Inhibikase Therapeutics

Nasdaq | IKT

Therapies in Clinical Development to Reverse the Progression of Neurodegeneration in Parkinson's Disease

1Q 2021 INVESTOR PRESENTATION

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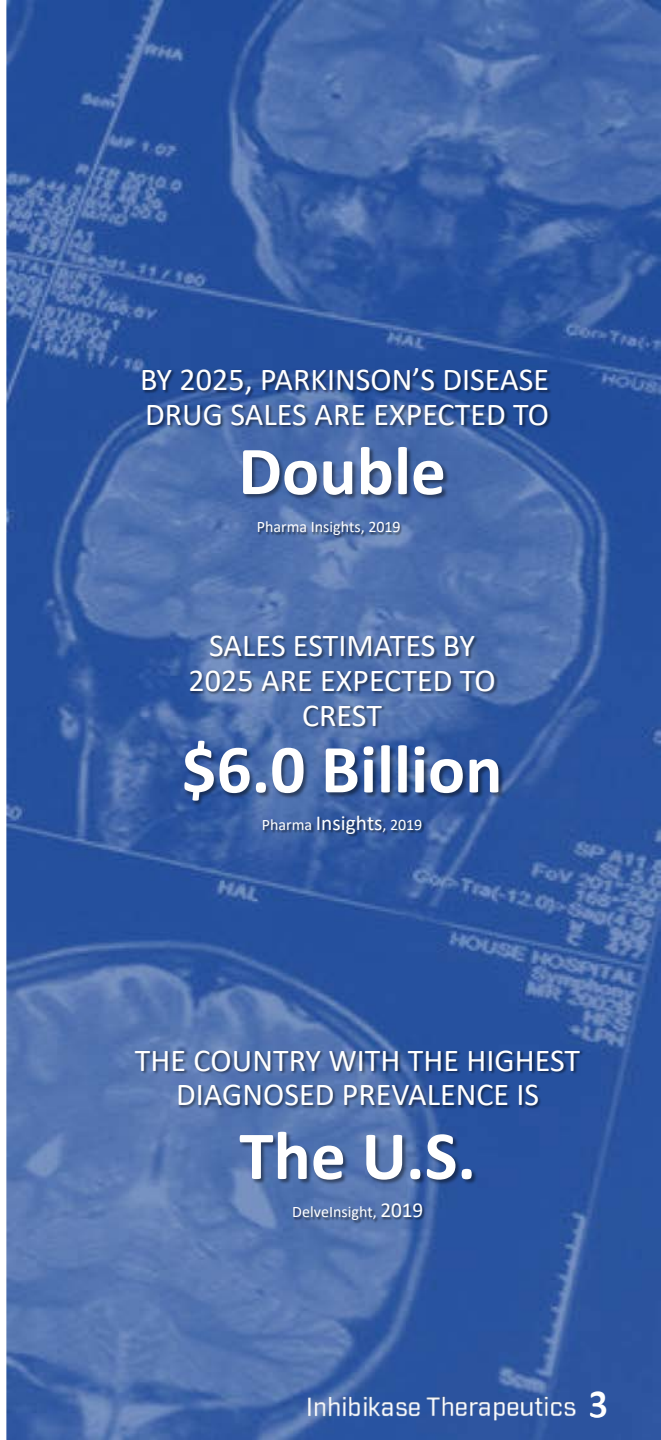
We do not intend our use or display of other entities’ names, trade names, trademarks or service marks to imply a relationship with, or endorsement or sponsorship of us by, any other entity.

Driving Functional Reversal of Parkinson's Disease

- Multi-therapeutic pipeline with emphasis on neurodegeneration
- Lead programs developing drugs targeting a gateway along a common pathway that initiates Parkinson's Disease (PD) inside and outside of the brain
- IkT-148009 shown to therapeutically halt and reverse functional loss inside and outside of the brain in animal models that recreate progressive human disease
- Two INDs filed for Parkinson's Disease and its GI complications
    - Shared Phase 1 study to dose patients mid-February, 2021
- Multiple patent families for lead compound with expiration of 2036 and beyond
- $20.4 million in grants and contracts from NIH, DoD, the Michael J. Fox Foundation and the Georgia Research Alliance, all peer-reviewed
- 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

				CLINICAL DEVELOPMENT[1]				BIOMARKER		
Drug Target	Drug candidate	Modality	Disease indication	PRECLINICAL DEVELOPMENT	PHASE 1	PHASE 2	PHASE 3	Preclinical target engagement[1]	Clinical target engagement[1]	Can be used for patient selection[1]
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		4 Indications Pursued Through 2 INDs			Validated	Validating	Yes
c-Abl	**IkT-148009**	Small molecule	Dysphagia					Validated	Validating	Yes
Oncology										
BCR-Abl	**IkT-001Pro**	Small molecule	Stable-phase CML (orphan indication)		505(b)(2) Path to Market			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. Four indications will be pursued for IkT-148009, 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 of PD patients. All four indication paths will share the same Phase 1 study in elderly healthy volunteers. The Company anticipates initiating a Phase 1b study in treatment naïve and early stage Parkinson's patients with GI complications for IkT-148009 in the next 12 months subject to additional financing. The Phase 2 study may be shared in whole or in part for all four indications. IkT-148x refers to a series of portfolio compounds being evaluated for these indications in preclinical models that are from the same chemical family as IkT-148009. 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 to validate 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 treatment.

Parkinson's Disease in the U.S.[1]

Large Market, Opportunity For Disease Modification

Mortality

1/3 of a Patient's Lifespan

60,000
NEW CASES / YR

38,000
DEATHS / YEAR

1.5x–2x
Men are more likely to be afflicted

Prevalence

700,000 – 1,000,000

60
AVERAGE AGE OF ONSET

Comorbidities

47%
ARTHRITIS

36%
HEART / CIRCULATORY

35%
PSYCHOSIS

30%
DEMENTIA

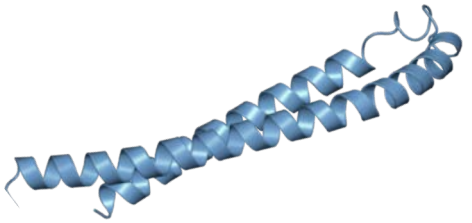
Parkinson's Disease Foundation Decisions Resources 2016, ParkinsonismRelatDisord . 2012;18:1073-1078, , Neuroepidemiology 2010;34:143–151 , J Neurol Neurosurg Psychiatry. 1997 Jan;62(1):10-5.

Clarification of Causation in Parkinson's Reveals c-Abl as a Primary Culprit

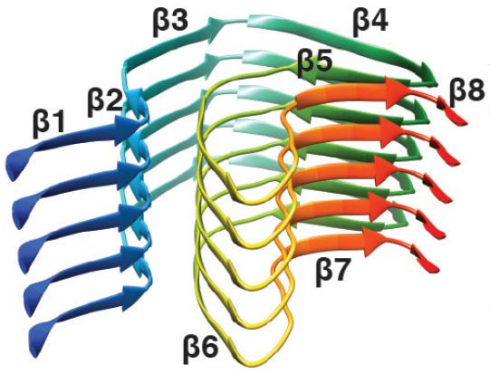
- Parkinson's Disease (PD) is a neurodegenerative disease which limits function of nerve cells throughout the brain and gut.
- PD conditions include:

Tremors	Slowed Movement	Impaired Balance	Permanent Constipation
Speech Loss	Cognitive Decline	Memory Loss	and Reflux Disease

- α-Synuclein, an abundant and non-essential protein, has long been thought to be linked to the cause of Parkinson's Disease
  - Normally, α-Synuclein plays a role in neurotransmission by dopamine. Dopamine deficiency is believed to be related to the start and stop of voluntary and involuntary movements
  - In the disease state, α-Synuclein is remodeled into protein aggregates we call plaques, which have been thought to be the cause of disease.
- New research by the Company and it's collaborators demonstrate that plaques **of α-synuclein cannot cause disease unless chemically modified by the Abelson tyrosine kinase, c-Abl**. The Company believes this modified plaque, which is entirely within the affected neurons, is the true causative agent of PD, making c-Abl an important therapeutic target.



α-Synuclein Is normally in a helix-turn-helix configuration[1]



In the disease state, α-Synuclein reorganizes to form fibrous aggregates ("Plaques")[2]

[1]Biochim Biophys Acta. 1818:1013-8 (2012)
[2]Pathogens 7:50 (2018)

Stressors Trigger the Production of α-Synuclein Plaques Which Are Chemically Modified Into a Toxic Form Leading to Neurodegeneration

Stressors[1] Trigger the Production of α-Synuclein

Oxidative / nitrosative stress

Point mutation in one or more proteins causing hyperaggregation (Mutant α-syn, LRRK2, GBA1)

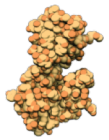
α-synuclein duplication / triplication

α-Synuclein Forms Fibrous Aggregates we call Plaques

At this stage, plaques of misfolded α-synuclein are abnormal, but benign, as they have not been chemically modified to a toxic, pathological state

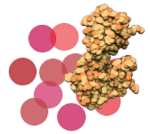
This stage is the target of antibody therapy

CELL ENTRY



C-Abl is a Sentinel which Patrols for Abnormalities Inside a Gut or Brain Neuron

c-Abl's sensor identifies α-synuclein plaques as a threat and activates in response



The Effects and Results of Activated c-Abl Kinase

Sensing α-synuclein plaques, activated c-Abl **Chemically Modifies** α-synuclein at **Tyr[39]** (pY39), converting it into its toxic, pathologic form

C-Abl further chemically modifies Parkin, shutting off a protective survival pathway for the neuron

NEURODEGENERATION

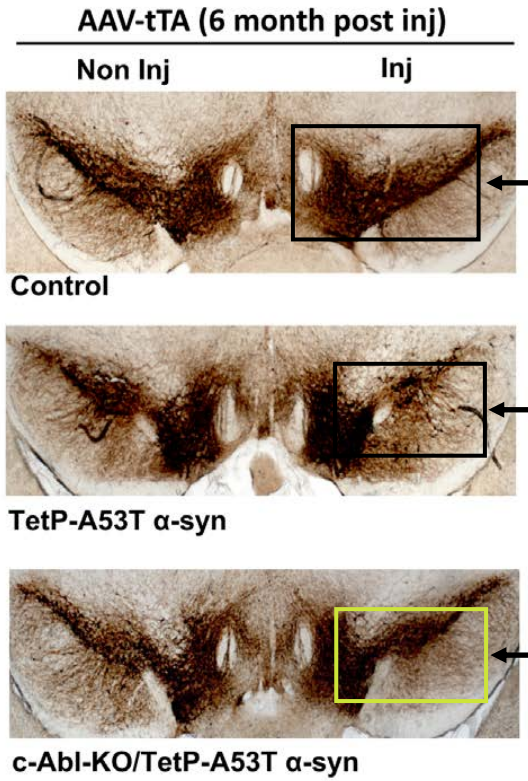
GI Conditions

Parkinson's Disease

[1] *Nat Rev Neurosci*. **2**, 492–501 (2001)
[2] Werner and Olanow (2020), under review

α-Synuclein Plaques Do Not Cause Disease Without c-Abl Modification in Humanized Preclinical Models[1]

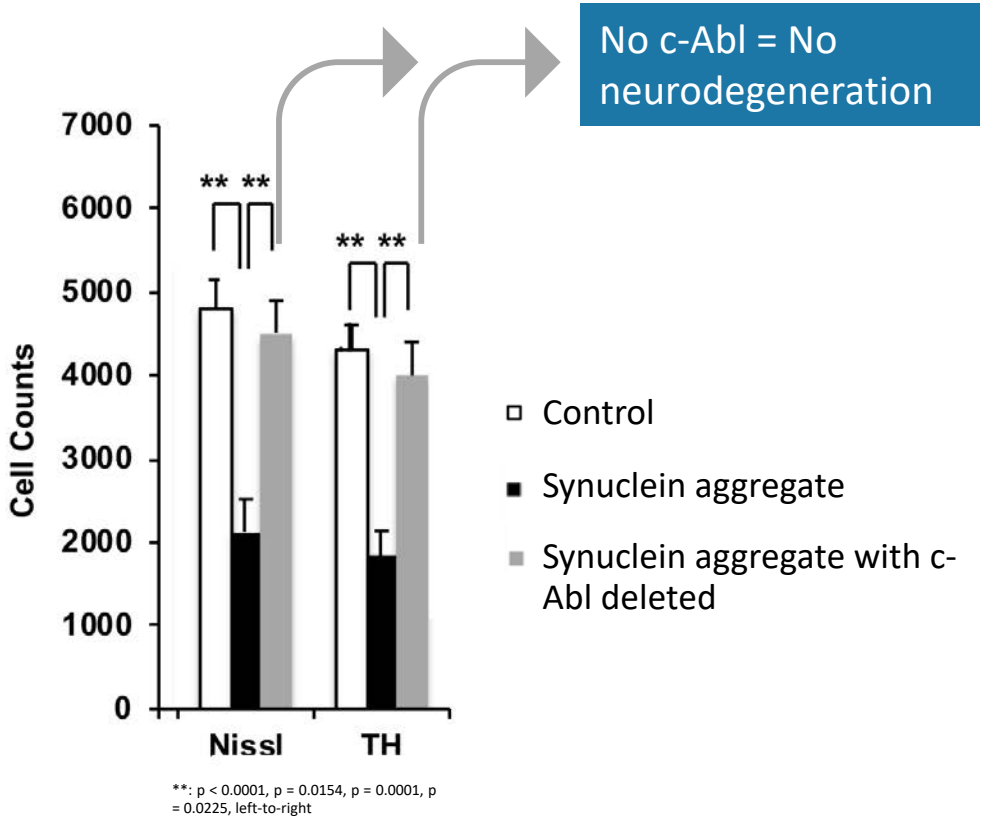
α-Synuclein plaque in the ABSENCE OF c-Abl CAUSES NO NEURODEGENERATION AFTER 6 MONTHS



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



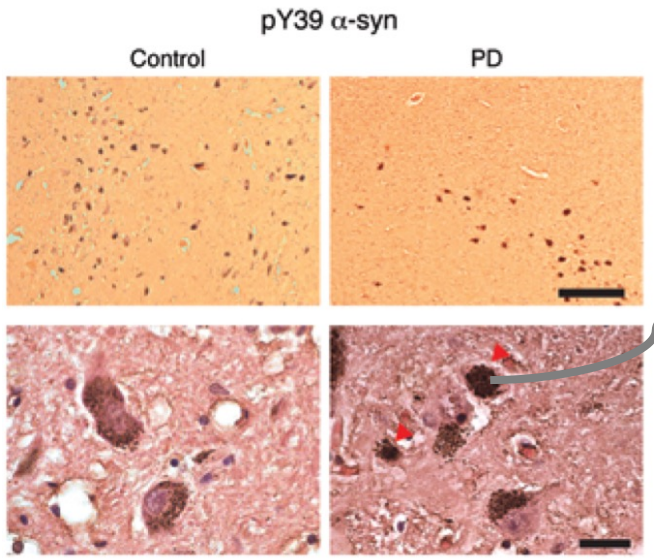
**: p < 0.0001, p = 0.0154, p = 0.0001, p = 0.0225, left-to-right

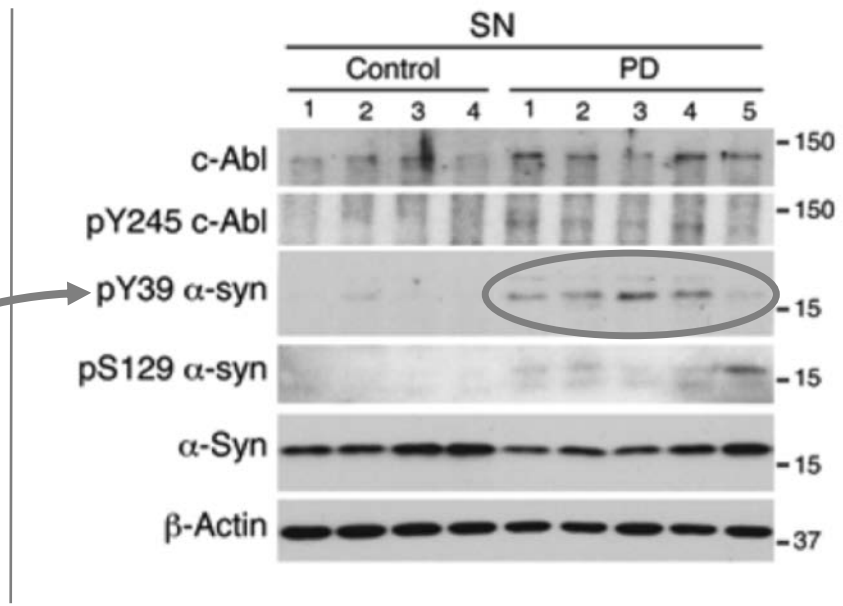
[1]Brain 142:2380ff (2019)

STUDY

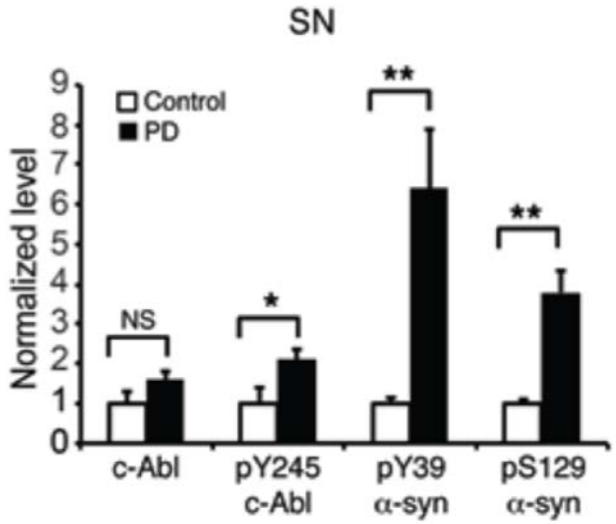
Pathologic, c-Abl-Modified α-Synuclein (pY39) is Present in Parkinson's Patient Brain[1]

Toxic pY39-a-Synuclein Prominent in Lewy Bodies





Toxic pY39-a-Synuclein Abundant In Substantia Nigra (SN) of Patients



[1]J Clin Invest. **126**, 2970-88 (2016)

IkT-148009 c-Abl Inhibition May Drive Functional Reversal

The Gateway that Converts α-Synuclein into ITS TOXIC FORM CAN BE SUPPRESSED BY INHIBITING c-ABL

Stressors[1] Trigger the Production of α-Synuclein

Oxidative / nitrosative stress

Point mutation in one or more proteins causing hyperaggregation (Mutant α-syn, LRRK2, GBA1)

α-synuclein duplication / triplication

α-Synuclein Forms Fibrous Aggregates we call Plaques

At this stage, plaques of misfolded α-synuclein are abnormal, but benign, as they have not been chemically modified to a toxic, pathological state

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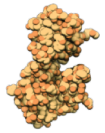


C-Abl is a Sentinel which Patrols for Abnormalities Inside a Gut or Brain Neuron

c-Abl's sensor identifies α-synuclein plaques as a threat and activates in response

IkT Therapeutic Intervention

IkT-148009 Inhibikase Therapeutics

1. INHIBITS c-Abl at this Gateway by blocking pY39 Formation

2. PROMOTES CLEARANCE of pathology through Parkin turnover and ubiquitin/proteasome survival pathway

FUNCTIONAL RECOVERY

GI
Cognitive and Motor

[1]Nat Rev Neurosci. **2**, 492–501 (2001)
[2] Werner and Olanow (2020), under review

Low Toxicity, Brain Penetrant c-Abl Inhibitor in Clinical Development

NOVEL ABL KINASE INHIBITOR	RELATIVE POTENCY
148019	8
148003	12
148027	17
148032	23
148009	**18***
01427	36
Imatinib	**1**

14-Day Toxicology in Rat/Monkey	
Human equivalent dose of 600 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
Sustainable brain concentration	> 1 micromolar

IkT-148009 Inhibikase Therapeutics

- No observed toxicity from off target kinase inhibition
- No CNS toxicity
- No toxicity observed even on 210+ day dosing
  >150 mg/kg/day
- Nearly complete neuroprotection in a-synuclein progressive disease models
- Multi-kilogram process scale development under GMP completed, 6 step synthesis

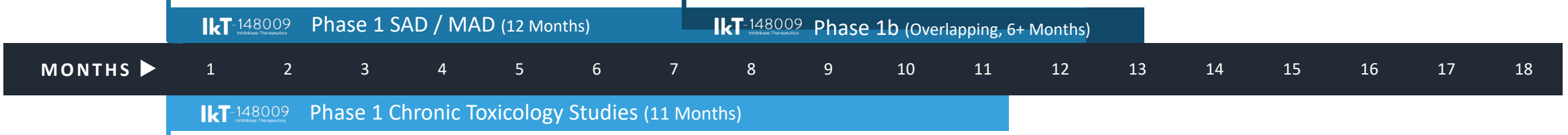
*Compositions of matter **patent protection through 2036**

Phases and Development Intervals

Single Ascending Dose (SAD)
- 8 dosing cohorts (25 – 400 mg, 1x/day)
- 8 patients/cohort, 2:1 randomized
- Sentinel dosing
- Primary objectives safety, tolerability , pharmacokinetics (PK), urine and plasma concentrations

Multiple Ascending Dose (MAD)
- 4 dosing cohorts, doses determined from SAD PK and safety
- 12 patients/dose, 2:1 randomized, 7 day dosing 1x/day
- Primary objectives safety, tolerability, pharmacokinetics (PK), urine, plasma spinal fluid concentrations, trough concentration and Maximum Tolerated Dose
- Protocol to be amended to interleave MAD study with last 4 cohorts of SAD study

Multiple Ascending Dose (MAD)
- Subject to FDA review and agreement
- Enroll cohort of Parkinson's patients (treatment naïve and early stage) with GI complications at middle of MAD period
- Timing coordinated with 3 month toxicology readout
- Primary objectives safety, tolerability, pharmacokinetics
- Secondary objectives functional assessment of motor and cognitive function in brain, motor function in GI, target engagement



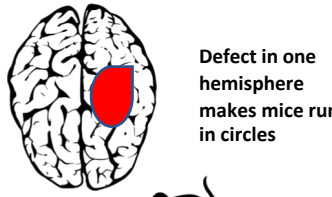
Comparative Toxicology to Imatinib at Toxic Dose

RAT: 3 and 6-month dosing
- 3-month readout extends patient dosing period covered by pivotal toxicology for Phase 1b

MONKEY: 3 and 9-month dosing
- 3-month readout extends patient dosing period covered by pivotal toxicology for Phase 1b

Reversal of Functional Loss Following Oral Therapeutic Administration of IkT-148009 in Mice Humanized for Parkinson's Disease in Brain

Baseline Neuromuscular Coordination Behavioral Marker



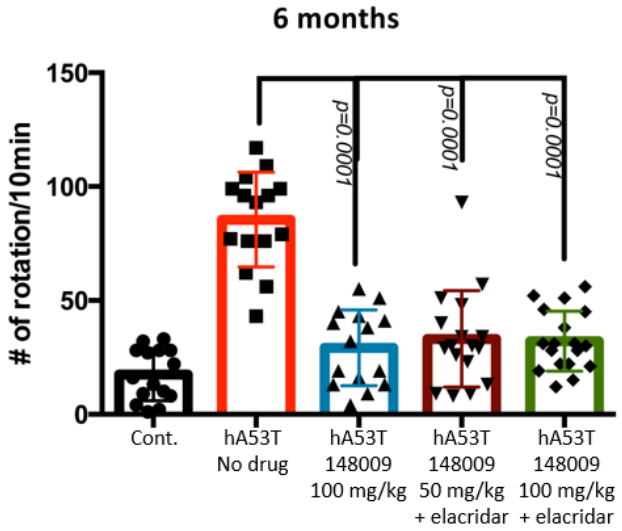
Defect in one hemisphere makes mice run in circles

Healthy Mice Run In Circles at the Average Rate of 25 Turns : 10 Min

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

Toxicity renders the mice trembling and only able to run in circles

Treatment Started 6 Weeks After Onset of Conditions



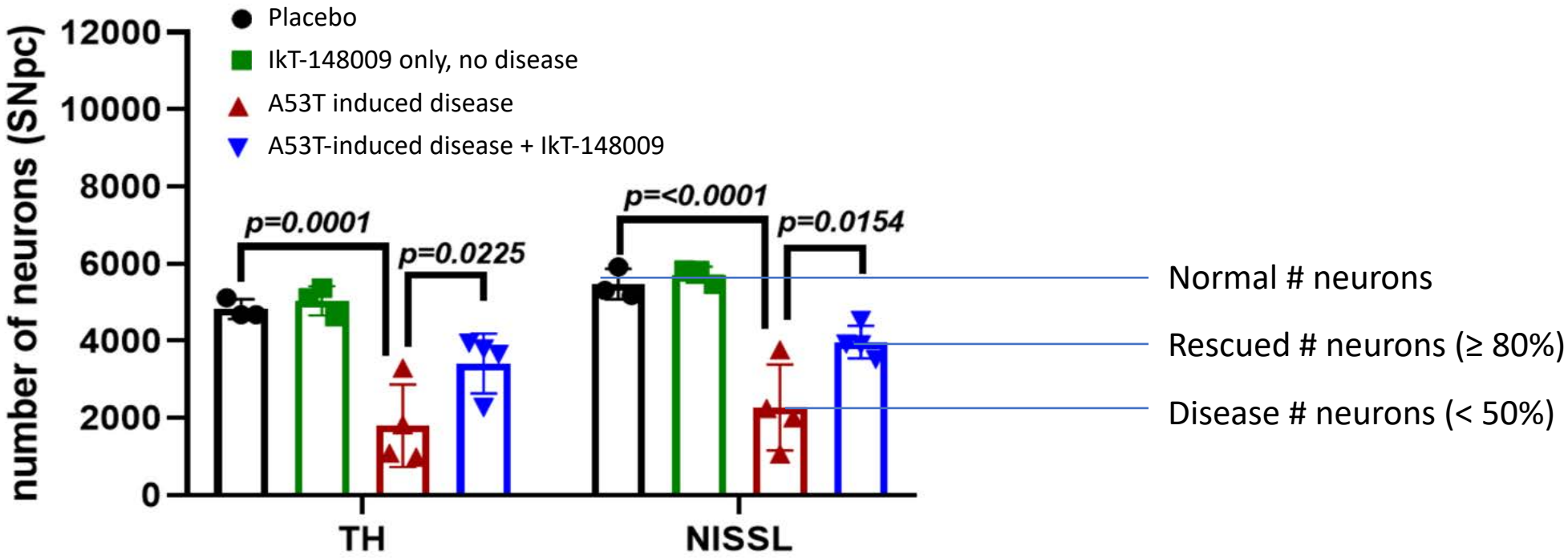
Near Normal Behavior Returned Following Treatment

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

NEARLY COMPLETE RESCUE

Therapeutic Treatment With Oral IkT-148009 Preserves Neural Anatomy

IkT-148009 restores function to neurons in the brain



Gastrointestinal Tract Offers Unique Opportunity to Accelerate Development

Parkinson's May Begin in the Gut

Easy access

Can demonstrate disease benefit with quantitative endpoints

Biopsy and Biomarkers

Large effect size

GI disorders resulting from kinase modification of α-synuclein:

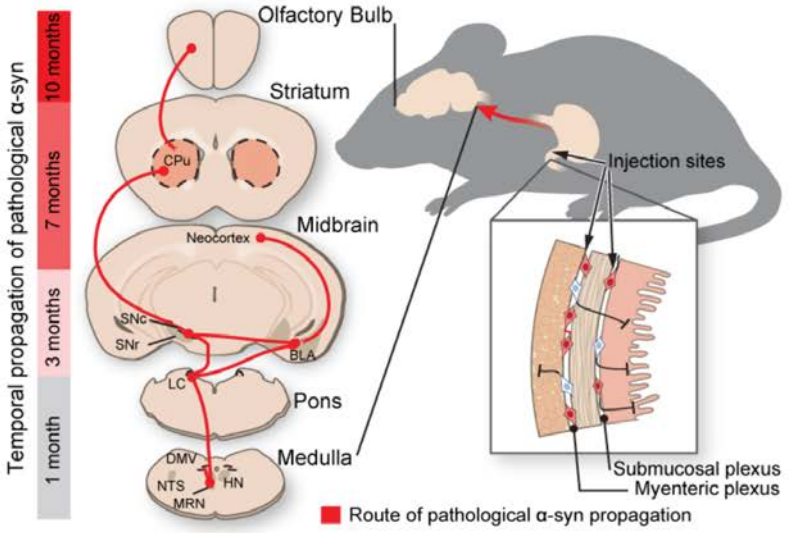
Dysphagia

Unresolvable constipation

Gastroesophageal reflux

Gastroparesis

The Gut-Brain Connection Enables Innovation in Trial Design

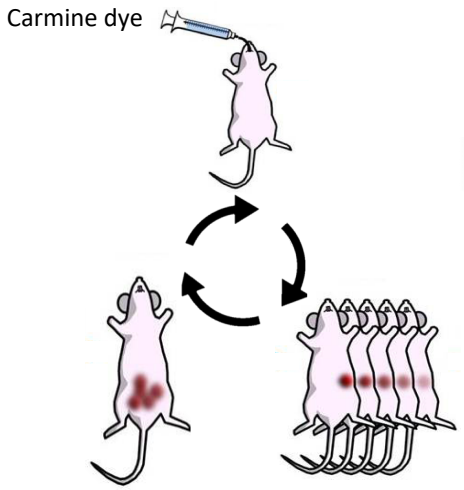


Introduction of dysfunctional synuclein in the **gut leads to progressive disease that mirrors the human disease course in the brain**

Therapeutic Administration of Oral IkT-148009 Reverses Functional Loss in the GI Tract in Mice Humanized for Parkinson's Disease in the GI

METHOD OF MEASUREMENT

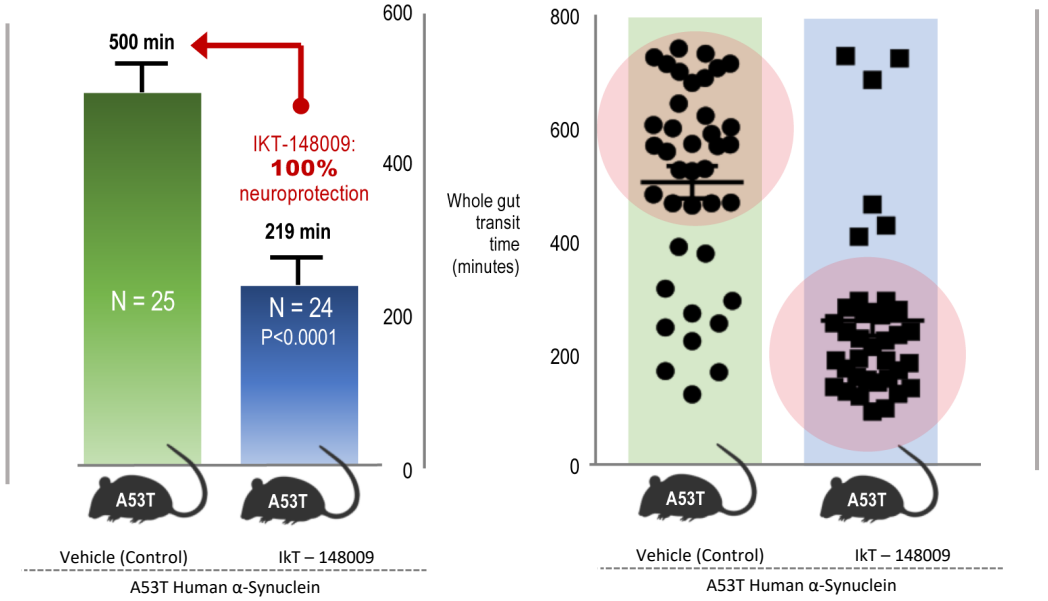
α-Synuclein Aggregates Introduced Into Gut Slows Gut Transit Time



POST TREATMENT RESULTS

At 3 months defecation slows to 500 min (3x normal); At 6 months defecation slows to 800 min (5x normal); Normal = 165 min

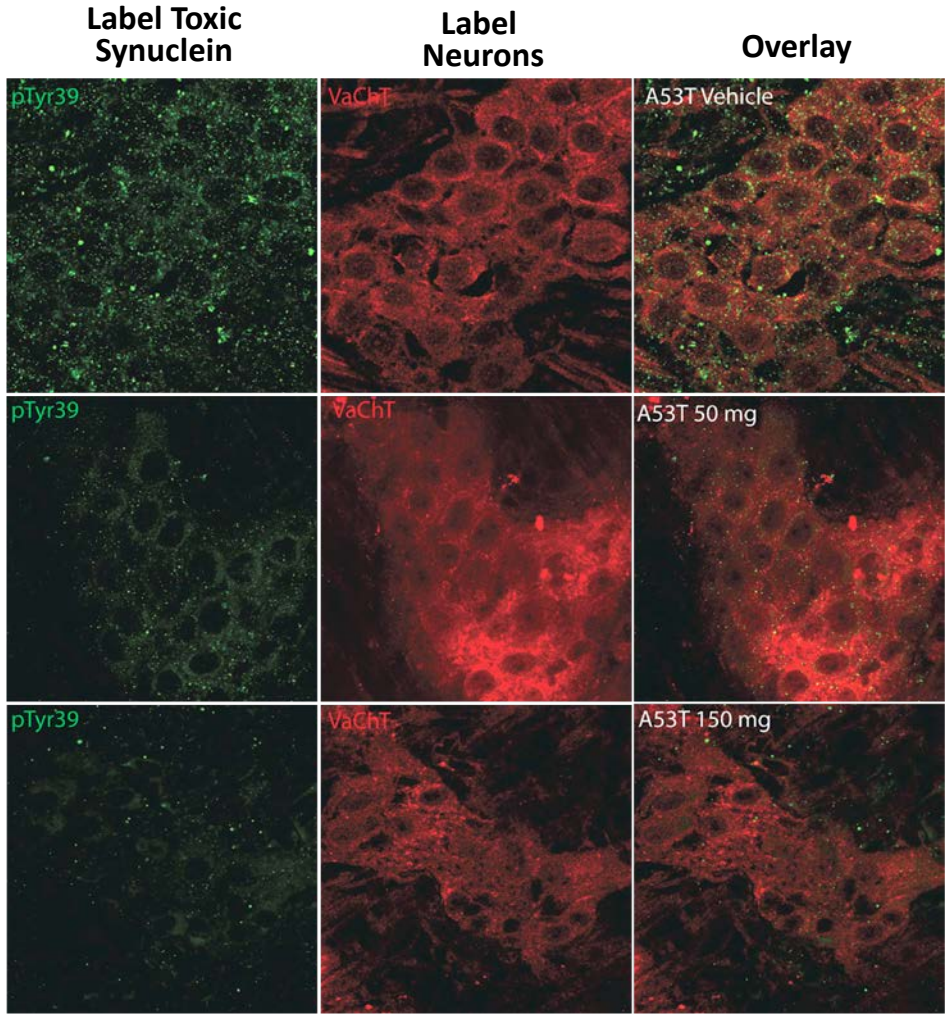
Neuron count read out by Whole Gut Transit Time - The time it takes for a mouse to defecate



NOTES

1. If it weren't for 6 outliers in 96 measures, average would be 170 min, essentially 100% recovery.
2. We find the neuroanatomy is nearly completely restored by measuring the number and distribution of neural ganglia in the tissue.

Functional Reversal of GI Defect Occurs Concomitantly with Clearance of Toxic α-Synuclein Following Oral IkT-148009 Treatment



IkT-148009 nearly clears pathological α-synuclein (green dots have been cleared)

In Pre-Clinical Models

- IKT-148009 drives functional recovery inside and outside of the brain
- IKT-148009 drives clearance of the toxic form of α-synuclein
- IKT-148009 evaluation in the GI offers unique paths for clinical success

Targeting c-Abl Can Block the Path to Neurodegeneration

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 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.

Clinical Development Team Collaboration with Clintrex Research Corporation

Terence Kelly, PhD, Contractor, Head of MedChem & Drug Discovery
Dr. Kelly is a 30-year pharmaceutical industry veteran and has served as a member of the board of directors of Cardax since June 2014. He previously held several positions at CoMentis, including President and CEO. From July 2002 to December 2009, he served as Vice President of Medicinal Chemistry at Boehringer Ingelheim Pharmaceuticals. Dr. Kelly developed processes for the RAMP drug discovery program with Dr. Werner.

Roger Rush, PhD, Contractor, Head of Preclinical Research
Dr. Rush is an experienced veteran of the pharmaceutical industry with more than 30 years of experience. He has contributed to more than 20 IND, CTA, and product license submissions and approved drugs, including nicardipine (Cardene), ranolazine (Ranexa), Foscan, and zileuton (Zyflo CR). Previously, he was Vice President, Preclinical Development for Idenix Pharmaceuticals, Inc., a wholly owned subsidiary of Merck & Company, Inc., where he managed the DMPK, toxicology, and discovery research that led to the identification of lead molecules to treat Hepatitis C virus.

Board of Directors

Mr. Dennis Berman has been a co-founder, board member, and/or seed investor in many private biotechnology and technology companies, five of which have gone public. He 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. Other public companies for which Mr. Berman has served as a seed investor, co-founder, and/or board member include Intervu (one of the first software-as-a-service companies), which was acquired by Akamai; Kintera, Inc. (an online fundraising pioneer), which was acquired by Blackbaud; Gensia (focused on purine/pyrimidine metabolism compounds), which was acquired by Teva; and Viagene (the first U.S. gene therapy company that utilized a non-replicating retrovirus), which was acquired by Chiron/Novartis. Mr. Berman also was a seed investor in Calabrian (a water treatment company), which was acquired by SK Capital.

Dr. Paul Grint, MD has more than two decades of 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. He has most recently served on the Board of Cardea Bio since April, 2020, on the Boards of Directors of Amplyx Pharmaceuticals since 2016 and Synedgen since 2014 and was CEO and member of the Board of Directors of AmpliPhi Biosciences from 2015 through 2019. Dr. Grint has also served in senior management roles at Cerexa, Forest Laboratories, Kalypsys, Pfizer, IDEC Pharmaceuticals, and Schering-Plough Corporation. He is currently a board member at Amplyx Pharmaceuticals and Synedgen. He is a Fellow of the Royal College of Pathologists, a member of numerous professional and medical societies, and holds a bachelor's degree from St. Mary's Hospital College, University of London and a medical degree from St. Bartholomew's Hospital College, University of London.

Dr. Roy Freeman, MD is 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 in Boston, Massachusetts. Dr. Freeman is 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. Dr. Freeman is 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. He is a 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 recently retired from a 25-year career with Eli Lilly and Company, lastly serving as Chief Procurement Officer and Leader, Global Head of Shared Services from 2012 to 2017. Prior to that, she advanced through a number of executive management positions 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. O'Farrell was an accountant with Boise Cascade Office Products and served as an auditor at Whipple & Company and Price Waterhouse. Ms. O'Farrell currently serves on the board of PDL BioPharma, where she is a member of the Audit Committee and Compensation Committee and Geron Corporation where she is a member of the Audit Committee. Ms. O'Farrell holds a BS in accounting with honors and an MBA in management information systems, both 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
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Dr. Valina Dawson, PhD
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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

Upcoming Milestones

OBJECTIVES

Pre-clinical Activities

Oncology Prodrug, IkT-001Pro:

- Commercial product GMP mfg.
- IND filing

Clinical development for IkT-148009

- Elderly healthy volunteer Phase 1
- Comparative chronic toxicology rat and monkey

Team Build-out, Medicinal Chemistry, Pre-clinical Research, G&A

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