



This presentation shall not constitute an offer to sell or a solicitation of an offer to buy any securities, nor shall there be any sale of such securities in any state or jurisdiction in which such offer, solicitation, or sale would be unlawful prior to registration or qualification under the securities laws of any such state or jurisdiction.

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.



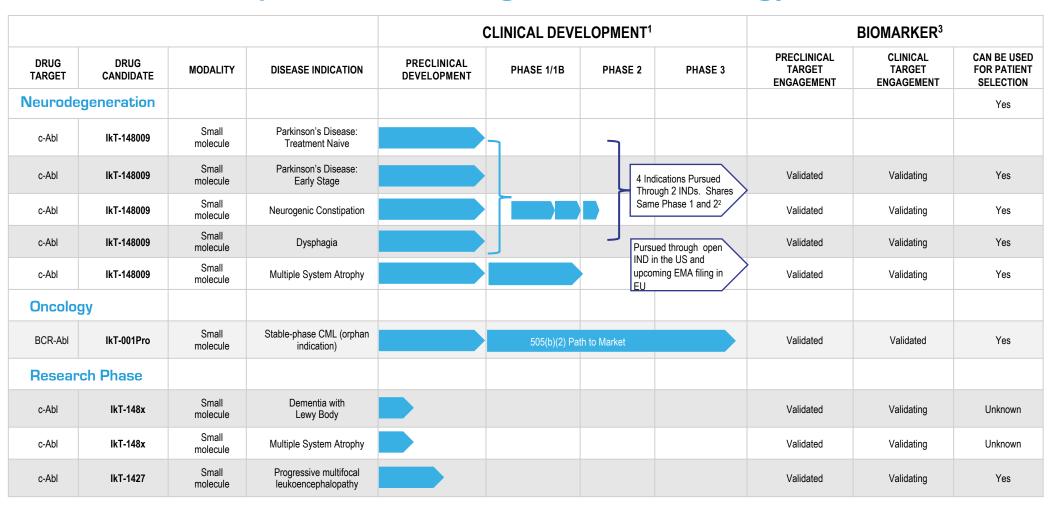
# Developing innovative medicines across the therapeutic spectrum

- Focused on developing novel therapeutics across a wide therapeutic spectrum including neurodegeneration, oncology and infectious diseases.
- Aim to discover novel therapeutics by modeling human disease using the Company's Re-engineering Approach with Metabolism Preserved (RAMP) medicinal chemistry platform
- IkT-148009: Lead Abelson Tyrosine Kinase (c-Abl) inhibitor program has the potential to be a disease-modifying treatment for Parkinson's disease (PD) and related disorders. Phase 2a '201' trial in Parkinson's began screening patients in 1Q23.
- IkT-001Pro: First oncology product is a BCR-Abl inhibitor with a potentially-improved safety profile to standard of care imatinib mesylate for leukemias and gastrointestinal cancers. Bioequivalence trial compared to 400 mg imatinib mesylate completed 4 cohorts 1Q22 in first part of three-part study. Anticipated completion of bioequivalence in 1H23.
- Robust patent portfolio with protection to 2033 (oncology) and 2036 (neurodegeneration).
- \$20.8 million in grants and contracts from NIH, DoD, the Michael J. Fox Foundation and the Georgia Research Alliance, all peer-reviewed; \$63 million gross proceeds in investor capital in 2021, \$10 million investor capital in 2023

Highly-experienced management team, consultants, Board of Directors and Scientific Advisory Board



## Multi-Indication Pipeline in Neurodegeneration, Oncology and Infectious Disease



- (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 IRT-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.
- (3). For biomarker status, 'Validated' refers to proof of target engagement in the target tissue which has been performed using rodent tissues and fluids. We are currently developing methods for using clinical samples for validating our ability to confirm target engagement in patients. 'Validating' in this context indicates ongoing efforts to prove target engagement using proprietary sources and methods under development from human tissues and fluids. Target engagement measures if and to what extent a compound occupies its target. 'Can be used for patient selection' refers to our ability to use one or more markers we are currently 'Validating' to screen patients for the presence of that marker as a means of defining the patients most likely to benefit from the proposed





# Parkinson's Disease in the U.S. Large Market, Opportunity For Disease Modification

#### Chronic Disease for a Long Time

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

60,000

38,000

New Cases / Year

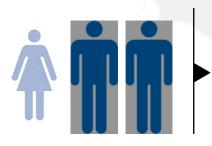
Deaths / Year

930,000 - 1,200,000 U.S. Patients<sup>1</sup>

60

Average Age Of Onset

### Other illnesses complicate development



Men twice
as likely as
women to
contract
disease



47%

**Arthritis** 



36%

Heart/Circulatory



35%

**Psychosis** 



30%

Dementia

By 2025, Parkinson's disease drug sales are expected to

### **DOUBLE**

Pharma Insights, 2019

Sales estimates by 2025 are expected to exceed

\$6.0 BILLION

Pharma Insights, 2019

The country with the highest diagnosed prevalence is

THE U.S.

Delvelnsight, 2019

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



# Multiple System Atrophy in the U.S.<sup>1</sup> Orphan Market, Opportunity For Disease Modification

Rapid disease leading to death

6 - 10 years

1,980

UNK

New Cases / Year

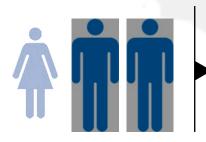
Deaths / Year

Oprhan disease with ≈17,000 US cases

55

Average Age Of Onset

#### Other illnesses complicate development



Men twice as likely as women to contract disease



47%

**Arthritis** 



36%

Heart/Circulatory



35%

**Psychosis** 



30%

Dementia

No Treatments Benefit MSA

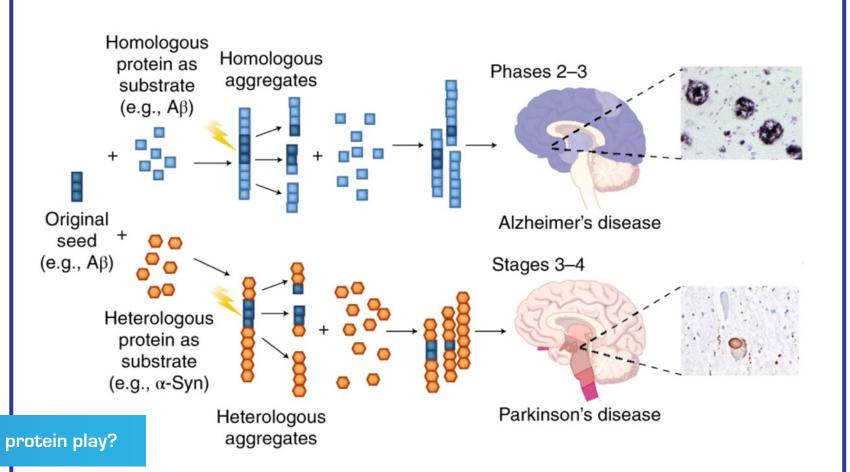
Wide Open Addressable Market

C—Abl Inhibiter Treatment could offer first benefits

<sup>1</sup>Lancet Neurol . 2009 Dec;8(12):1172-8.



Causation in Parkinson's and Alzheimer's is closely related<sup>1</sup>



What role does the misfolded protein play?

<sup>1</sup>Nat. Neurosci. 21: 1332-1340 (2018)



# Internalized aggregates are the pathological species in PD and MSA<sup>1</sup>



Stressors Trigger the Production of α-Synuclein

α-Synuclein Forms **Fibrous Aggregates or Plaques** 

Aggregates or plaques of

misfolded α-synuclein are

abnormal, but they have

not been chemically

modified.

c-Abl is a Sentinel which patrols for abnormalities inside an affected neuron

c-Abl Kinase

The Effects and **Results of Activated** 

**NEURODEGENERATION** 

Oxidative / nitrosative stress

Point mutation in one or more proteins causing hyperaggregation

> Gene duplication / triplication

> > **Toxin**

Inflammation

Stochastic Mechanisms c-Abl acts a sensor for abnormality, such as internalized α-synuclein plaques or aggregates, stimulating a cascade of responses leading to neurodegeneration

Sensing α-synuclein plaques or aggregates, activated c-Abl chemically modifies asynuclein at Tyr39, converting it into a more highly aggregated form.

C-Abl also chemically modifies Parkin, disrupting survival pathways centered on mitochondrial integrity and protein clearance

Cell Death

Movement Disorder

Cognitive Disorder

**Neurons in PD** Glial Cells in MSA

c-Abl inhibition acts here

<sup>1</sup>Werner and Olanow, Mov Disorders 2021, doi: 10.1002/mds.28858

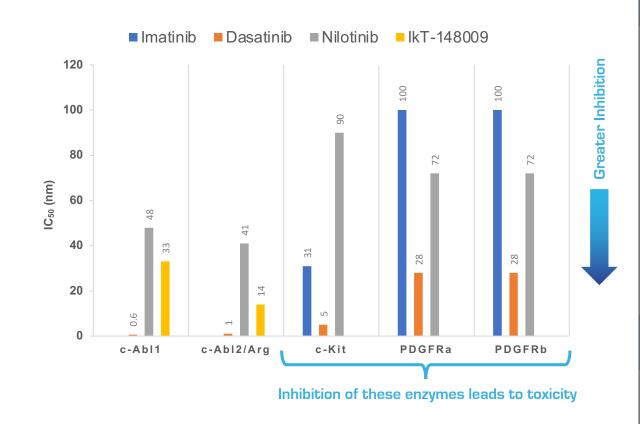
Nasdaq : IKT 9 Inhibikase.com •



# IkT-148009 is Low Toxicity, Selective, Brain Penetrant c-Abl Inhibitor in Clinical Development



- Selective Inhibitor of c-Abl1 and Abl2/Arg
- Design suppressed toxicity of cancer drugs in this class
- Low or no apparent organ toxicity at current level of knowledge
- High brain penetrance



#### TOXICOLOGY IN RAT/MONKEY1

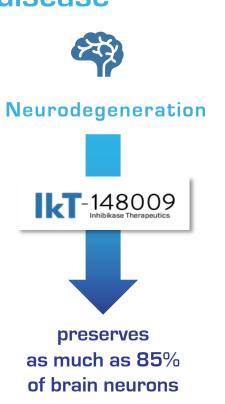
Human equivalent dose of 1460 mg					
Cardiovascular	None				
Renal	None				
Liver	None				
Bone marrow	None				
Sternum	None				
Blood	None				
PBMCs	Slight increase in neutrophils within normal limits				
Cytotoxicity	None in primary or mature cells				
Sustained brain concentration	> 1 micromolar				

113, 26 week and 39 week toxicology data shows lkT-148009 has a more favorable toxicity profile as dosing is extended



# c-Abl inhibition by lkT-148009 restores lost function in Validated Animal Models of Parkinson's disease<sup>1</sup>









<sup>1</sup>Karuppagounder, et al., (2023), DOI: 10.1126/scitranslmed.abp9352



# Clinical Development of IkT-148009



# Demographics and Adverse Events Across 119 Healthy Subjects and Patients

Category	Demographic	Healthy Subjects Value (% of Total N=94)	Parkinson Patient Value (% of Total, N=25)
Gender	Female	36 (37.9)	9 (36)
	Male	58 (61.1)	15 (60)
Age	Average (SD)	57.9	61.9
	Median	58.0	63
	Range	40, 70	48, 71
Ethnicity	Hispanic or Latino	14.9 (14.8)	4 (16)
	Not Hispanic or Latino	80 (85.1)	20 (80)
Race	Black or African American	55 (58.5)	3 (12)
	White	37 (39.4)	20 (80)
	Other	2 (2.1)	0 (0)
Adverse events		13, none clinically significant, only 9 possibly drug related	12, none clinically significant, only 4 possibly drug related

IkT-148009 does not lead to typical c-Abl inhibitor adverse events: No common Gl No Cardiovascular No Hematological



# Complete Listing of Possibly Drug-related Adverse Events:

No Clinically Significant Adverse
Events in Healthy Subjects or
Parkinson's Patients
Regardless of Dose or Dose
Duration Have Been Seen To
Date

Category	Dose mg	Dose Duration	# Occurrences Healthy Subjects (N=88)	# Occurrences PD patients (N=25)	Severity
Cardiovascular	75 mg	Single Dose	1 Palpitations <sup>1</sup>		Mild
Gastrointestinal					
	325 mg	Single Dose	2 Diarrhea		Mild
	100 mg	7-day, 1x/day		1 Constipation <sup>2</sup>	Mild
	100 mg	4 wk, 1x/day		1 Elevated Amylase/Lipase <sup>3</sup>	Moderate
	200 mg	7-day, 1x/day	1 Elevated Lipase <sup>5</sup>		Mild
	Active, 50 mg	4 wk		1 Gastric pain <sup>4</sup>	Mild
	Active, 50 mg	4 wk		1 Nausea <sup>4</sup>	Mild
Dermatological					
	50 mg	7-day, 1x/day		1 Dermatitis	Mild
Musculoskeletal					
	200 mg	7-day, 1x/day	5 Myalgias, joint pain, fatigue,edema <sup>5</sup>	lou Halton monitoning 2	

<sup>&</sup>lt;sup>1</sup>Appeared 2 weeks post-dose, no clinical basis found even after following by 3-day Holter monitoring; <sup>2</sup>Appeared one day after last dosing day; <sup>3</sup>Amylase and Lipase abnormalities were asymptomatic. Patient reported regular consumption of alcohol prior to the baseline visit and while enrolled in the trial; <sup>4</sup>Single occurrence on first dose; <sup>5</sup>Six AEs in a single subject, mild severity. Lipase elevation occurred on one day of a 7-day dosing period.



# Systemic exposure in efficacy studies corresponds to low oral dose in humans with therapeutic dose between 50 mg and higher

#### Steady-state Clinical Pharmacokinetics of IkT-148009 and Comparison to Steady-state PK in Mouse Efficacy Animals

Drug	Dose	T <sub>1/2</sub> (h)	T <sub>max</sub> (h)	C <sub>max</sub> (ng∕mL)	AUC <sub>0-inf</sub> (h*ng/mL)
Mouse efficacy <sup>1</sup> 148009 N=15	50 mg/kg/day QD Steady-state	3.1	4	2562	19650
Healthy MAD 148009 <sup>2</sup> N=6	25 mg QD Steady-state	28.6±4.7	4.7±1.3	1770±807	25400±9260
Patient MAD 148009³ N=6	50 mg QD Steady-state	24.9±3.9	3.7±1.5	2560±564	32500±8500

<sup>&</sup>lt;sup>1</sup>Steady-state PK values in mouse plasma where therapeutic efficacy was observed in models of both inherited and sporadic PD; brain concentration exceeded 1 μM after 7-days and displayed no neurological effects over 7 months of daily dosing. <sup>2</sup>Steady-state PK values in healthy subjects given daily dose of lkT-148009 for 7-days. <sup>3</sup>Steady-state PK values in Parkinson's patients given daily dose of lkT-148009 for 7-days.



## CLINICAL PHASE 2: the '201' Trial 3 doses

	Double-blinded Enrollment and Measurement 3 months										Anal	ysis						
Open-label 12 month safety extension at 3 doses																		
March23 🕨	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

Double-blinded: 3 Months Dosing Across 3 Doses 12 month extension study

Primary: Safety/Tolerability Prim
Secondary: MDS-UPDRS II+III Secondary

PGI-S

CGI-S

MDS-UPDRS II

MDS-UPDRS III MDS-UPDRS I

**Non-motor Symptom Scale** 

**CSBM** 

**Epworth Sleepiness Scale** 

**GI** Measures

Primary: Safety/Tolerability

Secondary: MDS-UPDRS II+III

(measure PGI-S every 3 CGI-S

months) MDS-UPDRS II

MDS-UPDRS III MDS-UPDRS I

Non-motor Symptom Scale

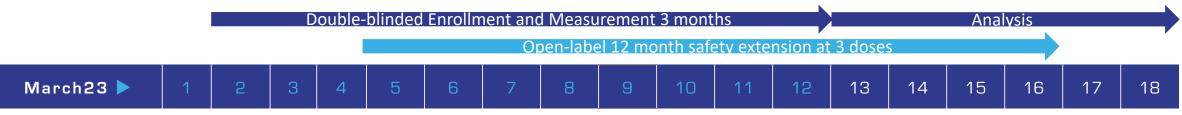
**CSBM** 

**Epworth Sleepiness Scale** 

**GI** Measures



### CLINICAL PHASE 2: the '201' Trial 3 doses



Double-blinded: 3 Months Dosing Across 3 Doses

Exploratory:

Skin  $\alpha$ -synuclein aggregates

Seed amplification assays

12 month extension study

Exploratory: Skin  $\alpha$ -synuclein aggregates

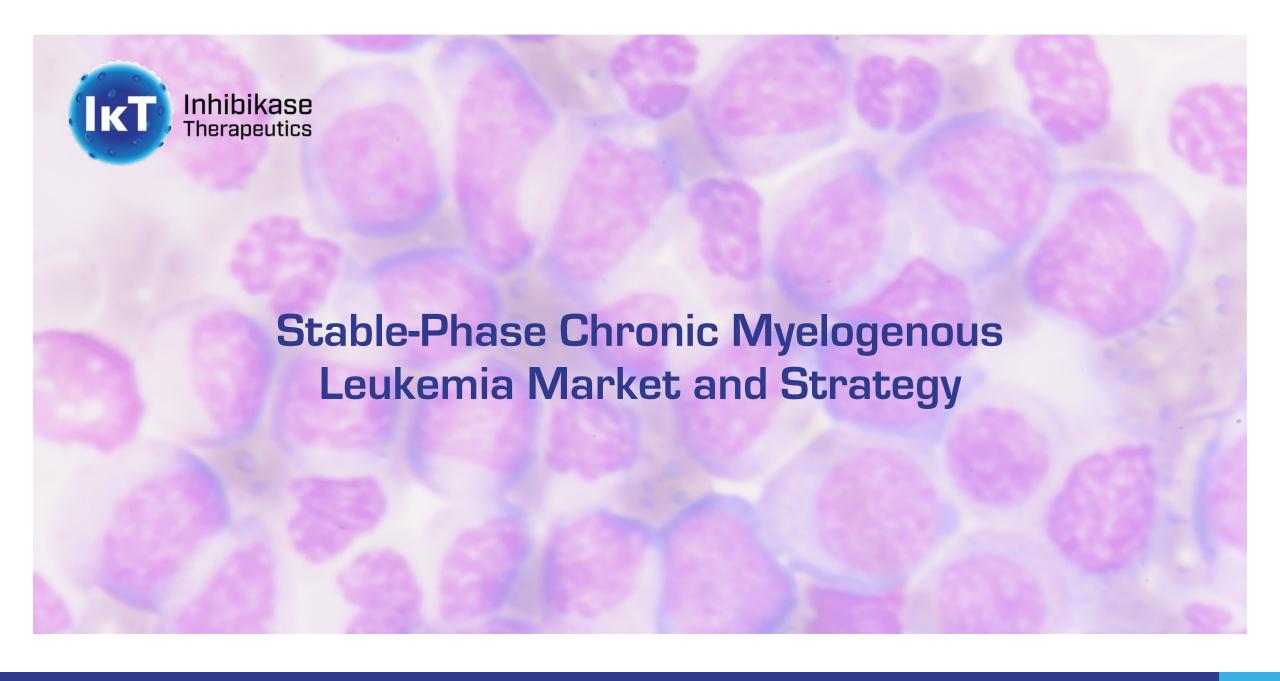
Seed amplification assays

Time to initiation of PD

symptomatic medication

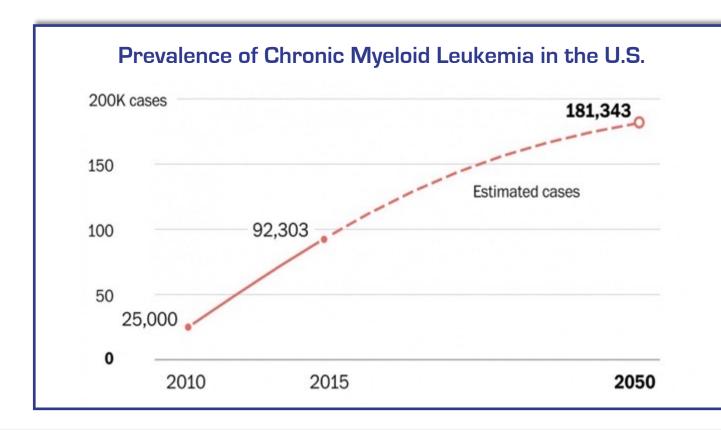
Time before initiation of PD

symptomatic medication





# CML in the U.S.<sup>1</sup> Accessible Market Opportunity Despite Presence of Generic



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

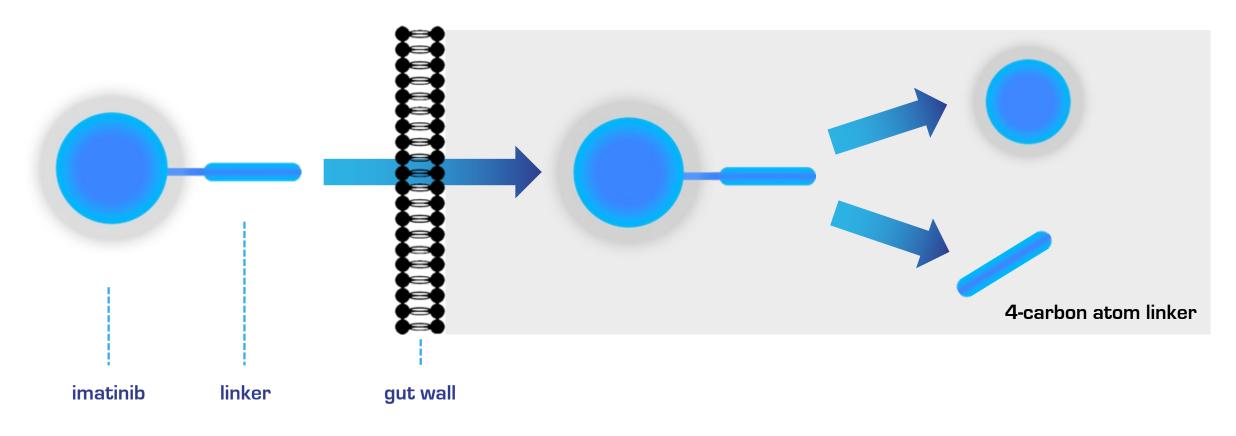
<sup>1</sup>Jabbour E, Kantarjian H. Am. J. Hematol. 89:548–556 <sup>2</sup>IMS-Iqvia retail sales data 2016-2020 <sup>3</sup>Am J. Hematology (2019) 94:46-54 <sup>4</sup>Annals of Hematology (2018) 97:1357–1367

\$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 Gl adverse events



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



pH 2-6 (stomach, ileum, duodenum)

pH 7<sup>+</sup> (blood)
Lifetime in blood < 10 min



#### IkT-001Pro:

# Lower GI Toxicity Alternative to Generic Gleevec®

#### Measurement of IkT-001Pro in Non-Human Primates

	No Adverse Event Level (mg/kg) <b>NOAEL</b>	Cmax (mean, ng/mL)	Tmax (mean, h)	AUC <sub>0-T</sub> (mean, ng-h/mL)
Imatinib	15	176/206	4/3	1540/1960
(Day 91) <sup>1</sup>		(M/F)	(M/F)	(M/F)
lkT-001Pro	75	400/318	5.3/3.7	5220/3890
(Day 28)		(M/F)	(M/F)	(M/F)

**RESULTS SUGGEST THAT:** 

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

<sup>1</sup>FDA summary data for approval 21-335



NO ADVERSE EVENTS

Up to 3.4x increased exposure

Up to 3.4x higher dose of imatinib

delivered as 001Pro

Inhibikase.com • Nasdaq : IKT

21



# Clinical Development of IkT-001Pro



**kT**- 001Pro **Bioequivalence '501'** Trial

#### Single and multi-dose three-part trial to bioequivalence

- Part 1: 1 safety cohort (3 subjects), 3 dosing cohorts of 8 subjects each to find dose equivalent to 400 mg imatinib mesylate measuring 96 hr pharmacokinetics; 4 cohorts completed as of April, 2023
- Part 2: Single dose measure in 32 subjects confirming 96 hr pharmacokinetics from Part 1; 2023 anticipated completion
- Part 3: 8 subject, 5-day steady-state bioequivalent to 600 mg imatinib; 3Q23 anticipated completion
- Will confer with FDA on NDA path under the 505(b)(2) statute following the 501 trial

**kT**- 001Pro **Planned '502' Trial** 

#### Superiority to standard of care in CML

- Wait-list control crossover design comparing 001Pro to 400 mg imatinib over 6 months
- 98 patients dosed 1x/day
- Primary endpoints are patient-reported outcome measures of GI disturbance and frequency of diarrhea.
- Planned to conduct '502' trial coincident with pursuit of NDA under the 505(b)(2) statute

Nasdaq: IKT Inhibikase.com •





## **Selected Financial and Stock Data**

Capitalization Table	March 15, 2023				
Common Shares Outstanding	28,977,238				
Options (WAEP: \$2.41)	4,001,208				
Warrants (WAEP: \$0.77)	21,474,519				
Fully Diluted Shares Outstanding	54,471,126				

\$20.8M non-dilutive grant revenue pre-IPO (NIH, DoD, State gov'ts)



Balance Sheet	December 31, 2022 (last reporting period)
Current Assets:	
Cash, Cash Equivalents, Marketable Securities	\$23,050,173
Grants Receivable	\$39,881
Prepaid research and development	\$1,117,616
Prepaid expenses and other current assets	\$163,452
Total Current Assets	\$24,371,122
Total Current Liabilities	\$3,695,445
Working Capital	\$20,675,677
Active grant funding available in accounts held by the U.S. treasury	\$300,386
Total Working Capital + Available Grant Funds	\$20,976,063

\$10M gross proceeds from equity sales raised January 25, 2023



# **Upcoming Milestones: 2Q 2023**



- Open up to 35 201 trial sites by close 2Q23
- Active screening at at least 20 201 trial sites
- PK analysis of commercial formulation
- Implement 12-month open-label safety extension study into 201 trial
- Characterize novel compounds as follow-ons to IkT-148009



- Complete through Part 2 of 501 trial
- Explore commercial manufacturing scale-up
- Initiate high dose imatinib 600 mg bioequivalence study



# 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.







#### Joseph Frattaroli, CPA

**Chief Financial Officer** 

Mr. Frattaroli is a certified public accountant with more than 15 years of experience in public company filings and compliance for Nasdaq and OTC Markets companies. Previously, he provided chief financial officer and consulting services for several emerging biopharmaceutical and medical device companies, with responsibilities that included capital formation, deal structuring, and assisting private companies in their transition to becoming publicly traded SEC registrants.







#### 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.







#### 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.,
- Currentlly 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.



#### 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

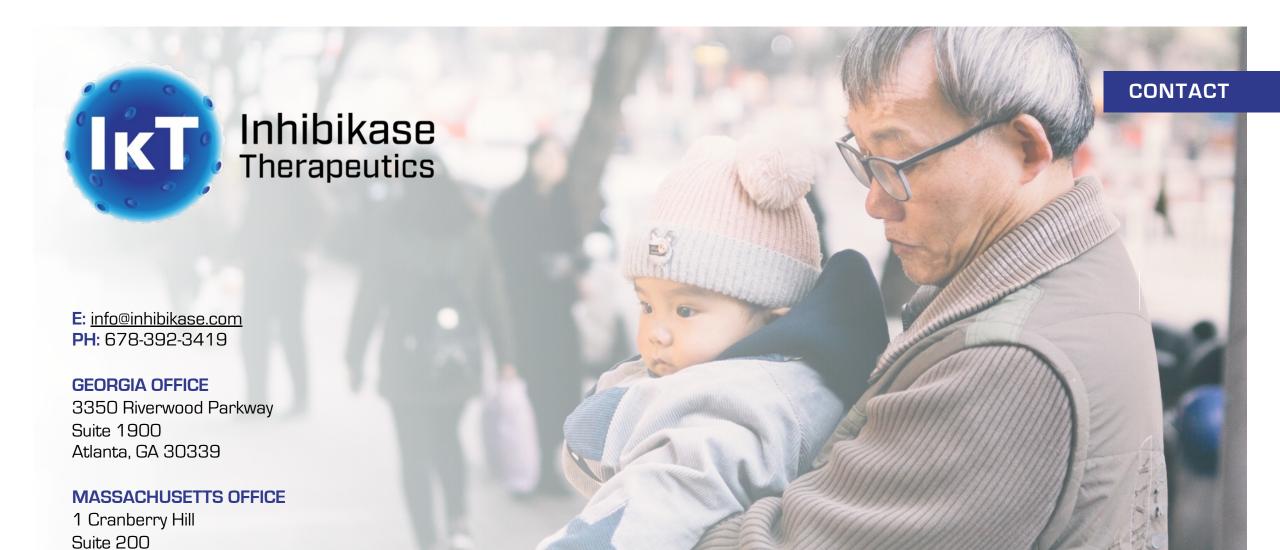
#### Dr. Karl Kieburtz, MD, MPH

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





Inhibikase.com Nasdaq: IKT

Lexington, MA 02421