
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): April 21, 2022

INHIBIKASE THERAPEUTICS, INC.

(Exact Name of Registrant as Specified in its Charter)

Delaware
(State or Other Jurisdiction
of Incorporation)

001-39676
(Commission
File Number)

26-3407249
(IRS Employer
Identification No.)

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

30339
(Zip Code)

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

(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 April 21, 2022, Inhibikase Therapeutics, Inc. (the “Company”), made available on the Company’s website at www.inhibikase.com a corporate presentation which may be used in presentations to investors and analysts from time to time in the future. A copy of the Company’s corporate presentation is attached hereto as Exhibit 99.1 and is incorporated herein by reference.

The information contained in this Current Report on Form 8-K speaks only as of the date hereof. While the Company may elect to update the information in this Current Report on Form 8-K in the future, the Company disclaims any obligation to do so except to the extent required by applicable law.

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.

<u>Number</u>	<u>Description</u>
99.1	Corporate Presentation of Inhibikase Therapeutics, Inc.
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: April 21, 2022

INHIBIKASE THERAPEUTICS, INC.

By: /S/ MILTON H. WERNER

Milton H. Werner, Ph.D.

President and Chief Executive Officer



**Inhibikase
Therapeutics**

Nasdaq | **IKT**

Clinical Development of Disease- Modifying Therapeutics for Parkinson's and Related Disorders

2Q 2022 BUSINESS PRESENTATION



Disclaimer

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, 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.

Simultaneous Evaluation of Brain and GI Function Expands the Definition of a Disease-Modifying Treatment

- PD is more than just a disease of the brain. GI manifestation in many patients occurs at an early stage, suggesting that evaluation of GI and brain function could be essential to identifying truly disease-modifying treatments
- Utilization of slowly progressive, α -synuclein dependent animal models that reproduce the rate of disease progression relative to lifespan of the human disease have been key to properly identifying potential disease-modifying therapeutics.
- Our lead asset is an inhibitor of the Abelson Tyrosine Kinase (c-Abl). IKT-148009, halts and reverses functional loss; discovered using slowly progressive animal models.
- Six clinical programs in 2022 across two assets in Parkinson's disease, Multiple Systems Atrophy (orphan indication, orphan designation not sought) and Chronic Myelogenous Leukemia (orphan indication, orphan designation granted).
- Phase 2 programs anticipated to begin early 2Q22 in neurodegeneration, readouts anticipated within 12 months.
- Multiple patent families for lead compound with expiration of 2036 and beyond
- \$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
- Highly experienced and respected management team, consultants, Board of Directors and nearly all KOLs in the field on Scientific Advisory Board

Multi-Indication Pipeline in Neurodegeneration, Oncology and Infectious Disease

Drug Target	Drug candidate	Modality	Disease indication	CLINICAL DEVELOPMENT ¹				BIOMARKER ³						
				PRECLINICAL DEVELOPMENT	PHASE 1/1b	PHASE 2	PHASE 3	Preclinical target engagement	Clinical target engagement	Can be used for patient selection				
Neurodegeneration														
c-Abl	IKT-148009	Small molecule	Parkinson's Disease: Treatment Naïve					Validated	Validating	Yes				
c-Abl	IKT-148009	Small molecule	Parkinson's Disease: Early Stage							Validated	Validating	Yes		
c-Abl	IKT-148009	Small molecule	Neurogenic Constipation									Validated	Validating	Yes
c-Abl	IKT-148009	Small molecule	Dysphagia									Validated	Validating	Yes
c-Abl	IKT-148009	Small molecule	Multiple System Atrophy							Validated	Validating	Yes		
Oncology														
BCR-Abl	IKT-001Pro	Small molecule	Stable-phase CML (orphan indication)					Validated	Validated	Yes				
Research Phase														
c-Abl	IKT-148x	Small molecule	Dementia with Lewy Body					Validated	Validating	Unknown				
c-Abl	IKT-148x	Small molecule	Multiple System Atrophy					Validated	Validating	Unknown				
c-Abl	IKT-1427	Small molecule	Progressive multifocal leukoencephalopathy					Validated	Validating	Yes				

(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 two INDs; one focused on treatment in the brain in treatment naïve or early-stage patients and the second focused on GI complications. MSA is a Parkinson's-like disease to enter clinical development at Phase 2 sharing the Phase 1 data for 148009 with PD. MSA moves forward in clinic ONLY if animal model study ongoing is positive.

(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 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 treatment.



**Inhibikase
Therapeutics**

Nasdaq | **IKT**

Parkinson's Disease Market & Strategy



THE MARKET

Parkinson's Disease in the U.S.¹

Large Market, Opportunity For Disease Modification

Chronic Disease for a Long Time

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

60,000

NEW CASES / YR

38,000

DEATHS / YEAR

700,000 – 1,000,000

U.S. Patients

60

AVERAGE AGE
OF ONSET



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

Other illnesses complicate development



47%
ARTHRITIS



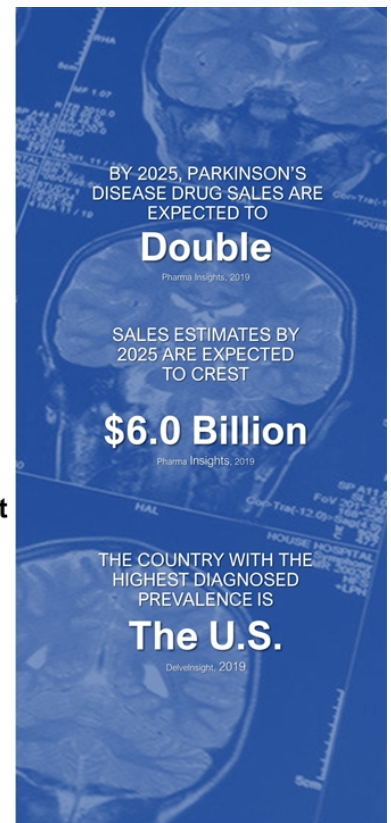
36%
HEART / CIRCULATORY



35%
PSYCHOSIS

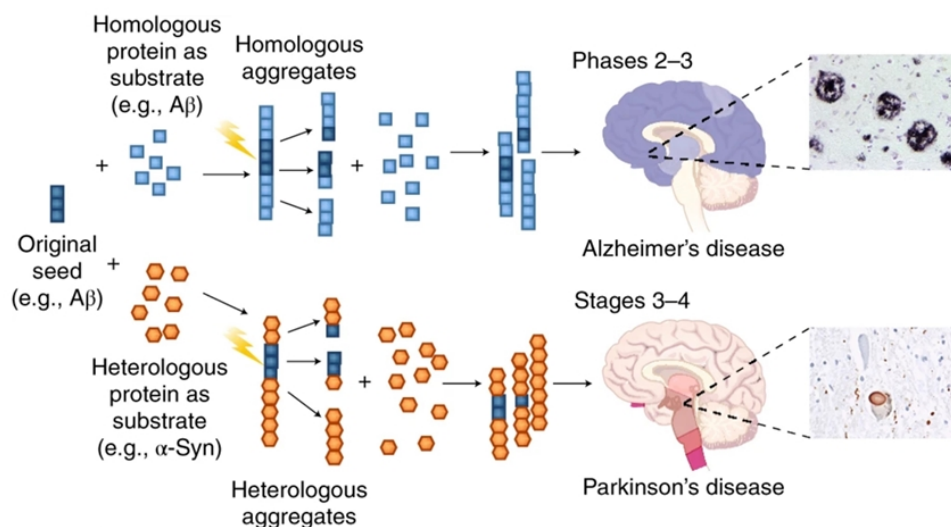


30%
DEMENTIA



¹Parkinson's Disease Foundation Decisions Resources 2016, ParkinsonismRelatDisord. 2012;18:1073-1078, Neuroepidemiology 2010;34:143-151, J Neurol Neurosurg Psychiatry. 1997 Jan;62(1):10-5.

Causation in Parkinson's and Alzheimer's is closely related¹



What role does the misfolded protein play?

¹Nat. Neurosci. 21: 1332-1340 (2018)

Evaluation of the Misfolded Protein ‘Seed’ in Parkinson’s Reveals c-Abl as the Primary Culprit (video)

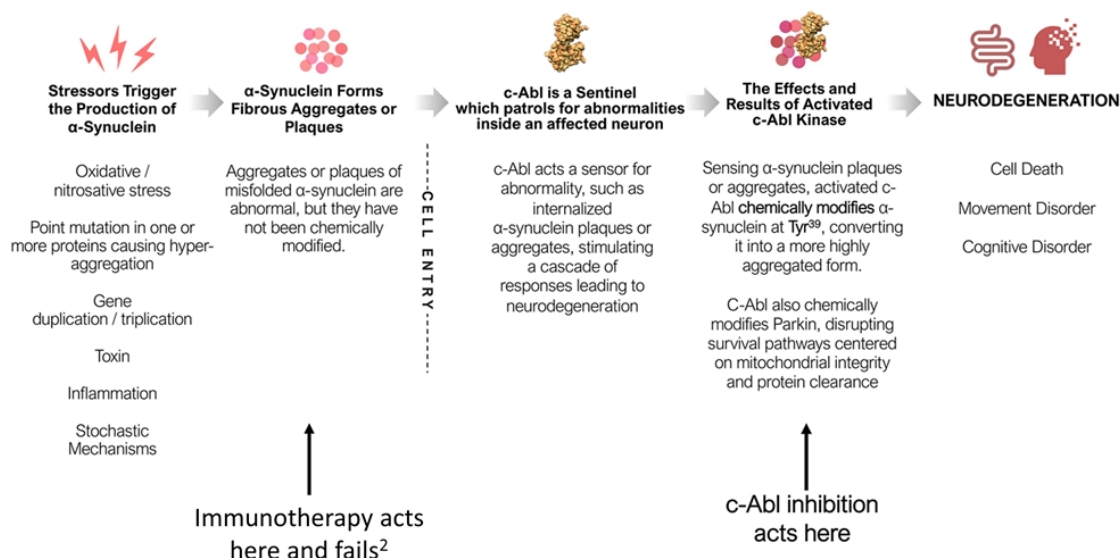


Movement Disorders Official Journal of the International Parkinson and Movement Disorder Society

Parkinson's Disease Modification Through
Abl Kinase Inhibition: An Opportunity
DOI: 10.1002/mds.28858

WILEY

Stressors Trigger the Production of Misfolded α -Synuclein Which Activates c-Abl to Drive Neurodegeneration²



¹ *Nat Rev Neurosci*. 2, 492–501 (2001)

² Werner and Olanow, *Mov Disorders* 2021, doi: 10.1002/mds.28858

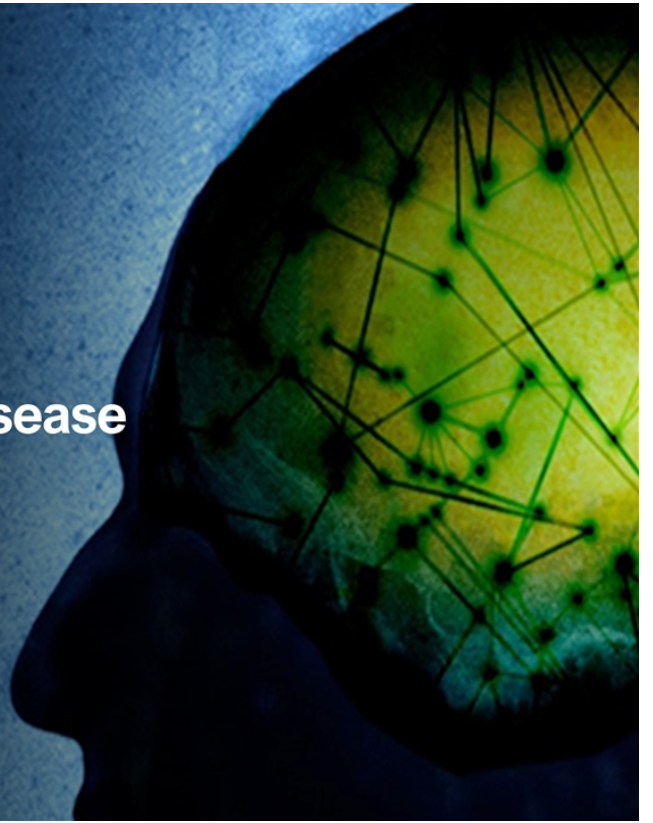
³ <https://ir.prothena.com/news-releases/news-release-details/update-phase-2-pasadena-study-prasinezumab-prx002rg7935>
<http://media.biogen.com/node/22876/html>



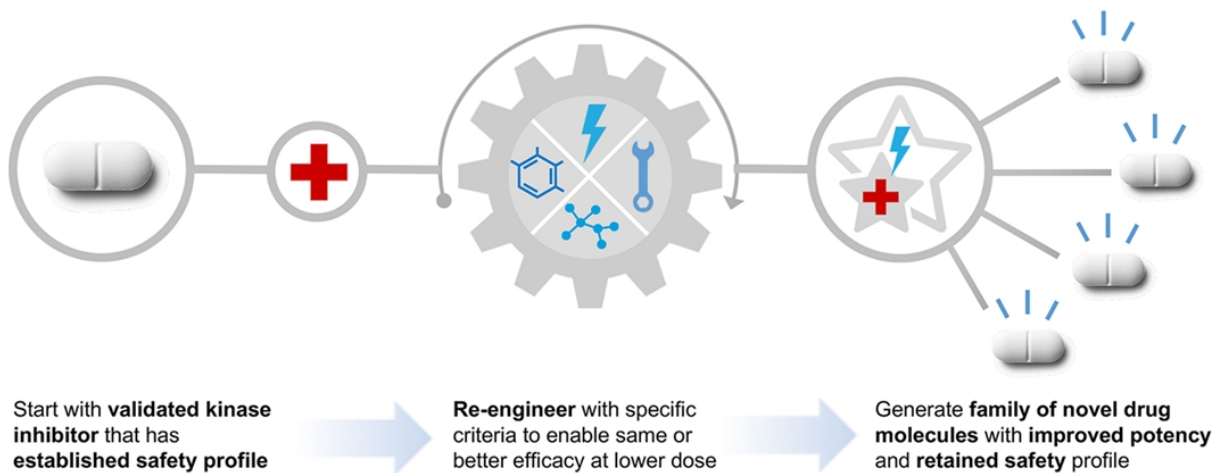
**Inhibikase
Therapeutics**

Nasdaq | **IKT**

How Inhibikase Will Modify Disease



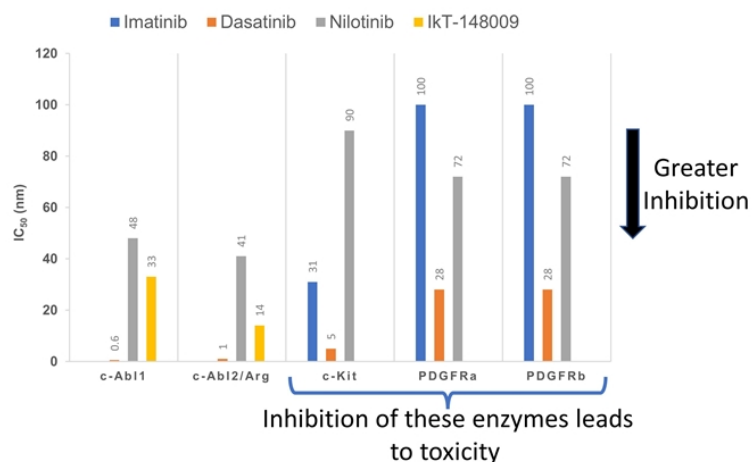
Re-engineering Approach with Metabolism Preserved (RAMP™)



Ikt-148009: Low Toxicity, Selective, Brain Penetrant c-Abl Inhibitor in Clinical Development

Ikt-148009
Inhibikase Therapeutics

Selective Inhibitor of c-Abl and Abl2/Arg
bypasses toxicity of cancer drugs¹



Ikt-148009
Inhibikase Therapeutics

No organ toxicity
High brain penetrance

Toxicology in Rat/Monkey ¹	
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
¹ 13 week and 39 week toxicology data shows Ikt-148009 has a more favorable toxicity profile as dosing is extended	

¹See SelleckChem.com, Leuk 23:1689ff (2009)

c-Abl inhibition by IkT-148009 blocks the four pillars of Parkinson's Disease in Validated Animal Models

a-Synuclein Toxicity



IkT-148009 clears to baseline in the organs of disease

Neurodegeneration



IkT-148009 preserves as much as 85% of brain neurons

Motor Dysfunction



IkT-148009 restores as much as 90% of lost function

Neuroinflammation



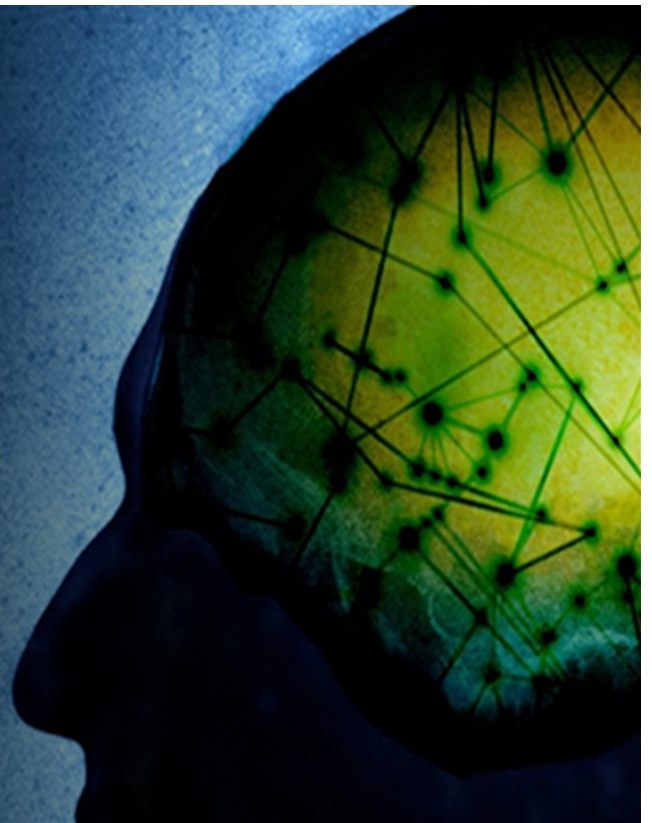
IkT-148009 suppresses to near baseline in the organs of disease



Inhibikase
Therapeutics

Nasdaq | **IKT**

Clinical Development



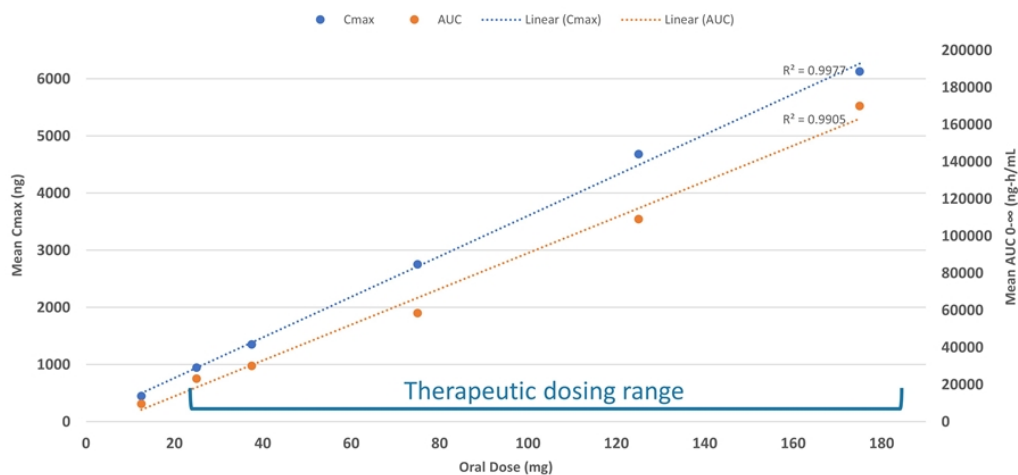
Phase 1: Dose Proportional Clinical Pharmacokinetics and No Clinically Significant Adverse Events

Category	Demographic	Value (% of Total N=88)
Gender	Female	34 (38.6)
	Male	54 (61.4)
Age	Average (SD)	57.9 (5.72)
	Median	58.0
	Range	45, 69
Ethnicity	Hispanic or Latino	13 (14.8)
	Not Hispanic or Latino	75 (85.2)
Race	Black or African American	54 (61.4)
	White	33 (37.5)
	Other	1 (1.1)
Adverse events		7 (7.9), all clinically insignificant

No GI
No Cardiovascular
No Hematological

Phase 1:Dose Proportional Clinical Pharmacokinetics and No Clinically Significant Adverse Events

Clinical Pharmacokinetics of IKT-148009-SAD



Significance of clinical pharmacokinetics

- High exposures at low oral dose, linearly dose proportional up to 175 mg. Exposures at 75 mg IKT-148009 comparable to 500 mg imatinib¹

¹FDA summary data for approval 21-335

Phase 1b: Mild to Moderate PD (H&Y < 3.0) and No Clinically Significant Adverse Events

Category	Demographic	Value (% of Total N=13)
Gender	Female	6 (42.8)
	Male	7 (57.2)
Age	Average	62.5
	Median	62
	Range	57, 70
Ethnicity	Hispanic or Latino	3 (23.1)
	Not Hispanic or Latino	10 (76.9)
Race	Black or African American	2 (15.4)
	White	11 (84.6)
	Other	0 (0)
Adverse events		5 (38.5), (pneumonia, spinal headache, dermatitis, constipation, orthostatic hypotension)

Phase 1b: Pharmacokinetics similar to elderly healthy subjects

			T _{1/2} (h)	T _{max} (h)	C _{max} (ng/ml)	AUC ₀₋₂₄ (ng-h/mL)	V _z /F (l)	CL (l/h)
Day 1	Mean	25 mg	15.4	5	1040	12700	32.5	1.52
N=6	SD	Healthy	11.3	4	419	6010	14.7	0.905
Day 7	Mean		27.4	4.67	1770	25400	42.8	1.1
N=6	SD		5.09	1.63	807	9260	15.3	0.384
Day 1	Mean	50 mg	10.1	4.67	1720	19400	37.2	2.51
N=6	SD	PD	2.7	1.03	737	9470	22.8	1.2
Day 7	Mean		24.9	3.67	2560	32500	57.1	1.61
N=6	SD		3.86	1.51	564	8500	12.4	0.312

Trends in motor and non-motor scores and assessments and in GI function that may have occurred over 7 day-dosing need more cohorts to be completed to draw any conclusions.

Clinical Phase 1b and Phase 2 Programs

Phase 1b ONGOING

Multiple Ascending Dose (MAD): 7-Day Dosing

- 3 dosing cohorts, 3:1 randomized with placebo, doses determined from SAD PK and safety
- 24 patients total
- 8 patients/dose 7-day dosing 1x/day
- Hoehn & Yahr (H&Y) < 3.0
- Primary endpoints: safety, tolerability, pharmacokinetics (PK), urine, plasma and spinal fluid concentrations

Multiple Ascending Dose (MAD): 7-Day Dosing

- Exploratory endpoints: UPDRS II, III, II+III, NMSS, PDQ-39, CSBM, PAGI-SYM

IKT-148009 Phase 1b MAD (6-8 Months)

Oct. 2021 ▶ 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18

Apr/May 2022 ▶

IKT-148009 Phase 2a (Up to 12 months)

Phase 2a 2Q22

Multiple Ascending Dose (MAD): 3 Mos Dosing

- 3 dosing cohorts, 1 placebo cohort
- 120 patients total
- 30 patients/dose 3:1 randomized 13-week dosing 1x/day
- Treatment naïve/Early state patients (H&Y ≤ 2.0)
- Primary endpoints: safety, tolerability

Multiple Ascending Dose (MAD): 3 Mos Dosing

Animal GI Recovery < 4 weeks
Animal Brain Recovery < 8 weeks

- Secondary endpoints UPDRS II, III, II+III, PGI-S, CGI-S, Epworth Sleepiness Scale, NMSS, PDQ-39, CSBM, PAGI-SYM, PAC-QOL, PAGI-SYM-QOL
- Exploratory endpoints: Phospho-a-synuclein GI, Skin and CSF; Whole Gut Transit Time (SmartPill™)
- Descriptive statistics

Selected Financial and Stock Data

Capitalization Table	March 31, 2022
Common Shares Outstanding	25,227,051
Options (WAEP: \$2.47)	4,208,056
Warrants (WAEP: \$5.21)	1,561,913
Fully Diluted Shares Outstanding	30,997,020

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

Balance Sheet	December 31, 2021 (last reporting period)
Current Assets:	
Cash	\$ 40,750,133
Grants Receivable	\$ 110,141
Prepaid research and development	\$ 107,000
Prepaid expenses and other current assets	\$ 1,502,725
Total Current Assets	\$ 42,469,999
Total Current Liabilities	\$ 4,054,450
Working Capital	\$ 38,415,549
Active grant funding available in accounts held by the U.S. treasury	\$ 385,888
Total Working Capital	\$ 38,801,437

Upcoming Milestones

➤ 2Q22

- 148009
 - Complete Phase 1b study in Parkinson's patients with mild to moderate disease
 - Initiate dosing in Phase 2a study in early-stage Parkinson's patients
 - Complete first of two animal model validation studies of IkT-148009 in MSA
 - Set-up EU/US sites for Phase 2 studies in MSA anticipated to commence in 3Q22
 - Meet with FDA to review Phase 2 and Phase 3 development plans
- 001Pro
 - File IND and commence bioequivalence clinical studies
 - Design and develop superiority studies for IkT-001Pro relative to standard-of-care
 - Identify and begin developing commercial partnership

Management Team with Deep Experience in Drug Development and Commercialization

Executive

Milton Werner, PhD President & CEO

Previously, Dr. Werner served as Director of Research at Caltaxsys. 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, Interim Chief Medical Officer and Chief Executive Officer of CLINTREX.

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.



Board of Directors

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. 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 Amplitx Pharmaceuticals and Synedgen.
- Served in senior management roles at Cerebra, 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. 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.

Ms. Elizabeth O'Farrell

- 25-year career with Eli Lilly and Company, lastly serving as Chief Procurement Officer and Leader, Global Head of Shared Services
- Served in senior management at Lilly including Senior Vice President, Policy and Finance; Senior Vice President, Finance; Chief Financial Officer, Lilly USA; Chief Financial Officer, Lilly Canada; and General Auditor. Before joining Eli Lilly, Ms.
- Director of PDL BioPharma, Geron Corporation and Lensar
- BS in accounting with honors and an MBA in management information systems from Indiana University.

Industry-Leading Advisors

Robert Hauser, MD

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

Jeffrey Kordower, PhD

Alla V and Solomon Jesmer Professor of Aging & Neurological Sciences Rush University Medical Center

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, FRCP

Henry P. and Georgette Goldschmidt Professor and Chairman Emeritus, Mount Sinai School of Medicine Clintrex, Inc.

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 Clintrex, Inc.

Dr. Jay Pasricha, MBBS, MD

Director, Johns Hopkins Center for Neurogastroenterology Professor of Medicine

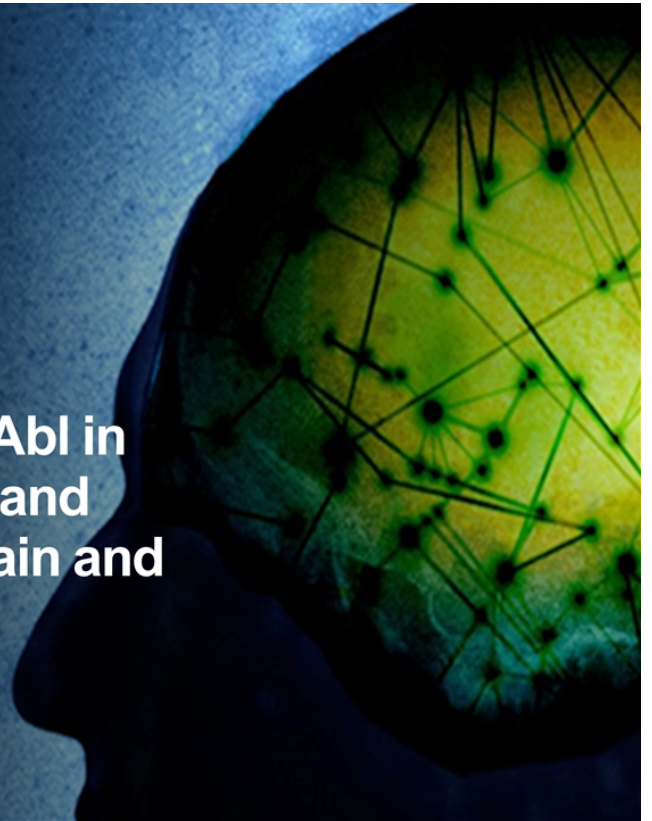


**Inhibikase
Therapeutics**

Nasdaq | **IKT**

Appendix

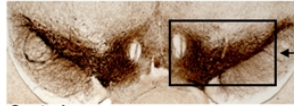
**Proof of the importance of c-Abl in
Disease, Target Engagement and
Functional Reversal in the Brain and
Gut**



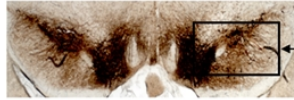
α -Synuclein Plaques Do Not Cause Disease Without c-Abl Modification in Humanized Preclinical Models¹

α -Synuclein plaque in the **ABSENCE OF c-Abl CAUSES NO NEURODEGENERATION AFTER 6 MONTHS**

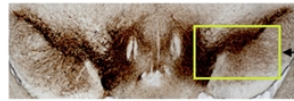
AAV-tTA (6 month post inj)
Non Inj Inj



Control



TetP-A53T α -syn

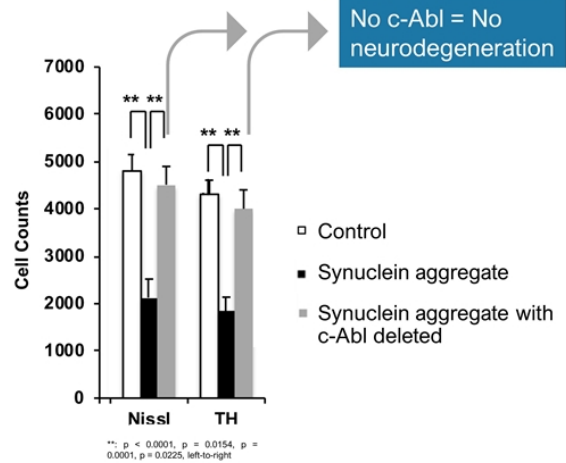


c-Abl-KO/TetP-A53T α -syn

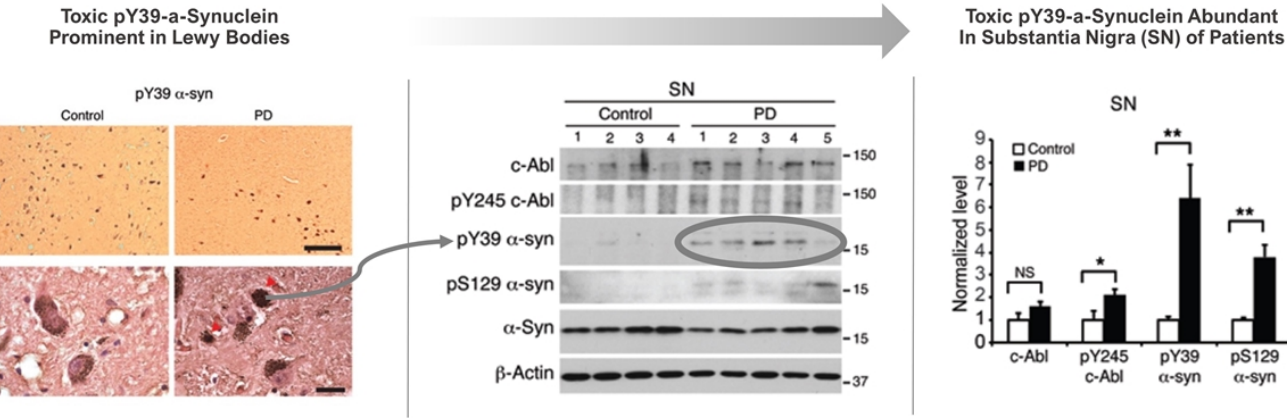
Injection (Inj) of an expression vector for the A53T form of synuclein doesn't degrade neurons until the expression of A53T is turned on

Once turned on, A53T induces 50% neurodegeneration in 6 months. NOTE loss is visible in the dark patches of stained neurons on the right half. The left half was an internal control and is unaffected.

When the expression of A53T is turned on, but c-Abl is genetically deleted from the mouse brain, you don't get any neurodegeneration. Thus, even though the clumps of α -syn are present in the right region of the brain, they don't cause disease until c-Abl acts on them



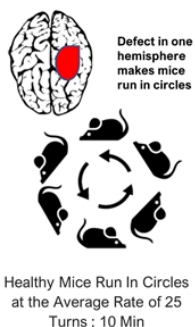
Pathologic, c-Abl-Modified α -Synuclein (pY39) is Present in Parkinson's Patient Brain¹



¹J Clin Invest. 126, 2970-88 (2016)

Oral IKT-148009 in Mice Humanized for Parkinson's Disease in Brain Reverses Functional Loss

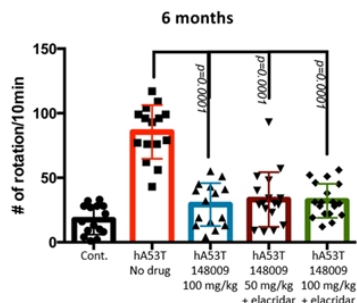
Baseline Neuromuscular Coordination Behavioral Marker



Toxic Levels of Synuclein Are Introduced Into One Side of the Mouse Brain



Treatment Started 6 Weeks After Onset of Conditions



Near Normal Behavior Returned Following Treatment

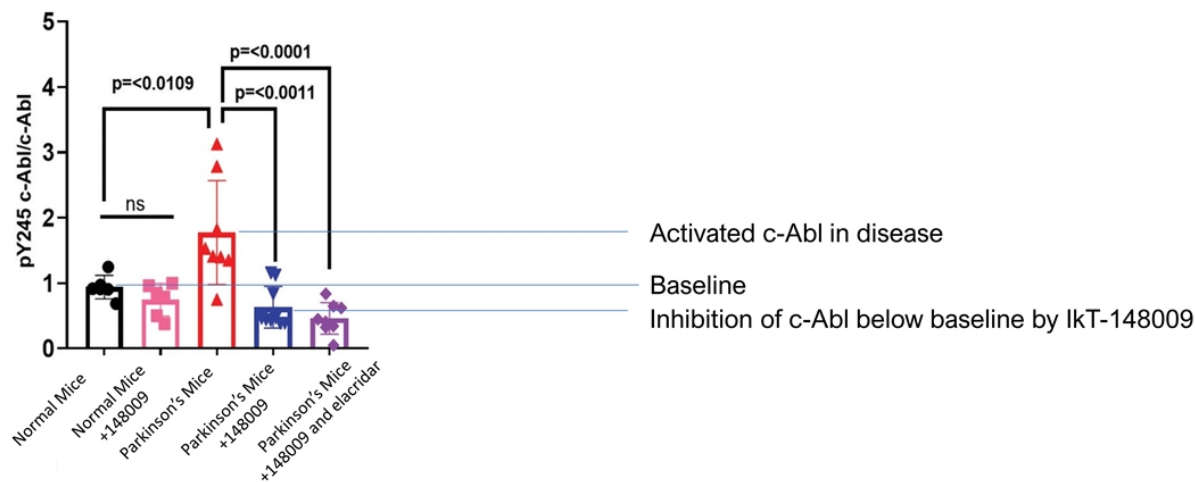


Mice completed 30 turns/10 min when treated a dose of 100 mg/kg dose

NEARLY COMPLETE RESCUE

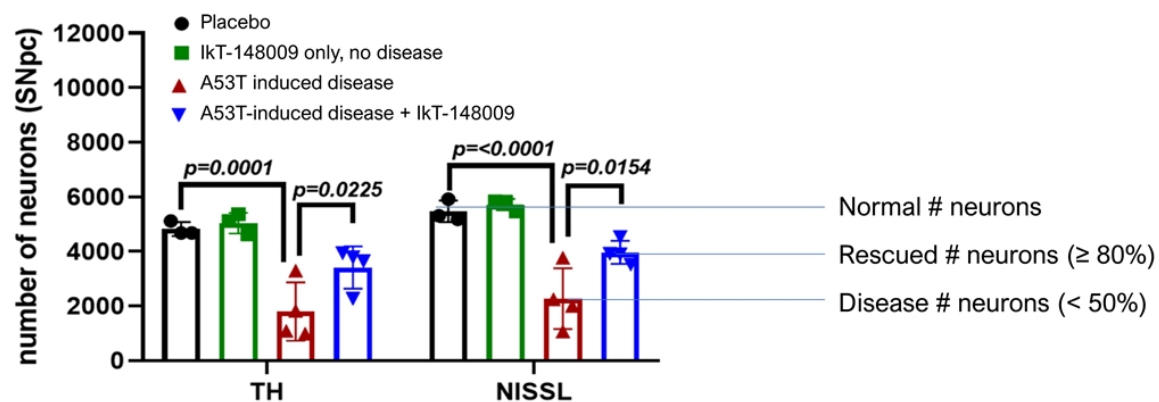
Oral IkT-148009 Suppresses c-Abl Activation in the Brain that Correlates with Functional Recovery

IkT-148009 engages the c-Abl target in the brain



Oral IkT-148009 Preserves Neural Anatomy in the Brain

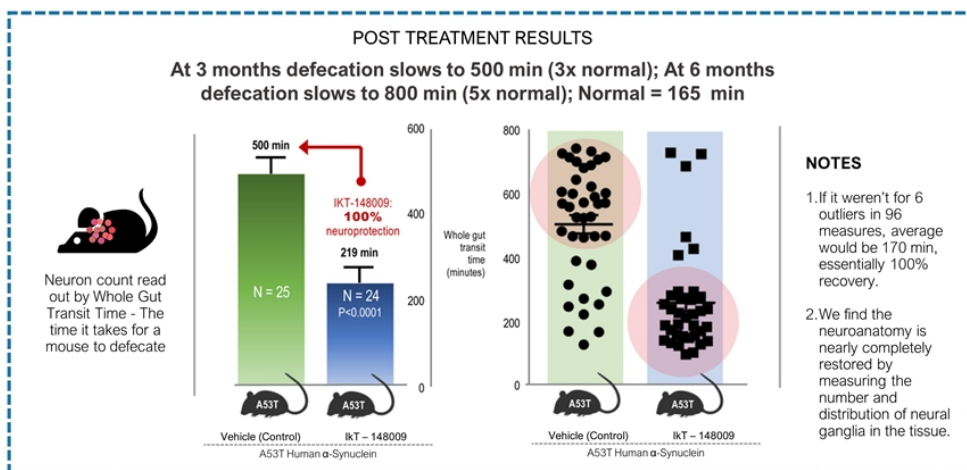
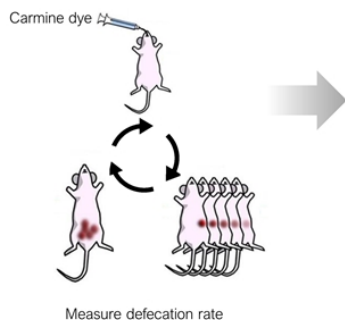
IkT-148009 stopped loss of neurons, accounting for functional recovery



Oral IkT-148009 in Mice Humanized for Parkinson's Disease in Gut Reverses Functional Loss

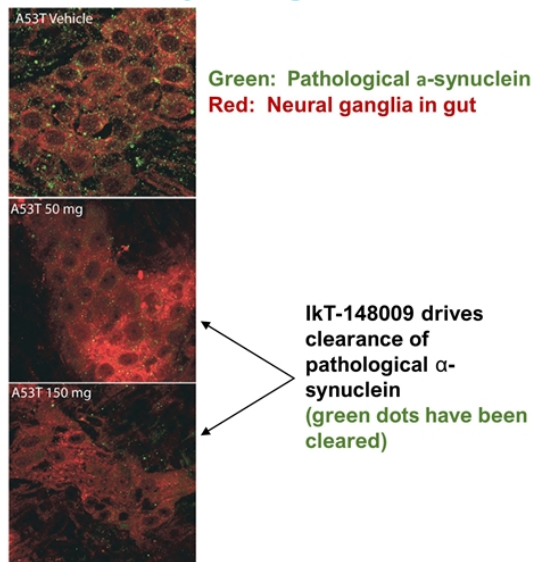
METHOD OF MEASUREMENT

α -Synuclein Aggregates Introduced Into Gut Slows Gut Transit Time

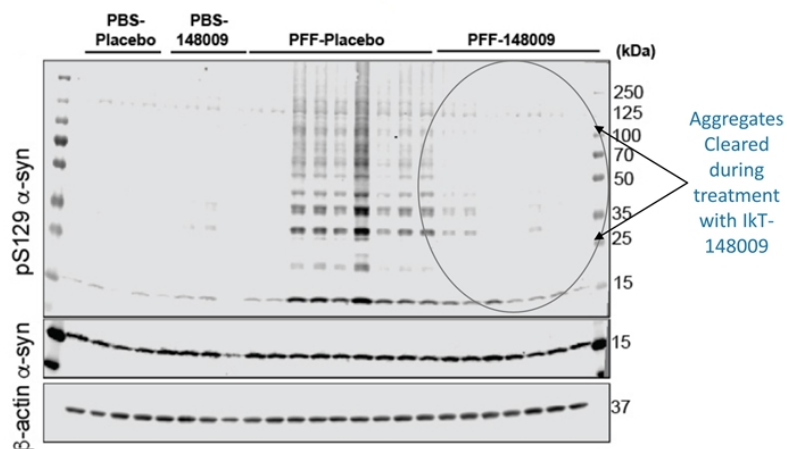


Oral IkT-148009 Treatment Clears Toxic α -Synuclein in the brain and gut

Clearance of toxicity in the gut



Clearance of toxicity in the brain



Advances in pre-clinical models and clinical dosing

- IKT-148009 drives functional recovery inside and outside of the brain
- IKT-148009 drives clearance of the toxic form of α -synuclein
- Low oral doses in humans achieve therapeutic exposure levels observed in animal efficacy studies

Targeting c-Abl we believe is transformational to treatment of neurodegeneration