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 18, 2022

INHIBIKASE THERAPEUTICS, INC.
(Exact Name of Registrant as Specified in its Charter)

Delaware	**001-39676**	**26-3407249**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(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 March 18, 2022, Inhibikase Therapeutics, Inc. (the "Company"), the Company presented an update on the results of its Phase 1 and Phase 1b clinical studies at the AD/PD Conference in Barcelona, Spain. A copy of the Company's AD/PD Conference 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.

Number	Description
99.1	AD/PD Conference 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 24, 2022

INHIBIKASE THERAPEUTICS, INC.

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

Exhibit 99.1

Inhibikase Therapeutics

Nasdaq | IKT

Parkinson's Disease Modification Through Abl-kinase Inhibition

AD-PD 2022, March 18 2022

Disclosures

Dr. Milton Werner is the Founder, Chief Executive and largest shareholder of Inhibikase Therapeutics, Inc.

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 registration statement on Form S-1, as amended (File No. 333-240036), including under the caption "Risk Factors.

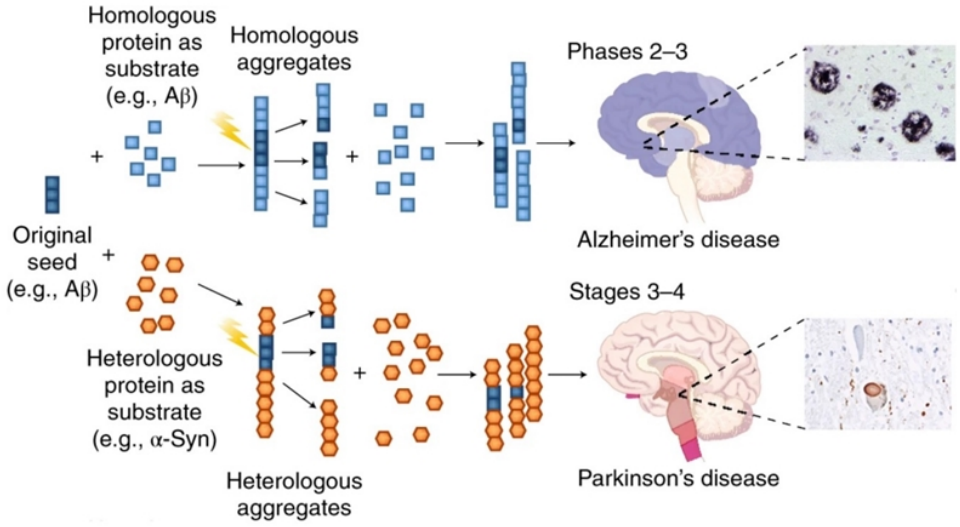
We do not intend our use or display of other entities’ names, trade names, trademarks or service marks to imply a relationship with, or endorsement or sponsorship of us by, any other entity.

TREATING INSIDE AND OUTSIDE OF THE BRAIN

Simultaneous Evaluation of Brain and GI Function Identifies a Potential Disease-Modifying Treatment

- PD is more than just a disease of the brain,
- GI manifestation in many patients occurs at an early stage, indicating 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 disease-modifying therapeutics.
- Overview
  - Review of the role of the Abelson Tyrosine Kinase, or c-Abl, in disease
  - Demonstration of the treatment potential of c-Abl inhibitors
  - Identification of IkT-148009 as a potential disease modifying strategy
  - Clinical safety, tolerability and measures of efficacy
  - Development Timeline

Causation in Parkinson's and Alzheimer's is closely related[1]

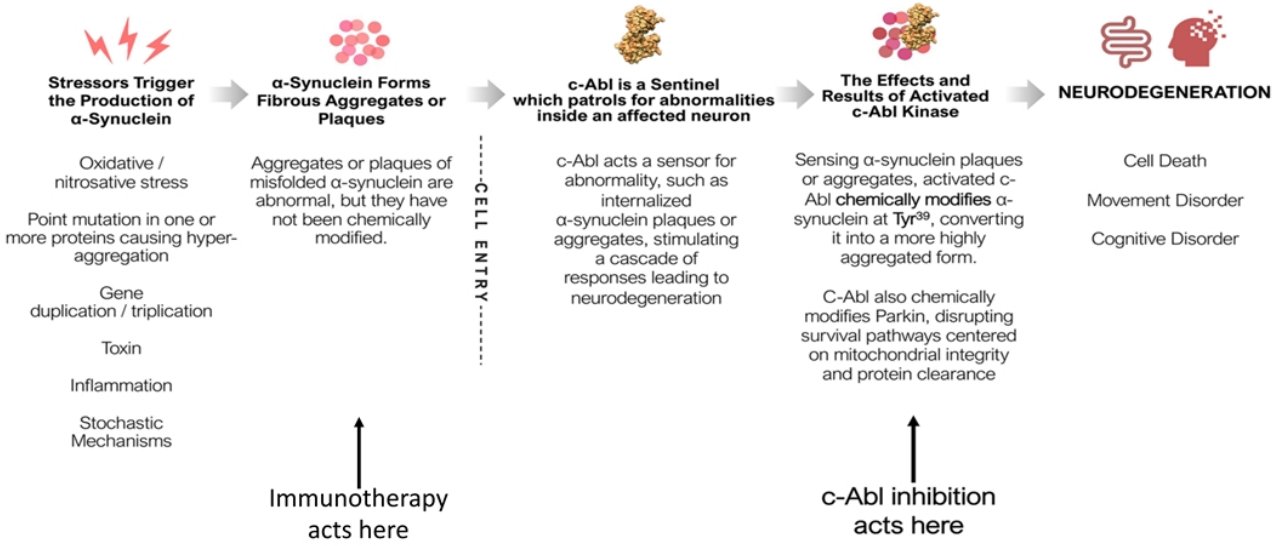


What role does the misfolded protein play?

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

THE PATH TO NEURODEGENERATION

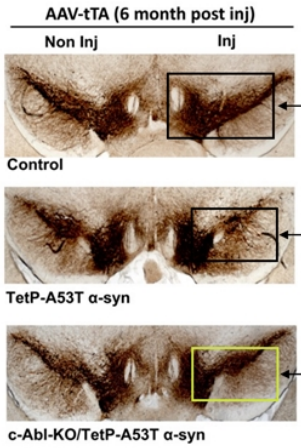
Stressors Trigger the Production of Misfolded α-Synuclein Which Activates c-Abl to Drive Neurodegeneration[1]



[1]Werner and Olanow, Mov. Disorders 2022 Jan;37(1):6-15.

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

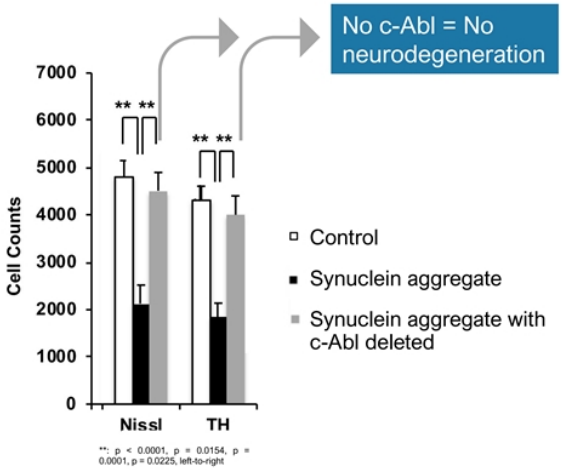
α-Synuclein plaque in the ABSENCE OF c-Abl CAUSES NO NEURODEGENERATION AFTER 6 MONTHS



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

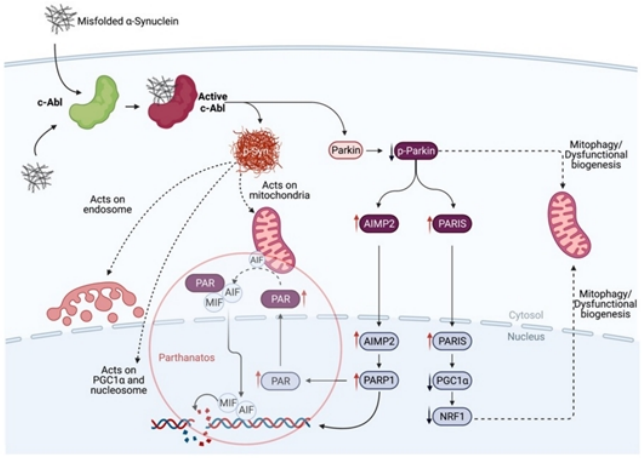
Once turned on, A53T induces 50% neurodegeneration in 6 months. NOTE loss is visible in the dark patches of stained neurons on the right half. The left half was an internal control and is unaffected.

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

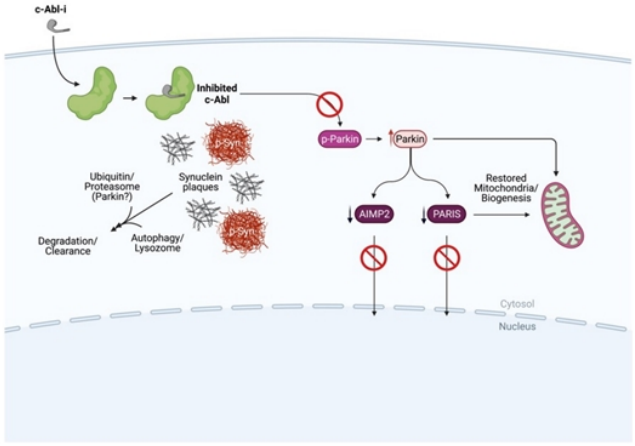


[1]Brain 142:2380ff (2019)

Biochemistry of Parkinson's Disease Initiation and Progression[1]



Disease process



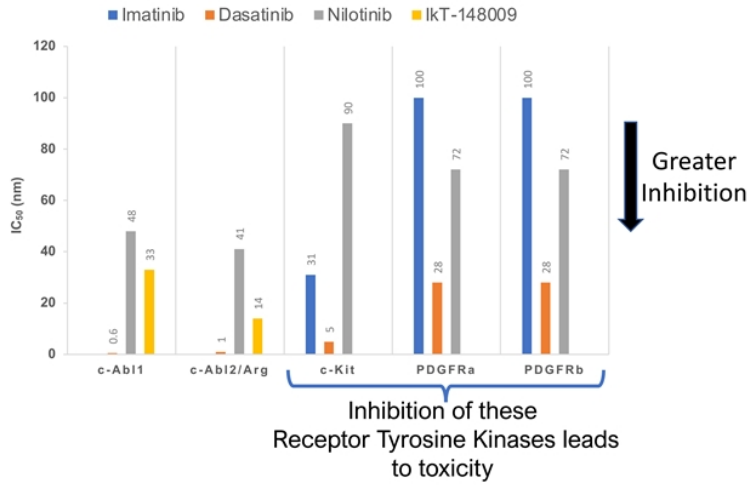
Treatment effect

[1]Werner and Olanow, Mov. Disorders 2022 Jan;37(1):6-15; , J Clin Invest. 2016; 126: 2970-2988, Brain 2019; 142:2380-2401, Cell 2011; 144: 689-702, Nat Neurosci. 2013; 16: 1392-1400, Adv Neurobiol. 2017; 15:403-425

IkT-148009 IS A SMALL MOLECULE c-ABL INHIBITOR

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

IkT-148009 Selective Inhibitor of non-Receptor Tyrosine Kinases c-Abl and Abl2/Arg[1]



IkT-148009 No organ toxicity High brain penetrance

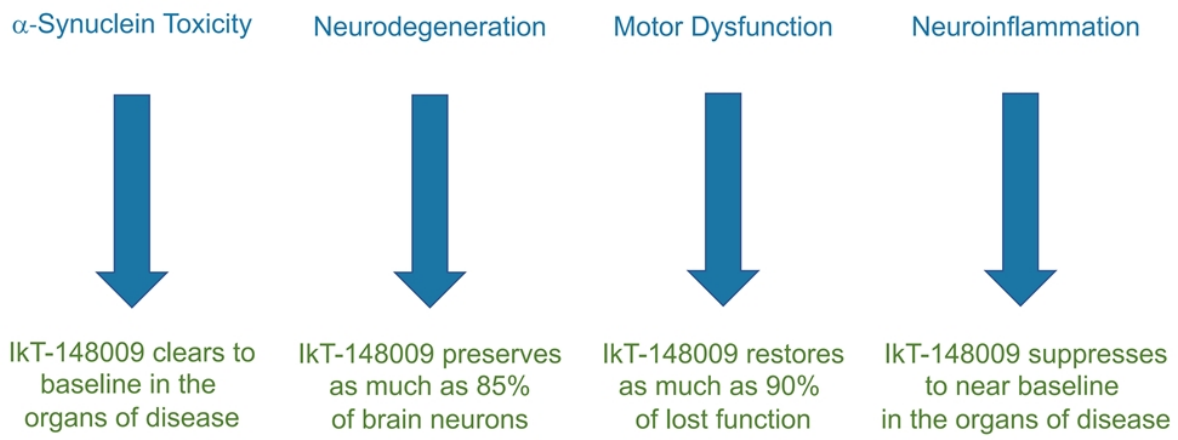
Toxicology in Rat/Monkey[1]	
Human equivalent dose of 1460 mg	
Cardiovascular	None
Renal	None
Liver	None
Bone marrow	None
Sternum	None
Blood	None
PBMCs	Slight increase in neutrophils within normal limits
Cytotoxicity	None in primary or mature cells
Sustained brain concentration	> 1 micromolar

[1]13 week and 39 week toxicology data shows IkT-148009 has a more favorable toxicity profile as dosing is extended

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

IkT-148009 MODIFIES DISEASE

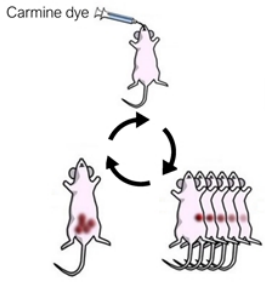
c-Abl Inhibition by IkT-148009 Reverses Functional Loss in Parkinson's Disease Animal Models



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

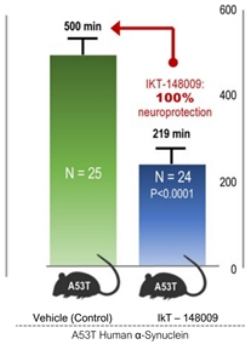


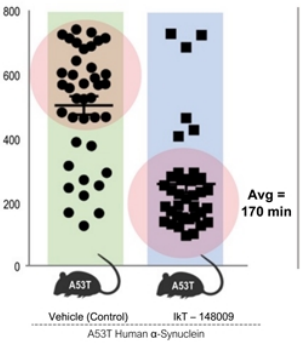
Measure defecation rate

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



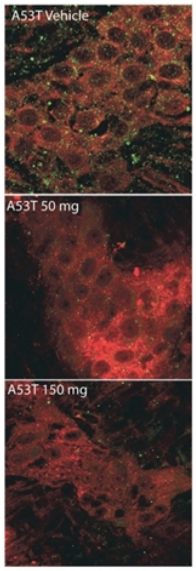


NOTES

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.

Oral IkT-148009 Treatment Nearly Clears Pathological α-Synuclein in the Brain and Gut

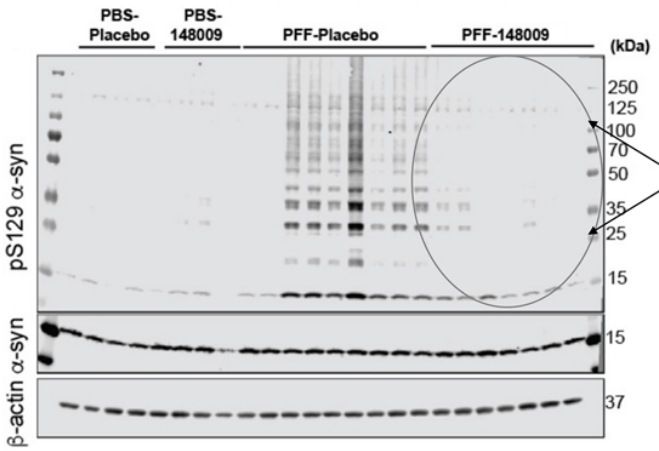
Clearance of toxicity in the gut



Green: Pathological α-synuclein
Red: Neural ganglia in gut

IkT-148009 drives near clearance of pathological α-synuclein

Clearance of toxicity in the brain



Aggregates Nearly cleared during treatment with IkT-148009

SUMMARY

Key points of Parkinson's Disease Initiation and Progression[1]

- Internalization of misfolded or aggregated α-synuclein and the activation of c-Abl in response is a key event in PD initiation/progression
- The long-sought goal of clearing α-synuclein aggregates for a therapeutic purpose should focus on the aggregates WITHIN the affected neurons
- Reduction and/or clearance of aggregates can be driven by restoring endogenous processes from within the affected neurons
- Recovery of lost functional activity can be achieved

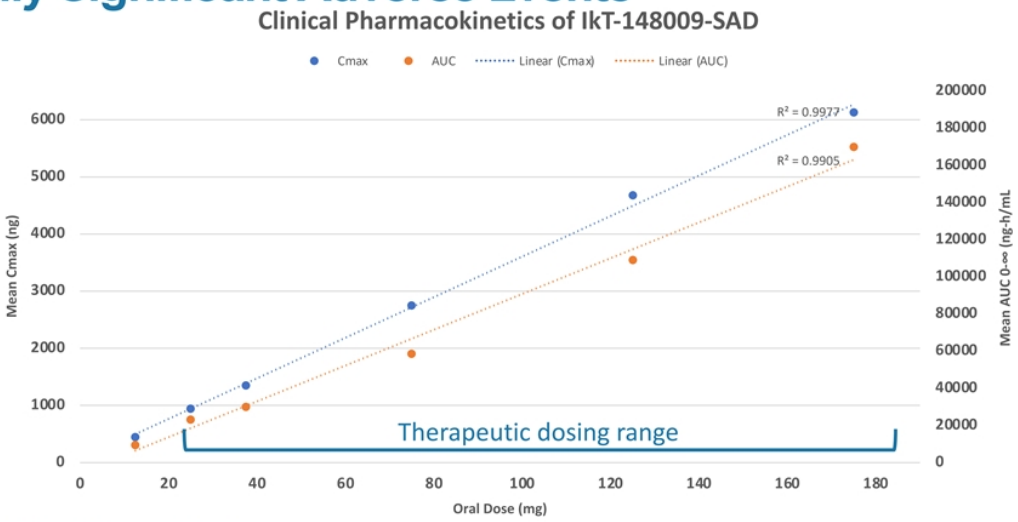
[1]Werner and Olanow, Mov. Disorders 2022 Jan;37(1):6-15; , J Clin Invest. 2016; 126: 2970-2988, Brain 2019; 142:2380-2401, Cell 2011; 144: 689-702, Nat Neurosci. 2013; 16: 1392-1400, Adv Neurobiol. 2017; 15:403-425


Inhibikase
Therapeutics
Nasdaq | IKT
Clinical Development and Status
AD-PD 2022, March 18 2022

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

Category	Demographic	Value (% of Total N=88)
Gender	Female	27 (37.5)
	Male	45 (62.5)
Age	Average (SD)	57.9 (5.72)
	Median	58.0
	Range	45, 69
Ethnicity	Hispanic or Latino	10 (13.9)
	Not Hispanic or Latino	62 (86.1)
Race	Black or African American	44 (61.1)
	White	27 (37.5)
	Other	1 (1.4)
Adverse events		7 (7.9), clinically insignificant,some emerging weeks after dosing with no clinical correlate

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



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 imatinib[1]

[1]FDA 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=8)
Gender	Female	4 (50)
	Male	4 (50)
Age	Average	61
	Median	61
	Range	57, 65
Ethnicity	Hispanic or Latino	1 (12.5)
	Not Hispanic or Latino	7 (87.5)
Race	Black or African American	1 (12.5)
	White	7 (87.5)
	Other	0 (0)
Adverse events		3 (37.5), (pneumonia, spinal headache, dermatitis)

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.

[1]FDA summary data for approval 21-335

IkT-148009 ONGOING TRIALS IN SAFETY AND DOSING

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. 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 1:1 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-α-synuclein GI, Skin and CSF; Whole Gut Transit Time (SmartPill™)
- Descriptive statistics

Multi-institutional effort across five models

Inhibikase Therapeutics, Inc.

Milton H Werner PhD
Roger Rush PhD
Terence Kelly, PhD
Surendra Singh PhD
Sydney Kruger
Irena Webster, MPH
Warren Olanow, MD
Andrew McGarry,MD
Cornelia Kamp
Syner-G Bio
Voisin Consulting

Johns Hopkins University

Senthil Karuppagounder, PhD
Richard Nguyen MS
Shivani Bisen
Yoko Yamashita
Nicholas Sloan
Brianna Dang
Alexander Sigmon
Yyeun Woo Lee
Shirley Marino Lee
Leslie Watkins
Erica Kim
Saurav Brahmachari, PhD
Manoj Kumar, PhD
Hu Wang, PhD
Ted Dawson, MD PhD
Valina Dawson, PhD

Subhash Kulkarni, PhD
Qian Li, PhD
Jay Pasricha, MD

Parkinson's Institute

Tyler Molitor, PhD
Cassandra Hempel
Ashani Chand
Carolee Barlow, MD

Funding: MJFF 13682, NINDS NS103695


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