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): October 9, 2024

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.)**

3350 Riverwood Parkway SE, Suite 1900 Atlanta, Georgia	**30339**
(Address of Principal Executive Offices)	**(Zip Code)**

Registrant's Telephone Number, Including Area Code: (678) 392-3419

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 October 9, 2024, Inhibikase Therapeutics, Inc. (the "**Company**") made available in the investor relations section of its website a new company presentation. A copy of the presentation is attached hereto as Exhibit 99.1 and is incorporated herein by reference.

The information furnished in this Item 7.01 of this Current Report on Form 8-K and Exhibit 99.1 attached hereto shall not be deemed "filed" for the 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. The information in this Item 7.01 of this Current Report on Form 8-K is not incorporated by reference into any filings of the Company made under the Securities Act of 1933, as amended, or the Exchange Act, whether made before or after the date of this Current Report on Form 8-K, regardless of any general incorporation language in the filing unless specifically stated so therein.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Number	Description
99.1	Company Presentation
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: October 9, 2024

INHIBIKASE THERAPEUTICS, INC.

By: /S/ MILTON H. WERNER
Milton H. Werner, Ph.D.
President and Chief Executive Officer

Exhibit 99.1

Inhibikase.com

Nasdaq : IKT

This presentation contains information that may constitute “forward-looking statements” within the meaning of Section 27A of the Securities Act, and Section 21E of the Securities Exchange Act of 1934, 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” and similar expressions. 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, regulatory approvals, product candidate development, results of operations or financial condition 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 and its quarterly Form 10-Q (the “SEC Filings”). Important factors that could cause actual results to differ materially from those in the forward-looking statements are set forth in the Company’s SEC filings, including under the caption "Risk Factors”.

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 Therapeutics

Developing innovative medicines across the therapeutic spectrum

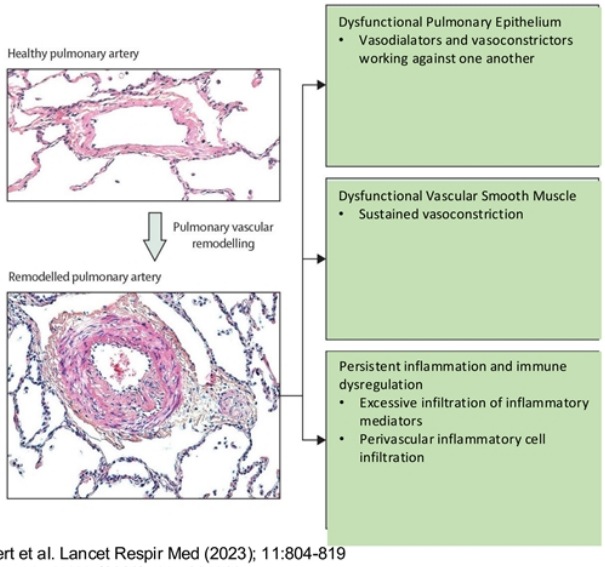
- Multi-therapeutic pipeline across cardiopulmonary disease, neurodegenerative disease and cancer.
- **IkT-001Pro: Prodrug of imatinib mesylate to be evaluated in Pulmonary Arterial Hypertension (PAH). Clinical studies completed to quantify imatinib delivery. PAH has a $7B+ global addressable market.[1]**
  - IND filed August 9, 2024, 'Study May Proceed' Letter received from FDA September 9, 2024
  - Phase 2 interim safety readout anticipated 4Q25/1Q26
  - Phase 2 top line results anticipated 4Q26
  - Robust patent portfolio: compositions to 2033, with extensions to 2039, PAH use to 2044
- **Risvodetinib (IkT-148009):** Selective c-Abl inhibitor. Phase 2 results expected November, 2024 measuring disease modification in untreated Parkinson's disease. FDA Phase 3 meeting planned December, 2024. Phase 3 entry anticipated 2025. $12B+ global addressable market.[2]
- Orphan designations: **IkT-001Pro** in pulmonary arterial hypertension under review.
- Highly-experienced management team, consultants, Board of Directors and Scientific Advisory Board.

[1]From Biomedtracker (Citeline Commercial), 2024 (https://www.biomedtracker.com/indicationreport.cfm?indid=245#PipelineChart) ; [2]Vision Research, 2022

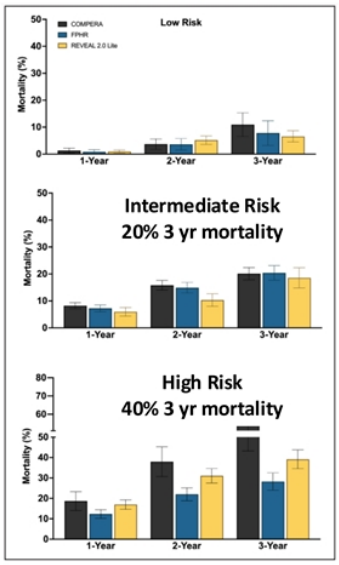
Inhibikase Therapeutics

IkT-001Pro for Treatment of Pulmonary Arterial Hypertension

Complex disease etiology leading to narrowing of lung microvasculature[1]



PAH mortality on par with many Cancer Indications[2]



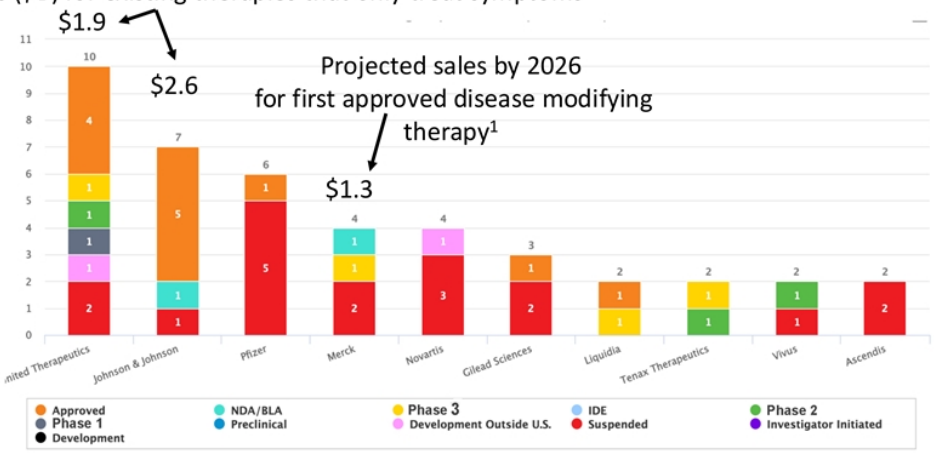
COMPERA: Comparative, Prospective Registry of Newly Initiated Therapies for Pulmonary Hypertension; **FPHR:** French Pulmonary Hypertension Registry; **REVEAL:** The Registry to Evaluate Early and Long-Term PAH Disease Management.

[1]Humbert et al. Lancet Respir Med (2023); 11:804-819
[2]Chang et al., JAHA (2022); 11:e024969

Inhibikase Therapeutics

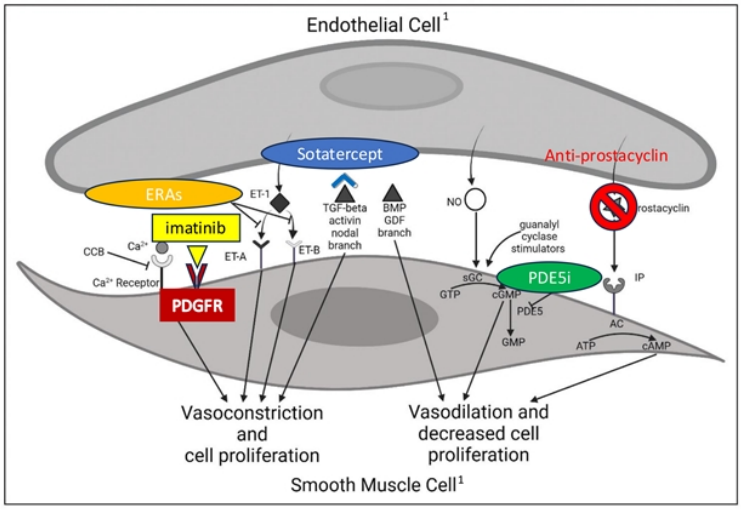
Only 1 approved Disease Modifying drug: Sotatercept (Merck). No oral disease-modifying therapies approved

2023 Sales ($B) for existing therapies that only treat symptoms[1]



Currently a $5B market in the US alone; $.7.6B globally[1]

[1]From Biomedtracker (Citeline Commercial), 2024 (https://www.biomedtracker.com/indicationreport.cfm?indid=245#PipelineChart) ;



Imatinib inhibits the Platelet-derived growth factor receptor (PDGFR), blocking cell signaling that drives proliferation. Shown to be disease-modifying[2]

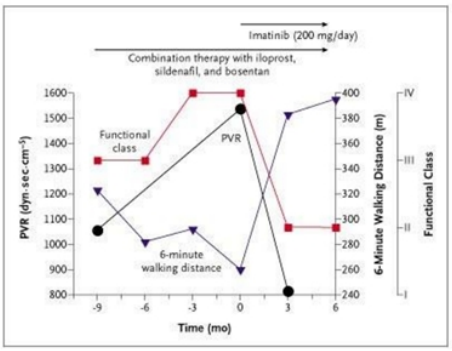
Sotatercept blocks a different proliferative pathway. Shown to be disease-modifying and approved.[3]

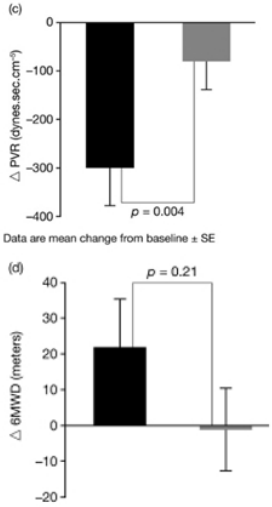
[1]Adapted from Tuttle, et al., Curr Opin Nephrol Hypertens 2024, 33:494–502
[2]Hoeper et al. Circulation. 2013;127:1128-1138; [3]Hoeper et al. New Engl. J. Med.(2023); 16:1478ff

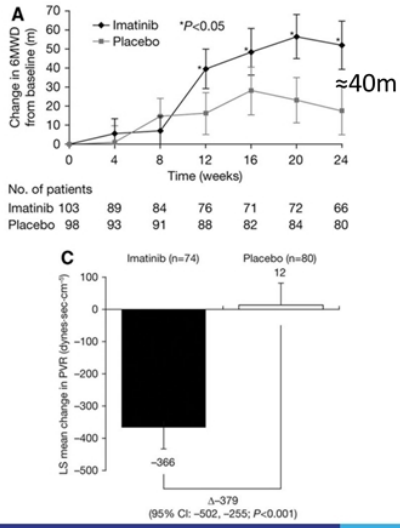
2005: Off-label use of imatinib elicits disease control in advanced PAH patient[1]

2010: Small proof-of-concept trial demonstrates that Imatinib improves hemodynamics and 6MWD in PAH patients.[2]

2013: P3 IMPRES trial -> Imatinib hits stat sig on primary endpoint of 6MWD as well as key secondary endpoint PVR.[3]

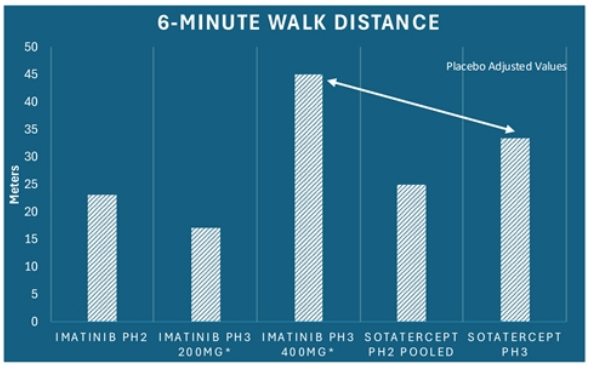




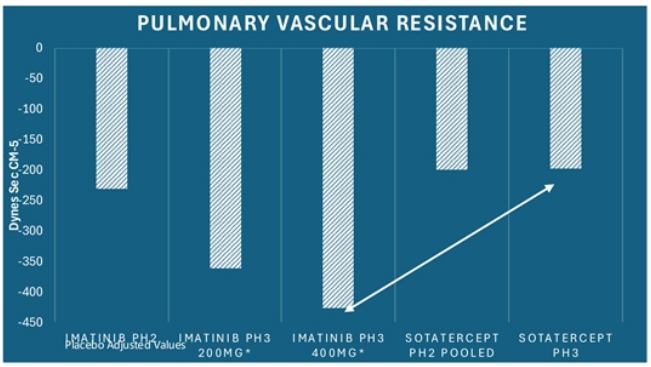


[1]Ghofrani et al, N Engl J Med. 2005;353:1412–1413
[2]Ghofrani et al., Am J Respir Crit Care Med. 2010;182:117
[3]Hoeper et al. Circulation. 2013;127:1128-1138

Imatinib data from Ph3 IMPRES has comparable efficacy to Sotatercept from Ph3 STELLAR data[1-5]



* Patient spent majority of trial at this dose



* Patient spent majority of trial at this dose

Sotatercept only improved disease in 30% of STELLAR participants.[4] There remains a significant unmet need.

[1]Humbert et al, J Engl J Med. 2021; 384:1204-1215; [2]Ghofrani et al., Am J Respir Crit Care Med. 2010;182:117
[3]Hoeper et al. Circulation. 2013;127:1128-1138; [4]Hoeper et al. New Engl. J. Med. (2023); 16:1478ff
[3]No head-to-head trials have been conducted leading to the results shown. As a result no cross-trial comparisons can be made.

Inhibikase Therapeutics

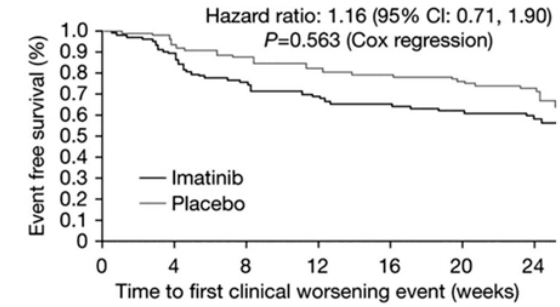
Why imatinib failed to be approved

Inhibikase Therapeutics

Imatinib was not approved because of high discontinuation rate in IMPRES Ph3
AEs mimicked disease symptoms as such mimicry could suggest clinical worsening[1-2]

Adverse Events included:

- **Subdural Hematoma**
- **Edema and Fatigue may mimic symptoms of disease**
- **GI: Gastrointestinal AEs including nausea and diarrhea**



No. of patients

Imatinib	103	93	70	66	60	58	45
Placebo	98	91	85	80	75	72	54

[1]Hoeper et al. Circulation. 2013;127:1128-1138.
[2]Frost et al., J. Heart Lung Transplantation 2015; 34:1366-1375.

ALL patients with subdural hematoma treated with old-style anti-coagulants
Subdural hematoma didn't occur in the absence of anti-coagulants[1-2]

Event	Study	Diagnosis	Anti-coagulant
1	IMPRES core study	SDH	Phenprocoumon
2	IMPRES core study	SDH	Warfarin
3	IMPRES extension study	SDH	Warfarin
4	IMPRES extension study	SDH	Warfarin
5	IMPRES extension study	SDH	Enoxaparin, Warfarin
6	IMPRES extension study	SDH	Phenprocoumon
7	IMPRES extension	Acute SDH	Acenocoumarol
8	IMPRES extension	SDH	Phenprocoumon

Subdural hematoma incidence:
2/103 patients in the core study (1.9%)[1]
6/144 patients in the extension study (4.2%)[2]

Imatinib significantly increases exposure of the anti-coagulants used in these patients via Cyp inhibition.

Anticoagulants*	
Acenocoumarol	• Inhibition of CYP 2C9 by imatinib: ↑ anticoagulation → monitor PT/INR[9]
Phenprocoumon	• Inhibition of CYP 2C9 by imatinib: ↑ anticoagulation → monitor PT/INR[9]
Warfarin	• Inhibition of CYP 2C9 by imatinib: ↑ anticoagulation → monitor PT/INR[9]

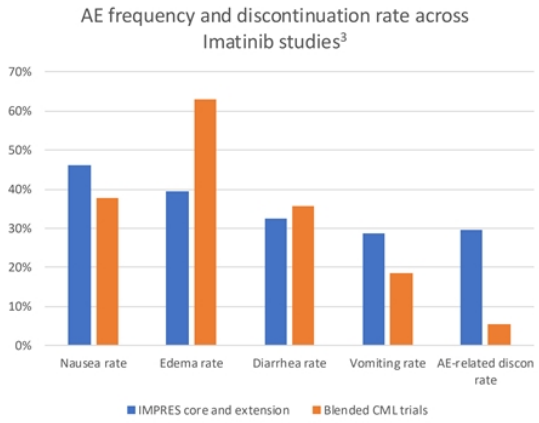
1. ***Anti-coagulant use no longer standard-of-care in PAH.***
2. ***Imatinib has significant DDI with older anti-coagulants used in IMPRES***
   - ***DDI with newer anti-coagulants like Eliquis thought to be modest.***
3. ***Subdural hematoma is a known AE associated with Imatinib therapy in cancer, occurring in <1% of patients with CML on chronic imatinib[3]***

Anti-coagulant use will be prohibited in the upcoming trial with IkT-001Pro

[1]Hoeper et al. Circulation. 2013;127:1128-1138; [2]Frost et al., J. Heart Lung Transplantation 2015; 34:1366-1375; [3]Druker et al, N Engl J Med 2001 Apr 5;344(14):1031-7

Inhibikase Therapeutics

GI and edema adverse event rate on Imatinib is similar in IMPRES and contemporary CML trials. However, discontinuation rate was much higher in IMPRES.



From IMPRES manuscript[1-2]

Most of the AEs reported in the study were similar to those observed previously in association with the use of imatinib in patients with other approved indications. The most frequent AEs were nausea, peripheral edema, diarrhea, vomiting, and periorbital edema. There were no indications of liver toxicity or impaired renal function. However, particular note should be taken of the safety profile of imatinib in PAH because certain early AEs could be mistaken for progression of right heart failure, whereas progressive venous congestion in conjunction with worsening PAH could be interpreted erroneously as an imatinib side effect.

Study-drug discontinuations were comparatively high in the present study compared with previous studies with imatinib for malignant diseases (12% to 44% in studies up to 24 months in duration).[30,31] The exact reasons for this observation are unknown, but potential causes may include effects of the underlying disease and comedications as well as a lack of experience with the use of imatinib among PAH specialists.

Hoeper et al. Circulation 2013

AEs largely similar to those seen in other imatinib studies.

Imatinib induced edema mistaken for worsening of PAH.

High discontinuation rate likely driven by lack of physician experience with drug.

Clinical worsening will be adjudicated by Data Monitoring Committee in planned trial

[1]Hoeper et al. Circulation. 2013;127:1128-1138; [2]Frost et al., J. Heart Lung Transplantation 2015; 34:1366-1375; [3]Druker et al, N Engl J Med 2001 Apr 5;344(14):1031-7

Adverse Event	Placebo (n=98)	Imatinib (n=103)[1]
Nausea	23 (24%)	57 (55%)
Diarrhea	19 (19%)	36 (35%)
Vomiting	10 (10%)	31 (30%)

Frequency of GI disturbance in IMPRES[1]

Adverse Event	Imatinib 200-300mg[2] % Occurrence	Imatinib 350-500mg[2] % Occurrence
Nausea	30%	50%
Diarrhea	4%	33%
Vomiting	13%	11%

Lower frequency of GI disturbance at lower doses[2]

IkT-001Pro was developed to suppress GI adverse events and shown to achieve this in non-human primate studies

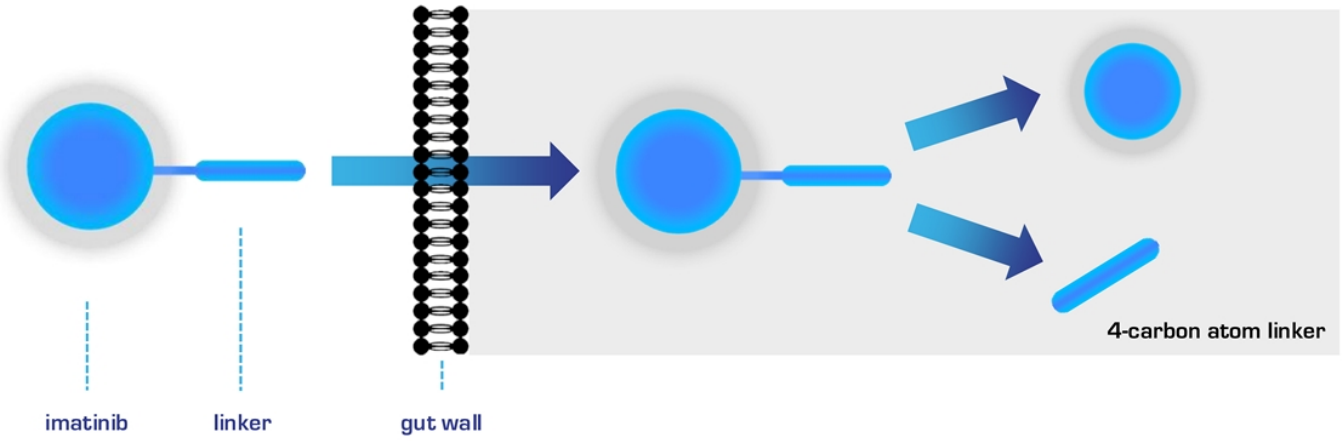
[1]Hoeper et al. Circulation. 2013;127:1128-1138; [2]Druker et al, N Engl J Med 2001 Apr 5;344(14):1031-7

Inhibikase Therapeutics

Imatinib delivery by prodrug

Inhibikase Therapeutics

IkT-001Pro, a prodrug of imatinib, designed to mask imatinib at the gut wall



pH 2-6
(stomach, ileum, duodenum)

pH 7⁺ (blood)
Lifetime in blood < 10 min

Inhibikase Therapeutics

IkT-001Pro:
Anticipated lower GI toxicity alternative to imatinib

Measurement of IkT-001Pro orally delivered once daily in Non-Human Primates

	Doses studied (mg/kg/day)	No Adverse Event Level (mg/kg/day) NOAEL	Cmax (mean, ng/mL)	Tmax (mean, h)	AUC_{0-T} (mean, ng-h/mL)
Imatinib (91 days)[1]		15	176/206 (M/F)	4/3 (M/F)	1540/1960 (M/F)
IkT-001Pro (28 days)	25, 75, 150	75	400/318 (M/F)	5.3/3.7 (M/F)	5220/3890 (M/F)

RESULTS SUGGEST POTENTIAL TO:

- ✓ Achieve dose flexibility, including use of higher dosing due to lower AEs
- ✓ Suppress GI and other adherence-related adverse events

NO ADVERSE EVENTS

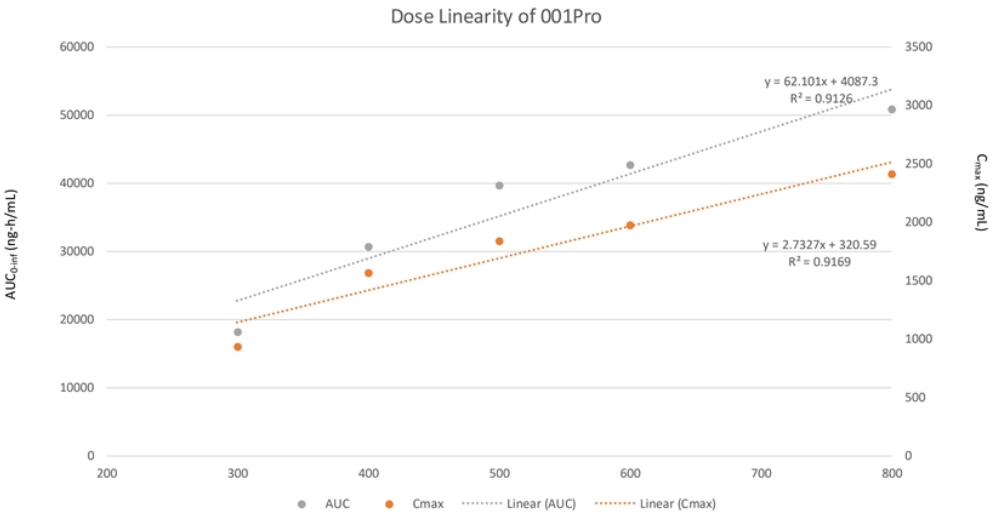
Up to 3.4x
higher dose of imatinib delivered as 001Pro

Up to 3.4x
increased exposure

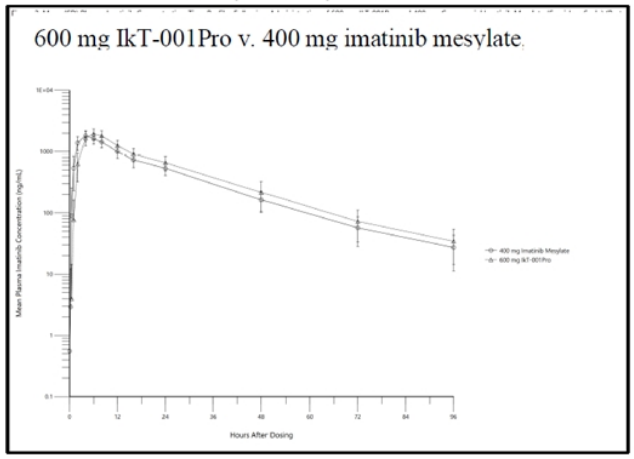
[1]Imatinib data taken from FDA summary data for approval #21-335 available at Drugs@FDA

Bioequivalence and Linear PK over the effective range

- Bioequivalence of IkT-001Pro established to cover a dosing range between 230 and 600 mg imatinib delivered
- No clinically meaningful side effects observed from single doses over this range.
- No meaningful GI disturbance observed from single doses over the range.
- PK analysis performed with two-period crossover studies over a total of 66 participants



IKT-001Pro at bioequivalent dose to 400 mg imatinib observed to have comparable exposures in humans



Trough concentrations observed in human cancer patients or in PAH

Oral Dose (mg)[1]	C_{trough} (ng/mL)[1]	Trough observed IMPRESS (ng/mL)[2]
140	279 ± 38	
300	886 ±752	
400	1216 ± 750	125 to 1200

Projected trough need to saturate target: 200 to 250 ng/mL

IKT-001Pro Dose	Projected Imatinib C_{trough}[1]	Equivalent Imatinib Dose
300 mg QD	≈600 ng/mL	240 mg QD
500 mg QD	≈1100 ng/mL	380 mg QD

[1]ACS Omega 2023, 8, 13741−13753; [2]Renard et al., Br J Clin Pharmacol 2015; 80:75–85

Regulatory and IP Status: IkT-001Pro is a New Molecular Entity for PAH

Pre-IND Meeting with FDA April 5, 2024

- Agency agreed that lower dosing, improved safety profile in NHP studies may mitigate safety concerns of IMPRES; Agency was keen to see this proceed
- **Agency agreed 505(b)(2) is an appropriate approval path for IkT-001Pro**
- **Agency agreed NME exclusivity applies to IkT-001Pro**
- **Orphan Drug designation already under review**
- Unique dosing + novel compositions of matter and use will extend patent exclusivity to 2044.
- Agency agreed special designations/accelerated approval could be sought (e.g. Breakthrough, Fast-Track, etc.).

IP protection out to 2039-2044+

Composition of Matter

- Protection to 2033/4
- 2038/9 with Patent Term Extension

Method of Use in PAH

- Protection anticipated to 2044 (without including PTE)
- No blocking IP from NVS
- Patent Pending

Other layers of Protection

- Dosing, DDI, etc.
- To be Filed

Other layers of protection available

- Drug-drug interactions

Summary of why imatinib delivered by IkT-001Pro could be impactful

- **IkT-001Pro is a Phase 3 ready asset for PAH following IND clearance**
- **Imatinib is proven to be effective in PAH**
- **High unmet need to improve the lives of patients, sotatercept isn't enough**
- **IP protection as an NME with exclusivity over the patent life, which we believe will run into 2044**
- **Imatinib has well-understood, long-term safety/tolerability and patient management**
- **If IkT-001Pro is efficacious, safe and tolerable, IkT-001Pro should be eligible for Breakthrough Designation**
- **Program has potential for simpler/faster path to approval on 505(b)(2) as an NME**

Inhibikase Therapeutics

Ph2b Trial (Registrational?)

N=150
1:1:1

- No anti-coagulants or >1 anti-platelet therapy
- Stratify sotatercept
- 50% PVR > 800 dyne-s/cm^5 at entry

Placebo
IKT001-Pro 300 mg
IKT001-Pro 500 mg

* 80% powered to show a 25% reduction in PVR

Wk 24

Primary EPs:
- PVR
- Safety

Ph3 Trial

N~160
1:1
randomization

Placebo
IKT001-Pro Selected Dose

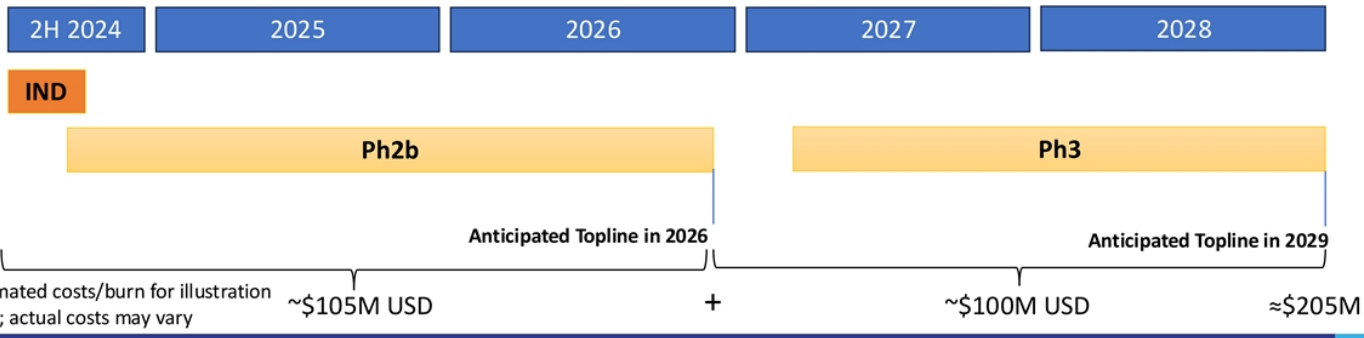
No anti-coagulants or >1 anti-platelet therapy allowed.

* 80% powered to show a 30m improvement in 6MWD

Wk 24

Primary EPs:
- 6MWD

HYPOTHETICAL EXECUTION TIMELINE



Dr. Marius Hoeper, MD

Marius M. Hoeper, MD, is deputy director of the Department of Respiratory Medicine at Hannover Medical School, Hannover Germany, where he is also in charge of the pulmonary hypertension (PH) program and senior attending physician at the intensive care unit. As clinician scientist, he has published more than 400 papers in these areas. Professor Hoeper was a task force member/chair at the 3rd, 4th, 5th and 6th World Symposia on PH. He was also an author and Section Editor of the 2009 and 2022 European PH Guidelines and the senior author of the 2015 European PH Guidelines. In 2014, Professor Hoeper received the distinguished Lifetime Achievement in Pulmonary Arterial Hypertension Award from the European Respiratory Society, and in 2016, he received the Oskar award for Medicine. In 2018 and 2019, Clarivate Analytics listed Professor Hoeper as Highly Cited Researcher.

PRINCIPAL INVESTIGATOR OF IMATINIB AND SOTATERCEPT TRIALS; HIGHLY PUBLISHED CLINICAL INVESTIGATOR IN PAH

Will BE PRINCIPAL INVESTIGATOR OF TRIAL

Dr. Marc Humbert, MD

President of the European Respiratory Society, Marc Humbert is Professor of Respiratory Medicine, Vice Dean for Research and Director of the Inserm Unit 999 at Université Paris-Saclay, France. He is the Director of the Department of Respiratory and Intensive Care Medicine, Pulmonary Hypertension Reference Centre Hôpital Bicêtre, Assistance Publique Hôpitaux de Paris, France. Marc Humbert was the Chief Editor of the European Respiratory Journal from 2013 to 2017 and he is currently Section Editor for Pulmonary Vascular Medicine. He has received distinctions. Since 2017, Marc Humbert is the vice-coordinator of the European Reference Network for rare and low prevalence respiratory diseases (ERN-LUNG). Since 2018, Clarivate Analytics has listed Marc Humbert as one of the world's highly cited researchers in the field of Clinical Medicine.

THE MOST PUBLISHED CLINICAL INVESTIGATOR WORLDWIDE IN PAH
PARTICIPATED IN IMATINIB AND SOTATERCEPT TRIALS

Inhibikase Therapeutics

Hypertension

Imatinib Mesylate as Add-on Therapy for Pulmonary Arterial Hypertension

Results of the Randomized IMPRES Study

Marius M. Hoeper, MD; Robyn J. Barst, MD; Robert C. Bourge, MD; Jeremy Feldman, MD; Adaani E. Frost, MD; Nazzareno Galié, MD; Miguel Angel Gómez-Sánchez, MD; Friedrich Grimminger, MD; Ekkehard Grünig, MD; Paul M. Hassoun, MD; Nicholas W. Morrell, MD;

Marius Hoeper, MD

Lessons from IMPRES -> 'Imatinib is an efficacious drug for patients with PAH. With contemporary trial design and Inhibikase's prodrug approach, I believe IkT001-Pro can be a differentiated therapy for patients.'

Treatment of Pulmonary Arterial Hypertension

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Published March 6, 2023 | N Engl J Med 2023;388:1478-1490 | DOI: 10.1056/NEJMoa2213558 | VOL. 388 NO. 16

Management Team with Deep Experience in Drug Development and Commercialization

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President & CEO

Previously, Dr. Werner served as Director of Research at Celtaxsys. From September 1996 until June 2007, Dr. Werner was a Head of the Laboratory of Molecular Biophysics at The Rockefeller University in New York City. Throughout his scientific career, Dr. Werner has been an innovator integrating chemistry, physics, and biology into a comprehensive approach to solving problems in medicine. Dr. Werner is the author or co-author of more than 70 research articles, reviews, and book chapters and has given lectures on his research work throughout the world.

Garth Lees-Rolfe
Chief Financial Officer

Previously served as our Vice President of Finance from November 2022 to March 2024. Prior to Inhibikase served as the Vice-President, Finance for F-Star, Inc., a publicly traded global clinical-stage biotech company. Prior to his corporate work, spent 16 years in public practice mostly with Ernst & Young, lastly as a Senior Manager. He is a licensed Certified Public Accountant in the state of Massachusetts and a licensed Chartered Accountant of Australia and New Zealand.

C. Warren Olanow, MD,
Medical Consultant
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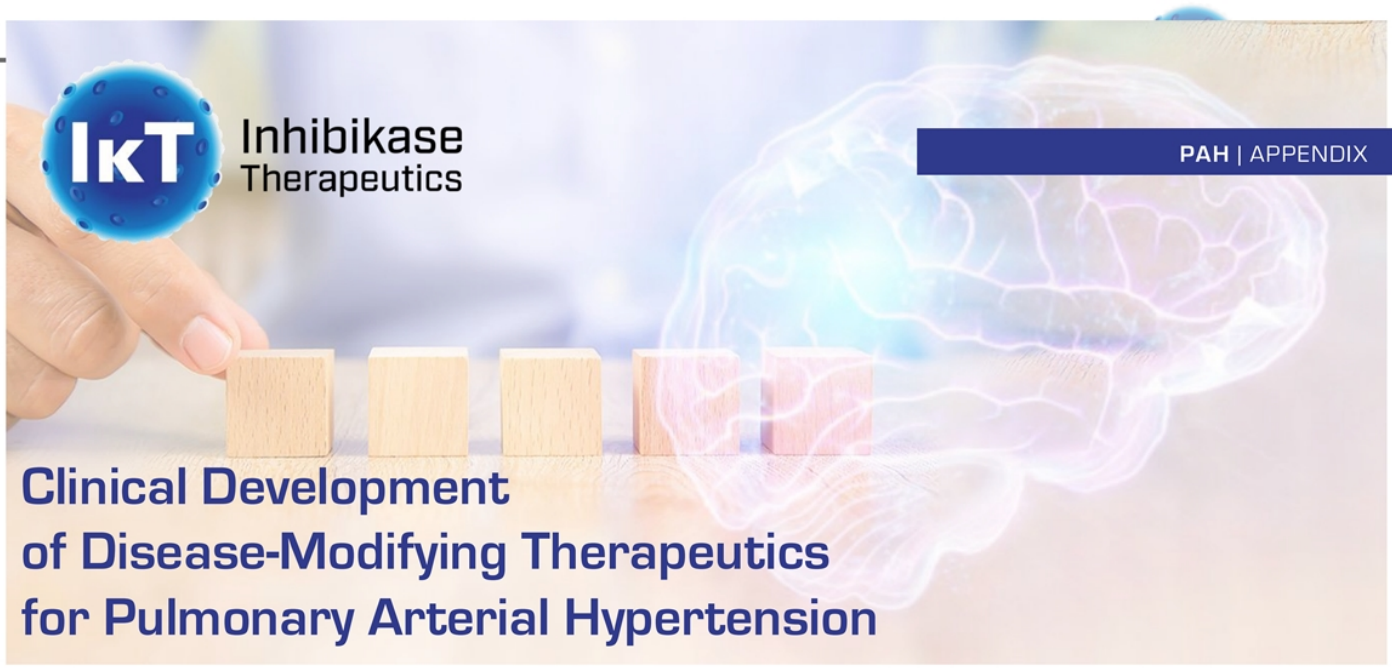
Dr. Olanow is the former Henry P. and Georgette Goldschmidt Professor and Chairman of the Department of Neurology at the Mount Sinai School of Medicine Prior to joining Mount Sinai, he served on the faculties of McGill University, Duke University, and the University of South Florida. He is the former President of the Movement Disorder Society, past President of the International Society of Motor Disturbances, and former Treasurer of the American Neurological Association. He has served on the executive committee of the Michael J. Fox Foundation Scientific Advisory Board, and he is the former Chairman of the Scientific Advisory Board of the Bachmann-Strauss Parkinson Foundation and of the Dystonia Foundation. Dr. Olanow is the former Co-Editor-in-Chief of the journal Movement Disorders. Dr. Olanow received his medical degree from the University of Toronto, performed his neurology training at the New York Neurological Institute at Columbia Presbyterian Medical Center at Columbia University, and undertook postgraduate studies in neuroanatomy at Columbia University and authored more than 600 articles in the field of neurodegeneration.


IKT
Inhibikase
Therapeutics
PAH | APPENDIX
Clinical Development
of Disease-Modifying Therapeutics
for Pulmonary Arterial Hypertension

Inhibikase Therapeutics

Adverse Events observed during bioequivalence clinical studies

Category	# Occurrences 600 mg IkT-001Pro (N=64)[1]	# Occurrences 400 mg imatinib mesylate (N=55)[1, 2]	Severity
Ocular	1 Dry eyes		Mild
Gastrointestinal	5 Gassy, bloated, abdominal discomfort or loose stool	3 Loose stool, reduced appetite, nausea	Mild
Respiratory	1 Labored breathing	1 Labored breathing	Mild
Dermatological		1 Itchy ankles	Mild
Musculoskeletal	5 Myalgias, joint pain	6 Myalgias, joint pain	Mild
Neurological			
	2 Headache	2 Headache	Mild
		1 Anxiety	Mild
		1 Sleepiness	Mild

[1]One subject accounts for 14 of the 25 adverse events observed; 6 of the total events on IkT-001Pro and 8 of the total events on 400 mg imatinib mesylate; [2]The 300 mg 001Pro cohort did not have a 400 mg imatinib mesylate comparator (N=3)

Category	# occurrences 800 mg IkT-001pro[1] (n=8) [1]	# occurrences 600 mg imatinib mesylate (n=8)[1]	Severity
Gastrointestinal	2 Sporadic loose stool	1 Nausea	Mild
Neurological		1 Loss of appetite (7 days)	Mild

[1]800 mg IkT-001Pro studied, 900 mg computed equivalent dose

INCLUSION

1. Men and women between the ages of 18 and 70 years of age (inclusive) at the time of signing the informed consent.
2. Capable of giving signed ICF which includes compliance with the requirements and restrictions listed in the ICF and in this protocol.
3. Documented diagnostic right heart catheterization (RHC) at any time prior to screening confirming the diagnosis of WHO PAH Group 1 in any of the following subtypes:
    a. Idiopathic PAH
    b. Heritable PAH
    c. Drug/toxin-induced PAH
    d. PAH associated with CTD
    e. PAH associated with simple, congenital systemic-to-pulmonary shunts at least 1 year following repair
4. Symptomatic PH classified as WHO FC II or III
5. Baseline RHC performed during the Screening Period documenting a minimum PVR of ≥ 400 dyn·sec·cm^{-5} (≥5 WU) and a pulmonary capillary wedge pressure (PCWP) or left ventricular end-diastolic pressure of ≤ 15 mmHg
6. On stable doses of background PAH therapy (i.e., patient-specific dose goal for each therapy already achieved) for at least 90 days prior to screening; for infusion prostacyclins, dose adjustment within 10% of optimal dose is allowed per medical practice.
    a. For those taking sotatercept their dosing regimen should be stable for at least 6 months prior to screening
7. 6MWD ≥ 150 and ≤ 500 m repeated twice at screening (measured at least 4 hours apart, but no longer than 1 week), and both values are within 15% of each other (calculated from the highest value).

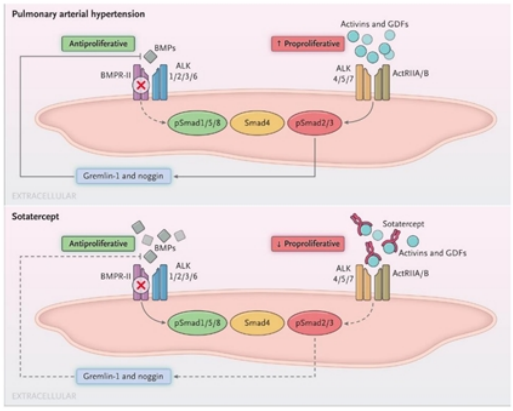
Inhibikase Therapeutics

EXCLUSION

1. Diagnosis of PAH WHO Groups 2, 3, 4, or 5
2. Diagnosis of the following PAH Group 1 subtypes: human immunodeficiency virus (HIV)-associated PAH and PAH associated with portal hypertension. Exclusions in PAH Group 1 should also include schistosomiasis-associated PAH and pulmonary veno-occlusive disease
3. Evidence of three or more (≥ 3) of the following left ventricular disease/dysfunction risk factors:
    1. Body mass index (BMI) ≥ 30 kg/m2 at the Screening Visit
    2. History of essential hypertension
    3. Diabetes mellitus – any type
    4. Historical evidence of significant coronary artery disease (CAD) established by any one of the following:
        1. History of myocardial infarction
        2. History of percutaneous coronary intervention (PCI)
        3. Angiographic evidence of CAD (> 50% stenosis in at least one vessel)
        4. Positive stress test with imaging
        5. Previous coronary artery surgery
        6. History of chronic stable angina or unstable angina
4. Uncontrolled systemic hypertension as evidenced by sitting systolic BP > 160 mmHg or sitting diastolic BP > 100 mmHg during screening visit after a period of rest
5. Baseline systolic BP < 90 mmHg at screening
6. Clinically significant orthostatic hypotension
7. History of restrictive, constrictive, or congestive cardiomyopathy
8. Electrocardiogram (ECG) with Fridericia's corrected QT interval (QTcF) ≥ 450 msec in males or ≥ 470 msec in females during the Screening Period or ≥500 msec in the presence of a right bundle branch block.
9. Personal or family history of long QT syndrome (LQTS) or sudden cardiac death
10. Pulmonary function tests (PFT) completed no more than 24 weeks before the screening period (or completed at the Screening Visit). Following criteria must are considered exclusionary
    1. TLC and/or FVC < 70%
    2. FEV1 < 65%
    3. FEV1:FVC ratio ≤ 0.60
    4. DLCO < 45% except for CTD-PAH
11. Left ventricular ejection fraction (LVEF) < 50% at screening.
12. Cerebrovascular accident within 3 months prior to the screening visit
13. Acutely decompensated heart failure within 30 days prior to the screening visit, as per investigator assessment
14. Currently receiving moderate or strong Cytochrome P450 (CYP) 3A4/5 inducers or CYP3A4/5 inhibitors (except for topical administration)
15. History of pneumonectomy
16. Currently receiving or anticipated need to receive anticoagulants
17. Currently receiving or anticipated need to receive more than one anti-platelet medication. The use of prostacyclins is not considered exclusionary. Note that sotatercept is not considered anti-platelet therapy
18. Initiation of an exercise program for cardiopulmonary rehabilitation within 90 days prior to the screening visit or planned initiation during the study (participants who are stable in the maintenance phase of a program and who will continue for the duration of the study are eligible)
19. History of atrial septostomy within 180 days prior to the screening visit
20. Current participation in another investigational clinical trial and/or receipt of any investigational medication within 90 days prior to screening
21. Previous randomization into this or another IkT-001Pro study
22. Any social or behavioral reason that would preclude completion of the study, in the judgement of the investigator
23. Alanine aminotransferase (ALT) or aspartate aminotransferase (AST) levels greater than 3 times the upper limit of normal (ULN)
24. Bilirubin levels greater than 3 times the upper limit of normal (ULN)
25. Estimated glomerular filtration rate (eGFR) < 30 mL/min/m^2 (as defined by the Modification of Diet in Renal Disease [MDRD] equation)
26. Currently lactating, pregnant or planning on becoming pregnant during the study

Inhibikase Therapeutics

Sotatercept (SC Q3W) was approved in 2024 as the first disease modifying therapy



Sotatercept developer Acceleron was acquired for $11.5B by Merck in 2021 following positive Phase 2 data[2]

Sotatercept demonstrated 6-minute walk and PVR benefit[1]

Table 2. Change from Baseline at Week 24 in Primary and Secondary Efficacy End Points (Intention-to-Treat Population).*

End Point	Sotatercept (N=163)	Placebo (N=160)
Primary end point		
6-Minute walk distance — m		
Median change estimate (95% CI) from baseline at wk 24†	34.4 (33.0 to 35.5)	1.0 (−0.3 to 3.5)
Hodges–Lehmann location shift from placebo estimate (95% CI)‡	40.8 (27.5 to 54.1)§¶	
Secondary end points		
Pulmonary vascular resistance — dyn·sec·cm^{-5}		
Median change estimate (95% CI) from baseline at wk 24†	−165.1 (−176.0 to −152.0)	32.8 (26.5 to 40.0)
Hodges–Lehmann location shift from placebo estimate (95% CI)‡	−234.6 (−288.4 to −180.8)§¶	

However, only a minority of PAH patients respond to Sotatercept in a clinically meaningful way[1]

Table 2. Change from Baseline at Week 24 in Primary and Secondary Efficacy End Points (Intention-to-Treat Population).*

End Point	Sotatercept (N=163)	Placebo (N=160)
Multicomponent improvement		
Patients who met all three criteria for 6-min walk distance, NT-proBNP level, and WHO functional class — no./total no.	63/162‖	16/159‖
Percentage of patients (95% CI)	38.9 (31.3 to 46.9)¶**	10.1 (5.9 to 15.8)
WHO functional class		
Patients with improvement at wk 24 from baseline — no./total no.	48/163¶**	22/159‖
Percentage of patients (95% CI)	29.4 (22.6 to 37.1)	13.8 (8.9 to 20.2)

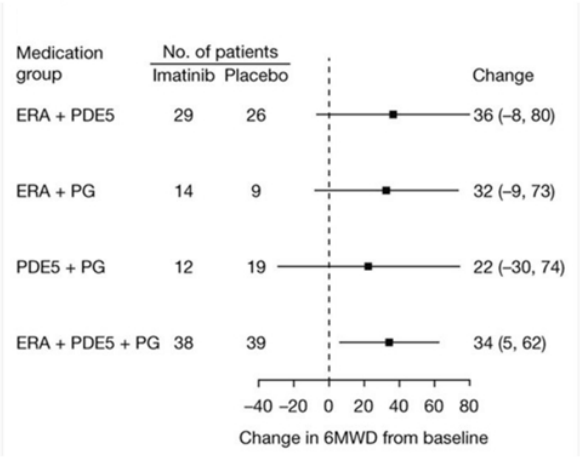
[1]Hoeper et al. New Engl. J. Med. (2023); 16:1478ff.; [2]https://www.merck.com/news/merck-to-acquire-acceleron-pharma-inc/

- IMPRES Ph3 Trial titrated patients from 200 mg QD for first 2 weeks to 400 mg QD if well tolerated. If not well tolerated, patients remained on 200 mg QD.[1]

- 24/67 patients on imatinib were not able to stay on the 400 mg dose for ≥50% of the treatment period and kept dose at 200 mg for majority of treatment. **These patients showed significant PVR and 6MWD improvements. These benefits were seen independent of background therapy**[1]

Endpoint	Treatment	n	mean (SE)
6MWD, m	Imatinib (long-term dose escalation successful)	40	50 (9)
	Imatinib (long-term dose escalation not successful)	24	22 (10)
	Placebo	79	5 (7)
PVR, dynes·sec·cm^{-5}	Imatinib (long-term dose escalation successful)	44	–437 (53)
	Imatinib (long-term dose escalation not successful)	23	–371 (77)
	Placebo	78	–10 (52)

*Successful long-term dose escalation refers to sustained dose escalation to 400 mg for ≥50% of the treatment period.



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

Inhibikase Therapeutics

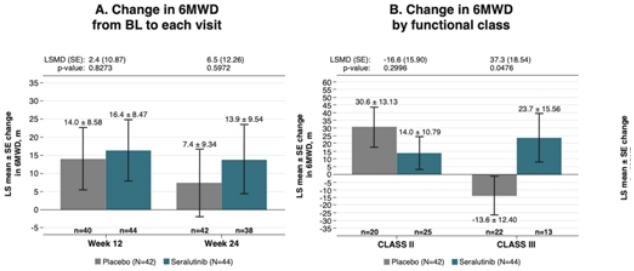
Competitive landscape for TKIs

- Inhaled Seralutinib[1]: Phase 2 trial outcome demonstrates inhaled seralutinib < 1/3 as effective as sotatercept/imatinib in haemodynamics, modest 6MWD improvement across all classes.

Table 2. Change from baseline to Week 24 in pulmonary hemodynamic parameters. A significant reduction in mPAP (p=0.0094) was the main driver of the observed reduction in PVR. Treatment with seralutinib was associated with a significant improvement in PAC (p=0.0410)

Parameter	Placebo (N=42)	Seralutinib (N=38)	
	LS mean change ± SE	LS mean change ± SE	LS mean difference (95% CI)
mRAP, mmHg	0.85 ± 0.532	-0.14 ± 0.576	-0.99 (-2.350, 0.367)
PASP, mmHg	1.74 ± 2.321	-5.24 ± 2.469	-6.98 (-12.77, -1.19)*
PADP, mmHg	1.95 ± 1.127	-1.47 ± 1.197	-3.43 (-6.21, -0.64)*
mPAP, mmHg	2.12 ± 1.415	-2.58 ± 1.508	-4.70 (-8.203, -1.188)*
Cardiac output, L/min	-0.15 ± 0.165	0.06 ± 0.173	0.20 (-0.204, 0.605)
Cardiac index, L/min/m²	-0.02 ± 0.092	0.11 ± 0.097	0.13 (-0.100, 0.359)
PCWP or LVEDP, mmHg	1.04 ± 0.574	0.54 ± 0.608	-0.50 (-1.963, 0.963)
PVR, dyne*s/cm⁵	21.2 ± 29.91	-74.9 ± 33.02	-96.1 (-183.5, -8.8)*
PA compliance, mL/mmHg*	-0.02 ± 0.085	0.19 ± 0.089	0.22 (0.009, 0.423)*
Stroke volume, mL	-4.57 ± 2.206	-0.78 ± 2.313	3.79 (-1.606, 9.190)
Stroke volume index, mL/m²	-1.81 ± 1.263	0.38 ± 1.313	2.19 (-0.917, 5.299)

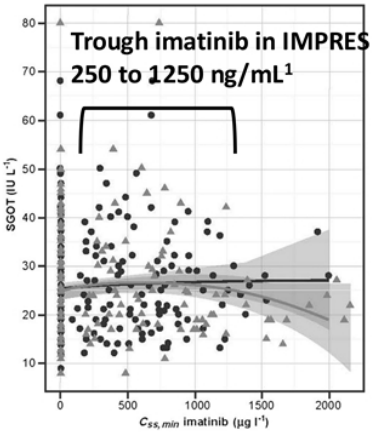
Figure 6. A. At Week 24, the least-square mean difference in 6MWD between se was 6.5 m (p=NS). **B.** Significant improvement in 6MWD in FC III patients (+37.3 trend favoring seralutinib in patients with a REVEAL 2.0 risk score of ≥ 6 (+21.9m

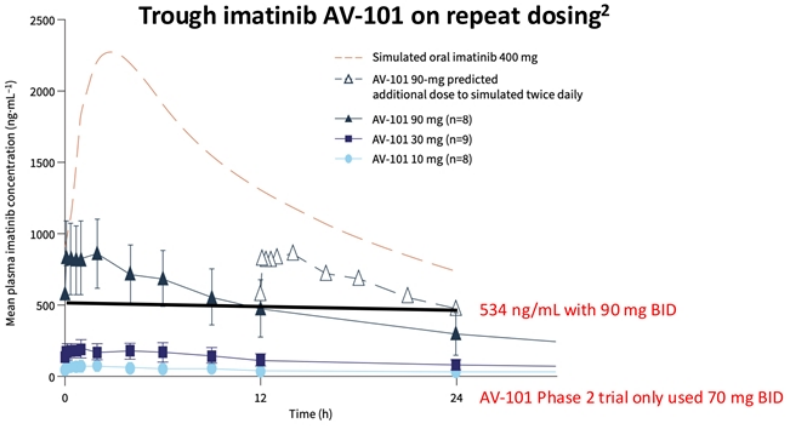


[1]Poster, American Thoracic Society Annual Meeting, May 19-24, 2023

Competitive landscape for TKIs

- Inhaled imatinib[1]: Phase 2b studies of AV-101 demonstrate no efficacy whatsoever. Why?





[1]Renard et al., Br J Clin Pharmacol 2015; 80:75–85; [2]Gillies et al. ERJ Open Res 2023; 9: 00433-2022