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) November 4, 2020

Cassava Sciences, Inc.

(Exact name of registrant as specified in its 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, Texas 78731
(Address of principal executive offices, including 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 (see General Instruction A.2 below):

☐ Written communication 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 communication pursuant to Rule 14d-2(b) under the Exchange Act (17CFR 240.14d-2(b))

☐ Pre-commencement communication pursuant to Rule 13e-4(c) under the Exchange Act (17CFR 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	SAVA	NASDAQ Capital Market

Indicate by check mark whether the registrant is an emerging growth company as defined in 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.

A copy of the Cassava Sciences, Inc. presentation at the *13th Clinical Trials on Alzheimer's Disease (CTAD)* is furnished as Exhibit 99.1 to this Form 8-K and is incorporated herein by reference.

Item 8.01. Other Events.

On November 4, 2020, Cassava Sciences, Inc. issued a press release, a copy of which is attached hereto as Exhibit 99.2 and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibit No. Description

99.1	Cassava Sciences, Inc. CTAD 2020 presentation
99.2	Cassava Sciences, Inc. CTAD press release dated November 4, 2020

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended, the registrant has duly caused this report to be signed on its behalf by the undersigned thereunto duly authorized.

CASSAVA SCIENCES, INC.
a Delaware corporation

Date: November 4, 2020

By: /s/ ERIC J. SCHOEN
Eric J. Schoen
Chief Financial Officer

Sumifilam Significantly Improves Eleven CSF Biomarkers in a Randomized, Placebo-controlled, One-month Clinical Trial in Alzheimer's Disease Patients

Lindsay BURNS, Hoau-Yan WANG, Zhe PEI, Kuo-Chieh LEE, Yaneicy GONZALEZ-ROJAS, Tamara DOEHNER, John PUENTE, Patrick SCIARA, Brian BECK, Evelyn LOPEZ-BRIGNONI, Boris NIKOLOV, Carrie CROWLEY, Nadav FRIEDMANN

November 7, 2020

CTAD
Clinical Trials on Alzheimer's Disease

CASSAVA sciences

Forward-Looking Statements

This presentation contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. To identify such forward-looking statements, in some cases we use terms such as "predicts, "believes," "potential," "estimates," "anticipates," "expects," "plans," "intends," "may," "could," "might," "should" or other words that will convey risk or uncertainty of future events or outcomes. All statements other than statements of historical fact contained in this presentation including, but not limited to, statements regarding plans or timing for future Phase 3 clinical studies with sumifilam; the interpretation of prior or current results of our Phase 2 clinical studies, including the measured effects of sumifilam on cognition; plans to publish results in a peer-reviewed journal; potential health benefits, if any, of changes in levels of CSF biomarkers; verbal commentaries made by Cassava Sciences' employees; and potential benefits, if any, of the Company's product candidates for Alzheimer's disease are forward-looking statements.

Such statements are based largely on our current expectations and projections about future events. Such statements speak only as of the date of this presentation and are subject to a number of risks, uncertainties and assumptions, including, but not limited to, those risks relating to the ability to conduct or complete clinical studies on expected timelines, to demonstrate the specificity, safety, efficacy or potential health benefits of our product candidates, the severity and duration of health care precautions given the international outbreak of an infectious disease and including those described in the section entitled "Risk Factors" in our Annual Report on Form 10-K for the year ended December 31, 2019 and future reports to be filed with the SEC. In light of these risks, uncertainties and assumptions, forward-looking statements and events discussed in this presentation are inherently uncertain and may not occur. Actual results could differ quickly, materially and adversely from those anticipated or implied in the forward-looking statements. Accordingly, you should never rely upon forward-looking statements as predictions of future events. We do not undertake any obligation to update this corporate presentation or any forward-looking statements included therein, except as required by law.

The content of this presentation is solely our responsibility and does not necessarily represent the official views of the National Institutes of Health (NIH).

Disclosures

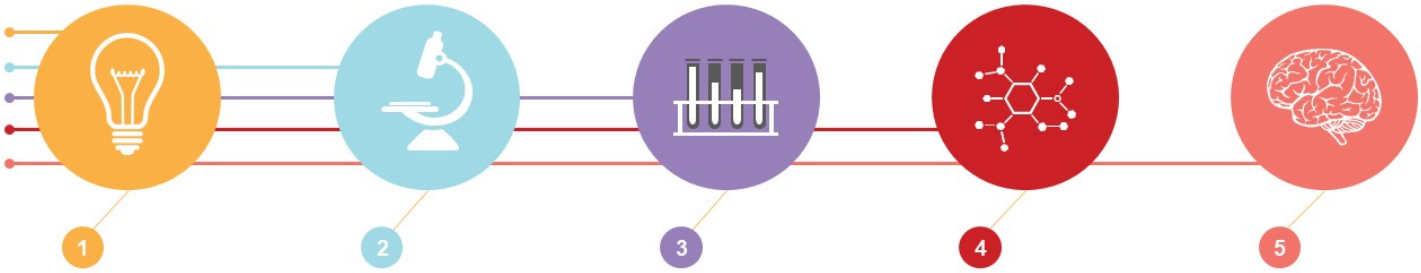
- Sumifilam is under clinical development by Cassava Sciences, Inc.
- L.H. Burns, PhD, N. Friedmann, PhD, MD, and C. Crowley are employees of Cassava Sciences.
- H-Y. Wang, PhD, is a consultant to Cassava Sciences.
- H-Y. Wang, PhD, Z. Pei, PhD, and K-C. Lee are employees of City University of New York School of Medicine.
- Sumifilam benefits from long-term scientific & financial support from the National Institute on Aging (AG050301, AG056166, AG060878, AG065152).

Introduction to Sumifilam

- **Sumifilam is a proprietary, small molecule drug candidate to treat Alzheimer's disease (AD) and other neurodegenerative diseases.**
  - Drug was discovered and developed in-house, 2008 to present.

- **Sumifilam binds a single target, has a dual mechanism of action:**
  - Reduces neurodegeneration *and* neuroinflammation.
  - Published preclinical and mechanism of action data support sumifilam's potential as a disease-modifying drug for AD that may also enhance cognition.

Sumifilam: A Journey of Basic Research to Clinical Research



<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
Sumifilam (formerly, PTI-125) binds altered FLNA with high affinity, blocks α7nAChR/Aβ interaction.

Preclinical testing of sumifilam.

2017 - present
Clinical testing of sumifilam.

Positive results reported in Alzheimer's patients.

CTAD
Clinical Trials on Alzheimer's Disease

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Phase 2b Clinical Study of Sumifilam

I. Mechanism of Action

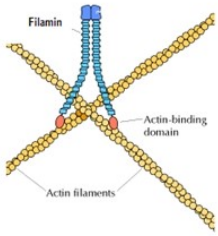
II. Phase 2b Study Results

III. Study Conclusions

Target of Sumifilam is *Altered* Filamin A (FLNA)

FLNA is an intracellular scaffolding protein anchored in the cell membrane.

FLNA interacts with > 90 proteins, influencing many signaling pathways.



The AD brain carries an *ALTERED* conformation of FLNA.

Altered FLNA is critical to amyloid beta's toxicity.

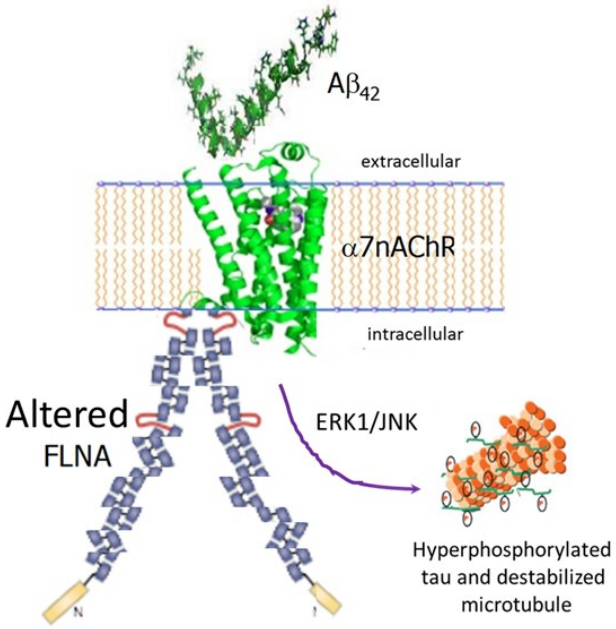
Sumifilam Mechanism of Action

- **Altered FLNA enables Aβ_{42} signaling via two different receptors:**
  1) α7-nicotinic acetylcholine receptor (α7nAChR) ⟶ hyperphosphorylates tau
  2) Toll-like receptor 4 (TLR4) ⟶ releases inflammatory cytokines

- **Sumifilam preferentially binds *altered* FLNA, restores its proper shape/function, potently suppressing Aβ_{42} signaling via α7nAChR and TLR4.**
  - Through a single target, sumifilam reduces both neurodegeneration and neuroinflammation.

Altered FLNA links to α7-nicotinic acetylcholine receptor

- **$A\beta_{42}$ binds α7nAChR and recruits FLNA, altering its shape.**
- **Altered FLNA linkage to α7nAChR enables a *femtomolar* affinity of $A\beta_{42}$ for α7nAChR and the signaling that hyperphosphorylates tau.**



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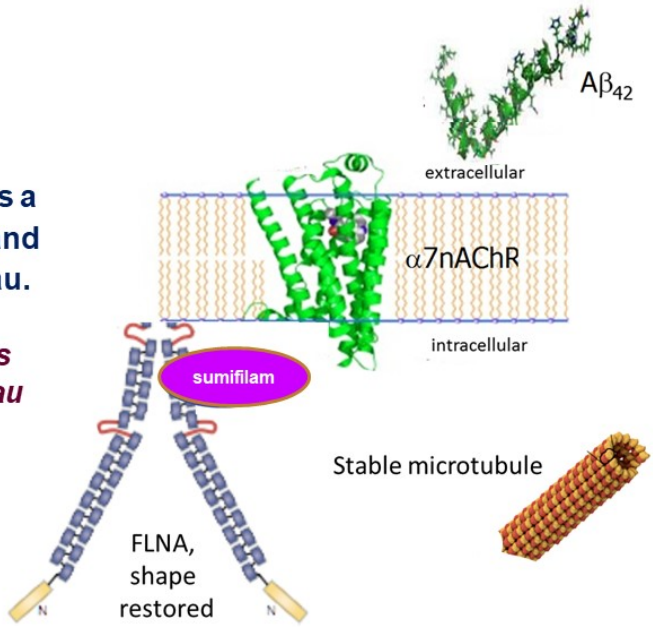
- Altered FLNA linkage to α7nAChR enables a *femtomolar* affinity of $A\beta_{42}$ for α7nAChR and the signaling that hyperphosphorylates tau.

Sumifilam binds altered FLNA, restores its normal shape, stops $A\beta_{42}$ signaling and tau hyperphosphorylation.



CTAD Clinical Trials on Alzheimer's Disease

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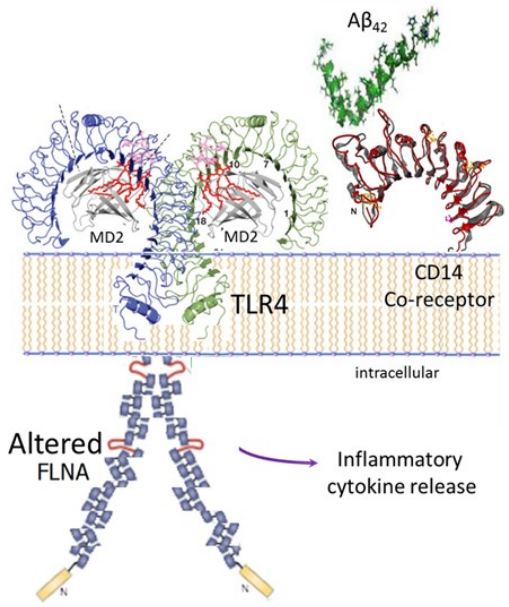
Altered FLNA links to toll-like receptor 4 (TLR4)

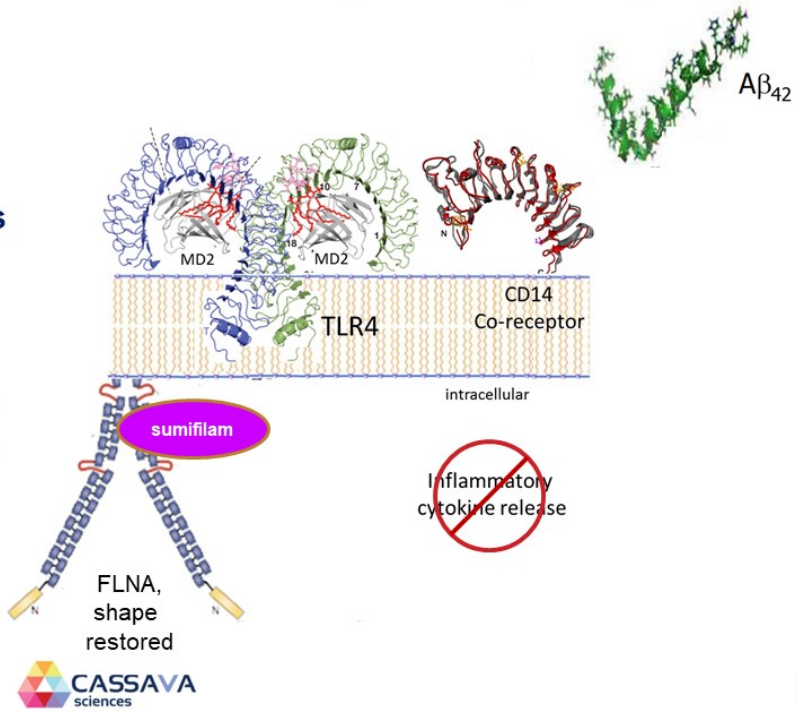
- **Altered FLNA linkage to TLR4 enables $A\beta_{42}$ to activate TLR4.**
- **Persistent TLR4 activation results in chronic neuroinflammation.**



Altered FLNA links to toll-like receptor 4 (TLR4)

- **Altered FLNA linkage to TLR4 enables $A\beta_{42}$ to activate TLR4.**
- **Persistent TLR4 activation results in chronic neuroinflammation.**

Sumifilam binds altered FLNA, restores its normal shape, stops $A\beta_{42}$-induced neuroinflammation.



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Summary of Preclinical Effects

Sumifilam	Intracerebro-ventricular (ICV) $A\beta_{42}$ infusion mouse model	Triple transgenic AD mouse model	Postmortem human AD brain tissue	Postmortem human age-matched control brain tissue treated with $A\beta_{42}$ in vitro
Reduced FLNA linkage to α7nAChR/TLR4	√	√	√	√
Reduced $A\beta_{42}$ bound to α7nAChR	√	√	√	√
Reduced amyloid deposits and NFTs	√	√	–	–
Reduced tau hyperphosphorylation	√	√	–	√
Improved function of α7nAChR, NMDAR and insulin receptors	√	√	√	√
Improved synaptic plasticity (activity-dependent Arc expression)	–	√	–	√
Reduced inflammatory cytokine levels	√	√	–	–
Improved cognition/behavior	–	√	–	–

CTAD
Clinical Trials on Alzheimer's Disease

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Phase 2b Clinical Study of Sumifilam

I. Mechanism of Action

II. Phase 2b Study Results

III. Study Conclusions

Hypothesis and Objective

Clinical Hypothesis

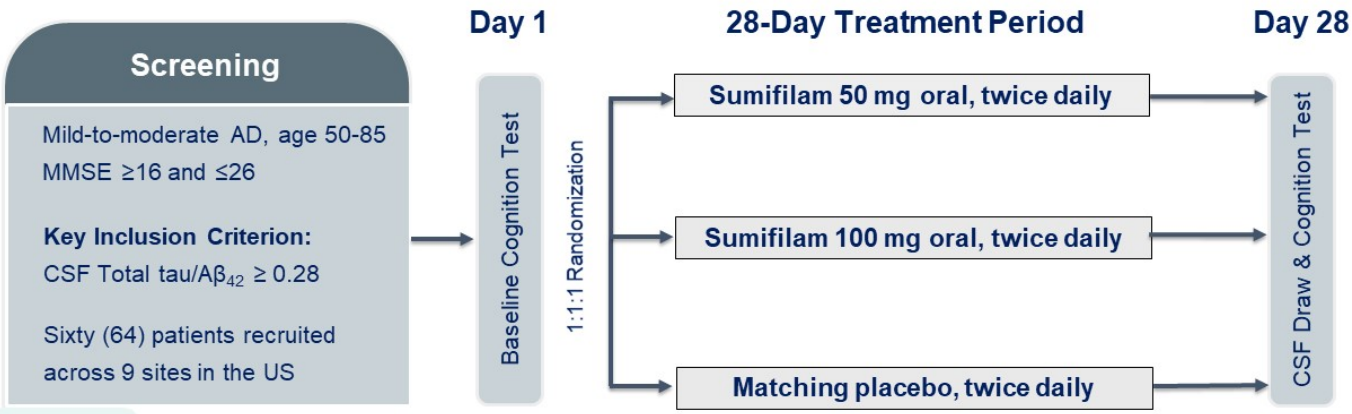
Published preclinical effects and mechanism of action suggest sumifilam is a disease-modifying drug for AD and may enhance cognition.

Phase 2b Study Objective

Evaluate safety, biomarkers and cognition in a well-controlled study of sumifilam.

Phase 2b Study Design

Randomized, placebo-controlled, multi-center, multi-dose study.



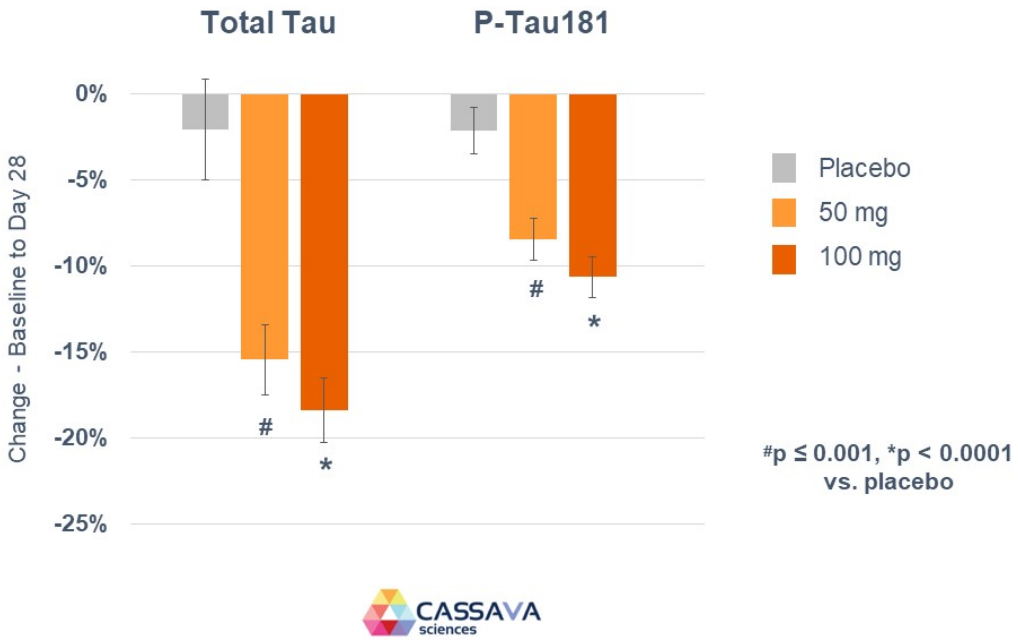
Primary Endpoint: Biomarkers of disease

Secondary Endpoint: Cognition

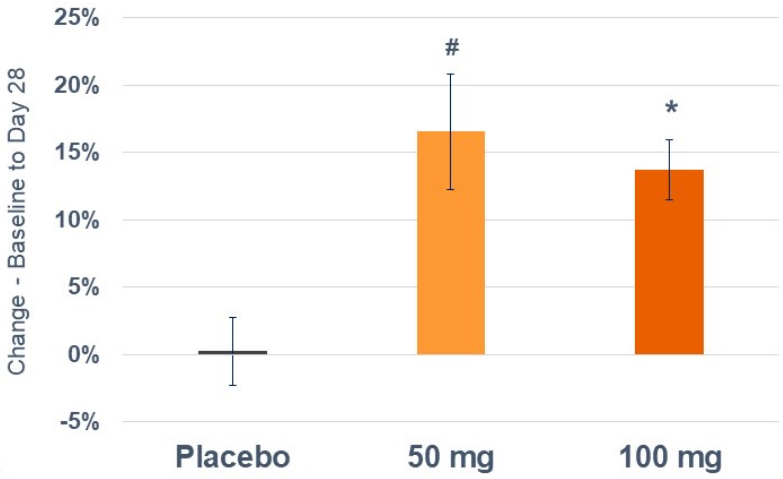
Phase 2b – Safety

- **Sumifilam was well-tolerated**
- **No serious adverse events**
- **No drug-related patient discontinuation**
- **No drug-related adverse events**
  - Common, non-persistent AEs observed in both placebo & drug groups.

Phase 2b Results – CSF Total Tau and P-Tau181 Decreased

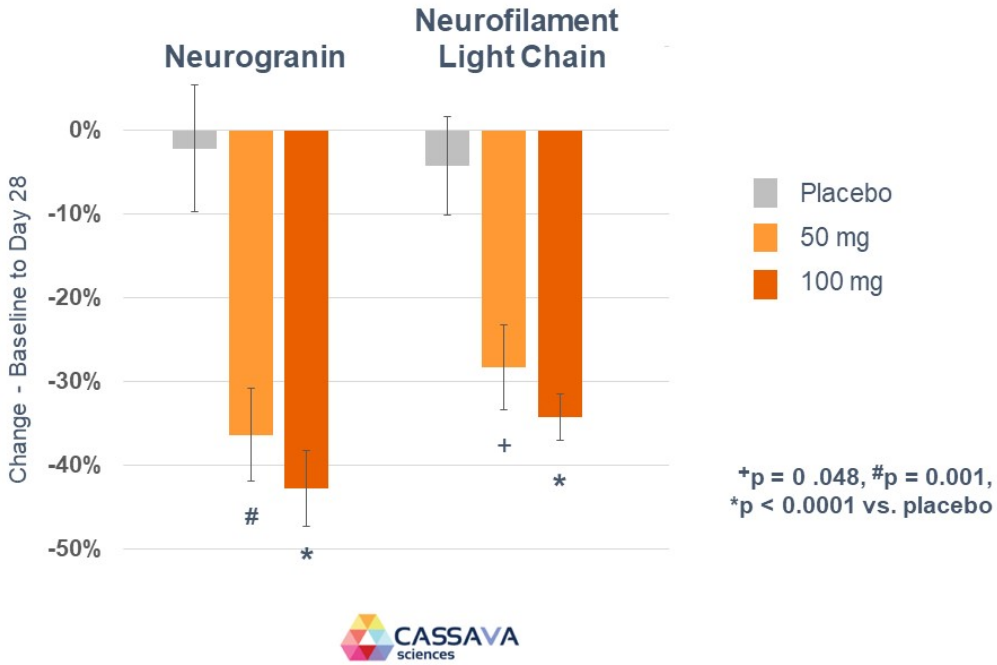


Phase 2b Results – CSF $A\beta_{42}$, Low in AD, Increased



#p = 0.0015, *p = 0.0003 vs. placebo

Phase 2b Results - Neurodegeneration Biomarkers in CSF Decreased



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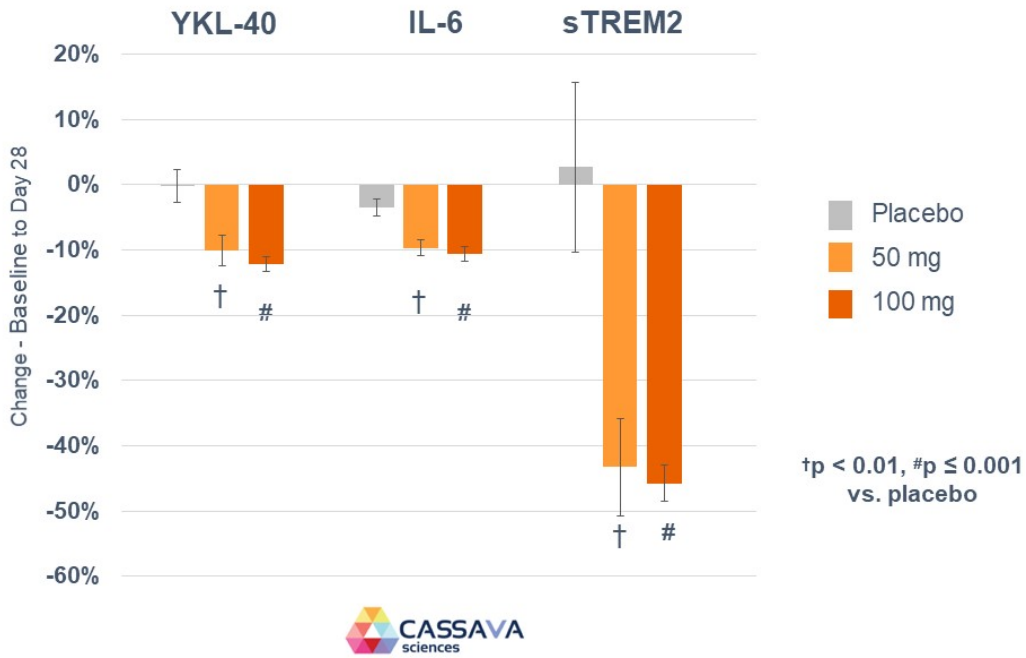
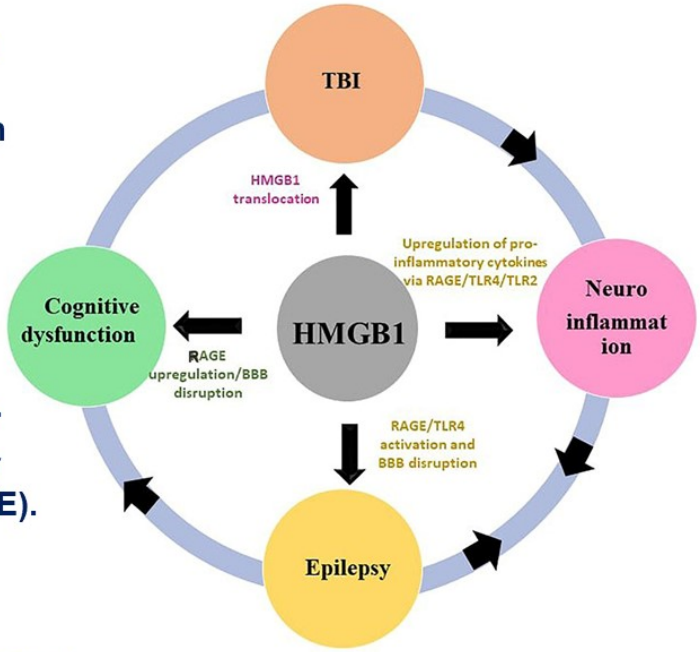
Phase 2b Results - Neuroinflammation Biomarkers in CSF Decreased



Elevated Levels of HMGB1 Trigger Loss of Neurons

- **High-Mobility Group Box 1 (HMGB1) is a pathogenic protein that induces neuroinflammation, neurite degeneration and cell death.**
  - Actively secreted by glia; released by necrotic cells.
  - Induces cytokine production, activates immune cells, stimulates auto-antibodies, regulates gene transcription.
  - Described as a 'danger molecule.'
- **HMGB1 elevated in AD, other diseases of neuroinflammation and neuronal loss.**
- **HMGB1 activates TLR4 and Receptor for Advanced Glycation End products (RAGE).**



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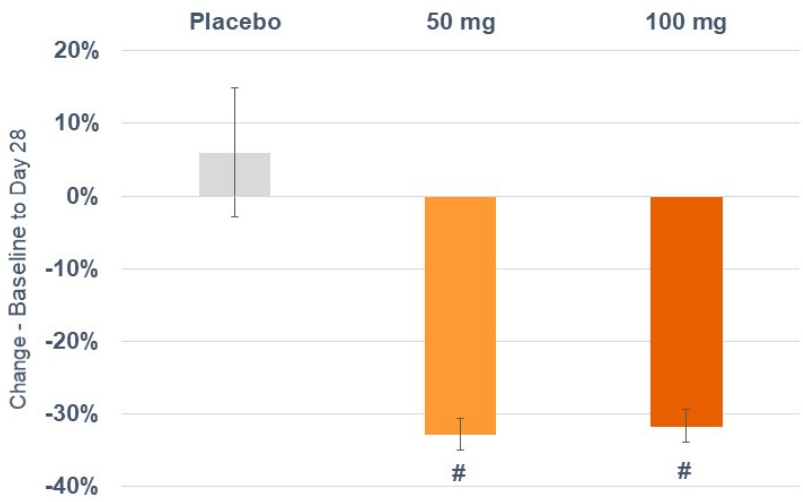
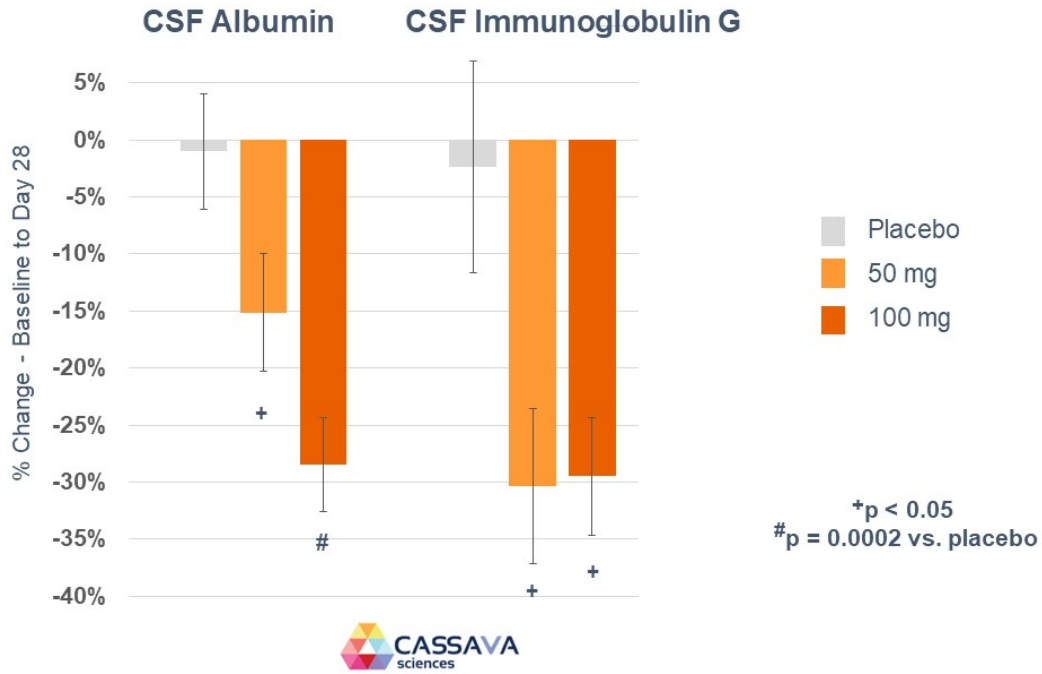
Phase 2b Results – CSF HMGB1 Decreased

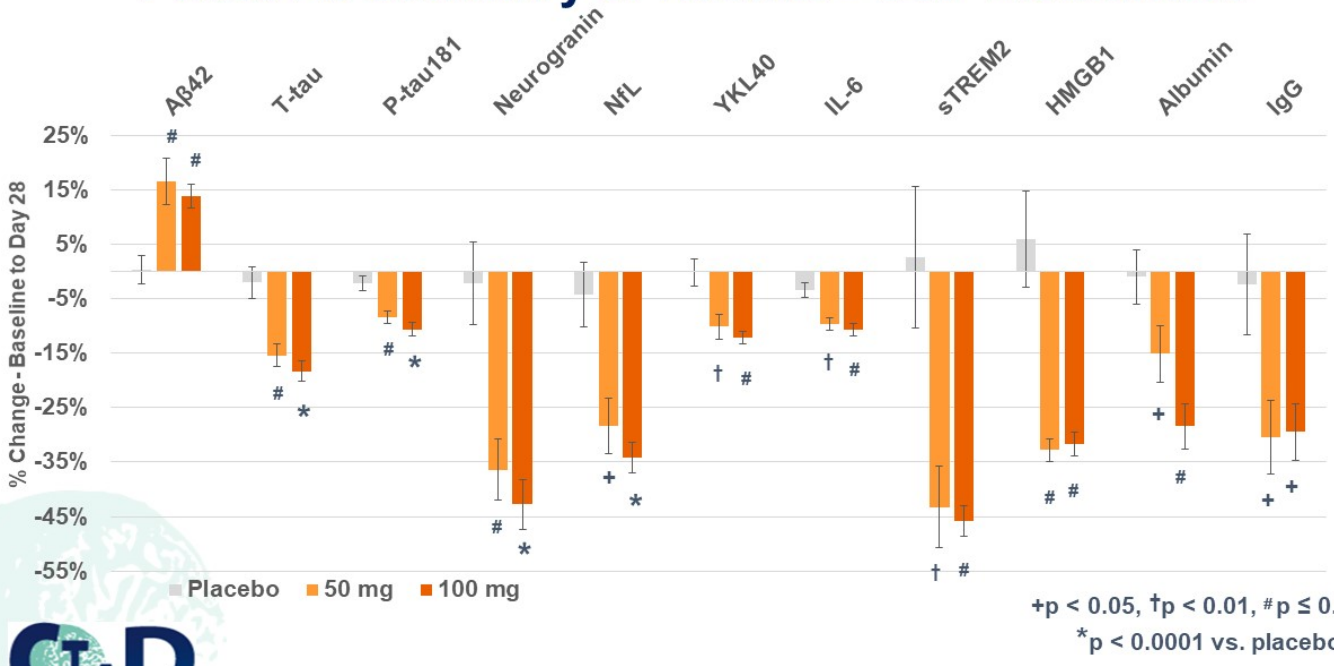


# p < 0.001 vs. placebo

Phase 2b Results - BBB Integrity Improved



Phase 2b Summary of Results - CSF Biomarkers



+p < 0.05, †p < 0.01, #p ≤ 0.001,
*p < 0.0001 vs. placebo

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Cognition

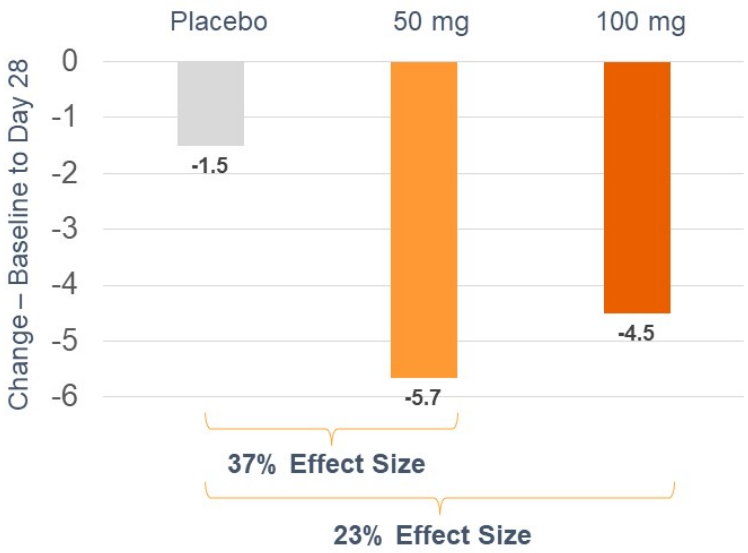
- **CANTAB (Cambridge Neuropsychological Test Automated Battery) is a widely used, computer-based battery of memory tests sensitive to subtle changes in cognition.**
  - Tests are independent of language skills, speed, gender or education.

- **Cognitive assessments were made on Day 1 (pre-dose) and again on Day 28.**

- **Patients were tested on 'Episodic Memory' and 'Spatial Working Memory'.**
  - Patients advance through progressively more difficult levels.
  - Outcome measure = total errors, with errors imputed for more difficult levels not reached, so…….

Lower score is better!

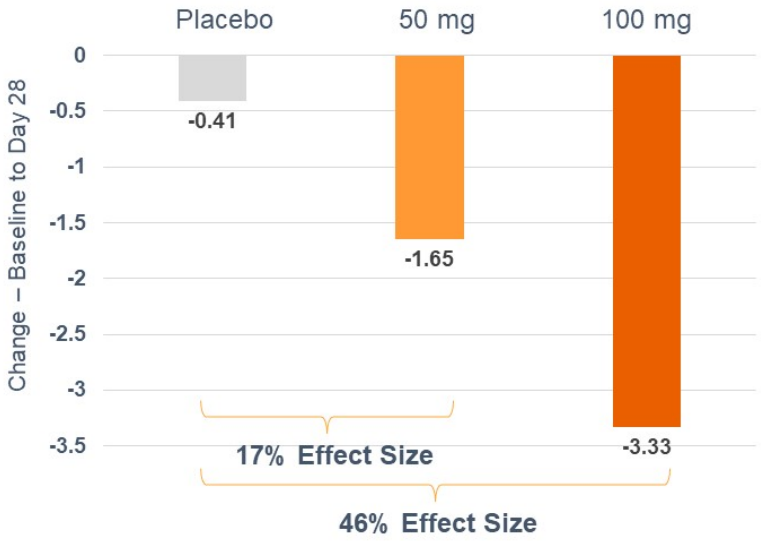
Phase 2b Results - Episodic Memory



Episodic Memory Endpoint:

Lower score is better in Total Errors on Paired Associates Learning (PAL).

Phase 2b Results - Spatial Working Memory

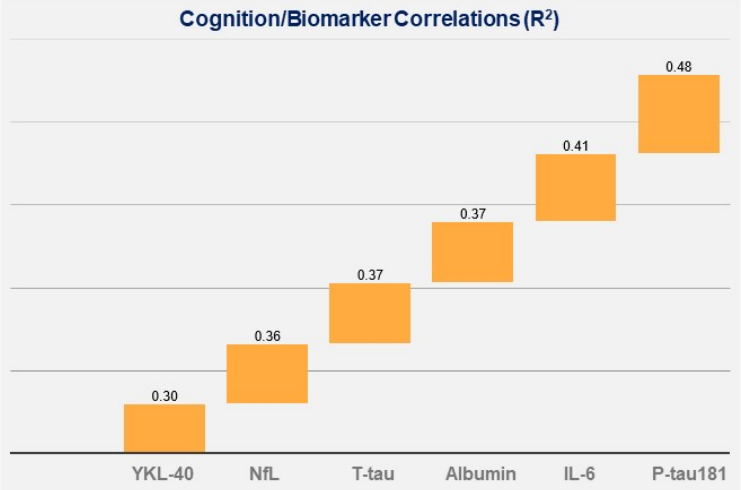


Spatial Working Memory Endpoint:

Lower score is better in Total Errors on Spatial Working Memory task.

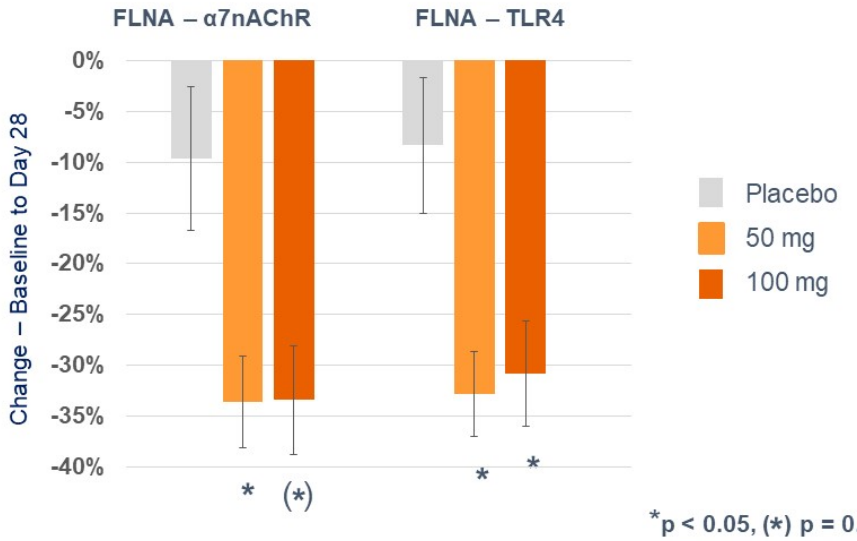
Phase 2b Results - Cognition/Biomarker Correlation

Cognitive Improvement Correlates Most (R^2 = 0.5) with Decreases in CSF P-tau181



Phase 2b Results - Target Engagement in Lymphocytes

Reduced FLNA linkages:



*p < 0.05, (*) p = 0.076 vs placebo

Phase 2b Results - Summary of Drug Effects

- **98% of patients treated with sumifilam 50 mg or 100 mg b.i.d. for 28 days showed improvements in validated biomarkers of AD pathology; neuroinflammation; and neurodegeneration; with no safety issues.**
- **Sumifilam appears to enhance cognition.**
  - 37% and 23% effect sizes in Episodic Memory vs. placebo
  - 17% and 46% effect sizes in Spatial Working Memory vs. placebo
  - Improved cognition correlated most strongly with reduction in levels of P-tau^{181} (R^2 = 0.5)
- **Target engagement and mechanism of action were demonstrated in this Phase 2b and in prior clinical and pre-clinical studies.**

CTAD Clinical Trials on Alzheimer's Disease

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Phase 2b Clinical Study of Sumifilam

I. Mechanism of Action

II. Phase 2b Study Results

III. Study Conclusions

Study Conclusions

- A well-controlled study of sumifilam showed promising treatment effects in mild-to-moderate AD patients.
- Sumifilam improved an entire panel of validated biomarkers of disease, neuroinflammation, and BBB integrity, and appeared to enhance cognition.
- Phase 2b treatment effects replicate prior clinical results and are consistent with published preclinical data and the drug's mechanism of action.

These data highlight sumifilam's potential as a disease-modifying treatment for Alzheimer's disease.

Next Steps

- On-going: *One-year, open-label safety study of sumifilam*
  - Enrolling 100 patients with mild-to-moderate AD, including ADAS-Cog assessments.

- Planned for 2021: *Large-scale safety & efficacy study of sumifilam*

THANK YOU!

CASSAVA sciences

Cassava Sciences Announces Additional Clinical Data from a Phase 2b Study of Sumifilam in Alzheimer's Disease

Alzheimer's Patients Treated with Sumifilam Showed a Statistically Significant ($p<0.001$) Treatment Benefit on HMGB1, a Protein that Triggers Neuroinflammation and Loss of Neurons

Alzheimer's Patients Treated with Sumifilam Also Showed a Treatment Benefit ($p<0.05$) on Blood-brain Barrier Integrity

Clinical Dataset to be Presented November 7th at CTAD 2020 Conference

AUSTIN, TX – November 4, 2020 – Cassava Sciences, Inc. (Nasdaq: SAVA) today announced additional clinical data of a Phase 2b study with sumifilam, its lead drug candidate, in patients with Alzheimer's disease. In a clinical study funded by the National Institutes of Health (NIH), sumifilam decreased levels of a protein called HMGB1 and improved measurements of the integrity of the blood-brain barrier (BBB). The ability of a drug candidate to decrease HMGB1 and improve BBB integrity in patients with Alzheimer's disease has not been previously reported in the science literature. Sumifilam is a proprietary, small molecule (oral) drug that restores the normal shape and function of altered filamin A protein in the brain.

"The ability to improve multiple biomarkers of disease with one drug is a unique achievement," said Remi Barbier, President & CEO of Cassava Sciences. "We believe these exciting clinical results create a time of rapid strategic momentum for the Company, to include development plans to evaluate sumifilam in a Phase 3 clinical program in patients with Alzheimer's disease."

Additional Phase 2b Study Results

Additional clinical data include changes in levels of HMGB1 protein and measurements of the integrity of the blood-brain barrier from baseline to Day 28 (all p-values versus placebo):

Sumifilam Significantly Reduced Levels of HMGB1 in Cerebrospinal Fluid (CSF):

- HMGB1 decreased 33% ($p<0.001$) in patients treated with 50 mg sumifilam
- HMGB1 decreased 32% ($p<0.001$) in patients treated with 100 mg sumifilam

Sumifilam Significantly Improved the Integrity of the Blood-brain Barrier (BBB):

- CSF IgG decreased 30% ($p<0.05$) in patients treated with 50 mg sumifilam
- CSF IgG decreased 30% ($p<0.05$) in patients treated with 100 mg sumifilam
- CSF albumin decreased 15% ($p<0.05$) in patients treated with 50 mg sumifilam
- CSF albumin decreased 28% ($p<0.05$) in patients treated with 100 mg sumifilam

Sumifilam Improved the Albumin Ratio, a Test of Blood-brain Barrier (BBB) Permeability:

- BBB permeability can be clinically evaluated by comparing levels of albumin in CSF and plasma. The albumin ratio is a test for BBB permeability because albumin protein is not synthesized in CSF. Hence, albumin in CSF necessarily comes from plasma through the BBB. The albumin ratio is frequently elevated in patients with dementia and various other disorders.

- In the Phase 2b study, the albumin ratio was unchanged for Alzheimer's patients on placebo. The albumin ratio improved by approximately 5 and 7 points for patients treated with sumifilam, 50 mg and 100 mg, respectively, over 28 days.

Changes in the Albumin Ratio by Treatment Group

Treatment	Day 0	Day 28	Change-Day 0 to 28
Placebo	24	24	No change
50 mg sumifilam	25	20	- 5
100 mg sumifilam	25	18	- 7

About HMGB1

HMGB1 is an endogenous and potent pro-inflammatory protein that is sometimes called a 'danger molecule.' HMGB1 is elevated in patients with Alzheimer's disease and other neurodegenerative disorders and many other disorders. Elevated levels of HMGB1 induce neuroinflammation, tissue damage and, eventually, cell death. Preclinical research has shown that inhibiting HMGB1 improves outcomes in neurodegenerative disease models, decreasing neuroinflammation and improving learning and memory. PubMed® reports nearly 6,000 scientific publications of HMGB1 research in the past decade, highlighting the molecule's potential importance in clinical research.

About the Blood-brain Barrier (BBB)

The BBB is a complex border of cells along blood vessels that prevent unwanted substances in blood from entering the brain. BBB integrity is essential to brain health. A healthy BBB system selectively allows the passage of some molecules into the brain, such as water and glucose, while blocking passage to molecules that may damage the brain. Levels of IgG antibodies and albumin protein in the cerebrospinal fluid (CSF) are an index of BBB integrity, with elevated levels evidence of a 'leaky', impaired BBB. Albumin protein is found at high levels in plasma and low levels in CSF because it does not normally cross the BBB.

Late-breaking Presentation at CTAD 2020

Clinical results of a Phase 2b study of sumifilam were selected as a late-breaking oral presentation by the 13th international conference on Clinical Trials on Alzheimer's Disease (CTAD). CTAD is a prestigious annual conference focused on Alzheimer's clinical research and takes place this year as a virtual event on November 4-7th, 2020.

On Saturday, November 7th, Company scientists will present an oral presentation titled, *"Sumifilam Significantly Improves Eleven CSF Biomarkers in a Randomized, Placebo-Controlled, One-Month Clinical Trial in Alzheimer's Disease Patients."*

The Company's CTAD presentation is available on its website: https://www.CassavaSciences.com

Phase 2b Study Design

Phase 2b was a double-blind, randomized, placebo-controlled, multi-center clinical study of sumifilam (formerly, PTI-125). Sixty-four patients with mild-to-moderate Alzheimer's disease, age 50-85, were randomized (1:1:1) to 100 mg or 50 mg oral sumifilam or matching placebo. Treatment was administered twice daily for 28 days. Nine U.S. study sites enrolled patients. A clinical diagnosis of Alzheimer's disease was confirmed with the Mini-Mental State Examination (MMSE) $\geq$16 to $\leq$26 and a CSF T-tau/Aβ_{42} ratio $\geq$0.28. Safety was assessed by ECGs, clinical labs, adverse event monitoring and physical examinations.

Previously Announced Clinical Phase 2b Data

As previously announced in September 2020, sumifilam was safe and well-tolerated, with no drug-related patient discontinuations. Alzheimer's patients treated with 50 mg or 100 mg sumifilam twice-daily for 28 days showed statistically significant ($p<0.05$) improvements in eight biomarkers of disease pathology, neurodegeneration and neuroinflammation, versus Alzheimer's patients who took placebo. In addition, Alzheimer's patients treated with sumifilam showed improvements in validated tests of Episodic Memory and Spatial Working Memory, versus patients who took placebo (Effect Sizes 17-46%). Cognitive improvements correlated most strongly (R^2=0.5) with decreases in P-tau181, a biomarker that leads to tangles in the brain. Sumifilam decreased brain levels of Ptau-181 by 8-11%, versus placebo. The study achieved a 98% response rate, defined as the proportion of study participants taking sumifilam who showed improvements in biomarkers.

On-going Open-label Study

In March 2020, we announced the initiation of an open-label study to evaluate sumifilam in patients with Alzheimer's disease. This is an open-label, multi-center, extension study to monitor the long-term safety and tolerability of sumifilam 100 mg twice-daily for 12 months. The study's target enrollment is approximately 100 patients with mild-to-moderate Alzheimer's disease. This study has exceeded 60% enrollment. The open-label study employs *The Alzheimer's Disease Assessment Scale-Cognitive Subscale* (ADAS-Cog-11) to assess cognitive symptoms of dementia and *The Neuropsychiatric Index* (NPI) to assess behavioral symptoms. Cassava Sciences plans to announce results of an interim analysis as additional safety and cognition data is collected from patients enrolled in the open-label study.

About Alzheimer's Disease

Alzheimer's disease is a progressive brain disorder that destroys memory and thinking skills. Currently, there are no drug therapies to halt Alzheimer's disease, much less reverse its course. In the U.S. alone, approximately 5.8 million people are currently living with Alzheimer's disease, and approximately 487,000 people age 65 or older developed Alzheimer's in 2019.[1] The number of people living with Alzheimer's disease is expected to grow dramatically in the years ahead, resulting in a growing social and economic burden.[2]

About Sumifilam

Sumifilam is a proprietary, small molecule (oral) drug that restores the normal shape and function of altered filamin A (FLNA), a scaffolding protein, in the brain. Altered FLNA in the brain disrupts the normal function of neurons, leading to Alzheimer's pathology, neurodegeneration and neuroinflammation. The underlying science for sumifilam is published in peer-reviewed journals, including *Journal of Neuroscience*, *Neurobiology of Aging*, *Journal of Biological Chemistry, Neuroimmunology and Neuroinflammation* and *Journal of Prevention of Alzheimer's Disease.* The Company is also developing an investigational diagnostic, called SavaDx, to detect Alzheimer's disease with a simple blood test.

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1,2 Source: Alzheimer's Association. *2019 Alzheimer's Disease Facts and Figures*. Available online at: https://www.alz.org/media/documents/alzheimers-facts-and-figures-2019-r.pdf

Sumifilam and SavaDx were both developed in-house. Both product candidates are substantially funded by peer-review research grant awards from the National Institutes of Health (NIH). Cassava Sciences owns worldwide development and commercial rights to its research programs in Alzheimer's disease, and related technologies, without royalty obligations to any third party. Patent protection in this area runs through 2037, plus extensions, and includes seven issued patents and related patent filings and applications.

About Cassava Sciences, Inc.

Cassava Sciences' mission is to discover and develop innovations for chronic, neurodegenerative conditions. Over the past 10 years, Cassava Sciences has combined state-of-the-art technology with new insights in neurobiology to develop novel solutions for Alzheimer's disease. For more information, please visit: https://www.CassavaSciences.com

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Cautionary Note Regarding Forward-Looking Statements: *This press release contains "forward-looking statements" for purposes of the Private Securities Litigation Reform Act of 1995 (the Act). Cassava Sciences claims the protection of the Safe Harbor for forward-looking statements contained in the Act. All statements other than statements of historical fact contained in this press release including, but not limited to, statements regarding the status of current and future clinical studies with sumifilam, including our intention to conduct a Phase 3 clinical program; the interpretation of results of our Phase 2 clinical studies including cognition data; plans to announce results of an interim analysis of an ongoing open-label study; potential health benefits, if any, of changes in levels of biomarkers; verbal commentaries made by Cassava Sciences' employees; and potential benefits, if any, of the Company's product candidates for Alzheimer's disease are forward-looking statements. Such statements are based largely on the Company's current expectations and projections about future events. Such statements speak only as of the date of this press release and are subject to a number of risks, uncertainties and assumptions, including, but not limited to, those risks relating to the ability to conduct or complete clinical studies on expected timelines, to demonstrate the specificity, safety, efficacy or potential health benefits of our product candidates, the severity and duration of health care precautions given the COVID-19 pandemic, any unanticipated impacts of the pandemic on our business operations, and including those described in the section entitled "Risk Factors" in Cassava Sciences' Annual Report on Form 10-K for the year ended December 31, 2019 and future reports to be filed with the SEC. In light of these risks, uncertainties and assumptions, the forward-looking statements and events discussed in this press release are inherently uncertain and may not occur, and actual results could differ materially and adversely from those anticipated or implied in the forward-looking statements. Accordingly, you should not rely upon forward-looking statements as predictions of future events. Except as required by law, the Company disclaims any intention or responsibility for updating or revising any forward-looking statements contained in this press release. For further information regarding these and other risks related to our business, investors should consult our filings with the SEC, which are available on the SEC's website at www.sec.gov.*

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