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):
January 14, 2025

Ventyx Biosciences, Inc.
(Exact name of registrant as specified in its charter)

Delaware	**001-40928**	**83-2996852**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

12790 El Camino Real, Suite 200
San Diego, CA 92130
(Address of principal executive offices, including zip code)

(760) 593-4832
(Registrant's telephone number, including area code)

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):

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 exchange on which registered
Common Stock, $0.0001 par value per share	VTYX	The Nasdaq Global Select 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 2.02 Results of Operations and Financials Condition.

On January 14, 2025, Ventyx Biosciences, Inc. (the "Company"), issued a press release (the "Press Release") announcing Company's preliminary unaudited cash, cash equivalents and marketable securities balance as of December 31, 2024. Additionally, the Press Release highlighted the Company's 2025 pipeline strategy and provided clinical updates on its NLRP3 portfolio. The Press Release is attached hereto as Exhibit 99.1, and is incorporated herein by reference.

The information in this Item 2.02, including Exhibit 99.1 attached hereto, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, except as expressly set forth by specific reference in such filing.

Item 7.01 Regulation FD Disclosure.

Beginning on January 14, 2025, the Company will be attending meetings with investors, analysts and others in connection with the 43rd Annual J.P. Morgan Healthcare Conference. During these meetings, the Company will present the slides attached as Exhibit 99.2 to this Current Report on Form 8-K, which are incorporated by reference herein.

The information in this Item 7.01, including Exhibit 99.2 attached hereto, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, except as expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Press Release, dated January 14, 2025.
99.2	Corporate Presentation, dated January 14, 2025.
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.

VENTYX BIOSCIENCES, INC.

By: /s/ Raju Mohan
Raju Mohan, Ph.D.
Chief Executive Officer

Date: January 14, 2025

Ventyx Biosciences Highlights 2025 Pipeline Strategy and Provides Clinical Updates on its NLRP3 Inhibitor Portfolio

First subjects dosed in a Phase 2 trial of VTX3232 in participants with obesity and cardiometabolic risk factors, with topline data expected in H2 2025

Phase 2 trial of VTX2735 in participants with recurrent pericarditis expected to initiate in January, with topline data expected in H2 2025

Topline data from ongoing Phase 2 biomarker trial of VTX3232 in participants with early Parkinson's disease expected in H1 2025

Cash, cash equivalents and marketable securities balance of $252.9M as of December 31, 2024 (unaudited) expected to fund operations into at least H2 2026

SAN DIEGO, CA, January 14, 2025 (GLOBE NEWSWIRE) – Ventyx Biosciences, Inc. (Nasdaq: VTYX) ("Ventyx", "Company"), a clinical-stage biopharmaceutical company focused on developing innovative oral therapies for patients with autoimmune, inflammatory, and neurodegenerative diseases, today highlighted its 2025 pipeline strategy and provided clinical updates on its NLRP3 inhibitor portfolio, including VTX2735 and VTX3232.

"We believe that 2025 will be a transformative year for Ventyx with important clinical data readouts from our NLRP3 portfolio, including VTX2735, our peripherally restricted NLRP3 inhibitor and VTX3232, our CNS-penetrant NLRP3 inhibitor," said Raju Mohan, PhD, President and Chief Executive Officer. "With three trials expected to be underway by the end of January, we plan to report topline results from the Phase 2 biomarker trial of VTX3232 in patients with early Parkinson's disease in the first half of 2025, followed by results from the Phase 2 trial of VTX2735 in patients with recurrent pericarditis and the Phase 2 trial of VTX3232 in participants with obesity and cardiometabolic risk factors during the second half of 2025. With these readouts, we aim to establish Ventyx as a leader in the field of the NLRP3 inflammasome, with the potential to explore opportunities in multiple systemic and neurological diseases, including those in which IL-1 antagonism has already been validated as a therapeutic approach."

Pipeline Updates and Anticipated Milestones

NLRP3 Inhibitor Portfolio: Ventyx is advancing a portfolio of potential best-in-class oral NLRP3 inhibitors for systemic inflammatory conditions and neurodegenerative diseases, including VTX2735, a peripherally restricted NLRP3 inhibitor, and VTX3232, a CNS-penetrant NLRP3 inhibitor.

- **VTX2735 in Recurrent Pericarditis:** A single dose, open-label Phase 2 trial of VTX2735 in participants with recurrent pericarditis is expected to initiate in January. The trial will enroll approximately 30 participants for a 6-week primary treatment period, followed by a 7-week extension period. Key endpoints include safety, change in the NRS pain score, and change in high sensitivity C-reactive protein (hsCRP). Topline results are expected in the second half of 2025.

  Recurrent pericarditis is considered to be an autoinflammatory condition caused by over-activity of the innate-immune system. In particular, the disease pathophysiology is associated with aberrant activation of the NLRP3 inflammasome and IL-1, the initial cytokine of the innate immune system. Recently, concentrations of NLRP3 have been shown to be elevated in pericardial samples from patients with recurrent pericarditis compared to healthy controls. Patients refractory to non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine are commonly treated with injectable IL-1 therapies, though substantial unmet medical need remains. We believe that, by treating and preventing disease recurrence, VTX2735 has the potential to offer a safe, effective, and convenient oral therapy for patients suffering from recurrent pericarditis.

- **VTX3232 in Cardiometabolic Diseases:** Dosing has initiated in a randomized, placebo-controlled Phase 2 trial of VTX3232 in participants with obesity and cardiometabolic risk factors. The trial is expected to enroll approximately 160 subjects randomized to one of four groups for a 12-week primary treatment period: monotherapy placebo, monotherapy VTX3232, combination semaglutide + placebo, or combination semaglutide + VTX3232. Key endpoints include safety and change in hsCRP. The trial also includes a panel of exploratory endpoints, including biomarkers of inflammation and cardiometabolic disease, as well as imaging to assess body composition and liver fat. Topline results are expected in the second half of 2025.

  Activation of the NLRP3 inflammasome, and resulting chronic inflammation, has been linked to a range of cardiometabolic diseases including atherosclerosis, insulin resistance, and obesity. The Phase 2 trial of VTX3232 in participants with obesity and cardiometabolic risk factors is designed as a signal-finding trial to identify the effects of NLRP3 inhibition on a broad panel of inflammatory and metabolic biomarkers, including IL-6 and hsCRP. Data from the Phase 2 trial are expected to inform future development of the Company's NLRP3 inhibitors in cardiometabolic diseases.

- **VTX3232 in Parkinson's Disease:** Enrollment is progressing in the ongoing Phase 2 biomarker and imaging trial of VTX3232 in participants with early Parkinson's disease. This trial is expected to enroll approximately 10 participants for a 28-day open-label treatment period. Key endpoints include safety, pharmacokinetics, and biomarkers in cerebrospinal fluid (CSF) and plasma. The trial also includes exploratory TSPO PET imaging as a marker of microglial activation. Topline results are expected in the first half of 2025.

In a disease as complex as human Parkinson's disease, the regulatory networks in microglia and other neural cell types linking the pathological consequence of NLRP3-mediated neuroinflammation to the progression of Parkinson's disease are still unclear. However, overexpression of IL-1b and IL-18 has been observed in CSF samples from Parkinson's disease patients, suggesting NLRP3 inhibition in the CNS may offer a disease-modifying therapeutic approach.

The Phase 2 trial of VTX3232 in early Parkinson's disease is designed to generate data in support of this therapeutic hypothesis by demonstrating the ability to modulate key inflammatory and disease-related biomarkers in the CSF, downstream of NLRP3 activation. Beyond Parkinson's disease, NLRP3 inhibition in the CNS may have therapeutic utility in a range of neurodegenerative diseases with high unmet medical need, including Alzheimer's disease, multiple sclerosis, and amyotrophic lateral sclerosis, among others.

Inflammatory Bowel Disease (IBD) Portfolio:

- **Tamuzimod (VTX002, S1P1R Modulator, ulcerative colitis):** Phase 2 long-term extension (LTE) data presented in October 2024 at the United European Gastroenterology Week meeting continue to reinforce the potential best-in-class profile of tamuzimod in ulcerative colitis (UC). While tamuzimod achieved high rates of clinical and endoscopic remission, a therapeutic ceiling may have been reached with monotherapies. Combination treatment is an emerging therapeutic concept in IBD, and its efficacy and safety profile could position tamuzimod as the backbone of future combination regimens with another oral or biologic agent. The Company continues to explore partnership opportunities for tamuzimod in ulcerative colitis.
- **VTX958 (TYK2 Inhibitor, Crohn's disease):** As previously announced, in a Phase 2 trial, VTX958 did not meet the primary endpoint of change from baseline in the Crohn's Disease Activity Index (symptomatic outcome) due to an abnormally high placebo response. VTX958 did demonstrate robust, dose-dependent, nominally statistically significant endoscopic response at Week 12 as measured by Simple Endoscopic Score-Crohn's Disease (SES-CD; an objective endpoint) and showed a greater magnitude of decrease compared to placebo in two key biomarkers of inflammation, CRP and fecal calprotectin. Recognizing the opportunity for a safe and effective oral TYK2 inhibitor as early-line therapy in Crohn's disease, we are continuing the analysis of the Phase 2 data including data from the 52-week treat-through LTE phase. Full analysis of the Phase 2 data is expected to inform a future development strategy for VTX958 in Crohn's disease, including potential partnership opportunities.

About Ventyx Biosciences

Ventyx Biosciences is a clinical-stage biopharmaceutical company developing innovative oral therapies for patients with autoimmune, inflammatory, and neurodegenerative diseases. Our expertise in medicinal chemistry, structural biology, and immunology enables the discovery of differentiated small molecule therapeutics for conditions with

high unmet medical need, and our extensive experience in clinical development allows the rapid progression of these drugs through clinical trials. Our lead portfolio of NLRP3 inhibitors includes VTX2735, a peripherally restricted NLRP3 inhibitor in Phase 2 development for recurrent pericarditis, and VTX3232, a CNS-penetrant NLRP3 inhibitor in Phase 2 development for neurodegenerative and cardiometabolic diseases. Our inflammatory bowel disease portfolio includes tamuzimod (VTX002), an S1P1R modulator, and VTX958, a TYK2 inhibitor, both of which have completed Phase 2 clinical trials.

Forward-Looking Statements

Ventyx cautions you that statements contained in this press release regarding matters that are not historical facts are forward-looking statements. These statements are based on Ventyx's current beliefs and expectations. Such forward-looking statements include, but are not limited to, statements regarding: the expected year-end 2024 cash balance based on preliminary, unaudited information for the year ended December 31, 2024; the potential of each of Ventyx's product candidates, including the potential of VTX2735 and VTX3232, to emerge as best-in-class NLRP3 inhibitors for the treatment of systemic inflammatory conditions or neurodegenerative diseases, the potential of VTX2735 to be a safe, effective or convenient oral therapy for recurrent pericarditis and to have therapeutic potential in additional chronic peripheral inflammatory diseases, and the potential of tamuzimod as a best-in-class profile for Ulcerative Colitis (UC) or a part of a combination therapy for inflammatory bowel disease; the hypothesis that NLRP3 inhibition in the CNS may offer a disease-modifying therapeutic approach, and that the Phase 2 study of VTX3232 will support such hypothesis; the anticipated timing for commencing the Phase 2 trial of VTX2735 in recurrent pericarditis; the anticipated timing of enrollment of subjects, and the estimated total subjects enrolled, in each of the Phase 2 trials; the anticipated timing for the topline results of the ongoing Phase 2 trials of VTX3232 subjects in Parkinson's disease in H1 2025, and in the setting of obesity with cardiometabolic risk factors in H2 2025, and the Phase 2 trial of VTX2735 in recurrent pericarditis in H2 2025; management's plans with respect to the commitment of internal resources toward further analysis, or development, including future studies, partnerships or other source of non-dilutive financing, for tamuzimod in UC, VTX958 in Crohn's disease, and VTX3232 and VTX2735 in multiple cardiometabolic, systemic or neurological diseases; and the expected timeframe for funding Ventyx's operating plan with current cash, cash equivalents and marketable securities.

We are in the process of finalizing our financial statements for the year ended December 31, 2024, and the preliminary, unaudited information presented in this press release for the year ended December 31, 2024 is based on management's initial review of the information presented and its current expectations and is subject to adjustment as a result of, among other things, the completion of Ventyx's end-of-period reporting processes and related activities, including the audit by Ventyx's independent registered public accounting firm of Ventyx's financial statements. As such, any financial information contained in this press release may differ materially from the information reflected in Ventyx's financial statements as of and for the year ended December 31, 2024. You should carefully review our audited, consolidated financial statements for the year ended December 31, 2024 when they become available.

The inclusion of forward-looking statements should not be regarded as a representation by Ventyx that any of its plans will be achieved. Actual results may differ from those set forth in this press release due to the risks and uncertainties inherent in Ventyx's business, including, without limitation: potential delays in the commencement, enrollment and completion of clinical trials; Ventyx's dependence on third parties in connection with product manufacturing, research and preclinical and clinical testing; disruptions in the supply chain, including raw materials needed for manufacturing and animals used in research, delays in site activations and enrollment of clinical trials; the results of preclinical studies and clinical trials; early clinical trials not necessarily being predictive of future results; interim results not necessarily being predictive of final results; the potential of one or more outcomes to materially change as a trial continues and more patient data become available and following more comprehensive audit and verification procedures; regulatory developments in the United States and foreign countries; unexpected adverse side effects or inadequate efficacy of Ventyx's product candidates that may limit their development, regulatory approval and/or commercialization, or may result in recalls or product liability claims; Ventyx's ability to obtain and maintain intellectual property protection for its product candidates; the use of capital resources by Ventyx sooner than expected; and other risks described in Ventyx's prior press releases and Ventyx's filings with the Securities and Exchange Commission (SEC), including in Part II, Item 1A (Risk Factors) of Ventyx's Quarterly Report on Form 10-Q for the quarter ended September 30, 2024, filed on or about November 7, 2024, and Ventyx's subsequent filings with the SEC.

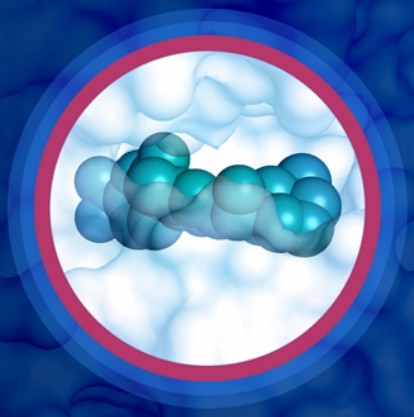
You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof, and Ventyx undertakes no obligation to update such statements to reflect events that occur or circumstances that exist after the date hereof. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995.

Investor Relations Contact:

Joyce Allaire
Managing Director
LifeSci Advisors
IR@ventyxbio.com

Exhibit 99.2

ventyx BIOSCIENCES



CORPORATE PRESENTATION

January 2025

Forward Looking Statements

Ventyx Biosciences, Inc. ("Ventyx" or the "Company") cautions you that statements contained in this presentation regarding matters that are not historical facts are forward-looking statements. These statements are based on the Company's current beliefs and expectations. Such forward-looking statements include, but are not limited to, statements regarding: the expected year-end 2024 cash balance based on preliminary, unaudited information for the year ended December 31, 2024; the potential of Ventyx's product candidates, including the potential of meaningful value creation through clinical trial results, potential of VTX3232 to demonstrate best in class profile, treat various neuroinflammatory diseases, including Parkinson's disease modification, or to achieve certain drug concentrations in the CSF, the potential of VTX2735 to demonstrate best in class profile or treat various systemic diseases, and the potential of VTX002 in UC and class-leading safety and efficacy profile; the design of clinical studies to be conducted by the Company; the total addressable market for a Parkinson's disease modifying therapy; the timing of clinical updates for all three Phase 2 studies of VTX3232 and VTX2735, including the publication of any clinical data from these studies in 2025; the regulatory pathway for VTX2735 and any expedited pathways that may be available; management's plans with respect to a potential pivotal Phase 3 trial for tamuzimod (VTX002) in UC, supported by a partner or other source of non-dilutive financing; the need for a single pivotal study for VTX002; and the expected timeframe for funding Ventyx's operating plan with current cash, cash equivalents and marketable securities.

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This presentation includes statistical and other industry and market data that we obtained from industry publications and research, surveys and studies conducted by third parties as well as our own estimates of potential market opportunities. Industry publications and third-party research, surveys and studies generally indicate that their information has been obtained from sources believed to be reliable, although they do not guarantee the accuracy or completeness of such information. Our estimates of the potential market opportunities for our products include several key assumptions based on our industry knowledge, industry publications, third-party research and other surveys, which may be based on a small sample size and may fail to accurately reflect market opportunities. While we believe that our internal assumptions are reliable, such assumptions have not been verified by any third party. The industry in which we operate is subject to a high degree of uncertainty and risk due to a variety of important factors that could cause results to differ materially from those expressed in the estimates made by third parties and by us.

Trademarks in this presentation are the property of their respective owners and used for informational and education purposes only.

Company Highlights

NLRP3 inhibition represents a paradigm shift in the treatment of autoimmune, inflammatory and neurodegenerative disorders via upstream regulation of key cytokines (e.g., IL-1, IL-18, IL-6)

VTX2735 and **VTX3232** demonstrate potential best-in-class profiles: low nanomolar potency; proven target engagement; clinical proof-of-concept in CAPS; favorable safety profile

Clinical catalysts represent three potential paths to meaningful value creation in 2025:

- VTX2735 Phase 2 recurrent pericarditis trial – data expected **H2 2025**
- VTX3232 Phase 2 cardiometabolic trial in obese participants – data expected **H2 2025**
- VTX3232 Phase 2 early Parkinson's disease study – data expected **H1 2025**

Late-stage Inflammatory Bowel Disease portfolio provides potential additional source of value:

- Tamuzimod is a Phase 3-ready, potential best-in-class S1P1R modulator for Ulcerative Colitis
- VTX958 (TYK2 inhibitor) demonstrated robust endoscopic response in a Phase 2 Crohn's disease study

Strong balance sheet: $252.9M* in cash to fund operations into at least H2 2026

CAPS: Cryopyrin-associated autoinflammatory syndromes
*Preliminary cash, cash equivalents and marketable securities balance as of December 31, 2024 (unaudited), subject to adjustments resulting from, among other things, the completion of our end-of-period reporting processes and related activities, including the audit by our independent registered public accounting firm of our financial statements.

Experienced Leadership Team

Executive Team

Raju Mohan, PhD
CHIEF EXECUTIVE OFFICER, FOUNDER

Mark Forman, MD, PhD
CHIEF MEDICAL OFFICER

Matthew Moore
CHIEF OPERATING OFFICER

John Nuss, PhD
CHIEF SCIENTIFIC OFFICER

Board Of Directors

Sheila Gujrathi, PhD
EXECUTIVE CHAIR, VENTYX

Raju Mohan, PhD
CHIEF EXECUTIVE OFFICER, VENTYX

Onaiza Cadoret-Manier
CHIEF EXECUTIVE OFFICER, STEALTH BIOTECH

Allison Hulme, PhD
CHIEF EXECUTIVE OFFICER, AEOVIAN PHARMACEUTICALS

Somu Subramaniam
MANAGING PARTNER, NEW SCIENCE VENTURES

William White
CHIEF FINANCIAL OFFICER, AKERO THERAPEUTICS

Internally Discovered Clinical-Stage Pipeline

Addressing Autoimmune, Inflammatory and Neurodegenerative Diseases with High Unmet Need

TARGET	PROGRAM	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	NEXT ANTICIPATED MILESTONES
NLRP3	VTX2735 *Peripheral*			Recurrent Pericarditis		Ph 2 data **H2 2025**
	VTX3232 *CNS-Penetrant*			Parkinson's Disease		Ph 2 data **H1 2025**
	VTX3232 *CNS-Penetrant*			Cardiometabolic Diseases (CMD) / Obesity		Ph 2 data **H2 2025**
S1P1	Tamuzimod*				Ulcerative Colitis	Phase 3 Ready*
TYK2	VTX958			Crohn's Disease		Phase 2 analysis underway

Cash, cash equivalents and marketable securities of $252.9M (unaudited) as of December 31, 2024 are expected to fund operations into at least the second half of 2026**

*Formerly VTX002. Available for partnering or non-dilutive financing.
**Preliminary cash, cash equivalents and marketable securities balance as of December 31, 2024 (unaudited), subject to adjustments resulting from, among other things, the completion of our end-of-period reporting processes and related activities, including the audit by our independent registered public accounting firm of our financial statements.

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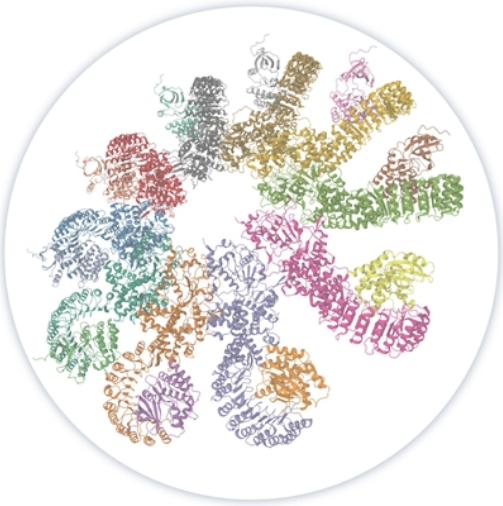
NLRP3 Inhibition

Broad potential in inflammatory diseases

NLRP3 Inflammasome: A Key Component of Innate Immunity

Dysregulation Linked to a Broad Range of Inflammatory Diseases

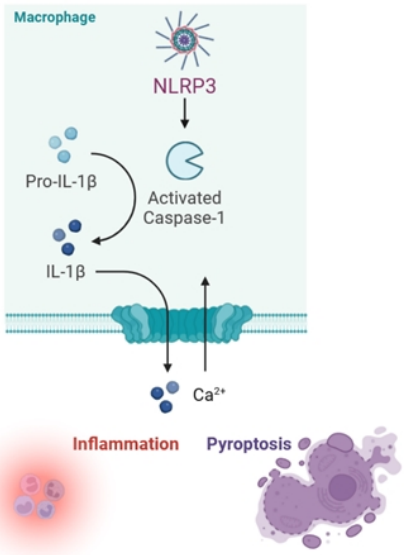


Active **NLRP3** inflammasome disk

Nod-Like Receptor family Pyrin domain containing 3

INFLAMMASOMES are activated by molecular hallmarks of infection or cellular injury

NLRP3 mediates release of proinflammatory cytokines **IL-1β** and **IL-18** and drives a form of cell death called **PYROPTOSIS**



NLRP3 is a High Value Therapeutic Target

Broad Potential Across Systemic and CNS Inflammatory Disease

VTX2735

Systemic Diseases

NLRP3 inhibition has therapeutic potential in a broad range of systemic diseases, particularly where IL-1 biologics have demonstrated therapeutic benefit

- Cardiovascular
- Dermatologic
- Rheumatic
- CAPS (FCAS)
- Other orphan indications

VTX3232

Neuroinflammatory Diseases

NLRP3 activation (inhibition) has been linked to a range of neuroinflammatory and neurodegenerative conditions with high unmet medical need

- Parkinson's Disease
- Multiple Sclerosis
- Alzheimer's Disease
- Cardiometabolic & obesity

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CNS: Central nervous system. CAPS: Cryopyrin-associated periodic syndromes. FCAS: Familial cold autoinflammatory syndrome.
Red color indicates disease areas under active investigation.

ventyx BIOSCIENCES

VTX2735

Peripheral NLRP3 Inhibitor

VTX2735: Potent and Selective Peripheral NLRP3 Inhibitor

Phase 2 Ready for Systemic Inflammatory Diseases

PATENT GRANTED
(US Pat. No. 11,603,375)

POTENT & SELECTIVE

- hu WB IC_{50} (IL-1β) = 80 nM
- No inhibition of other inflammasomes

NONCLINICAL & PHASE 1 PACKAGE

- Demonstrated pharmacodynamic effects and *in vivo* efficacy in rodent models
- High exposures and target coverage achieved in Phase 1
- Safety profile established in nonclinical and clinical studies
  - Well-tolerated in healthy adults
  - No safety signals in nonclinical *in vitro* and *in vivo* studies
  - Chronic tox studies completed
- Potent inhibitor of NLRP3 in PBMC from CAPS patients (FCAS mutations)

Phase 2 proof-of-concept study in CAPS patients completed

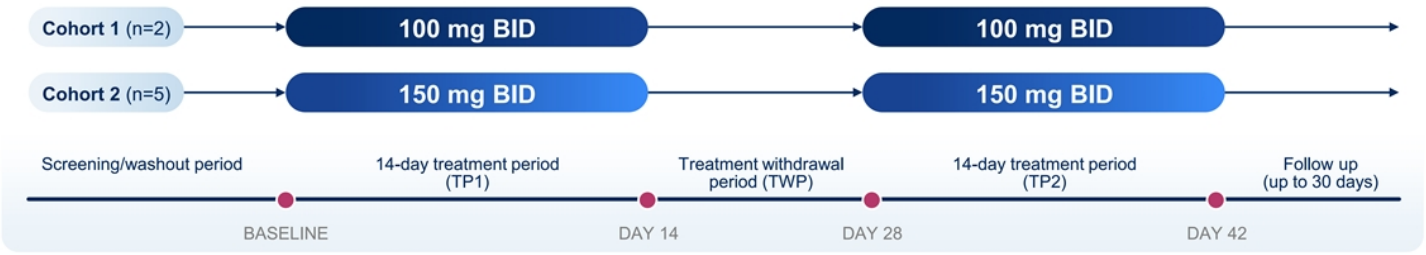
ventyx BIOSCIENCES

Source: Ventyx internal data. CAPS: Cryopyrin-associated periodic syndromes. FCAS: Familial cold autoinflammatory syndrome. WB: whole blood. PMBC: peripheral blood mononuclear cells.

VTX2735 Phase 2 Open-Label Trial in CAPS (FCAS)

Trial Design

- **CAPS** is an ultra rare condition driven by **gain of function mutations in NLRP3 gene;** FCAS is the most common subtype
- Following washout of SoC, VTX2735 dosed for 14 days in two treatment periods (TP1 and TP2, 28 days total)
- Key endpoints: safety/tolerability and improvement in Key Symptom Score (**KSS**, mean of 5 symptom scores)
  - **Pharmacodynamic assessments**: hsCRP; acute phase reactants (SAA, IL-1α, IL-1β, IL-6, and IL-18)
- **7 participants enrolled** (diverse NLRP3 mutations, prior SoC therapies, and symptoms)
  - 5 participants completed the trial; 2 participants withdrew consent after treatment period 1



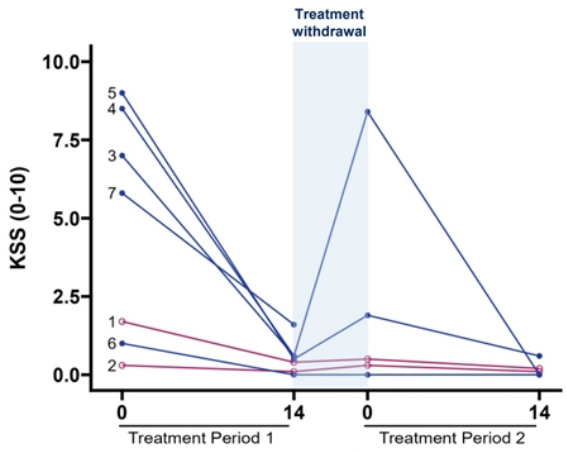
Source: Ventyx data on file. CAPS: Cryopyrin-associated periodic syndrome. FCAS: Familial cold autoinflammatory syndrome. SoC: standard of care. SAA: serum amyloid A. hsCRP: high sensitivity C-reactive protein.

Treatment with VTX2735 Drives Reductions in Disease Activity

Disease Activity as Assessed by Key Symptom Score (KSS) and General Well-Being

KEY SYMPTOM SCORE (0-10)*

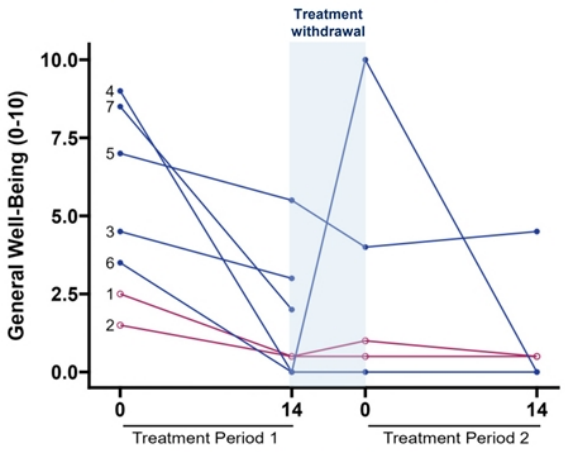
Daily mean of five symptom scores



Mean 85% reduction during Treatment Period 1

GENERAL WELL-BEING (0-10)*

"Considering all the ways FCAS affects you, please rate how you are doing"



Mean 68% reduction during Treatment Period 1

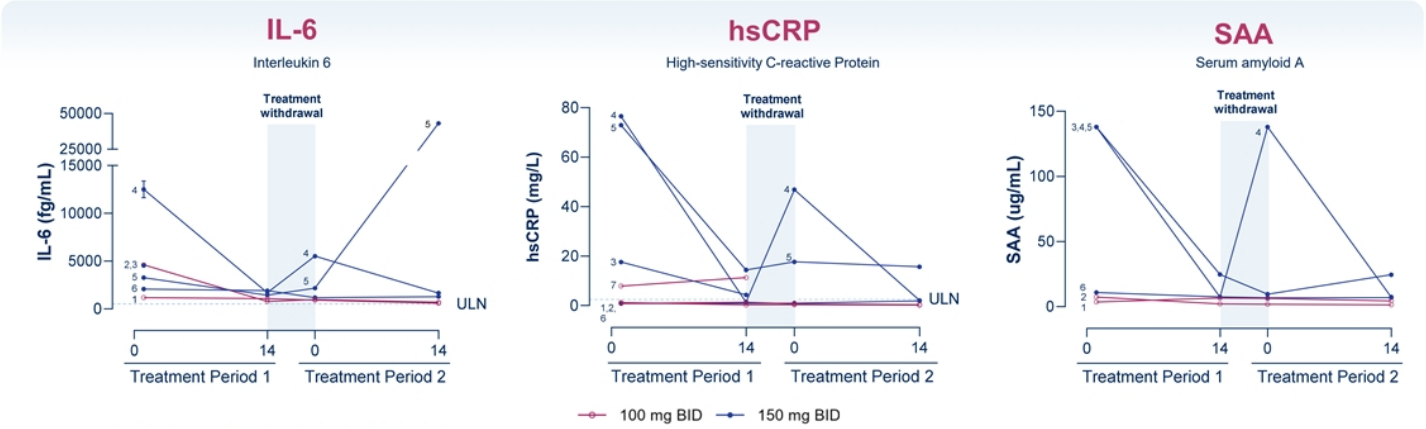
100 mg BID — 150 mg BID

Source: Ventyx data on file (07 March 2024).
*Note: Number next to each line represents individual participant number. One participant did not complete Treatment Period 1; the final measurement prior to study discontinuation is reported.

VTX2735 Biomarker Changes

Reductions in IL-6, hsCRP and SAA Observed as Expected with NLRP3 Inhibition

- The pleiotropic cytokine IL-6 induces acute-phase reactant proteins, including C-reactive protein (CRP) and Serum amyloid A (SAA)
- Treatment with VTX2735 reduced plasma IL-6, hsCRP, and SAA in patients with elevations at baseline, consistent with reductions in disease activity
  - Lack of baseline elevations in some patients is likely attributable to long half-life of SoC treatment (canakinumab)



Source: Ventyx data on file. SoC: standard of care.
*Note: Number next to each line represents individual participant number. One participant did not complete Treatment Period 1; the final measurement prior to study discontinuation is reported. No IL-6 or SAA data available for Participant No. 7.

Conclusions from the Phase 2 Trial of VTX2735 in FCAS Patients

Clinical Proof of Concept Achieved in CAPS Patients

VTX2735 showed clinically-meaningful effects on disease activity and relevant biomarkers

VTX2735 was well-tolerated; all adverse events categorized as mild or moderate and resolved without treatment interruption

Clinical outcomes and biomarker changes represent a major milestone for VTX2735 and for NLRP3 inhibition

- Dr. Hal Hoffman (UCSD): “Results similar to what we have seen with IL-1 targeted biologics; particularly impressive in a treatment-experienced population.”

ventyx BIOSCIENCES

CAPS: Cryopyrin-associated periodic syndrome. FCAS: Familial cold autoinflammatory syndrome.

VTX2735 is a Phase 2 Ready Peripheral NLRP3 Inhibitor

HIGHLY POTENT & SELECTIVE

- Structurally unique, selective inhibitor of NLRP3
- Potent inhibitor of NLRP3 with IC_{50} = 80 nM in human whole blood assay
- Highly potent vs. CAPS mutation variants

BIOLOGIC-LIKE ACTIVITY IN CAPS TRIAL

- Concentration dependent suppression of IL-1β *ex vivo*
- Reduction in hsCRP and other inflammation biomarkers (IL-6, SAA, neutrophils)
- Clinically-meaningful benefits observed in CAPS patients

PROMISING SAFETY PROFILE

- Well-tolerated in healthy adults and CAPS patients
- No toxicological signals of concern
- No CYP, hERG or transporter interactions

PHASE 2 READY

- IP position secure; patent issued (US Pat. No. 11,603,375)
- Multi-kilo API production completed
- Once-daily dosing form in development

ventyx BIOSCIENCES

Source: Ventyx data on file. CAPS: Cryopyrin-associated periodic syndrome. nM: nanomolar. hsCRP: high sensitivity C-reactive protein. SAA: serum amyloid A. CYP: Cytochrome P450. hERG: human ether-à-gogo gene. API: active pharmaceutical ingredient.

Attractive Opportunity for NLRP3 in Recurrent Pericarditis

De-risked Mechanism and Efficient Path to Market

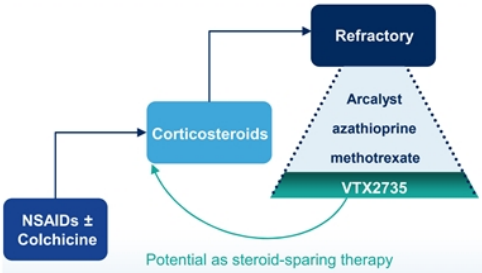
Recurrent pericarditis is a debilitating autoinflammatory condition

~40,000 patient U.S. prevalent population with recurrent pericarditis[1]

Autoinflammatory process characterized by IL-1 release (downstream of NLRP3)

- 2021 approval of Arcalyst (rilonacept) validates IL-1 approach (de-risking for NLRP3)
  - Arcalyst generated $233M in 2023 sales in 2nd full year of commercial availability; consensus sales >$1B in 2030[2]
- Regulatory precedent for efficient path to market
  - Open-label Phase 2 trial followed by a single Phase 3 trial
- Topline Phase 2 data for VTX2735 expected in **H2 2025**

TREATMENT PARADIGM



Source: 1. Klein et al., J Am Heart Assoc. 2021 Aug 3;10(15):e018950. doi: 10.1161/JAHA.120.018950; 2. Historical sales from Kiniksa investor presentation; consensus sales from Evaluate Pharma.

Phase 2 Trial in Participants with Recurrent Pericarditis

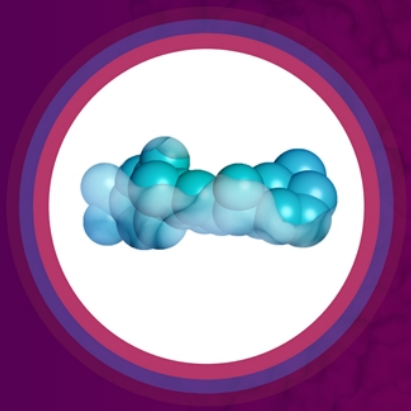
Designed to Efficiently Achieve Clinical Proof of Concept in RP

- Phase 2 trial evaluating the effects of VTX2735 in participants with active recurrent pericarditis
  - Acute symptoms of RP (pain, elevated hsCRP) despite standard therapy
- Measuring resolution of acute symptoms of RP as evidenced by:
  - Change from baseline in hsCRP (inflammatory marker)
  - Change from baseline in the NRS pain score
- Positive results may support rapid advancement to registrational Phase 3*

Topline Phase 2 data expected H2 2025

RP: recurrent pericarditis. NRS: numerical rating scale (11-point scale). hsCRP: high sensitivity C-reactive protein. BID: twice daily.
*Pending alignment with regulators.
**Defined as reduction in pain, hsCRP, and/or echographic effusion

ventyx BIOSCIENCES

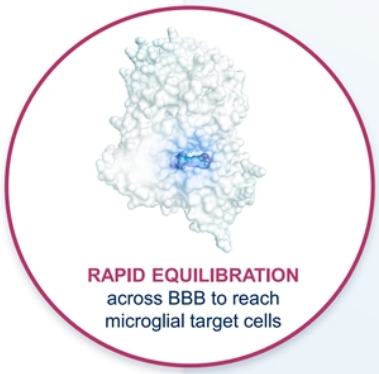


VTX3232

CNS-Penetrant NLRP3 Inhibitor

VTX3232: Potent and Selective CNS-Penetrant NLRP3 Inhibitor

RATIONALLY DESIGNED AND OPTIMIZED FOR CNS EXPOSURE



RAPID EQUILIBRATION
across BBB to reach microglial target cells

In Vitro Profile

- Hu WB IC_{50} (IL-1β) = 15 nM
- Mu WB IC_{50} (IL-1β) = 94 nM
- Inhibits palmitate-induced IL-1β
- No inhibition of other inflammasomes

In Vivo Profile

- Safe and well-tolerated in Phase 1 Study
- Equal CNS partitioning; human Kp,uu = 0.5
- $T_{½}$ = ~17 h with high free-drug fraction
- Robust effects on inflammatory biomarkers

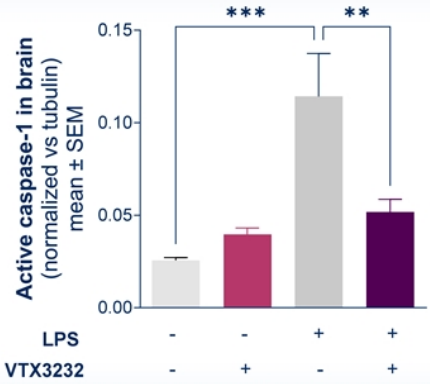
Pharmaceutics

- Single polymorph
- BCS Class 1
- Solubility (pH 7.4 PB) = 0.4 mg/mL

Source: Ventyx internal data. BBB: blood brain barrier. CNS: central nervous system. Hu: human. Mu: mouse. WB: whole blood. Kp,uu: partition coefficient. BCS: biopharmaceutics classification system.

VTX3232 Efficacy in Neuroinflammation Models

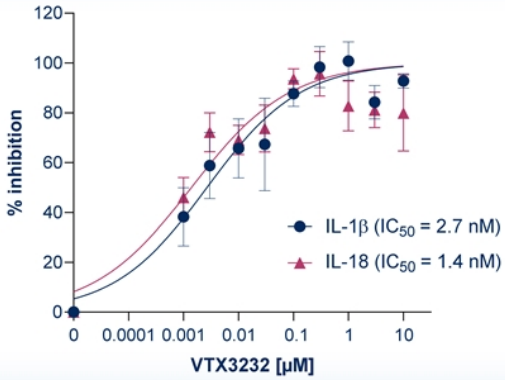
MOUSE NEUROINFLAMMATION MODEL



($^{**}P$<0.01,$^{***}P$<0.001; One-Way ANOVA)

Inhibition of caspase-1 activation
(directly downstream of NLRP3)

LPS-PRIMED HUMAN MICROGLIA



Mean ± SEM, min. 4 donors

Potent inhibition of induced IL-1β & IL-18
selective vs TNFα

VTX3232 activity translates to CNS-relevant assays and models

Source: Ventyx internal data. CNS: central nervous system.

VTX3232 Phase 1 SAD and 14-Day MAD Trial in Healthy Volunteers

VTX3232 achieved optimal exposures in Phase 1 with favorable safety profile

Generally well-tolerated following single- and multiple-dose administration

- No dose-limiting toxicities
- All treatment emergent adverse events considered mild or moderate

Pharmacokinetic profile consistent with once-daily dosing

- Comparable exposure in both plasma and CSF
- No effect of food on exposure

Potent target engagement at doses ≥3 mg

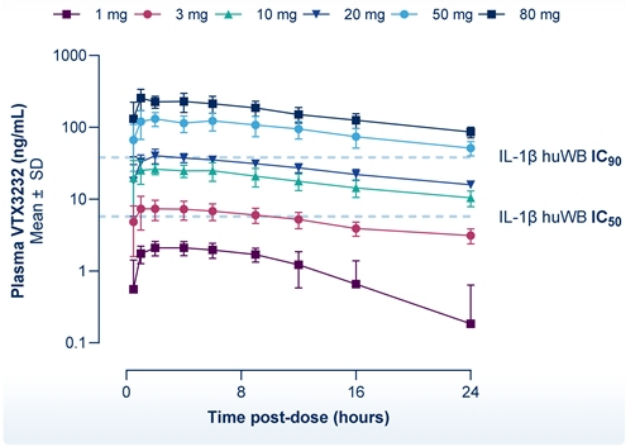
- Dose-dependent pharmacodynamic effects in *ex vivo* IL-1β assay and on downstream inflammatory biomarkers
- Predict trough drug concentrations in CSF above IC_{90} at doses > 12 mg

ventyx BIOSCIENCES

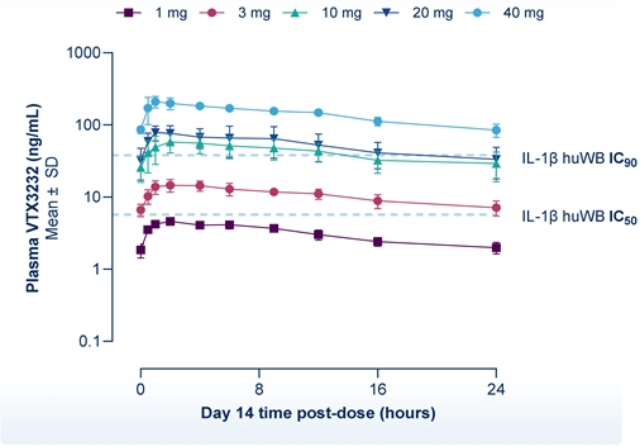
SAD: single ascending dose. MAD: multiple ascending dose. CSF: cerebrospinal fluid. IC: inhibitory concentration.

VTX3232 Phase 1 SAD and 14 Day MAD Pharmacokinetics

SINGLE ASCENDING DOSE



MULTIPLE ASCENDING DOSE



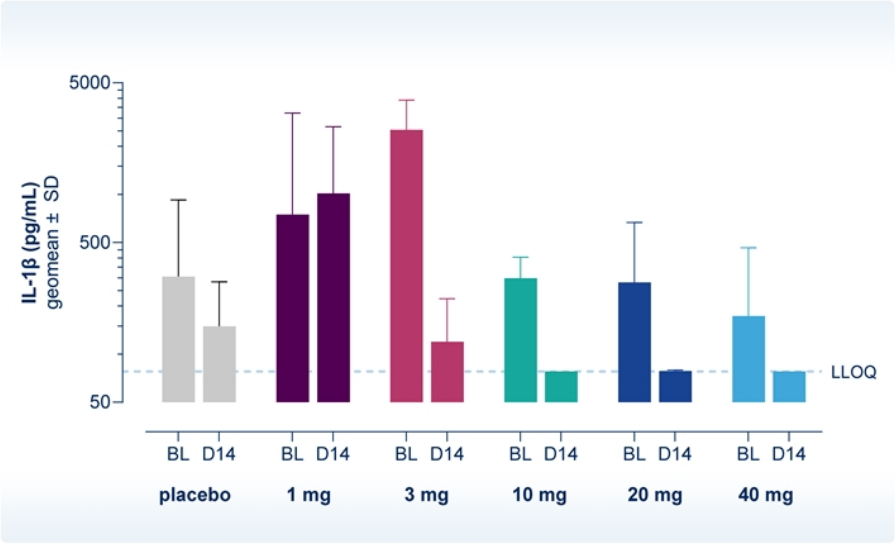
Dose-related, linear exposure from 1 mg to 80 mg
3 mg QD achieves 24 h IL-1β IC_{50} coverage

ventyx BIOSCIENCES

Source: Ventyx internal data. SAD: single ascending dose. MAD: multiple ascending dose. huWB: human whole blood. IC: inhibitory concentration. QD: once daily.
Note: huWB IC_{50}/IC_{90} based on *ex vivo* IL-1β assay.

VTX3232 Whole Blood *Ex Vivo* IL-1β Stimulation Assay

Potent Target Engagement Demonstrated At Doses ≥3 mg QD



DATA SUMMARY

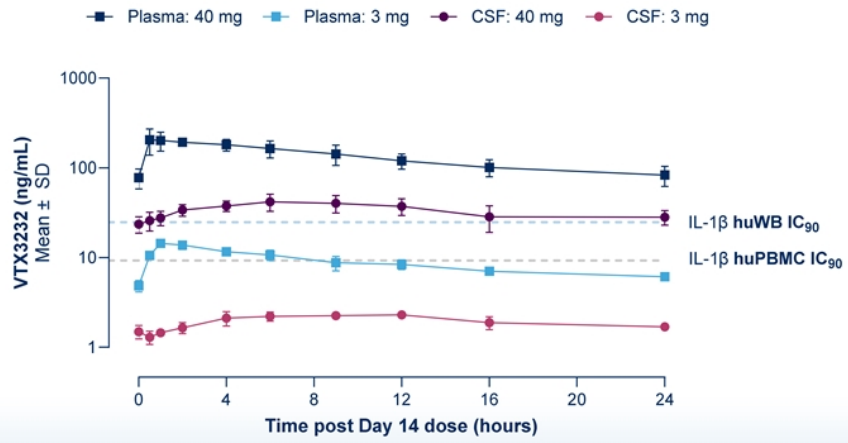
- Blockade of NLRP3 mediated IL-1β secretion is maintained at Day 14 with repeat dosing
- Maximal inhibition achieved at doses of 10 mg QD and higher

1. Lower Limit of Quantitation (LLOQ)= 78 pg/mL. All subjects below LLOQ were assigned a value of 78 pg/mL.
2. Day 14 pre dose (D14). Pre dose baseline (BL).
Source: Ventyx internal data. QD: once daily.

VTX3232 Pharmacokinetics in Cerebrospinal Fluid (CSF)

Comparable exposures in both plasma and CSF

MATCHED PLASMA & CSF EXPOSURE IN MAD COHORTS



DATA SUMMARY

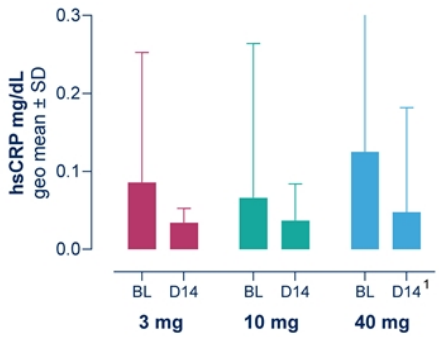
- 40 mg QD exceeds CSF IC_{90} for 24 h, achieving robust target coverage for NLRP3 in microglia for neuroinflammatory conditions

Source: Ventyx internal data. huWB: human whole blood. IC: inhibitory concentration. QD: once daily. CSF: cerebrospinal fluid.
Note: huWB IC_{50}/IC_{90} based on *ex vivo* IL-1β assay.

VTX3232 Effects on Inflammatory Biomarkers

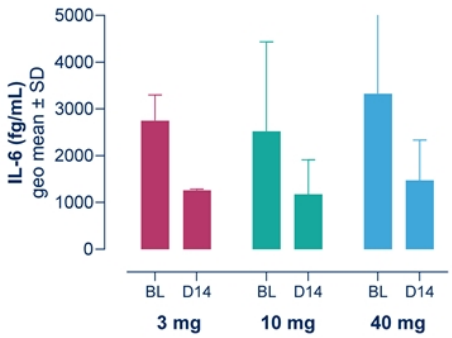
Reduction in hsCRP and IL-6 Comparable to that Achieved by Canakinumab (IL-1β mAb)

PLASMA hsCRP

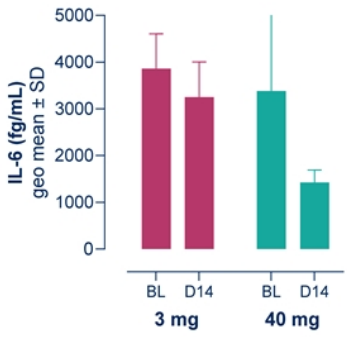


Systemic inflammation biomarker
hsCRP lowered by as much as 55%

PLASMA IL-6



CSF IL-6



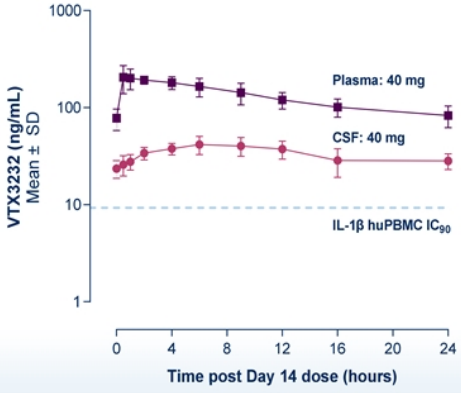
IL-6 lowered by as much as 46% in plasma

1. Day 14 pre dose samples not available for 40mg cohort. Data 2hr post dose displayed.
Source: Ventyx internal data. BL: pre-dose baseline. D14: Day 14 pre-dose samples, unless otherwise noted.

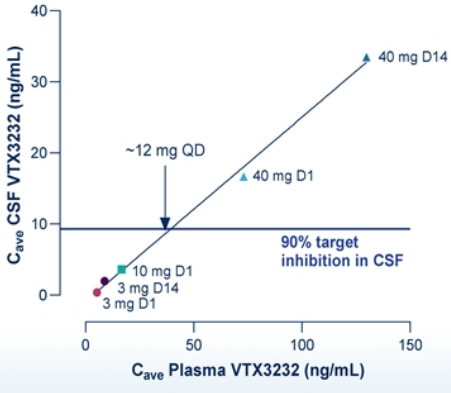
Conclusions from the Phase 1 Trial of VTX3232 in NHV

Potentially Class-leading Safety and Efficacy Profile for Neuroinflammatory Diseases

40 MG QD DOSE: TARGET COVERAGE



DATA PREDICT LOW EFFICACIOUS DOSES



DATA SUMMARY

- Well-tolerated in healthy volunteers
- Robust target coverage achieved in the plasma and CNS
- Potent, dose-dependent PD effect in *ex vivo* IL-1β assay and on inflammatory biomarkers
- Data predict trough concentrations in CSF above IC_{90} at doses > 12 mg

ventyx BIOSCIENCES

Source: Ventyx internal data. CNS: central nervous system. PD: pharmacodynamic. CSF: cerebrospinal fluid. IC: inhibitory concentration. QD: once daily.

VTX3232: Optimally Engineered to Target Neuroinflammatory Diseases

POTENT & SELECTIVE

- Structurally unique
- K_d < 1 nM to NLRP3 NACHT domain
- IC_{50} = 13 nM hu WB, 2.7 nM in microglia
- Selective vs AIM2/NLRC4
- Doses >3 mg suppress IL-1β release for >24 h

PK & PD PROFILE

- T½ = ~17 h with high free fraction
- High CNS penetration; human Kp,uu = 0.5
- Data predict trough concentrations in CSF above IC_{90} at doses > 12 mg

SAFETY PROFILE

- Well-tolerated in healthy adults
- No toxicological signals requiring further investigation
- No CYP, hERG, or transporter interactions

PHASE 2 READY

- IP position secure; patent application published 09/23
- Multi-kilo API production complete
- Solid-oral dosing form with high bioavailability

ventyx BIOSCIENCES

Source: Ventyx internal data. K_d: dissociation constant. nM: nanomolar. IC: inhibitory concentration. AIM2: absent in melanoma 2. NLRC4: NLR family caspase activation and recruitment domain-containing 4. $T_{1/2}$: half-life. CNS: central nervous system. CSF: cerebrospinal fluid. CYP: Cytochrome P450. hERG: human ether-à-gogo gene. API: active pharmaceutical ingredient.

VTX3232 Has Potential for Disease Modification in Parkinson's Disease

Strong Mechanistic Rationale and High Unmet Need

HIGH UNMET NEED

- **~1 million** U.S. patient prevalent population (2nd most common neurodegenerative disease)
- **No disease-modifying therapies** approved for Parkinson's disease

LARGE ADDRESSABLE MARKET

- **>$4B annual market** for symptomatic therapies in 2021[1]
- Estimated **~$10-$15B+ annual TAM** for first disease-modifying therapy[2]

STRONG BIOLOGIC RATIONALE

- **Neuroinflammation** is central to Parkinson's disease pathogenesis
- **Strong evidence** in preclinical models and PD patient samples for NLRP3 as a **key driver** of neuronal degeneration

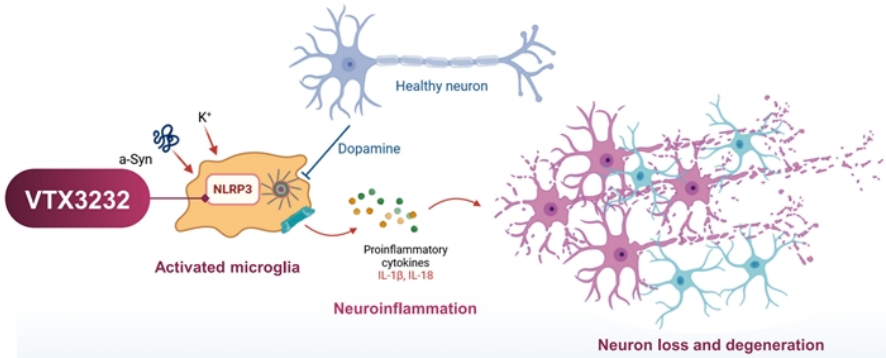
ventyx BIOSCIENCES

Source: 1. Grand View Research Parkinson's disease market report; 2. ClearView Healthcare Partners market research for internal use. TAM: total addressable market.

Rationale for Targeting NLRP3 in Parkinson's Disease (PD)

Neuroinflammation May Play a Central Role in Disease Pathogenesis

There is a growing body of evidence for NLRP3 inhibition as a **potential disease-modifying approach** that may prevent dopaminergic neurodegeneration and clinical symptoms



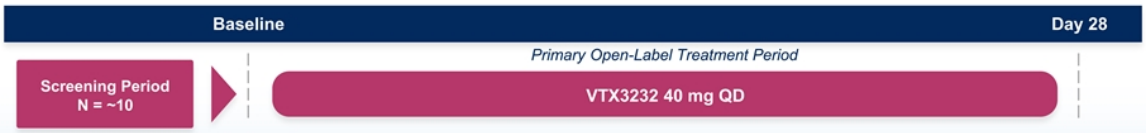
- NLRP3 inhibition **reduces motor symptoms, microglial activation, and dopaminergic neuron loss** in multiple animal models of PD
- In patients, **NLRP3 expression is linked to progression and risk of disease**, and NLRP3-derived cytokines are elevated
- **Neuroinflammation through excess NLRP3 activity may be the mechanistic link driving neuronal cell death in PD**

Source: Liang T et al. 2022 Front. Pharmacol. 13:845185. Ising, C et al. Nature 575, 669–673 (2019). Panicker et al. Neuron. 2022;110(15):2422-2437.e9. Grotemeyer, A. et al., J Neuroinflammation 20, 79 (2023). Huang et al., J Nueroimmunology, Volume 354, 2021, 577543. Gordon et al., Sci. Transl. Med. 10,eaah4066(2018).

Phase 2 Trial in Participants with Early Parkinson's Disease

Disease-Relevant Biomarkers and Exploratory Neuroimaging

- Conducting a Phase 2 safety and biomarker trial assessing NLRP3-, inflammatory, and disease-relevant biomarkers, with exploratory neuroimaging
  - Impact on relevant plasma and CSF biomarkers: hsCRP, IL-1β, IL-6, IL-18, α-syn, NfL, sTREM2, GFAP
  - Impact on microglial inflammation via neuroimaging (TSPO-PET)
- Test of therapeutic hypothesis that CNS NLRP3 inhibition will result in reduced inflammation and potential early disruption of PD pathophysiology



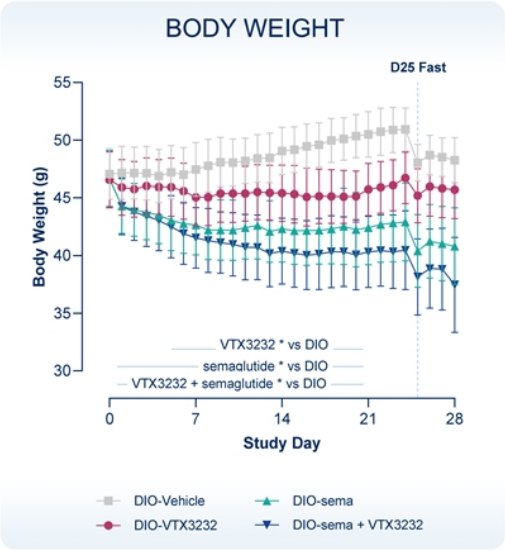
Topline Phase 2 data expected H1 2025

ventyx BIOSCIENCES

α-syn: α-synuclein.GFAP: glial fibrillary acidic protein. NfL: neurofilament light chain. PD: Parkinson's disease. TSPO: translocator protein 18 kDa.
Note: NCT06556173. QD: once daily.

NLRP3 as a Target in Obesity and Obesity-Related Metabolic Disease

- Obesity is a chronic inflammatory condition associated with release of NLRP3-related cytokines such as IL-1β and IL-6
  - This inflammation may drive a range of metabolic disorders, including insulin resistance, diabetes, and atherosclerosis
  - Calorie restriction and exercise-mediated weight loss are associated with reduced expression of NLRP3 and decreased systemic inflammation[1]
  - In preclinical studies, NLRP3 activation is associated with obesity-related insulin resistance[1]
- VTX3232 demonstrates broad cardiometabolic benefits in diet-induced obesity (DIO) mouse model
  - Reduced food intake and decreased body weight
  - Decreased markers of systemic inflammation (IL-1β, IL-6, fibrinogen)
  - Improved markers of metabolic function (decreased cholesterol, triglycerides, insulin resistance, and HbA1c)



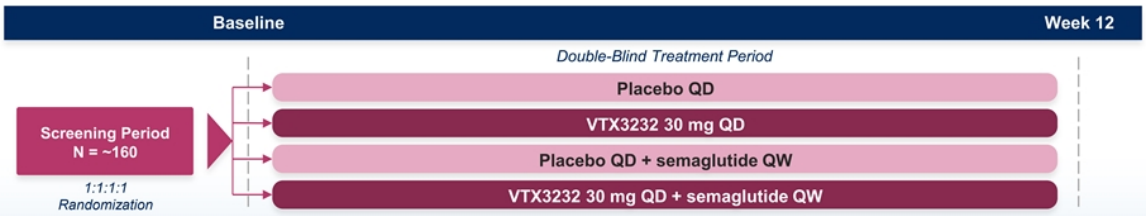
VTX3232 20 mg/kg BID orally; Semaglutide 10 μg/kg QD subcutaneously; mean ± SEM, * p <0.05 or more highly significant at all indicated timepoints, Mixed effects ANOVA, Sidak's post-hoc test.

Source: 1. Vandanmagsar et al., Nat Med. 2011 Feb;17(2):179-88. doi: 10.1038/nm.2279. Ventyx internal data.
DIO: diet-induced obesity. sema: semaglutide.

Phase 2 Trial in Obese Participants with Cardiometabolic Risk Factors

Measuring Key Inflammatory Biomarkers and Changes in Body Composition

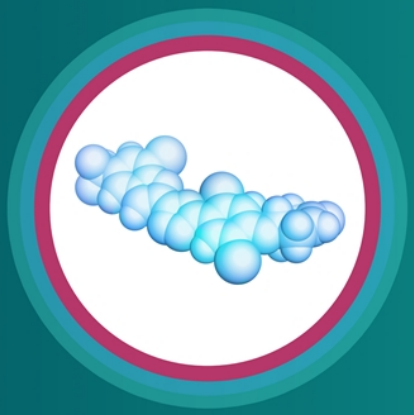
- Conducting a Phase 2 trial in obese participants with cardiometabolic risk factors, exploring a broad panel of cardiometabolic and inflammatory endpoints:
  - **Inflammatory (cardiovascular) biomarkers**, including IL-6, hsCRP and other acute phase reactants
  - **Cardiometabolic markers**, including lipids and glycemic parameters
  - Impact on **body weight** and **body composition**, including liver steatosis
- Trial expected to support assessment of cardiometabolic development opportunities for NLRP3 portfolio



Topline Phase 2 data expected H2 2025

QD: once daily. QW: once weekly.

ventyx BIOSCIENCES



Tamuzimod (VTX002)

S1P1 Receptor Modulator for Ulcerative Colitis

Tamuzimod Phase 2 Study in Moderate-to-Severe UC

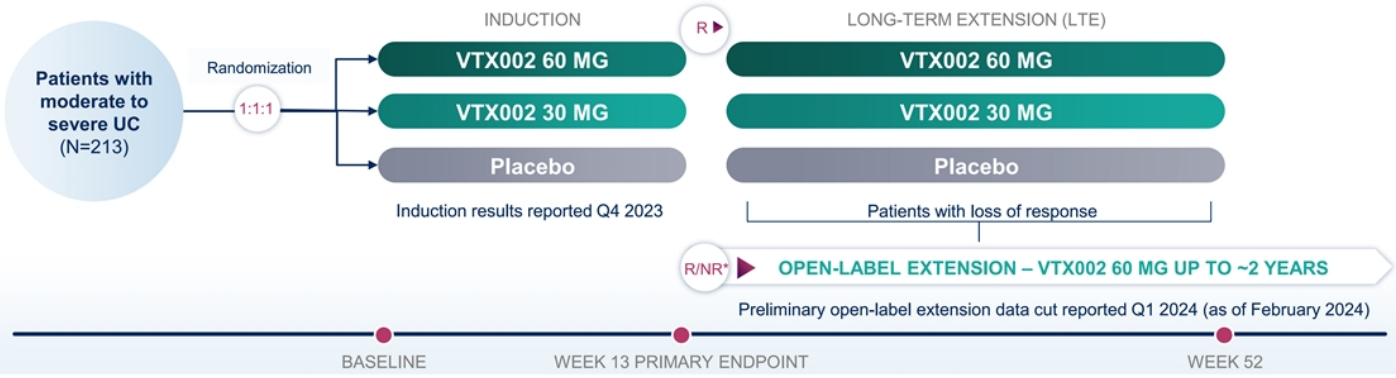
Designed to Serve as the First of Two Pivotal Trials

KEY ELIGIBILITY CRITERIA

- Patients with moderately to severely active UC as defined by the Modified (3-component) Mayo Score
- Insufficient response, loss of response, and/or intolerance to conventional or advanced therapies

ENDPOINTS

- **Primary Endpoint:** Clinical remission at Week 13 as defined by the Modified Mayo Score
- **Key Secondary Endpoints**: Endoscopic improvement; symptomatic remission; histologic remission; endoscopic improvement-histologic remission



ventyx BIOSCIENCES

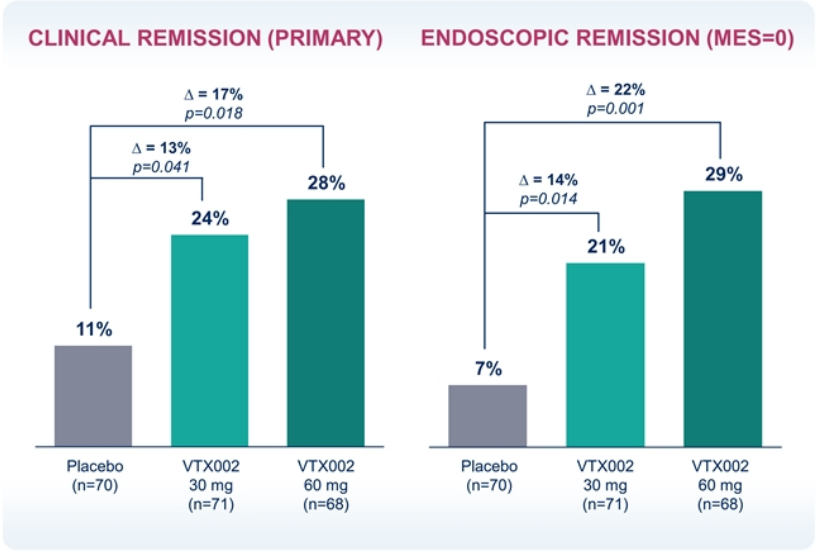
* Protocol version 1 study design. All patients had the option to roll-over regardless of response status. Subsequent protocol versions had patients with NR or loss of response only enter OLE
Note: NCT05156125. MMS: Modified Mayo Score; R: responder; NR: non-responder; UC: ulcerative colitis.

Overview of Tamuzimod Induction Data

Robust Week 13 Clinical Remission with Differentiated Complete Endoscopic Remission

Key Takeaways from Tamuzimod Week 13 Data

1. **Competitive week 13 clinical remission with differentiated endoscopic remission (MES=0)**
2. **Deep remission** (endoscopic and clinical remission), **symptomatic remission** and **histologic endoscopic mucosal improvement** rates further support clinical profile
3. Subgroup analysis demonstrated **differentiated clinical remission and endoscopic remission in patients with prior exposure to advanced therapies**
4. **No cases** of atrioventricular block, bradycardia, serious or opportunistic infections, or macular edema



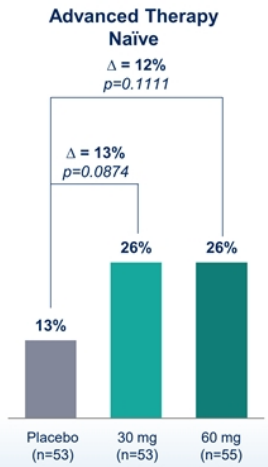
Baseline MMS 5 to 9 (N=209): Week 13

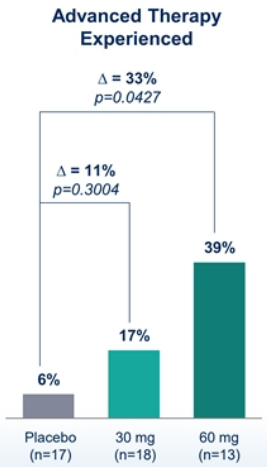
Clinical remission by modified Mayo Score (MMS) is defined as stool frequency subscore = 0 or 1, rectal bleeding subscore = 0, and endoscopic subscore ≤ 1 (excluding friability). Endoscopic remission is defined as a Mayo endoscopic subscore of 0. P-value for testing the treatment difference is based on Cochran-Mantel-Haenszel test adjusted for prior use of advanced therapies, baseline corticosteroid use and baseline disease activity. Full analysis set with non-responder imputation. Source: Ventyx data on file

Induction Subgroup Analysis: Advanced Therapy Prior Use

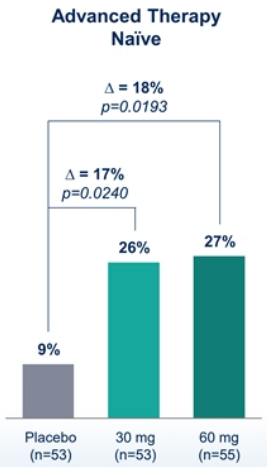
Clinical and Endoscopic Remission in both advanced therapy naïve and advanced therapy experienced subgroups

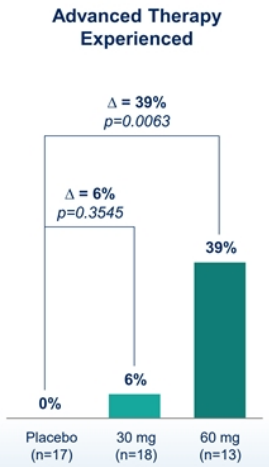
CLINICAL REMISSION





ENDOSCOPIC REMISSION





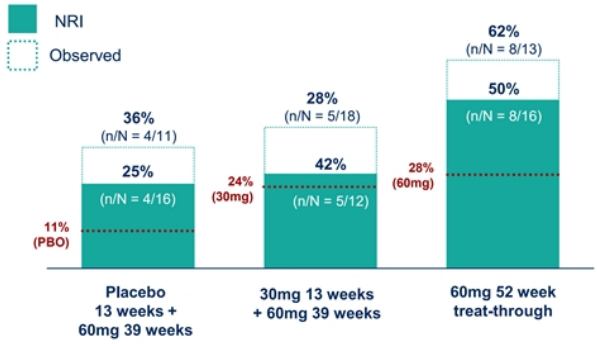
Clinical remission by modified Mayo Score (MMS) is defined as stool frequency subscore = 0 or 1, rectal bleeding subscore = 0, and endoscopic subscore ≤ 1 (excluding friability). Endoscopic remission is defined as a Mayo endoscopic subscore of 0. P-value for testing the treatment difference is based on Cochran-Mantel-Haenszel test adjusted for prior use of advanced therapies, baseline corticosteroid use and baseline disease activity. Full analysis set with non-responder imputation. Source: Ventyx data on file

Preliminary Open-Label Extension Data

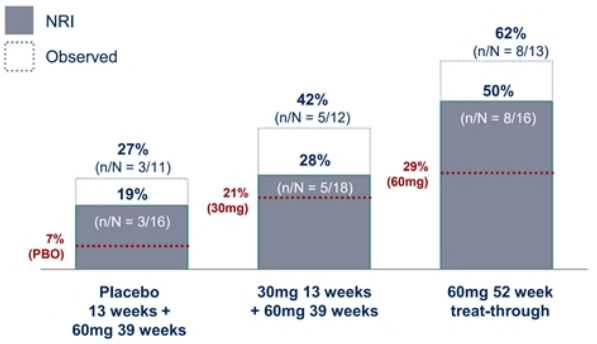
Further improvement in clinical and endoscopic remission rates at OLE week 39

..... % absolute endpoint rate (clinical or endoscopic remission) in induction dose arm at 13 weeks

% CLINICAL REMISSION AT OLE WEEK 39[1] BY INDUCTION DOSE ARM



% ENDOSCOPIC REMISSION[2] AT OLE WEEK 39[1] BY INDUCTION DOSE ARM



At least half (NRI) of patients in 60mg treat-through group reach clinical remission or endoscopic remission at week 52

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Note: NRI = non-responder imputation; participant discontinuations are assumed to be non-remitters
1 Irrespective of the clinical response at the end of the 13-week induction phase; VTX002 60mg / 60mg represents 52 weeks treat-through efficacy; other groups received 60mg for 39 weeks post- induction; 2 MES =0; Source: Ventyx data on file.

Endoscopic Remission is a Consensus Long-Term Treatment Goal

Current therapeutic outcomes remain disappointing: Tamuzimod has demonstrated the potential to set a new bar

CURRENT ENDOSCOPIC REMISSION OUTCOMES

1. **The vast majority of patients on advanced therapy fail to reach endoscopic remission, particularly within the induction period[1]:**

82-95% Absolute % patients in Phase 3 for advanced UC agents that fail to achieve MES=0 at induction

2. **Achievement of endoscopic remission (MES=0) vs. mild endoscopic activity (MES=1) is associated with improved long-term patient outcomes[2]:**

12-month risk of clinical relapse
(meta-analysis of 17 studies):

3. **Achievement of endoscopic remission (MES=0) is recognized in STRIDE II[3] guidelines as an aspirational target of long-term treatment:**

MES=0

Lower risk of disease recurrence	Decreased steroid use
Lower rate of surgical intervention & hospitalization	Superior patient-reported outcomes

TAMUZIMOD PROFILE

Induction data

- **Competitive clinical remission** and **differentiated endoscopic remission**
- Differentiated clinical and endoscopic **outcomes in prior advanced therapy subgroup**

OLE data

- Clinical remission and endoscopic remission rates at **OLE week 39 further differentiate VTX002**
- **Differentiated endoscopic remission rates** achieved in 52-week 60mg VTX002 treat-through group
- Competitive rates of **sustained clinical and endoscopic remission:**
  - At least 38% (NRI) of patients in 60mg 52wk treat-through arm were in clinical remission at both week 13 and week 52
  - Patients in clinical remission were also in endoscopic remission

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1 Based on analysis of Phase 3 trials for biologic and oral approved advanced agents; 2 Source: Yoon et al. Gastroenterology 2020;159(4):1262-1275.e7; 3 Source: Turner et al. Gastroenterology 2021;160:1570–1583

Tamuzimod (VTX002) Program Status

OLE/LTE data continue to support the differentiated profile of tamuzimod in ulcerative colitis

LTE phase completed mid 2024; data presented at UEGW in October 2024

Tamuzimod is Phase 3 ready (clinical, CMC, regulatory)

- End of Phase 2 meeting with FDA completed; EMA Scientific Advice meeting completed
- Phase 2 trial expected to serve as the first of two pivotal trials*

Ventyx working to identify partner or other source of non-dilutive financing to support future trials of VTX002 in ulcerative colitis

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*Pending alignment with regulators.

Internally Discovered Clinical-Stage Pipeline

Addressing Autoimmune, Inflammatory and Neurodegenerative Diseases with High Unmet Need

TARGET	PROGRAM	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	NEXT ANTICIPATED MILESTONES
NLRP3	VTX2735 *Peripheral*			Recurrent Pericarditis		Ph 2 data **H2 2025**
	VTX3232 *CNS-Penetrant*			Parkinson's Disease		Ph 2 data **H1 2025**
	VTX3232 *CNS-Penetrant*			Cardiometabolic Diseases (CMD) / Obesity		Ph 2 data **H2 2025**
S1P1	Tamuzimod*				Ulcerative Colitis	Phase 3 Ready*
TYK2	VTX958			Crohn's Disease		Phase 2 analysis underway

Cash, cash equivalents and marketable securities of $252.9M (unaudited) as of December 31, 2024 are expected to fund operations into at least the second half of 2026**

*Formerly VTX002. Available for partnering or non-dilutive financing.

**Preliminary cash, cash equivalents and marketable securities balance as of December 31, 2024 (unaudited), subject to adjustments resulting from, among other things, the completion of our end-of-period reporting processes and related activities, including the audit by our independent registered public accounting firm of our financial statements.