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): October 04, 2018

Pain Therapeutics, Inc.
(Exact Name of Registrant as Specified in Charter)

Delaware	**000-29959**	**91-1911336**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(I.R.S. Employer Identification Number)

7801 N Capital of Texas Highway, Suite 260, Austin, TX 78731
(Address of Principal Executive Offices) (Zip Code)

512-501-2444
(Registrant's telephone number, including area code)

Not Applicable
(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:

[ ] 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))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (17 CFR §230.405) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2).
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.

A copy of the investor presentation that Pain Therapeutics, Inc. used in conjunction with today's October 04, 2018 conference call is furnished as Exhibit 99.1 to this Form 8-K and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibit No. Description.

99.1	Pain Therapeutics, Inc. Investor presentation dated October 04, 2018

SIGNATURE

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.

Pain Therapeutics, Inc.

Date: October 04, 2018

By: /s/ Remi Barbier
Remi Barbier
Chairman of the Board of Directors,
President and Chief Executive Officer

NASDAQ: PTIE

Pain Therapeutics, Inc.



Overview of Neuroprotection Program

Non-confidential
October 2018

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 or growth, 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 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; unfavorable market launch of our product; reimbursement for our products; 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 any forward-looking statements.

Summary of Our Neuroprotection Program

- **We are developing a first-in-class program for neuroprotection of the brain.**
  - Our proprietary drug candidate, PTI-125, represents an entirely new target to treat Alzheimer's disease (AD).
  - Our proprietary diagnostic, PTI-125Dx, is a simple blood-based test to detect AD.
  - Data-driven approach to expand indications into rare diseases of the brain.

- **Strong scientific rationale, peer-reviewed publications and multi-year support from NIH.**
  - Scientific evidence for Filamin A is published in prestigious, peer-reviewed academic journals.
  - Science program has been awarded multiple, highly competitive, peer-reviewed NIH grants.

- **Positive results in Phase I study of PTI-125 provides strong rationale for drug development.**
  - Drug candidate was safe, well-tolerated, demonstrated favorable pharmacokinetics at all doses studied.

- **Phase II study with PTI-125 in Alzheimer's patients, initiation in Q4 2018.**
  - NIH-funded study in Alzheimer's patients to prepare for pivotal efficacy program.
  - Biomarker measurements are expected to validate mechanism of action in humans.

We are developing a first-in-class program for neuroprotection of the brain

Proprietary Drug Candidate PTI-125

ALZHEIMER'S DISEASE

Phase I
Positive Results

Phase II
Initiation Expected
Q4 2018

RARE DISEASE

Frontotemporal Dementia

Exploratory Program

Diagnostic PTI-125Dx

ALZHEIMER'S DISEASE

Blood-based Test

Clinical-stage Program

The biology of the brain is heavily regulated by Filamin A

Filamins are a class of proteins that are evolutionarily conserved

Normal Function

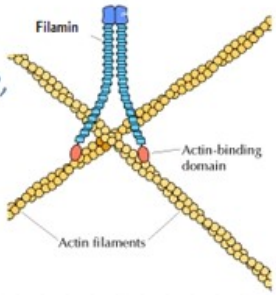
Filamin A (FLNA) protein is found inside all cells.

FLNA functions as a scaffold that interacts with many proteins.

FLNA is essential for proper cell structure, cell signaling, cell function.

FLNA helps build, organize, maintain and anchor the brain's dynamic network of proteins

Normal FLNA:
proper cell structure,
cell signaling,
cell function

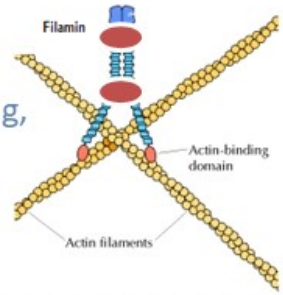


Normal FLNA, Healthy Brain

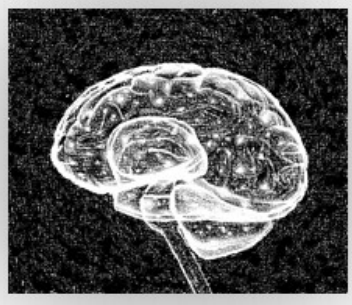
The Alzheimer's brain carries an altered form of Filamin A

Altered FLNA:

dysfunctional cell signaling,
impaired cell function



Altered FLNA, Alzheimer's Brain

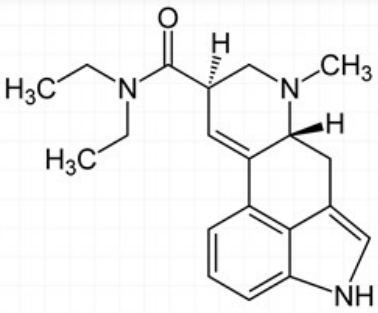


Altered Filamin A can no longer properly organize the brain's dynamic network of proteins

Publications show that an **altered conformation** of Filamin A enables the massive neuroinflammation and the amyloid and tau pathologies observed in the Alzheimer's brain.

Restore FLNA, restore the health of the brain.

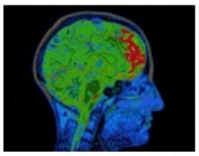
Drug candidate PTI-125 restores FLNA back to its native conformation.



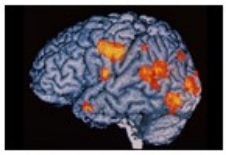
PTI-125 dramatically improved brain health in animal models.

Reduced amyloid and tau deposits

Reduced neuroinflammation

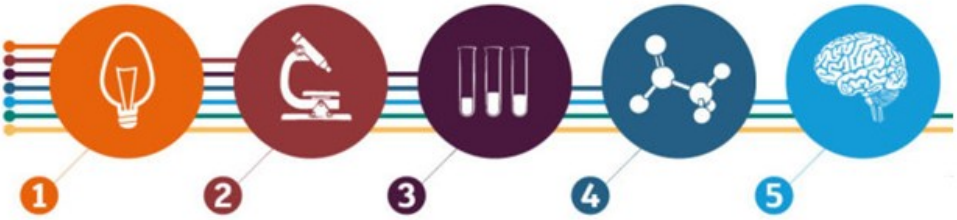


Improved insulin receptor signaling



Improved learning and memory

Drug Discovery



<2008
Basic research indicates the opioid receptor antagonists naloxone and naltrexone bind a specific site on FLNA with high affinity.

2009
We then discovered that FLNA associates with a7nAChR when Aβ signals.

2010
We tested many FLNA-binding compounds against this interaction.

2011
PTI-125 was the most potent inhibitor.

2017
Human testing of PTI-125

PTI-125 was designed in-house and characterized by academic collaborators from City University of New York.

Mechanism of action, in brief

nature REVIEWS DRUG DISCOVERY

IN BRIEF, VOLUME 11 | SEPTEMBER 2012

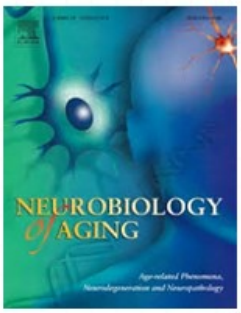
Targeting filamin A reduces Alzheimer's signaling

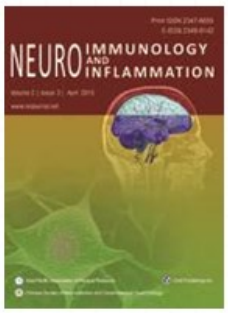
In Alzheimer's disease (AD), toxic amyloid-β_{42} ($A\beta_{42}$) binds to and aberrantly signals through the α7-nicotinic acetylcholine receptor (α7nAchR). Using tissue from both mouse models of AD and patients with AD, we show that $A\beta_{42}$ signaling is dependent upon the recruitment of the scaffolding protein filamin A to the α7nAchR. An orally available small molecule that bound 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. Neurosci.* **32**, 9773–9784 (2012)

Multiple peer-reviewed publications in prestigious journals

The underlying science for PTI-125 is complex and has been subject to the scrutiny of many experts in the field.





Today's news

To date, NIH has awarded us >$10 million in competitive research grants.
Each grant was awarded following an in-depth, peer-reviewed evaluation of PTI-125 and PTI-125Dx for scientific and technical merit.

October 4, 2018

Pain Therapeutics Awarded $3.5 Million NIH Grant to Study Alzheimer's

August 15, 2018

Pain Therapeutics Awarded $3.2 Million NIH Grant to Study Alzheimer's

We are advised by leading experts in the field

Jeff Cummings, MD

Director of Cleveland Clinic Lou Ruvo Center for Brain Health. Professor of Neurotherapeutics and Drug Development, Cleveland Clinic.

Trevor William Robbins, CBE FRS FMedSci

Professor of Cognitive neuroscience and former Head of the Department of Psychology at the University of Cambridge.

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

Tenured Associate 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.

Summary of PTI-125 treatment effects

- Restores function of alpha-7 nicotinic receptor (α7nAChR), N-methyl-D-aspartate (NMDA) receptor and insulin receptor (IR).
- Reduces neuronal damage (improves K^+-evoked Ca^{+2} influx).
- Reduces $A\beta_{42}$-induced tau hyper-phosphorylation.
- Reduces $A\beta_{42}$ deposits and neurofibrillary tangles (NFTs).
- Reduces inflammatory cytokine release.
- Reduces α7nAChR – $A\beta_{42}$ complexes.
- Improves synaptic plasticity.

Clinical Strategy

PTI-125 for Alzheimer's disease

Our Approach to Clinical Development

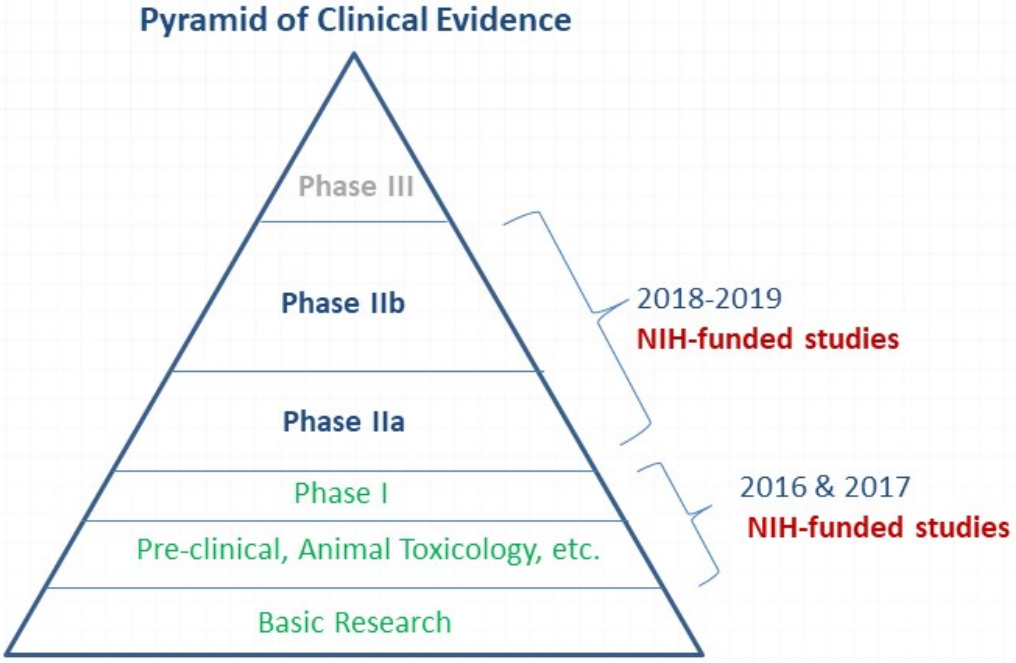
Our overall objective is to develop and gain regulatory approval for PTI-125 for the treatment of AD and to leverage our scientific/clinical platform to develop a first-in-class program for neuroprotection of the brain.

Key elements of our strategy include:

- **Continuous publications in peer-reviewed journals.**
- **Advance PTI-125 in a comprehensive Phase II clinical development program.**
- **Evaluate the development of PTI-125 for treatments of rare diseases, non-AD dementia.**
- **Expand the development of our blood-based diagnostic/biomarker for AD.**
- ***Generate favorable Phase II data to enable us to seek one or more strategic collaborations with pharmaceutical or biotechnology companies.***

Clinical Program for PTI-125

A comprehensive clinical program for PTI-125 is being developed in collaboration with experts from the NIH, FDA and science advisors.



Phase I Clinical Study Results

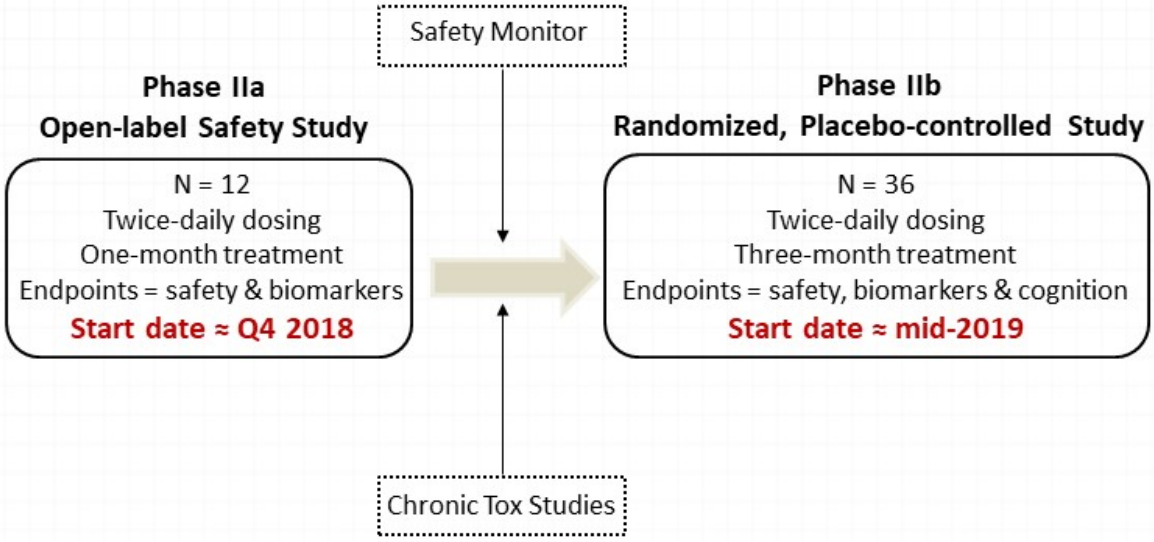
- **An NIH-funded, first-in-human Phase I study was completed in 2017 in the U.S.**
- **This study evaluated the safety, tolerability and pharmacokinetics of PTI-125 in 24 healthy subjects exposed to 50-200 mg in a single ascending dose study.**
- **Main takeaway:**

  Phase I study results demonstrated favorable safety, tolerability, bioavailability and dose-proportionality for PTI-125 at all doses studied, indicating a favorable profile for further development.

  - Full results published at the 10th Annual International Conference on Clinical Trials on Alzheimer's Disease (CTAD, Nov 2017).

Phase II Clinical Program

A comprehensive, NIH funded, Phase II program for PTI-125 in patients with mild-to-moderate AD is intended to generate safety and tolerability data, demonstrate target engagement, and prepare PTI-125 for a pivotal efficacy program.



Meaningful Phase II data may enable us to seek one or more strategic collaborations with pharmaceutical or biotechnology companies.

Competitive Landscape

Compounds in Late-Stage Development

Several compounds with disease modifying potential are currently in the late stages of development for various stages of AD. These generally fall into three broad categories: monoclonal antibody immunotherapies, ß-secretase inhibitors and other.

Monoclonal Antibody Immunotherapies

Several pharmaceutical companies are developing passive monoclonal antibody to treat AD. These include Biogen's aducanumab and Roche's crenezumab and gantenerumab. Aducanumab is currently in Phase 3 development with top-line data expected in 2019. Top-line data for the first trial of gantenerumab is expected in 2018, and top-line data for the first trial of crenezumab is expected in late 2020. Alector, a high-publicity, well-funded start-up is developing active (immune modulating) antibodies to clear amyloid from the brain.

ß-Secretase Inhibitors

Several pharmaceutical companies are developing ß-secretase inhibitors to treat AD. These include Eisai's elenbecestat, Eli Lilly's lanabecestat, Merck's verubecestat and Novartis and Amgen's CNP520. The Phase 3 trial of verubecestat in mild to moderate AD was terminated in 2017 due to lack of efficacy, however, verubecestat is continuing to be evaluated in early-stage AD with top-line data expected in 2019. Top-line data for elebecestat's Phase 3 trial and lanabecestat's Phase 3 trial are expected in 2020. Two Phase 2/3 studies of CNP520 have been initiated, one in APOE4/4 homozygotes only, and one in APOE carriers. These studies are estimated to complete in 2024.

Aß Inhibitors

Tramiprosate, formerly Alzhemed, now relabeled ALZ-801, is a patented variant of the amino acid taurine. It is reported to inhibit the interaction of Aβ with endogenous glycosaminoglycans and thereby prevent β-sheet formation. Bellus (formerly, Neurochem) evaluated this agent in 16 clinical trials with over 2,000 AD patients; all studies failed to show efficacy. A post-hoc analysis of patients with two copies of the APOE/4 gene showed some promising data. Alzheon, Inc. is attempting to raise $40-50 million to conduct Phase III studies with tramiprosate .

Summary of Technical Advantages

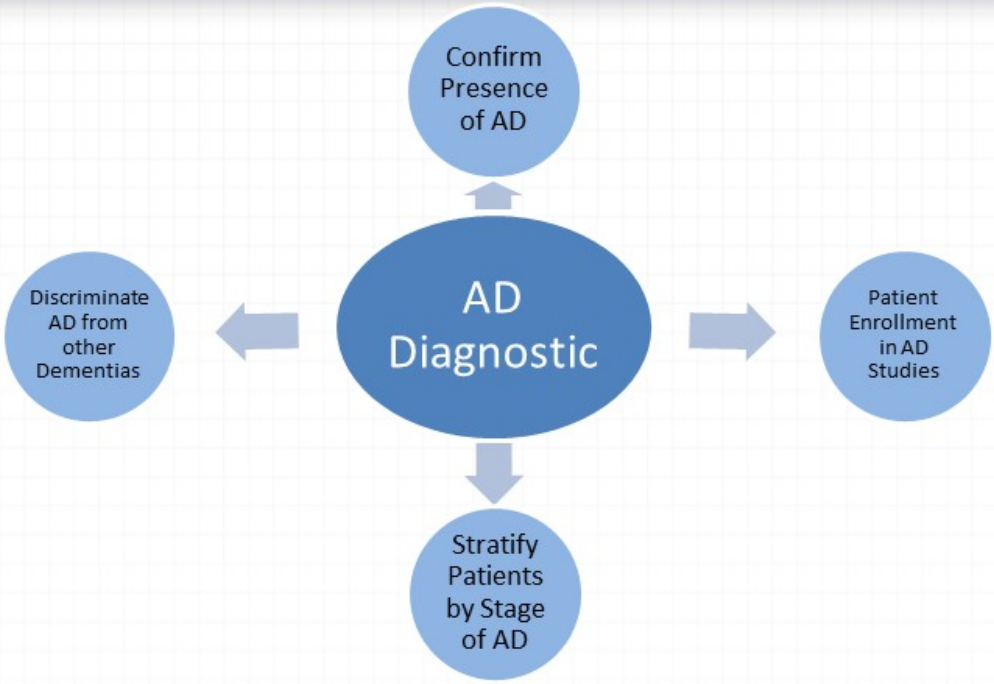
- **PTI-125 represents a new target for AD drug development.**
  - By reversing multiple AD neuropathologies, is expected to slow the course of AD.
  - Significant, long-term scientific and financial support from the NIH.
- **PTI-125 is not dependent on clearing amyloid from the brain.**
  - In contrast, agents that reduce amyloid levels, such as BACE inhibitors, must reduce amyloid >1,000-fold to impact Aβ's tight binding to α7nAChR.
- **PTI-125 releases tightly bound amyloid without direct competition.**
  - In contrast, monoclonal antibodies against amyloid compete in a tug-of-war with α7nAChR.
- **PTI-125 produces multiple beneficial effects by binding to a single target (i.e., FLNA).**
  - Reduced neuroinflammation, tau hyperphosphorylation, insulin resistance in the brain, etc.
- **PTI-125 is a small molecule.**
  - Advantages include well-defined structure; simple & predictable manufacturing; stable; generally non-immunogenic; and simple oral administration for chronic use in elderly populations.

The underlying science for PTI-125 supports the development of a biomarker/diagnostic to detect Alzheimer's disease with a simple blood test.

PTI-125Dx

An experimental blood-based test to detect Alzheimer's disease

There is a profound need for a diagnostic test for Alzheimer's

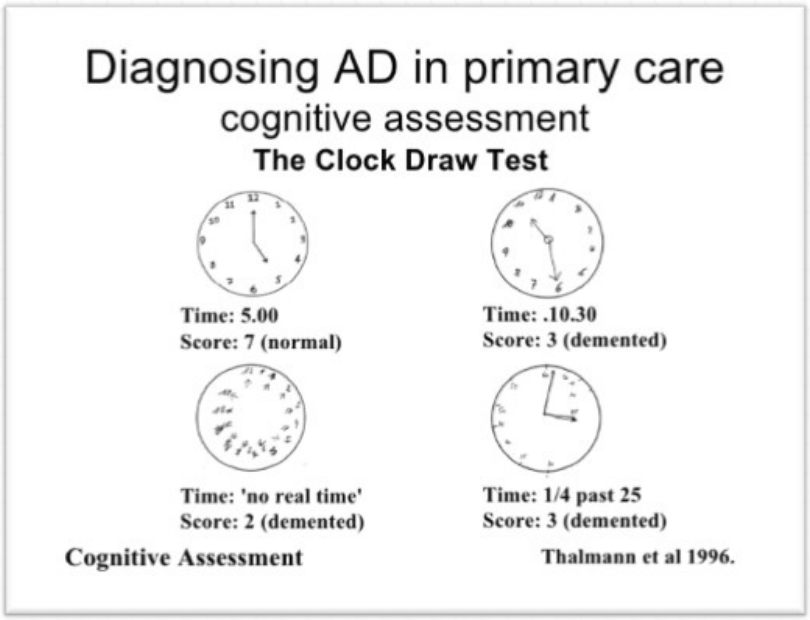


Amyloid-β begins to accumulate up to two decades before cognitive symptoms appear.

The ultimate goal is to identify people destined to develop AD long before symptoms occur, and cease -- or at least slow down -- brain damage before it is too late.

Current Approaches to Detect AD

Right now, the only definitive way to diagnose AD is through autopsy after death.
Everything else is expensive, invasive, risky, uncomfortable and/or uncertain.
Plus, no one is tested until they show obvious cognitive decline.



Current approaches for diagnosing AD include measurement of amyloid-β (specifically Aβ42), total tau (T-tau) or phosphorylated tau (P-tau) levels in cerebrospinal fluid (CSF); structural neuroimaging techniques (MRI or CAT); PET imaging of brain amyloid (AmyVid®) or inflammation; and batteries of cognitive tests. Usually, a combination of more than one test is necessary to provide a working diagnosis.

Detecting AD --- as simple as getting a blood test?

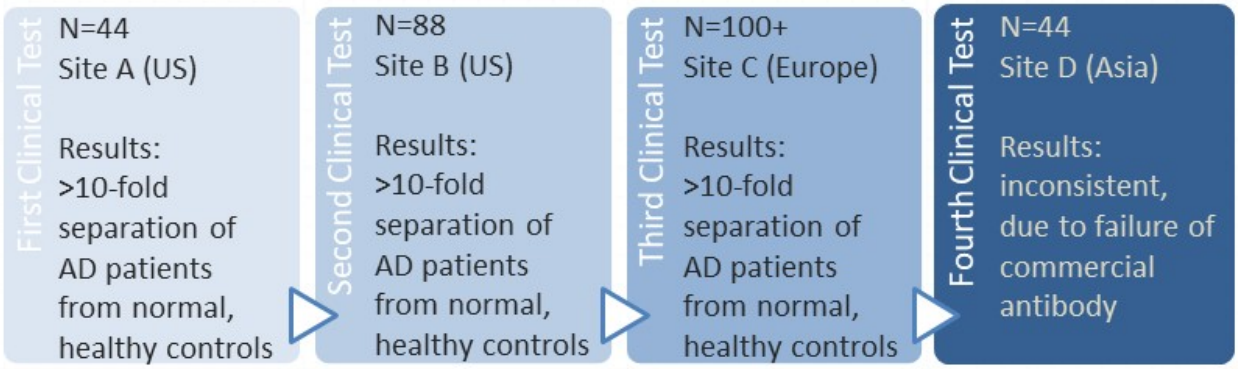
PTI-125Dx is our proprietary, experimental blood-based diagnostic to detect Alzheimer's disease, even before clinical symptoms appear.

Altered Filamin A (FLNA) is a hallmark feature of AD pathology in the brain. It also appears in plasma. PTI-125Dx detects and quantifies this biomarker.

The National Institute on Aging of the NIH has substantially funded this research program, following multiple in-depth, peer-reviewed evaluations of PTI-125Dx for scientific and technical merit.

Top-line study results indicate PTI-125Dx can detect AD quantitatively, with sensitivity and specificity

PTI-125Dx detected more than 10-fold separation between AD patients and age-matched normal healthy controls or young cognitive intact (YCI) subjects (N=232).
All samples were blinded and analyzed by an outside lab.



Sensitivity is the proportion of subjects *with* AD in whom the test is positive.

Specificity is the proportion of subjects *without* AD in whom the test is negative.

Formula for approving a new diagnostic test

FDA has recommended that estimates of sensitivity and specificity be reported with 95% confidence intervals (CI). This is the formula:

$$\hat{p} \pm 1.96 \sqrt{\frac{\hat{p}(1-\hat{p})}{n}}$$

p is the estimate of sensitivity or specificity; *n* is either the number of true-positive samples (for sensitivity) or the number of true-negative samples (for specificity).

PTI-125Dx – can it serve as a clinical endpoint for an accelerated drug approval?

FDA Guidance Document: Endpoints for Early AD Trials in Patients

"Because it is highly desirable to intervene as early as possible in AD, it follows that patients with characteristic pathophysiologic changes of AD but no subjective complaint, functional impairment, or detectable abnormalities on sensitive neuropsychological measures (Stage 1 patients) are an important target for clinical trials. A clinically meaningful benefit cannot be measured in these patients because there is no clinical impairment to assess (assuming that the duration of a trial is not sufficient to observe and assess the development of clinical impairment during the conduct of the trial). In Stage 1 patients, an effect on the characteristic pathophysiologic changes of AD, as demonstrated by an effect on various biomarkers, may be measured. ***Such an effect, analyzed as a primary efficacy measure, may, in principle, serve as the basis for an accelerated approval*** *(i.e., the biomarker effects would be found to be reasonably likely to predict clinical benefit, with a post-approval requirement for a study to confirm the predicted clinical benefit)."*

Source: FDA Guidance for Industry, Early Alzheimer's Disease: Developing Drugs for Treatment, February 2018, Page 6

PTI-125Dx -- discussion and next steps

- ✓ We are developing PTI-125Dx as a safe, fast, accurate and quantitative blood-based biomarker/diagnostic to detect Alzheimer's disease.
- ✓ Encouraging results to date indicate PTI-125Dx is a feasible program.
- ✓ In blinded studies, PTI-125Dx detected >10-fold differences between AD patients and age-matched normal controls or young cognitively intact (YCI) subjects (N=232).
- ✓ Results from Study #4 indicate that commercial antibodies are not always properly validated. In 2018-2019, we expect to make a proprietary antibody with well-defined affinity, specificity, etc.
- ✓ With repeat measurements over time, PTI-125Dx may provide a probability of cognitive decline or disease progression. Even if there is not a cutoff value for AD, it may be important to incorporate data from PTI-125Dx into the diagnostic framework for AD.
- ✓ Recent FDA guidance indicates that a qualified diagnostic is needed for regulatory approval of an AD drug candidate that reduces a validated biomarker of disease.

Intellectual Property

- **We own exclusive, worldwide commercial rights to PTI-125 and related technologies.**
- **No royalty, milestone payments or other form of payment is owed any third-party.**
- **Intellectual property protection runs though 2034+**

TITLE: ALZHEIMER'S DISEASE ASSAY IN A LIVING PATIENT	Issued Nov. 22, 2016 Patent No. 9,500,640
TITLE: ALZHEIMER'S DISEASE ASSAY IN A LIVING PATIENT	Issued May 31, 2016 Patent No. 9,354,223
TITLE: ALZHEIMER'S DISEASE ASSAY IN A LIVING PATIENT	Issued Nov. 22, 2016 Patent No. 9,500,640
Title: ANALGESIA WITH MINIMAL TOLERANCE AND DEPENDENCE BY A MU OPIOID RECEPTOR AGONIST THAT ALSO BINDS FILAMIN A	Issued: 5/13/14 Patent No. 8,722,851
Title: ANALGESIA WITH MINIMAL TOLERANCE AND DEPENDENCE BY A MU OPIOID RECEPTOR AGONIST THAT ALSO BINDS FILAMIN A	Issued: 7/23/13 Patent No. 8,492,349
TITLE: FILAMIN A-BINDING ANTI-INFLAMMATORY ANALGESIC	Issued: 11/12/13 Patent No. 8,580,808

Summary of Our Neuroprotection Program

- **We are developing a first-in-class program for neuroprotection of the brain.**
  - Our proprietary drug candidate, PTI-125, represents an entirely new target to treat Alzheimer's disease (AD).
  - Our proprietary diagnostic, PTI-125Dx, is a blood-based test to detect AD before clinical symptoms are apparent.
  - We have a data-driven approach to expand indications into rare diseases of the brain.

- **Strong scientific rationale, peer-reviewed publications and multi-year support from NIH.**
  - Scientific evidence for Filamin A is published in prestigious, peer-reviewed academic journals.
  - Science program has been awarded multiple, highly competitive, peer-reviewed NIH grants.

- **Positive results in Phase I study of PTI-125 provides strong rationale for drug development.**
  - Drug candidate was safe, well-tolerated, demonstrated favorable pharmacokinetics at doses studied.

- **Phase II study with PTI-125 in Alzheimer's patients, initiation Q4 2018.**
  - NIH-funded Phase II program is intended to generate safety and tolerability data, demonstrate target engagement, and prepare PTI-125 for a pivotal efficacy program.
  - Protocol development in collaboration by expert opinion from the NIH, FDA and outside advisors.

Key Financial Information

- ~$20 million of cash & equivalents at 9/1/18
- ~$5-6 million net cash use expected in 2018
- ~17 million shares outstanding
- ~9 million warrants outstanding
- ~15 million shares float, per NASDAQ
- ~$74 million pre-tax NOLs
- No debt

All number unaudited.

Reinventing the Company

- ☑ Financial stability
- ☑ New scientific talent around Alzheimer's disease
- ☐ New CFO announcement
- ☐ New corporate identify around Alzheimer's disease
- ☐ New company name
- ☐ New ticker symbol on NASDAQ
- ☐ Re-designed website
- ☐ Exposure to new investors via non-deal roadshows

Experienced Team

Remi Barbier - Chairman, President & CEO - 1998 to date.
- **Founder/co-founder, three public life science companies.**
- Trustee, *Carnegie Institution for Science; Santa Fe Institute; Ca. Institute for Quantitative Biosciences.*

Nadav Friedmann, PhD, MD - Chief Medical Officer, 2001 to date; Board member.
- J&J; Daiichi Pharmaceuticals; Abbott Labs.
- **Eight FDA drug approvals prior to Pain Therapeutics.**

Lindsay H. Burns, VP, Neuroscience, 2001 to date
- Neurex; Elan; Abgenix.
- PhD with Trevor Robbins, University of Cambridge.
- Post-doc in Parkinson's research, McLean hospital.

George B. Thornton, PhD – Sr. VP, Technology
- J&J; GeneMedicine; Immune Complex Corp.; Apovia.
- Developed prototype diagnostic assays for HBV.

Michael Zamloot - Sr. VP, Tech. Operations, 2000 to date.
- Boehringer Mannheim; Athena; Ciba-Geigy.
- **Four FDA drug approvals prior to Pain Therapeutics.**

Board of Directors

Sandy Robertson

Saira Ramasastry

Bob Gussin, PhD.

Pat Scannon, MD, PhD

Mike O'Donnell

Pain Therapeutics, Inc.



THANK YOU!

Appendix

Neuroprotection Program -- 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
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