Inhibikase Therapeutics

2Q24 | BUSINESS PRESENTATION

Clinical Development of Disease-Modifying Therapeutics for Neurodegenerative Disease & Cancer

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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. Important factors that could cause actual results to differ materially from those in the forward-looking statements are set forth in the Company’s filings with the Securities and Exchange Commission, including its annual report on Form 10-K and its quarterly Form 10-Q, 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.

Developing innovative medicines across the therapeutic spectrum

- Multi-therapeutic pipeline across neurodegenerative disease, cancer and cardiopulmonary disease
- **Risvodetinib (IkT-148009):** Lead Selective Abelson Tyrosine Kinase (c-Abl) inhibitor with potential to be a disease-modifying treatment for Parkinson's disease (PD) and related disorders. Phase 2, 201 trial ongoing with 94% of sites open, 61% enrolled in the U.S. The 201 trial planned to be expanded for 12 additional months providing 15 months of data to evaluate clinical benefit. Preliminary outcomes support continuation and expansion of trial.
- **IkT-001Pro:** Prodrug of imatinib mesylate with improved safety/tolerability profile for treatment of 11 blood and stomach cancers. Bioequivalent 501 trial completed and FDA pre-NDA meeting completed January 2024. Planned submission for approval under 505(b)(2). Cardiopulmonary applications to be explored with FDA April, 2024.
- Robust patent portfolio with protection to 2033 (oncology) and 2036 (neurodegeneration).
- Orphan designations: **Rivsodetinib** in Multiple System Atrophy, **IkT-001Pro** in multiple oncology indications and pulmonary arterial hypertension.
- Cash/cash equivalent runway into 1Q25.
- Highly-experienced management team, consultants, Board of Directors and Scientific Advisory Board

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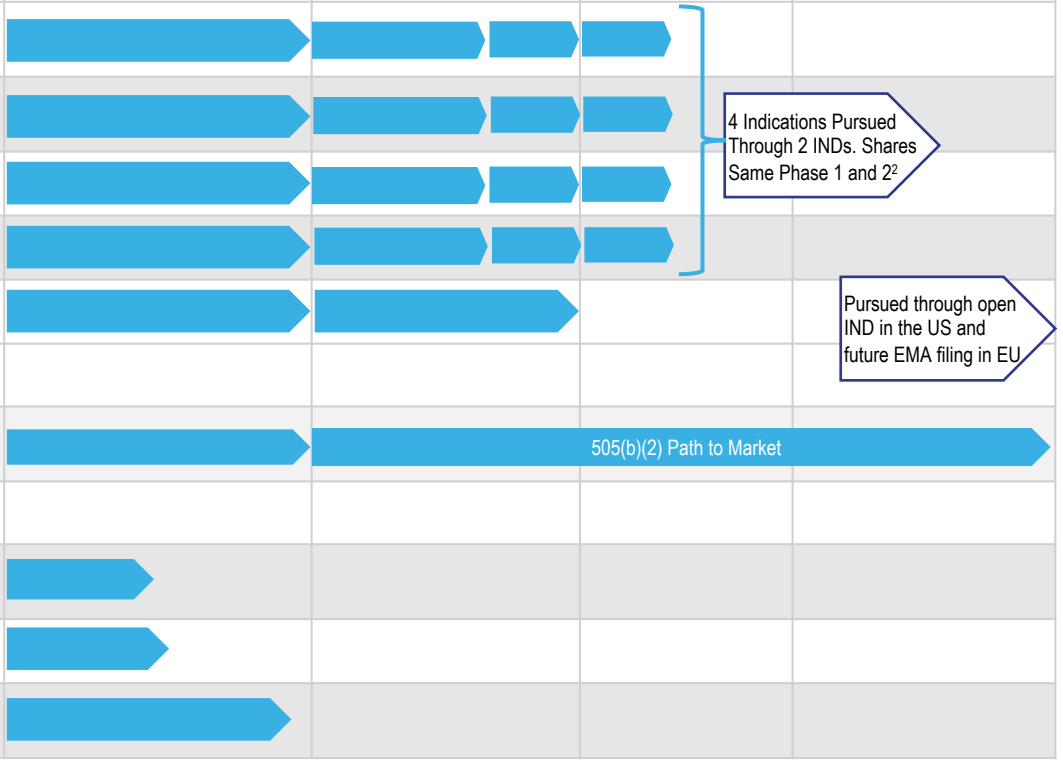
Multi-Indication Pipeline in Neurodegeneration, Oncology and Non-oncology Indications

DRUG TARGET	DRUG CANDIDATE	MODALITY	DISEASE INDICATION	CLINICAL DEVELOPMENT[1]
				PRECLINICAL DEVELOPMENT / PHASE 1/1B / PHASE 2 / PHASE 3
Neurodegeneration				
c-Abl	**Risvodetinib**	Small molecule	Parkinson's Disease: Treatment Naive	
c-Abl	**Risvodetinib**	Small molecule	Parkinson's Disease: Early Stage	
c-Abl	**Risvodetinib**	Small molecule	Neurogenic Constipation	
c-Abl	**Risvodetinib**	Small molecule	Dysphagia	
c-Abl	**Risvodetinib**	Small molecule	Multiple System Atrophy	
Oncology				
BCR-Abl	**IkT-001Pro**	Small molecule	Stable-phase CML (orphan indication)	
Research Phase				
c-Abl	**IkT-148x, BIP 4-7**	Small molecule	Dementia with Lewy Body	
c-Abl	**IkT-148x, BIP4-7**	Small molecule	Multiple System Atrophy	
c-Abl	**IkT-001Pro**	Small molecule	Pulmonary Arterial Hypertension	



(1). 'Clinical Development' progress bars represent the current state of the indicated programs. Blue arrows represent completed or in progress studies; white arrows represent planned approaches for future clinical studies.

(2). Four indications will be pursued for IkT-148009 in PD, which will be pursued through studies of treatment naïve and early-stage patients, including their GI complications. MSA is an orphan, aggressive form of Parkinson's-like disease to enter clinical development at Phase 2 following completion and positive outcomes from animal model studies of IkT-148009 in prophylactic and therapeutic dosing studies.

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Parkinson's and Related Disorders

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Parkinson's disease and MSA in the U.S.[1]

Parkinson's: Slowly Progressing

1/3 of a Patient's Lifespan to death = 25 years

90,000
New Cases / Year

38,000
Deaths / Year

930,000 – 1,200,000
U.S. Patients[1]

60
Average Age Of Onset

MSA: Rapidly Progressing

1/10 of a Patient's Lifespan to death = 8 years

15,000 -50,000
Cases

Orphan Disease

55
Average Age Of Onset

Men twice as likely as **women** to contract disease

Co-morbid indications

47%
Arthritis

36%
Heart/Circulatory

35%
Psychosis

30%
Dementia

Global treatment sales for PD by 2030 are expected to exceed

$12.2 BILLION

Vision Research, 2022

Current treatments cannot alter course of Parkinson's disease

MSA has no beneficial treatments

The country with the highest diagnosed prevalence is

THE U.S.

Vision Research, 2022

[1]Parkinson's Disease Foundation Decisions Resources 2016, Lewin Report in the Economic Burden and Future Impact of Parkinson's disease, 2019.

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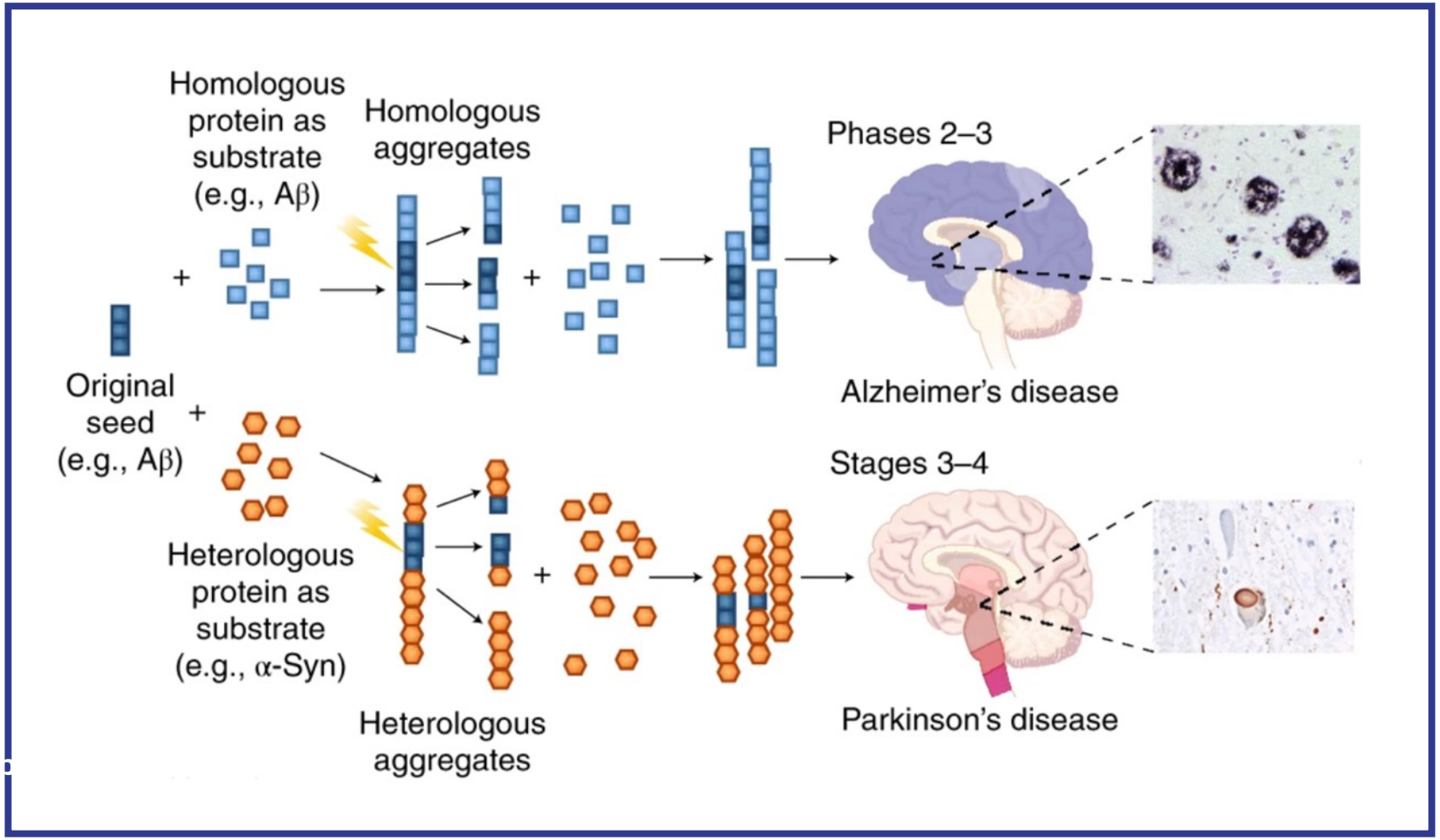
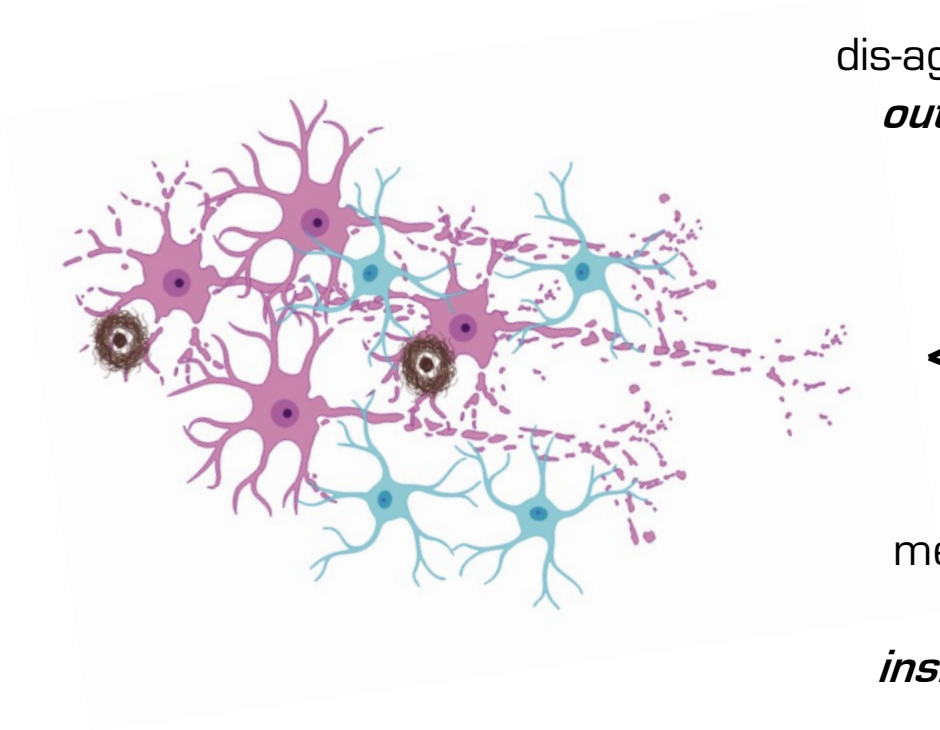
Different proteins, similar pathological effect

β-amyloid and Tau in Alzheimer's

α-synuclein in PD, MSA, DLB



[1]Nat. Neurosci. 21: 1332-1340 (2018)



antibody or
dis-aggregator targets plaque
outside affected neurons

→ Clearance of pathological plaque unknown
NOT restorative

mechanistic inhibitors
target plaque
inside affected neurons

→ Does clear pathological plaques
CAN BE restorative

- Risvodetinib, an inhibitor of c-Abl

How does Risvodetinib work?

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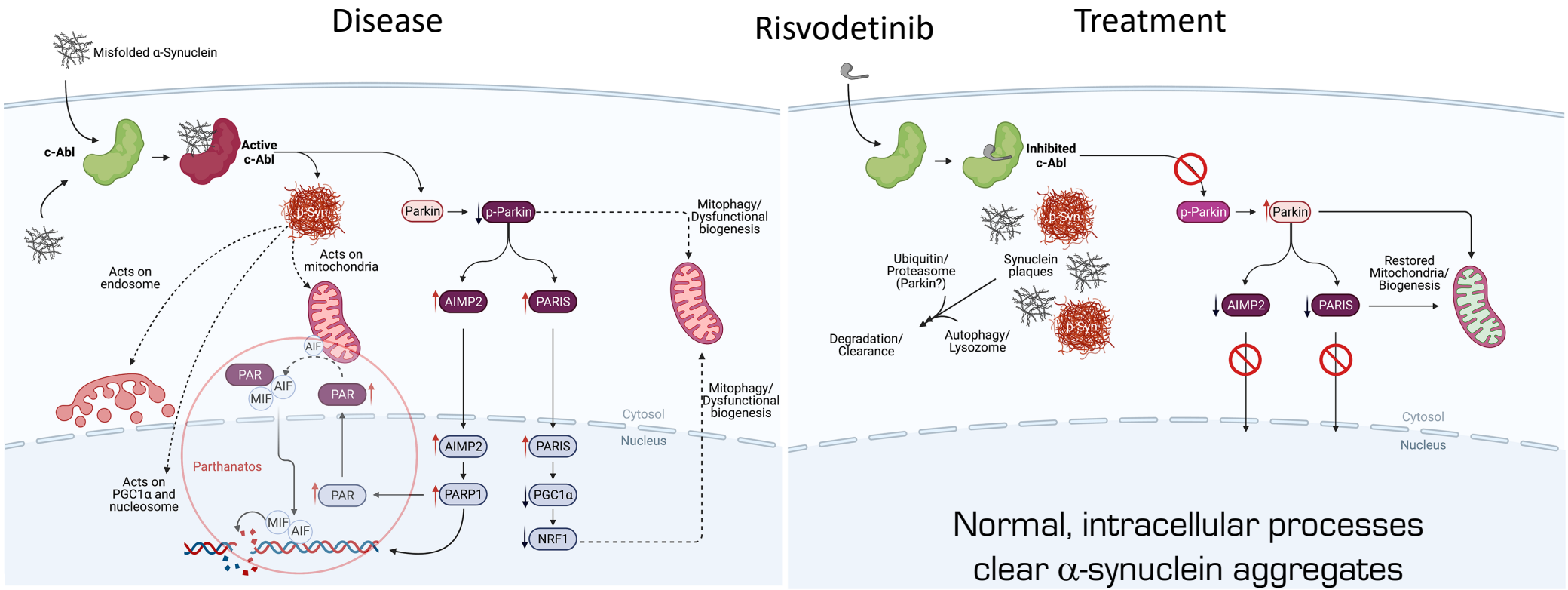
Biochemistry of disease initiation and its treatment response[1]



[1]Werner and Olanow **37**, 6-15 (2022)

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c-Abl inhibition by Risvodetinib restores lost function in Validated Animal Models of Parkinson's disease[1]

Neurodegeneration

Risvodetinib @human doses

preserves as much as 85% of brain neurons

Motor Dysfunction

Risvodetinib @human doses

restores as much as 90% of lost function

α-Synuclein Toxicity

Risvodetinib @human doses

clears to baseline in the organs of disease

Neuroinflammation

Risvodetinib @human doses

suppresses to near baseline in the organs of disease

[1]Werner and Olanow , Mov Disorders 2021, doi: 10.1002/mds.28858
Karuppagounder, Werner, et al., Sci Transl. Med 2023 doi: 10.1126/scitranslmed.abp9352

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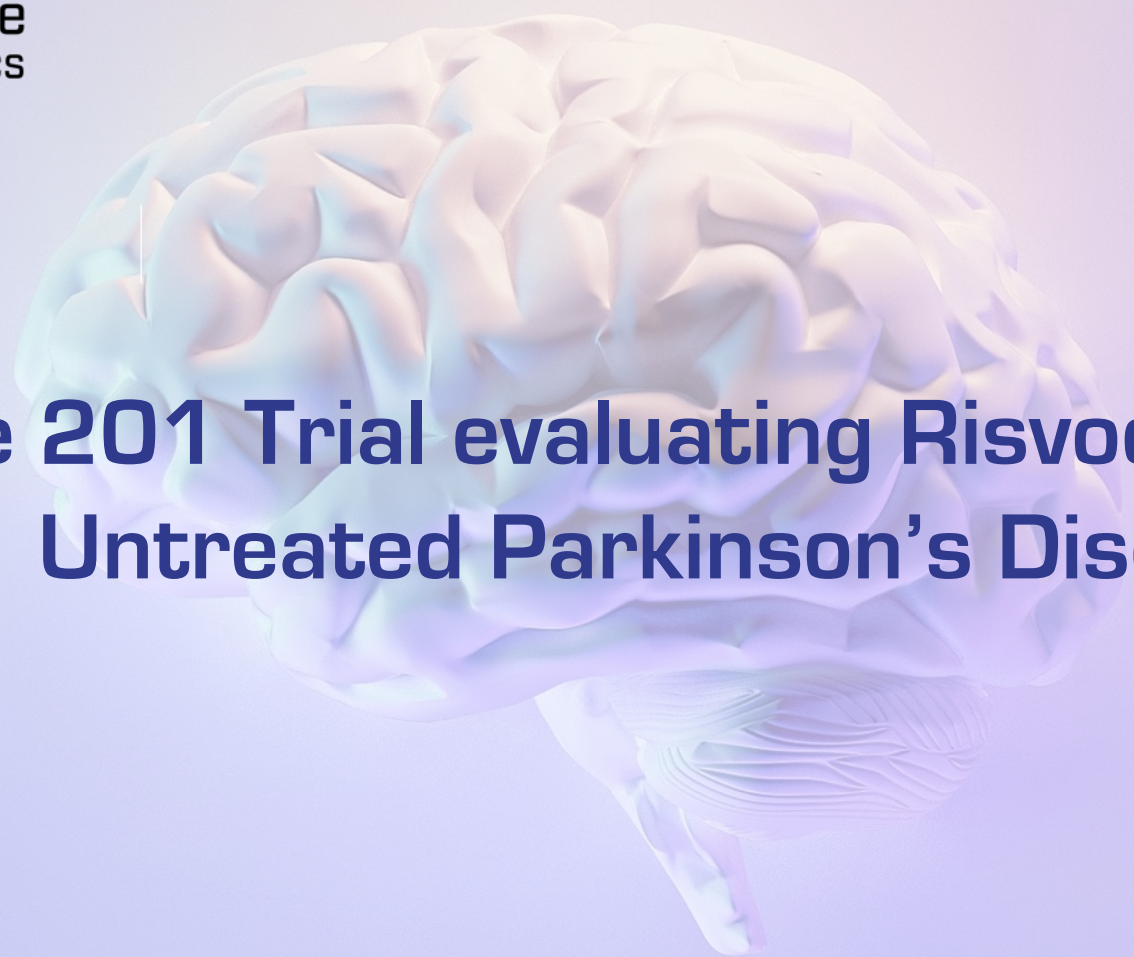

The 201 Trial evaluating Risvodetinib in Untreated Parkinson's Disease

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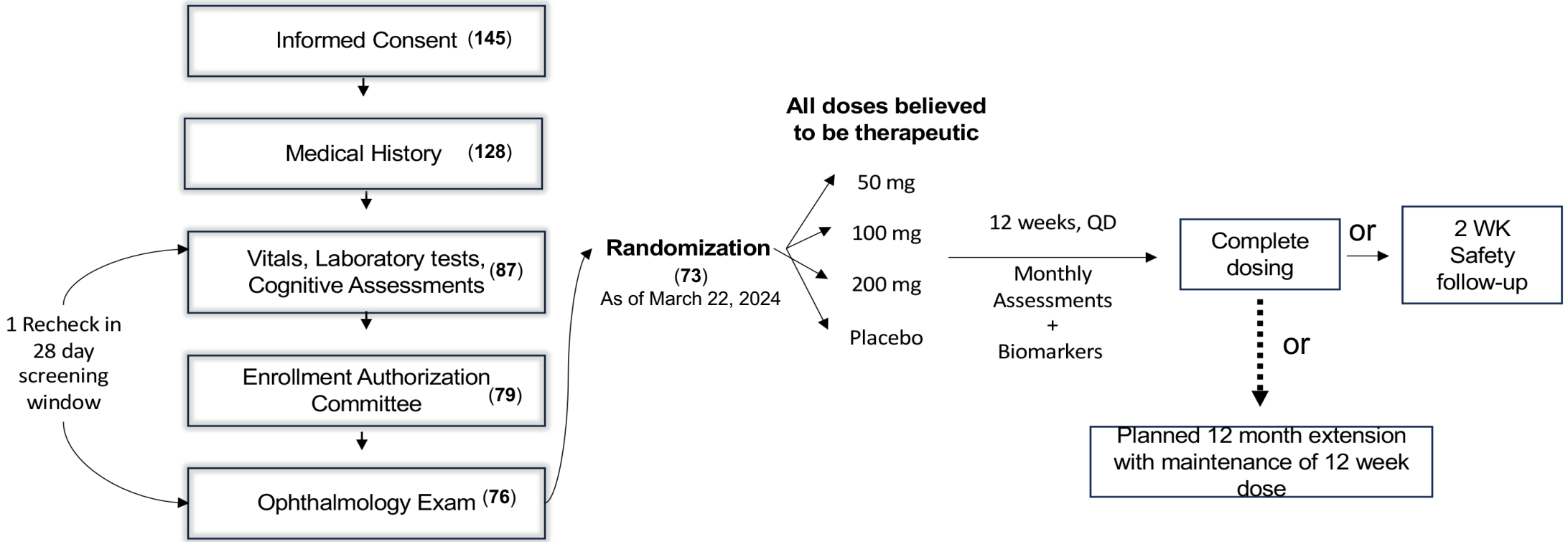
Untreated Patients

Defined: Must meet UK Brain Bank Criteria and MDS Research Criteria
Must have bradykinesia with sequence effect and motor asymmetry
No more than 28 days prior treatment with levadopa/carbidopa or MAO-B inhibitors
Hoehn & Yahr < 3.0



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	Measure	Untreated Patients (% of Total) N=73
Gender	Female	31 (42.5)
	Male	42 (57.5)
Age	Median Male/Female	64/64
	Range Male/Female	43-79/50-74
Ethnicity	Not Hispanic or Latino	68 (93.2)
	Hispanic or Latino	5 (6.8)
Time since PD diagnosis	Median (months)	7.3
	Range (months)	1 – 49.1
MDS-UPDRS at baseline	Part III	23.5±9.3
	Part II	6.8 ± 4.4
	Part I	5.2 ± 3.9

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17 total AEs that may be treatment related

	Measure (# occurrences)	Severity
Gastrointestinal		
	Vomiting (3)	Mild (2)/Moderate (1)
	Gas/Cramps (2)	Mild (2)
	Heartburn (1)	Mild
	Diarrhea (3)	Mild
Cardiovascular		
	Abnormal ECG (1)	Mild
	Orthostatic hypotension (1)	Mild
Laboratory		
	Elevated Lipase (1)	Mild
	Elevated Creatinine (1)	Mild
Psychological/ Neurological		
	Irritability (1)	Mild
	Migraine (1)	Mild
	Worsening Parkinson's (1) (non-compliant dosing)	Moderate
	Increased energy (1)	Mild

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Assessment[1]	Mean Change from Baseline @ 4 weeks				Mean Change from Baseline @ End of Study			
	50 mg N=3	100 mg N=2	200 mg N=2	Placebo N=3	50 mg N=3	100 mg N=2	200 mg N=3	Placebo N=3
PGI-S	-0.33	0	-1	-0.33	0	0	0	-0.33
CGI-S	0.33	-0.5	-2	-0.33	0.33	-2	-0.67	-0.33
PDQ-39	-1	0	-3.5	-8.3	-0.67	0	-1.67	ND
UPDRS II	-0.33	1	-0.33	-0.66	-0.33	0	-4.33	0
UPDRS III	0.33	-1	-4	-0.33	2	-1.33	-4.33 -10.4	1.7
II + III	0	0	-4.3	-0.99	1.67	-1.33	-8.67	1.7
NMSS	-1.33	-2	-0.5	0.66	-3.67	-1	1 -6.6	0
S&E ADL	0	0	-5	6.7	0	-5	-3.33	3.33
ESS	ND	ND	ND	ND	-0.33	-0.5	1.67	-1

[1]Lower numbers (or negative changes) may suggest improvement

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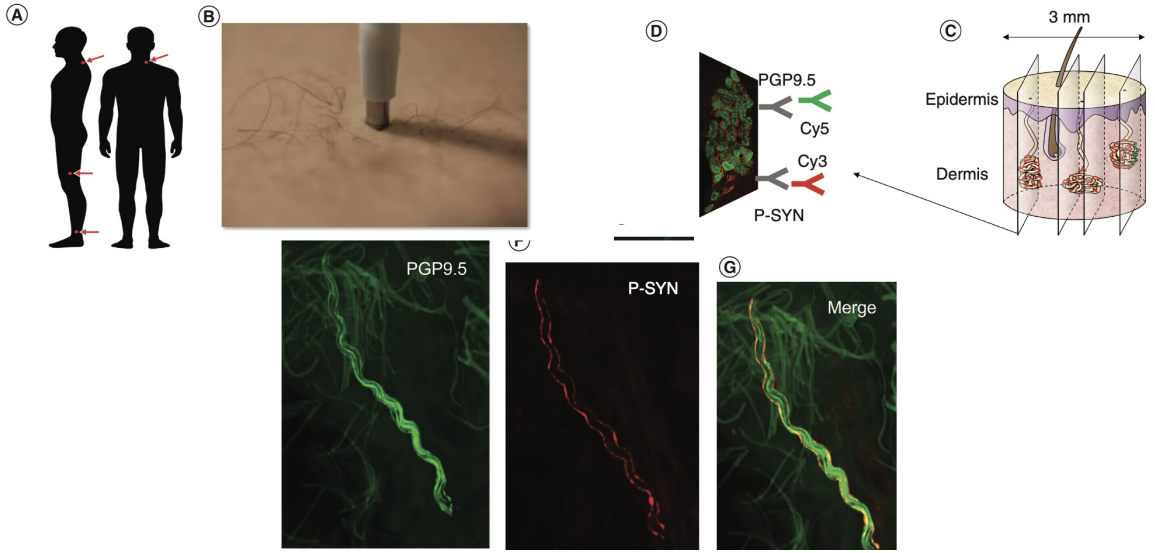
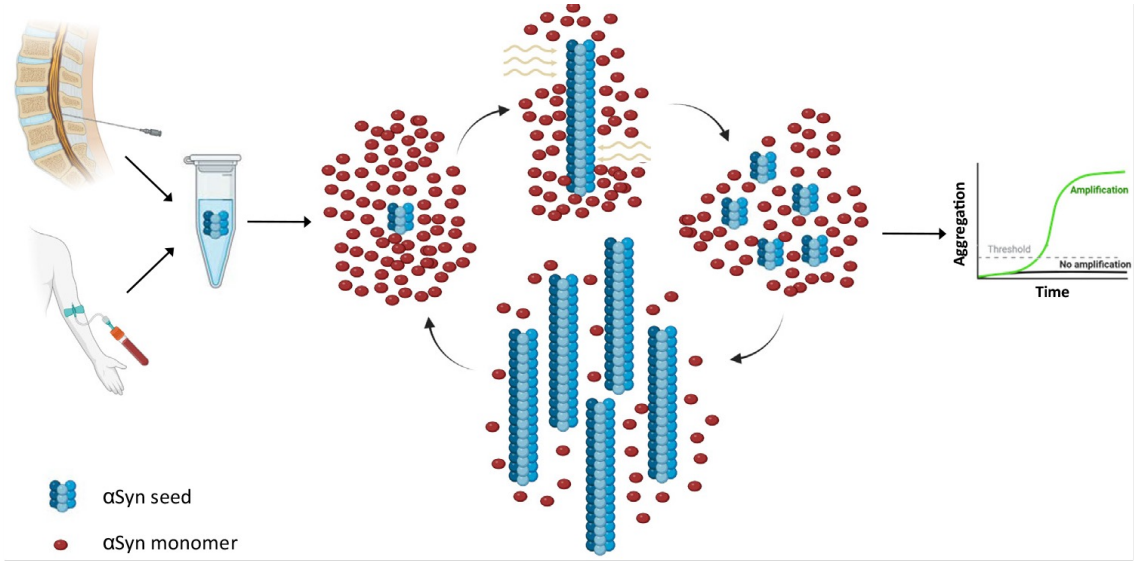
Tissue-derived biomarkers



Tissue biopsy is collected at three locations on the body using a 3mm 'punch'. The tissue samples are shipped to a standardized testing lab and processed with two antibodies. One highlights axons for intraepidermal nerves and the second highlights pathological alpha-synuclein. Fluorescent dyes colorize the synuclein deposits in the tissue for quantification. Adapted from *Biomark.Med.*(2022)16(7),499–509.

Blood and CSF biomarkers



Seed amplification uses the small quantities of alpha-synuclein aggregates in the CSF as a 'seed' for an amplification process. The amplification process is achieved by feeding alpha-synuclein monomers into a cyclic 'shaking' routine to amplify the seeds into longer molecules that can be detected by fluorescence. Adapted from https://doi.org/10.1016/j.tibtech.2024.01.007

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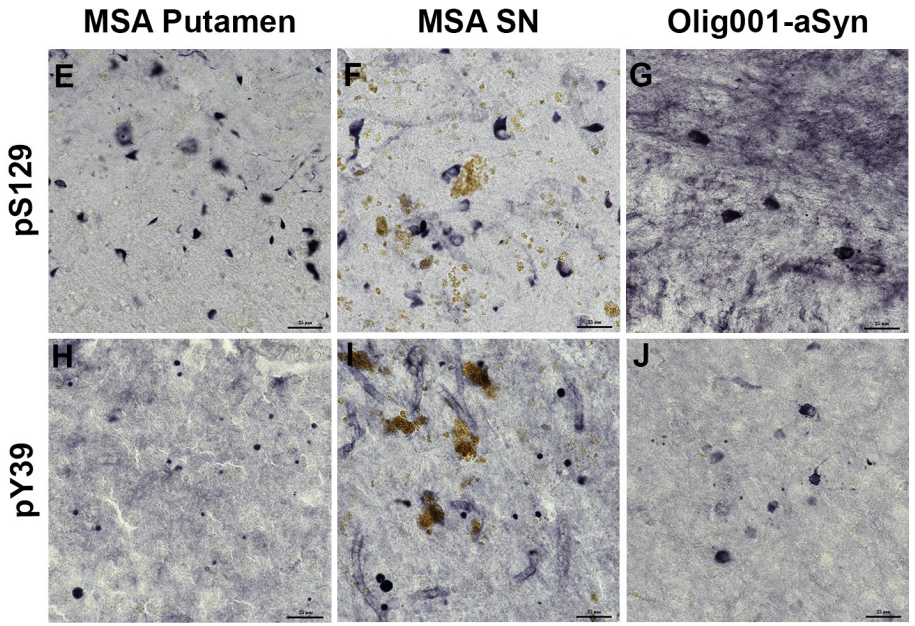
c-Abl in other neurodegenerative disease

MSA[1]



AD

Prog Neurobiol. 2021;202:102031
Autophagy. 2021;17(5):1278-1280.
J Biol Chem. 2020 ;295(23):7905-7922
Front Cell Neurosci. 2019;13:526
Biochim Biophys Acta Mol Basis Dis. 2018; 1864(4 Pt A):1148-1159
Biomol Struct Dyn. 2017;35(4):883-896
J Alzheimers Dis. 2016 ;54(3):1193-1205
PLoS One. 2014 ;9(3):e92309
Curr Alzheimer Res. 2011;8(6):643-51.
Neurobiol Aging. 2011;32(7):1249-61.
J Alzheimers Dis. 2011;25(1):119-33.
J Alzheimers Dis. 2010;19(2):721-33.
J Alzheimers Dis. 2009;18(1):1-9
J Alzheimers Dis. 2009;17(2):409-22
Brain. 2008;131(Pt 9):2425-42
Neurobiol Dis. 2004;17(2):326-36
Proc Natl Acad Sci U S A. 2003;100(21):12444-9.

ALS

J Neurol Sci. 2018;393:80-82
Sci Transl Med. 2017;9(391):eaaf3962
Front Cell Neurosci. 2015 9;9:203
PLoS One. 2012;7(9):e46185

[1]Marmion, Werner, Kordower et al, (2021) Neurobiol Dis 148:105184

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IkT-001Pro for Treatment of Stable-Phase Chronic Myelogenous Leukemia

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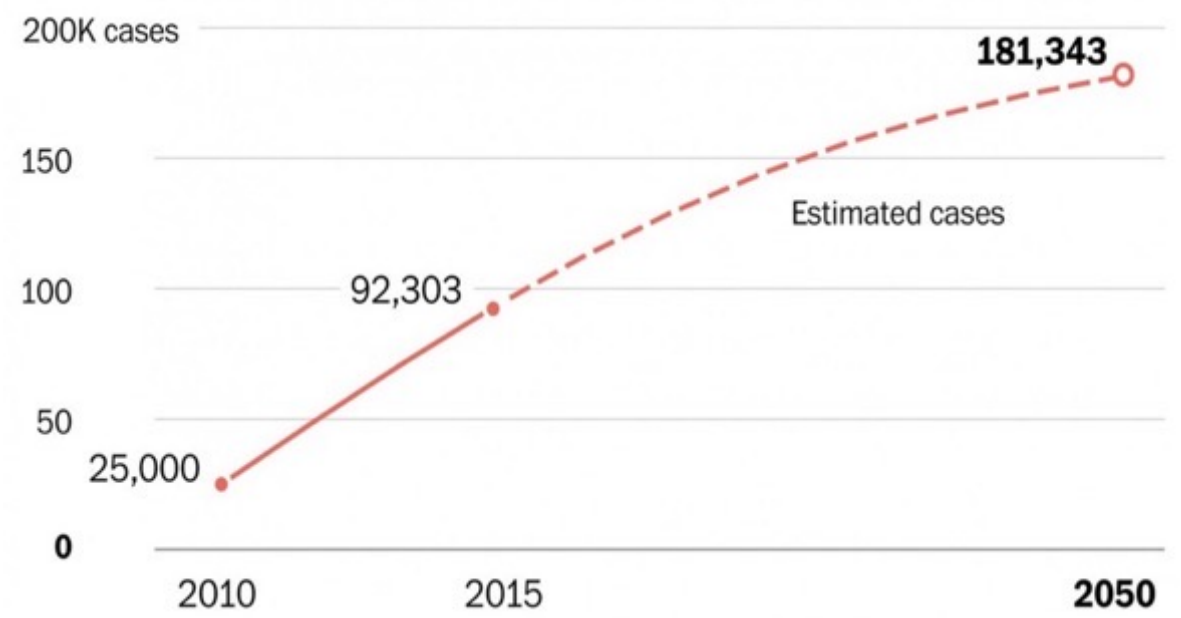
CML in the U.S.[1] Accessible Market Opportunity Despite Presence of Generic Gleevec®

Prevalence of Chronic Myeloid Leukemia in the U.S.



- Patients commonly switch due to intolerance or lack of response[3]
- Intolerance to Gleevec® occurs in 30% of patients, leading to lack of treatment compliance and relapse[4]
- Second generation treatments have severe adverse events (i.e. Sprycel® or Tasigna®)
- Best approach: reduce Gleevec® side effects

[1]Jabbour E, Kantarjian H. Am. J. Hematol. 89:548–556
[2]IMS-Iqvia retail sales data 2016-2020
[3]Am J. Hematology (2019) 94:46-54
[4]Annals of Hematology (2018) 97:1357–1367

- **$330.5 million in net U.S. Sales for branded and generic Gleevec®[2]**
- **> 57% market share Generic Gleevec®**
- **50% of recipients experience Grade 2 GI adverse events**

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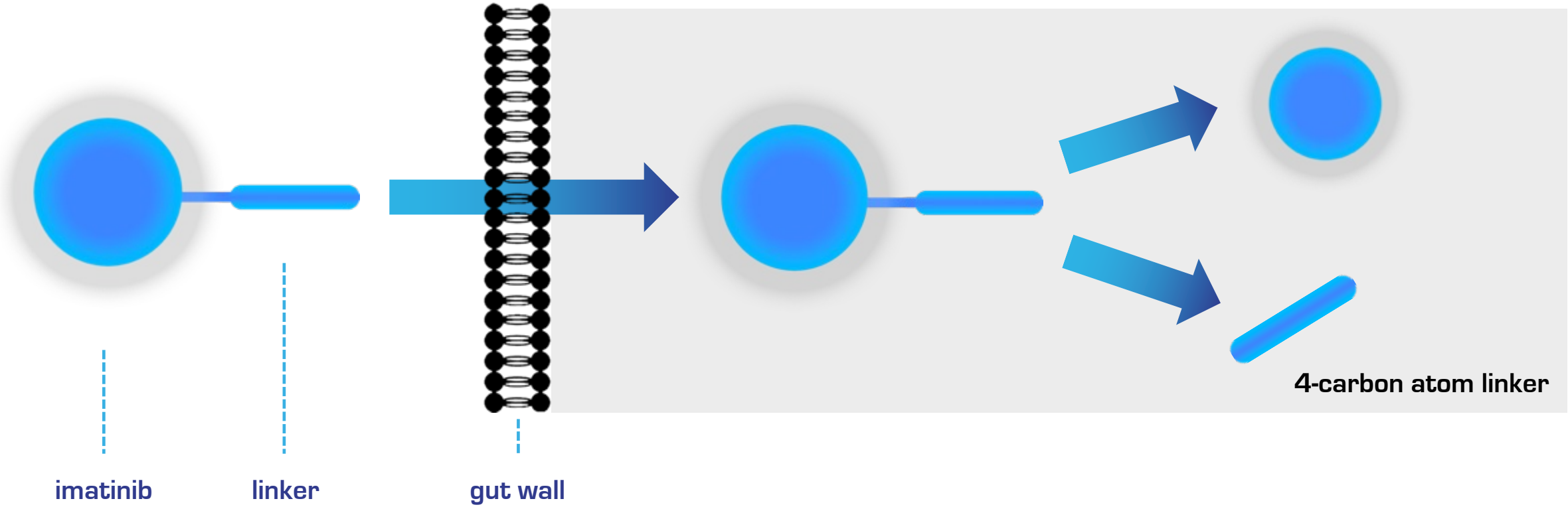
IkT-001Pro releases the active ingredient imatinib only in blood



pH 2-6
(stomach, ileum, duodenum)

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

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IkT-001Pro:
Lower GI Toxicity Alternative to Generic Gleevec®

Measurement of IkT-001Pro in Non-Human Primates

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

[1]FDA summary data for approval 21-335

RESULTS SUGGEST THAT:

- ✓ 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

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Clinical Development of IkT-001Pro: Summary

- 600 mg IkT-001Pro bioequivalent to 400 mg imatinib mesylate
  - Only 25 mild adverse events observed across 58 participants, 12 each for prodrug and standard-of-care
  - IkT-001Pro showed less inter-patient variability and longer rise time to maximum plasma concentration relative to 400 mg imatinib mesylate
  - Mean C_{max} and $AUC_{0\text{-}inf}$ 16% higher than standard-of-care
- 800 mg IkT-001Pro may be bioequivalent to 600 mg imatinib mesylate ('high dose imatinib)
- Pre-NDA meeting January, 2024 preliminary outcomes: evaluate IkT-001Pro up to 1200 mg to cover all approved doses of imatinib mesylate; evaluate P-GP and BCRP in pre-clinical assay to confirm if IkT-001Pro and imatinib mesylate absorb in the gut similarly; create distinct dosages of IkT-001Pro to discriminate from imatinib mesylate; advance typical requirements for manufacturing and CMC.

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IkT-001Pro for Treatment of Pulmonary Arterial Hypertension

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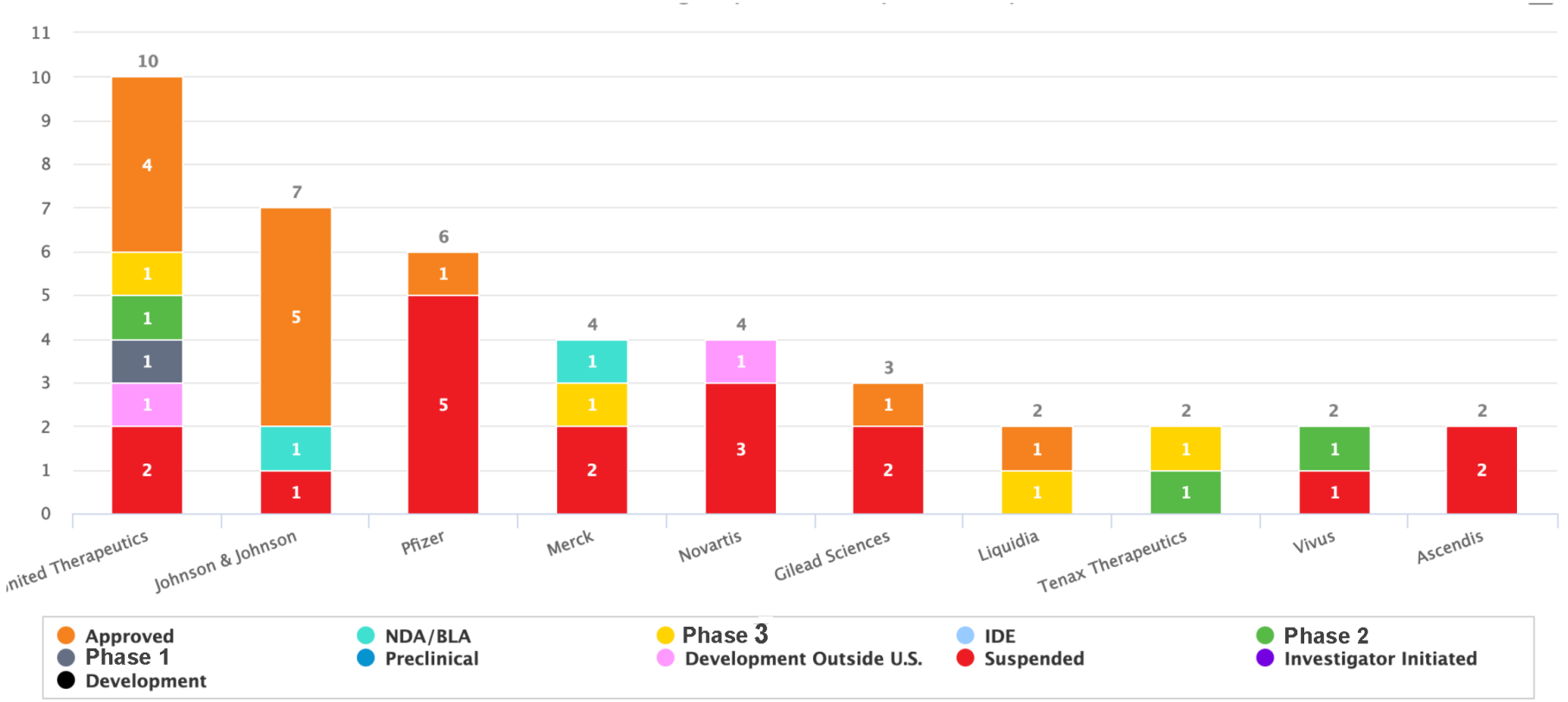
Total US Sales of Approved Drugs for PAH: $4.6B



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

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Advantages of Clinical Development of IkT-001Pro in PAH

- Large market, high value opportunity
  - IkT-001Pro ALREADY proven to be disease-modifying as imatinib was shown to be disease-modifying (*Circulation* 2013;127:1128-1138)
- Accelerated approval pathways may be open to this product in PAH as a branded drug
- Orphan designation already under review.
- Pre-IND meeting leading into registrational Phase 3 trial takes place April, 2024. Meeting outcomes will determine whether this indication will be pursued.

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Selected Financial Data

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Selected Financial and Stock Data

Capitalization Table	April 1, 2024
Common Shares Outstanding	6,476,844
Options (WAEP: $10.35)	992,566
Warrants (WAEP: $7.64)	2,266,136
Fully Diluted Shares Outstanding	**9,735,546**

Balance Sheet	December 31, 2023
Current Assets:	
Cash, Cash Equivalents, Marketable Securities	$13,252,052
Prepaid research and development	$219,817
Prepaid expenses and other current assets	$739,179
Total Current Assets	**$14,506,647**
Total Current Liabilities	$3,438,601
Total Working Capital	**$10,772,447**

Management Team with Deep Experience in Drug Development and Commercialization

Milton Werner, PhD
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. 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
and Chief Executive Officer of Clintrex Research Corporation.

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.

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Mr. Dennis Berman

- Co-founder, board member, and/or seed investor in many private biotechnology and technology companies, five of which have gone public.
- Currently serves as the President of Molino Ventures, LLC a board advisory and venture capital firm and was co-founder and Executive Vice President of Corporate Development of Tocagen.
- Seed investor, co-founder, and/or board member of Intervu, Kintera, Inc., Gensia, Calabrian

Dr. Milton H. Werner, PhD

- President & CEO, Inhibikase Therapeutics, Inc.

Ms. Gisele Dion

- Senior Vice President, Chief Accounting Officer and Corporate Controller at Takeda Pharmaceutical Ltd
- Senior Advisor to the Chief Financial Officer of Takeda Pharmaceutical Ltd.
- Vice President, Chief Accounting Officer and Corporate Controller at Shire Pharmaceuticals LLC,
- Corporate Controller and Senior Director of Technical Accounting at Biogen Inc.,
- Currently Director and Audit Committee Chair, Cytek Biosciences, Inc.
- Staff Member of the Financial Accounting Standards Board (FASB)
- Audit Advisor Group Member for the Pharmaceutical Research and Manufacturers of America (PhRMA).
- B.S. in Accounting and Management Information Systems from Fairfield University

Dr. Roy Freeman, MD

- Professor of Neurology at the Harvard Medical School and Director of the Center for Autonomic and Peripheral Nerve Disorders in the Department of Neurology at Beth Israel Deaconess Medical Center
- Former chairman of the World Federation of Neurology research group on the autonomic nervous system, former President of the American Autonomic Society, and former chairman of the Autonomic Section of the American Academy of Neurology.
- Editor-in-Chief of Autonomic Neuroscience: Basic and Clinical and on the editorial boards of The Clinical Journal of Pain, Pain: Clinical Updates, and Clinical Autonomic Research.
- Serial founder of several companies in pain and neurodegenerative disease and is on the scientific advisory boards of many large and small pharmaceutical and biotechnology companies.

Dr. Paul Grint, MD

- 20+ years experience in biologics and small-molecule research and development, including the successful approval and commercialization of products in the infectious diseases, immunology, and oncology therapeutic areas.
- Director of Amplyx Pharmaceuticals and Synedgen.
- Served in senior management roles at Cerexa, Forest Laboratories, Kalypsys, Pfizer, IDEC Pharmaceuticals, and Schering-Plough Corporation.
- Fellow of the Royal College of Pathologists and a medical degree from St. Bartholomew's Hospital College, University of London.

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Dr. Robert Hauser, MD
Professor of Neurology, University of South Florida College of Medicine - Director USF Parkinson's Disease and Movement Disorders Center

Dr. Jeffrey Kordower, PhD
Founding Director
ASU-Banner Neurodegenerative Disease Research Center (NDRC)
The Charlene and J. Orin Edson Distinguished Director at the Biodesign Institute
Professor of Life Sciences
Arizona State University

Dr. Ken Marek
President and Senior Scientist, Institute of Neurodegenerative Disorders

Dr. Ted Dawson, MD, PhD
Neurodegeneration and Stem Cell Programs, Institute for Cell Engineering, Departments of Neurology, Physiology, Pharmacology, and Molecular Sciences - The Johns Hopkins University School of Medicine

Dr. Valina Dawson, PhD
Neurodegeneration and Stem Cell Programs, Institute for Cell Engineering, Departments of Neurology and Physiology
The Johns Hopkins University School of Medicine

Dr. Warren Olanow, MD, FRCPC
Henry P. and Georgette Goldschmidt Professor and Chairman Emeritus, Mount Sinai School of Medicine
CEO, Clintrex Research Corporation

Dr. Karl Kieburtz, MD, MPH
Robert J. Joynt Professor in Neurology, Senior Associate Dean for Clinical Research, Director of the Clinical &Translational Science Institute, Founder Center for Human Experimental Therapeutics (CHET)- University of Rochester Medical Center
President Clintrex Research Corporation

Dr. Jay Pasricha, MBBS, MD
Director, Johns Hopkins Center for Neurogastroenterology
Professor of Medicine