UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 6-K

REPORT OF FOREIGN PRIVATE ISSUER
PURSUANT TO RULE 13a-16 OR 15d-16 UNDER
THE SECURITIES EXCHANGE ACT OF 1934

For the month of October 2026

Commission File Number: 001-39173

NovaBridge Biosciences

2440 Research Boulevard, Suite 400
Rockville, MD 20850
(Address of principal executive offices)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F.

Form 20-F ☒ Form 40-F ☐

NovaBridge Biosciences (the "Company") reports that clinical data from the ongoing Phase 1 clinical trial of ragistomig (also known as "ABL503" or "TJ-L14B"), a bispecific antibody targeting both PD-L1 and 4-1BB being jointly developed by the Company and ABL Bio Inc. ("ABL Bio"), were presented in a poster at the European Society for Medical Oncology (ESMO) Congress 2026 by ABL Bio in Madrid, Spain, on October 23, 2026.

Incorporation by Reference

The information set forth in this report on Form 6-K shall be deemed to be incorporated by reference into the Company's Registration Statements on Form F-3 (File No. 333-286954) and Form S-8 (File No. 333-239871, File No. 333-256603, File No. 333-265684, File No. 333-279842, File No. 333-290195 and File No. 333-298369) (including any prospectuses forming a part of such registration statements) and to be a part thereof from the date on which this Form 6-K is furnished, to the extent not superseded by documents or reports subsequently filed or furnished.

EXHIBIT INDEX

Exhibit No.	Description
99.1	Poster – Dose Optimization of Ragistomig (ABL503/TJ-L14B), a PD-L1x 4-1BBBispecific Antibody

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, thereunto duly authorized.

NovaBridge Biosciences

By : /s/ Kyler Lei
Name : Kyler Lei
Title : Chief Financial Officer

Date: October 23, 2026

Final Publication Number (FPN): 1066P

Dose Optimization of Ragistomig (ABL503/TJ-L14B), a PD-L1 x 4-1BB Bispecific Antibody

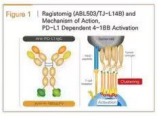
G. Falchook[1], E. Garon[2], M. Velez[3], A. El-Khoueiry[4], J. Thomas[5], A. Tolcher[6], A. Spira[8], D. Yardley[9], O. Dsvar[8], B.Keam[10], H.R. Kim[11], S.J. Shin[12], D.H. Lee[13], K.H. Park[14], J. Jeon[15], J. Park[15], J. Park[16], S. Oh[16], P. Dennis[17], C. Xu[17]

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Background

- Ragistomig (ABL503/TJ-L14B), a bispecific antibody targeting PD-L1 and 4-1BB, has been engineered to restrict 4-1BB costimulation to PD-L1 expressing tumor microenvironment, thereby reducing systemic hepatotoxicity associated with 4-1BB monoclonal antibodies, while enhancing antitumor activity and potentially overcoming resistance to PD-(L)1 checkpoint inhibitors. Figure 1
- Clinical experience with ragistomig administered 3-5mg/kg Q2W demonstrated encouraging antitumor activity, with an ORR of 26.9% and a DCR of 69.2%, including responses in patients whose disease had relapsed after or was refractory to prior anti-PD-(L)1 therapy[1].
  Further dose optimization was pursued to improve the therapeutic window for combination development, including evaluation of extended-interval Q6W dosing. Previously, extending the dosing interval to Q6W at 3mg/kg was associated with a more favorable safety profile while maintaining clinically meaningful antitumor activity compared with Q2W dosing, and it supported the expansion of the Q6W evaluation to 5 mg/kg.[2]

Figure 1 | Ragistomig (ABL503/TJ-L14B) and Mechanism of Action, PD-L1 Dependent 4-1BB Activation



Method

- ABL503-1001 is a first-in-human, multicenter, open-label, phase 1 trial of Ragistomig in advanced or metastatic relapsed/refractory solid tumors. (NCT04762641). Figure 2
- Following evaluation of 3 and 5 mg/kg Q2W in dose expansion part 2A, additional patients were enrolled in Part 2B and treated with 3 or 5 mg/kg Q6W. Tolerability, antitumor activity, pharmacokinetics (PK), and pharmacodynamic (PD) biomarkers were assessed.
- Data from both the dose-escalation and dose-expansion part cohorts were included in the exposure-safety and efficacy analyses. Exposure-safety relationships were evaluated using logistic regression models relating individual average PK exposure (C_{avg}) to the incidence of key adverse events. Efficacy was evaluated across dose levels to assess dose dependence and potential response saturation. The study was not designed or powered to compare different dose levels or dosing schedules. All between-cohort comparisons are descriptive.

Figure 2 | Phase 1 Study Design



Results

Baseline Demographics and Clinical Characteristics Table 1

- As of 23 Aug 2026, a total of 95 subjects had been treated with Ragistomig monotherapy across the dose-escalation and dose-expansion parts of the study. Among them, 42 subjects were enrolled in the 3 and 5 mg/kg Q6W cohorts, with treatment ongoing in 4 subjects.
- Most subjects across the 3 and 5 mg/kg cohorts were heavily pretreated, with 66.7% of Q6W subjects and 54.8% of Q2W subjects having received ≥3 prior lines of systemic therapy. Most subjects had also received prior IO therapy, including 64.5% (20/31) of subjects in the Q2W cohorts and 100% (42/42) in the Q6W cohorts.

Table 1 | Baseline Demographics and Clinical Characteristics As of 23-Aug-2026

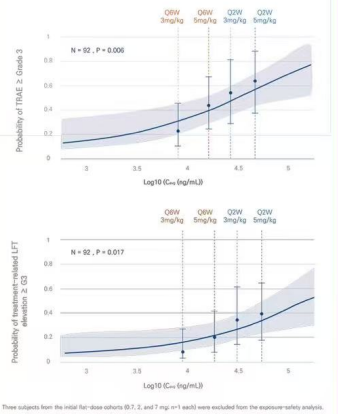
	0.7, 2, 7 mg Q2W (N=3)	0.3 mg/kg Q2W (N=3)	1 mg/kg Q2W (N=3)	2mg/kg Q2W (N=3)	3 mg/kg Q2W (N=11)	5 mg/kg Q2W (N=14)	3 mg/kg Q6W (N=22)	5 mg/kg Q6W (N=22)	7 mg/kg Q2W (N=4)	10 mg/kg Q2W (N=3)
Age										
Mean	58.7	48.0	58.5	54.0	63.7	65.2	68.0	64.9	64.8	60.0
ECOG Performance Status,% (n)										
0	33.3% (1/3)	66.7% (2/3)	33.3% (1/3)	0.0% (0)	45.7% (7/15)	43.8% (7/16)	10.0% (2/20)	40.9% (9/22)	25.0% (1/4)	33.3% (1/3)
1	66.7% (2/3)	33.3% (1/3)	66.7% (4/6)	100.0% (3/3)	53.3% (8/15)	56.3% (9/16)	90.0% (18/20)	59.1% (13/22)	75.0% (3/4)	66.7% (2/3)
Prior IO therapy,% (n)										
N	33.3% (1/3)	33.3% (1/3)	50.0% (3/6)	33.3% (1/3)	20.0% (3/15)	50.0% (8/16)	0.0% (0/20)	0.0% (0/22)	100.0% (4/4)	33.3% (1/3)
Y	66.7% (2/3)	66.7% (2/3)	50.0% (3/6)	66.7% (2/3)	80.0% (12/15)	50.0% (8/16)	100.0% (20/20)	100.0% (22/22)	0.0% (0)	66.7% (2/3)
Prior Lines of Systemic Therapy,% (n)										
1	0.0% (0)	33.3% (1/3)	16.7% (1/6)	0.0% (0)	6.7% (1/15)	31.3% (5/16)	10.0% (2/20)	18.2% (4/22)	0.0% (0)	66.7% (2/3)
2	0.0% (0)	0.0% (0)	0.0% (0)	0.0% (0)	26.7% (4/15)	25.0% (4/16)	20.0% (4/20)	18.2% (4/22)	0.0% (0)	0.0% (0)
≥3	100.0% (3/3)	66.7% (2/3)	83.3% (5/6)	100.0% (3/3)	66.7% (10/15)	43.8% (7/16)	70.0% (14/20)	63.6% (14/22)	100.0% (4/4)	33.3% (1/3)

Results

Exposure-Safety Analysis Figure 3

- The exposure-safety analysis included 92 subjects from the dose-escalation and dose-expansion parts with available PK and clinical outcome data. Median C_{avg} was substantially lower with Q6W than Q2W at both 3 and 5 mg/kg, with observed Grade ≥3 safety outcomes generally consistent with model predictions.
- C_{avg} was significantly associated with Grade ≥3 TRAEs and Grade ≥3 treatment-related LFT elevations (p=0.006 and p=0.017, respectively), supporting reduced exposure with extended-interval dosing as a strategy to improve tolerability for combination cohorts.

Figure 3 | Exposure–Safety Relationship Analysis As of 23-Aug-2026



Three subjects from the initial flat-dose cohorts (0.7, 2, and 7 mg; n=1 each) were excluded from the exposure-safety analysis.

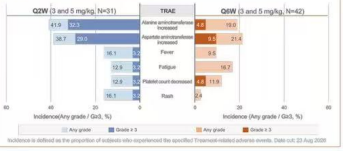
Safety Table 2, Figure 4

- For dose optimization, safety was evaluated at the 3 and 5mg/kg dose levels selected for further expansion, incorporating subjects from both the dose-escalation and dose-expansion parts to characterize schedule-dependent differences.
- Across the 3 and 5 mg/kg dose levels, Q6W dosing was associated with lower rates of Grade ≥3 treatment-related AEs (26.2% vs 51.6%), Grade ≥3 treatment-related LFT elevations (11.9% vs 35.5%), and treatment-related discontinuations (7.1% vs 12.9%) compared with Q2W dosing.
- TRAEs occurring in ≥10% of subjects treated with 3 or 5 mg/kg in either dosing schedule included LFT elevations, fever, fatigue, platelet count decreased, and rash.
- Grade ≥3 TRAEs, excluding transaminase elevations, were infrequently observed.

Table 2 | Overall Safety Profiles As of 23-Aug-2026

Category	3 mg/kg Q2W (N=15)	5 mg/kg Q2W (N=16)	3 and 5 mg/kg Q2W (N=31)	3 mg/kg Q6W (N= 20)	5 mg/kg Q6W (N= 22)	3 and 5 mg/kg Q6W (N=42)
Any AE	100.0% (15/15)	100.0% (16/16)	100.0% (31/31)	95.0% (19/20)	95.5% (21/22)	95.2% (40/42)
Any treatment-related AE	80.0% (12/15)	75.0% (12/16)	77.4% (24/31)	50.0% (10/20)	72.7% (16/22)	61.9% (26/42)
Grade ≥3 AE	66.7% (10/15)	87.5% (14/16)	77.4% (24/31)	55.0% (11/20)	59.1% (13/22)	57.1% (24/42)
Grade ≥3 treatment-related AE	46.7% (7/15)	56.3% (9/16)	51.6% (16/31)	15.0% (3/20)	36.4% (8/22)	26.2% (11/42)
Grade ≥3 treatment-related LFT elevation	33.3% (5/15)	37.5% (6/16)	35.5% (11/31)	5.0% (1/20)	18.2% (4/22)	11.9% (5/42)
Any treatment-related SAE	33.3% (5/15)	25.0% (4/16)	29.0% (9/31)	5.0% (1/20)	9.1% (2/22)	7.1% (3/42)
AE leading to treatment discontinuation	26.7% (4/15)	18.8% (3/16)	22.6% (7/31)	5.0% (1/20)	13.6% (3/22)	9.5% (4/42)
Treatment-related AE leading to treatment discontinuation	13.3% (2/15)	12.5% (2/16)	12.9% (4/31)	5.0% (1/20)	9.1% (2/22)	7.1% (3/42)
Any CRS	0.0% (0/15)	0.0% (0/16)	0.0% (0/31)	0.0% (0/20)	0.0% (0/22)	0.0% (0/42)

Figure 4 | TRAE With Incidence ≥10% in Either Dosing Schedule (Q2W or Q6W) at 3 and 5 mg/kg



Incidence is defined as the proportion of subjects who experienced the specified Treatment-related adverse events. Data cut: 23 Aug 2026

Efficacy Table 3, Figure 5

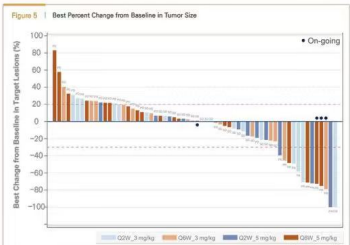
- Ragistomig demonstrated antitumor activity across both Q2W and Q6W dosing schedules. Clinically meaningful activity was maintained with 5 mg/kg Q6W monotherapy, with an ORR of 21.1% compared with 25.0% at the corresponding Q2W dose level.
- A total of 13 objective responders were observed across multiple tumor types and dosing schedules, including ovarian cancer, HNSCC, gastric cancer, cutaneous melanoma, HCC, and esophageal cancer in the Q2W groups, and HNSCC, acral / mucosal melanoma, HCC, and SCLC in the Q6W groups.
- In addition, tumor reductions exceeding 20% were observed in 4 subjects across Q2W and Q6W schedules. 2 subjects receiving 3 mg/kg Q6W (one with anti-PD-(L)1–relapsed HCC and one with gastric neuroendocrine carcinoma) and 2 subjects receiving Q2W (one at 3 mg/kg with anti-PD-(L)1–refractory esophageal cancer and one at 5 mg/kg with NSCLC).
- Most responders (84.6%, 11/13) had relapsed or refractory disease following prior anti-PD-(L)1 therapy, suggesting that ragistomig may reinvigorate exhausted T cells through 4-1BB–mediated costimulation and restore antitumor activity in tumors relapsed or refractory to prior checkpoint inhibition.[1]

Table 3 | Efficacy Profiles As of 23-Aug-2026

Ragistomig monotherapy efficacy profile	3 mg/kg Q2W (N=14)	5 mg/kg Q2W (N=12)	3 and 5 mg/kg Q2W (N=26)	3 mg/kg Q6W (N=17)	5 mg/kg Q6W (N=19)	3 and 5 mg/kg Q6W (N=36)
Objective Response Rate*, % (n)	28.6% (4/14)	25.0% (3/12)	26.9% (7/26)	11.8% (2/17)	21.1% (4/19)	16.7% (6/36)
Disease Control Rate, % (n)	64.3% (9/14)	75.0% (9/12)	69.2% (18/26)	58.8% (10/17)	52.6% (10/19)	55.6% (20/36)
Complete response	7.1% (1/14)	0.0% (0/12)	3.8% (1/26)	0.0% (0/17)	0.0% (0/19)	0.0% (0/36)
Partial response*	21.4% (3/14)	25.0% (3/12)	23.1% (6/26)	11.8% (2/17)	21.1% (4/19)	16.7% (6/36)
Stable disease	35.7% (5/14)	50.0% (6/12)	42.3% (11/26)	47.1% (8/17)	31.6% (6/19)	38.9% (14/36)
Progressive disease	35.7% (5/14)	25.0% (3/12)	30.8% (8/26)	41.2% (7/17)	47.4% (9/19)	44.4% (16/36)

*All objective responses were confirmed except for one unconfirmed partial response (uPR) each in the 5 mg/kg Q6W and 3 mg/kg Q2W cohorts.

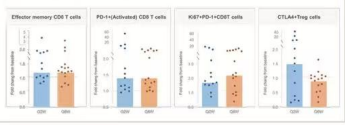
Figure 5 | Best Percent Change from Baseline in Tumor Size



Pharmacodynamic Biomarker (Peripheral Blood) Figure 6

- Q6W dosing preserved the activation and expansion of key CD8 T-cell subsets, including proliferating and effector memory CD8 T cells, at levels comparable to Q2W. Moreover, CTLA4^{+} regulatory T cells trended lower with Q6W than with Q2W, suggesting a more favorable effector-to-regulatory balance.
- These findings support pharmacodynamic activity with the extended dosing interval despite lower drug exposure, with evidence of immune activation consistent with the proposed mechanism of action.

Figure 6 | Changes in Peripheral Immune Cell Subsets at Week 8: Q2W vs Q6W (3 and 5 mg/kg)



Conclusion

- Ragistomig demonstrated clinically meaningful antitumor activity in heavily pretreated patients, including those whose disease had relapsed after or was refractory to prior anti-PD-(L)1 therapy.
- Ragistomig dose and schedule optimization was evaluated using integrated clinical safety, efficacy, PK/PD, and exposure-safety analyses in patients with advanced solid tumors.
- The totality of evidence supports 3-5 mg/kg Q6W, or regimens achieving comparable average exposure (C_{avg}), for further clinical development in combination with other agents.
- A study evaluating ragistomig in combination with pembrolizumab in anti-PD-(L)1 relapsed/refractory head and neck squamous cell carcinoma is ongoing.

Reference

1. ASCO2024. Phase 1 Trial Safety and Efficacy of Ragistomig, a Bispecific Antibody Targeting PD-L1 and 4-1BB in Advanced Solid Tumors, Abstract #3523.
2. ESMO IO2025. Phase 1 Clinical Trial of Ragistomig (ABL503/TJ-L14B: PD-L1 x 4-1BB Bispecific Antibody) Q6W Dosing Balances Favorable Safety and Sustained Efficacy Through Extended Immunologic Memory and Reinvigoration of CD8+ T Cells, Abstract #988.

This study was sponsored by ABL Bio, Inc.
The presenter declares that there are no conflicts of interest related to this presentation.