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): August 6, 2024

CLENE INC.

(Exact name of registrant as specified in its charter)

Delaware	**001-39834**	**85-2828339**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

6550 South Millrock Drive, Suite G50 **Salt Lake City, Utah**	**84121**
(Address of principal executive offices)	(Zip Code)

(801) 676-9695

(Registrant's telephone number, including area code)

N/A

(Former name or former address, if changed since last report.)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ 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, $0.0001 par value	CLNN	The Nasdaq Capital Market
Warrants, to acquire one-fortieth of one share of Common Stock for $230.00 per share	CLNNW	The Nasdaq Capital Market

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.

In connection with the press release discussed under Item 8.01 in this Current Report on Form 8-K, on August 6, 2024, Clene Inc. (the "Company") released an updated corporate presentation (the "Corporate Presentation") on its website, invest.clene.com. A copy of the Corporate Presentation is furnished as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference. The Company plans to use its website to disseminate future updates to the Corporate Presentation and may not file or furnish a Current Report on Form 8-K alerting investors if the Corporate Presentation is updated.

The information furnished in this Item 7.01, including Exhibit 99.1, shall not be deemed to be "filed" for purposes of Section 18 of the Securities Exchange Act of 1934 (the "Exchange Act"), as amended, or otherwise subject to the liabilities of that section, and shall not be deemed to be incorporated by reference into any filing made by the Company under the Exchange Act or the Securities Act of 1933, as amended, regardless of any general incorporation language in any such filings, except as shall be expressly set forth by specific reference in such a filing.

Item 8.01 Other Events.

On August 6, 2024, the Company issued a press release announcing new CNM-Au8 biomarker and clinical efficacy data submitted to the U.S. Food and Drug Administration ("FDA") in support of treatment for amyotrophic lateral sclerosis ("ALS"). A copy of the press release is filed as Exhibit 99.2 to this Current Report on Form 8-K and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Exhibit Description
99.1	Corporate presentation.
99.2	Press release, dated August 6, 2024, announcing new CNM-Au8 biomarker and clinical efficacy data submitted to FDA in support of treatment for ALS.
104	Cover Page Interactive Data File (formatted as Inline XBRL).

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.

CLENE INC.

Date: August 6, 2024

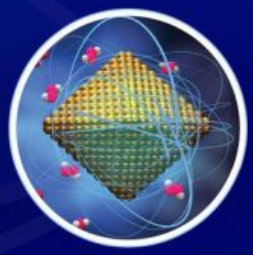
By: /s/ Robert Etherington
Robert Etherington
President and Chief Executive Officer

Exhibit 99.1

Forward Looking Statements

This presentation contains "forward-looking statements" within the meaning of Section 21E of the Securities Exchange Act of 1934, as amended, and Section 27A of the Securities Act of 1933, as amended, which are intended to be covered by the "safe harbor" provisions created by those laws. Clene's forward-looking statements include, but are not limited to, statements regarding our or our management team's expectations, hopes, beliefs, intentions or strategies regarding our future operations. In addition, any statements that refer to projections, forecasts or other characterizations of future events or circumstances, including any underlying assumptions, are forward-looking statements. The words "anticipate," "believe," "contemplate," "continue," "estimate," "expect," "intends," "may," "might," "plan," "possible," "potential," "predict," "project," "should," "will," "would," and similar expressions may identify forward-looking statements, but the absence of these words does not mean that a statement is not forward-looking. These forward-looking statements represent our views as of the date of this presentation and involve a number of judgments, risks and uncertainties. We anticipate that subsequent events and developments will cause our views to change. We undertake no obligation to update forward-looking statements to reflect events or circumstances after the date they were made, whether as a result of new information, future events or otherwise, except as may be required under applicable securities laws. Accordingly, forward-looking statements should not be relied upon as representing our views as of any subsequent date. As a result of a number of known and unknown risks and uncertainties, our actual results or performance may be materially different from those expressed or implied by these forward-looking statements. Some factors that could cause actual results to differ include our substantial dependence on the successful commercialization of our drug candidates, if approved, in the future; our inability to maintain the listing of our common stock on Nasdaq; our significant net losses and net operating cash outflows; our ability to demonstrate the efficacy and safety of our drug candidates; the clinical results for our drug candidates, which may not support further development or marketing approval; actions of regulatory agencies, which may affect the initiation, timing and progress of clinical trials and marketing approval; our ability to achieve commercial success for our drug candidates, if approved; our ability to obtain and maintain protection of intellectual property for our technology and drugs; our reliance on third parties to conduct drug development, manufacturing and other services; our limited operating history and our ability to obtain additional funding for operations and to complete the licensing or development and commercialization of our drug candidates; the impact of the COVID-19 pandemic on our clinical development, commercial and other operations; changes in applicable laws or regulations; the effects of inflation; the effects of staffing and materials shortages; the possibility that we may be adversely affected by other economic, business, and/or competitive factors; and other risks and uncertainties set forth in "Risk Factors" in our most recent Annual Report on Form 10-K and any subsequent Quarterly Reports on Form 10-Q. In addition, statements that "we believe" and similar statements reflect our beliefs and opinions on the relevant subject. These statements are based upon information available to us as of the date of this presentation, and while we believe such information forms a reasonable basis for such statements, such information may be limited or incomplete, and our statements should not be read to indicate that we have conducted an exhaustive inquiry into, or review of, all potentially available relevant information. These statements are inherently uncertain and you are cautioned not to rely unduly upon these statements. All information in this presentation is as of the date of this presentation. The information contained in any website referenced herein is not, and shall not be deemed to be, part of or incorporated into this presentation.

Focused on Improving Mitochondrial Health and Protecting Neuronal Function to Treat Neurodegenerative Diseases



THE PROBLEM

- The World Health Organization predicts **neurodegenerative diseases will become the second-most prevalent cause of death** within the next 20 years.
- A therapeutic breakthrough is urgently needed.
- In neurodegenerative diseases**, impaired mitochondrial activity and compromised cellular metabolism can lead to neuronal death**.

A NEW APPROACH

- **Clene is pioneering catalytic nanotherapeutics** to treat neurodegenerative diseases, such as amyotrophic lateral sclerosis, Parkinson's disease and multiple sclerosis.
- By **targeting the improvement of mitochondrial function** via the nicotinamide adenine dinucleotide pathway, Clene's first-in-class drug, CNM-Au8, is **pioneering a new way to restore and protect neuronal function**.

clene NANOMEDICINE

Building the Clinical Case for Neuroprotection & Remyelination

HEALEY ALS Platform Trial

RESCUEALS

VISIONARY-MS STUDY

RepairPD

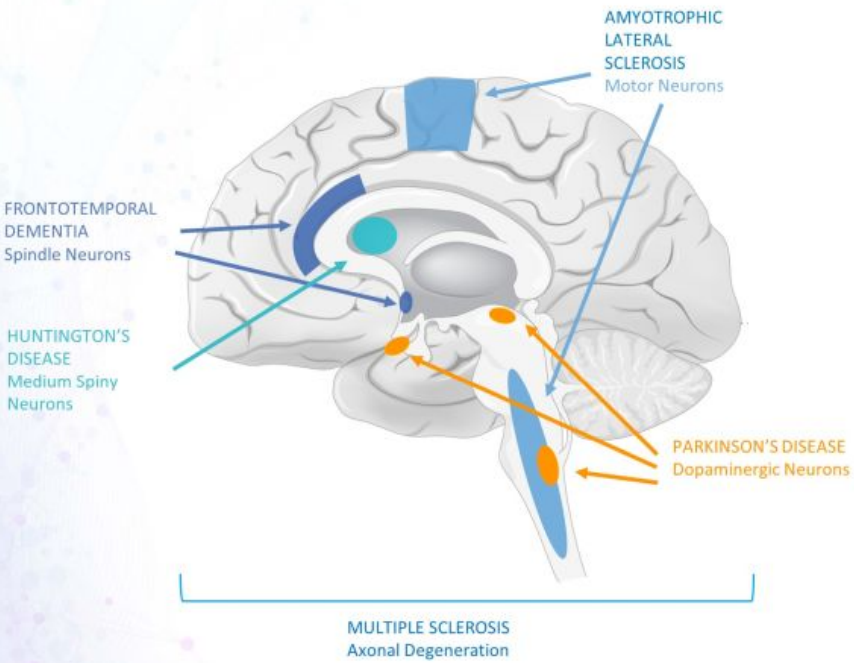
RepairMS

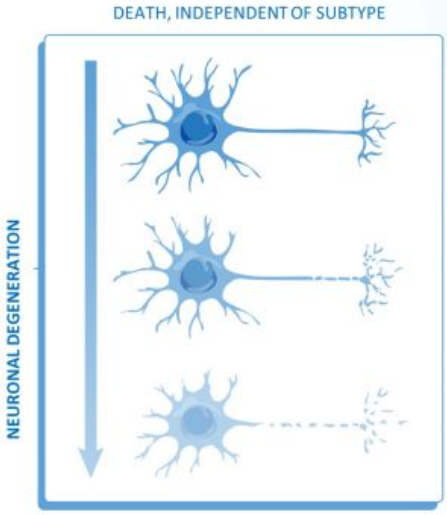
Growing Body of Clinical Evidence Across ALS and MS Supports CNM-Au8 Therapeutic Potential to Treat Neurodegenerative Diseases

Proprietary Nanotherapeutic Manufacturing
Strong IP: 150+ granted patents PLUS Trade Secrets

CLene NANOMEDICINE

All Neurodegenerative Diseases Involve Neuronal Death

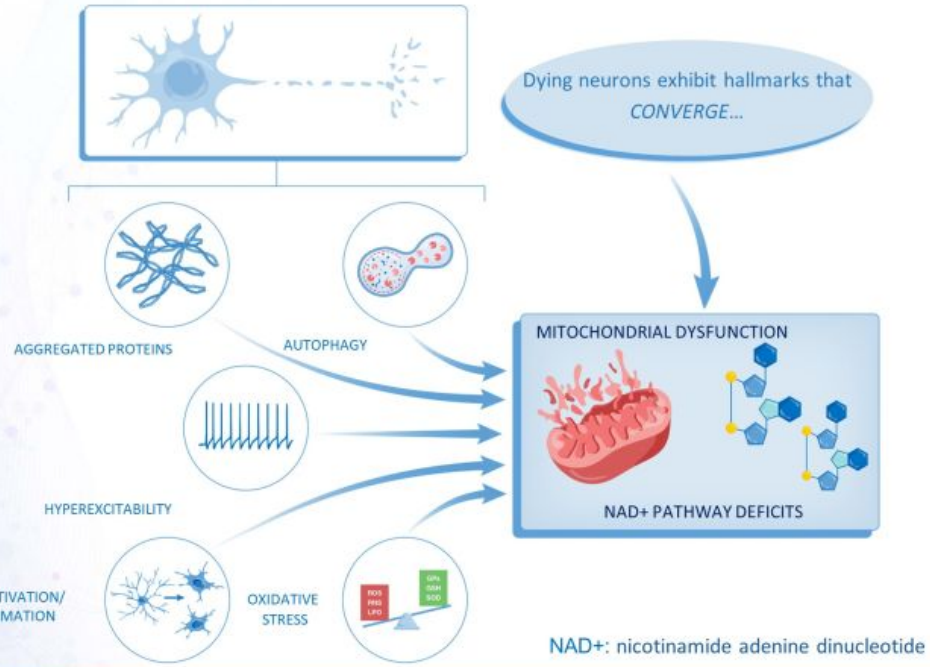




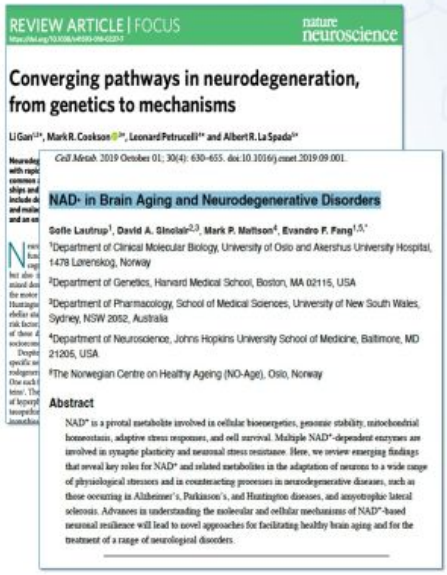
Fu, H., et al; Nature Neuroscience (2018) 21: 1350-1358. Rone et al. J Neurosci. 2016 Apr 27;36(17):4698-707.

CLene NANOMEDICINE

Hallmarks of Neuronal Death Converge on ***Mitochondrial Dysfunction*** and ***NAD+ Pathway Deficits***



NAD+: nicotinamide adenine dinucleotide



REVIEW ARTICLE | FOCUS

nature neuroscience

Converging pathways in neurodegeneration, from genetics to mechanisms

Li Gan[1,2*], Mark R. Cookson[3*], Leonard Petrucelli[4*] and Albert R. La Spada[5*]

Cell Metab. 2019 October 01; 30(4): 630–655. doi:10.1016/j.cmet.2019.09.001.

NAD+ in Brain Aging and Neurodegenerative Disorders

Sofie Lautrup[1], David A. Sinclair[2,3], Mark P. Mattson[4], Evandro F. Fang[1,5,*]

[1]Department of Clinical Molecular Biology, University of Oslo and Akershus University Hospital, 1478 Lørenskog, Norway

[2]Department of Genetics, Harvard Medical School, Boston, MA 02115, USA

[3]Department of Pharmacology, School of Medical Sciences, University of New South Wales, Sydney, NSW 2052, Australia

[4]Department of Neuroscience, Johns Hopkins University School of Medicine, Baltimore, MD 21205, USA

[5]The Norwegian Centre on Healthy Ageing (NO-Age), Oslo, Norway

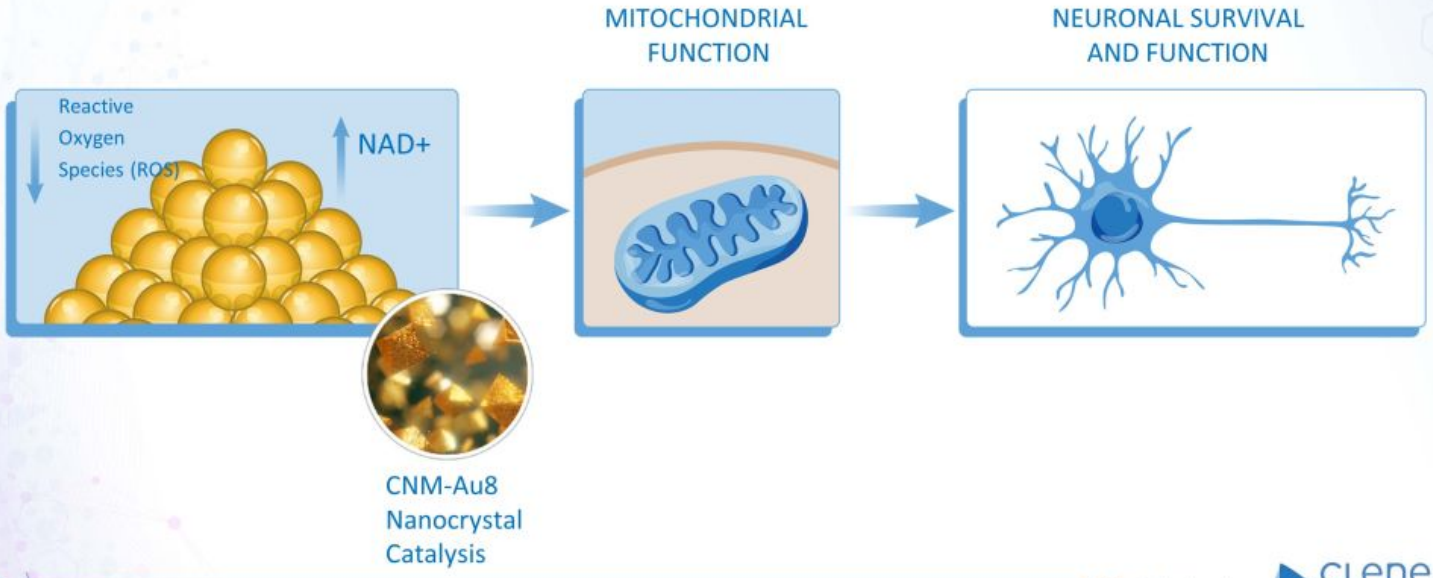
Abstract

NAD+ is a pivotal metabolite involved in cellular bioenergetics, genomic stability, mitochondrial homeostasis, adaptive stress responses, and cell survival. Multiple NAD+-dependent enzymes are involved in synaptic plasticity and neuronal stress resistance. Here, we review emerging findings that reveal key roles for NAD+ and related metabolites in the adaptation of neurons to a wide range of physiological stressors and in counteracting processes in neurodegenerative diseases, such as those occurring in Alzheimer's, Parkinson's, and Huntington diseases, and amyotrophic lateral sclerosis. Advances in understanding the molecular and cellular mechanisms of NAD+-based neuronal resilience will lead to novel approaches for facilitating healthy brain aging and for the treatment of a range of neurological disorders.

Lautrup et al. Cell Metab. 2019 Oct 1;30(4):630-655. Gan et al. Nat Neurosci. 2018 Oct;21(10):1300-1309.

CLene NANOMEDICINE

CNM-Au8® | Surface Catalysis Improves Mitochondrial Function



CNM-Au8 Nanocrystal Catalysis

Robinson et al. Sci Rep. 2020 Feb 11;10(1):1936. Wang et al 2023, Small. 2023 Sep 28:e2304082.

CLene NANOMEDICINE

Two REPAIR Trials Demonstrated Target Brain Engagement and Improved Energy Metabolism in Early PD and Stable Relapsing MS

Study Objective: Demonstrate target engagement & **Blood-Brain penetration** for CNM-Au8 on CNS biomarkers related to energetic effects in the brain using Magnetic Resonance Spectroscopy (^{31}P-MRS)

RepairPD
Early Parkinson's Disease

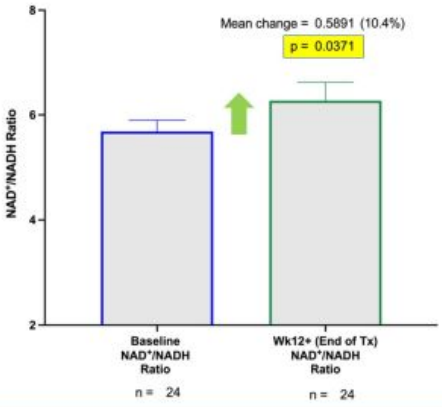
RepairMS
Stable Relapsing MS

RepairMS
Non-Active Progressive MS
(Ongoing)

Results demonstrated a potentially meaningful 10% improvement in NAD+/NADH ratio, an essential molecule for energy production[1]

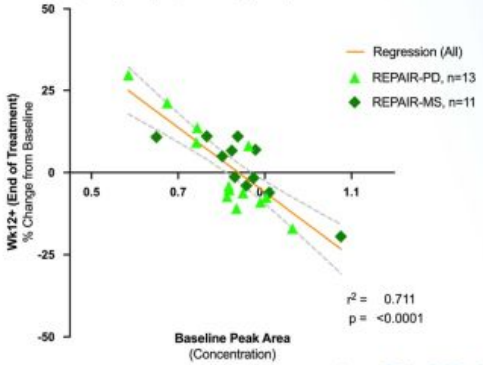
1° Endpoint (integrated PD & MS)[2]

^{31}P-MRS Change in Brain NAD$^+$/NADH Ratio at End of Treatment
Partial Volume Coil; Ratio of NAD$^+$/NADH (% Fraction of NAD$^+$/ % Fraction NADH)
Primary Endpoint, Mean ± SEM (Paired t-test)



Exploratory
(ATP Normalization)

REPAIR Integrated Analysis
^{31}P-MRS Change in β-ATP at End of Treatment
Full Volume Coil ^{31}P Signal Area (Integral)
Exploratory Endpoint, Percent (%) Change vs. Baseline Value



Ren et al. J Nanobiotechnology. 2023 Dec 13;21(1):478.

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Over 650 Years of Subject Exposure Without Identified Safety Signals Across ALS, MS, and PD

Clean Toxicology Findings

All Animal Toxicology Studies Resulted in No-Adverse Effect Level (NOAEL) Findings

- Multiple species up to 9-months treatment
- Up to maximum feasible dosing without any toxicology findings related to CNM-Au8

Well Tolerated Adverse Event (AE) Profile

Assessed as Predominantly Mild-to-Moderate Severity and Transient

- **No SAEs related to CNM-Au8** considered severe, life-threatening, or resulting in death
- **AEs transient/mild-to-moderate** severity (GI/Headache)

Patient Exposure Across ALS, MS & PD

Over 650 Years of Subject Exposure Without Identified Safety Signals

- Long-term dosing experience **over 4 years**

Data on File, Clene Nanomedicine, Inc. MS: Multiple Sclerosis, ALS: Amyotrophic lateral sclerosis, and PD: Parkinson's Disease.

Promising Evidence from Two Phase 2 Trials and Long-Term Data

CNM-Au8 Demonstrated Survival, Delayed Clinical Worsening, and Preserved Function

	RESCUE-ALS	RESCUE-OLE	HEALEY ALS Platform	HEALEY OLE	EAP
ALS Patient Demographics	Early-to-Mid-Stage (45)	Early-to-Mid-Stage	Mid-to-Late-Stage (161 Regimen C)	Mid-to-Late-Stage	Real-World Experience (256)
Duration	36-weeks	Up to 173 weeks	24-weeks	Up to 133 weeks	Over 4.0 years
Survival	--	✅	✅	✅ PRO-ACT	✅
Delayed Time to Clinical Worsening	✅	✅	✅	Pending data 1Q 2024	Not routinely collected
Preserved Function (ALSFRS-R)	--	✅	--		
Progression Biomarkers	p75 trend	↓ UCHL1 *	✅ NfL ↓	✅ NfL ↓	
Safety	>600 Years of Subject Exposure Without Identified Safety Signals Across ALS, MS, and PD				

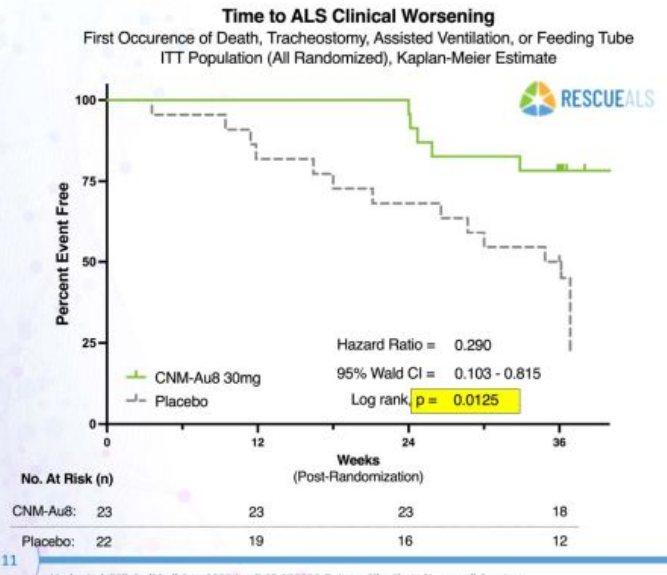
Consistent Evidence for CNM-Au8 30mg Dose Across Broad ALS Patient Population

* Data on File, Clene Nanomedicine, Inc. *Small sample available for analyses in RESCUE OLE

CNM-Au8 | Clinical Worsening Concordant in Two Phase 2 Trials

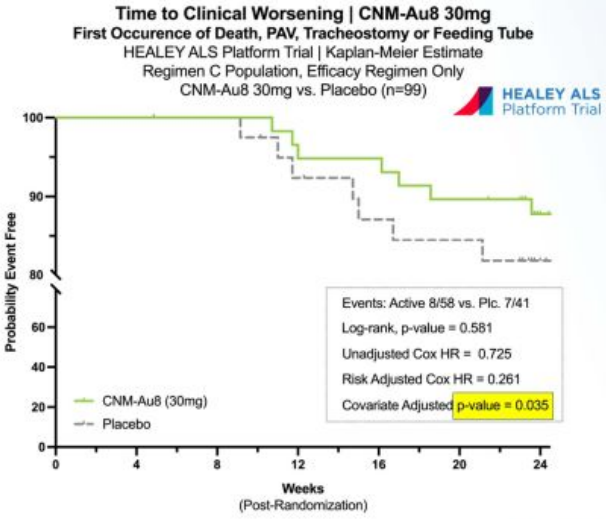
Evidence for Decreased Clinical Worsening Events Across Two Phase 2 Studies

Phase 2 RESCUE-ALS CNM-Au8 30mg
Decreased Time to Clinical Worsening



No. At Risk (n)				
CNM-Au8:	23	23	23	18
Placebo:	22	19	16	12

Phase 2 HEALEY ALS Platform CNM-Au8 30mg
Decreased Time to Clinical Worsening



Vucic et al. EClinicalMedicine. 2023 Jun 8;60:102036. Data on File, Clene Nanomedicine, Inc.

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CNM-Au8 | ALS Survival at 30mg Concordant in Two Phase 2 Trials

RESCUEALS

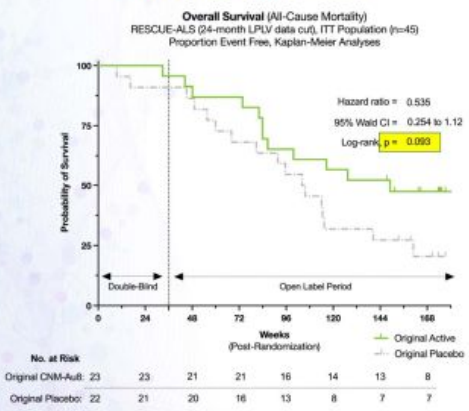
HEALEY ALS Platform Trial

Up to 75% decreased risk of death through 168 weeks

>90% risk reduction of death at 30mg at 24 weeks

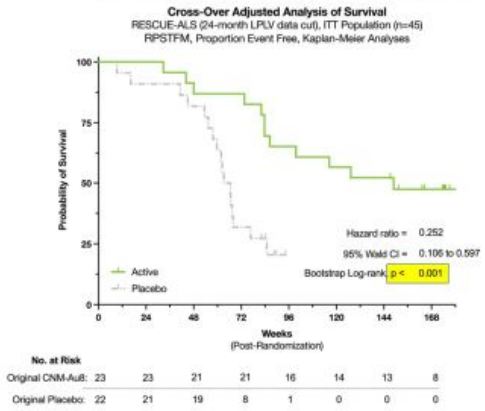
Unadjusted Survival

10.1 Months Survival Difference



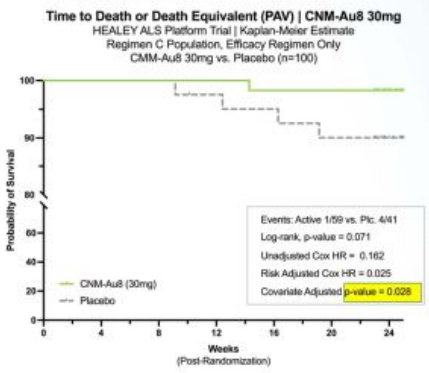
Cross-Over Adjusted Survival

Up to 19.3 Month Survival Benefit vs. Original Pbo



RPSFTM (Rank Preserving Structural Failure Time Model) removes estimated benefit from cross-over to active treatment in ex-placebo participants

Survival During Blinded Period



Data on File, Clene Nanomedicine, Inc.

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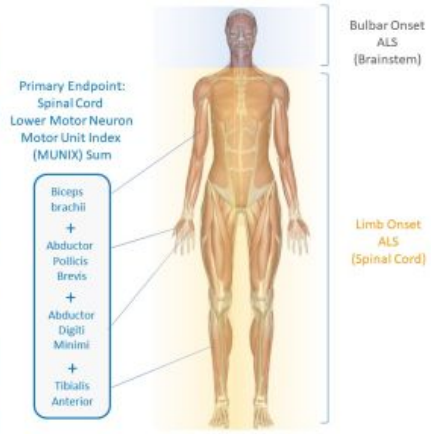
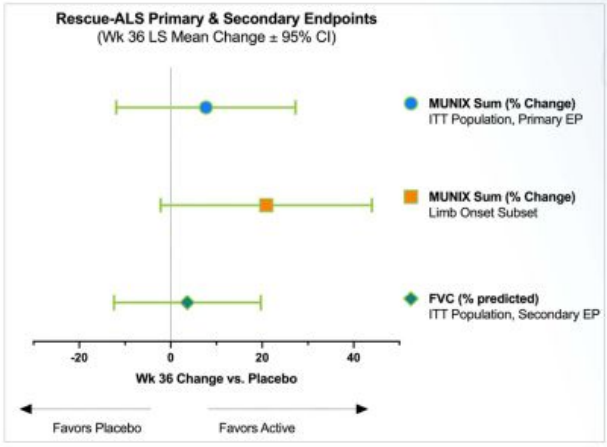
RESCUEALS Encouraging Efficacy Signals in Phase 2 Trial

Study Objective: Detect preservation of motor neuron function in people with early ALS as measured by MUNIX

Study Design:
36-week blinded treatment with ongoing long-term open-label follow-up



1° & 2° Endpoints



Results in favor of CNM-Au8 treatment but study underpowered

Vucic et al. RESCUE-ALS Trial Results: A Phase 2, Randomized, Double-Blind, Placebo-Controlled Study of CNM-Au8 to Slow Disease Progression in Amyotrophic Lateral Sclerosis; Presented at International Symposium on ALS/MND; 7-10 December 2021.)

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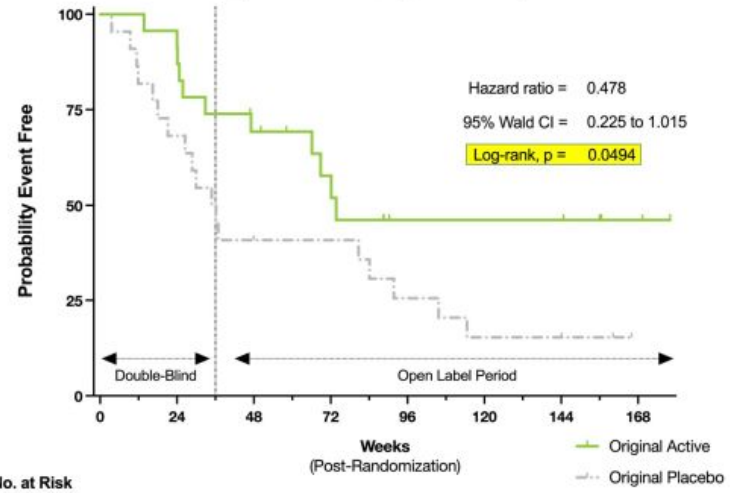
RESCUEALS OLE | 52% Reduced Risk of ALS Clinical Worsening

- Death
- Tracheostomy
- Non-Invasive Ventilation
- Feeding Tube Placement

ALS Clinical Worsening Events
Time to Death, Tracheostomy, Assisted Ventilation, or Feeding Tube
RESCUE-ALS Double-Blind & OLE Periods (24-month LPLV data cut)
Proportion Event Free, Kaplan-Meier Analyses



No. at Risk								
Original CNM-Au8:	23	23	16	11	6	6	6	3
Original Placebo:	22	16	10	9	6	4	4	1

52% decrease in risk of ALS clinical worsening for CNM-Au8 compared to placebo in OLE up to 168 weeks

Participants were right-censored at loss of follow-up with OLE withdrawal, as applicable

Data on File, Clene Nanomedicine, Inc.

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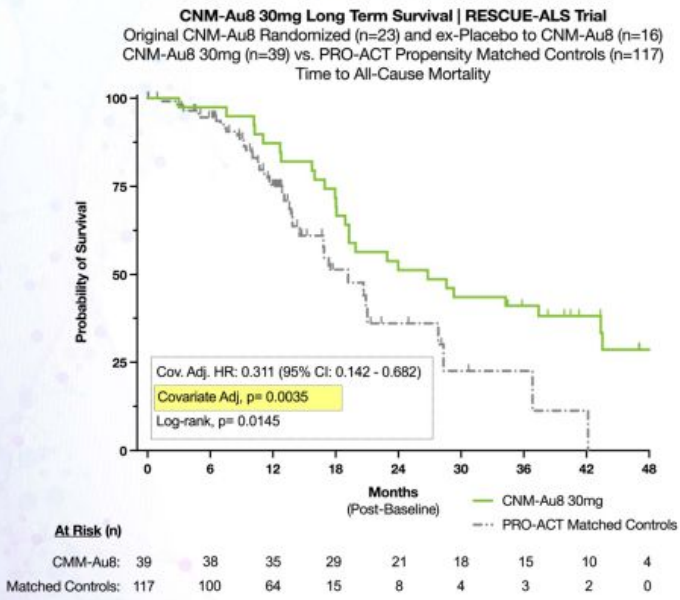
CNM-Au8 30mg Treatment Improved Long-Term Survival vs. Propensity Matched PRO-ACT & MiNDAUS (Australian) Controls

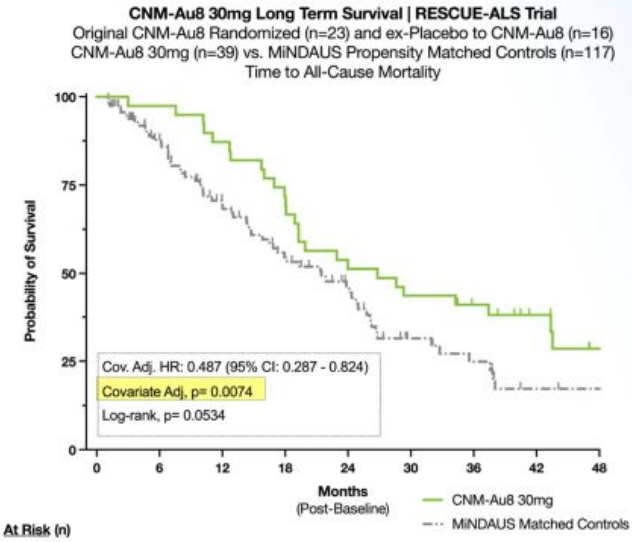
PRO-ACT Controls



At Risk (n)

CMM-Au8:	39	38	35	29	21	18	15	10	4
Matched Controls:	117	100	64	15	8	4	3	2	0

MiNDAUS Controls



At Risk (n)

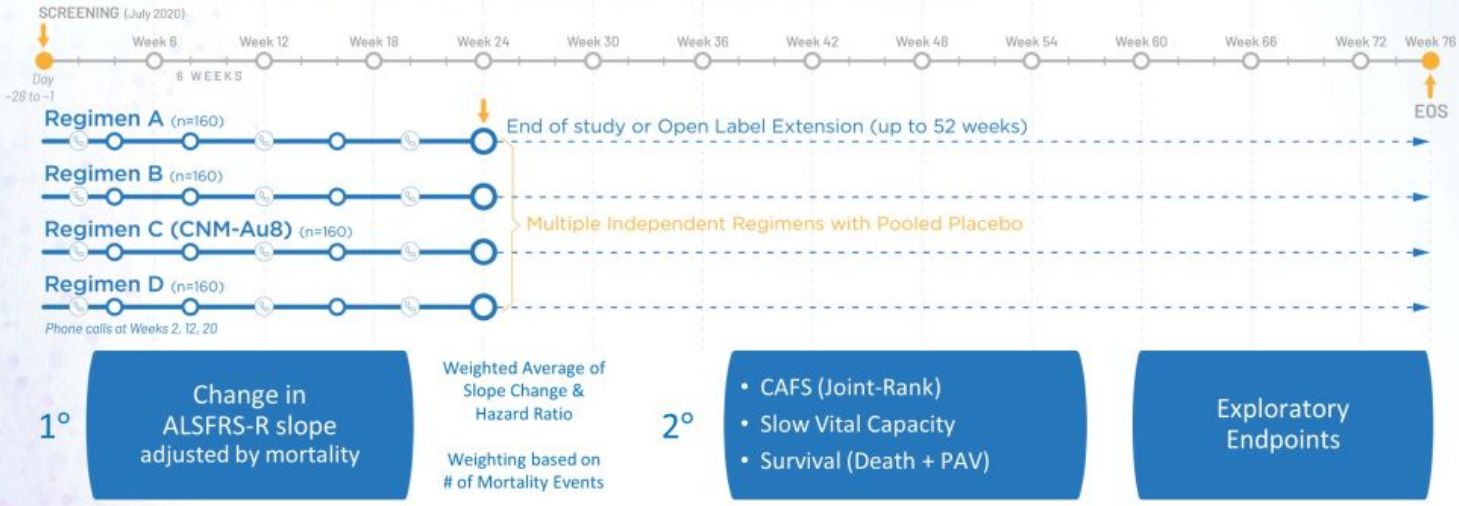
CMM-Au8:	39	38	35	29	21	18	15	10	4
Matched Controls:	117	87	60	43	30	16	12	6	5

Data on File, Clene Nanomedicine, Inc.

HEALEY ALS Platform Trial

A Multi-center, Randomized Double-Blind, Placebo-Controlled Clinical Trial Assessing the Efficacy, Safety, Tolerability, Pharmacokinetics, and Pharmacodynamics of CNM-Au8 in Participants with Amyotrophic Lateral Sclerosis

24-Week Treatment Period (3:1 randomization, 120 active [30mg, 60mg]: 40 placebo)



1° Change in ALSFRS-R slope adjusted by mortality

Weighted Average of Slope Change & Hazard Ratio

Weighting based on # of Mortality Events

2°
- CAFS (Joint-Rank)
- Slow Vital Capacity
- Survival (Death + PAV)

Exploratory Endpoints

No evidence for treatment effect at 24 weeks for either adjusted ALSFRS-R change, CAFS, or SVC (combined 30 & 60 mg doses)

Paganoni et al. Adaptive Platform Trials to Transform Amyotrophic Lateral Sclerosis Therapy Development. Ann Neurol. 2022; 91:165-175.

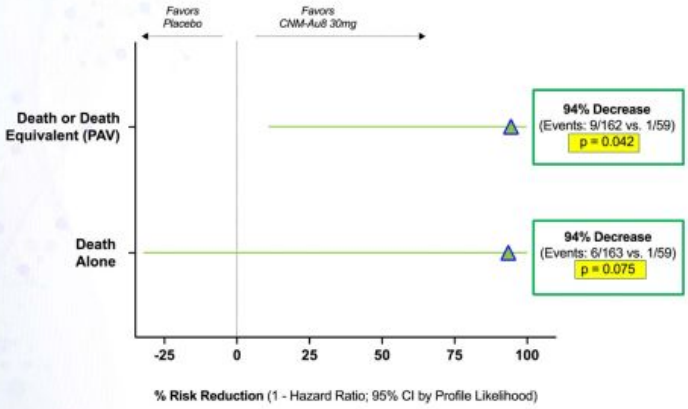
CLene NANOMEDICINE

Survival Signal | >90% Reduced Risk of Death with CNM-Au8 30mg

HEALEY ALS Platform Trial

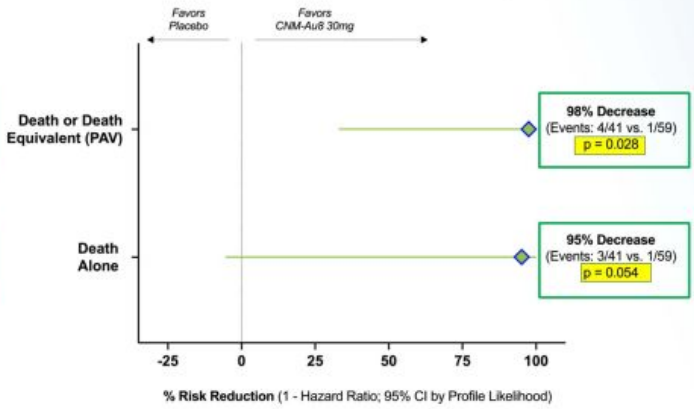
Shared Placebo Across Regimens

CNM-Au8 30mg Survival | Adjusted Cox Proportional Hazard
Full Analysis Set (All Shared Placebo, Regimens A, B, C, D)
% Hazard Reduction at Week 24
(1 - Hazard Ratio, 95% Confidence Interval)



CNM-Au8 Regimen Only (Regimen C)

CNM-Au8 30mg Survival | Adjusted Cox Proportional Hazard
Efficay Regimen Only Set (Within Regimen Analysis)
% Hazard Reduction at Week 24
(1 - Hazard Ratio, 95% Confidence Interval)



PAV = Permanently Assisted Ventilation; Prespecified covariate adjustments include: (i) time from symptom onset, (ii) pre-baseline ALSFRS-R slope, (iii) riluzole use, (iv) edaravone use, (v) age. Events (placebo vs. active). p-values are not adjusted for multiple comparisons; exploratory analyses by dose.

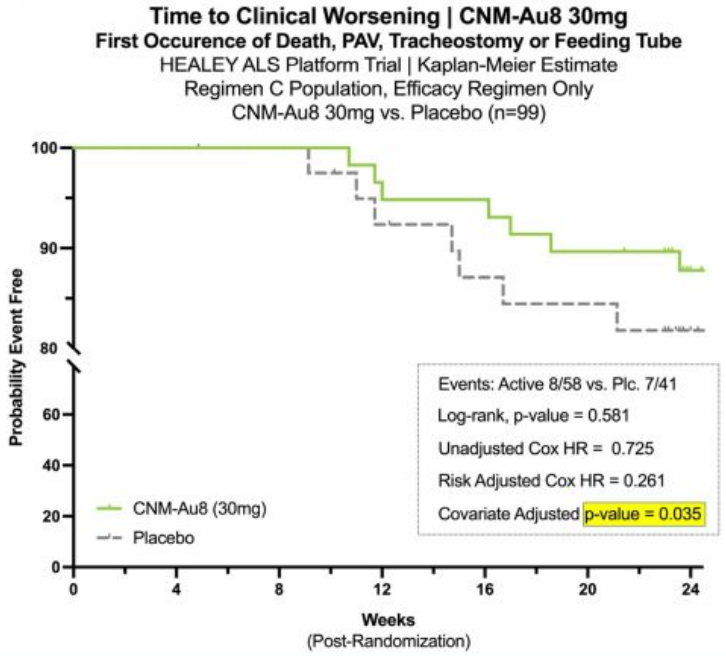
Data on File, Clene Nanomedicine, Inc.

clene NANOMEDICINE

Delayed Time to ALS Clinical Worsening

CNM-Au8 30mg | Within Regimen Analysis (Primary Model)

HEALEY ALS Platform Trial



Prespecified covariate risk adjustments included: (i) time from symptom onset, (ii) pre-baseline ALSFRS-R slope, (iii) riluzole use, (iv) edaravone use, and (v) age.

Data on File, Clene Nanomedicine, Inc.

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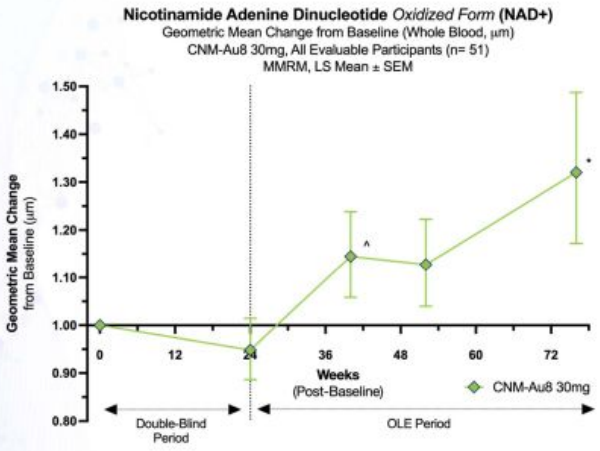
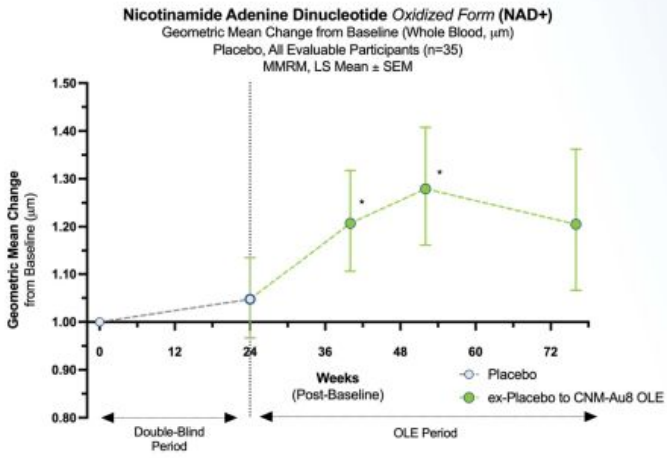
CNM-Au8 Target Engagement | NAD+ Improvement

HEALEY ALS Platform Trial

Original CNM-Au8 30mg



Ex-Placebo to CNM-Au8



LS Mean Difference vs. Baseline: *** p<0.001, ** p< 0.01, * p<0.05, ^ p<0.10

Data on File, Clene Nanomedicine, Inc.

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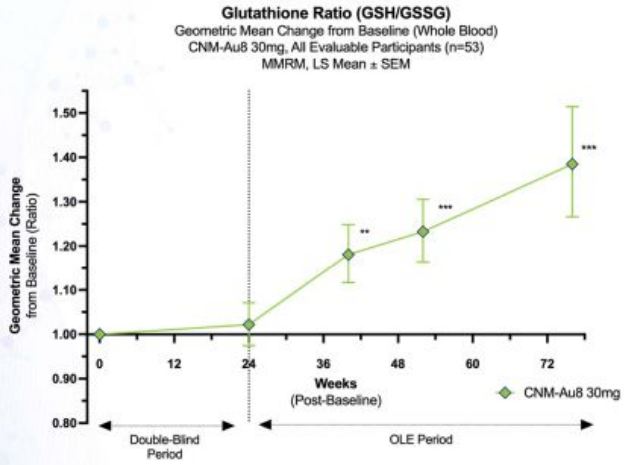
CNM-Au8 Target Engagement | Glutathione Ratio Improvement

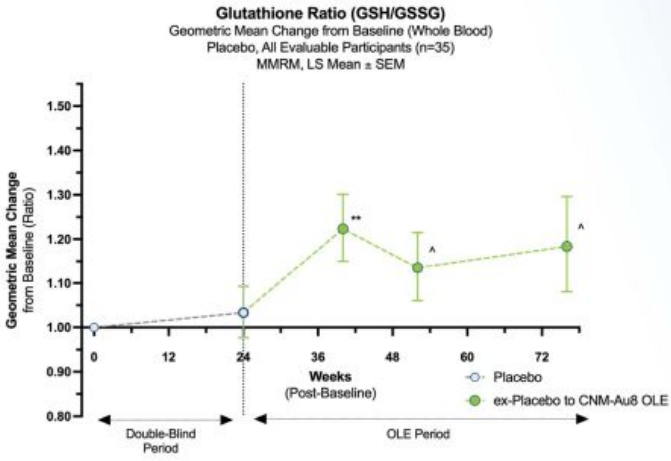
GSH/GSSG Ratio

HEALEY ALS Platform Trial

Original CNM-Au8 30mg



Ex-Placebo to CNM-Au8



LS Mean Difference vs. Baseline: *** p<0.001, ** p< 0.01, * p<0.05, ^ p<0.10

Data on File, Clene Nanomedicine, Inc.

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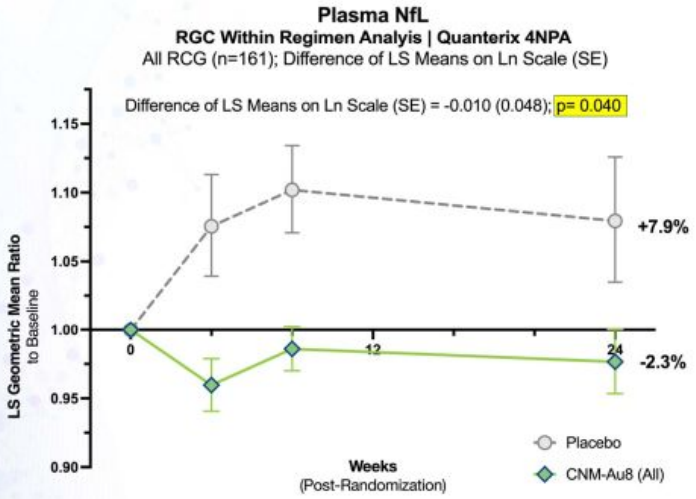
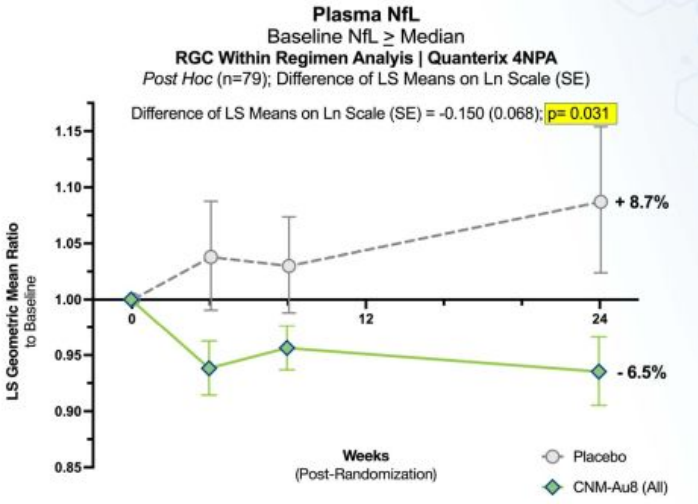
Significant Biomarker Plasma NfL Difference

CNM-Au8 vs. Placebo | All RGC Participants During Double-Blind Period

HEALEY ALS Platform Trial





MMRM log NfL by group, contrast model; prespecified covariates included: (i) pre-treatment ALSFRS-R slope (delta-FS), (ii) months from symptom onset, (iii) use of riluzole, (iv) use of edaravone; (v) covariate by visit interaction, (vi) treatment by visit interaction

Data on File, Clene Nanomedicine, Inc.

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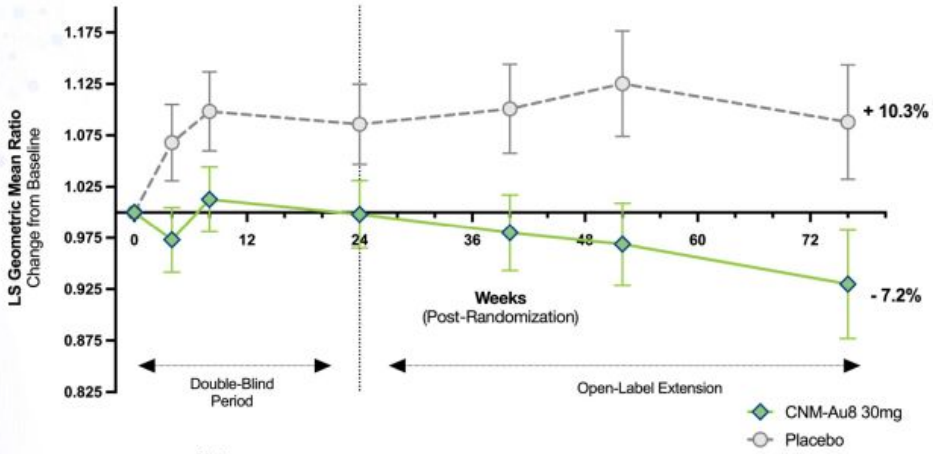
Continued Long Term Plasma NfL Decline in the OLE

76-Weeks post baseline MMRM (CNM-Au8 30mg)

HEALEY ALS Platform Trial

CNM-Au8 30mg Plasma NfL Geometric Mean Change
RGC Within Regimen Analyis | Long Term Extension | Quanterix 4NPA
All Evaluable with Baseline, n=99; LS Geometric Mean Difference ± SEM

Week 76 LS Difference of LS Mean on Ln Scale (SE) = -0.1730 (0.076); p= 0.023



Notes:
[1] All visits graphed with n ≥ 10 participant data.
[2] MMRM analysis uses LS means to account for missing data.

Covariates included: (i) months from symptom onset, (ii) pretreatment ALSFRS-R slope, (ii) background riluzole, (iv) background edaravone. Mixed model repeat measures (MMRM).

Data on File, Clene Nanomedicine, Inc.

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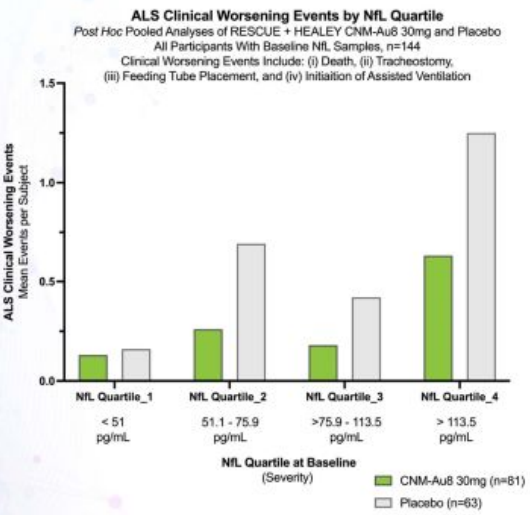
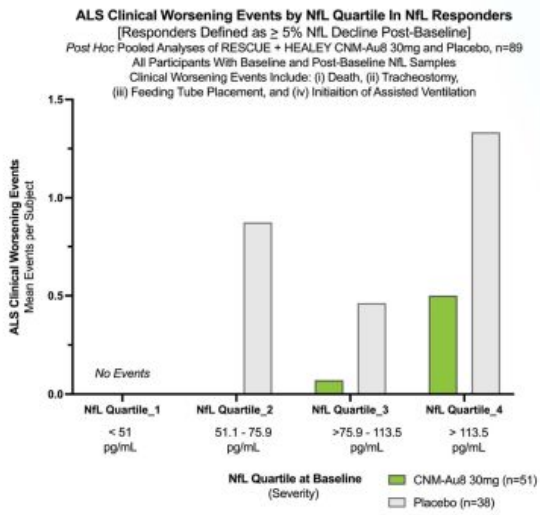
Validation of NfL Association with Clinical Outcomes

Post Hoc | Clinical Worsening Event (Average Events per Patient per Group)

Clinical Worsening Events Frequency is Associated with Higher Baseline NfL Levels (by Quartile)



NfL Responder Analyses in Participants with a NfL Decline of ≥5% (Post-Baseline) Demonstrated Greater Treatment Effect

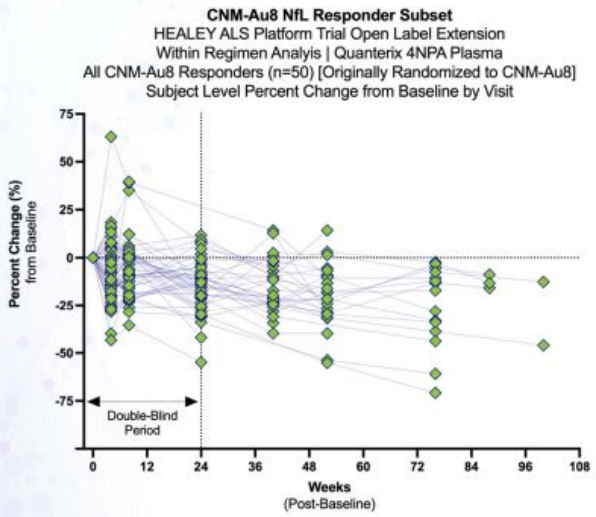


Data on File, Clene Nanomedicine, Inc.

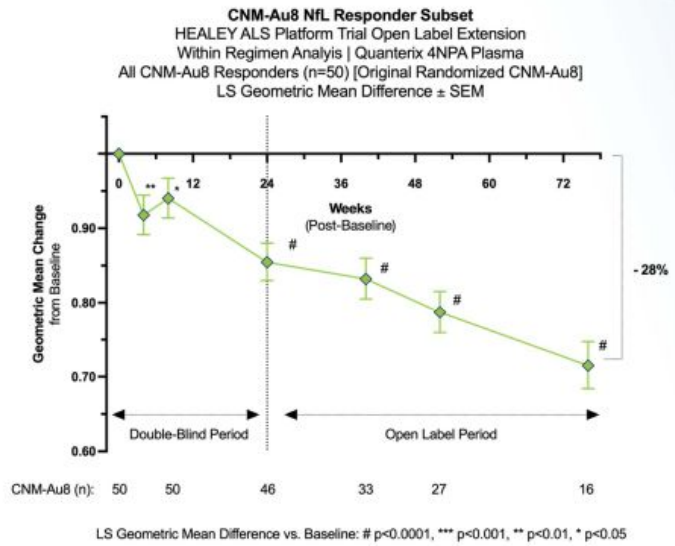
CNM-Au8 NfL Responders Demonstrated Robust NfL Declines

HEALEY ALS Platform Trial

NfL Decline by Participant



NfL Decline Across Responders



LS Geometric Mean Difference vs. Baseline: # p<0.0001, *** p<0.001, ** p<0.01, * p<0.05

NfL Responders include all CNM-Au8 30mg and 60mg originally randomized participants with consistent and sustained NfL declines.

Data on File, Clene Nanomedicine, Inc.

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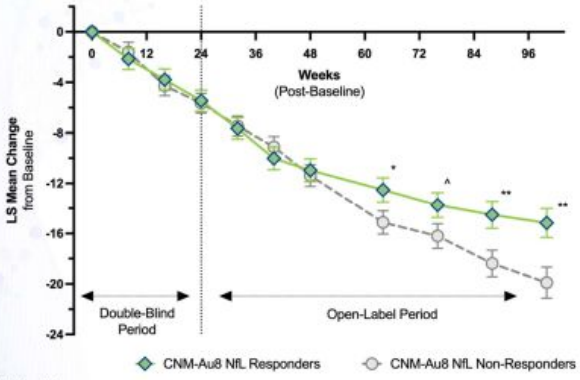
CNM-Au8 NfL Responders Showed Slowed ALSFRS-R Decline

NfL Responders vs. NfL Non-Responders

HEALEY ALS Platform Trial

ALSFRS-R Decline

Long-Term ALSFRS-R Total Score Change by NfL Responder Status
HEALEY ALS Platform Trial | All CNM-Au8 with Baseline Covariates (n=116 of 120)
CNM-Au8 Plasma NfL Responders vs. Non-Responders
MMRM; LS Mean Change from Baseline ± SEM



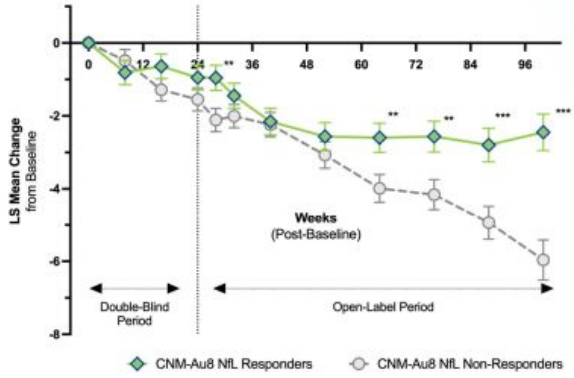
CNM-Au8 NfL Responders CNM-Au8 NfL Non-Responders

No. Evaluable											
NfL Responder:	50	50	45	47	41	32	35	27	23	17	14
Non-Responder:	66	60	59	59	52	41	38	29	22	18	9

LS Mean Difference: *** p<0.001, ** p< 0.01, * p<0.05, ^ p<0.10

ALSFRS-R Respiratory Subdomain

Long-Term ALSFRS-R Respiratory Subscore Change by NfL Responder Status
HEALEY ALS Platform Trial | All CNM-Au8 with Baseline Covariates (n=116 of 120)
CNM-Au8 Plasma NfL Responders vs. Non-Responders
MMRM; LS Mean Change from Baseline ± SEM



CNM-Au8 NfL Responders CNM-Au8 NfL Non-Responders

No. Evaluable											
NfL Responder:	50	50	45	47	41	32	32	27	23	17	14
Non-Responder:	66	60	59	59	52	41	34	29	22	18	9

LS Mean Difference: *** p<0.001, ** p< 0.01, * p<0.05, ^ p<0.10

Data on File, Clene Nanomedicine, Inc. NfL Responders include all CNM-Au8 30mg and 60mg originally randomized participants with consistent and sustained NfL declines.

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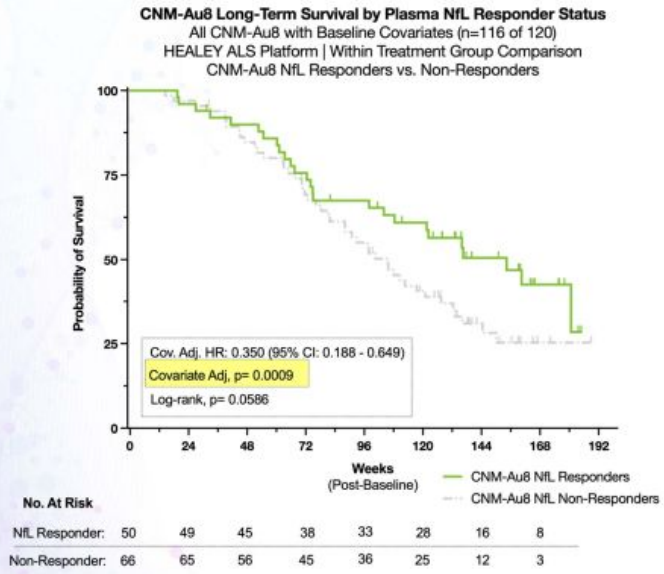
CNM-Au8 NfL Responders Demonstrated Improved Survival

NfL Responders vs. NfL Non-Responders and vs. PRO-ACT Matched Controls

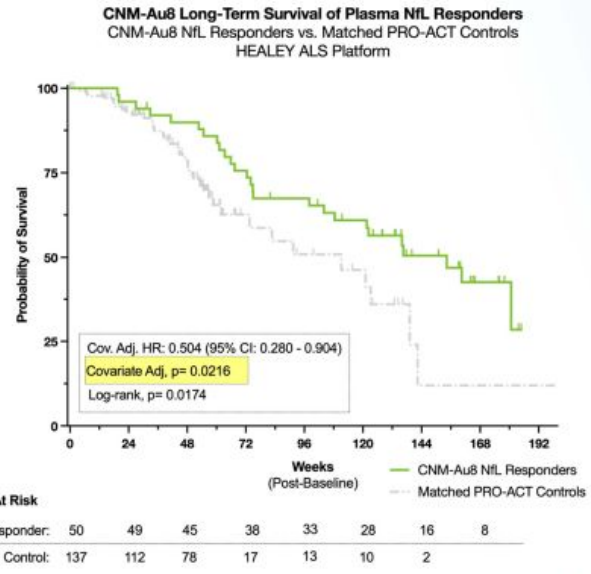
HEALEY ALS Platform Trial

NfL Responders vs. NfL Non-Responders



No. At Risk								
NfL Responder:	50	49	45	38	33	28	16	8
Non-Responder:	66	65	56	45	36	25	12	3

NfL Responders vs. Matched PRO-ACT Controls



No. At Risk								
NfL Responder:	50	49	45	38	33	28	16	8
Matched Control:	137	112	78	17	13	10	2	

Data on File, Clene Nanomedicine, Inc. NfL Responders include all CNM-Au8 30mg and 60mg originally randomized participants with consistent and sustained NfL declines.

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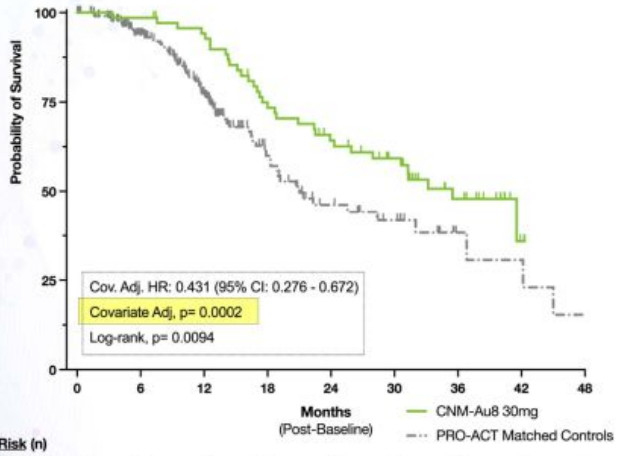
CNM-Au8 30mg Treatment Improved Long-Term Survival vs. Propensity Matched PRO-ACT & NHC Controls

HEALEY ALS Platform Trial

PRO-ACT Controls

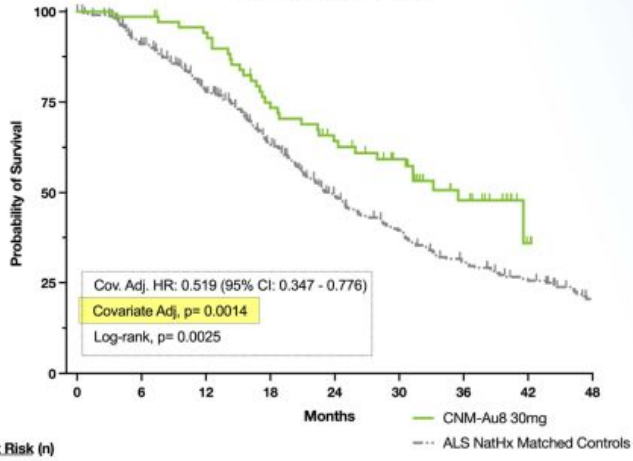
CNM-Au8 30 mg Long Term Survival | HEALEY ALS Platform Trial
Original CNM-Au8 (n=59) and ex-Placebo to CNM-Au8 (n=11)
CNM-Au8 30mg (n=70) vs. PRO-ACT Propensity Matched Controls (n=322)
Time to All-Cause Mortality



At Risk (n)									
CMM-Au8:	70	70	65	51	41	32	18	3	0
Matched Controls:	322	259	174	43	25	16	6	5	3

Natural History Consortium Controls

CNM-Au8 30 mg Long Term Survival | HEALEY ALS Platform Trial
Original CNM-Au8 (n=59) and ex-Placebo to CNM-Au8 (n=11)
CNM-Au8 30mg vs. ALS Natural History Study Propensity Matched Controls
Time to All-Cause Mortality



At Risk (n)									
CMM-Au8:	70	70	65	50	41	32	18	3	0
Matched Controls:	322	277	220	164	115	87	65	50	31

Data on File, Clene Nanomedicine, Inc.

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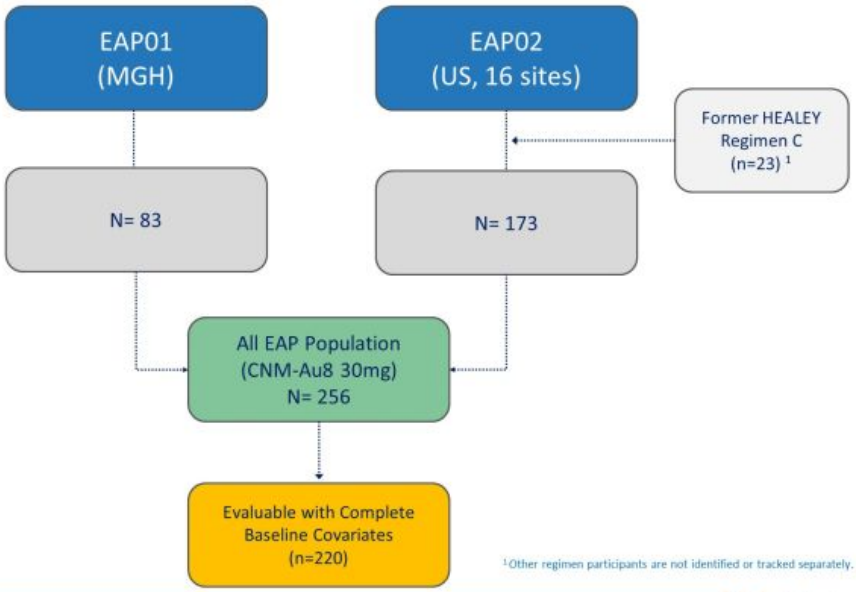
EAP Participant Enrollment

- All EAP participants (CNM-Au8 30mg) enrolled through 15-December-2023 with EDC data entry
- Survival updated through the 14-January-2024 data cut

EAP01 in collaboration with the:

Healey Center
Sean M. Healey & AMG Center for ALS at Mass General

The Healey center covered all site costs and EDC management through philanthropic donations; Clene provided CNM-Au8 and conducted analyses

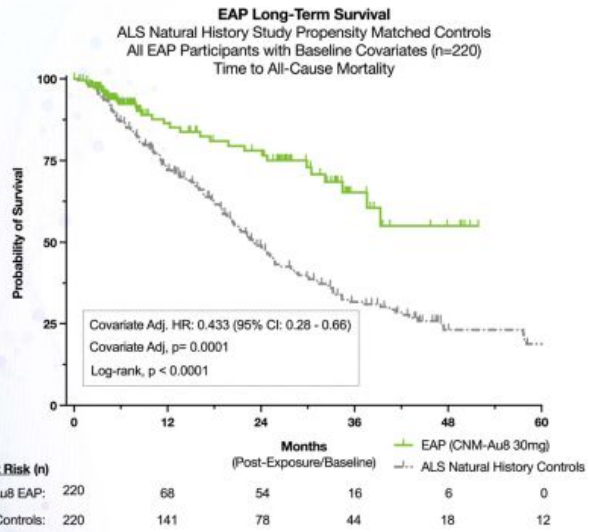


[1] Other regimen participants are not identified or tracked separately.

Data on File, Clene Nanomedicine, Inc.

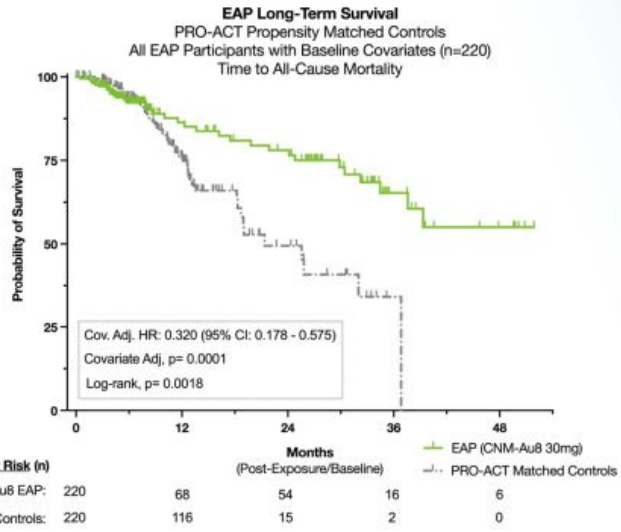
EAP Survival vs. ALS Natural History and PRO-ACT Matched Controls | Control-Matched EAP and All EAP

ALS Natural History Study | EAP Matched (n=220)



At Risk (n)						
CMM-Au8 EAP:	220	68	54	16	6	0
Controls:	220	141	78	44	18	12

PRO-ACT | EAP Matched (n=220)



At Risk (n)					
CMM-Au8 EAP:	220	68	54	16	6
Controls:	220	116	15	2	0

Baseline covariates include: (i) onset age, (ii) sex, (iii) BMI, (iv) pre-treatment ALSFRS-R slope, (v) ALSFRs-R, (vi) vital capacity (% predicted), (vii) vital capacity pre-treatment slope, and (viii) TRICALS risk score. Propensity matching caliper minimized at 0.41. All EAP participants alive are right censored as of the January 18, 2024 data cut.

Data on File, Clene Nanomedicine, Inc.

VISIONARY-MS STUDY Core Design Elements

Phase 2 Study: 48-Week Placebo-Control Treatment Period 2:1 Randomization (Active [15mg, 30 mg]: Placebo)

- Enrolled stable relapsing remitting MS participants with chronic optic neuropathy on background DMTs
- 92% treated with background DMTs (inc 53% monoclonal antibodies, 32% oral)
- n=73 of 150 planned (~50%) study ended prematurely due to COVID-19 pandemic enrollment challenges
- Modified ITT (mITT) Analysis Population; Pre-specified statistical threshold set at p=0.10
- CNM-Au8 Open Label Extension (OLE) for original active and placebo continued for up-to-96 weeks

1° Change in Low Contrast Letter Acuity (LCLA)

2° Change in modified MS Functional Composite (mMSFC)

9HPT SDMT T25FWT LCLA

Data on File, Clene Nanomedicine, Inc.

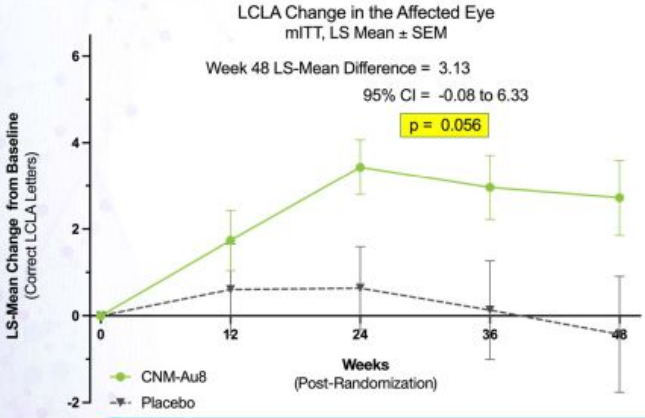
Clene NANOMEDICINE

CNM-Au8 Demonstrated Vision and Global Neurological Improvement in Stable MS patients on DMTs

VISIONARY-MS STUDY

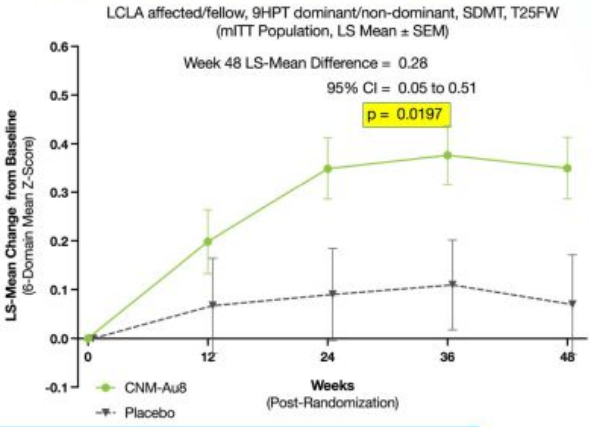
Significantly Improved Vision

Change in Low Contrast Letter Acuity (LCLA)



Global Neurological Improvement

Change in modified MS Functional Composite (mMSFC)



Global neurological clinical improvement was driven by cognition, manual dexterity, and low contrast letter acuity

Data on File, Clene Nanomedicine, Inc.

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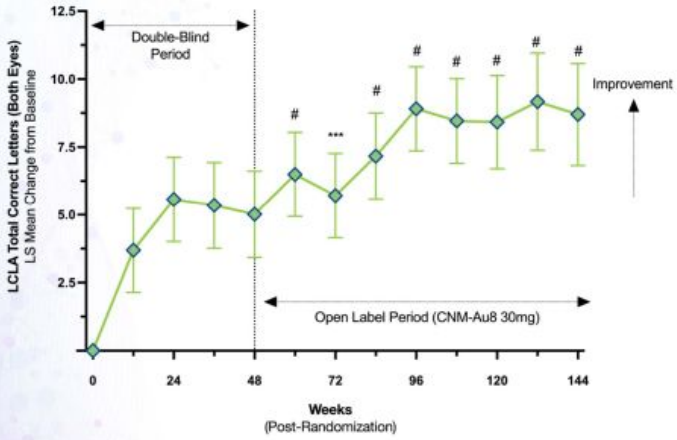
Long-Term LCLA Improvement in LTE Participants

Low Contrast Letter Acuity

VISIONARY-MS STUDY

Original Active (CNM-Au8)

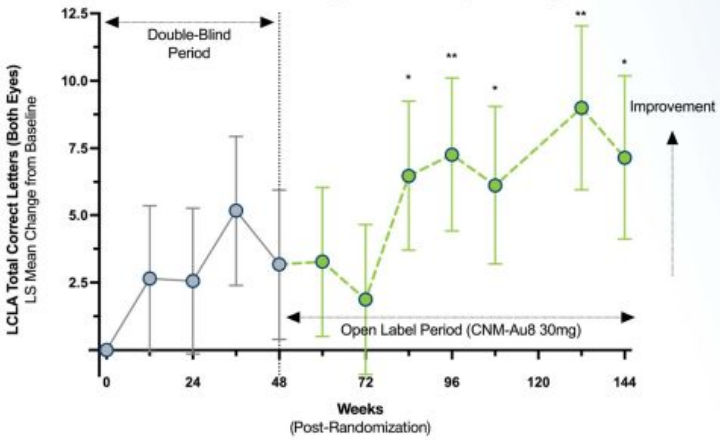
Longitudinal LCLA | Change from Baseline (Total Correct, Both Eyes) | All Active
In LTE Participants Originally Randomized to CNM-Au8 (n=35), mITT Population
LS Mean ± SEM, Change from Baseline (Preliminary)



LTE: LS mean difference vs. randomization baseline: # p≤0.0001, *** p≤0.001, ** p≤0.01, *p≤0.05

Original Placebo

Longitudinal LCLA | Change from Baseline (Total Correct, Both Eyes)
In LTE Participants Originally Randomized to Placebo (n=11), mITT Population
LS Mean ± SEM, Change from Baseline (Preliminary)



MMRM accounts for missing data; all visits with ≥ 60% participant values are graphed.

LTE: LS mean difference vs. randomization baseline: # p≤0.0001, *** p≤0.001, ** p≤0.01, *p≤0.05

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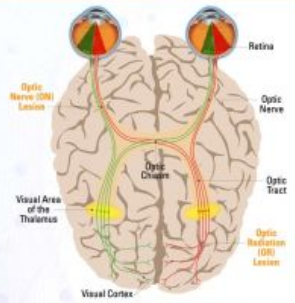
CNM-Au8 Improved Information Signal Strength & Speed in the Visual Pathway

VISIONARY-MS STUDY

Visual Evoked Potentials

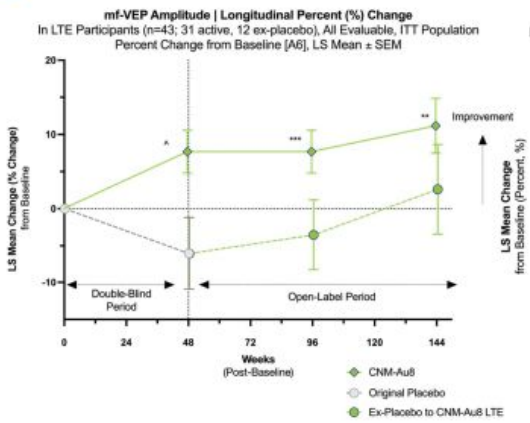


Amplitude = Signal Strength

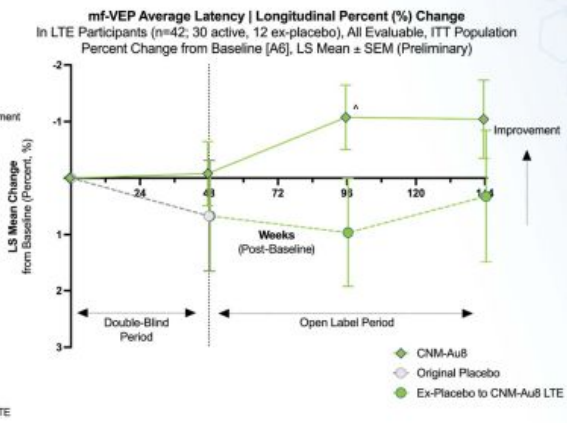
Latency = Signal Speed

From the Eye to Visual Cortex

Improved Amplitude



Improved Latency



LTE: LS mean difference vs. randomization baseline: # p≤0.0001, *** p≤0.001, ** p≤0.01, *p≤0.05, ^p≤0.10

Increased VEP amplitude is associated with improved axonal integrity (more signal);
Improved latency is associated with evidence of remyelination (faster conduction velocity)

Data on File, Clene Nanomedicine, Inc.

CLENE NANOMEDICINE

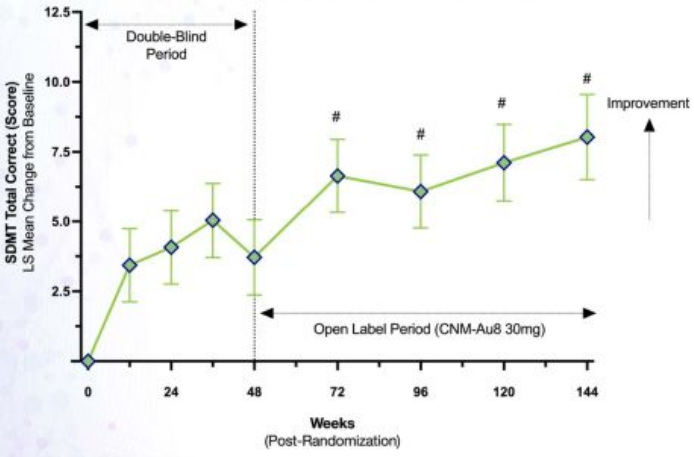
Long-Term SDMT Improvement in LTE Participants

Symbol Digit Modality Test | Working Memory & Cognition

VISIONARY-MS STUDY

Original Active (CNM-Au8)

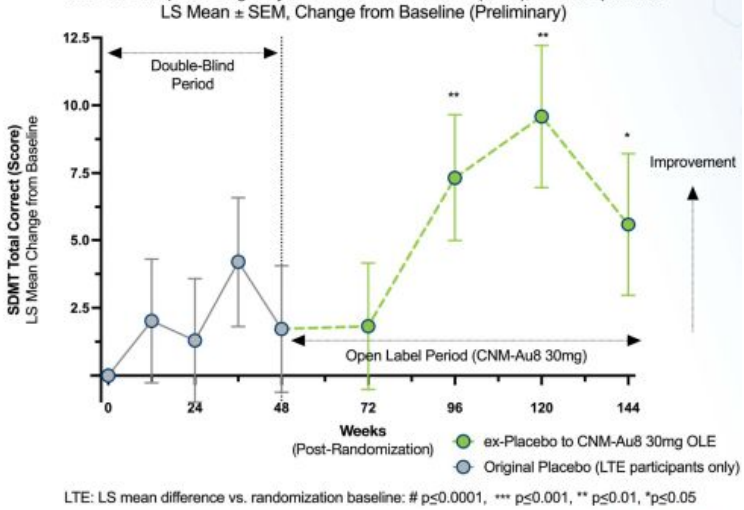
Longitudinal SDMT | Change from Baseline (Total Score) | All Active
In LTE Participants Originally Randomized to CNM-Au8 (n=35), mITT Population
LS Mean ± SEM, Change from Baseline (Preliminary)



LTE: LS mean difference vs. randomization baseline: # p≤0.0001, *** p≤0.001, ** p≤0.01, *p≤0.05

Original Placebo

Longitudinal SDMT | Change from Baseline (Total Score)
In LTE Participants Originally Randomized to Placebo (n=11), mITT Population
LS Mean ± SEM, Change from Baseline (Preliminary)



LTE: LS mean difference vs. randomization baseline: # p≤0.0001, *** p≤0.001, ** p≤0.01, *p≤0.05

Data on File, Clene Nanomedicine, Inc.

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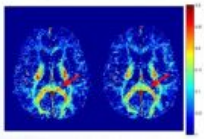
CNM-Au8 Treatment Demonstrated MS Lesion Repair and Promoted Remyelination

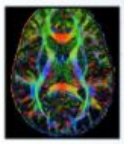
VISIONARY-MS STUDY

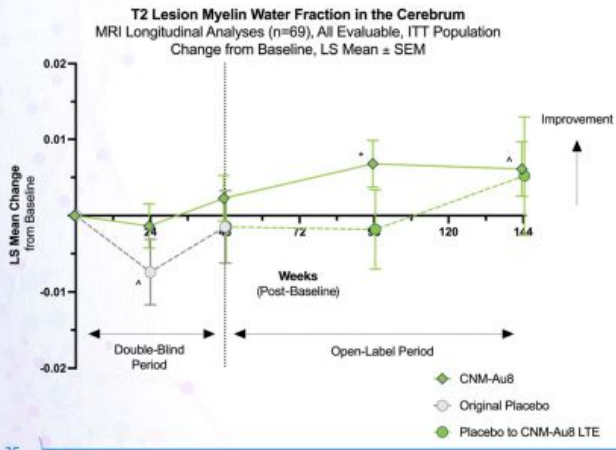
Advanced MRI Techniques

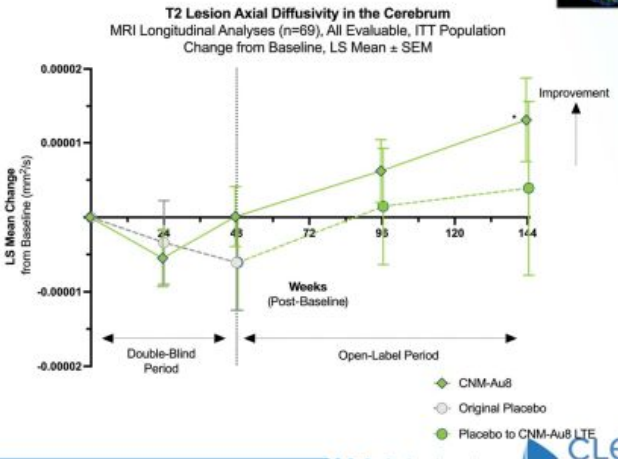


T2 Lesion Myelin Water Fraction (Remyelination)

T2 Lesion Axial Diffusivity (Axonal Integrity)







Data on File, Clene Nanomedicine, Inc.

LTE: LS mean difference vs. randomization baseline: # p≤0.0001, *** p≤0.001, ** p≤0.01, *p≤0.05, ^p≤0.10

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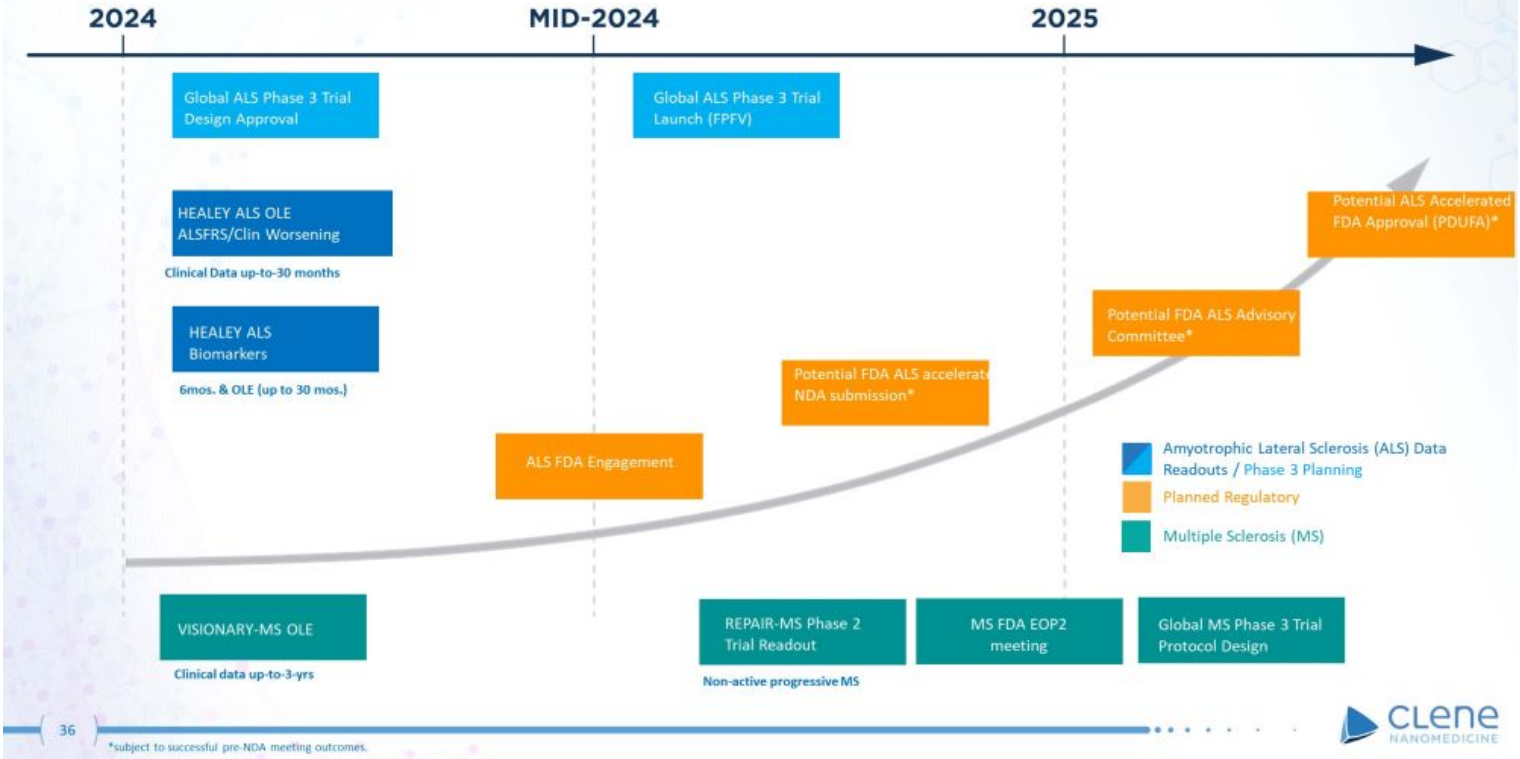
Clene | CNM-Au8 Path to Regulatory Approval



*subject to successful pre-NDA meeting outcomes.

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Evidence Supports CNM-Au8 Therapeutic Potential to Treat Neurodegenerative Diseases

CNM-Au8®
a gold nanocrystal suspension, in development as the first cellular energetic catalyst to remyelinate[1] & protect neurological function

RESCUEALS

75% decreased risk of death in ALS through 168 weeks

HEALEY ALS Platform Trial

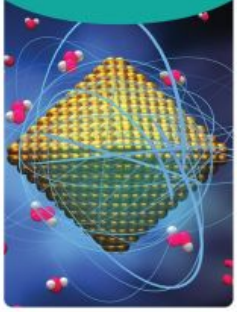
>90% decreased risk of death with 30 mg in ALS

VISIONARY-MS

Demonstrated global neurological improvement in MS patients on adjunctive DMT standard of care

>650
patient years of CNM-Au8 clinical exposure

Strong IP:
150+
patents on nanotherapeutic platform, plus trade secret protection



As of Mar 31 2024, cash and equivalents on hand (unaudited):

$27.9M

Data on File, Clene Nanomedicine, Inc. [1]Robinson et al. Sci Rep. 2020 Feb 11;10(1):1936.[2]https://clinicaltrials.gov/ct2/show/NCT04414345.

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Version: 6 August 2024

Exhibit 99.2

CLENE ANNOUNCES NEW CNM-Au8® BIOMARKER AND CLINICAL EFFICACY DATA SUBMITTED TO FDA IN SUPPORT OF TREATMENT FOR ALS

- CNM-Au8 treated participants in the HEALEY ALS Platform Trial with substantial neurofilament light (NfL) declines (CNM-Au8 NfL Responders) demonstrated significant clinical improvements in survival, functional status (slowed ALSFRS-R decline), and combined function and survival (CAFS scores) compared to NfL non-responders
- Independent of NfL responder status, significant survival benefits in CNM-Au8 30mg treated participants continue to be observed in the long-term extension compared to multiple natural history controls
- Nicotinamide adenine dinucleotide (NAD) and glutathione improvements were consistent and sustained with CNM-Au8 treatment, supporting a dual mechanism of action and indicating target engagement in ALS patients
- CNM-Au8 treated participants who demonstrated NAD/glutathione improvements demonstrated concordance in the same participants who were CNM-Au8 NfL Responders

SALT LAKE CITY, August 6, 2024 – Clene Inc. (Nasdaq: CLNN) (along with its subsidiaries, "Clene") and its wholly-owned subsidiary Clene Nanomedicine Inc., a clinical-stage biopharmaceutical company focused on improving mitochondrial health and protecting neuronal function to treat neurological diseases, including amyotrophic lateral sclerosis (ALS) and multiple sclerosis (MS), today announced new CNM-Au8 biomarker and clinical efficacy data submitted to the FDA, including *post hoc* analyses from two independently conducted Phase 2 clinical trials of CNM-Au8 for the treatment of ALS. This new information supplements the original data previously discussed with FDA in late 2023 and is intended to guide the planned FDA Type C interaction expected to occur in the third quarter of 2024 to discuss an accelerated approval regulatory pathway.

The level of neurofilament light (NfL) in plasma is considered an important biomarker of ALS disease progression and mortality risk.

CNM-Au8 NfL Responders, defined as those who had consistent and sustained NfL reductions, comprising nearly half of all CNM-Au8 treated patients, demonstrated a 28% mean reduction in NfL levels compared to baseline, while NfL levels continued to increase in CMN-Au8 NfL non-responders (all doses; geometric mean ratio (GMR) difference at week 76 post-baseline: 0.57, 95% CI: 0.50 – 0.64, $p < 0.00001$).

New analyses of the CNM-Au8 NfL Responders demonstrated efficacy in all-cause mortality, functional, and combined assessment of function and survival (CAFS):

- **All-cause mortality (survival):**
  - **Improved survival of CNM-Au8 NfL Responders compared to CNM-Au8 NfL non-responders:** hazard ratio (HR): 0.350, 95% CI: 0.188 – 0.649; covariate adjusted, $p = 0.0009$
  - **Improved survival of CNM-Au8 NfL Responders compared to propensity matched** controls from the PRO-ACT database: HR: 0.504, 95% Wald CI: 0.28 – 0.904, covariate adjusted $p = 0.022$)
- **ALS Functional Improvement:** the ALS Functional Rating Scale (ALSFRS-R) is an instrument for evaluating the functional status of patients with ALS and is used to monitor functional change in a patient over time. CNM-Au8 NfL Responders demonstrated:
  - **Significantly less decline in ALSFRS-R total score compared to CNM-Au8 NfL non-responders:** $p < 0.01$ at the Week 64, 76, 88, and 100 visits post-randomization (Mixed model repeated measures (MMRM) was used to compare least squares mean change from baseline).
  - **Significantly less decline in the respiratory subdomain score of the ALSFRS-R compared to CNM-Au8 NfL non-responders:** $p < 0.01$ at the Week 64, 76, 88, and 100 visits post-randomization (MMRM was used to compare least squares mean change from baseline).
- **Improvements in the Combined Assessment of Function and Survival:** CAFS ranks clinical outcomes based on survival time and change in the ALSFRS-R:
  - CNM-Au8 NfL Responder demonstrated improvements compared to CNM-Au8 NfL non-responders starting at Week 48 ($p<0.10$) and all later timepoints with significance reached at Weeks 88 and later ($p < 0.05$).

Independent of NfL responder status, **long-term treatment with CNM-Au8 30 mg was associated with improved survival in participants from the RESCUE-ALS and HEALEY ALS Platform Trials using updated long-term follow-up of survival status** compared to propensity matched controls from the clinical trial data registry PRO-ACT, the ALS/MND Natural History Consortium (NHC), and the Australian MiNDAUS registry. Matching methods and covariates were prespecified and conducted by an independent statistician.

- Long-term treatment with CNM-Au8 30mg in the HEALEY ALS Platform Trial demonstrated a **57% decreased risk of all-cause mortality vs. PRO-ACT propensity matched controls:** (HR: 0.431, 95% CI: 0.276 to 0.672; covariate adjusted, p = 0.0002)
- Long-term treatment with CNM-Au8 30 mg in the HEALEY ALS Platform Trial demonstrated a **48% decreased risk of all-cause mortality vs. ALS NHC propensity matched controls** (HR: 0.519, 95% CI: 0.347 to 0.776; covariate adjusted, p = 0.0014).
- Long-term treatment with CNM-Au8 30 mg in the RESCUE-ALS Phase 2 Trial demonstrated a **70% decreased risk of all-cause mortality vs. PRO-ACT propensity matched controls:** (HR: 0.311, 95% CI: 0.142 to 0.682; covariate adjusted, p = 0.0035)
- Long-term treatment with CNM-Au8 30 mg in the RESCUE-ALS Phase 2 Trial demonstrated a **51% decreased risk of all-cause mortality vs. MiNDAUS propensity matched control:** (HR: 0.487, 95% CI: 0.287 to 0.824; covariate adjusted, p = 0.0074).

CNM-Au8 mechanism of action responders demonstrated concordance with CNM-Au8 NfL Responders. Data provided to the FDA also included an association of responses between CNM-Au8 mechanism responders (defined as those who had consistent and sustained NAD+ and GSH/GGSG glutathione improvements) and CNM-Au8 NfL Responders. The connection between CNM-Au8 mechanism responders and CNM-Au8 NfL Responders links the mechanism of action to NfL declines. Biomarkers of oxidative stress, including the GSH/GSSG ratio, demonstrated consistent improvement following CNM-Au8 treatment with increased activity associated with the duration of treatment. These data support a dual mechanism of action of neuronal metabolic support and decreased oxidative stress. Clene further provided mechanistic evidence from preclinical models that established improved neuronal integrity and survival, where CNM-Au8 simultaneously decreased the release of NfL from damaged motor neurons axons.

Merit Cudkowicz, M.D., Chair, Neurology Department, Massachusetts General Hospital, Director, Sean M Healey & AMG Center for ALS, and the Principal Investigator of the HEALEY ALS Platform Trial, said, “The strong safety profile of CNM-Au8, with its NfL biomarker response now linked to survival evidence, and new information on mechanisms of action support proceeding to a confirmatory Phase 3 clinical trial and regulatory discussions on approval pathways.”

Over 650 patient years of safety data continue to show that CNM-Au8 demonstrates a safety profile with no significant safety concerns or safety trends identified. No serious adverse events (SAEs) have been identified as related to CNM-Au8 treatment by any investigators to date.

“The risk-benefit assessment evidence of CNM-Au8 is strong. Our next step is discussing this new CNM-Au8 biomarker and efficacy data with the FDA, with the hope that ALS patients will benefit from this drug, sooner rather than later,” said CEO and President of Clene, Rob Etherington.

About Clene

Clene Inc., (Nasdaq: CLNN) (along with its subsidiaries, “Clene”) and its wholly owned subsidiary Clene Nanomedicine Inc., is a late clinical-stage biopharmaceutical company focused on improving mitochondrial health and protecting neuronal function to treat neurodegenerative diseases, including amyotrophic lateral sclerosis, Parkinson’s disease, and multiple sclerosis. CNM-Au8® is an investigational first-in-class therapy that improves central nervous system cells’ survival and function via a mechanism that targets mitochondrial function and the NAD pathway while reducing oxidative stress. CNM-Au8® is a federally registered trademark of Clene Nanomedicine, Inc. The company is based in Salt Lake City, Utah, with R&D and manufacturing operations in Maryland. For more information, please visit www.clene.com or follow us on X (formerly Twitter) and LinkedIn.

Forward Looking Statements:
This press release contains “forward-looking statements” within the meaning of Section 21E of the Securities Exchange Act of 1934, as amended, and Section 27A of the Securities Act of 1933, as amended, which are intended to be covered by the “safe harbor” provisions created by those laws. Clene’s forward-looking statements include, but are not limited to, statements regarding the Company’s expectations, hopes, beliefs, intentions or strategies, including expectations regarding interactions with the FDA and the next steps regarding the Company’s efforts to seek an accelerated approval pathway from the FDA. In addition, any statements that refer to characterizations of future events or circumstances, including any underlying assumptions, are forward-looking statements. The words “anticipate,” “believe,” “contemplate,” “continue,” “estimate,” “expect,” “intends,” “may,” “might,” “plan,” “possible,” “potential,” “predict,” “project,” “should,” “will,” “would,” and similar expressions may identify forward-looking statements, but the absence of these words does not mean that a statement is not forward-looking. These forward-looking statements represent our views as of the date of this press release and involve a number of judgments, risks and uncertainties. We anticipate that subsequent events and developments will cause our views to change. We undertake no obligation to update forward-looking statements to reflect events or circumstances after the date they were made, whether as a result of new information, future events or otherwise, except as may be required under applicable securities laws. Accordingly, forward-looking statements should not be relied upon as representing our views as of any subsequent date. As a result of a number of known and unknown risks and uncertainties, the Company’s expectations, hopes, beliefs, intentions or strategies, including expectations regarding the timing of the Type C meeting , may be materially different from those expressed or implied by these forward-looking statements. Some factors that could cause actual results to differ include the Company’s ability to demonstrate the efficacy and safety of its drug candidates; the clinical results for its drug candidates, which may not support further development or marketing approval; actions of regulatory agencies, which may affect the initiation, timing and progress of clinical trials and marketing approval; the Company’s ability to achieve commercial success for its drug candidates, if approved; the Company’s limited operating history and its ability to obtain additional funding for operations and to complete the development and commercialization of its drug candidates, and other risks and uncertainties set forth in “Risk Factors” in our most recent Annual Report on Form 10-K and any subsequent Quarterly Reports on Form 10-Q. In addition, statements that “we believe” and similar statements reflect our beliefs and opinions on the relevant subject. These statements are based upon information available to us as of the date of this press release, and while we believe such information forms a reasonable basis for such statements, such information may be limited or incomplete, and our statements should not be read to indicate that we have conducted an exhaustive inquiry into, or review of, all potentially available relevant information. These statements are inherently uncertain and you are cautioned not to rely unduly upon these statements. All information in this press release is as of the date of this press release. The information contained in any website referenced herein is not, and shall not be deemed to be, part of or incorporated into this press release.

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