
**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): March 22, 2023

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

N/A
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (*see* General Instruction A.2. below):

- Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
- Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
- Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
- Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, \$0.001 par value	IKT	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act.

Item 7.01 Regulation FD Disclosure.

On March 22, 2023, Inhibikase Therapeutics, Inc. (the “Company”) made presentations to investors at the Company’s R&D Day. A copy of the 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	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: March 22, 2023


INHIBIKASE THERAPEUTICS, INC.

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



Inhibikase
Therapeutics

March 22, 2023 | Inhibikase Neurodegeneration R&D Day



Transforming fundamental and clinical research
in neurodegenerative disease

[Inhibikase.com](https://www.inhibikase.com)

Nasdaq : **IKT**

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.



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Agenda for the 2023 Neurodegeneration R&D Day

- Why is c-Abl an important target in PD?
- How was IKT-148009 discovered?
- What is therapeutically possible with IKT-148009?
- Clinical Development: Phase 2 and beyond in PD
- Opening of MSA clinical development

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 Naive	██████████	██████████	██████████				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 naive 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 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.

Chronic Disease for a Long Time
1/3 of a Patient's Lifespan = 25 years

60,000

New Cases / Year

38,000

Deaths / Year

930,000 - 1,200,000
U.S. Patients¹

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

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.

DelveInsight, 2019

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

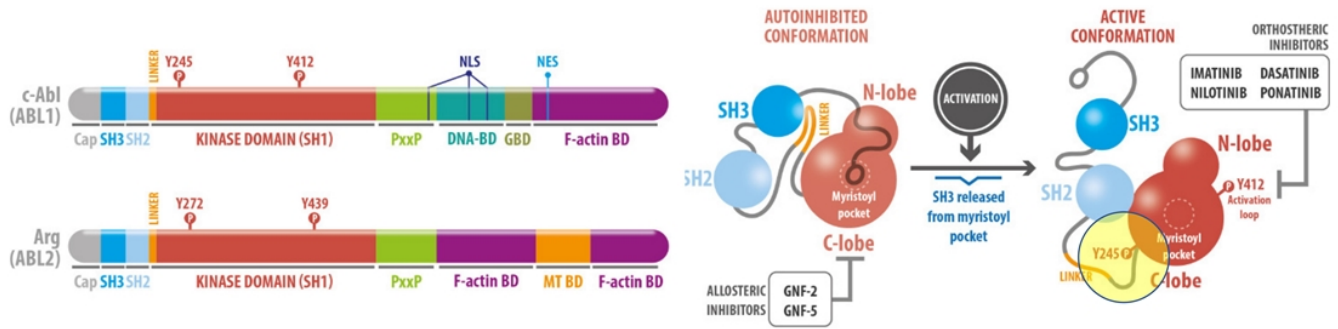


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Discovery of c-Abl's Role in PD

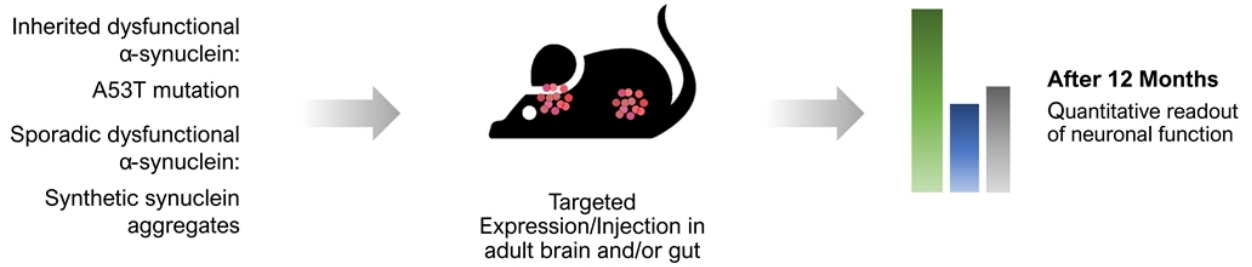
c-Abl is an essential regulator of growth and differentiation¹



➤ Monitoring c-Abl activation by observation of Tyr²⁴⁵ phosphorylation (Y245p)

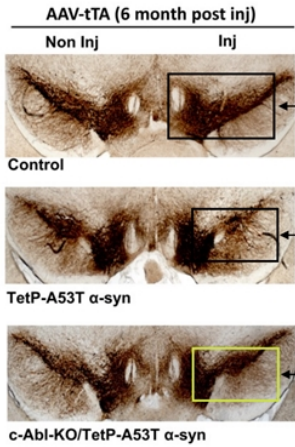
¹Neural Regen Res. 2022 Feb; 18(2): 237–243

Slowly progressive, α -synuclein dependent models were essential to thinking differently about PD^{1,2}



¹J Clin Invest. 126, 2970-88 (2016); ²Hum Mol Genet 19, 1633-50 (2010)

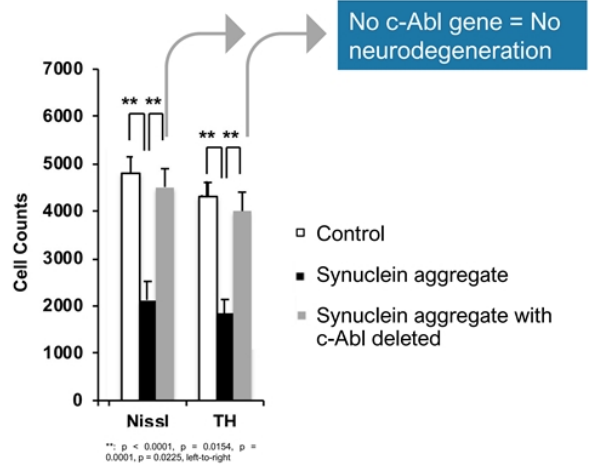
α-synuclein aggregates in the ABSENCE OF c-Abl DO NOT LEAD TO NEURODEGENERATION



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

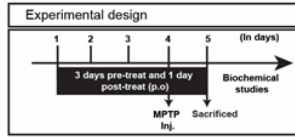
Once turned on, A53T induces 50% neurodegeneration in 6 months. NOTE loss is visible in the dark patches of stained neurons on the right half. The left half was an internal control and is unaffected.

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 aggregates of α-syn are present in the right region of the brain, they don't cause disease until c-Abl acts on them



¹Brain 142:2380ff (2019)

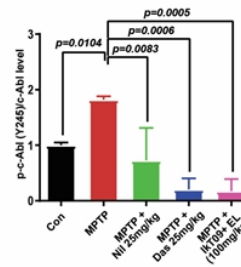
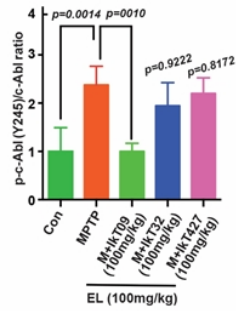
Use acute neurotoxin MPTP as a functional screen for drug selection¹



Dopamine neurons are selective

Commercial inhibitors show varied activities

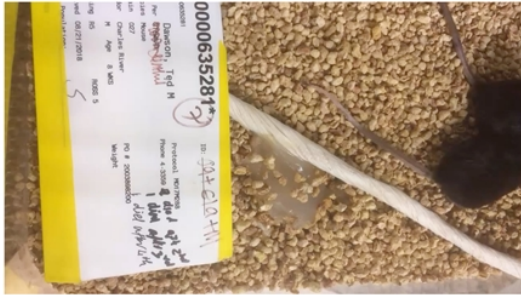
IC₅₀
 148009 (09): 33 nM
 148032 (32): 53 nM
 1427 (427) : 30 nM



¹Karuppagounder et al., Sci. Transl Med 1/18/2023

Pre-treatment with IKT-148009 blocks neurodegeneration in the MPTP acute toxicity model¹

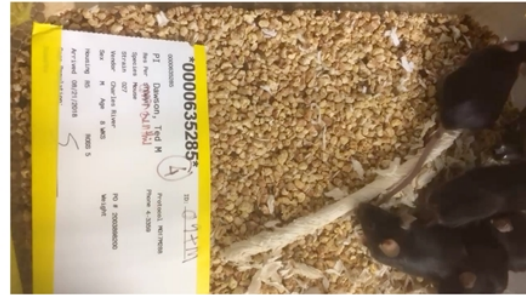
No IKT-148009



Mice treated with an acute neurotoxin MPTP results in substantial loss of dopamine neurons

- No stimulus response, hunched
- Little movement
- Anti-social

Pre-Treatment with IKT-148009



Mice pre-treated with IKT-148009 are protected from neurodegeneration

- Normal response to stimuli
- Run around the cage
- Socially engaged

¹Karuppagounder et al., Sci. Transl Med 1/18/2023

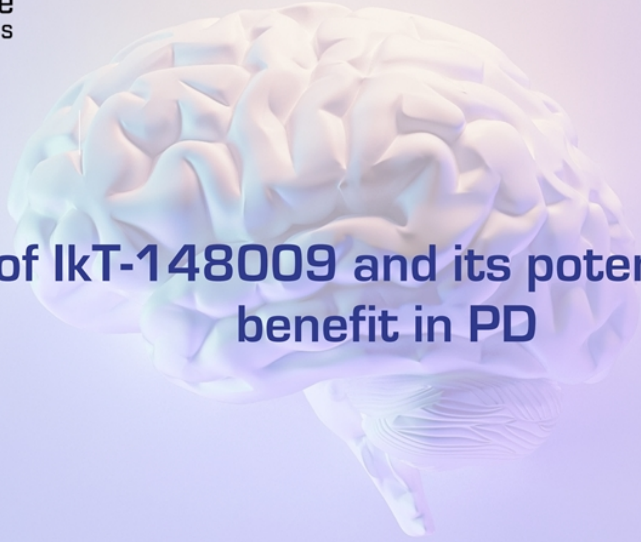
What we concluded early on about c-Abl inhibition as a potential disease-modifying therapy for PD?¹

- **C-Abl is an essential determinant for PD initiation and progression: *If you delete the gene, you cannot induce neurodegeneration in animals***
- **C-Abl inhibition can block induction of neurodegeneration by the acute neurotoxin MPTP**

¹Werner and Olanow, Mov. Disord. 2022 DOI: 10.1002/mds.28858

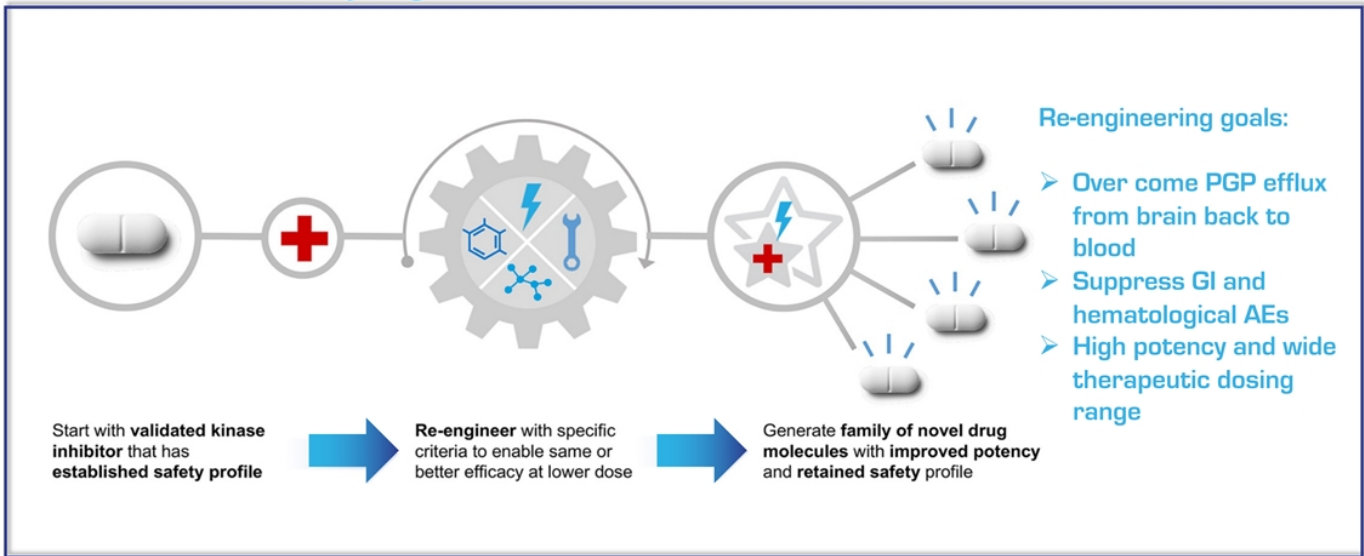


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Discovery of Ikt-148009 and its potential therapeutic benefit in PD

RAMP as a discovery engine for PD¹



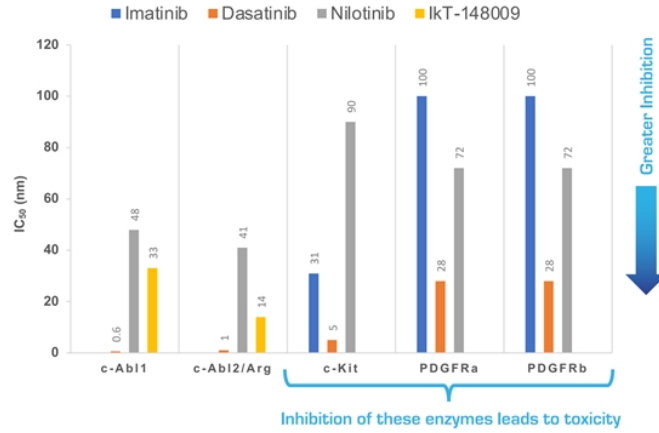
¹Karuppagounder et al., Sci Transl. Med, Jan 18, 2023

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

Ikt-148009

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- 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 and 39 week toxicology data shows Ikt-148009 has a more favorable toxicity profile as dosing is extended

¹Karuppagounder et al., Sci. Transl Med 1/18/2023

Therapeutic dosing in a slowly progressive model of inherited disease leads to functional recovery¹

Baseline Neuromuscular Coordination Behavioral Marker



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 Nearly Eliminates Dopamine Levels Rendering the Mice Stationery and Trembling



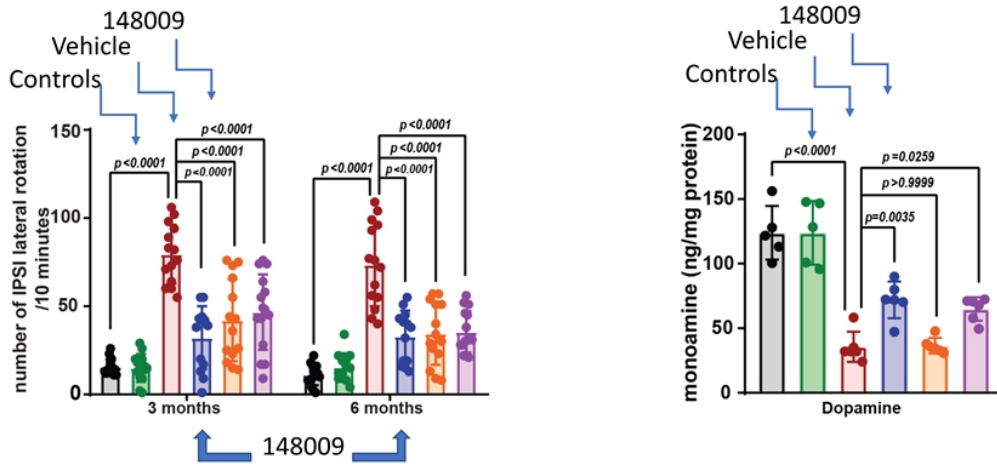
Near Normal Behavior Returned Following Treatment



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

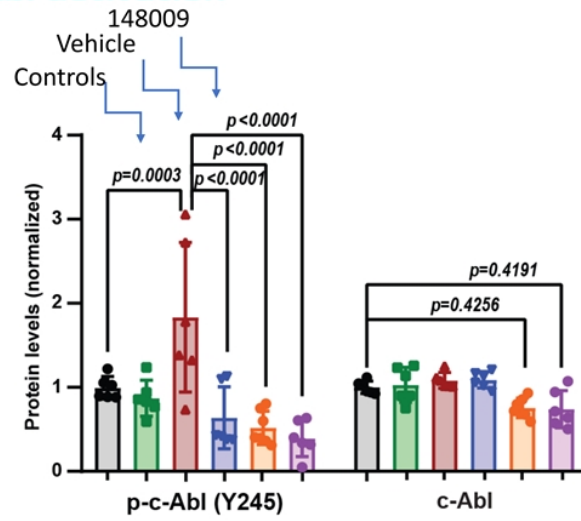
¹Karuppagounder et al., Sci Transl Med Jan 18 2023

Therapeutic dosing in a slowly progressive model of inherited disease leads to functional recovery¹



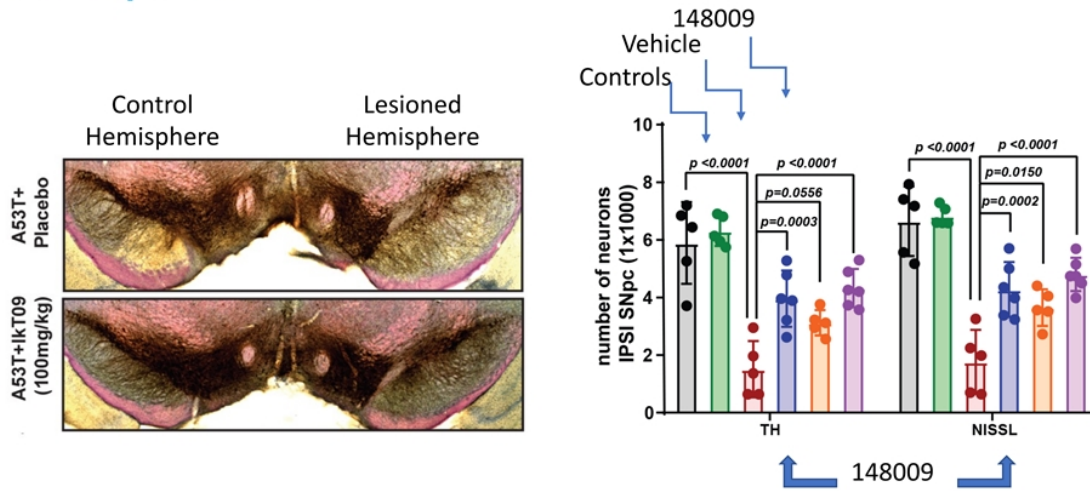
¹Karuppagounder et al., Sci Transl Med Jan 18 2023

Therapeutic dosing in a slowly progressive model of inherited PD suppresses c-Abl activation¹



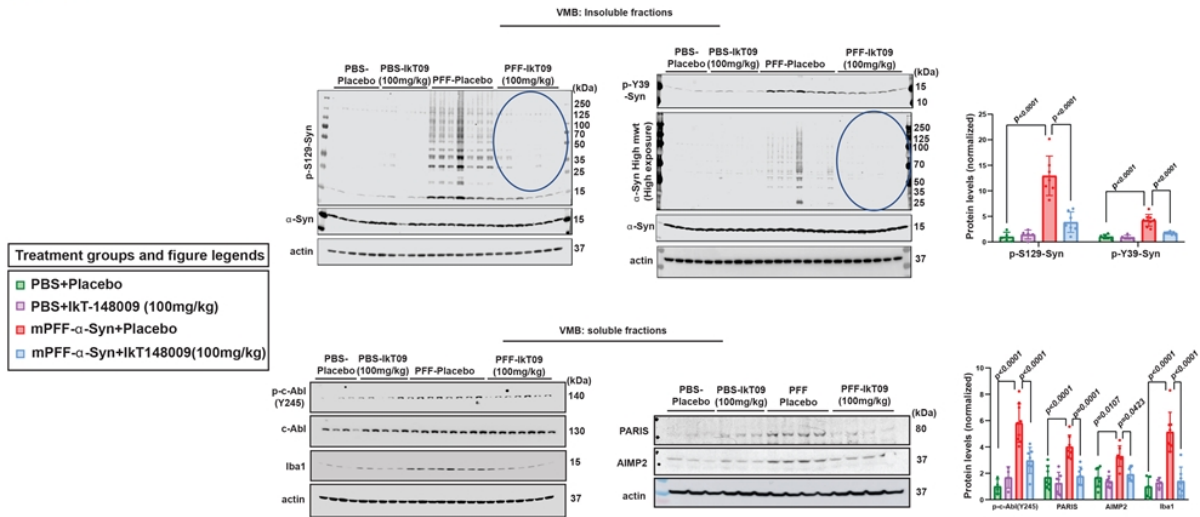
¹Karuppagounder et al., Sci Transl Med Jan 18 2023

Therapeutic dosing in a slowly progressive model of inherited PD is neuroprotective¹



¹Karuppagounder et al., Sci Transl Med Jan 18 2023

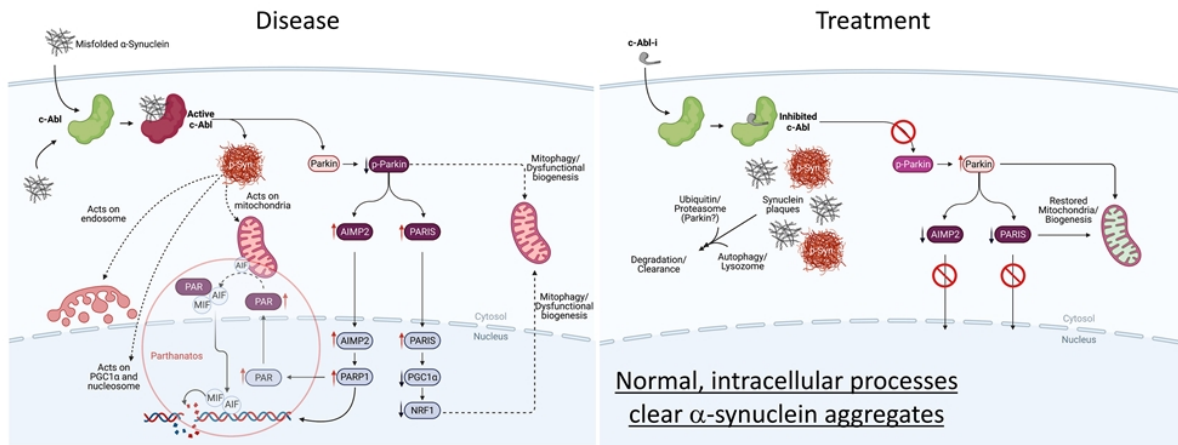
Therapeutic dosing leads to reduction/clearance of α -synuclein aggregates at 6 months ¹



Modeling PD followed by therapeutic dosing revealed:

- **C-Abl inhibition is neuroprotective in animal models of disease**
- **C-Abl inhibition blocks downstream effector pathways of neurodegeneration in animal models of disease**
- **C-Abl inhibition reduces α -synuclein pathology in the affected neurons in animal models of disease**

Model of the biochemistry of disease initiation and its treatment response



¹Werner and Olanow 37, 6-15 (2022)



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Clinical Development of IKT-148009 for Treatment of Parkinson's disease

Demographics and Adverse Events Across 113 Healthy Subjects and Patients

Category	Demographic	Healthy Subjects Value [% of Total N=88]	Parkinson Patient Value [% of Total, N=25]
Gender	Female	34 (38.6)	9 (36)
	Male	54 (61.4)	15 (60)
Age	Average (SD)	57.9 (5.72)	61.9
	Median	58.0	63
	Range	45, 69	48, 71
Ethnicity	Hispanic or Latino	13 (14.8)	4 (16)
	Not Hispanic or Latino	75 (85.2)	20 (80)
Race	Black or African American	54 (61.4)	3 (12)
	White	33 (37.5)	20 (80)
	Other	1 (1.1)	0 (0)
Adverse events		6, none clinically significant, only 3 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 GI
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 ¹		Mild
Gastrointestinal					
	325 mg	Single Dose	2 Diarrhea		Mild
	100 mg	7-day, 1x/day		1 Constipation ²	Mild
	100 mg	4 wk, 1x/day		1 Elevated Amylase/Lipase ³	Moderate
	Active, 50 mg	4 wk		1 Gastric pain ⁴	Mild
	Active, 50 mg	4 wk		1 Nausea ⁴	Mild
Dermatological					
	50 mg	7-day, 1x/day		1 Dermatitis	Mild

¹Appeared 2 weeks post-dose, no clinical basis found even after following by 3-day Holter monitoring;

²Appeared one day after last dosing day; ³Amylase and Lipase abnormalities were asymptomatic. Patient reported regular consumption of alcohol prior to the baseline visit and while enrolled in the trial;

⁴Single occurrence on first dose

CLINICAL PHASE 2: the '201' Trial 3 doses



Double-blinded: 3 Months Dosing Across 3 Doses				12 month extension study			
Primary:	Safety/Tolerability			Primary:	Safety/Tolerability		
Secondary:	MDS-UPDRS II+III			Secondary:	MDS-UPDRS II+III		
	PGI-S			(measure	PGI-S		
	CGI-S			every 3	CGI-S		
	MDS-UPDRS II			months)	MDS-UPDRS II		
	MDS-UPDRS III				MDS-UPDRS III		
	MDS-UPDRS I				MDS-UPDRS I		
	Non-motor Symptom Scale				Non-motor Symptom Scale		
	CSBM				CSBM		
	Epworth Sleepiness Scale				Epworth Sleepiness Scale		
	GI Measures				GI Measures		

CLINICAL PHASE 2: the '201' Trial 3 doses



Double-blinded: 3 Months Dosing Across 3 Doses	12 month extension study
<p>Exploratory: Skin/possibly GI reduction α-synuclein aggregates</p>	<p>Exploratory: Skin/possibly GI reduction α-synuclein aggregates</p> <p>Time to initiation of PD symptomatic medication</p> <p>Time before initiation of PD symptomatic medication</p>

CLINICAL PHASE 2: the '201' Trial 3 doses



Adding back 200 mg dose post-Clinical Hold:

- Required safety/PK profile study completed.
- Data to be submitted and dose added back early in 201 trial (after 5 patients each at 50 mg, 100 mg, placebo cohorts)

Vision Monitoring:

- Observed increase in minimal/mild changes in retina between 3 and 6 months in rat
- Implemented a standard program of vision monitoring until known whether this will occur in humans
- Program used for approved products: brigatinib, infigratinib, pemibratinib, futibatinib, trametinib, binimetinib, selumetinib, cobimetinib
- No vision pathology observed in 11 patients at all 3 doses for up to 11 weeks.



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Clinical Development of Ikt-148009 for Treatment of Multiple System Atrophy (MSA)

What distinguishes MSA from PD?

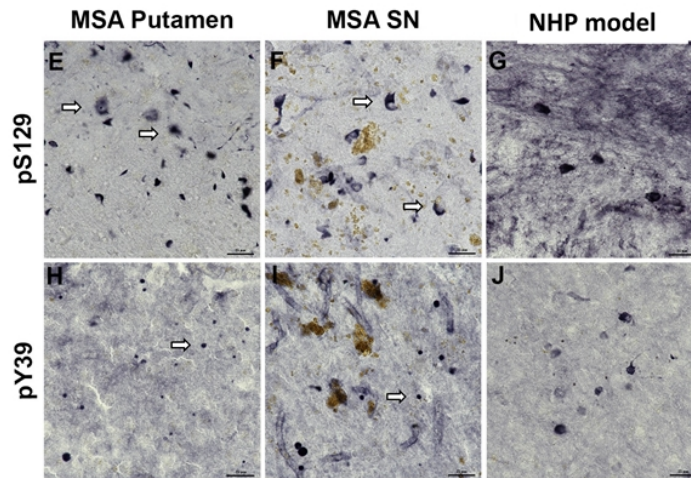
PD

- Slowly progressive, 25 years to death
- Highly variable interpatient variation in rate of progression and disease manifestation
- Multiple treatments to address symptoms
- Diagnosis easier
- Large disease ($\approx 1\text{M}$ US cases)
- Synuclein aggregates found inside affected neurons

MSA

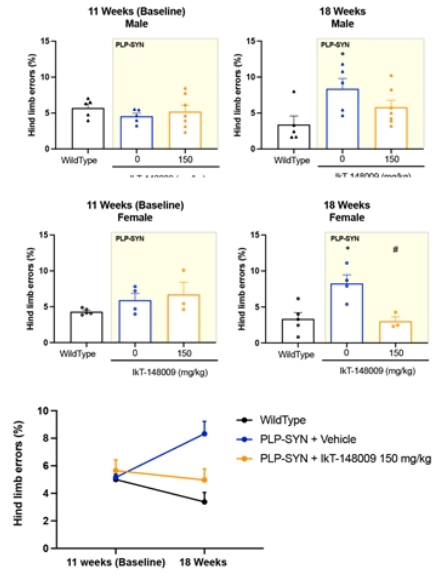
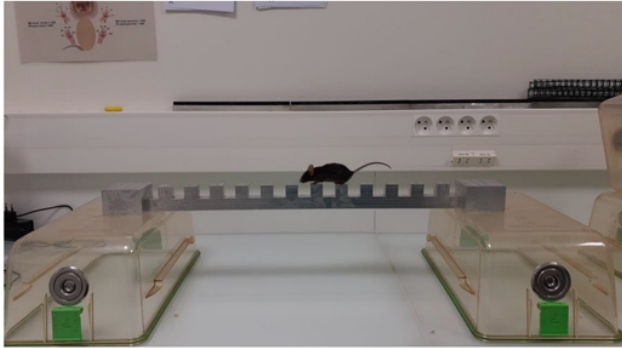
- Rapid progression, 8 years to death
- Similarity in disease manifestation and rate of progression
- No treatments that are very helpful
- Difficult to diagnose early
- Orphan disease ($\approx 20\text{K}$ US cases)
- Synuclein aggregates found in glial cells, not neurons

C-Abl activation occurs in glial cells in MSA and in models¹



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

IkT-148009 inhibition protects against loss of function

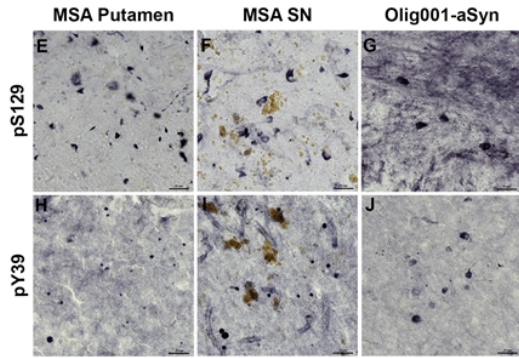


C-Abl Activation

MSA¹

AD

ALS



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.

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

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

CLINICAL PHASE 2: the '202' Trial 2 doses



Double-blinded: 6 Months Dosing Across 2 Doses																		
Primary:	Safety/Tolerability											Exploratory:	α -synuclein aggregate level in skin/CSF					
Secondary:	Modified Total UMSARS Activities of daily life PGI-S CGI-S MSA QOL scores Orthostatic hypotension symptom score Neurofilament light chain blood/CSF Progression of cerebellar atrophy by MRI																	

2023 Guidance in Neurodegeneration

PD

- Fully enroll the 201 trial
- Initiate long-term extension study
- Prepare for Phase 3 program
- Declare a commercial formulation: 2 tablet forms to be tested
- Analyze food effect on Ikt-148009

MSA

- Complete prophylactic and therapeutic animal model studies
- Initiate MSA Phase 2 in US and EU with positive model outcome and appropriate capital



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Selected Stock and Financial Data

Selected Financial and Stock Data

Capitalization Table	January 30, 2023
Common Shares Outstanding	28,027,840
Options (WAEP: \$2.41)	4,001,208
Warrants (WAEP: \$0.77)	22,423,915
Fully Diluted Shares Outstanding	54,452,963

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



Balance Sheet	September 30, 2022 (last reporting period)
Current Assets:	
Cash, Cash Equivalents, Marketable Securities	\$26,534,208
Grants Receivable	\$13,842
Prepaid research and development	\$932,419
Prepaid expenses and other current assets	\$505,924
Total Current Assets	\$27,986,393
Total Current Liabilities	\$3,212,997
Working Capital	\$24,773,396
Active grant funding available in accounts held by the U.S. treasury	\$300,386
Total Working Capital + Available Grant Funds	\$25,073,782



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