
**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 22, 2024



NEUROBO PHARMACEUTICALS, INC.

(Exact name of Registrant as Specified in Its Charter)

Delaware (State or other jurisdiction of incorporation)	001-37809 (Commission File Number)	47-2389984 (IRS Employer Identification No.)
--	---	---

**545 Concord Avenue, Suite 210
Cambridge, Massachusetts 02138**

(Address of principal executive offices)

02138
(Zip Code)

(857) 702-9600
(Registrant's telephone number, including area code)

Not applicable
(Former name, former address and former fiscal year, 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, par value \$0.001 per share	NRBO	The Nasdaq Stock Market LLC

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 8.01. Other Events.

On May 22, 2024, NeuroBo Pharmaceuticals, Inc. issued a press release announcing that pre-clinical data suggests that DA-1241, a novel G-Protein-Coupled Receptor 119 (GPR119) agonist, in combination with semaglutide (Wegovy®), improves liver fibrosis and demonstrates additive hepatoprotective effects in pre-clinical metabolic dysfunction-associated steatohepatitis (MASH) models compared to either treatment alone. Members of the Dong-A ST Research Center and Contract Research Organization, Gubra, will present the data in two poster presentations at the EASL Congress 2024, taking place June 5-8, in Milan, Italy, as well as virtually. A copy of the press release is filed as Exhibit 99.1 to this Current Report on Form 8-K and incorporated herein by reference.

Information contained on or accessible through any website reference in the press release is not part of, or incorporated by reference in, this Current Report on Form 8-K, and the inclusion of such website addresses in this Current Report on Form 8-K by incorporation by reference of the press release is as inactive textual references only.

Item 9.01. Financial Statements and Exhibits.**(d) Exhibits**

Exhibit Number	Exhibit Description
99.1	Press Release dated May 22, 2024.
104	Cover Page Interactive Data File (embedded within Inline XBRL document).

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.

NEUROBO PHARMACEUTICALS, INC.

Date: May 22, 2024

By: /s/ Hyung Heon Kim

Hyung Heon Kim

President and Chief Executive Officer



NeuroBo Pharmaceuticals' DA-1241 in Combination with Semaglutide Improves Liver Fibrosis and Demonstrates Additive Hepatoprotective Effects in Pre-Clinical MASH Models Compared to Either Treatment, Alone

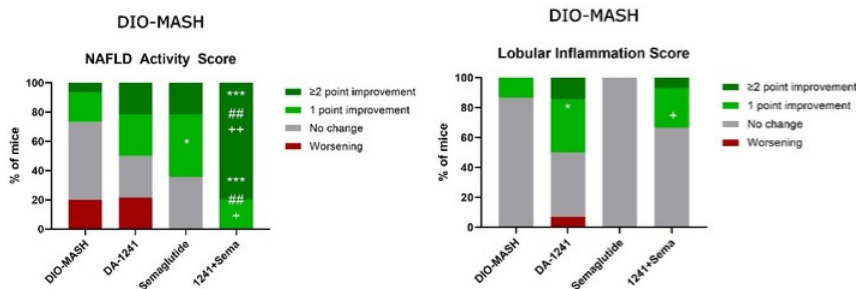
Data Will be Presented in Two Posters at the EASL Congress 2024

CAMBRIDGE, Mass., May 22, 2024 –**NeuroBo Pharmaceuticals, Inc.** (Nasdaq: NRBO), a clinical-stage biotechnology company focused on the transformation of cardiometabolic diseases, today announced that pre-clinical data suggests that DA-1241, a novel G-Protein-Coupled Receptor 119 (GPR119) agonist, in combination with semaglutide (Wegovy®), improves liver fibrosis and demonstrates additive hepatoprotective effects in pre-clinical metabolic dysfunction-associated steatohepatitis (MASH) models compared to either treatment alone. Members of the Dong-A ST Research Center and Contract Research Organization, Gubra, will present the data in two poster presentations at the EASL Congress 2024, taking place June 5-8, in Milan, Italy, as well as virtually.

“The data being presented at EASL further strengthen the pre-clinical evidence that DA-1241’s activation of GPR119 has therapeutic potential for the reduction of hepatic steatosis, inflammation, fibrosis, and improved glucose control,” stated Hyung Heon Kim, President and Chief Executive Officer of NeuroBo. “In April, we completed enrollment for Part 1 of our Phase 2a clinical trial of DA-1241 in MASH and continue to enroll patients in Part 2, exploring the efficacy of DA-1241 in combination with sitagliptin, a DPP-4 inhibitor, which we believe will show synergistic effects compared to DA-1241, alone. The new data being presented at the EASL Congress explored DA-1241 in combination with semaglutide, a GLP1R agonist. Importantly, the data suggests, for the first time, a beneficial combination effect of DA-1241 and semaglutide in the treatment of liver fibrosis, which may be attributed to augmented inhibition of fibrogenesis and inflammation in the liver. The data also demonstrates more than additive effects on metabolic, biochemical, and histological endpoints in GAN DIO-MASH mice, highlighting the therapeutic potential of dual targeting GPR119 and GLP1R function in MASH with liver fibrosis. We eagerly anticipate reporting top-line data from both parts of the ongoing Phase 2a clinical trial in MASH in the fourth quarter of this year. We continue to believe the unique mechanism of DA-1241, targeting the inflammation associated with MASH, will translate into a safe and effective treatment for this disease.”

- **Abstract Title:** *Additive Hepatoprotective Effects of DA-1241, a Novel GPR119 Agonist, in Combination with Semaglutide in the GAN Diet-Induced Obese and Biopsy-Confirmed Mouse Model of MASH*
 - **Authors:** Monika Lewinska, Malte H. Nielsen, Susanne Pors, Henrik B. Hansen, Il Hoon Jung, Hyung Heon Kim, Michael Feigh, Mi-Kyung Kim
 - **Presenter Name:** Dr. Michael Feigh, Vice President Scientific Research, Gubra
 - **Abstract Number:** 1950
 - **Abstract ID:** THU-232
 - **Session:** Poster - MASLD: Experimental and pathophysiology
 - **Session Date:** Thursday, June 6, 2024
 - **Session Time:** 8:30 am – 6:00 pm CET
-

Male C57BL/6J mice were fed the GAN diet for 36 weeks before treatment initiation. Only biopsy-confirmed GAN DIO-MASH mice (steatosis score =3, lobular inflammation score ≥ 2 , fibrosis stage F2-F3) were stratified to treatment (n=14-15 per group). GAN DIO-MASH mice received once daily treatment with vehicle, DA-1241 (100 mg/kg, PO) or semaglutide (30 nmol/kg, SC) alone or in combination for 8 weeks. Within-subject comparisons (pre vs. post treatment) were performed for liver biopsy histopathological NAFLD Activity Score (NAS) and Fibrosis Stage. Terminal quantitative endpoints included plasma/liver biochemistry, liver histomorphometry and RNA sequencing. DA-1241 was weight-neutral and did not influence liver weight. In contrast, semaglutide robustly reduced body weight by approximately 25% and improved hepatomegaly in GAN DIO-MASH mice with or without combination treatment. There was no additional weight loss in the combination group compared to semaglutide alone. Each monotherapy ameliorated plasma transaminases and liver cholesterol levels, with combination therapy providing further improvement compared to monotherapies.

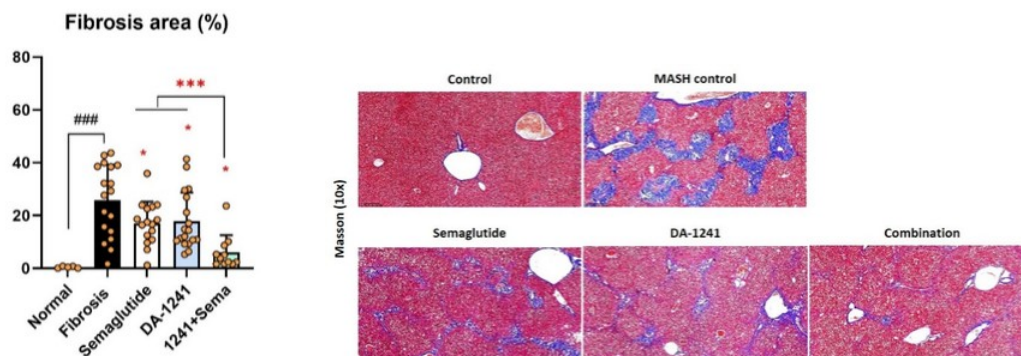


DA-1241 and semaglutide monotherapy each improved NAS (≥ 2 -point) in 21% of mice, whereas combination treatment led to marked improvements (≥ 2 -point in 80% of mice and ≥ 1 -point in all mice), driven by reduction in steatosis and lobular inflammation scores. Correspondingly, combination therapy synergistically promoted quantitative histology for steatosis (%-area of liver lipids, % lipid-laden hepatocytes) compared to monotherapies. While treatments did not significantly influence quantitative markers of fibrosis (PSR, Col1a1), DA-1241 and semaglutide in monotherapy lowered α -SMA levels with further improvement in combination treatment, suggesting additive inhibitory effects on fibrogenesis. Liver transcriptome analysis demonstrated a significant increase in the number of differential expressed genes with prominent signature changes in lipid metabolism, chemokine signaling, and fiber proteins following combination therapy compared to monotherapies.

- **Abstract Title:** DA-1241, a GPR119 Agonist, Combined with Semaglutide Synergistically Improved Liver Fibrosis in Mice with CCl₄ Induced Liver Fibrosis
- **Authors:** Il Hoon Jung, Tae Hyung Kim, Sujin Lee, Yuna Chae, Hyung Heon Kim, Mi-Kyung Kim
- **Presenter Name:** Il Hoon Jung, Dong-A ST Research Center
- **Abstract Number:** 117
- **Abstract ID:** THU-531
- **Session:** Poster - Fibrosis / Stellate cell biology
- **Session Date:** Thursday, June 6, 2024
- **Session Time:** 8:30 am – 6:00 pm CET

Liver fibrosis mice were generated by feeding a western diet in adjunctive with CCl₄ injections. After administering CCl₄ twice weekly for 3 weeks, mice with elevated plasma ALT levels were allocated to receive DA-1241 (oral) or semaglutide (subcutaneous) alone and in combination for 4 weeks. At the end of treatment, semaglutide reduced body weight by approximately 17% ($p < 0.05$ vs. vehicle control), while DA-1241 (-2.8%) showed little effect. There was no additional weight loss in the combination group (-19%, $p < 0.05$) compared to semaglutide alone. After four-week-treatment, three drug-treated groups had significantly lower plasma ALT levels than the vehicle control group, suggesting alleviation of liver damage.

CCl₄-induced liver fibrosis



Collagen fiber deposition was prominent in mice treated with vehicle compared to the normal control group. However, DA-1241 or semaglutide alone lowered collagen-positive area compared to the vehicle-treated group (17.8%, 17.1% vs. 25.8%, $p < 0.05$), and their combination therapy elicited a further reduction to 6.05% ($p < 0.05$) compared with each treatment alone, which was recapitulated in changes of fibrosis score. Hepatic hydroxyproline contents and gene expression of various collagen subtypes (*Col1a1*, *Col3a1*, *Col5a1*, *Col6a1*) were also altered correspondingly, supporting the beneficial combination effects against the liver fibrogenesis. Notably, gene expression of Hedgehog-interacting protein (*Hhip*), a suppressor of hepatic stellate cell activation, was lower in mice with liver fibrosis than in normal mice, and were increased by DA-1241 or semaglutide alone. Intriguingly, their combination therapy fully restored the gene expression of *Hhip*. Additionally, the expression of inflammatory cytokines (*Tnfa*, *IL1b*, *Ccl2*, *Cxcl10*) was significantly reduced by each monotherapy, and combination treatment reduced gene expression of *Tnfa* and *Cxcl10* more than monotherapy. These data indicate that liver inflammation status has improved as well.

After the presentations, the posters will be accessible within the "Presentations" section of NeuroBo's website at: <https://www.neurobopharma.com/events-presentations/presentations>.

About DA-1241

DA-1241 is a novel G-Protein-Coupled Receptor 119 (GPR119) agonist with development optionality as a standalone and/or combination therapy for both MASH and type 2 diabetes (T2D). Agonism of GPR119 in the gut promotes the release of key gut peptides GLP-1, GIP, and PYY. These peptides play a further role in glucose metabolism, lipid metabolism and weight loss. DA-1241 has beneficial effects on glucose, lipid profile and liver inflammation, supported by potential efficacy demonstrated during in vivo preclinical studies. The therapeutic potential of DA-1241 has been demonstrated in multiple pre-clinical animal models of MASH and T2D where DA-1241 reduced hepatic steatosis, inflammation, fibrosis, and improved

glucose control. Furthermore, in Phase 1a and 1b trials, DA-1241 was well tolerated in both healthy volunteers and those with T2DM.

About NeuroBo Pharmaceuticals

NeuroBo Pharmaceuticals, Inc. is a clinical-stage biotechnology company focused on transforming cardiometabolic diseases. The company is currently developing DA-1241 for the treatment of Metabolic Dysfunction-Associated Steatohepatitis (MASH) and is developing DA-1726 for the treatment of obesity. DA-1241 is a novel G-protein-coupled receptor 119 (GPR119) agonist that promotes the release of key gut peptides GLP-1, GIP, and PYY. In preclinical studies, DA-1241 demonstrated a positive effect on liver inflammation, lipid metabolism, weight loss, and glucose metabolism, reducing hepatic steatosis, hepatic inflammation, and liver fibrosis, while also improving glucose control. DA-1726 is a novel oxyntomodulin (OXM) analogue that functions as a glucagon-like peptide-1 receptor (GLP1R) and glucagon receptor (GCGR) dual agonist. OXM is a naturally-occurring gut hormone that activates GLP1R and GCGR, thereby decreasing food intake while increasing energy expenditure, thus potentially resulting in superior body weight loss compared to selective GLP1R agonists. For more information, please visit www.neurobopharma.com.

Forward Looking Statements

Certain statements in this press release may be considered forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Words such as "believes", "expects", "anticipates", "may", "will", "should", "seeks", "approximately", "intends", "projects", "plans", "estimates" or the negative of these words or other comparable terminology (as well as other words or expressions referencing future events, conditions or circumstances) are intended to identify forward-looking statements. Forward-looking statements are predictions, projections and other statements about future events that are based on current expectations and assumptions and, as a result, are subject to risks and uncertainties. Many factors could cause actual future events to differ materially from the forward-looking statements in this press release, including, without limitation, those risks associated with NeuroBo's ability to execute on its commercial strategy; the timeline for regulatory submissions; ability to obtain regulatory approval through the development steps of NeuroBo's current and future product candidates, the ability to realize the benefits of the license agreement with Dong-A ST Co. Ltd., including the impact on future financial and operating results of NeuroBo; the cooperation of our contract manufacturers, clinical study partners and others involved in the development of NeuroBo's current and future product candidates; potential negative interactions between our product candidates and any other products with which they are combined for treatment; NeuroBo's ability to initiate and complete clinical trials on a timely basis; our ability to recruit subjects for its clinical trials; whether NeuroBo receives results from NeuroBo's clinical trials that are consistent with the results of pre-clinical and previous clinical trials; impact of costs related to the license agreement, known and unknown, including costs of any litigation or regulatory actions relating to the license agreement; effects of changes in applicable laws or regulations; effects of changes to NeuroBo's stock price on the terms of the license agreement and any future fundraising; and other risks and uncertainties described in our filings with the Securities and Exchange Commission, including our most recent Annual Report on Form 10-K. Forward-looking statements speak only as of the date when made. NeuroBo does not assume any obligation to publicly update or revise any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by law.

Contacts:

NeuroBo Pharmaceuticals

Marshall H. Woodworth
Chief Financial Officer
+1-857-299-1033
marshall.woodworth@neurobopharma.com

Rx Communications Group

Michael Miller
+1-917-633-6086
mmiller@rxir.com
