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): **May 29, 2026**

MONOPAR THERAPEUTICS INC.

(Exact name of registrant as specified in its charter)

Delaware	**001-39070**	**32-0463781**
(State or other jurisdiction of incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)

1000 Skokie Blvd., Suite 350, Wilmette, IL	**60091**
(Address of principal executive offices)	(Zip Code)

(847) 388-0349
Registrant's telephone number, including area code

N/A
(Former name or former address, if changed since last report)

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	**MNPR**	**The Nasdaq Stock Market LLC (Nasdaq Capital Market)**

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

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 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

On May 29, 2026, Monopar Therapeutics Inc. ("Monopar") presented results from a Phase 2 study (NCT04422431) evaluating the effect of ALXN1840 (tiomolibdate choline) on liver pathology and clinical symptoms in patients with Wilson disease at the European Association for the Study of the Liver ("EASL") International Liver Congress 2026.

The presentation is furnished as Exhibit 99.1 to this report and incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Presentation dated May 29, 2026.
104	Cover Page Interactive Data File - the cover page XBRL tags are embedded within the Inline XBRL document.

SIGNATURE

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

Monopar Therapeutics Inc.

Date: May 29, 2026

By: /s/ Quan Vu
Name: Quan Vu
Title: Chief Financial Officer


EASL CONGRESS
Barcelona Spain
27–30 May 2026
ALXN1840 (tiomolibdate choline) stabilizes liver disease and improves neurological symptoms as well as quality of life in treatment-experienced Wilson disease patients
Valentina Medici MD, FAASLD | UC Davis Medical Center, Sacramento, CA, USA
Aftab Ala, Karl Heinz Weiss, Edward J. Gane, Zoe Mariño, Chandler Robinson, Andrew Cittadine, Declan Tuffy, Marina Berenguer, George Boon Bee Goh, Thomas Sandahl, Fred Askari

Wilson Disease (WD)

EASL CONGRESS 2026

- **Wilson disease** is a rare autosomal recessive disorder of impaired copper (Cu) transport
- Cu accumulates in the **liver** and **brain**, causing organ damage
- Inadequately treated or untreated, Cu accumulation leads to **neuropsychiatric manifestations, hepatic failure**, and ultimately **death**
- Standard of care therapies (D-penicillamine, trientine, and zinc) carry safety risks such as paradoxical neurological worsening[1]

~1 in 30,000

Estimated prevalence worldwide[1]

~25%

Of patients with neurologic WD paradoxically worsen on chelator therapy[2,3]

Lifelong

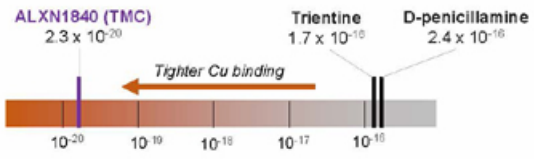
Treatment burden — requires cumbersome multi-dose daily regimen, separated from food

1. European Association for the Study of the Liver. *J Hepatol*. 2025 Feb 22:S0168-8278(24)02706-5. 2. Brewer GJ et al. *Arch Neurol*. 1987 May;44(5):490-3. 3. Antos A et al. Neurol Sci. 2023 Oct;44(10):3443-3455.

Tiomolibdate Choline (TMC; ALXN1840)

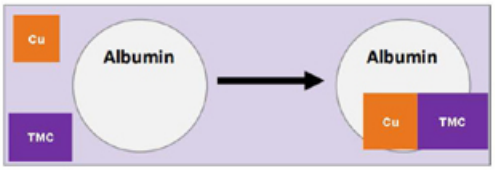
EASL CONGRESS 2026

CU-BINDING AFFINITY (K_D) OF WD THERAPIES



Tiomolibdate choline (TMC, ALXN1840) demonstrates **superior Cu binding affinity**[1] and specificity compared to currently approved chelators

TMC-CU-ALBUMIN FORMS A TRIPARTITE COMPLEX



Tiomolibdate choline is an albumin tripartite complex (ATC) activator, binding Cu and albumin to form a stable tripartite complex — **mobilizing** and **sequestering** hepatic Cu[2]

1. Kim P, et al. *Biomedicines*. 2021;9(12):1861. 2. Zhang L, et al. *Biochemistry*. 2009;48(5):891–897.

TMC Protects the Liver by Binding and Neutralizing Hepatic Cu

EASL CONGRESS 2026

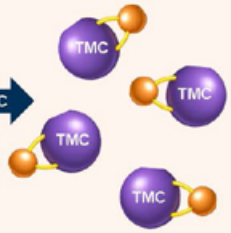
Differentiated from SoC, TMC's binding affinity is strong enough to **inhibit reactivity of free Cu** and **remove Cu from metallothionein**[1] — directly protecting the liver

TMC AND REACTIVE FREE CU

Excess **reactive free Cu** causes tissue **toxicity**

+TMC

TMC directly binds Cu, **preventing redox reactivity**[2]

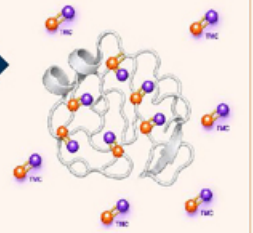


TMC AND METALLOTHIONEIN (MT)

Catabolism of Cu–MT **releases reactive Cu**[3]

+TMC

TMC prevents reactive Cu release by both **binding Cu and removing it from MT**[4]



1. Smirnova J et al. *Sci Rep*. 2018 Jan 23;8(1):1463. 2. Data on file. 3. Klein D et al. *Eur J Clin Invest*. 1998 Apr;28(4):302-10. 4. Czachor JD et al. *J Inorg Biochem*. 2002 Jan 15;88(2):213-22.

48-week Liver Biopsy Study Methods

Open-label, multicenter, pathologist-blinded trial of TMC monotherapy in **treatment-experienced** (≥ 1 year) patients with WD

Baseline liver biopsy:
Week 0
(pre-treatment)

Follow-up liver biopsy:
Week 48
(post-treatment)

LIVER BIOPSY ENDPOINTS

Biopsy endpoints assessed by pathologist **blinded to treatment status**

Hepatic Cu concentration *Primary Endpoint*

Fibrosis *(NASH CRN Fibrosis Stage, NAS total score)*

Steatosis (*NAS steatosis grade, hepatocellular ballooning)*

Inflammation (*lobular inflammation, portal inflammation)*

OTHER ENDPOINTS

Neurologic
(Unified Wilson Disease Rating Scale)

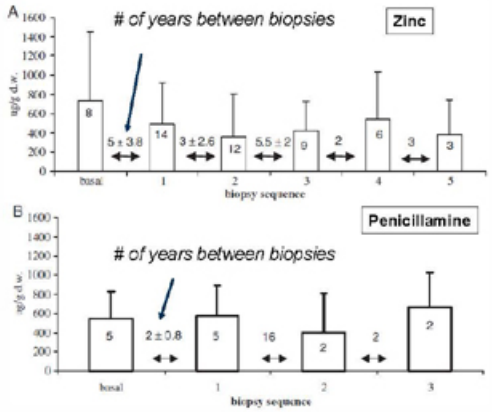
Global
(Clinical Global Impressions scale)

Quality of Life
(EuroQol-5D-5L)

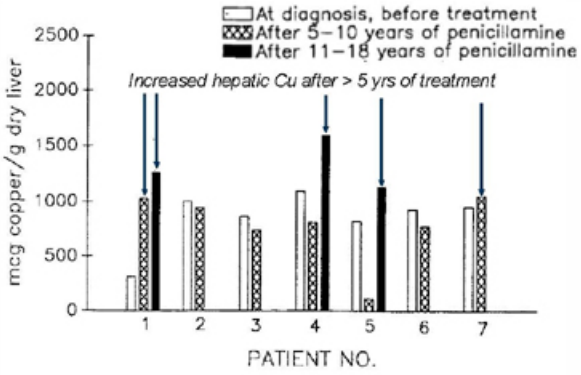
Safety
(adverse events, physical exam)

Literature Shows Stable/Increased SoC Hepatic Cu Change

Serial liver biopsy studies of SoC show **stable/increased hepatic Cu** over years on treatment



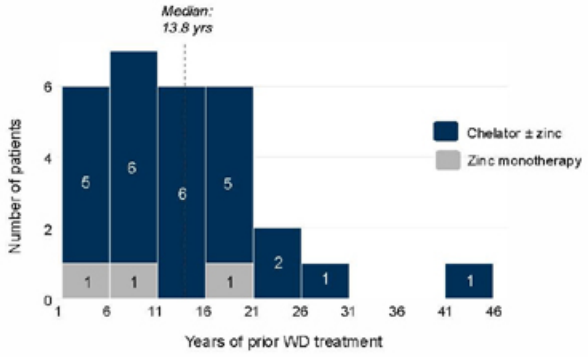
Adapted from Medici V et al. *J Clin Gastroenterol.* 2006 Nov-Dec;40(10):936-41.



Adapted from Scheinberg IH et al. *Lancet.* 1987 Jul 11;2(8550):95.

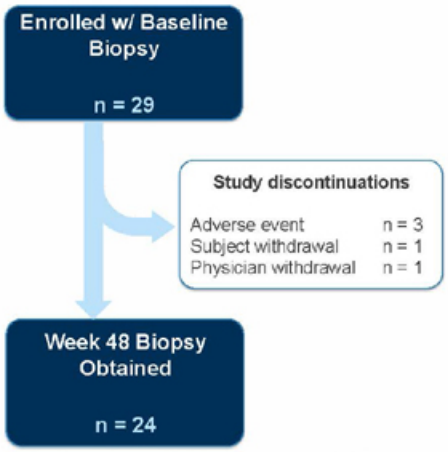
Study Enrolled a Heavily Pre-treated Population

CUMULATIVE DURATION OF PRIOR WD TREATMENT



Prior WD treatment	n	Median (yrs)	Min (yrs)	Max (yrs)
Chelator ± zinc	26	13.9	1.2	41.9
Zinc monotherapy	3	8.7	3.4	19.8

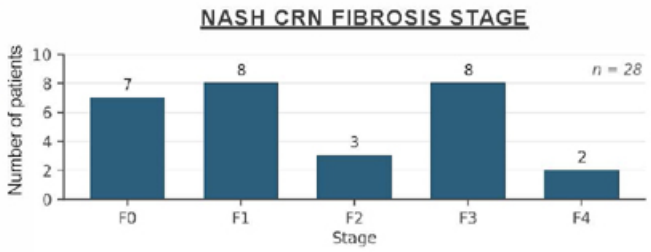
PATIENT ENROLLMENT

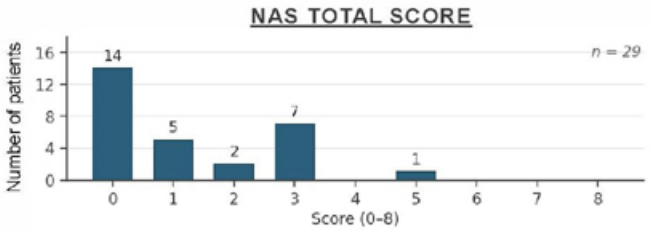


Numbers above are for the full analysis set (n=29); 31 enrolled but 2 had no baseline liver biopsy

Baseline Demographics and Characteristics

	Enrolled patients n=29
Demographics	
Male sex, n (%)	19 (65.5%)
Age in years, median (IQR)	35.0 (17.0)
Clinical characteristics	
Years of prior WD treatment, median (IQR)	13.8 (10.9)
Medical history of cirrhosis, n (%)	12 (41.4%)
Abnormal liver ultrasound, n (%)	8 (27.6%)
UWDRS Part III score, median (IQR)	4.0 (9.0)
MELD score, median (IQR)	7.0 (2.0)

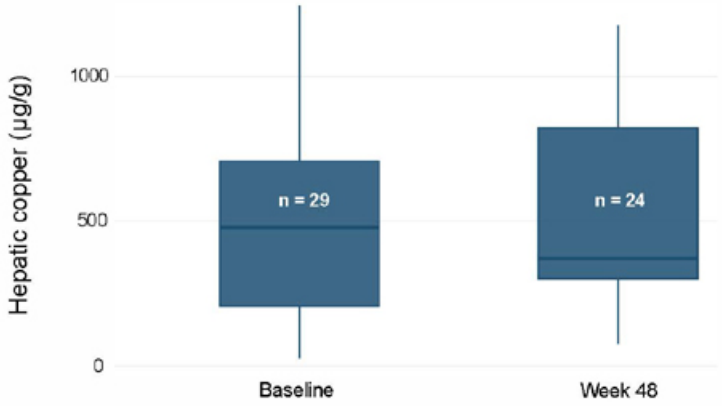




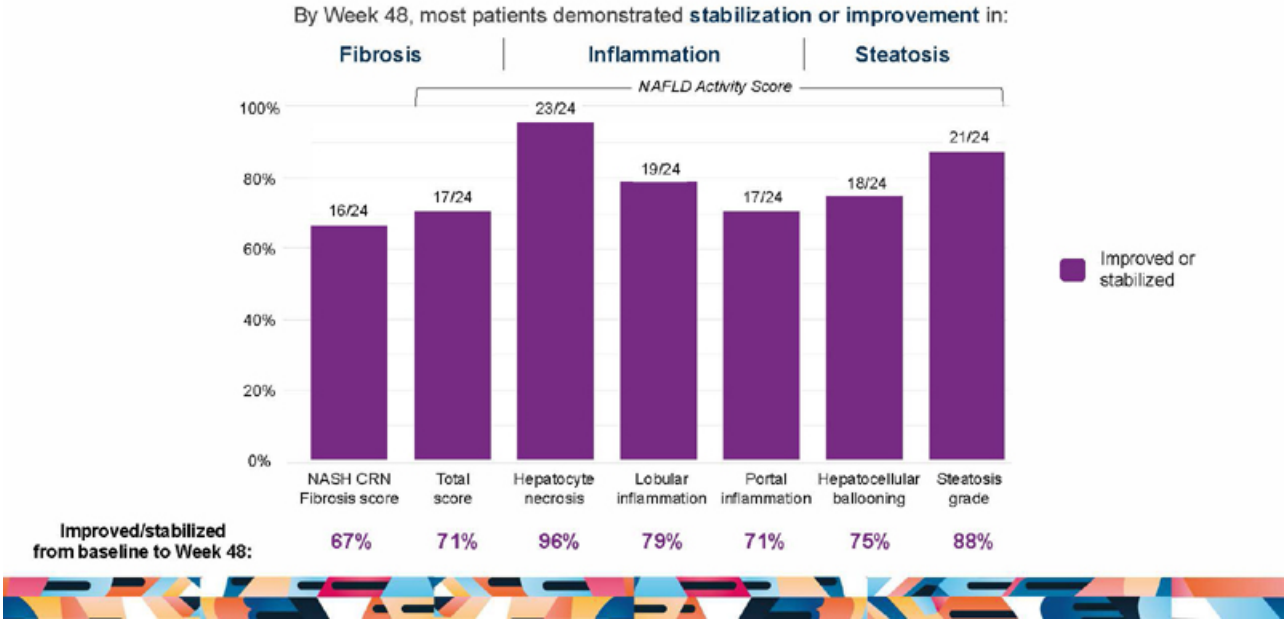
TMC Also Shows No Significant Change in Hepatic Cu

No statistically significant difference in hepatic Cu concentration after 48 weeks of treatment, **consistent with expected results** in heavily pre-treated patients

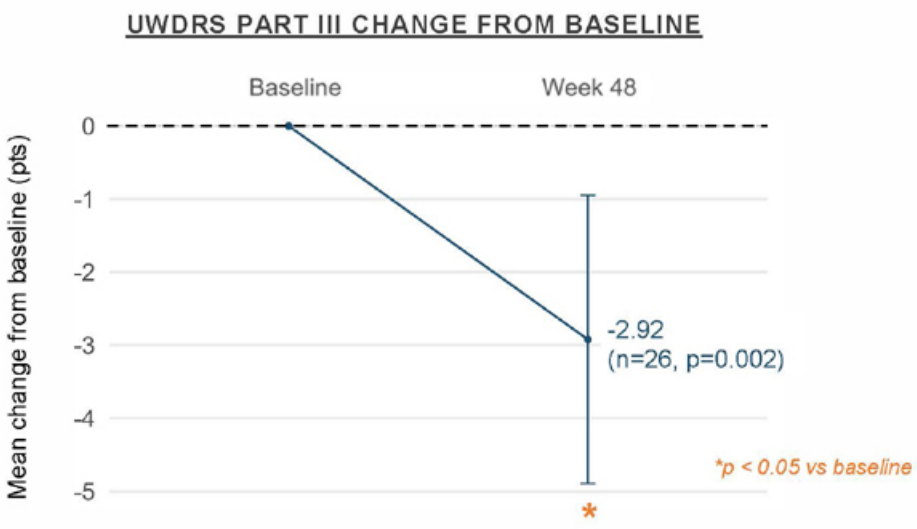
OBSERVED CHANGE FROM BASELINE IN HEPATIC CU CONCENTRATION (µg/g)



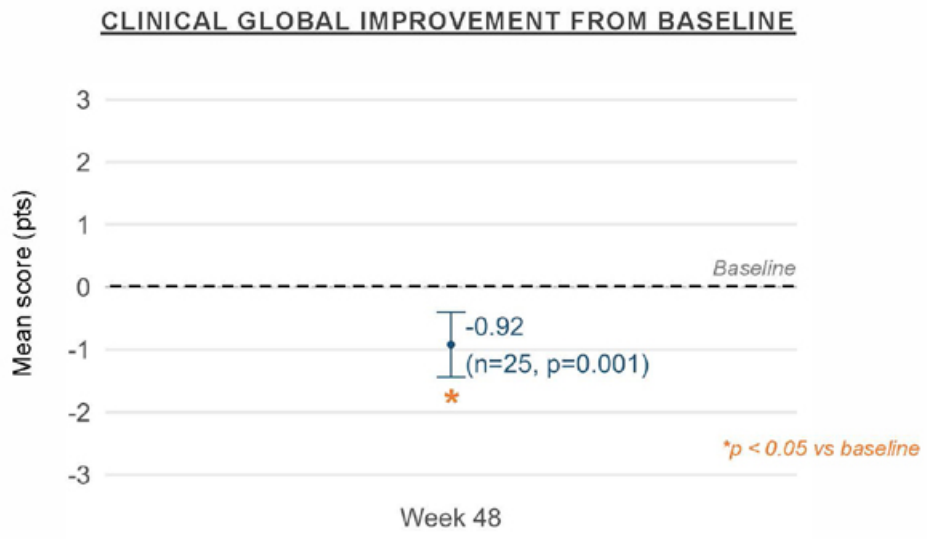
TMC Treatment Leads to Hepatic Stabilization and Improvement



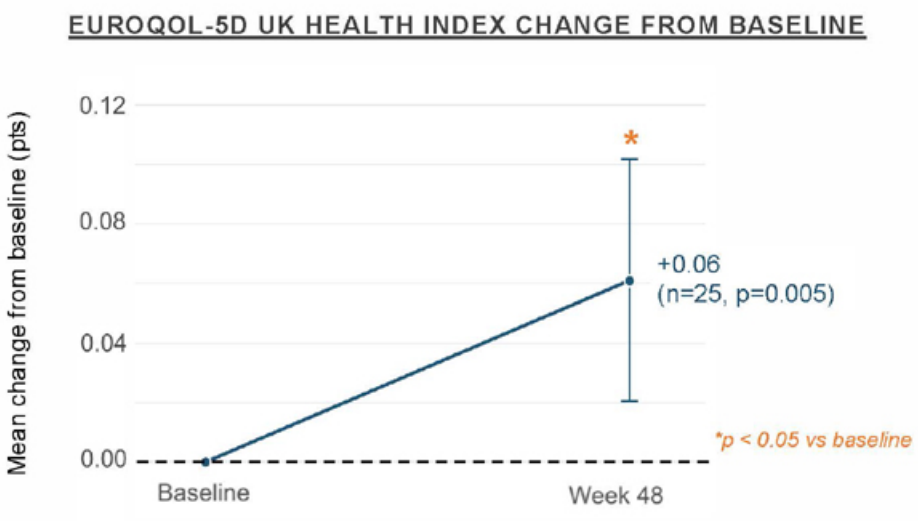
Neurological WD Symptoms Significantly Improved at Week 48



Global Clinical Disease Burden Significantly Improved at Week 48



Patient-reported Quality of Life Significantly Improved at Week 48



TMC is Well Tolerated, with Few Severe Adverse Events

- TMC was generally well tolerated; most treatment-emergent adverse events (TEAEs) were **nonserious** and **Grade 1 or 2** in severity
- No deaths were reported
- One event of DILI was reported
  - Patient was stable on TMC until Week 47; developed UTI and initiated on ciprofloxacin with DILI onset very shortly after
  - Adjudicated by independent panel as unlikely relationship to TMC

TEAEs IN SAFETY POPULATION (N=31)

	Primary period n=31	Extension period n=25
TEAEs	30 (96.8%)	19 (76.0%)
Related	24 (77.4%)	3 (12.0%)
Grade 1	17 (54.8%)	3 (12.0%)
Grade 2	16 (51.6%)	0
Grade 3	5 (16.1%)	0
Grade 4	0	0
Grade 5	0	0

Safety population (n=31) includes all patients who received ≥ 1 dose of TMC, including 2 patients enrolled without an evaluable baseline liver biopsy (full analysis set, n=29). Severity grade counts are subject-level and not mutually exclusive.

Conclusions

- Hepatic Cu concentration was **stable** after 48 weeks of TMC, consistent with published serial liver biopsy studies of WD patients treated with standard-of-care
- Treatment with TMC in heavily pre-treated patients resulted in **hepatic stabilization/improvement**
- TMC treatment **significantly improved neurological symptoms, global clinical status,** and **patient-reported quality of life**
- With the unique ability to **bind Cu with high affinity**, **remove excess Cu** from metallothionein, and **activate albumin-tripartite-complex (ATC) formation,** TMC has the potential to provide a therapeutic advantage over standard-of-care for the long-term treatment of WD

EASL CONGRESS
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Questions?