CASSAVA sciences

Remi Barbier - President & CEO

Corporate Overview - Non-Confidential

September 2019

Forward-Looking Statements

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, including statement about our prospects for growth, intellectual property protection, market size, competitive position, regulatory agency action, and the anticipated development, timing, data readouts and therapeutic scope and value of our development stage products. You should not place undue reliance on these statements.

These statements involve significant risks and uncertainties. Our results may differ materially from those contained in such statements, including, among others: our inability to protect our intellectual property rights and to have sufficient rights or resources to develop or to commercialize our products; product competition; clinical trials of our products may fail or not be initiated or conducted in a timely manner; our products may show insufficient therapeutic or diagnostic effects or unacceptable safety profiles; adverse decisions or delays by regulatory authorities; existing preclinical and clinical data with respect to our products may not be indicative of future results; and the inability to manufacture successfully our products.

Additional factors that could cause actual results to differ significantly from those projected in our forward-looking statement are discussed in our Annual Report on Form 10-K, Quarterly Reports on Form 10-Q and recent Current Reports on Form 8-K. Our forward-looking statements are based on our current beliefs and expectations and speak only as of the date of this presentation.

We do not undertake any obligation to update this corporate presentation or any forward-looking statements, except as required by law.

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Investment Highlights

- **Cassava Sciences, Inc. (NASDAQ: SAVA) is a clinical-stage biotechnology company developing a novel drug treatment for Alzheimer's disease, and an investigational diagnostic to detect Alzheimer's with a simple blood test.**

- **We reported positive Phase 2a results of our lead product candidate, PTI-125, in Alzheimer's patients.**
  - In a Phase 2a study funded by the National Institutes of Health (NIH), treatment with PTI-125 for 28 days significantly reduced key biomarkers of neuroinflammation and neurodegeneration in patients ($p<0.001$).
  - PTI-125 is a proprietary small molecule with a novel mechanism of action: it restores the normal shape/function of Filamin A (FLNA), a protein that misfolds in the Alzheimer's brain.
  - Phase 2a achieved a 100% responder rate.
- **In September 2019, we initiated a Phase 2b study of PTI-125 in Alzheimer's.**

Positive clinical results support PTI-125 as a new, highly differentiated and potentially disease-modifying drug treatment for Alzheimer's disease.

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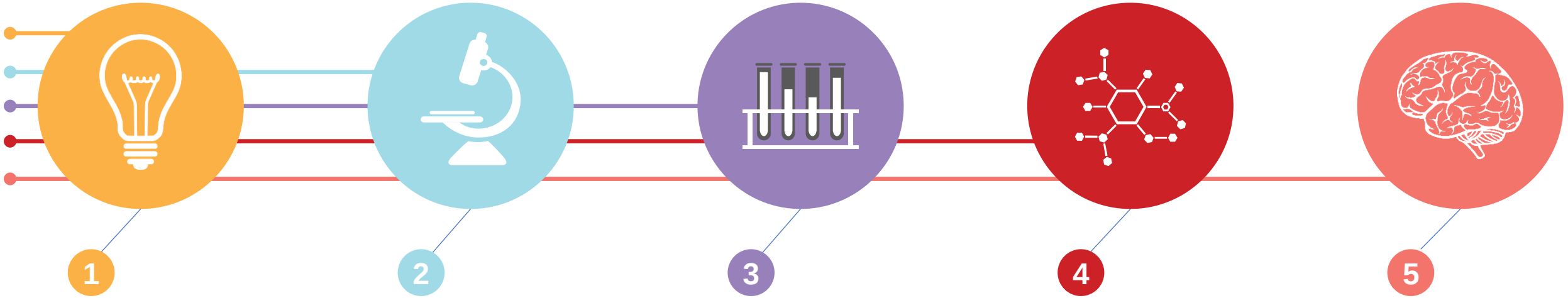
10-Year Drug Discovery Program



<2008
Basic research around neurobiology of Filamin A (FLNA).

2009
Discovery that altered FLNA associates with α7nAChR when Aβ signals.

2010
Screening/testing of compounds that bind altered FLNA and block α7nAChR/Aβ interaction.

2011
PTI-125 binds altered FLNA with high affinity. Preclinical testing of PTI-125.

2017 - present
Human testing of PTI-125.

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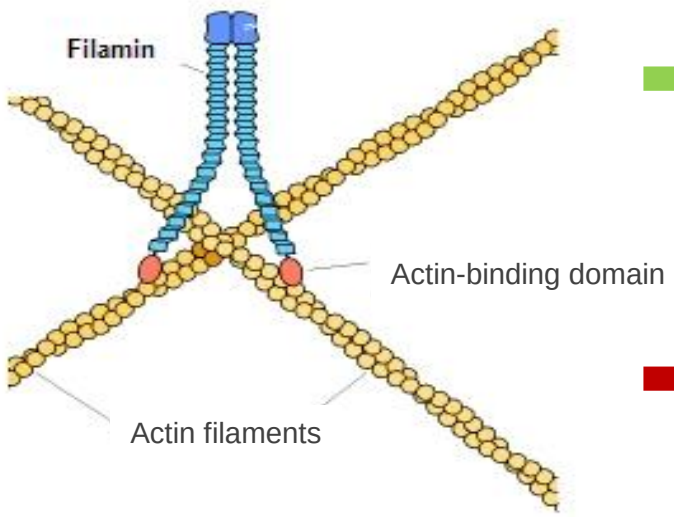
Unique Scientific Approach

- **The biology of the brain is heavily regulated by Filamin A, a scaffolding protein.**
  - FLNA interacts with at least 90 different proteins
  - FLNA interactions are critical to proper cell structure, cell function and signaling pathways

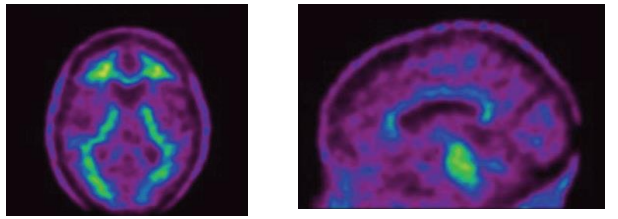
- **In Alzheimer's, FLNA takes on an altered conformation.**
  - Altered FLNA enables the massive neuroinflammation and neuropathology observed in Alzheimer's disease

PTI-125 binds to altered FLNA and restores its normal shape and function.



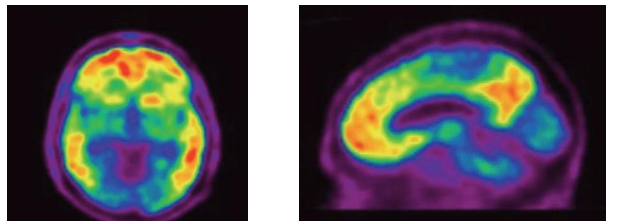
Healthy FLNA:
- Proper cell structure
- Proper cell signaling
- Proper cell function

Healthy, Elderly Brain



Altered FLNA:
- Dysfunctional cell signaling
- Impaired cell function
- Unstable cell structure

Patient with Alzheimer's



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Strong Scientific Rationale

IN BRIEF, VOLUME 11 | SEPTEMBER 2012

Targeting Filamin A Reduces Alzheimer's Signaling

In Alzheimer's disease, toxic amyloid-β_{42} ($A\beta_{42}$) binds to, and aberrantly signals through, the α7-nicotinic acetylcholine receptor (α7nAchR). Using tissue from both mouse models of AD and patients with AD, we show that $A\beta_{42}$ signaling is dependent upon the recruitment of the scaffolding protein filamin A to α7nAchR. An orally available small molecule that binds to filamin A (PTI-125) reduced abnormal signaling of α7nAChRs, decreased levels of tau phosphorylation and Aβ aggregates, and prevented Aβ-induced inflammatory cytokine release. PTI-125 greatly reduced the affinity of $A\beta_{42}$ for α7nAChRs, and could dissociate existing $A\beta_{42}$–α7nAChR complexes.

ORIGINAL RESEARCH PAPER Wang, H.-Y. *et al.* Reducing amyloid related Alzheimer's disease pathogenesis by a small molecule targeting filamin A. *J. Neuroscience.* **32**, 9773–9784 (2012)

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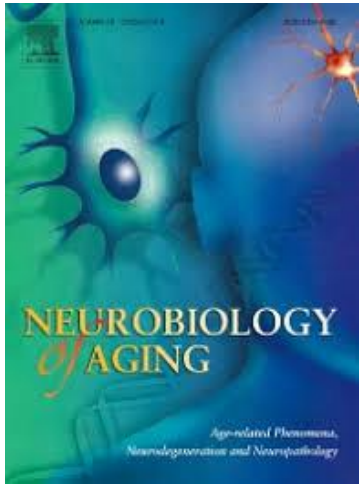
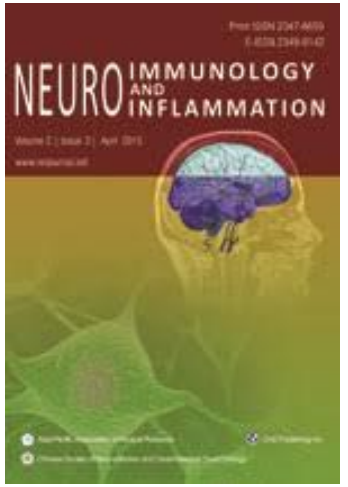
Science is Published; Funded by Peer-reviewed Grant Awards

The underlying science for PTI-125 has been subject to the scrutiny of many experts in the field……





….including NIH, which has awarded our science programs >$10 million in research grant awards.

National Institutes of Health

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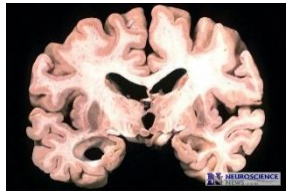
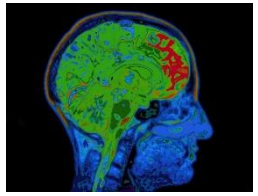
PTI-125: Treatment Effects in Animal Models

PTI-125 Dramatically Improved Brain Health in Animal Testing

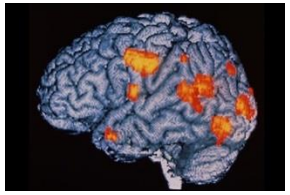
Reduced amyloid and tau deposits



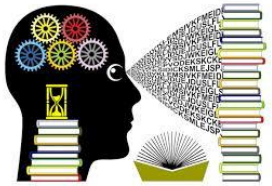
Reduced neuroinflammation



Improved insulin receptor signaling



Improved learning and memory



Published studies show that PTI-125 exerts powerful anti-neuroinflammatory effects

In animal models, chronic treatment with PTI-125 abolished IL-6 production and significantly suppressed levels of TNFa and IL-1b by 86% and 80%, respectively.

Published studies also show that PTI-125 restores the normal function of three receptors in the brain that are pivotal to cell survival, cognition and memory:

- alpha-7 nicotinic acetylcholine receptor (α7nAChR);
- N-methyl-D-aspartate (NMDA) receptor;
- the brain insulin receptor (IR).

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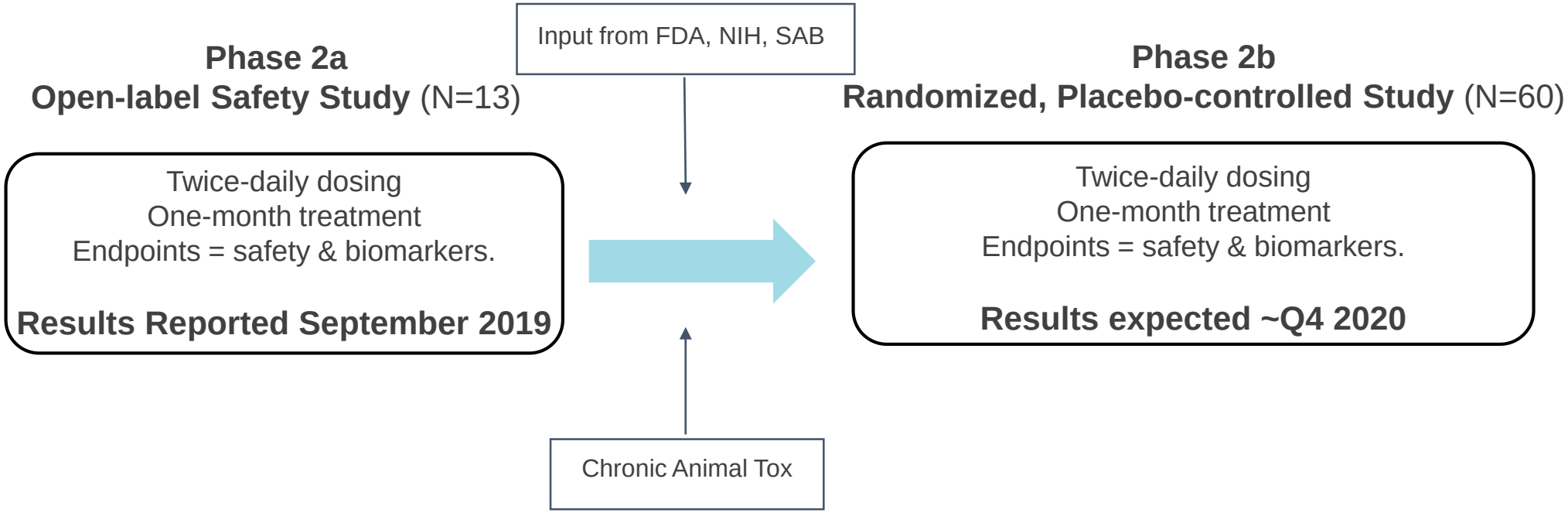
PTI-125: Ongoing Phase II Clinical Program

- **We are conducting a comprehensive, NIH funded, clinical testing program of PTI-125 in Alzheimer's, which was designed in collaboration with clinical/scientific advisors and NIH.**

- **Goal is to prepare PTI-125 for a pivotal efficacy program.**



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PTI-125: Phase 2a Study Design

- **Objective:** Investigate safety, PK, biomarkers under an IND application filed with FDA by Cassava Sciences
- **Study Design:** First-in-patient, multi-center, single-arm, open-label study
- **Population:** Mild-to-moderate Alzheimer's related dementia, MMSE ≥ 16 ≤ 24, age 50-85
- **Enrollment:** Thirteen (13) patients
- **PTI-125 Dose:** 100 mg oral tablets, administered twice-daily over 28 consecutive days
- **AD Index:** CSF Total tau/Aβ42 ≥ 0.30, indicating Alzheimer's
- **Biomarkers:** CSF samples collected twice (screening and post-treatment)

In Phase 2a we expected to observe decreases in key biomarkers of Alzheimer's disease, consistent with >10 years of basic research, PTI-125's mechanism of action and preclinical data.

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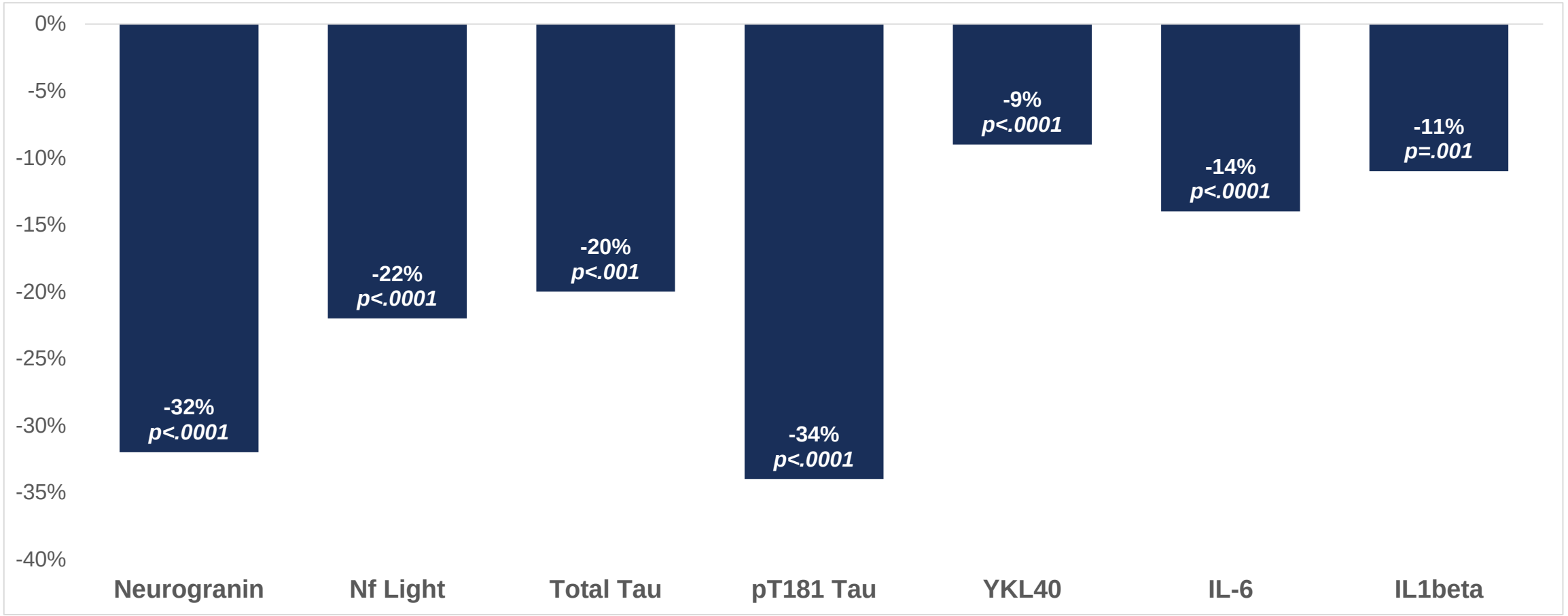
PTI-125: Phase 2a Top-line Study Results

Mean Change In Levels Of CSF Biomarkers

Baseline to Day 28



PTI-125: Summary of Clinical Results

- **CSF biomarkers were consistent in showing beneficial drug effects of PTI-125 on underlying disease pathology, neuroinflammation and neurodegeneration.**
  - Reduction in tau (T-tau and p-tau, -20% and -34%, respectively)
  - Reduction in axonal degeneration (neurofilament light chain, -22%)
  - Reduction in post-synaptic damage (neurogranin, -32%)
  - Reduction in proinflammatory cytokines (IL-6, IL-1β and TNFα, -14%, -11%, -5%, respectively)
  - Reduction in neuroinflammatory marker (YKL-40, -9%)

- **Treatment with PTI-125 for 28 days significantly reduced key biomarkers of neuroinflammation and neurodegeneration in patients with Alzheimer's (p<.001).**
  - Study achieved a 100% responder rate
  - Drug was well tolerated, with no drug related adverse events
  - Full data set to be presented at CTAD (*Clinical Trials on Alzheimer's Disease*) December 2019

PTI-125 appears to slow the rate of disease progression, consistent with the drug's mechanism of action and preclinical data.

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PTI-125: Next Step is a Phase 2b Study

- **A Phase 2b study was initiated September 2019, also funded by NIH.**

- **An objective of Phase 2b is to investigate effects of PTI-125 on validated biomarkers of disease in a larger study in Alzheimer's patients, including a dose-response relationship.**

	Phase 2a	Phase 2b
Status:	Completed	Initiation
Design:	Open-label	Blinded, randomized, placebo-controlled
Drug Dose:	100 mg b.i.d.	50 & 100 mg b.i.d.
Treatment Period:	28 days	28 days
# Patients:	13	60
Alzheimer's Patients:	Mild-to-moderate	Mild-to-moderate
MMSE Score:	16-24	16-26
Endpoints:	Biomarkers (CSF/plasma)	Biomarkers (CSF/plasma)

The underlying science also supports the development of an investigational diagnostic to detect Alzheimer's with a simple blood test, called PTI-125Dx.

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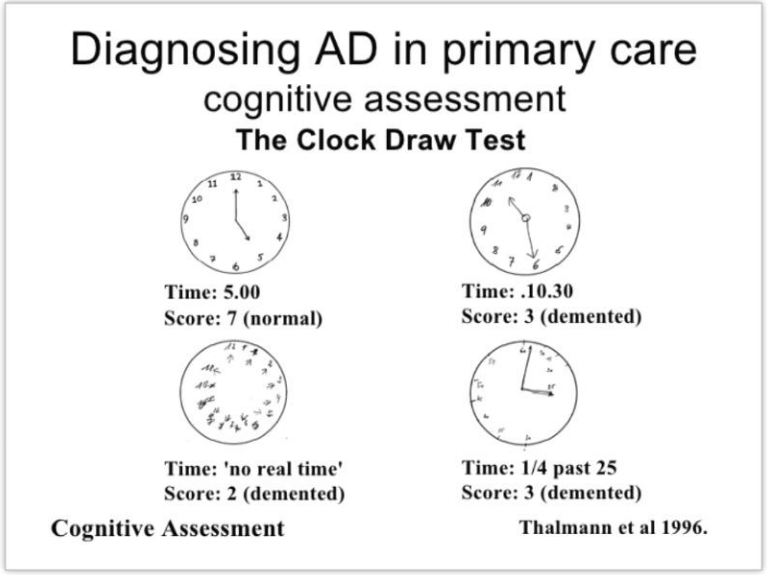
Current Process of Alzheimer's Diagnosis

Currently, the only way to definitively diagnose Alzheimer's disease is with an autopsy.

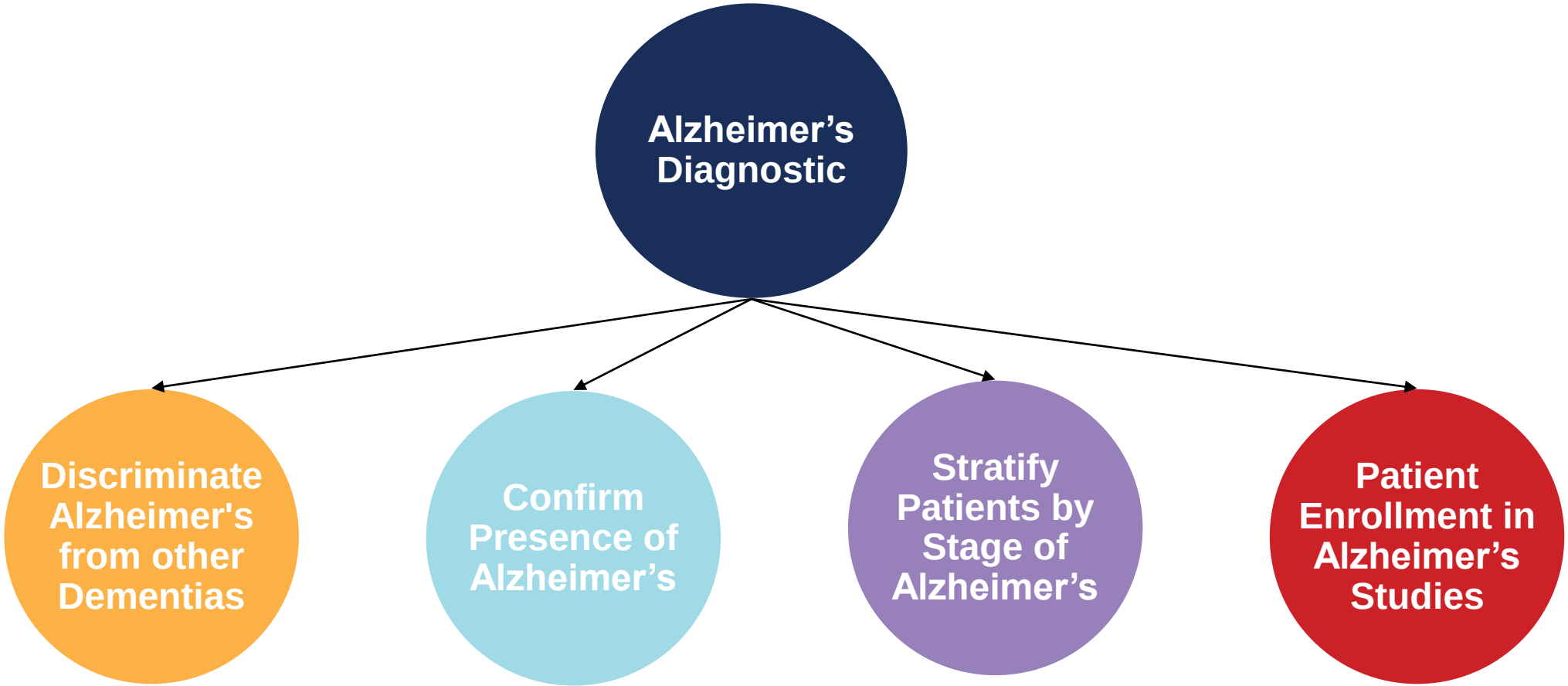
- **Other methods may be expensive, invasive, risky, uncomfortable, etc.**
- **Most people test only after they show obvious cognitive decline.**

- **Current approaches for diagnosing Alzheimer's include:**
  - Measurement of biomarkers of disease (Aβ42, T-tau, P-tau, NfL, etc.).
  - Structural neuroimaging techniques (MRI or CAT).
  - PET scan of brain amyloid (Amyvid®).
  - Screening tests for cognitive impairment, such as MMSE.



Profound Need for an Alzheimer’s Diagnostic Test

Goal is to identify people destined to develop Alzheimer’s long before symptoms occur and to cease — or at least slow down — brain damage before it is too late.



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PTI-125Dx: Investigational Diagnostic for Alzheimer's

- **PTI-125Dx is an investigational diagnostic to detect Alzheimer's disease in blood**
- **Goal is to detect Alzheimer's disease before the appearance of overt clinical symptoms**
- **Development of PTI-125Dx benefits from long-term scientific & financial support from the National Institute on Aging (NIA) at NIH**

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PTI-125Dx: Topline Study Results

In blinded studies, PTI-125Dx detected more than 10-fold separation between Alzheimer's patients and age-matched normal healthy controls or young cognitive intact subjects (N=232).

First Clinical Test

N=44
Site A (US)

Positive Result:
>10-fold separation of Alzheimer's patients from normal, healthy controls.

Second Clinical Test

N=88
Site B (US)

Positive Result:
>10-fold separation of Alzheimer's patients from normal, healthy controls.

Third Clinical Test

N=100+
Site C (Europe)

Positive Result:
>10-fold separation of Alzheimer's patients from normal, healthy controls.

Fourth Clinical Test

N=44
Site D (Asia)

Inconclusive result, due to failure of commercial antibody, indicative of need to develop proprietary, stable antibody with well-defined affinity.

We are currently developing a proprietary antibody specifically for use with PTI-125Dx.

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Key Financials

Nasdaq ticker: SAVA	
Shares Outstanding	17.2 million
Warrants Outstanding	9.1 million
Financials at June 30, 2019	
Cash & Cash Equivalents	$18.5 million
Approximate Net Cash Burn 2019	$ 5.0 million
Pre-tax NOLs	$78.7 million
Debt	none

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Scientific Advisory Board

Jeff Cummings, MD
Research Professor of the Department of Brain Health, UNLV and Director of the Center for Neurodegeneration and Translational Neuroscience of the Cleveland Clinic Lou Ruvo Center for Brain Health

Trevor William Robbins, CBE FRS FMedSci
Professor of Cognitive Neuroscience and former Head of the Department of Psychology at the University of Cambridge. Past President of the British Neuroscience Association.

Barbara Sahakian, FBA, FMedSci
Professor of Clinical Neuropsychology at the Department of Psychiatry and Medical Research Council/Wellcome Trust Behavioral and Clinical Neuroscience Institute, University of Cambridge.

Hoau-Yan Wang, PhD
Medical Professor at CUNY Medical School. Co-lead scientist on discovery & development of PTI-125.

Steven E. Arnold, M.D.
Translational Neurology Head of the Interdisciplinary Brain Center, Massachusetts General Hospital, Harvard Medical School.

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Experienced Management & Board

Management Team

Remi Barbier - Chairman, President & CEO

- **Founder/co-founder, three public life science companies.**
- Trustee emeritus, *Carnegie Institution for Science*; *Santa Fe Institute; California Institute for Quantitative Biosciences*

Nadav Friedmann, PhD, MD - Chief Medical Officer, Board member

- Formerly, Head of Biotechnology, J&J
- Formerly, CEO, Daiichi Pharmaceuticals USA
- **Eight FDA drug approvals prior to Cassava Sciences**

Lindsay H. Burns, VP Neuroscience

- Neurex, Inc; Elan Pharma; Abgenix, Inc.
- PhD with Trevor Robbins, University of Cambridge
- Post-doc, Parkinson's research, McLean hospital

Eric Schoen, Chief Financial Officer

- Vermillion, Inc; Borland Software, Inc.
- PricewaterhouseCoopers, Manager audit/assurance

Michael Zamloot - SVP Technical Operations

- Boehringer Mannheim; Athena; Ciba-Geigy.
- **Four FDA drug approvals prior to Cassava Sciences**

Board of Directors

Sandford Robertson

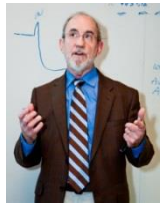
- Partner, Francisco Partners
- Formerly, Founder & Chairman, Robertson, Stephens & Company
- Independent Director, Salesforce.com

Saira Ramasastry

- Managing Partner, Life Sciences Advisory, LLC
- Formerly, Investment Banker, Merrill Lynch & Company, Inc.
- Director, Sangamo Biosciences, Inc. & and Glenmark Pharmaceuticals Ltd.

Robert Gussin, PhD.

- Formerly, Chief Scientific Officer and Corporate Vice President, Science and Technology, J&J
- Formerly, Director, Duquesne University, Duquesne University Pharmacy School & The University of Michigan Medical School Department of Pharmacology



Patrick Scannon, MD, PhD

- Co-founder, XOMA Corporation
- Formerly, Executive VP, Chief Biotechnology Officer XOMA
- Formerly, Chief Scientific and Medical Officer, XOMA

Michael O'Donnell

- Partner, Morrison & Foerster LLP
- Formerly, Partner, Wilson Sonsini Goodrich & Rosati

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Appendix: Key Publications

Neuroimmunology and Neuroinflammation
2017;4:263-71:
Altered filamin A enables amyloid beta induced tau hyperphosphorylation and neuroinflammation in Alzheimer's disease:
http://nnjournal.net/article/view/2313

Neurobiology of Aging
(Volume 55) July 2017, Pages 99—114)
PTI-125 binds and reverses an altered conformation of filamin A to reduce Alzheimer's disease pathogenesis:
http://www.neurobiologyofaging.org/article/S0197-4580(17)30087-8/

Alzheimer's & Dementia
Volume 8, Issue 4, Supplement, 1 July 2012, Pages p259-p260
PTI-125 reduces amyloid-related Alzheimer's pathogenesis by targeting filamin A:
https://www.sciencedirect.com/science/article/pii/S1552526012008242

Journal of Neuroscience
18 July 2012, 32 (29) 9773-9784
Reducing amyloid-related Alzheimer's disease pathogenesis by a small molecule targeting filamin A
http://www.jneurosci.org/content/32/29/9773.short

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