
**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): September 8, 2026

VERU INC.

(Exact name of registrant as specified in its charter)

Wisconsin
(State or other jurisdiction
of incorporation)

1-13602
(Commission
File Number)

39-1144397
(IRS Employer
Identification No.)

2916 N. Miami Avenue, Suite 1000, Miami, Florida 33127
Address of principal executive offices) (Zip Code)

Registrant's telephone number, including area code: (305) 509-6897

Not Applicable
(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 (see General Instruction A.2. below):

- ☐ 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, \$0.01 par value per share	VERU	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 8.01 Other Events.

On September 8, 2026, Veru Inc. issued a press release (“Press Release”) announcing that based on new, positive preclinical data for its oral, novel, microtubule targeting agent, sabizabulin, demonstrating augmented anticancer activity in *KRAS*-metastatic pancreatic cancer cell lines that were resistant to daraxonrasib (RASONQUE™* Revolution Medicines), and previous preclinical and clinical sabizabulin studies, the Company has made the strategic decision to expand its current drug development pipeline to advance sabizabulin as a potential novel treatment for patients whose metastatic pancreatic cancer has become resistant to daraxonrasib. Consequently, the Company will no longer be exploring sabizabulin for the treatment of chronic inflammation related to atherosclerotic cardiovascular disease. A copy of the Press Release is attached as Exhibit 99.1 to this report and incorporated by reference herein.

* RASONQUE™ is a trademark of Revolution Medicines, Inc.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

<u>Exhibit No.</u>	<u>Document</u>
99.1	Press Release of Veru Inc., issued September 8, 2026.
104	Cover Page Interactive Data File (embedded within the 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.

Date: September 8, 2026

VERU INC.

By: /s/ Michele Greco

Michele Greco
Chief Financial Officer and
Chief Administrative Officer



Investor and Media Contact:

Samuel Fisch

Executive Director, Investor Relations and Corporate Communications

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Veru Advances Oral Sabizabulin Following Positive Preclinical Data Showing Potent Anticancer Activity in Human Daraxonrasib (Revolution Medicines' RASONQUE™) Resistant Pancreatic Cancer into a Planned Phase 2 Clinical Trial

—New positive preclinical studies show that sabizabulin can overcome drug resistance to daraxonrasib with potent anticancer efficacy (IC₅₀=18.2nM) in daraxonrasib resistant human pancreatic cancer cell line which is a drug concentration that can be achieved with current sabizabulin human dosing with good safety—

—Oral sabizabulin had a good safety profile and promising efficacy in previous first-in-man Phase 1b/2 clinical study of 80 subjects with castration resistant and taxane resistant metastatic prostate cancer—

—Based on these new and prior supportive preclinical and clinical studies, the Company has made the strategic decision to advance oral sabizabulin as an innovative treatment for patients that develop metastatic pancreatic cancer progression that has become resistant to daraxonrasib—

—Veru has IP protection for sabizabulin until 2043 and controls global development and commercialization rights—

—Company's enobosarm for high quality weight loss in older patients with obesity receiving GLP-1 RA fully on track – Phase 2b PLATEAU clinical trial interim analysis near term milestone calendar Q1 2027, clinical supply agreement with Novo Nordisk, strong IP protection with newly issued patent for enobosarm with GLP-1 RA until late 2044—

MIAMI, FL – September 08, 2026 – Veru Inc. (NASDAQ: VERU) today announces new preclinical data showing that sabizabulin can overcome drug resistance to daraxonrasib (RASONQUE™ Revolution Medicines) with potent anticancer efficacy (IC₅₀=18.2nM) in daraxonrasib resistant human pancreatic cancer cell line which is a drug concentration that can be achieved with current sabizabulin human dosing with good safety.

Based on the new, positive preclinical data, and the previous preclinical and clinical sabizabulin studies, the Company will advance sabizabulin into a planned Phase 2 clinical trial for metastatic *KRAS*-driven pancreatic cancer progression that has become resistant to the recent FDA approved daraxonrasib.

New preclinical data results: Sabizabulin demonstrated potent anticancer activity against *KRAS*-driven metastatic cancer cell lines that were resistant to daraxonrasib (August 2026)

Preclinical studies were performed using a 2D proliferation assay to evaluate the efficacy of sabizabulin in parental and daraxonrasib resistant *KRAS* driven pancreatic cancer cell line AsPC-1 (G12D *KRAS* mutation) and colon cancer HCT-116 cell line (G13D *KRAS* mutation). It was confirmed that the reason for daraxonrasib resistance was the reactivation of the *KRAS* signaling pathway in both pancreatic and colon daraxonrasib resistant cell lines. The resistance index (RI) was calculated, which is the ratio of drug concentration required to inhibit 50% cell growth (IC50) in resistant cell line compared to its parental (sensitive) cell line. The resistance index represents the fold-change in drug tolerance of the resistant cell line compared to its sensitive, parental cell line. An RI greater than 1 indicates resistance, while an RI below 1 indicates increased sensitivity to the drug.

Like daraxonrasib, sabizabulin had potent anticancer activity for both pancreatic cancer and colon cancer parental cell lines regardless of the type of *KRAS* mutation. As expected, the daraxonrasib resistant cell lines were resistant to daraxonrasib with a RI of 68 for pancreatic cancer cell line and RI of >128 for colon cancer cell line. In contrast, daraxonrasib resistant pancreatic cancer cell lines became more sensitive (collateral sensitivity) to sabizabulin with a RI of 0.25 and daraxonrasib resistant colon cell line retained sensitivity to sabizabulin with a RI of 1. In summary, sabizabulin retained highly potent anticancer efficacy in daraxonrasib resistant pancreatic and colon cancer cell lines that had reactivation of *KRAS* signaling pathway as the mechanism for drug resistance to daraxonrasib. These preclinical data may also support sabizabulin as a treatment of daraxonrasib resistant cancer types beyond pancreatic cancer.

“The recent FDA approval of Revolution Medicines’ daraxonrasib was a major breakthrough in the treatment of *KRAS*-driven metastatic pancreatic cancer,” said Mitchell Steiner, M.D., Chairman, President, and Chief Executive Officer of Veru Inc. “Unfortunately, resistance to daraxonrasib occurs as the median time to cancer progression was 7.2 months in patients receiving daraxonrasib. The primary mechanism that leads to daraxonrasib reactivation is reactivation of the *KRAS* signaling pathway. As sabizabulin targets downstream components of the *KRAS* signaling pathway, we explored and confirmed in recently completed preclinical studies that sabizabulin has the potential to treat metastatic pancreatic cancer that has become resistant to daraxonrasib. This exciting new development and the importance of this large unmet medical need compel us to pursue this novel sabizabulin oncology indication. With the goal of maximizing Veru shareholder value, we have made the strategic decision to advance sabizabulin into a Phase 2b clinical trial. Consequently, we will no longer be exploring sabizabulin for the treatment of chronic inflammation related to atherosclerotic cardiovascular disease. The Company controls global development and commercialization rights to sabizabulin with issued patent protection until 2043.”

Dr. Steiner added: “Our enobosarm obesity program is completely on track. The Phase 2b PLATEAU clinical trial is fully enrolled, and we expect the interim analysis, a near term milestone, in calendar Q1 2027. We entered into a clinical supply agreement with Novo Nordisk², and we now have an issued US patent for enobosarm with semaglutide with expiry in late 2044.”

“Sabizabulin represents a compelling candidate for Phase 2 evaluation following daraxonrasib treatment, based on its profile as an oral, targeted agent with a novel microtubule binding mechanism and its ability to downregulate *TUBB3* and other downstream effectors of the *KRAS* signaling pathway. The rationale is further strengthened by reassuring safety findings from a previous Phase 1b/2 study in 80 patients, as well as preclinical data evidence that daraxonrasib resistance may potentiate sensitivity to sabizabulin. Together, these findings support clinical investigation of sabizabulin as a rational post-daraxonrasib strategy,” said Daniel King, M.D., Ph.D., Medical Oncologist and Director of Research and Development for Genomic Medicine at Northwell Health.

Sabizabulin is a clinical stage oncology drug candidate that has a favorable safety profile with promising preliminary anticancer activity

Sabizabulin's first-in-man study was a Phase 1/2b clinical trial evaluating safety and efficacy of sabizabulin in advanced metastatic prostate cancer (Markowski et al. Clin Cancer Res 28:2789-2795, 2022). We believe positive efficacy and safety clinical data from this Phase 1b/2 first-in-man clinical study of sabizabulin monotherapy conducted in 80 patients with heavily pretreated metastatic castration resistant and taxane resistant prostate cancer support the translational potential of sabizabulin treatment for daraxonrasib resistant metastatic pancreatic cancer.

The Phase 1b portion utilized a 3+3 dose escalation design with escalating daily oral doses of 4.5 mg—81 mg (7 days on drug/14 days off per 21-day cycle, which was then expanded to daily dosing). The Phase 1b portion included 39 metastatic castration resistant cancer patients that were treated with one or more novel androgen receptor targeting agents. Most patients had bone-only disease (55%) with an additional 21% having both lymph node and bone involvement with 23% of patients with prior taxane-based chemotherapy. The Phase 2 portion tested a daily dose of 63 mg in 41 heavily pretreated similar patient population, but with no prior chemotherapy. Efficacy was assessed using PCWG3 and RECIST 1.1 criteria.

The maximum tolerated dose was not defined in the Phase 1b as all doses tested were well tolerated. The recommended Phase 2 dose was set at 63 mg/day. The most common adverse events (>10% frequency) at the 63 mg oral daily dosing (combined Phase 1b/2 safety data) were predominantly Grade 1-2 events. Grade ≥ 3 events included diarrhea (7.4%), fatigue (5.6%) and ALT/AST elevations (5.6% and 3.7%, respectively). Neurotoxicity and neutropenia were not observed at these dosage levels.

Efficacy data in patients treated with ≥ 1 continuous cycle (21 days) of 63 mg or higher had a Kaplan-Meier median radiographic progression-free survival that was estimated to be 11.4 months with durable responses lasting greater than 12 months, occurring in 14.5% (n=55) patients. The objective response rate was 20.7% in patients with measurable disease and durable responses lasting greater than 2.75 years were observed. Compared to historical controls, the radiographic progression-free survival in similar patients was only 3.6 months and objective response rate was 2% with an alternative androgen receptor blocking agent (deBono J NEJM 382:2091, 2020). This Phase 1b/2 clinical trial had a favorable safety profile with promising preliminary antitumor activity and demonstrated that chronic oral daily dosing of sabizabulin was feasible up to 3 years.

Next steps

Preclinical studies including in daraxonrasib resistant pancreatic and colon cell lines and promising clinical data from Phase 1b/2 clinical trial conducted in metastatic castration resistant and taxane resistant prostate cancer support the translational potential of sabizabulin against daraxonrasib resistant metastatic pancreatic cancer. Accordingly, the Company plans to pursue a Phase 2b clinical study to evaluate the efficacy and safety of sabizabulin in patients who have metastatic pancreatic cancer progression while receiving treatment with daraxonrasib (RASONQUE). We will first seek regulatory clarity from the FDA through a preIND meeting in calendar Q4 2026 to better understand the scope of the clinical trial. We believe these recent developments will create greater shareholder value.

About Veru Inc.

Veru is a late clinical stage biopharmaceutical company focused on developing innovative medicines for the treatment of cardiometabolic and oncology. The Company's drug development program includes two late-stage novel small molecules, enobosarm and sabizabulin. Enobosarm, an oral selective androgen receptor modulator (SARM), is being developed as a next generation drug that makes weight reduction by GLP-1 RA drugs more tissue selective for loss of fat and preservation of lean mass to improve body composition and physical function which is expected to result in clinically meaningful incremental weight reduction versus GLP-1 RA therapy alone. Based on previous and new preclinical and clinical studies, sabizabulin, an oral novel microtubule targeting agent that targets downstream components of the KRAS signaling pathway, has demonstrated the potential to become the next treatment for *KRAS*-driven metastatic pancreatic cancer resistant to the recent FDA approved daraxonrasib multi-selective *KRAS* inhibitor therapy. Accordingly, the Company plans to pursue a Phase 2b clinical study to evaluate the efficacy and safety of sabizabulin in patients for the treatment of metastatic pancreatic cancer after daraxonrasib failure. We will first seek regulatory clarity from the FDA through a preIND meeting in calendar Q4 2026.

Obesity Program

Evaluating enobosarm in combination with GLP-1 RA for higher quality weight reduction in older patients with obesity

Fully Enrolled Phase 2b PLATEAU Clinical Study

The Phase 2b PLATEAU clinical trial is a double-blind, placebo-controlled study to evaluate the effect of enobosarm 3mg on total body weight, fat mass, lean mass, physical function, bone mineral density and safety in older patients (age ≥ 65 years) who have obesity (BMI ≥ 35) and are initiating semaglutide treatment for weight reduction. During the past quarter, the Company exceeded its Phase 2b PLATEAU clinical trial targeted full enrollment of 200 patients by enrolling 239 patients. The Phase 2b PLATEAU study is designed to assess the ability of enobosarm treatment to break through the weight loss plateau observed in patients with obesity receiving GLP-1 RA treatment by preserving muscle mass and physical function to achieve clinically meaningful incremental weight reduction by 68 weeks. The primary efficacy endpoint of the study is the percent change from baseline in total body weight at 68 weeks. The key secondary endpoints are total fat mass, total lean mass, physical function (stair climb test), mobility disability assessment, bone mineral density, and patient reported outcome questionnaires for physical function, HbA1c, and insulin resistance. Results of an interim analysis assessing lean body mass and fat mass as measured by DXA after patients have completed 32 weeks is expected in the first quarter of calendar year 2027. Final topline clinical data is expected in the fourth quarter of calendar year 2027. The Principal Investigator for the Phase 2b PLATEAU clinical trial is Steven Heymsfield, MD, a Professor and the Director of the Body Composition-Metabolism Laboratory at the Pennington Biomedical Research Center in Baton Rouge, Louisiana. Dr. Heymsfield was also the Principal Investigator of Veru's Phase 2 QUALITY clinical study.

Completed Positive Phase 2b QUALITY Clinical Study

The Phase 2b QUALITY clinical study was a positive multicenter, double-blind, placebo-controlled, randomized, dose-finding clinical trial that evaluated the safety and efficacy of enobosarm 3 mg, enobosarm 6 mg, or placebo as a treatment to augment fat loss and to prevent muscle loss in 168 older patients (≥ 60 years of age) receiving semaglutide (Wegovy®) for weight reduction. After the efficacy

dose-finding portion of the Phase 2b QUALITY clinical trial was completed at 16 weeks, participants continued into a Phase 2b QUALITY maintenance extension portion of study where all patients discontinued semaglutide treatment, but continued receiving placebo, enobosarm 3 mg, or enobosarm 6 mg as monotherapy in a double-blind fashion for 12 weeks. The Phase 2b QUALITY and maintenance extension clinical trial was a positive study that demonstrated that enobosarm plus semaglutide preserved lean mass and physical function and led to greater fat loss during the 16 week active weight loss period and enobosarm monotherapy prevented the regain of weight lost and continued to lose fat mass when the GLP-1 RA was discontinued.

About pancreatic cancer and *KRAS*

Pancreatic cancer is a highly lethal disease that has one of the lowest survival rates of all major cancers. The incidence of pancreatic cancer is increasing steadily, with an estimated 67,530 new cases and 52,740 deaths in United States for 2026. Pancreatic cancer is predicted to become the second leading cause of cancer-related deaths in the United States by 2030. The survival rate of patients diagnosed with pancreatic cancer is dismal, with median overall survival of 4 months with only ~12.5 % of patients expected to survive 5 years following diagnosis. A significant reason for such poor survival outcomes is the lack of early diagnosis, leading to only 15—20% of patients being eligible for surgery, which is the only curative intent treatment option.

Up to 85 % of patients present with a locally advanced or metastatic disease. One third of patients typically present with locally advanced pancreatic cancer, with vascular involvement preventing a surgical resection option. Treatment aims to control the disease and typically involves systemic chemotherapies of either a combination of gemcitabine and nab-paclitaxel and or FOLFIRINOX (a 4-drug chemotherapy consisting of 5-fluorouracil, oxaliplatin, irinotecan, and leucovorin). Half of pancreatic cancer patients will present with distant metastases, and palliative treatment is provided to reduce cancer-related symptoms and prolong survival.

When compared with other carcinomas, pancreatic cancer is genomically a relatively homogeneous disease. The major genetic event and hallmark in the development of pancreatic ductal adenocarcinoma is the somatic activating *KRAS* oncogene mutation which is observed in more than 90% of pancreatic cancer tumors. The *KRAS* mutation remains active during the progression from epithelial cells to invasive cancer, contributing to the processes of proliferation, survival, migration, and invasion. By its effect on the tumor stroma and microenvironment, the *KRAS* protein plays an important role in the process of metastatic spread and chemotherapy resistance.

About sabizabulin (VERU-111)

Sabizabulin is an orally bioavailable microtubule targeting agent that targets a novel binding sites on microtubules that causes crosslinking of α -tubulin with the “colchicine binding site” of β -tubulin to inhibit microtubule polymerization (assembly). Based on over 12 peer reviewed publications in preclinical cell lines and xenograft cancer models across a broad range of tumor types*, sabizabulin treatment disrupted and fragmented microtubules, inhibited cancer cell proliferation and tumor growth, prevented cancer cell invasion and metastases, and suppressed angiogenesis. Sabizabulin was able to restore chemosensitivity (pancreatic cancer) and overcome paclitaxel resistance in a variety of tumor types including taxane resistant lung, prostate, ovarian, and cervical cancers. Sabizabulin arrests the cell cycle in the G2/M halting mitosis in rapidly dividing cells resulting in cell death. Independent of its activities to disrupt the microtubules, sabizabulin preferentially decreases the transcription of β III- and β IV-tubulin isoforms to restore chemotherapy sensitivity as well as induces apoptosis in nondividing cells by activation of Caspase 3 and 9 and cleaving PARP and modulation of both cell cycle regulatory proteins (Cdc2, Cdc25c, and Cyclin B1) and intrinsic apoptosis-associated proteins (Bax, Bad, Bcl-2,

and Bcl-xl) to induce apoptosis. Down regulation of Bcl-2 is particularly interesting as Bcl-2 overexpression protects cancer cell from cell death. Moreover, sabizabulin may overcome other common drug resistance mechanisms since it is not substrate for proteins involved in multidrug resistance including P-glycoprotein, MRP and BCRP so that sabizabulin cannot be effluxed or pumped out of cancer cells.

** Triple negative breast cancer (taxane resistant), cervical cancer, lung cancer (taxane resistant), ovarian cancer (taxane resistant), prostate cancer (taxane resistant), melanoma (BRAF resistant), pancreatic cancer, colon cancer, glioma, and human promyelocytic leukemia (vincristine resistant).*

About TUBB3 gene (βIII-tubulin protein)

Altered expression of β-tubulin isotypes has been observed in a range of cancers. Among various tubulins, βIII- and βIV-tubulin isoforms have been primarily implicated in pancreatic cancer progression, metastasis and chemoresistance. However, there are no specific inhibitors of these isoforms that have potent anticancer activity with low toxicity.

In carcinogenesis, βIII-tubulin (TUBB3) gene is aberrantly expressed in a range of epithelial tumors and is associated with drug resistance and aggressive disease. In pancreatic cancer, it should be noted that βIII-tubulin protein is NOT expressed in normal ductal epithelium of the pancreas. In contrast, βIII-tubulin overexpression is frequently reported in tumor specimens, particularly in advanced or metastatic specimens. βIII-tubulin aberrant expression has been commonly observed in up to 90% of pancreatic adenocarcinoma cancers. Diverse cancer cell lines have shown that overexpression of βIII-tubulin confers resistance to paclitaxel, docetaxel, and vinca alkaloids as well as DNA damaging agents, such as cisplatin, topoisomerases inhibitors, and doxorubicin. Aberrant βIII-tubulin expression was also associated with poorly differentiated tumors, shorter disease progression, chemoresistance, unfavorable prognosis and worse overall survival — not only in pancreatic cancer, but also in many other types of cancers including lung, ovarian, breast, prostate, urothelial and bladder, gastric, rectal cancers, esophageal squamous cell carcinoma, and thymic carcinoma.

Forward-Looking Statements

This press release contains “forward-looking statements” as that term is defined in the Private Securities Litigation Reform Act of 1995, including, without limitation, express or implied statements related to the safety and efficacy of sabizabulin as a treatment for metastatic pancreatic cancer and other cancers, including cancers that have become resistant to daraxonrasib; the planned design for a future Phase 2 clinical trial of sabizabulin as a second line treatment of KRAS-driven metastatic pancreatic cancer; the Company’s ability to obtain regulatory clarity through a preIND meeting with the FDA and the timing of such meeting; the ability of the Company to obtain sufficient additional funding to advance its sabizabulin pancreatic cancer development program beyond the planned preIND clinical trial with the FDA and to a Phase 2 clinical trial; whether preclinical studies demonstrating sabizabulin’s potent anticancer activity against KRAS-driven metastatic cancer cell lines that were resistant to daraxonrasib and its mechanism of action can be replicated to any extent in clinical human studies; whether new and prior preclinical and clinical studies support further development of an innovative treatment for metastatic pancreatic and/or other cancers; the planned design, enrollment, timing, commencement, interim, topline and full data readout timing, scope and regulatory pathways for the continued development of enobosarm in patients with obesity, including the PLATEAU Phase 2b study; the planned design, number of sites, timing, endpoints, patient population and patient size of such trial and whether the PLATEAU trial will successfully meet any of its primary or secondary endpoints; whether the results of the Phase 2b QUALITY study and the extension maintenance study of enobosarm, including weight loss, preservation of lean mass and physical function and loss of fat mass and the prevention of the regain of fat mass and total body weight loss, will be replicated to the same or any degree in the PLATEAU Phase 2b study or in any future Phase 3 studies; whether and when the PLATEAU Phase 2b study of enobosarm will produce an interim analysis and/or topline data; whether enobosarm in combination with a GLP-1 RA drug will

provide a higher quality and/or greater quantity weight loss in patients and whether enobosarm will be the next generation combination therapy with GLP-1 receptor agonists for patients with obesity that makes weight reduction more tissue selective for loss of fat, preservation of lean mass, physical function, improved body composition and maintaining or increasing bone mineral density, and demonstrating favorable HbA1c and insulin resistance results, all while maintaining a favorable safety profile; whether patients treated with enobosarm in the PLATEAU Phase 2B study will break through the weight loss plateau and achieve clinically meaningful incremental weight reduction by preserving muscle mass and physical function whether enobosarm will enhance or achieve a higher quality weight loss or the preservation of muscle in, or meet any unmet need for, obesity patients, including whether it will provide important insights into quality weight loss therapy and the design of a Phase 3 clinical development program; and whether the Company will be successful in its transformation into a late stage biopharmaceutical company focused on obesity and oncology. The words “anticipate,” “believe,” “could,” “expect,” “intend,” “may,” “opportunity,” “plan,” “predict,” “potential,” “estimate,” “should,” “will,” “would” and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Any forward-looking statements in this press release are based upon current plans and strategies of the Company and reflect the Company’s current assessment of the risks and uncertainties related to its business and are made as of the date of this press release. The Company assumes no obligation to update any forward-looking statements contained in this press release because of new information or future events, developments, or circumstances. Such forward-looking statements are subject to known and unknown risks, uncertainties and assumptions, and if any such risks or uncertainties materialize or if any of the assumptions prove incorrect, our actual results could differ materially from those expressed or implied by such statements. Factors that may cause actual results to differ materially from those contemplated by such forward-looking statements include, but are not limited to: the development of the Company’s product portfolio and the results of clinical studies, including any interim or topline analysis, possibly being unsuccessful or insufficient to meet applicable regulatory standards or warrant continued development; although the Company has sought and received feedback from the FDA on the designs of its clinical trials and intends to continue to do so, the FDA may ultimately disagree that the Company’s clinical trials support approval; risks that results of prior preclinical and clinical trials may not be predictive for future development or a drug’s efficacy or other future results; the Company’s ability to reach agreement with FDA on study design requirements for the Company’s planned clinical studies, including for the Phase 2b program for enobosarm as a weight loss or body composition drug, a Phase 2 study for sabizabulin as a treatment for metastatic pancreatic cancer, and the number of future Phase 3 studies to be required and the cost thereof; potential delays in the timing of and results from clinical trials and studies, including as a result of an inability to enroll sufficient numbers of patients in clinical studies or an inability to enroll patients in accordance with planned schedules; the ability to fund planned clinical development as well as other operations of the Company; the Company plans to prioritize the use of its current internal cash to the development of enobosarm, with a primary near-term focus on funding its PLATEAU Phase 2b clinical trial, and as a result advancement of sabizabulin as a treatment for pancreatic cancer beyond a preIND meeting with the FDA, or as a treatment for any other cancer, will depend upon the Company securing additional funding to the extent not used for its enobosarm development efforts; whether the Company will be able to partner with another company in the development of enobosarm or sabizabulin; the timing of any submission to the FDA or any other regulatory authority and any determinations made by the FDA or any other regulatory authority; the potential for disruptions at the FDA or other government agencies to negatively affect our business, including as a result of a future shutdown of the U.S. government; any products of the Company, if approved, possibly not being commercially successful; the risk that the Supply Agreement with Novo Nordisk could be terminated prior to the completion of the Company’s PLATEAU Phase 2b clinical trial, including pursuant to a provision that permits Novo Nordisk to terminate for convenience upon 60 days’ prior notice; the ability of the Company to obtain sufficient financing, including any partnership or collaboration agreements, on acceptable terms when needed to fund development and operations and to enable us to continue as a going concern; the effect of the SEC’s “baby shelf” rules on the Company’s ability to raise sufficient capital when needed; demand for, market acceptance of, and competition against any of the Company’s products or product candidates; new or existing competitors with greater resources and capabilities and new competitive product approvals and/or introductions; changes in regulatory

practices or policies or government-driven healthcare reform efforts, including pricing pressures and insurance coverage and reimbursement changes; the Company's ability to obtain, protect and enforce its data, intellectual property and other proprietary rights; costs and other effects of litigation, including regulatory challenges, product liability claims, intellectual property claims and challenges, securities litigation and litigation with the purchaser of the Company's FC2 business; the Company's ability to identify, successfully negotiate and complete suitable acquisitions or other strategic initiatives; the Company's ability to successfully integrate acquired businesses, technologies or products; and other risks detailed from time to time in the Company's press releases, shareholder communications and Securities and Exchange Commission filings, including the Company's Form 10-K for the year ended September 30, 2025, and subsequent quarterly reports on Form 10-Q. These documents are available on the "SEC Filings" section of our website at www.verupharma.com/investors.

¹RASONQUE™ is a trademark of Revolution Medicines, Inc.

²Please see the Company's SEC Form 8-K filed with the SEC June 4, 2026 for further details.

³Wegovy® is a registered trademark of Novo Nordisk A/S.