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): April 16, 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-half of one share of Common Stock for $11.50 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 April 16, 2024, Clene Inc. (the "Company") released a presentation (the "Presentation") and poster (the "Poster") on its website, invest.clene.com. The Presentation and Poster discuss the clinical results of CNM-Au8 treatment in the Company's VISIONARY-MS trial long-term extension ("LTE") for multiple sclerosis ("MS"). A copy of the Presentation and Poster are furnished as Exhibit 99.1 and Exhibit 99.2, respectively, to this Current Report on Form 8-K and are incorporated herein by reference.

The information furnished in this Item 7.01, including Exhibit 99.1 and Exhibit 99.2, 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 April 16, 2024, the Company issued a press release announcing evidence for CNM-Au8 as a treatment for repair and remyelination in MS presented in the Emerging Science Session at the 2024 American Academy of Neurology ("AAN") Annual Meeting. A copy of the press release is filed as Exhibit 99.3 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	VISIONARY-MS LTE Presentation.
99.2	VISIONARY-MS LTE Poster.
99.3	Press release, dated April 16, 2024, announcing evidence for Clene's CNM-Au8 as a treatment for repair and remyelination in MS presented in the Emerging Science Session at the 2024 AAN Annual Meeting.
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: April 16, 2024

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


Phase 2
VISIONARY-MS
STUDY
Long-Term Open Label Extension
In Stable RMS Participants with Chronic Optic Neuropathy
Michael Barnett, MBBS PhD FRACP FRCP
On behalf of the VISIONARY-MS Investigators

Disclosures

- The University of Sydney received industry standard financial renumeration as a clinical trial site
- I am a consulting research director for Sydney Neuroimaging Analysis Centre (SNAC), which was contracted to analyse blinded MRI and VEP data
- I am a consulting physician to RxPx Cor
- I have received institutional support for research from Biogen, Merck, Novartis, Roche, BMS, and Sanofi Genzyme
- I have received institutional support for speaking, participation in advisory boards or consulting from Biogen, Merck, Novartis, Roche, BMS, Sanofi Genzyme and Autobahn Therapeutics

Treatment and Participant Disposition in the Long-Term Extension

VISIONARY-MS
Long-Term
Open Label Extension

CNM-Au8 30mg
Oral Suspension

Clean Surfaced,
Highly Faceted Nanocrystals

Cellular Energetic Nanocatalyst:
Mitochondrial Support & Increased Energetic Capacity in Neurons and Glia

Randomized
n=73

Eligible for LTE
n=69

Enrolled in LTE
n=55 (80%)

Treatment duration for up to 3-years

Original CNM-Au8 to CNM-Au8 LTE
n=41 (87%)

CNM-Au8 30mg LTE
mITT (n=35)

Original Placebo to CNM-Au8 LTE
n=14 (63%)

Ex-Placebo to CNM-Au8 30mg LTE
mITT (n=11)

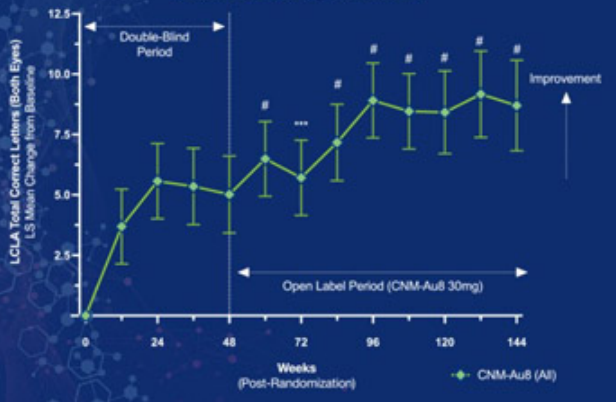
Clinical Results | Long-Term LCLA Improvement

Low Contrast Letter Acuity (Original Double-Blind Primary Endpoint)

VISIONARY-MS
Long-Term
Open Label Extension

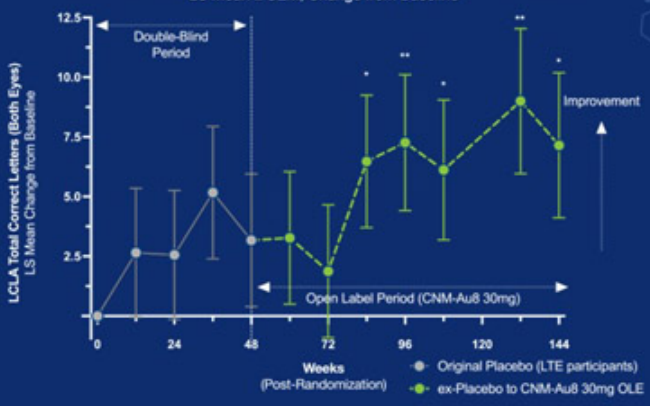
Original CNM-Au8

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



Ex-Placebo to CNM-Au8

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



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

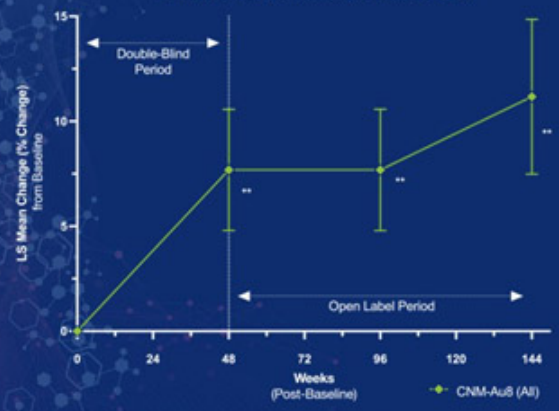
Multi-Focal VEP | Long-Term Amplitude Improvement

Visual Pathway Signal Strength

VISIONARY-MS
Long-Term
Open Label Extension

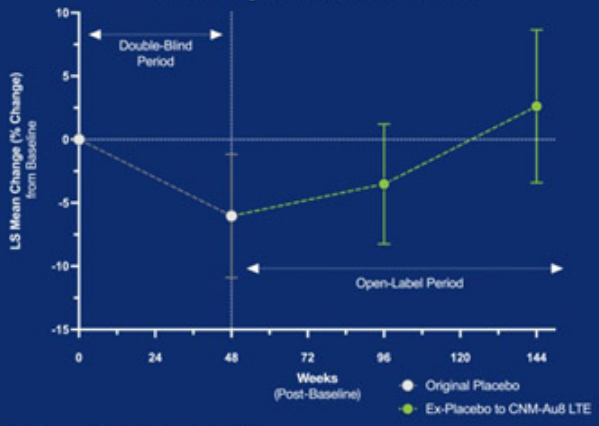
Original CNM-Au8

mf-VEP Amplitude | CNM-Au8 Longitudinal Percent (%) Change [A6]
In LTE Participants (n=32 active), All Evaluable, ITT Population
Percent Change from Baseline, LS Mean ± SEM



Ex-Placebo to CNM-Au8

mf-VEP Amplitude | ex-Placebo Longitudinal Percent (%) Change [A6]
In LTE Participants (n=12 ex-placebo), All Evaluable, ITT Population
Percent Change from Baseline, LS Mean ± SEM



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

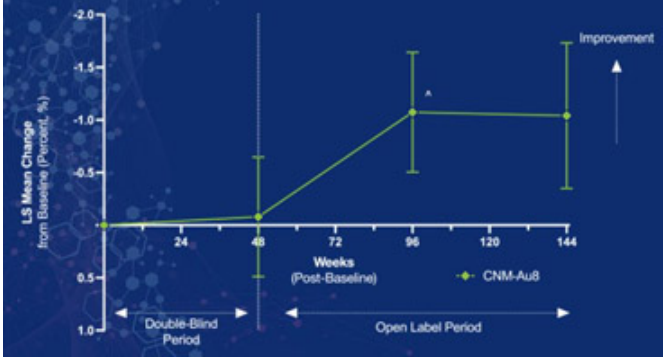
Multi-Focal VEP | Long-Term Latency Improvement

Visual Pathway Conduction Velocity

VISIONARY-MS
Long-Term
Open Label Extension

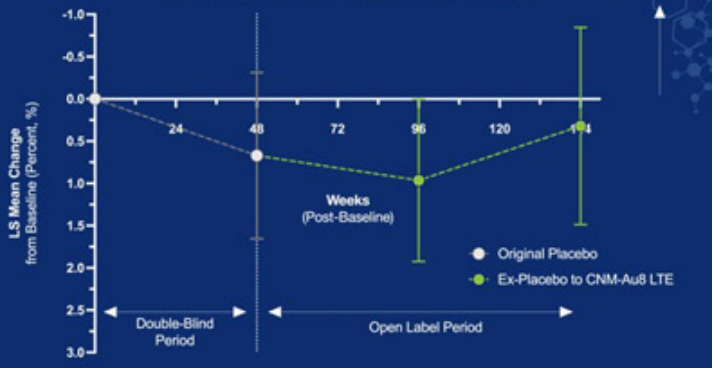
Original CNM-Au8

mf-VEP Average Latency | Longitudinal Percent (%) Change
In LTE Participants (n=30 active), All Evaluable, ITT Population
Percent Change from Baseline [A6], LS Mean ± SEM



Ex-Placebo to CNM-Au8

mf-VEP Average Latency | Longitudinal Percent (%) Change
In LTE Participants (12 ex-placebo), All Evaluable, ITT Population
Percent Change from Baseline [A6], LS Mean ± SEM



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

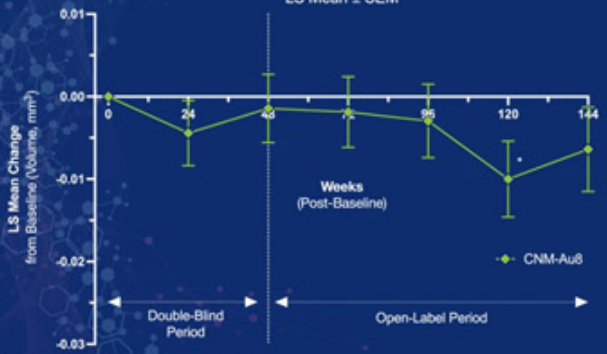
OCT | Long-Term Ganglion Cell Layer Preservation

GCL Volume Change in the Most Affected Eyes at Baseline

VISIONARY-MS
Long-Term
Open Label Extension

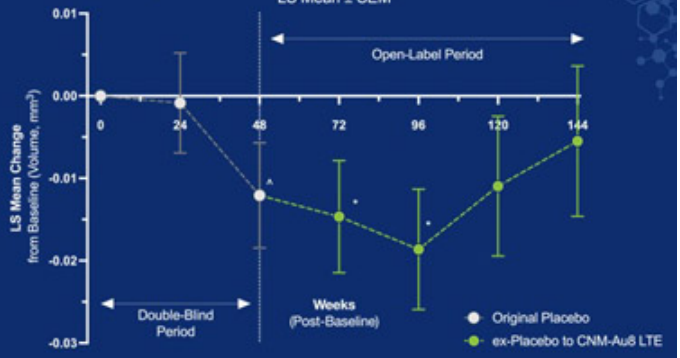
Original CNM-Au8

Ganglion Cell Layer Volume (mm^3) in Most Affected Eyes (At Baseline, n=49 of 146)
All Active Participants
Heidelberg OCT GCL Macular Scan, ITT Population (All Evaluable Eyes)
LS Mean ± SEM



Ex-Placebo to CNM-Au8

Ganglion Cell Layer Volume (mm^3) in Most Affected Eyes (At Baseline, n=49 of 146)
All Placebo Participants
Heidelberg OCT GCL Macular Scan, ITT Population (All Evaluable Eyes)
LS Mean ± SEM



LTE: LS mean difference vs. randomization baseline: # p≤0.0001, *** p≤0.001, ** p≤0.01, *p≤0.05, ^p≤0.10;
Most affected eyes defined as ≤25% percentile distribution at baseline or with inter-eye RNFL difference ≥6 µm or GCL difference of ≥4 µm (worst eye) – *post hoc*

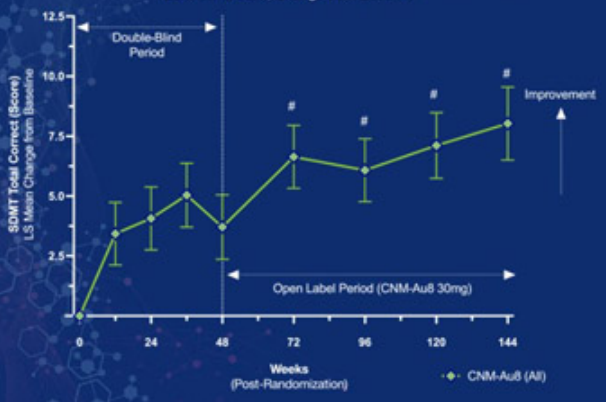
Clinical Results | Long-Term SDMT Improvement

Working Memory and Cognition (Original Exploratory Endpoint)

VISIONARY-MS
Long-Term
Open Label Extension

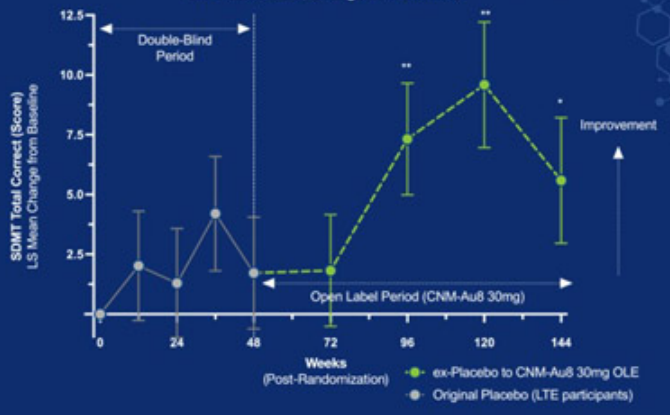
Original CNM-Au8

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



Ex-Placebo to CNM-Au8

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



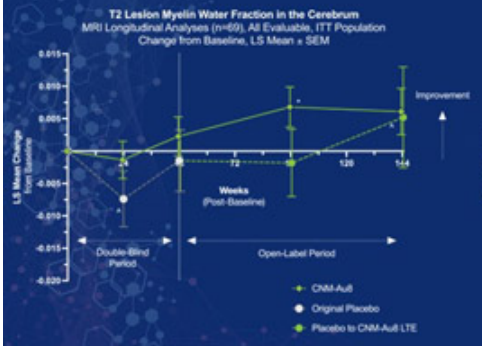
LTE: LS mean difference vs. randomization baseline: # p≤0.0001, *** p≤0.001, ** p≤0.01, *p≤0.05

MRI DTI | Evidence for Remyelination and Axonal Integrity

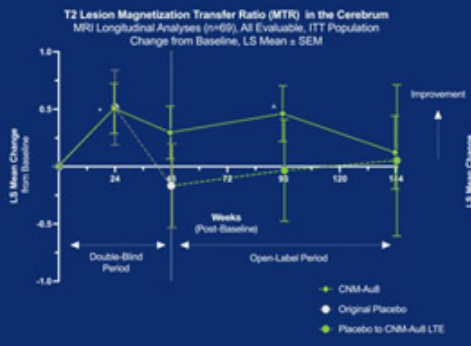
Diffusion Tensor Imaging | T2 Lesion Axial Diffusivity and Myelination Metrics

VISIONARY-MS
Long-Term
Open Label Extension

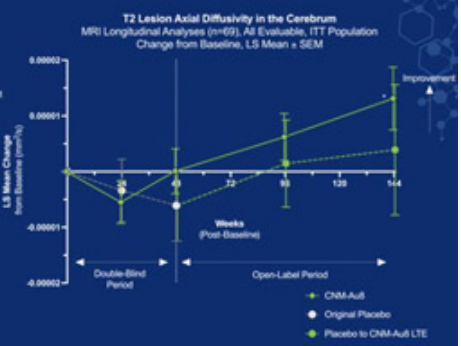
T2 Lesion MWF
In the Cerebrum



T2 Lesion MTR
In the Cerebrum



T2 Lesion Axial Diffusivity
In the Cerebrum



LTE: LS mean difference vs. randomization baseline: # p≤0.0001, *** p≤0.001, ** p≤0.01, *p≤0.05, ^p≤0.10
MWF: Myelin Water Fraction, MTR: Magnetization Transfer Ratio

CNM-Au8 Safety

CNM-Au8 treatment was safe and well-tolerated during the LTE

- Treatment emergent adverse events (TEAEs) were transient and predominantly mild-to-moderate
- 6 SAEs were reported over 82.9 years of cumulative participant follow-up including: (2) nephrolithiasis, (1) non-ST elevation myocardial infarction, (1) diverticulitis, (1) neutropenia, and (1) pneumonia; all resolved and were assessed as not related to CNM-Au8
- No dose limiting adverse events; average daily treatment compliance was 94% (bottles consumed/dispensed)

Most Common TEAEs (From Randomization to End of LTE) In LTE Participants	**Participants with TEAEs**	**Total TEAEs from Randomization**	**Events per 100-person exposure years**	**Poisson 95% CI**
Upper Respiratory Tract Infection	31	42	0.079	0.057 – 0.107
Headache	20	24	0.045	0.029 – 0.069
Urinary Tract Infection	11	19	0.036	0.022 – 0.056

Phase 2 CNM-Au8 VISIONARY-MS Trial | Long Term Extension Results

VISIONARY-MS STUDY

Long Term Open-Label Extension (LTE)

Michael Barnett MBBS FRACP PhD[1], Heidi Beadnall MBBS FRACP PhD[1], Alexander Klistorner PhD[1], Robert Sergott MD[2], Benjamin Greenberg MD MHS FANA FAAN CRND[3], Austin Rynders RN[4], Karen S. Ho PhD[4], Iacob Evan[4], Marjan Sepassi Pharm D[4], Jeremy Evan PA-C[4], Ryan McBride[4], Kyle McBride[4], Alan Hartford PhD[4], Robert Glanzman MD FAAN[4], Michael Hotchkiss[4] for the VISIONARY-MS Investigators

[1] Brain and Mind Centre, University of Sydney, Camperdown, NSW, Australia, [2] Thomas Jefferson University Annesley Eyeblaze Center, Philadelphia, PA, USA, [3] UT Southwestern Medical Center, Dallas, TX, USA, [4] Clene Nanomedicine, Inc., Salt Lake City, UT, USA, [5] Institut Clinical Research, Chatham, New Jersey, USA

CNM-Au8 30mg Was Safe and Well-Tolerated. Long-Term Treatment Resulted in Improved Clinical, Functional, and Structural Outcomes with Evidence Supporting Remyelination

Design Overview

Phase 2 Study: 48-Week Double-Blind Placebo-Controlled Treatment Period With Up to 96-Week Long-Term Open Label Extension (LTE) Through Week 144 (Post-Baseline)

- Enrolled stable relapsing remitting MS participants with chronic optic neuropathy on background DMTs (92% treated with DMT; 53% monoclonal antibody infusion, 32% oral)
- n=73 of 150 planned – study ended prematurely due to COVID pandemic-related enrollment challenges
- LTE was offered to participants in Australia; n= 55 were eligible and continued into the LTE

Safety (Primary Endpoint for the LTE Period)

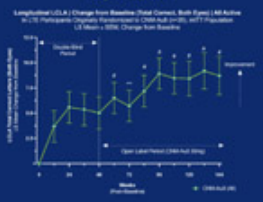
CNM-Au8 treatment was safe and well-tolerated during the LTE

- Treatment emergent adverse events (TEAEs) were transient and predominantly mild-to-moderate
- 6 SAEs were reported over 82.9 years of cumulative participant follow-up including: (2) nephrolithiasis, (1) non-ST elevation myocardial infarction, (1) diverticulitis, (1) neutropenia, and (1) pneumonia; all resolved and were assessed as not related to CNM-Au8 by the investigators
- No dose limiting adverse events; average daily treatment compliance was 94% (bottles consumed/dispensed)
- Most common TEAEs included: upper respiratory tract infection, headache, and urinary tract infection

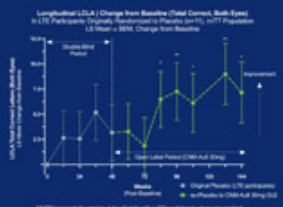
Most Common TEAEs (From Randomization to End of LTE) in LTE Participants	Total Participants with TEAE	Total TEAEs from Randomization	Events per 100-person exposure years	Poisson 95% CI
Upper Respiratory Tract Infection	31	62	0.079	0.057 – 0.107
Headache	20	24	0.045	0.029 – 0.069
Urinary Tract Infection	11	19	0.036	0.023 – 0.056

Clinical Endpoints | LCLA Change Across Eyes (Total Correct Letters)

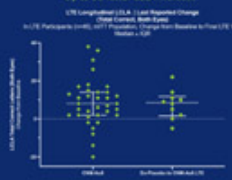
Original CNM-Au8



Ex-Placebo to CNM-Au8

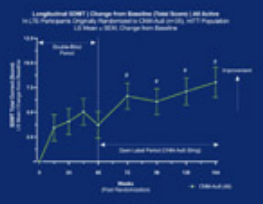


Up to 38-letter LCLA Increase

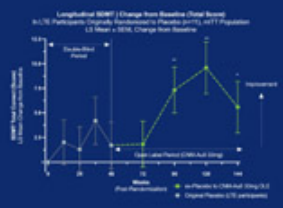


Clinical Endpoints | Symbol Digit Modalities Test (SDMT) (Working Memory & Cognition)

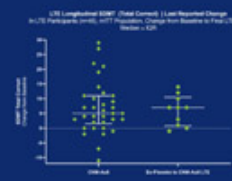
Original CNM-Au8



Ex-Placebo to CNM-Au8



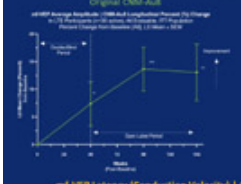
Up to 29-point SDMT Increase



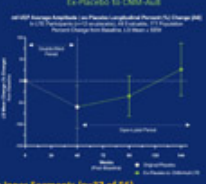
Functional Endpoints | Multi-Focal Visual Evoked Potential (mf-VEP) [of the Visual Pathway]

mf-VEP Amplitude (Signal Strength)

Original CNM-Au8

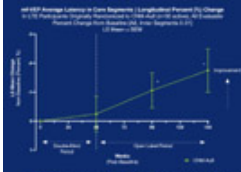


Ex-Placebo to CNM-Au8

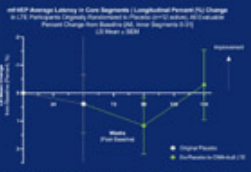


mf-VEP Latency (Conduction Velocity) | Core Inner Segments (n=32 of 56)

Original CNM-Au8

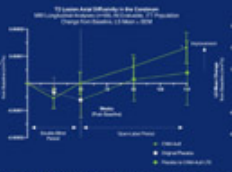


Ex-Placebo to CNM-Au8

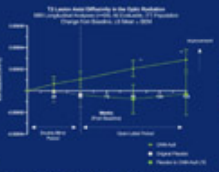


Structural Endpoints | MRI DTI T2 Lesion Axial Diffusivity

Cerebrum

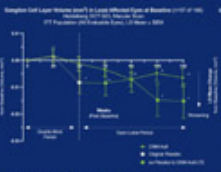


Optic Radiation

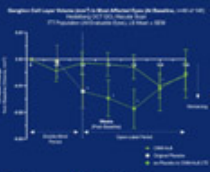


Structural Endpoints | Ganglion Cell Layer Volume (OCT)

In the Least Damaged Eyes [at Baseline]



In the Most Damaged Eyes [at Baseline]



LTE: MMRM analyses with age, gender, and baseline value as fixed covariates. LS mean difference vs. randomization baseline. # p<0.0001, *** p<0.001, ** p<0.01, * p<0.05, ^ p<0.10. Pre-specified study alpha was set at 0.10. For OCT endpoints the most affected eyes at baseline are defined post hoc as ≤15th percentile distribution at baseline or with inter-eye RNFL difference ≥6 µm or GCC difference of ≥8 µm (worst eye). mITT population included all valid clinical data. OCT and VEP data exclude one visit for one participant with an episode of acute optic neuritis. Acknowledgements: We thank the study participants and their families for their support and willingness to engage in clinical research. We thank the site investigators for their research excellence and dedication to patients. Disclosures: Dr. Greenberg is a consultant to and has equity in Clene. Clene employees have options and/or equity in Clene.

clene NANOMEDICINE

EVIDENCE FOR CLENE'S CNM-Au8® AS A TREATMENT FOR REPAIR AND REMYELINATION IN MULTIPLE SCLEROSIS PRESENTED IN THE EMERGING SCIENCE SESSION AT THE 2024 AMERICAN ACADEMY OF NEUROLOGY ANNUAL MEETING

- Long term extension of the Phase 2 VISIONARY-MS clinical trial of CNM-Au8 demonstrated significant evidence of repair and remyelination across multiple paraclinical endpoints (change from original baseline, $p<0.05$)
- Significantly improved clinical outcomes associated with long-term daily oral CNM-Au8® 30 mg treatment (change from original baseline; $p<0.05$)
- Long-term CNM-Au8 treatment, encompassing up to three years, was well-tolerated; no significant safety findings were observed
- First Phase 2 clinical MS trial of a non-immunomodulatory drug to meet a clinical outcome of improved function supporting remyelination and reparative effects

SALT LAKE CITY, April 16, 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 neurodegenerative diseases, including amyotrophic lateral sclerosis (ALS) and multiple sclerosis (MS), presented the Phase 2 VISIONARY-MS long term extension (LTE) study results at the 2024 American Academy of Neurology (AAN) Annual Meeting in Denver.

During the Emerging Science Session, Dr. Michael Barnett, MBBS, FRACP, FRCP, PhD, from the University of Sydney, presented data demonstrating improved clinical, functional, and structural outcomes associated with daily oral dosing of CNM-Au8 30 mg for up to three years of treatment.

These new long-term results from the Phase 2 VISIONARY-MS clinical trial demonstrated evidence supporting repair and remyelinating effects of CNM-Au8 treatment in patients originally randomized to CNM-Au8. The long-term results further enhance the trial's findings from the double-blind period, which demonstrated significant improvements on low contrast letter acuity and on the modified MS Functional Rating Scale, the study's primary and secondary endpoints, with continued improvement observed in the LTE.

The significant and concordant results across multiple paraclinical exploratory endpoints reinforce the evidence for sustained clinical benefit to study participants across multiple clinical outcome measures, associated with consistent improvements in neuronal function and remyelination.

"Phase 2 CNM-Au8 VISIONARY-MS Trial: Long-Term Extension Results" presentation key highlights:

Clinical improvements in cognition and vision

- Participants originally randomized to CNM-Au8 treatment experienced continued significant improvement in vision as measured by low contrast letter acuity. More than half of participants improved by 10 or more letters on a low-contrast Sloan eye chart, with increases of up to 38 letters (mixed model repeat measures, or MMRM vs. original baseline, $p < 0.001$).
- Participants originally randomized to placebo who transitioned to CNM-Au8 after the 48-week double blind period into the open label extension also experienced significant improvement in vision as measured by low contrast letter acuity following treatment with 30 mg CNM-Au8 (MMRM vs. original baseline, $p < 0.05$).
- Study participants treated with CNM-Au8 experienced up to 29 points of significant improvement (max score =110) in cognition and working memory as measured by the Symbol Digit Modality Test (SDMT) (MMRM vs. original baseline, $p < 0.001$).

Physiologic functional evidence of repair and remyelination

- Study participants treated with CNM-Au8 experienced significant improvements in both amplitude (MMRM vs. original baseline, $p < 0.01$) and latency (MMRM vs. original baseline, $p = 0.06$) as measured by multi-focal visual evoked potentials, physiologic measures of signal strength and speed along the visual pathway, markers of neuronal health and remyelination, respectively.

Structural evidence of repair and remyelination

- MRI measures of axial diffusivity showed significant improvements in T2 brain lesions in study participants treated with CNM-Au8 (MMRM vs. original baseline, $p < 0.05$).
- MRI measures of T2 lesion myelin water fraction (MWF) and magnetization transfer ratio (MTR), markers of remyelination, improved with long-term CNM-Au8 treatment (MWF: MMRM vs. original baseline, $p < 0.05$; MTR: MMRM vs. original baseline, $p = 0.06$).

CNM-Au8 was well-tolerated, and no significant safety findings were observed.

“The development of adjunctive therapies that not only prevent neurodegeneration, but also improve neuronal function with measurable clinical benefit, will fill a major unmet need for people living with MS. In the VISIONARY-MS trial, consistent improvements in multiple clinical and paraclinical endpoints over three years of adjunctive treatment with CNM-Au8 provide clear impetus for a definitive Phase 3 study,” stated Dr. Barnett.

Dr. Benjamin Greenberg, Head of Medical for Clene, noted, “Observing such a profound clinical benefit with corresponding improvements in physiologic measures utilizing a mechanism that does not target immune system modulation has never been demonstrated in prior multiple sclerosis trials. This is a very exciting data set that gives hope to the millions of people who are suffering from this disabling disease.”

The presentation is available in the Presentations section of the Clene website.

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 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 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,” “could,” “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, 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 clinical trials of our drug candidates may fail to demonstrate safety and efficacy to the satisfaction of regulatory authorities, or may not otherwise produce positive results, which may cause us to incur additional costs or experience delays in completing, or ultimately be unable to complete, the development and commercialization of our drug candidates; changes in government regulation or in practices relating to the pharmaceutical and biotechnology industries, including potential healthcare reform, could decrease the need for our drug candidates, or make it more difficult to obtain regulatory approvals for our drug candidates and commercialize them; we depend substantially on the successful commercialization of our drug candidates in the future, which may fail to materialize or may experience significant delays; we have a limited operating history, which may make it difficult to evaluate our current business and predict our future performance; and our ability to continue as a going concern requires that we obtain sufficient funding to finance our operations, which may not be available on acceptable terms, or at all, and a failure to obtain this necessary capital when needed could force us to delay, limit, reduce, or terminate our drug development or commercialization efforts; 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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