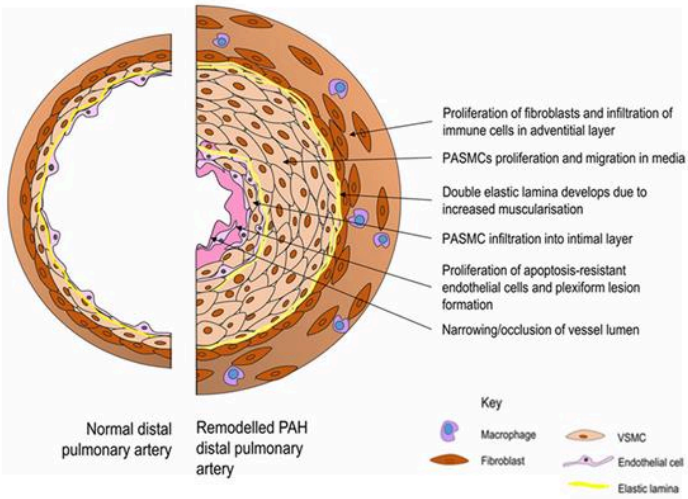
aerovate therapeutics Reneo Pharmaceuticals Iambic zurabio HeartWare MYOKARDIA A T P Apple Tree Partners

INHIBIKASE THERAPEUTICS

PAH is Progressive Disease Driven by Uncontrolled Cell Proliferation

Proliferation of vascular cells drive vascular remodeling, raising pulmonary artery pressure and leading to progressive right ventricular heart failure and ultimately death





5 PASMCs = Pulmonary artery smooth muscle cells; Vascular smooth muscle cells

PAH: Orphan Disease with ~30% 5-Year Mortality Despite Aggressive Treatment

~50,000
People with PAH in the US[1]

52 years
Average age at diagnosis[2]

~26,000
People with PAH in the EU5[1]

15
Approved vasodilators
(across the prostacyclin, nitric oxide, and endothelin pathways)

~80%
Female[2]

1
Approved antiproliferative

High Unmet Medical Need

Progressive and Life Threatening

- ~30% 5-year mortality[3] despite high diagnosis rate and specialist directed treatment with vasodilator therapies
- Progressively worsening symptoms

Reduced Quality of Life

- Chronic breathlessness, and fatigue
- Significant limitation on activities of daily living
- Dizziness, chest pain, anxiety and depression

High Economic Burden

- Average monthly U.S. healthcare costs ~$6,850-$15,650[4]
- Acute all cause hospitalization rate of 700 per 1000 patients per year among Medicare or Medicaid patients[4]
- Substantial indirect costs due to work loss, caregiver time and disability[4]

1 CVrG Market Strategies report Jun 2025; 2 Badlam et al; CHEST 2021; 3 Hoeper M, et al. Eur Respir J. 2017; 4 Watzger et al; Pharmacoeconomics 2025

Patient and Treatment Journey in PAH

Progressive Pulmonary Vasculopathy Driving Right Heart Failure And Death

Low Risk

3% Risk Of 1 Year Mortality

WHO Functional Class 1&2

6MWD > 440 meters

- No or Slight limitation of physical activity. Ordinary physical activity causes dyspnea, fatigue, chest pain, or near syncope

Intermediate Risk

4-19% Risk Of 1 Year Mortality

WHO Functional Class 3

6MWD 165 - 440 meters

- Marked limitation in physical activity. Less than ordinary activity causes undue dyspnea, fatigue, chest pain or near syncope

High Risk

>20% Risk Of 1 Year Mortality

WHO Functional Class 4

6MWD < 165 meters

- Inability to carry out any physical activity without symptoms. Signs of RHF. Discomfort increased by any physical activity. Dyspnea and fatigue present at rest

1st Line of Therapy ← → **Add On Therapy With Declining Health Status** ← →

First Line Therapy

- Phosphodiesterase type 5 (PDE5) inhibitors plus endothelin receptor antagonist (ERA) combination

Add-on Therapy

- Oral / inhaled prostacyclin or Antiproliferative (sotatercept)

Last Line Drug Therapy or Lung Transplant

- Infused Prostacyclin, Antiproliferative (Sotatercept)

7 WHO=World Health Organization; 6MWD= 6 Minute Walk Distance

Antiproliferatives Expected to Rapidly Grow the PAH Market

IKT-001 has the potential to be the First Once-Daily Oral Antiproliferative for PAH

Vasodilators

Prostacyclin Pathway ($3.94B)

Epoprostenol, Treprostinil, Iloprost, Selexipag

Endothelin Pathway ($2.53B)

Ambrisentan, Bosentan, Macitentan

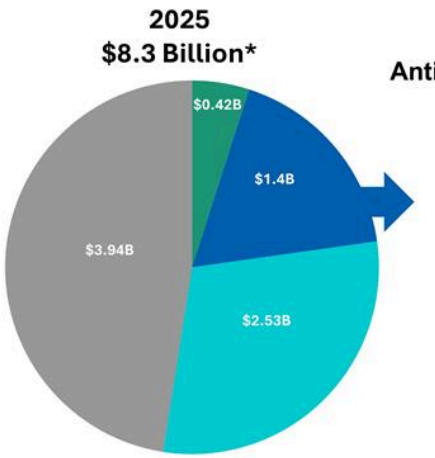
Nitric Oxide Pathway ($.42B)

Sildenafil, Tadalafil, Riociguat

Antiproliferatives

Activin Signaling ($1.44B)

Sotatercept - subcutaneos injection



Antiproliferative Segment Could Reach $8 Billion by 2034**

- US sales of sotatercept expected to reach **$2–$2.5 Billion in 2026** and continue growing
- Antiproliferatives with unique **Disease Modifying Properties** & patient friendly administration expected to become a **Cornerstone of PAH Therapy**

* 2025 annual reports from Janssen, United Therapeutics, Liquidia, Bayer, and Merck; ** Assumes at least 32,000 annual regimens of an antiproliferative using a net price of $250k per regimen per year


IKT-001

IKT-001's Path to Best-in-Class in PAH

IKT-001 On Track to be the First Oral Antiproliferative with a Strong Potential Profile

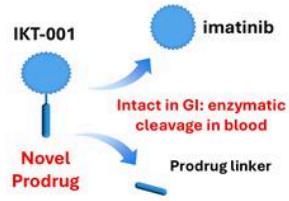
1 Class-Leading Efficacy*

45 Meters*

45m Placebo-adjusted Improvement in Phase 3 IMPRES Study**

- IKT-001 has demonstrated pre-clinical efficacy in PAH comparable to imatinib

2 Improved Safety Profile*



Improved GI Side Effect Profile

IKT-001 remains intact in the GI tract enabling 18X ↓ inhibition of c-Kit (implicated in GI side effects)

- No negative effect on gastric emptying

ATS 2026

Modernized Phase 3 Study Design

- **12-week dose titration** to maximum tolerable dose, including potential for down titration
- Defined classification of **edema events between drug-related and clinical worsening**
- Exclusion of **anticoagulants**

IMPROVE-PAH

3 Clinical Benefits*

Oral

Once-daily

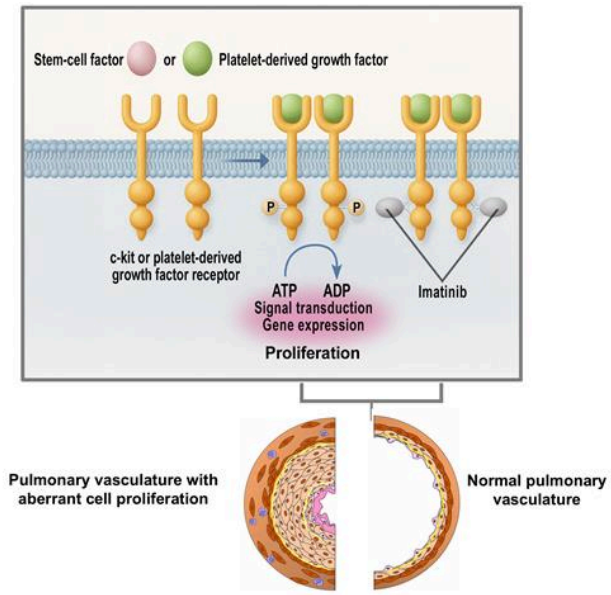
Reduced Monitoring

Antiproliferative

* Potential profile – IKT-001 is in phase 3 clinical development
** Hoeper et al., Circ. 2013 Placebo-adjusted 6MWD Improvement of 45m at Week 24 in patients at 400 mg for >50% of Treatment | GI = Gastro-intestinal ; 6MWD = 6-minute walk distance

INHIBIKASE THERAPEUTICS

Imatinib Mechanism of Action Targets the Underlying Cause of PAH

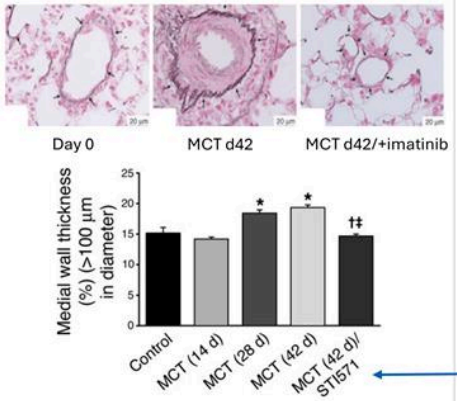


- Overactive kinases implicated in aberrant cell proliferation and migration in the pulmonary vasculature
- Imatinib inhibits the tyrosine kinase activity of PDGFRs and c-Kit, blocking cell signaling that drives vascular remodeling
- Antiproliferative and proapoptotic anti-remodeling mechanisms confirmed with imatinib in human pulmonary artery cells[1]

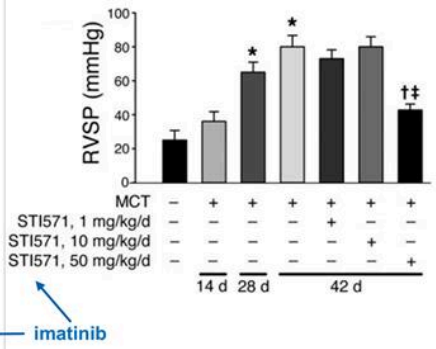
Modified from Savage and Antman, 2005 and Barst, R J Clin Invest 2005 [1]Schermuly et al., JCI 2005

Imatinib Demonstrated Reversal of PAH in Standard Animal Model[1]

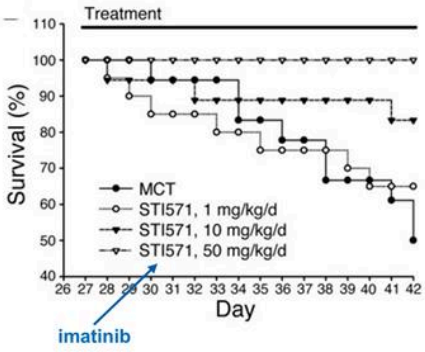
Reversal of pulmonary vascular remodeling



Reversal of increased right ventricular pressure



Improved survival



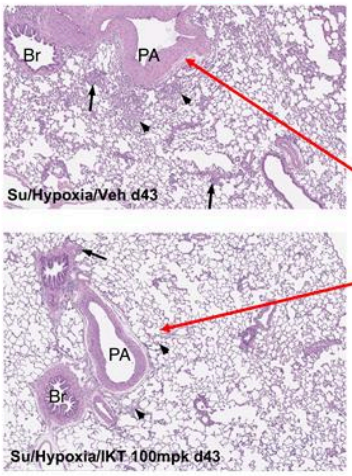
Imatinib reverses pulmonary vascular remodeling, right ventricular pressure/remodeling and improves survival

STI571 = imatinib; MCT = monocrotaline

*P < 0.05 versus control; †P < 0.05 versus MCT at day 28 or hypoxia at day 21; ‡P < 0.05 versus MCT at day 42 or hypoxia at day 35. [1]Schermuly et al., JCI 2005

IKT-001 Retains Imatinib Efficacy & PAH Reversal Profile in Pre-Clinical Model

Lung Histology



Legend: Perivascular to interstitial infiltration of lymphocytes, macrophages, and rare neutrophils (black arrows) is seen multifocally, in addition to increased alveolar macrophages (black arrowheads). Arterioles have a visibly thickened tunica media throughout; medial hypertrophy also seen in the captured pulmonary artery (PA) profile. A bronchiole (Br) is indicated.

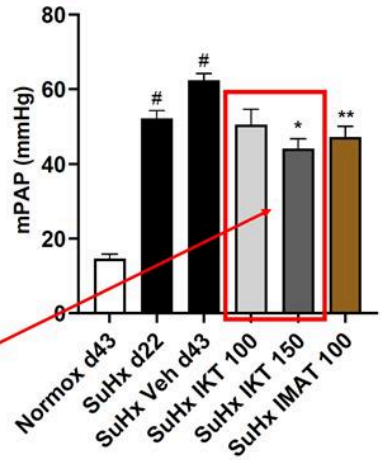
ATS 2026

Background: Imatinib has previously been shown to reverse PAH in the Sugen-hypoxia Rat Model(1)

Like imatinib, chronic treatment with IKT-001 reversed thickening of pulmonary arteries

IKT-001 dose dependently improved pulmonary hemodynamics including mPAP and demonstrated disease reversal

Mean Pulmonary Artery Pressure (mPAP)



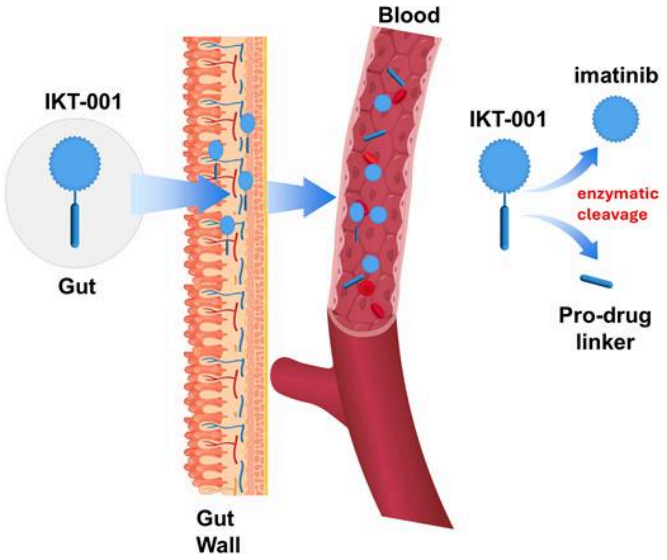
# p<0.0001 vs normoxia, * p<0.0005 v SuHx Veh d43, **p<0.007 v SuHx Veh d43

(1) Schermuly et al., 2005.
Data presented at American Thoracic Society Conference in Orlando, May 2026

INHIBIKASE THERAPEUTICS

IKT-001 Dramatically Reduces GI Imatinib Exposure to Drive Tolerability*

IKT-001 is Stable in the Gut and has Rapid Bioconversion in the Blood



Pre-absorption: IKT-001 Is Stable In Gut*

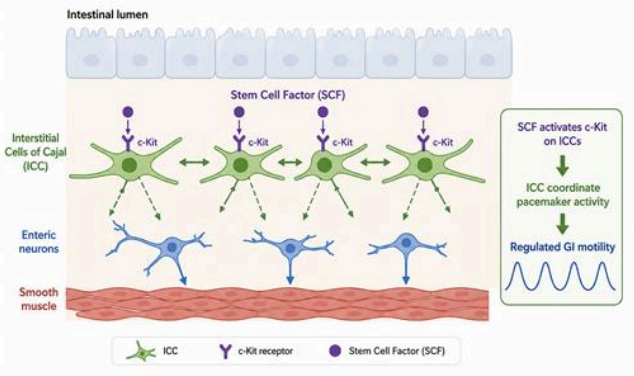
- **GI tract protected** from direct imatinib exposure
- **~20x less inhibition of tyrosine kinase activity** vs imatinib
- **2.5x improvement in GI tolerability** vs imatinib in non-human primates

Post-absorption: Rapid & Complete Conversion In Blood

- **Half-life of <5 minutes in** human plasma
- **No IKT-001 detected** in rat systemic plasma after 10 min

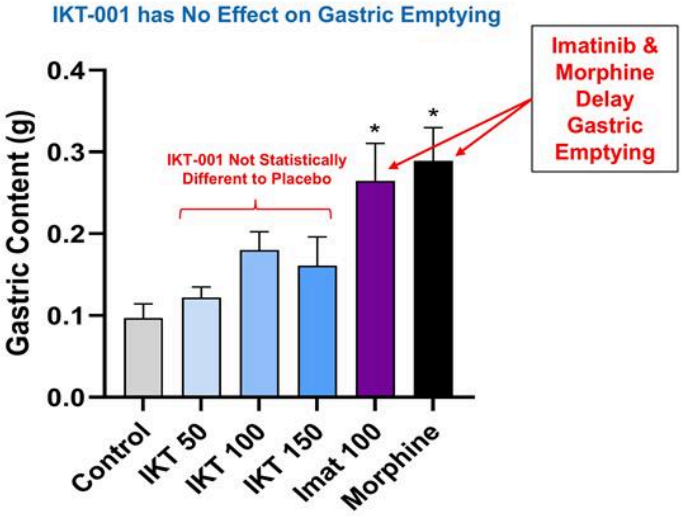
GI = gastrointestinal | *In preclinical studies – IKT data on file

IKT-001 Has Reduced Gut Motility Impairment vs Imatinib



- **Imatinib is a potent inhibitor of c-Kit**
- **c-Kit inhibition** in gut pacemaker cells (ICC) causes **gut motility disorders**[2]

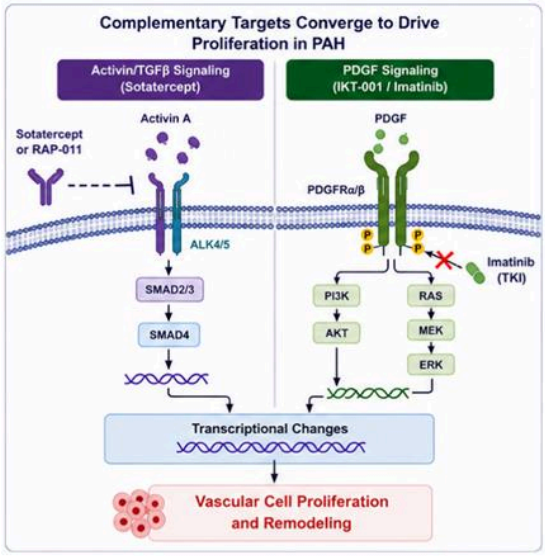
IKT-001 remains intact in the stomach and intestine and is 18X less potent at c-Kit than imatinib[1]

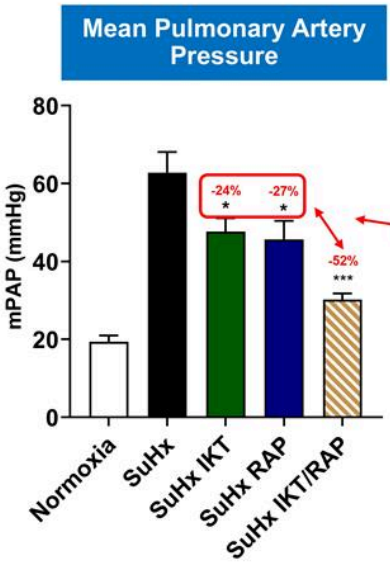


*p<0.01 vs Control, Data presented at American Thoracic Society Conference in Orlando, May 2026 [1]Data on file, [2]Maeda 1992..

Additive Efficacy Via Combination of IKT-001 + Activin Signaling Inhibitor

Activin Signaling Inhibitor (RAP – sotatercept surrogate) Plus IKT-001 in PAH Sugen-Hypoxia Model





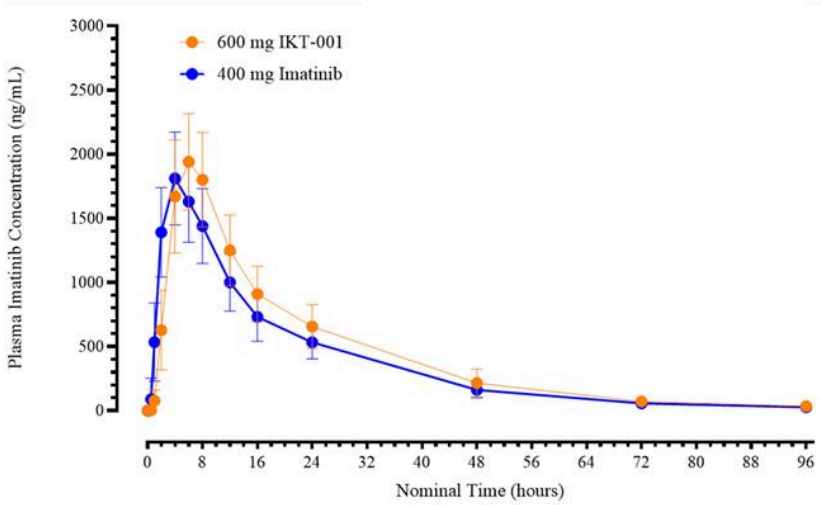
- **Statistically significant additive improvements in PAH hemodynamics** achieved with combination treatment (IKT-001 + ASI)
- **Combination treatment** (IKT-001 + ASI) was **well tolerated**

*Pre-clinical data on file with IKT, * p<0.05 vs SuHx, *** p<0.0001 v SuHx IKT
. ASI = Activin Signaling Inhibitor

INHIBIKASE THERAPEUTICS


Clinical
Rationale

SAD Study in Healthy Volunteers Establishes Bioequivalence

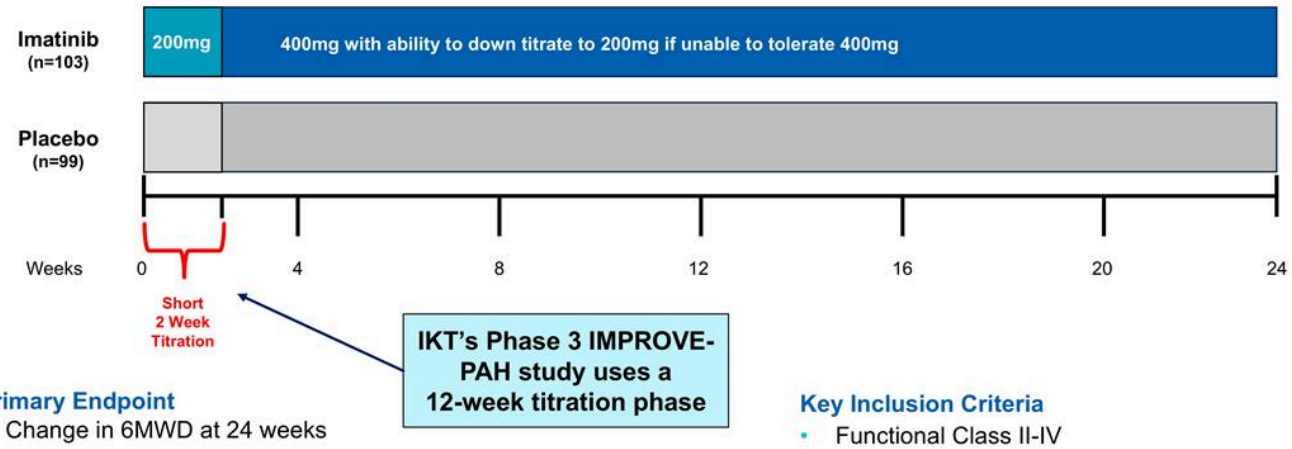


IKT-001 Dose	Bioequivalent Imatinib Dose
300 mg QD	230 mg QD
400 mg QD	306 mg QD
500 mg QD	383 mg QD

18 IKT Data on File; SAD: Single ascending dose study

IMPRES – Imatinib Phase 3 Study

Randomized controlled trial to assess the efficacy and safety of imatinib once daily (n=202)



Primary Endpoint

- Change in 6MWD at 24 weeks

Secondary Endpoints

- Changes in hemodynamics (PVR, CO, mPAP, RAP) at 24 weeks
- Time to clinical worsening

Key Inclusion Criteria

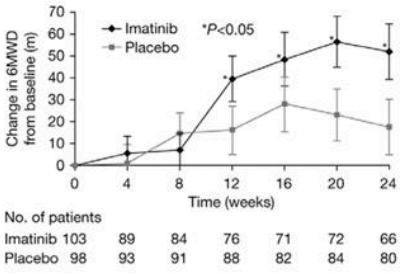
- Functional Class II-IV
- 2 or more background PAH therapies
- PVR ≧ 800 dynes.s.cm^{-5}
- 6MWD ≥150 meters and ≤ 450 meters

PVR = pulmonary vascular resistance; CO = cardiac output; mPAP = mean pulmonary atrial pressure; RAP = right atrial pressure; 6MWD = 6-minute walk distance

IMPRES: Statistically Significant Improvements in Function & Hemodynamics

Placebo-adjusted 32-meter improvement in 6MWD and 32% reduction in PVR at week 24
Majority of Subjects on 200 mg of Imatinib at End of Study

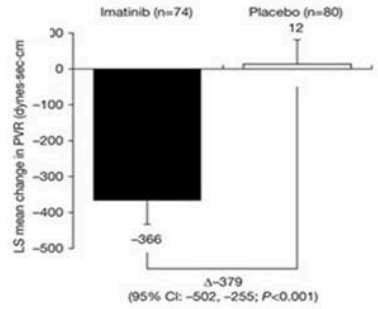
Change in 6-Minute Walk Distance



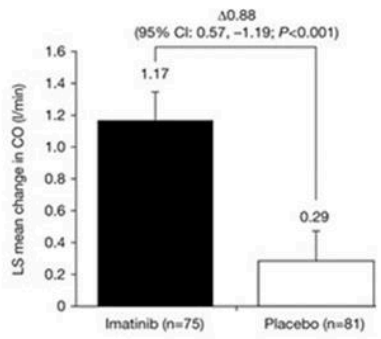
No. of patients

Imatinib	103	89	84	76	71	72	66
Placebo	98	93	91	88	82	84	80

Change in Pulmonary Vascular Resistance



Change in Cardiac Output



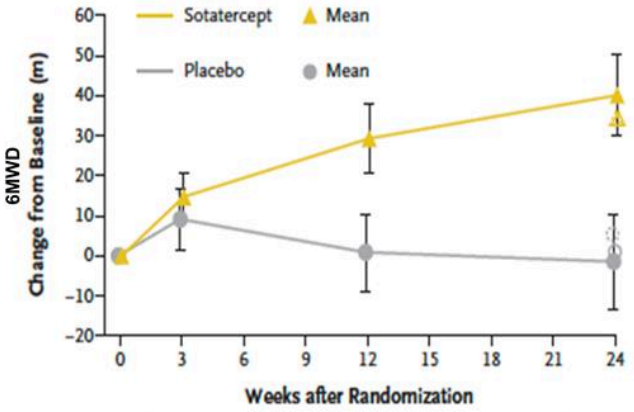
IMPRES hit its primary endpoint along with key secondary endpoints

PVR = Pulmonary Vascular Resistance; 6MWD= 6 Minute Walk Distance test
Hoeper et al; Circulation 2013

Imatinib Showed Comparable Efficacy to Sotatercept

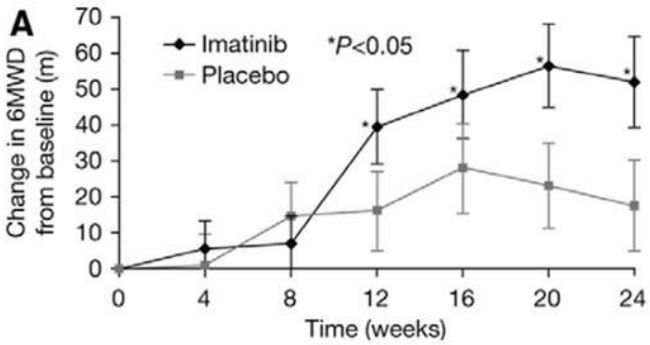
Majority of Sotatercept Subjects at Highest Dose While Majority of Imatinib Subjects at Lowest Dose

Sotatercept Phase 3 STELLAR[1]



1⁰ Endpoint: Median Treatment Difference = 33.4m
Majority of patients at highest dose of 0.7 mg/kg

Imatinib Phase 3 IMPRES[2]



1⁰ Endpoint: Mean Treatment Difference = 32m
Majority of patients at lowest dose of 200 mg

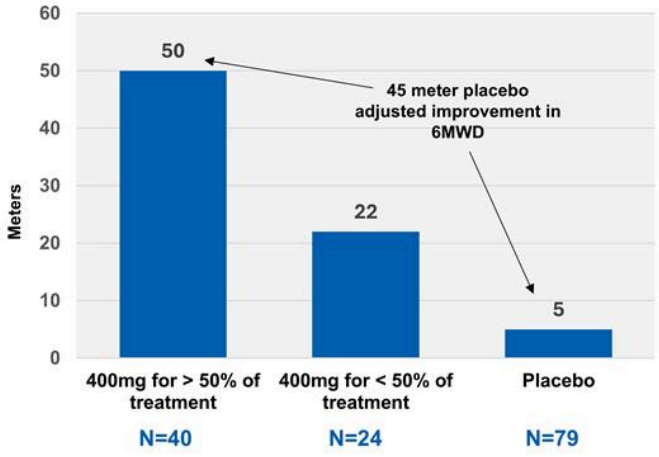
[1]Hoeper et al; NEJM 2023; [2]Hoeper et al; Circulation 2013

Note: Head-to-head trials were not conducted with Sotatercept and Imatinib and these trials had different trial designs, patient enrollment criteria and treatment regimens. In addition, the applicable measurements for the referenced trials were observed over different time periods and using different assays. As a result, the safety data from these trials may not be directly comparable

IMPRES: Patients Able To Sustain 400mg Dose Showed Greater Improvements

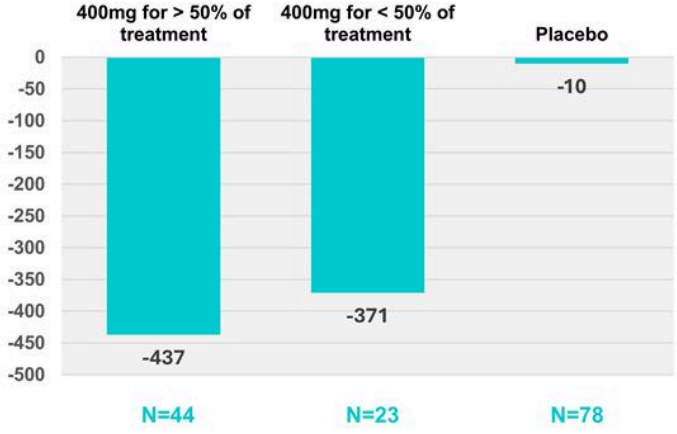
Placebo-adjusted 6MWD Improvement of 45m in Patients at 400mg for >50% of Treatment

Changes in 6MWD at Week 24



(Baseline*: 355 meters imatinib; 366 meters placebo)

Changes in PVR at Week 24



(Baseline*: 1202 dynes/sec/cm^{-5} imatinib; 1181 dynes/sec/cm^{-5} placebo)

*Baseline data is not available for the sub-population of patients on 400mg of imatinib for > 50% of treatment so baseline data above reflects entire study population.
PVR= Pulmonary vascular resistance; 6MWD = 6-minute walk distance

Hoeper et al; Circulation 2013 Suppl. Appendix

Targeting An Improved AE Profile To Help Patients Maintain Higher Doses

Imatinib Phase 3 IMPRES[1] Trial

	Imatinib n=103 (%)	Placebo n=98 (%)
Adverse Events	100 (97)	94 (96)
Nausea	57 (55)	23 (24)
Peripheral edema	45 (44)	20 (20)
Diarrhea	36 (35)	19 (19)
Vomiting	31 (30)	10 (10)
Periorbital edema	30 (29)	7 (7)

Improved Protocol & Patient Management

IKT-001 reduced c-Kit inhibition in the GI tract expected to reduce AEs

IKT-001 and IMPROVE-PAH Study Design Combine to Mitigate IMPRES AE Challenges

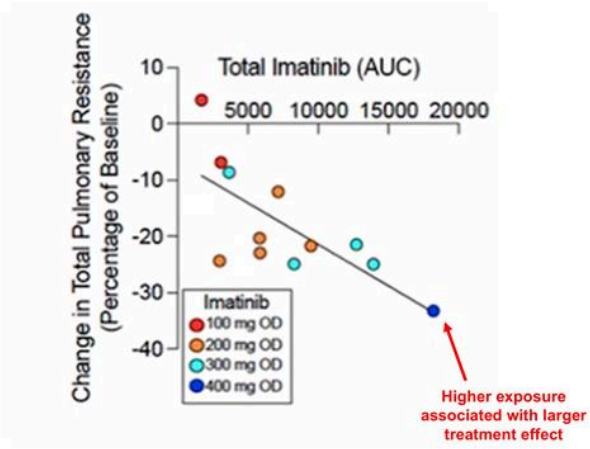
- IKT-001 is designed to have an improved AE profile to enable patients to tolerate higher doses
- IMPROVE-PAH gradual up-dosing titration and modernized study design to reduce significant AEs and discontinuations
- Defined classification of edema events between drug-related and clinical worsening

[1]Hoeper et al; Circulation 2013 AE = Adverse event; GI = Gastrointestinal

Recent Study* of Imatinib Supports Findings from IMPRES

Doses between 200mg – 400mg delivered meaningful efficacy response

Dose Dependent Improvement in Primary Endpoint



Percentage change in total pulmonary resistance from baseline at 60 days in relation to plasma level (area under curve in µg*h/L) of imatinib at steady state (red-100mg QD, orange-200mg QD, cyan-300mg QD, blue-400mg QD)

2025 Publication Using Imatinib in PAH

PAH patients (13/17) with implanted devices to assess continuous cardiac function

Comparable patient characteristics and baseline PVR scores to modern PAH trials

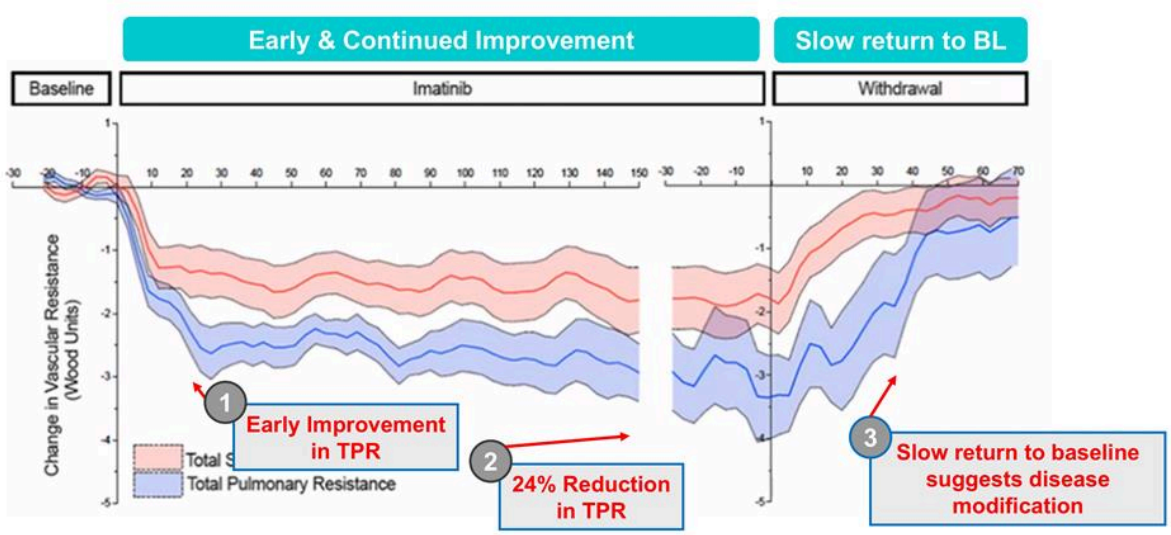
Average lower dose than IMPRES nevertheless delivered significant reduction in TPR and improvement in 6MWD

Dose adjustments to address tolerability allowed most patients to remain in the study

*Rothman et al., AJRCCM 2025.

Rapid & Sustained Hemodynamic Effect With Evidence of Disease Modification

Imatinib Delivered a 24% Reduction in Total Pulmonary Resistance (TPR)

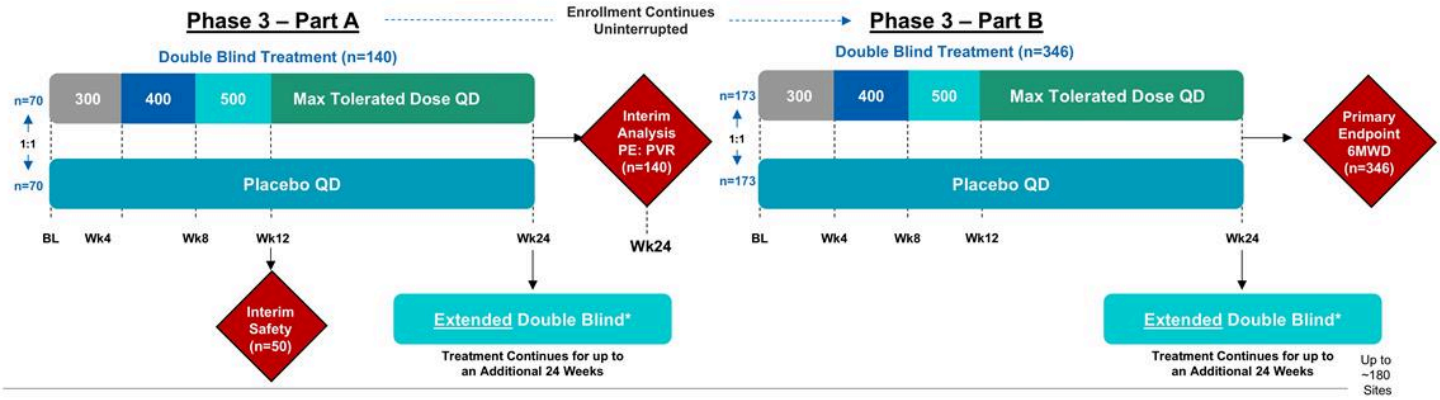


*Rothman et al., AJRCCM 2025.


IKT's Clinical Program

Phase 3 Enrollment Ongoing

IMPROVE-PAH Phase 3 Study of IKT-001



Primary Endpoints:

- PVR (Part A) & 6MWD (Part B)

Key Secondary Endpoints:

- Time to Clinical Worsening
- WHO Functional Class

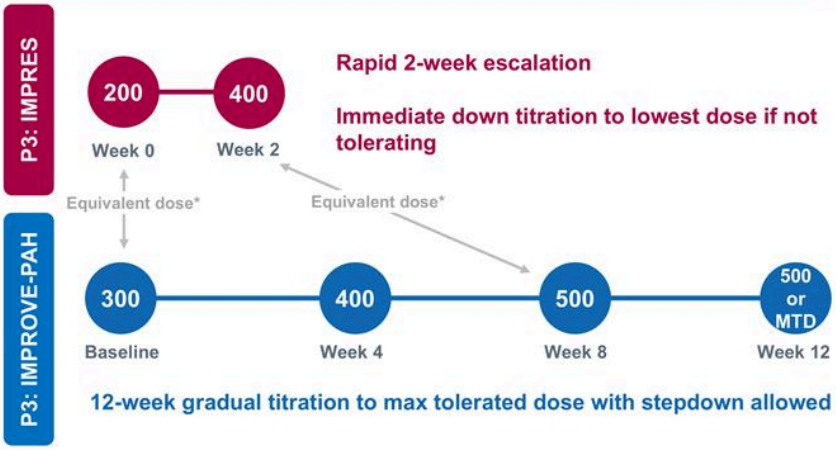
Key Inclusion / Stratification:

- WHO Group 1 PAH
- Baseline Right Heart Catheter performed during screening period:
  - PVR of ≥400 dynes/sec/cm^{-5} ; PCWP ≤15 mmHg; mPAP >20 mmHg
  - PVR enrichment criteria to ensure population baseline PVR >700 dynes/sec/cm^{-5}
- 6MWD ≥100 and ≤475 meters
- Previous sotatercept allowed if d/c 6 months prior to screening and no serious bleeding event history
- Stratification by PAH etiology and ERS/ESC Risk Score

*Patients completing Wk 48 may transition to Open Label Extension (OLE). Additionally, patients who have not completed follow-up at the time of study unblinding or who otherwise experience a clinically worsening event during the extended double blind treatment period may transition to OLE

Power of Titration & Improved Study Design in IMPROVE-PAH

1 Gradual titration to max. tolerated dose to retain IMPRES efficacy



2 Improved administration to ↓ discontinuations

Gradual Titration
Slower pace to Cmax steady-state allows patient to adapt to IKT-001 and may decrease adverse events

Flexible Dose Management
Stepwise down-titration may keep participants on treatment at MTD and reduce discontinuations

Nighttime Administration
Taking IKT-001 at night may reduce the daytime burden of transient nausea or fatigue

DESIGN OBJECTIVES Improved early tolerability → Fewer discontinuations → More patients reach / maintain effective exposure → Greater opportunity to realize maximal efficacy

P3: Phase 3. MTD = Maximum Tolerated Dose. Sources: Hoeper et al. Circulation. 2013;127:1128–1138; Gleevec (imatinib) U.S. Prescribing Information. * See slide 18 for precise bioequivalence

Why IMPROVE-PAH Phase 3 Has Higher Probability of Success

Modern Phase 3 Design + IKT-001's Better Tolerability = Maximum Efficacy & Improved Trial Outcome

Benefit of 20+ Years of Imatinib Clinical Experience

- ✓ **Gradual 12-week Titration** to max dose (vs 2 Weeks in IMPRES) ↑ **efficacy** ↓ **discontinuations**
- ✓ **45m 6MWD** for patients on high dose in IMPRES - ↑ **efficacy**
- ✓ **IKT-001 remains stable** in the gut with **18x ↓ in c-Kit inhibition** - ↑ **GI tolerability**
- ✓ **Defined guidance** for edema events - ↓ **discontinuations**
- ✓ **Exclusion of anticoagulants** - ↓ **events**
- ✓ **Independent adjudication** of worsening events - ↓ **event misclassifications**
- ✓ **Evening dosing** with water and meal - ↓ **transient nausea or fatigue**

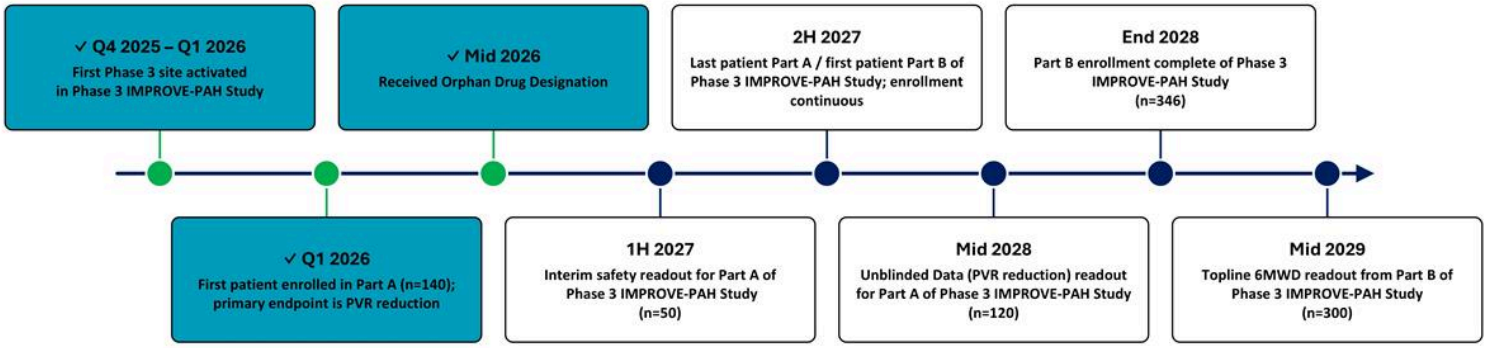
> **"Study-drug discontinuations were comparatively high** in the [IMPRES] study"[1]
>
> "… causes may include … **a lack of experience with the use of imatinib** among PAH specialists"[1]
>
> "… worsening events in the imatinib group… **transient…associated with known imatinib side effects rather than events reflecting disease progression**"[1]

[1]Hoeper et al. Circulation. 2013;127:1128-1138

IKT's Development and Projected Timelines

IKT-001 Phase 3 Enrollment Ongoing

26	46 of 180
countries approved, including USA & EU	planned sites active



Potential for the 1st Once-Daily Oral Antiproliferative

Global Pivotal Phase 3 Program with Multiple Clinical Readouts

First Half 2027 – Interim Safety Readout
Mid 2028 – Part A - PVR Efficacy Data
Mid 2029 – Part B – 6MWD Efficacy

- ✓ Very High Unmet Need Market with 30%, 5 Year Mortality
- ✓ $8.3B market expected to rapidly grow to $14B by 2034(1)

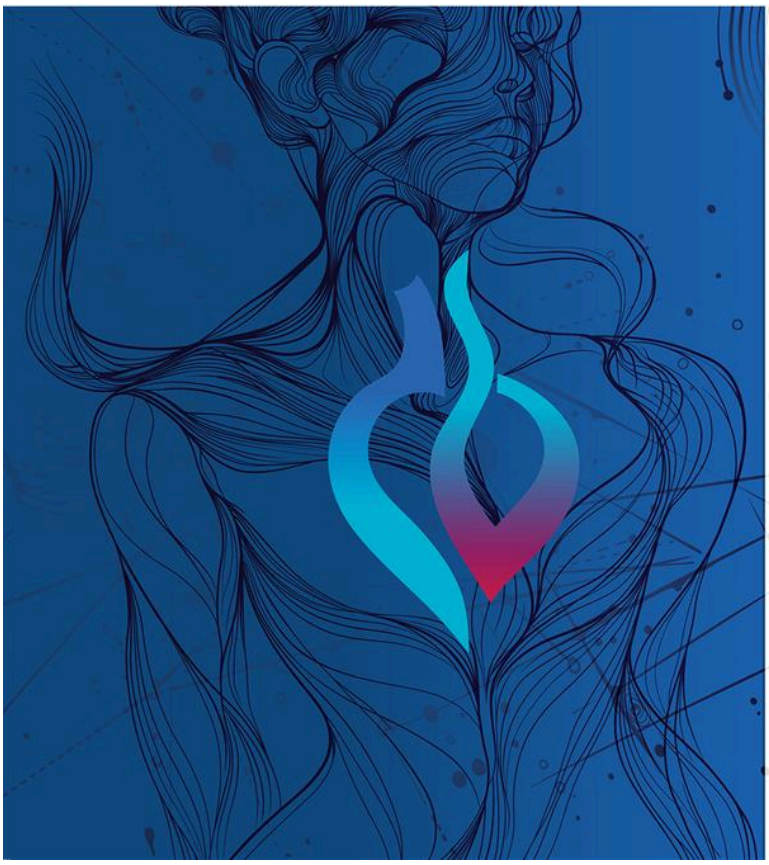
- ✓ Antiproliferatives are the first disease modifying agents and expected to reshape the PAH treatment algorithm
- ✓ IKT-001 expected to be the first once-daily oral antiproliferative

- ✓ IKT-001 designed to maintain potential class leading 45 meter improvement in 6MWD
- ✓ Pro-drug expected to significantly improve GI tolerability

- ✓ Orphan Drug Designation & 505(b)(2) regulatory pathway
- ✓ Long intellectual property (NCE) potential through 2044
- ✓ Cash runway through Phase 3 Part B Topline Data readout*

[1]https://www.precedenceresearch.com/pulmonary-arterial-hypertension-market,
* Assuming the full and timely exercise of the outstanding Series A and Series B Warrants

6MWD= 6 Minute Walk Distance test, NCE= new chemical entity



Thank You