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 25, 2024

Vera Therapeutics, Inc.
(Exact name of registrant as specified in its charter)

Delaware	**001-40407**	**81-2744449**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

8000 Marina Boulevard, Suite 120
Brisbane, California **94005**
(Address of principal executive offices) **(Zip Code)**

(650) 770-0077
(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:

☐ 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
Class A common stock, $0.001 par value per share	VERA	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On January 25, 2024, Vera Therapeutics, Inc. (the "Company") announced positive 72-week data from the open label extension ("OLE") period of the Company's Phase 2b ORIGIN clinical trial of atacicept in patients with immunoglobulin A nephropathy ("IgAN"). A copy of the press release is furnished as Exhibit 99.1. In connection with the data release, the Company compiled a presentation entitled "R&D Day" (the "R&D Day Presentation") that includes the week 72 data from the Phase 2b ORIGIN clinical trial referenced above. A copy of the R&D Day Presentation is furnished as Exhibit 99.2. For important information about forward-looking statements, see the slide titled "Forward-Looking Statements" in Exhibit 99.2 attached hereto.

The information in this Item 7.01 of this Current Report on Form 8-K, including Exhibits 99.1 and 99.2, 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 or Sections 11 and 12(a)(2) of the Securities Act of 1933, as amended. The information contained in this Item 7.01, including Exhibits 99.1 and 99.2, shall not be incorporated by reference into any filing with the U.S. Securities and Exchange Commission ("SEC") made by the Company, whether made before or after the date hereof, regardless of any general incorporation language in such filing.

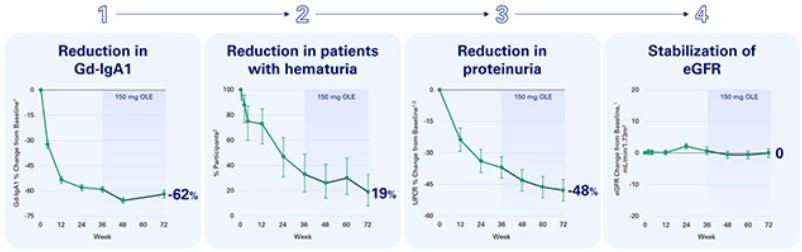
Item 8.01 Other Events.

As noted in Item 7.01, on January 25, 2024, the Company announced positive 72-week data from the OLE period of the Company's Phase 2b ORIGIN clinical trial of atacicept in patients with IgAN. Atacicept is the Company's potential best-in-class, disease-modifying dual inhibitor of the cytokines B-cell activating factor and a proliferation-inducing ligand. ORIGIN is a multinational, randomized, double-blind, placebo-controlled clinical trial (n=116) evaluating the efficacy and safety of atacicept in patients with IgAN who continue to have persistent proteinuria and remain at high risk of disease progression despite available ACE or ARB therapy.

After completing the 36-week randomized, double-blind, placebo-controlled period of the Phase 2b ORIGIN trial, all participants were eligible to receive atacicept 150 mg in the OLE. Of the 116 randomized participants, 106 completed 72 weeks.

Participants treated with atacicept for 72 weeks demonstrated a 62% reduction in Gd-IgA1, a reduction in the percentage of participants with hematuria to 19%, and a 48% reduction in urine protein to creatinine ratio ("UPCR") in the per-protocol ("PP") analysis. Importantly, participants had consistent and stable estimated glomerular filtration ("eGFR") with 0 mL/min/1.73m^2 change from baseline at 72 weeks. Of note, it has been shown that eGFR declines by approximately 1 mL/min/1.73m^2 per year in the general population.

Atacicept 72-week data from the Phase 2b ORIGIN trial are consistent with a profile of true disease modification



1. Mean ± SE; 2. Percentages represent number of participants with hematuria at each visit out of those with baseline hematuria. Data from participants originally randomized to any atacicept group in the double-blind period in the ITT analysis for Gd-IgA1, hematuria, and eGFR, and in prespecified week 36 PP analysis (excluded participants with protocol violations through week 36 as identified by blinded third-party CRO) for UPCR.

Participants who switched from placebo to atacicept demonstrated similar outcomes across each of the key indicators of IgAN as compared to participants originally randomized to atacicept during the first 36 weeks of the trial, including a 59% reduction in Gd-IgA1, a reduction in the percentage of participants with hematuria to 41%,

and a 47% reduction in UPCR in the PP analysis. In addition, eGFR stabilization was observed in participants who switched from placebo to atacicept with a -3.2 mL/min/1.73m^2 change from baseline at 72 weeks compared to -4.9 mL/min/1.73m^2 at 36 weeks.

Safety data in the OLE were consistent with the randomized period and indicated that atacicept was generally well-tolerated. These data build upon the prior experience of atacicept in randomized, double-blind, placebo-controlled clinical trials in over 1,500 participants to date across different indications – in which atacicept was generally well-tolerated.

Next Steps

The Company is continuing to advance the ongoing pivotal Phase 3 clinical trial of atacicept 150 mg. The Phase 3 ORIGIN 3 clinical trial was initiated in June 2023 and is expected to be fully enrolled in the second half of 2024. With ongoing data from the Phase 2b trial planned for presentation later in 2024 and Phase 3 topline results expected in the first half of 2025, if positive, the Company expects to submit a biologics license application ("BLA") for atacicept in IgAN to the U.S. Food and Drug Administration in the second half of 2025, with a projected commercial launch, if approved, in 2026.

Forward-looking Statements

Statements contained in this Current Report on Form 8-K regarding matters, events or results that may occur in the future are "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. Such forward-looking statements include statements regarding, among other things, the ability to execute against strategy for the Company's programs, atacicept's potential to be a best-in-class therapy and a disease-modifying treatment for patients with IgAN, the therapeutic potential of atacicept's dual inhibitor approach to treating the cause of IgAN, the Company's plans to complete enrollment of the pivotal Phase 3 ORIGIN 3 trial, the design and management of the Company's clinical trials, the Company's product candidates, strategy and regulatory matters and expectations regarding reporting longer term results from the Company's Phase 2b ORIGIN clinical trial, submitting a BLA and projected commercial launch. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements. Words such as "could," "expects," "will," "potential," "project," "plan," and similar expressions are intended to identify forward-looking statements. These forward-looking statements are based upon the Company's current expectations and involve assumptions that may never materialize or may prove to be incorrect. Actual results could differ materially from those anticipated in such forward-looking statements as a result of various risks and uncertainties, which include, without limitation, risks related to the regulatory approval process, results of earlier clinical trials may not be obtained in later clinical trials, preliminary results may not be predictive of topline results, risks and uncertainties associated with the Company's business in general, the impact of macroeconomic and geopolitical events and the other risks described in the Company's filings with the SEC. All forward-looking statements contained in this press release speak only as of the date on which they were made and are based on management's assumptions and estimates as of such date. The Company undertakes no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made, except as required by law.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Press Release of Vera Therapeutics, Inc., dated January 25, 2024.
99.2	Slide presentation entitled "R&D Day".
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

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

Vera Therapeutics, Inc.

Dated: January 25, 2024

By: /s/ Marshall Fordyce, M.D.
Marshall Fordyce, M.D.
Chief Executive Officer

Vera Therapeutics Presents Positive 72-Week Data Showing eGFR Stabilization in the Phase 2b ORIGIN Clinical Trial OLE in IgA Nephropathy

– Participants treated with atacicept for 72 weeks showed consistent and sustained reductions in Gd-IgA1, hematuria, and UPCR, with stable eGFR over the duration of treatment

– Placebo cohort participants who crossed over to atacicept 150 mg in the OLE had similar outcomes at 72 weeks as atacicept cohort in the first 36 weeks of the trial

– Atacicept was generally well-tolerated in the OLE period of the trial, consistent with the randomized period

– 72-week data provide additional confidence in the ongoing pivotal Phase 3 ORIGIN 3 clinical trial

BRISBANE, Calif., January 25, 2024 — Vera Therapeutics, Inc. (Nasdaq: VERA), a late clinical-stage biotechnology company developing and commercializing transformative treatments for patients with serious immunologic diseases, today announced positive 72-week data from the open label extension (OLE) period of its Phase 2b ORIGIN clinical trial of atacicept in participants with IgA nephropathy (IgAN). In aggregate, the 72-week data with atacicept are consistent with a profile of true disease modification in IgAN.

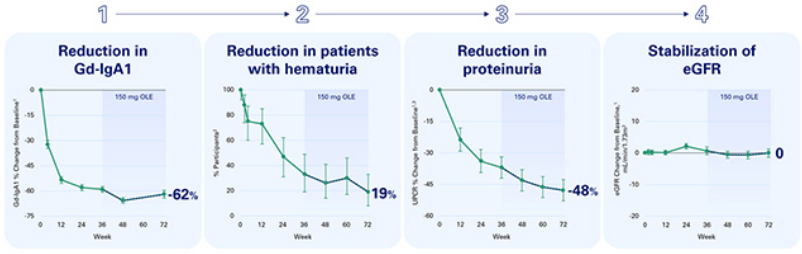
“Data from the OLE show the consistent and sustained reduction of Gd-lgA1, hematuria, and UPCR, as well as the stability of eGFR over 72 weeks in participants with IgAN. The demonstration of stable eGFR well beyond a year in participants receiving atacicept represents an important potential advancement for IgAN patients and has potential implications for the future treatment paradigm in this disease. It is also exciting that the data from the OLE show that switching to atacicept halted the eGFR decline seen in participants initially treated with placebo, with similar reductions in Gd-IgA1, hematuria, and UPCR as shown in the active cohort in the first 36 weeks,” stated Richard Lafayette, M.D., F.A.C.P., Professor of Medicine, Nephrology and Director of the Stanford Glomerular Disease Center at Stanford University Medical Center.

“We are thrilled to present this package of positive new data from the OLE of the Phase 2b ORIGIN clinical trial during our R&D Day, which will be held today in New York. We believe these data further support our belief that atacicept has the disruptive potential to stand out as a disease-modifying treatment for patients with IgAN,” said Marshall Fordyce, M.D., Chief Executive Officer of Vera Therapeutics. “This is an exciting period for Vera as the integrated data package for atacicept continues to mature. Importantly, the ongoing pivotal ORIGIN 3 trial is well underway, with enrollment on track to be completed in the second half of this year.”

After completing the 36-week randomized, double-blind, placebo-controlled period of the Phase 2b ORIGIN trial, all participants were eligible to receive atacicept 150 mg in the OLE. Of the 116 randomized participants, 106 completed 72 weeks.

Participants treated with atacicept for 72 weeks demonstrated a 62% reduction in Gd-IgA1, a reduction in the percentage of participants with hematuria to 19%, and a 48% reduction in UPCR in the per-protocol (PP) analysis. Importantly, participants had consistent and stable eGFR with 0 mL/min/1.73m^2 change from baseline at 72 weeks. Of note, it has been shown that eGFR declines by approximately 1 mL/min/1.73m^2 per year in the general population (Baba M, et al. PLOS ONE 2015).

Atacicept 72-week data from the Phase 2b ORIGIN trial are consistent with a profile of true disease modification



1. Mean ± SE; 2. Percentages represent number of participants with hematuria at each visit out of those with baseline hematuria. Data from participants originally randomized to any atacicept group in the double-blind period in the ITT analysis for Gd-IgA1, hematuria, and eGFR, and in prespecified week 36 PP analysis (excluded participants with protocol violations through week 36 as identified by blinded third-party CRO) for UPCR.

Participants who switched from placebo to atacicept demonstrated similar outcomes across each of the key indicators of IgAN as compared to participants originally randomized to atacicept during the first 36 weeks of the trial, including a 59% reduction in Gd-IgA1, a reduction in the percentage of participants with hematuria to 41%, and a 47% reduction in UPCR in the PP analysis. In addition, eGFR stabilization was observed in participants who switched from placebo to atacicept with a -3.2 mL/min/1.73m^2 change from baseline at 72 weeks compared to -4.9 mL/min/1.73m^2 at 36 weeks.

Safety data in the OLE were consistent with the randomized period and indicated that atacicept was generally well-tolerated. These data build upon the prior experience of atacicept in randomized, double-blind, placebo-controlled clinical trials in over 1,500 participants to date across different indications – in which atacicept was generally well-tolerated.

The R&D Day presentation, which includes the OLE 72 Week data slides and commentary, will be available on the Company's website at the Investor Calendar.

About the Phase 2b ORIGIN clinical trial

The Phase 2b ORIGIN clinical trial (NCT04716231) is a global, multicenter, randomized, double-blind, placebo-controlled trial evaluating the safety and efficacy of atacicept in 116 patients with IgAN who continue to have persistent proteinuria and remain at high risk of disease progression despite being on a stable prescribed regimen of RAASi for at least 12 weeks that is the maximum labeled or tolerated dose. The Phase 2b ORIGIN clinical trial evaluated three dose

strengths of atacicept versus placebo, administered weekly by prefilled syringe. Patients were randomized 2:2:1:2 to atacicept 150 mg, atacicept 75 mg, atacicept 25 mg, or matching placebo. Upon completion of the 36-week blinded treatment period, all patients were offered open-label atacicept 150 mg for an additional 60 weeks.

The primary endpoint was the change in proteinuria as evaluated by urine protein to creatinine ratio (UPCR) at week 24 and the key secondary endpoint was the change in proteinuria as evaluated by UPCR at week 36. Additional exploratory endpoints include change in proteinuria as evaluated by UPCR at weeks 12, 48, and 96; change in estimated glomerular filtration rate (eGFR); change in serum immunoglobulin levels, and serum Gd-IgA1 levels; safety and tolerability; and serum pharmacokinetics (PK).

The trial met its primary and key secondary endpoints, with statistically significant and clinically meaningful proteinuria reductions and stabilization of eGFR versus placebo through week 36. The safety profile was comparable between atacicept and placebo. The objectives of the trial are to determine the effect of atacicept on proteinuria and preservation of renal function compared to placebo to determine the appropriate dose(s) for further clinical development.

For more information about the Phase 2b ORIGIN clinical trial, please visit www.clinicaltrials.gov.

About the Phase 3 clinical trial (ORIGIN 3)

The ORIGIN 3 clinical trial (NCT04716231) is a global, multicenter, randomized, double-blind, placebo-controlled Phase 3 trial evaluating the safety and efficacy of atacicept 150 mg in patients with IgAN who continue to have persistent proteinuria and remain at high risk of disease progression despite being on a stable prescribed regimen of renin-angiotensin system inhibitors (RASi) (ACEi or ARB) for at least 12 weeks that is the maximum labeled or tolerated dose. The objectives of the trial are to determine the effect of atacicept on proteinuria and preservation of renal kidney function compared to placebo.

The Phase 3 trial is composed of up to a 4-week screening period, a 104-week double-blind treatment period, a 52-week open-label extension and 26 weeks of follow-up. Participants will be randomized 1:1 to atacicept 150 mg once weekly subcutaneous injections (N=188) or placebo once weekly subcutaneous injections (N=188) for 104 weeks, followed by a 52-week open-label extension. The primary endpoint is the change from baseline in proteinuria as evaluated by urine protein to creatinine ratio (UPCR) at week 36. The key secondary endpoint is annualized rate of change in estimated glomerular filtration rate (eGFR) up to week 104. Additional secondary endpoints are the change in Gd-IgA1, change in eGFR up to week 52, and time from randomization to first occurrence of composite kidney failure endpoint event.

For more information about the ORIGIN 3 clinical trial, please visit www.clinicaltrials.gov.

About IgA nephropathy (IgAN), or Berger's disease

IgAN, also known as Berger's disease, is a serious and progressive autoimmune disease of the kidney, for which there remains a high unmet medical need. IgAN is driven by the production of immunogenic galactose-deficient IgA1 (Gd-IgA1), which triggers autoantibodies that lead to the formation of pathogenic immune complexes, which become trapped in the kidney's glomeruli, causing inflammation and progressive damage. In up to 50 percent of patients, IgAN can lead to end-stage renal disease (ESRD) or kidney failure, which has considerable morbidity and impact on patients' lives.

About Atacicept

Atacicept is an investigational recombinant fusion protein that contains the soluble transmembrane activator and calcium-modulating cyclophilin ligand interactor (TACI) receptor that binds to the cytokines B-cell activating factor (BAFF) and A proliferation-inducing ligand (APRIL). These cytokines are members of the tumor necrosis factor family that promote B-cell survival and autoantibody production associated with certain autoimmune diseases, including IgAN and lupus nephritis. The Phase 2b ORIGIN clinical trial of atacicept in IgAN met its primary endpoint and showed a statistically significant reduction in mean proteinuria versus baseline at weeks 24 and 36. Vera believes atacicept is positioned for best-in-class potential, targeting B cells and plasma cells to reduce autoantibodies and having been administered to more than 1,500 patients in clinical studies across different indications.

About Vera Therapeutics

Vera Therapeutics is a late clinical-stage biotechnology company focused on developing treatments for serious immunological diseases. Vera's mission is to advance treatments that target the source of immunologic diseases in order to change the standard of care for patients. Vera's lead product candidate is atacicept, a fusion protein self-administered as a subcutaneous injection once weekly that blocks both B-cell activating factor (BAFF) and A proliferation-inducing ligand (APRIL), which stimulate B cells and plasma cells to produce autoantibodies contributing to certain autoimmune diseases, including IgAN, also known as Berger's disease, and lupus nephritis. In addition, Vera is evaluating additional diseases where the reduction of autoantibodies by atacicept may prove medically useful. Vera is also developing MAU868, a monoclonal antibody designed to neutralize infection with BK virus (BKV), a polyomavirus that can have devastating consequences in certain settings including kidney transplantation. Vera retains all global developmental and commercial rights to atacicept and MAU868. For more information, please visit www.veratx.com.

Forward-looking Statements

Statements contained in this press release regarding matters, events or results that may occur in the future are "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. Such forward-looking statements include statements regarding, among other things, the ability to execute against strategy for Vera's programs, atacicept's potential to be a best-in-class therapy and a disease-modifying treatment for patients with IgAN, the therapeutic potential of atacicept's dual inhibitor approach to treating the cause of IgAN, Vera's plans to complete enrollment of the pivotal Phase 3 ORIGIN 3 trial, the design and management of Vera's clinical trials, and Vera's product candidates, strategy, and regulatory

matters. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements. Words such as "believe," "potential," "will," and similar expressions are intended to identify forward-looking statements. These forward-looking statements are based upon Vera's current expectations and involve assumptions that may never materialize or may prove to be incorrect. Actual results could differ materially from those anticipated in such forward-looking statements as a result of various risks and uncertainties, which include, without limitation, risks related to the regulatory approval process, results of earlier clinical trials may not be obtained in later clinical trials, preliminary results may not be predictive of topline results, risks and uncertainties associated with Vera's business in general, the impact of macroeconomic and geopolitical events, and the other risks described in Vera's filings with the Securities and Exchange Commission. All forward-looking statements contained in this press release speak only as of the date on which they were made and are based on management's assumptions and estimates as of such date. Vera undertakes no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made, except as required by law.

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Exhibit 99.2

vera therapeutics

R&D Day

January 25, 2024

Forward-Looking Statements

Disclaimer

This material has been made available to you with the consent of Vera Therapeutics, Inc. ("we", "us", "our", or the "Company"). Statements contained in this presentation regarding matters, events or results that may occur in the future are “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995. Such forward-looking statements include statements regarding, among other things, atacicept's potential to be a transformational treatment for patients with IgAN and a best-in-class therapy, the Company's expectations regarding completing the pivotal Phase 3 ORIGIN trial, the design and management of the Company's clinical trials, expectations regarding reporting results from such clinical trials and regulatory matters, including the timing and likelihood of success in obtaining drug approvals. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements. Words such as “could,” “will,” “potential,” “plan,” and similar expressions are intended to identify forward-looking statements. These forward-looking statements are based upon the Company's current expectations and involve assumptions that may never materialize or may prove to be incorrect. Actual results could differ materially from those anticipated in such forward-looking statements as a result of various risks and uncertainties, which include, without limitation, risks related to the regulatory approval process, results of earlier clinical trials may not be obtained in later clinical trials, risks and uncertainties associated with the Company's business in general, the impact of macroeconomic and geopolitical events, including the COVID-19 pandemic, and the other risks described in the Company's filings with the Securities and Exchange Commission. All forward-looking statements contained in this presentation speak only as of the date on which they were made and are based on management's assumptions and estimates as of such date. Accordingly, you should not place undue reliance on forward-looking statements. The Company undertakes no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made, except as required by law.

Certain data in this presentation are based on cross-study comparisons and are not based on any head-to-head clinical trials. Cross-study comparisons are inherently limited and may suggest misleading similarities or differences.

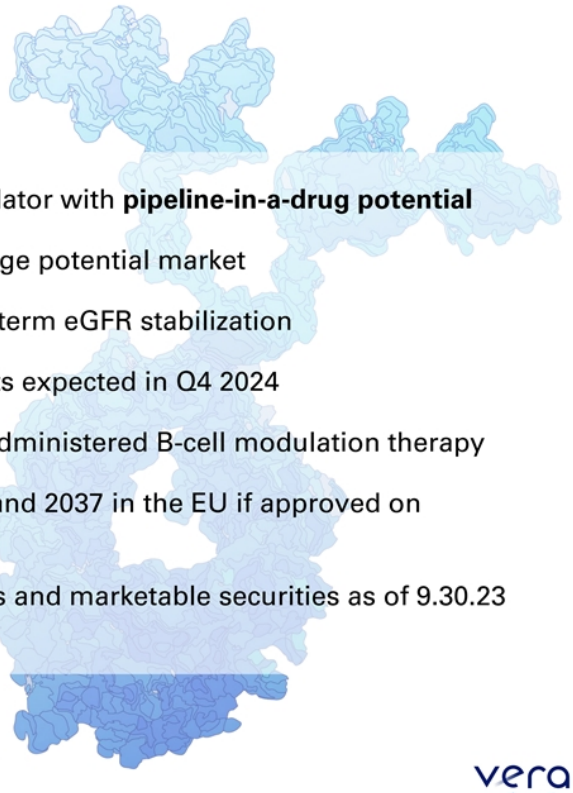
This presentation discusses product candidates that are under clinical study and which have not yet been approved for marketing by the U.S. Food and Drug Administration. No representation is made as to the safety or effectiveness of these product candidates for the use for which such product candidates are being studied.

The trademarks included herein are the property of the owners thereof and are used for reference purposes only. Such use should not be construed as an endorsement of such products.

Agenda

Opening Remarks	**Marshall Fordyce, MD** Founder and CEO, Vera Therapeutics
IgAN Disease State	**Jonathan Barratt, PhD, FRCP** Professor, University of Leicester
Atacicept ORIGIN Phase 2b 72 Week Results	**Richard Lafayette, MD, FACP** Professor, Stanford University
Closing Remarks	**Robert Brenner, MD** Chief Medical Officer, Vera Therapeutics
Q&A	

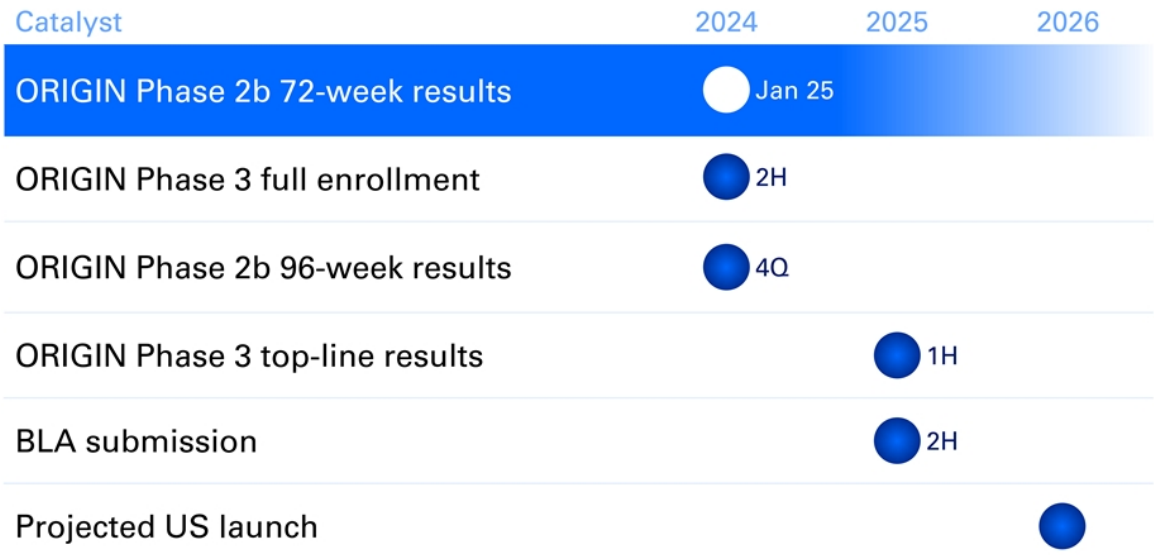
Corporate Highlights



- **Atacicept** is a potential first-in-class dual BAFF/APRIL B cell modulator with **pipeline-in-a-drug potential**
- Currently in Phase 3 pivotal trial for **IgA Nephropathy (IgAN)**, a large potential market
- Differentiation based on **disease-modifying MOA**, evident in long-term eGFR stabilization
- ORIGIN Phase 2b 72-week results **presented today;** 96-week results expected in Q4 2024
- Phase 3 readout expected 1H 2025, potential **first-to-market** self-administered B-cell modulation therapy
- Regulatory data exclusivity expected to extend to 2038 in the US and 2037 in the EU if approved on anticipated timeline
- Strong financial profile, ~$185M[1] pro forma cash, cash equivalents and marketable securities as of 9.30.23 sufficient to **fund IgAN-focused operations to 2026**

1. Unaudited; *pro forma* cash includes ~$160M of cash, cash equivalents and marketable securities as of September 30, 2023 in addition to the $25M drawdown of credit facility in December 2023.
APRIL = A proliferation-inducing ligand; BAFF = B-cell activating factor; eGFR = estimated glomerular filtration rate; MOA = mechanism of action.

Atacicept: Expected Value Creation Catalysts Over Next 18 Months



Vera holds worldwide, exclusive rights to develop and commercialize atacicept

Based on management's current assumptions.

Management Team: Successful Clinical and Commercial Track Record

Marshall Fordyce, MD
President and CEO
- >15 years drug dev leadership
- 7 new drug approvals, Project Lead for tenofovir alafenamide program

GILEAD

Robert Brenner, MD
Chief Medical Officer
- Nephrologist with >25 years biotech leadership supporting multiple drug approvals

AMGEN amag

Lauren Frenz, MBA
Chief Business Officer
- >15 years industry experience, including global commercial planning and multiple blockbuster launches at Gilead
- Strategic advisory at Leerink

GILEAD LEERINK PARTNERS

Sean Grant, MBA
Chief Financial Officer
- >15 years in corporate strategy, finance, and capital raising
- Former healthcare banker with capital raising and M&A experience

CareDx Your Partner in Transplant Care™ citi

William Turner
Chief Development Officer
- ~30 years drug dev and commercialization leadership in multiple therapeutic areas

SIERRA ONCOLOGY aimmune THERAPEUTICS
DYNAVAX

Kelly Rauber
VP, Head of HR
- >18 years in-depth HR experience from multiple industries

CYTOMX ORIC

vera therapeutics

Strong Financial Position

~$185M

Pro forma cash, cash equivalents, and marketable securities including $25M drawdown of credit facility[1]

Current capital position sufficient to fund IgAN-focused operations to

2026

~44.4M

Shares outstanding
(as of 9.30.23)

1. Unaudited; *pro forma* cash includes ~$160M of cash, cash equivalents and marketable securities as of September 30, 2023 in addition to the $25M drawdown of credit facility in December 2023.

Vera Pipeline: Compelling Late-Stage Opportunities For Patient Benefit

Atacicept

Lead Indication: IgAN	Phase 3 Ongoing
IgAN extended dosing	Phase 2/3 potential
LN	Phase 3 ready
SLE	Phase 3 ready
Sjogren's syndrome	Phase 3 potential
Myasthenia gravis	Phase 3 potential
Membranous nephropathy	Phase 3 potential

MAU868

BK virus in transplantation	Phase 2/3 potential

LN = lupus nephritis; SLE = Systemic lupus erythematosus.

MAU868: Novel Investigational Neutralizing Antibody Targeting BK Virus

Phase 2 Trial in Kidney Transplantation: Markedly decreased BK viral load and stable eGFR

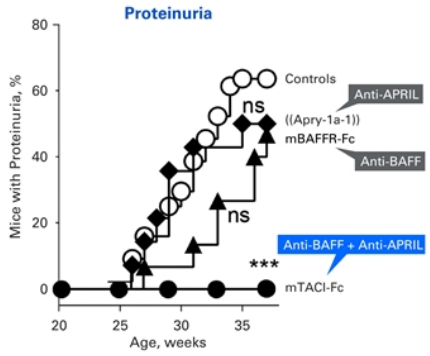
	MAU868 n=20	Placebo n=8	P-value
Log reduction in BK viremia, median (IQR) DNA copies/mL	-1.14 (-1.88, -0.50)	0.37 (-0.72, 0.04)	0.051
Patients with reduction in BK plasma viral load, n (%)			
by ≥1 log	11 (55)	1 (13)	0.040
to <lower limit of quantification	4 (20)	0	0.172
to $<10^4$ DNA copies/mL	15 (75)	3 (38)	0.061
Change in eGFR, median (IQR) mL/min/1.73m^2	2.0 (-5.0, 4.0)	-6.0 (-8.5, -0.5)	0.217

Jordan S, et al. Am J Transplant 2022.

Atacicept Dual Cytokine Inhibition of BAFF and APRIL:

Superior Potential B cell Modulation vs Single Pathway Intervention

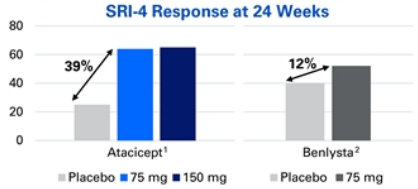
Pre-Clinical Evidence: BAFF-APRIL >> BAFF or APRIL alone



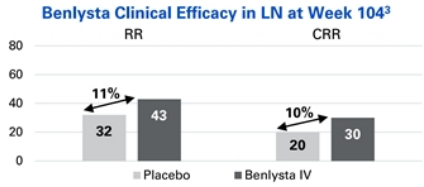
In mouse model of LN, atacicept prevented proteinuria compared to BAFF or APRIL alone

***p<0.001. Haselmayer P, et al. Eur J Immunol 2017.

Clinical Evidence: BAFF-APRIL >> BAFF or APRIL alone



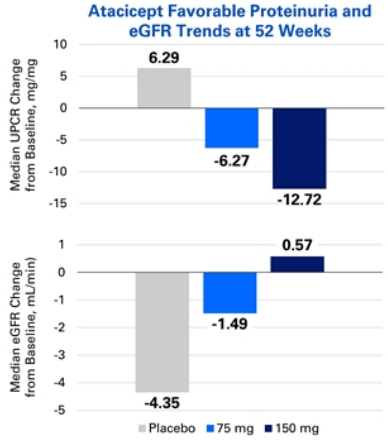
In similar serologically active SLE patients, BAFF/APRIL inhibition may provide better efficacy vs BAFF alone*



BENLYSTA approved in LN, but RR still <50%; we believe there is room for improvement with dual blockade[3]

1. Merrill JT, et al. Arthritis Rheumatol 2018; 2. van Vollenhoven RF, et al. Ann Rheum Dis 2012; 3. Furie R, et al. N Engl J Med 2020.

Clinical Evidence: Improved kidney function in SLE

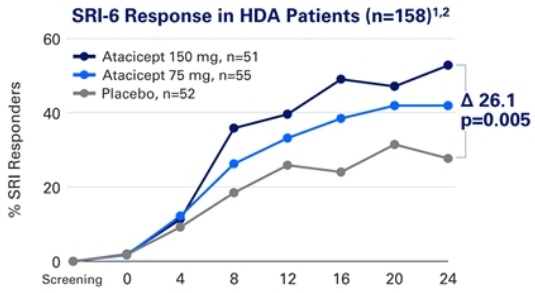


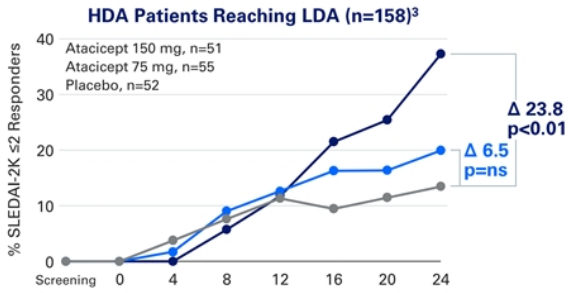
Phase 2 atacicept APRIL-SLE trial showed improved eGFR and proteinuria trends at 1 year in moderate–severe SLE

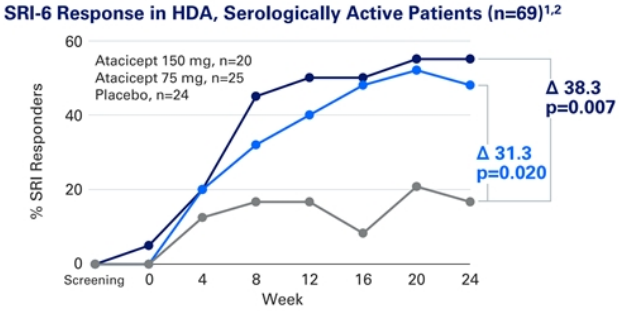
Isenberg D, et al. ERA-EDTA 2022 oral.

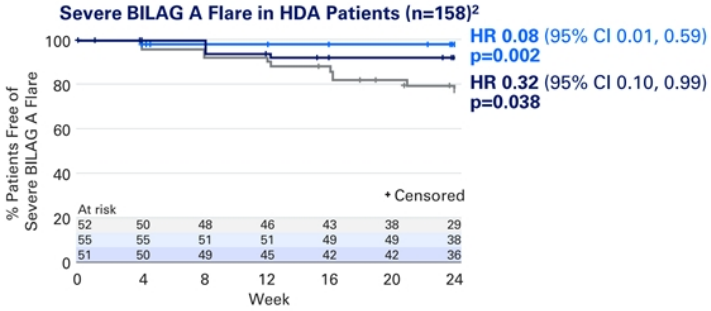
***Based on a cross-trial comparison and not a head-to-head clinical trial, such data may not be directly comparable due to differences in study protocols, conditions and patient populations.**
CRR = complete renal response; RR = renal response; UPCR = urine protein:creatinine ratio.

Atacicept Phase 2 Results in SLE Potentially Best-In-Class Clinical Activity









HDA = High Disease Activity (SLE Disease Activity Index 2000 [SLEDAI-2K] ≥10); LDA = Low Disease Activity (SLEDAI-2K ≤2). 1. SLE responder index 6 (SRI-6) response defined as ≥6-point reduction in Safety of Estrogens in Lupus Erythematosus National Assessment version of SLEDAI score, no new British Isles Lupus Assessment Group (BILAG) A organ domain score or 2 new BILAG B organ domain scores, and no worsening (<0.30-point increase) in Physician's Global Assessment (PGA) score; 2. Merrill JT, et al. Arthritis Rheumatol 2018; 3. Morand EF, et al. Rheumatology 2020.

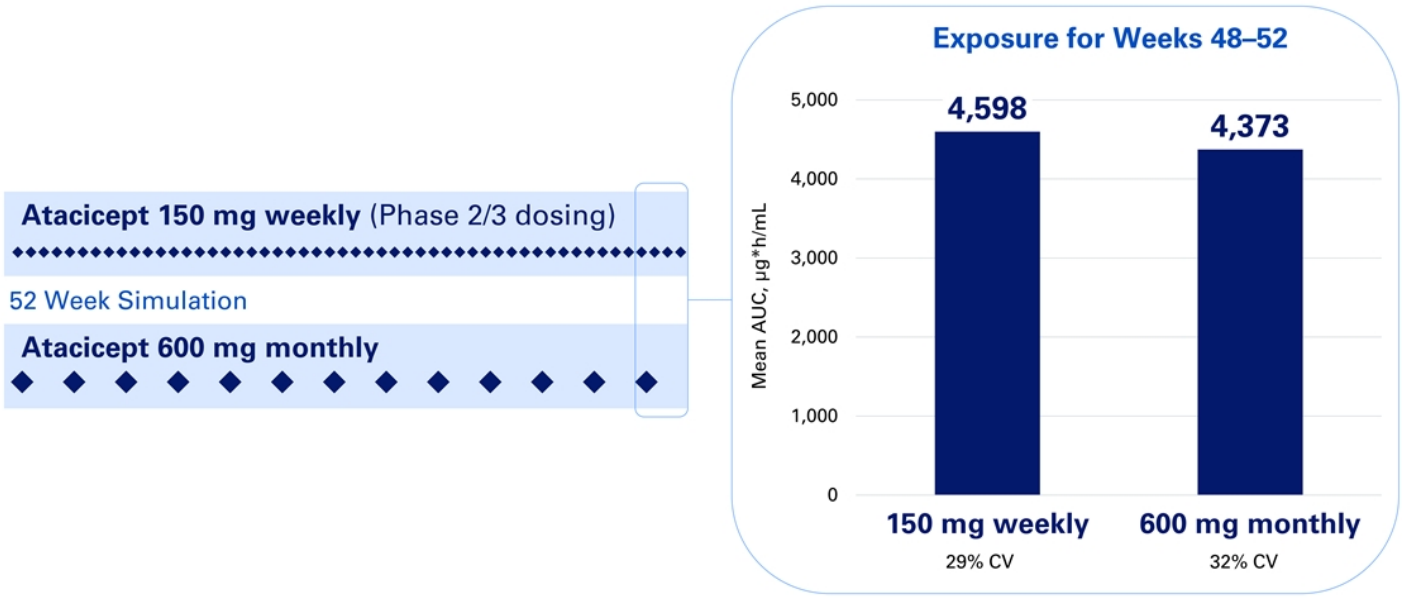
Attractive Target Commercial Atacicept Product Profile

- Self-administration (subcutaneous) of small volume (1 mL) once weekly via autoinjector at commercial launch
- Smallest volume among B-cell modifying drugs in Phase 3 development (2–4 mL with APRIL-only)
- Large subcutaneous injection volumes are associated with pain and may affect tolerability and adherence[1]

1. Usach I, et al. Adv Ther 2019;36:2986-96. Atacicept is investigational and has not been approved by any regulatory authorities for any use.

Atacicept PK/PD Supports Once-Monthly Dosing

Plan to Evaluate as Part of Life Cycle Management



Simulation of N=500 for each dosing scenario. AUC = area under curve; CV = coefficient of variation; PK/PD = pharmacokinetics/pharmacodynamics.

IgAN: High Unmet Need and Significant Commercial Opportunity

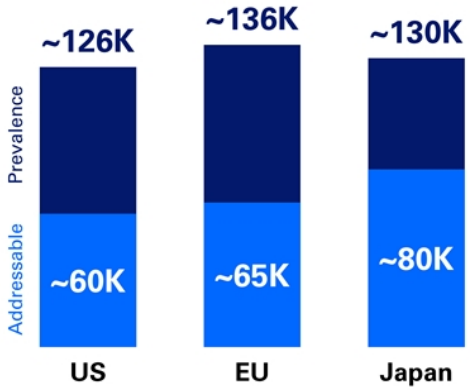
Serious and progressive autoimmune disease of the kidney; average age of diagnosis ~35 years old, severely impacting quality of life[1]

Orphan disease indication in the US and EU[2]

Up to 50% of IgAN patients progress to **ESKD**, resulting in need for **dialysis or transplant**[3,4]

Current SOC includes RASi and supportive care[5]; high unmet need for **disease-modifying therapy that targets the source**[5,6]

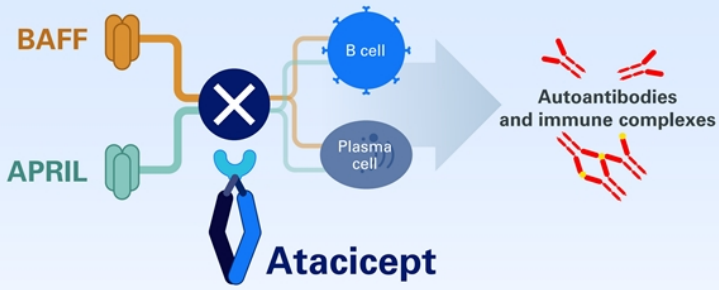
~$6–10B Annual Market Opportunity in US, EU, and Japan for Novel IgAN Therapeutics[7]



ESKD = end-stage kidney disease; RASi = renin-angiotensin system inhibitor; SOC = standard of care.
1. Jarrick S, et al. J Am Soc Nephrol 2019; 2. Orphan Disease Designation not yet obtained for atacicept in IgAN; 3. Kwon CS, et al. J Health Econ Outcomes Res 2021; 4. Pitcher D, et al. Clin J Am Soc Nephrol 2023; 5. Maixnerova D, et al. J Clin Med 2022;11:2810; 6. Huang X, Xu G. Front Pharmacol 2021;12:715253; 7. ClearView Healthcare Partners Analysis. Prevalence and addressable population estimates based on peak year forecast.

Thesis That Drove Vera Acquisition of Atacicept in 2020…

Rationale for Dual Inhibition of BAFF + APRIL with Atacicept

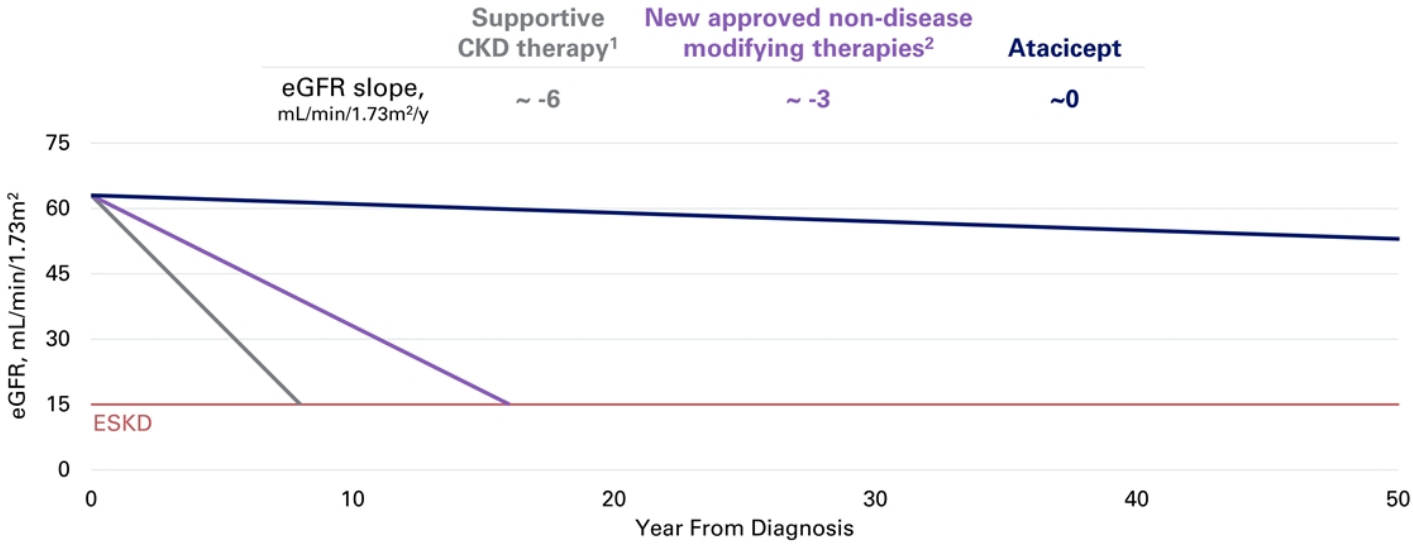


- Elevated BAFF plays **key role in IgAN pathogenesis**
  - BAFF and APRIL levels are both elevated in patients with IgAN and are each associated with clinical severity[1-3]
  - In preclinical models, overexpression of BAFF alone can lead to the development of kidney IgA deposits and IgA-like nephritis[4]
  - BAFF can directly increase the expression of factors associated with fibrosis and inflammation in mesangial cells[2]
  - Dual blockade of BAFF and APRIL decreased renal damage in an immunologic animal model more than individual blockade of either pathway alone[5]
- Dual inhibition offers the potential for **sustained clinical efficacy**
  - BAFF or APRIL alone are each capable of independently supporting plasma cell survival[5,6]
  - Blocking both biologic targets may avoid compensatory increase in parallel signal[7,8]
  - Blocking APRIL alone may lead to upregulation of BAFF signaling with potential consequences on efficacy[9]

1. Xin G, et al. J Nephrol 2013; 2. Cao Y, et al. Mol Med Rep 2020; 3. Zhai Y, et al. Medicine (Baltimore) 2016; 4. McCarthy D, et al. J Clin Invest 2011; 5. Haselmayer P, et al. Eur J Immunol 2017; 6. Benson M, et al. J Immunol 2008; 7. Yeh T, et al. J Allergy Clin Immunol 2020; 8. Vallerskog T, et al. Arthritis Res Ther 2006.

… Included Bold Projections for IgAN Disease Modification

Atacicept Potential to Convert eGFR Rate of Decline to That of the General Population



Projected eGFR trajectories do not represent clinical data and assume a constant eGFR slope over time.
Average slope estimates were applied to mean baseline eGFR of 63 mL/min/1.73m² in the ORIGIN Phase 2b study population and projected to ESKD (eGFR 15 mL/min/1.73m²).

1. Average historical placebo (including standard of care) data from 7 clinical trials[3-11]; 2. Average data from clinical trials of two therapies[3,4,10]; 3. Lafayette R, et al. Lancet 2023; 4. Travere Corporate Overview January 2024; 5. Li PK-T, et al. Am J Kidney Dis 2006; 6. Manno C, et al. Nephrol Dial Transplant 2009; 7. Lv J, et al. JAMA 2017; 8. Wheeler DC, et al. Kidney Int 2021; 9. Lv J, et al. JAMA 2022; 10. Zhang H, et al. ASN Kidney Week 2023, poster TH-PO1123; 11. Mathur M, et al. N Engl J Med 2023.

Agenda

Opening Remarks	**Marshall Fordyce, MD** Founder and CEO, Vera Therapeutics
IgAN Disease State	**Jonathan Barratt, PhD, FRCP** Professor, University of Leicester
Atacicept ORIGIN Phase 2b 72-week Results	**Richard Lafayette, MD, FACP** Professor, Stanford University
Closing Remarks	**Robert Brenner, MD** Chief Medical Officer, Vera Therapeutics
Q&A	

Jonathan Barratt, PhD, FACP

UNIVERSITY OF LEICESTER

Dr. Barratt leads the Renal Research Group within the College of Life Sciences, University of Leicester. His research is focused on a bench-to-bedside approach to improving our understanding of the pathogenesis of IgAN, a common global cause of kidney failure. Dr. Barratt is the IgAN Rare Disease Group lead for the UK National Registry of Rare Kidney Diseases (RaDaR), and a member of the steering committee for the International IgAN Network. He works closely with pharmaceutical companies interested in new treatments for IgAN, is Chief Investigator for a number of international randomized controlled Phase 2 and 3 clinical trials in IgAN, and was a member of the U.S. Food and Drug Administration and American Society of Nephrology Kidney Health Initiative: Identifying Surrogate Endpoints for Clinical Trials in IgAN Workgroup.

UNIVERSITY OF LEICESTER

Leicester IgA Nephropathy Research Group

IgA Nephropathy

Current Challenges and Unmet Needs

Professor Jonathan Barratt
University of Leicester
&
John Walls Renal Unit, Leicester

Agenda

Opening Remarks	**Marshall Fordyce, MD** Founder and CEO, Vera Therapeutics
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Q&A	

Richard Lafayette, MD, FACP

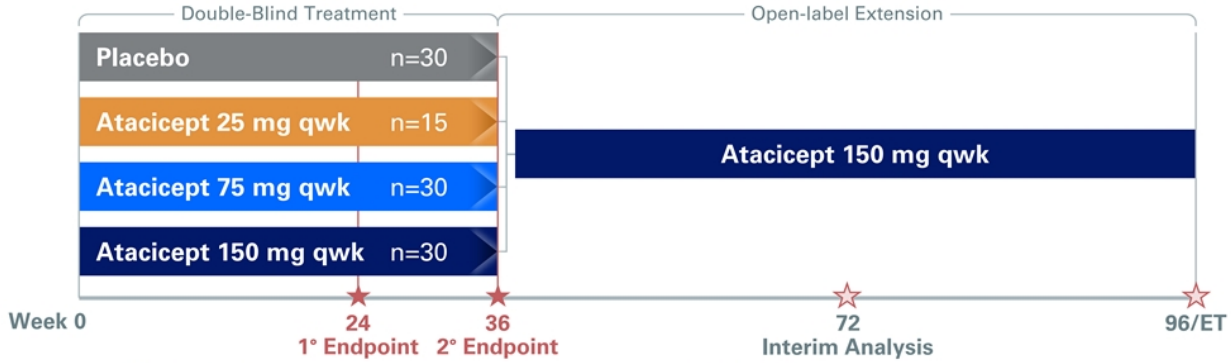
Stanford MEDICINE

Dr. Lafayette is a Professor of Medicine (Nephrology) and Director of the Stanford Glomerular Disease Center at Stanford University Medical Center. Dr. Lafayette completed his medical education at New York Medical College and went on to complete his residency at the Long Island Jewish Medical Center, and his fellowship at Stanford University School of Medicine. Dr. Lafayette is board-certified in Internal Medicine and Nephrology.

Dr. Lafayette served as the Associate Chair of the Stanford University Department of Medicine from 2002–2007, the Clinical Chief of Nephrology at Stanford University from 1999–2012, and currently serves as the Director of the Stanford Glomerular Disease Center since 2010. Dr. Lafayette was honored in America's Top Doctors, Best Doctors from 2004–2018, and received America's Top Doctors Award, Castle Connolly Medical Ltd. from 2014–2022. Dr. Lafayette has been part of the following boards and professional organizations: Editorial Board, Kidney News, American Society of Nephrology (2010–2021) Member, Glomerular Disease Advisory Committee, American Society of Nephrology (2013–2017) Member (ex-officio), Communications Committee, American Society of Nephrology (2015–Present).

ORIGIN Phase 2b IgAN Trial: Study Design and Objectives

Multinational, randomized, placebo-controlled trial



Inclusion Criteria

- Participants ≥18 years old with IgAN on renal biopsy and high risk of disease progression
- Stable and optimized RAASi for ≥12 weeks
- Use of SGLT2i allowed
- UPCR-24h >0.75 g/g or UP >0.75 g per 24h
- eGFR ≥30 mL/min/1.73 m^2
- Blood pressure ≤150/90 mmHg

Endpoints

- Primary efficacy: UPCR-24h at week 24 ★
- Key secondary: UPCR-24h at week 36 ★
- eGFR change up to week 96 ☆
- Gd-IgA1 change
- Safety

ET = end of treatment; Gd-IgA1 = galactose-deficient immunoglobulin A1; RAASi = renin-angiotensin-aldosterone system inhibitor; SGLT2i = sodium-glucose cotransporter-2 inhibitor; UPCR = urine protein:creatinine ratio.

Summary of Positive Phase 2b Week 36 Results

- **Gd-IgA1 reduction of 64% from baseline with atacicept 150 mg**
- **Hematuria resolution in 80% of participants on atacicept 150 mg vs 5% on placebo**
- **Met primary endpoint, with statistically significant UPCR reductions on atacicept 150 mg**

	PP Analysis	ITT Analysis	
	Δ 43%*	Δ 35%*	*p<0.05

- **Stable eGFR observed for participants on atacicept, with clinically meaningful and statistically significant difference vs placebo**

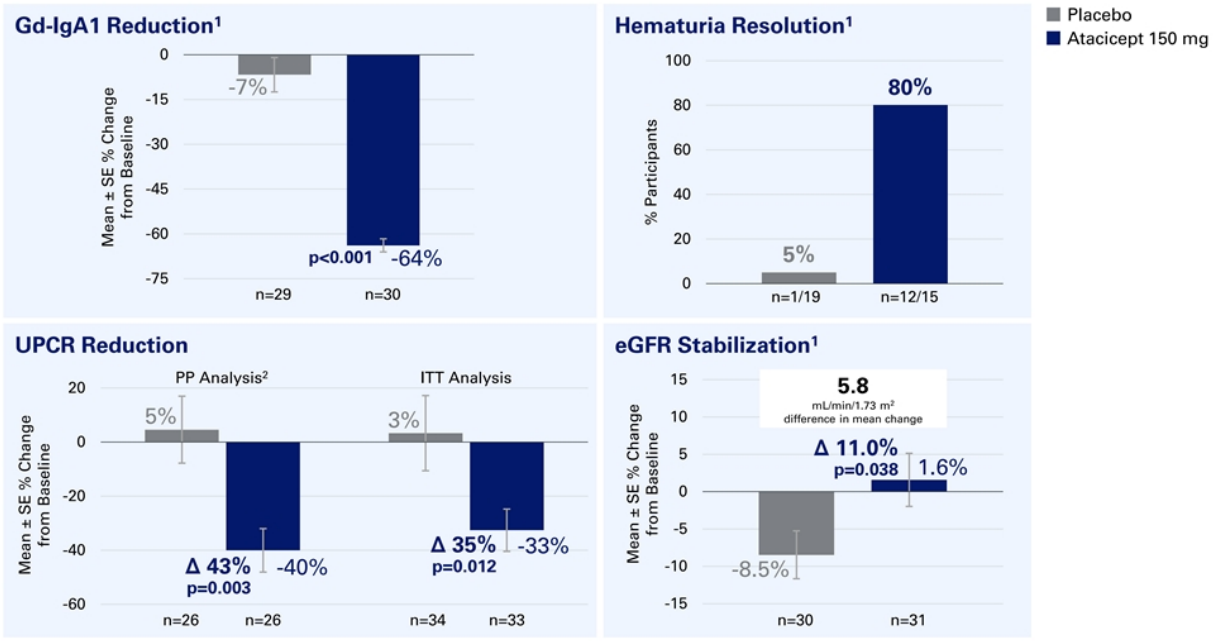
Mean eGFR % change with atacicept 150 mg vs placebo was **11% (p=0.038)**, approximating to an absolute difference of **5.8 mL/min/1.73 m²**

- **Clinical safety profile similar between atacicept and placebo**

Atacicept 150 mg dose selected for Phase 3 clinical trial, initiated in June 2023

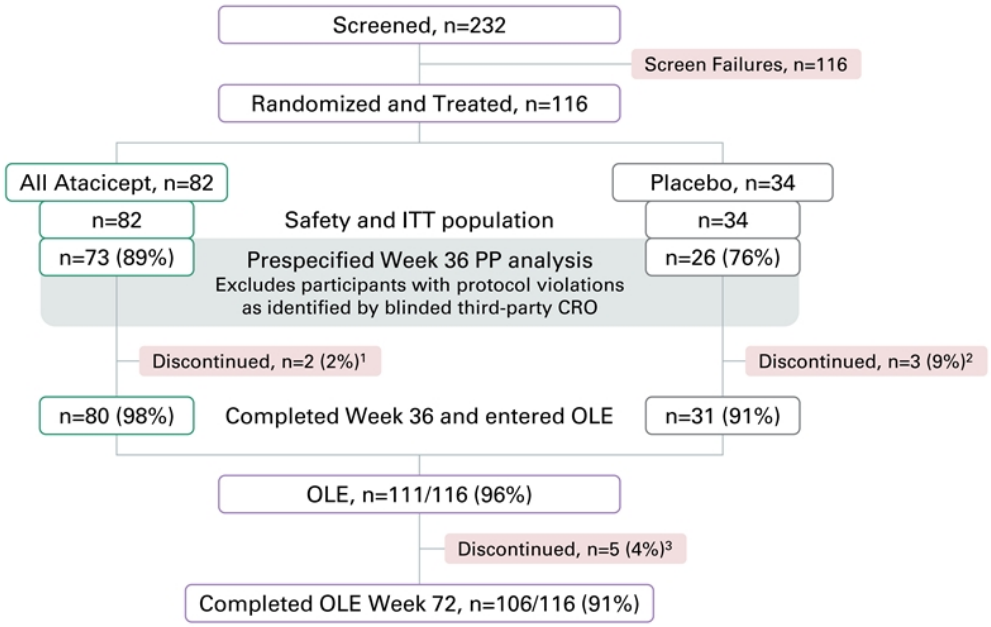
ITT = intent to treat; PP = per-protocol. Hematuria resolution defined as decrease to negative/trace levels in participants with baseline hematuria 1+ or higher.
Lafayette R, et al. ERA 2023 late breaking clinical trial oral presentation, June 17 2023; Barratt J, et al. ASN 2023, SA-PO887.

Disease Modification Observed in Phase 2b Week 36 Results



1. ITT analysis; 2. Prespecified Week 36 PP analysis excluded participants with protocol violations through week 36 as identified by blinded third-party CRO. Hematuria resolution defined as decrease to negative/trace levels in participants with baseline hematuria 1+ or higher. Lafayette R, et al. ERA 2023 late breaking clinical trial oral presentation, June 17 2023; Barratt J, et al. ASN 2023, SA-PO887.

ORIGIN 2b Participant Disposition



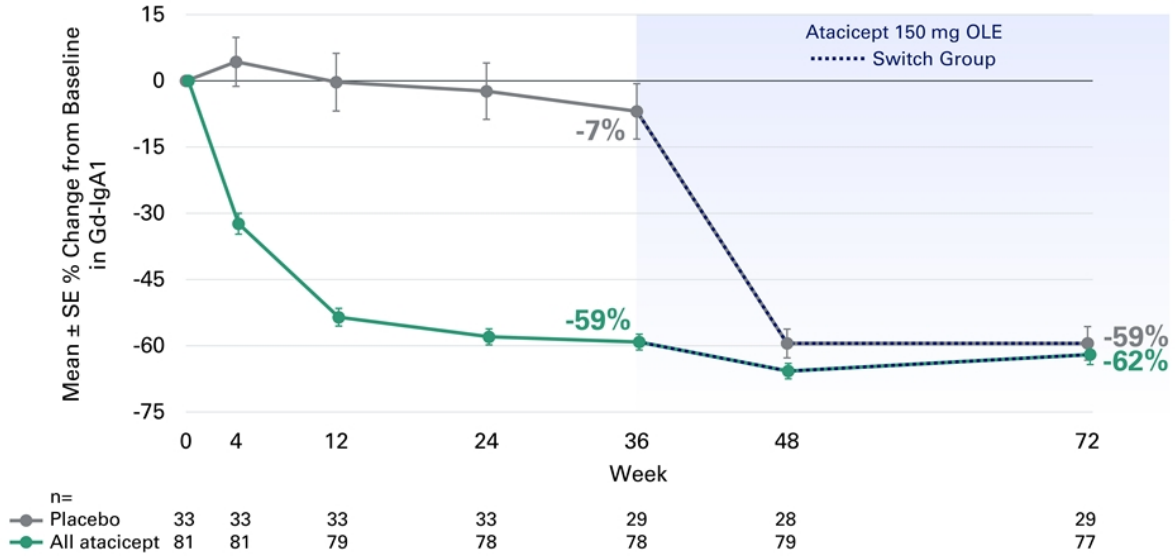
OLE = open label extension.
1. Discontinued to pursue elective surgery (1), discontinued due to positive hepatitis B DNA and adverse event (1).
2. Initiated prohibited medication for concomitant disease (1), discontinued due to plan to start prohibited medication for concomitant disease (1) and adverse event (1).
3. Discontinued to pursue surgery (1), discontinued due to serious adverse event of pneumonia in a heavy smoker, resolved (1), investigator decision (1), pregnancy (1), and participant withdrawal (1).

ORIGIN 2b Demographics and Baseline Characteristics

Mean ± SD or n (%)	Overall n=116	Atacicept 25 mg n=16	Atacicept 75 mg n=33	Atacicept 150 mg n=33	Placebo n=34
Age, y	39	40	41	38	39
Male sex	69 (59)	9 (56)	19 (58)	22 (67)	19 (56)
Race					
White	62 (53)	7 (44)	12 (36)	17 (52)	26 (76)
Asian	51 (44)	7 (44)	20 (61)	16 (48)	8 (24)
Other	3 (3)	2 (12)	1 (3)	0	0
eGFR, mL/min/1.73 m^2	63 ± 27.3	71 ± 28.7	64 ± 25.4	56 ± 22.7	66 ± 31.7
UPCR by 24h urine, g/g	1.6 ± 0.9	1.6 ± 0.8	1.7 ± 0.9	1.7 ± 1.0	1.6 ± 0.8
Time from biopsy, y	2.8 ± 2.8	1.7 ± 1.6	3.4 ± 2.8	3.3 ± 3.4	2.1 ± 2.4

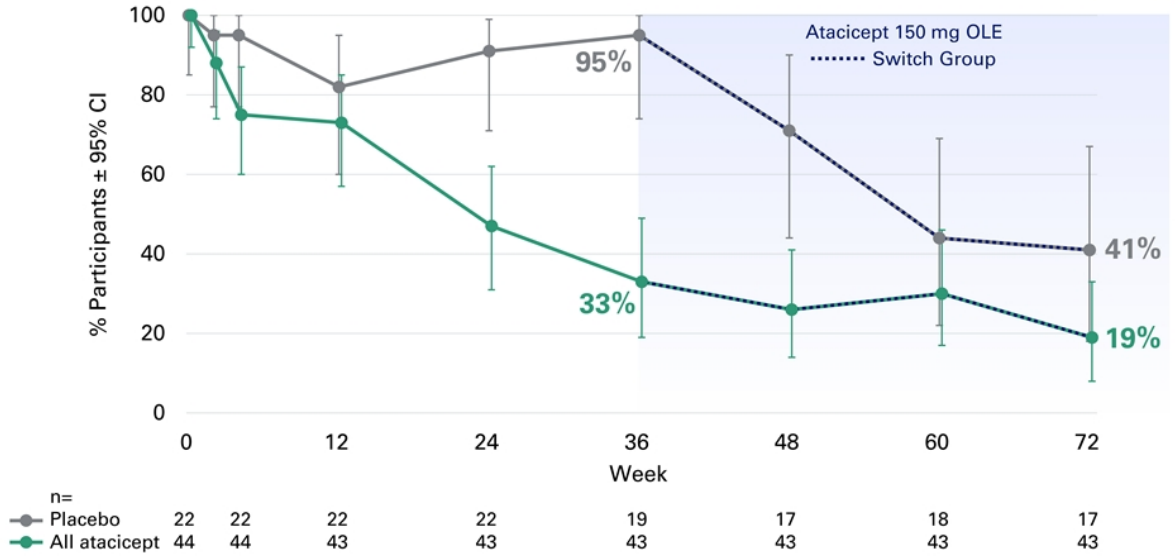
Consistent and Sustained Gd-IgA1 Reduction Through Week 72

Placebo → Atacicept Switch Group Had Similar Reduction as Randomized Atacicept Group



Percentage changes from baseline were computed using FDA-endorsed mixed-effects modeling; all atacicept group includes participants originally randomized to any atacicept group in the double-blind period; ITT analysis.

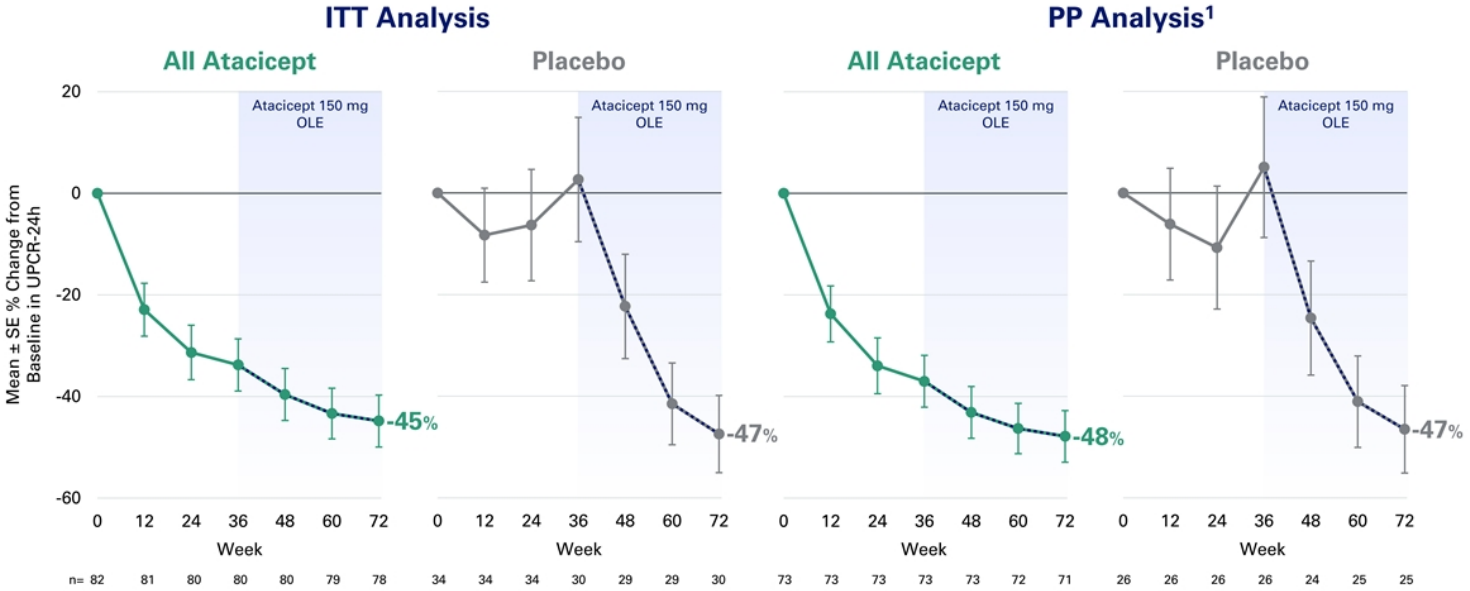
Consistent and Sustained Reductions in Percentage of Participants with Hematuria Through Week 72



n=							
Placebo	22	22	22	22	19	17	18	17
All atacicept	44	44	43	43	43	43	43	43

Percentages represent number of participants with hematuria at each visit out of those with baseline hematuria; microscopic hematuria was evaluated via urine dipstick at a centralized lab, and hematuria levels were graded negative/trace, 1+, 2+, or 3+. All atacicept group includes participants originally randomized to any atacicept group in the double-blind period; ITT analysis. CI = confidence interval.

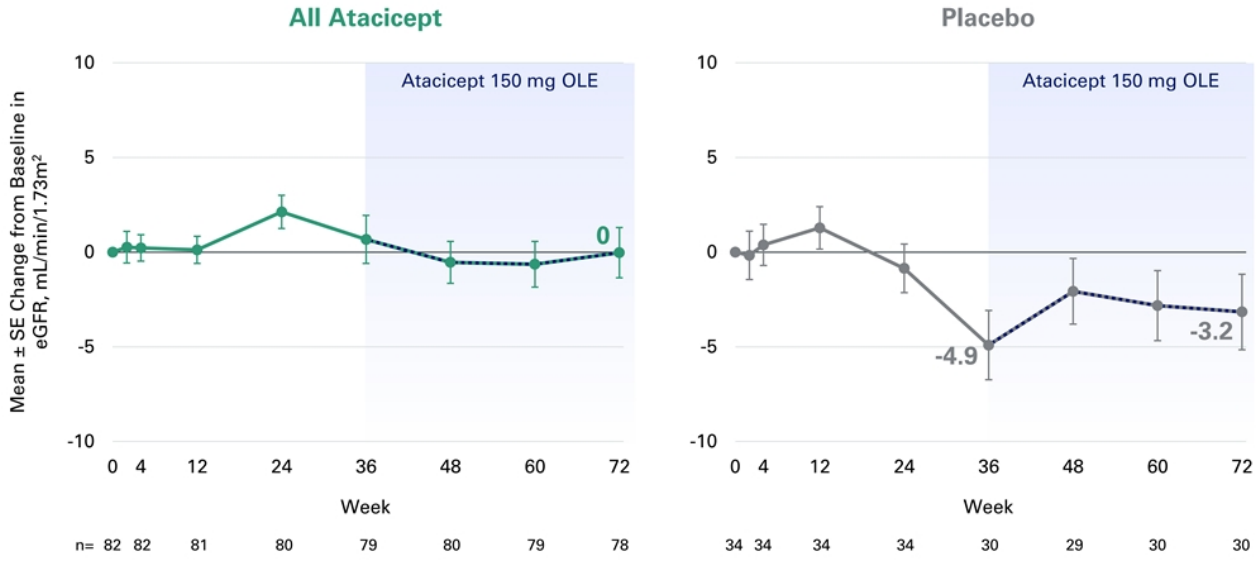
Consistent and Sustained Reductions in UPCR Over 72 Weeks



Percentage changes from baseline were computed using FDA-endorsed mixed-effects modeling which takes into account the effects of baseline UPCR and eGFR and implicitly imputes missing data as missing at random.
All atacicept group includes participants originally randomized to any atacicept group in the double-blind period.
1. Prespecified Week 36 PP analysis excluded participants with protocol violations through week 36 as identified by blinded third-party CRO.

Atacicept Resulted in eGFR Stabilization Through 72 Weeks

Atacicept Switch Halted eGFR Decline in Randomized Placebo Cohort



Percentage changes from baseline were computed using FDA-endorsed mixed-effects modeling which takes into account the effects of baseline UPCR and eGFR and implicitly imputes missing data as missing at random; geometric least squares (LS) means, ratio of geometric LS means, and standard errors (SE), were transformed back into the original scale from model estimates. All atacicept group includes participants originally randomized to any atacicept group in the double-blind period; ITT analysis.

OLE Adverse Events Profile Consistent with Randomized Period

Double-Blind Data Through Week 36; OLE Data Through 12/2023[1]

	Double-blind BL to W36				W36 to W72	BL to W72
Participants, n (%)	Placebo n=34	Atacicept 25 mg n=16	Atacicept 75 mg n=33	Atacicept 150 mg n=33	Total OLE Atacicept 150 mg n=111	Atacicept 150 mg n=33
TEAEs	28 (82)	11 (69)	24 (73)	25 (76)	77 (69)	26 (79)
Infections and infestations	11 (32)	6 (38)	16 (48)	13 (39)	33 (30)	15 (45)
Study drug-related TEAEs[2]	14 (41)	6 (38)	17 (52)	19 (58)	51 (46)	22 (67)
Serious TEAEs	3 (9)	0	1 (3)	1 (3)	8 (7)	2 (6)
TEAEs leading to study drug discontinuation	1 (3)[3]	0	0	1 (3)[4]	1 (1)[5]	1 (3)[4]
Deaths	0	0	0	0	0	0

- Total patient exposure:
  - OLE through 12/05/23: mean 48.8 wk, median 47.7 wk (range 10.7 – 62.7)
  - Double-blind BL to 12/05/23: mean 82.0 wk, median 83.4 wk (range 3.0 – 99.0)

1. W72 cut-off includes all safety data as of 12/05/23, including visits past W72. AEs are considered treatment-emergent during the OLE period if they start after the first dose of open-label atacicept 150 mg through the end of the study.
2. Majority of study drug-related TEAEs were injection site reactions and one contributed to drug discontinuation during double-blind period.
3. Discontinued due to worsening flank pain that was not resolved; unrelated to study treatment.
4. Discontinued due to injection site reaction and positive hepatitis B DNA that resolved without treatment 3 weeks later, normal liver function throughout.
5. Discontinuation due to pneumonia in a heavy smoker, resolved.

Summary of Week 72 Results

- Participants treated with atacicept for 72 weeks demonstrated:
  - Consistent and sustained reductions in Gd-IgA1, hematuria and UPCR
  - Consistent and stable eGFR
  - **In aggregate, these data provide evidence of long-term, comprehensive IgAN disease modification**
- Participants switched from placebo to atacicept demonstrated similar results (Gd-IgA1, hematuria, UPCR, eGFR) to those originally randomized to atacicept during the first 36 weeks of ORIGIN 2b
- The cumulative safety profile is consistent with that observed during the randomized 36 weeks of ORIGIN 2b
- Week 72 data provide additional confidence in the ongoing ORIGIN 3 study

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Q&A	

IgAN Through the Ages

nephritis [*Greek*]: *nephros* "of the kidney" + *-itis* "inflammation"

Hippocratic Aphorism 7.34: earliest description of proteinuria[1]

"When bubbles settle on the surface of the urine, they indicate disease of the kidneys, and that the complaint will be protracted."

~400 BC

Signs/Symptoms of IgA vasculitis in patient in Vienna[2]

1791

1763	Scarlet
1763	Erythema nodosum
Starting 1763	Chronic tooth maturation
1764	Angina tonsilaris
1765	Thyphus abdominalis
Starting 1766	Rheumatic fever
1767	Smallpox
1778	Influenza
Starting 1784	Recurrent renal colic
Starting 1784	Hypertension, epistaxis, cluster headache
Starting 1791	Depression, anancasm
5.12.1791	Death of uraemia

Evidence that IgAN is a B-cell mediated disease[3]

1968

Les dépôts intercapillaires d'IgA - IgG

par MM. J. Berger et N. Hinglais (*)

with comments by
LILIANE STRIKER

Reprinted from J. Urol. Nephrol. (Paris) 74: 694–695, 1968

Sur les biopsies rénales de 25 malades, ont été mis évidence par immunofluorescence des dépôts intercapillaires fixant le sérum anti-IgA et moins intensément les sérums anti-IgG et anti-β_1 C-globuline. En revanche, il n'y avait aucune fixation sur ces dépôts, des sérums anti-IgM, anti-fibrinogène, anti-albumine, anti-coeruléoplasmine, anti-α_2-macroglobuline et anti-β-lipoprotéine. Les dépôts intercapillaires étaient présents dans tous les glomérules.

Discovery of APRIL[4]

1998

APRIL, a New Ligand of the Tumor Necrosis Factor Family, Stimulates Tumor Cell Growth

By Michael Hahne,* Takao Kataoka,* Michael Schröter,* Kay Hofmann,§ Martin Irmler,* Jean-Luc Bodmer,* Pascal Schneider,* Tierry Bornand,* Nils Holler,* Lars E. French,‖ Bernard Sordat,§ Donata Rimoldi,‖ and Jürg Tschopp*

J. Exp. Med. © The Rockefeller University Press
Volume 188, Number 6, September 21, 1998 1185–1190

Discovery of BAFF[5]

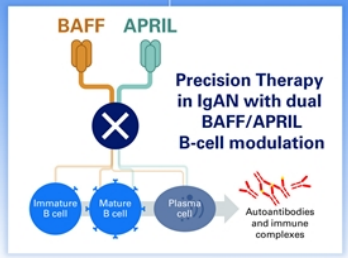
1999

BAFF, a Novel Ligand of the Tumor Necrosis Factor Family, Stimulates B Cell Growth

By Pascal Schneider,* Fabienne MacKay,¶ Véronique Steiner,* Kay Hofmann,‡ Jean-Luc Bodmer,* Nils Holler,* Christine Ambrose,¶ Pornsri Lawton,¶ Sarah Bixler,¶ Hans Acha-Orbea,* Danila Valmori,§ Pedro Romero,§ Christiane Werner-Favre,‖ Rudolph H. Zubler,‖ Jeffrey L. Browning,¶ and Jürg Tschopp*

J. Exp. Med. © The Rockefeller University Press
Volume 189, Number 11, June 7, 1999 1747–1756

Today



1. Diamandopoulos A, et al. Am J Kidney Dis 2009; 2. Hatzinger M, et al. Acta med-hist Adriat 2013; 3. Berger J, Hinglais N. J Am Soc Nephrol 2000; 4. Hahne M, et al. J Exp Med 1998; 5. Schneider P, et al. J Exp Med 1999.

Providing Context:

An Abbreviated Review of the CKD Therapy History and Landscape

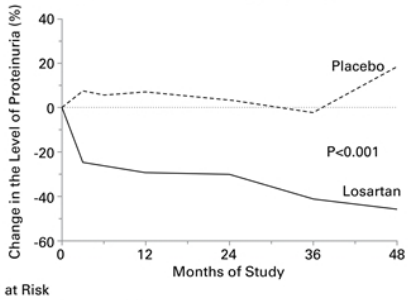
The NEW ENGLAND JOURNAL of MEDICINE

The Effect of Angiotensin-Converting-Enzyme Inhibition on Diabetic Nephropathy

Lewis EJ, et al. N Engl J Med 1993.

The NEW ENGLAND JOURNAL of MEDICINE

Effects of Losartan on Renal and Cardiovascular Outcomes in Patients with Type 2 Diabetes and Nephropathy

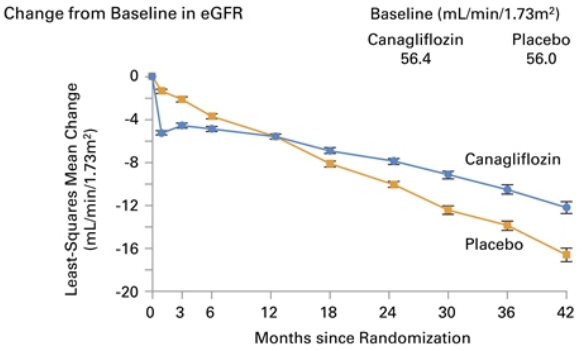


N at Risk					
Placebo	762	632	529	390	130
Losartan	751	661	558	438	167

Adapted from Brenner BM, et al. N Engl J Med 2001.

The NEW ENGLAND JOURNAL of MEDICINE

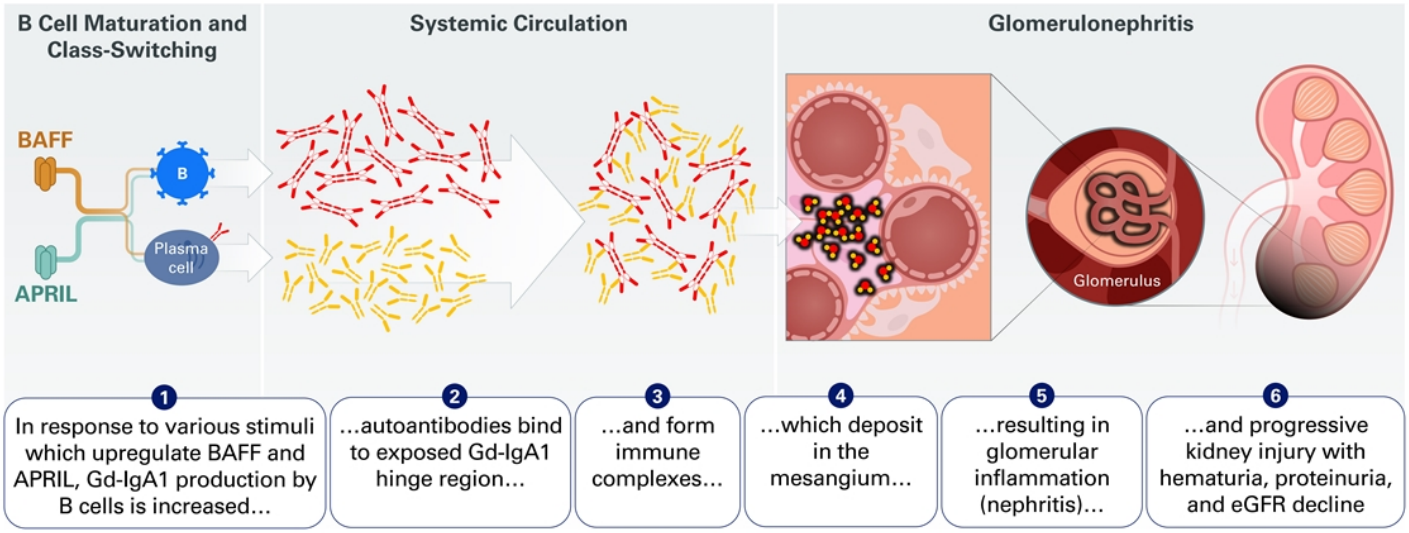
Canagliflozin and Renal Outcomes in Type 2 Diabetes and Nephropathy



No. of patients								
Placebo	2178	1985	1882	1720	1536	1006	583	210
Canagliflozin	2179	2005	1919	1782	1648	1116	652	241

Adapted from Perkovic V, et al. N Engl J Med 2019.

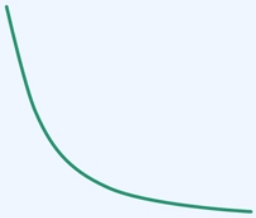
IgAN is a Disease of B Cell Origin With Kidney Pathology



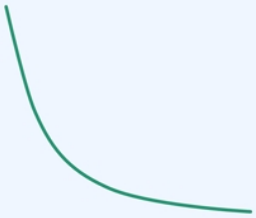
An Ideal IgAN Disease Modifying Therapy Would be Expected To…

1 → 2 → 3 → 4

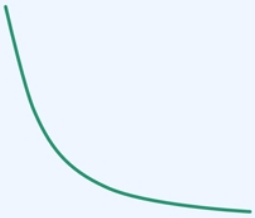
Reduce Gd-IgA1



Reduce hematuria



Reduce proteinuria

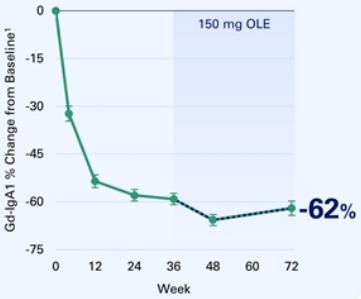


Stabilize eGFR

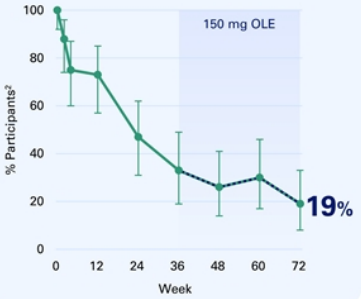
… And the Atacicept ORIGIN Phase 2b 72 Week Results Are Consistent With a Disease Modifying IgAN Profile

1 → 2 → 3 → 4

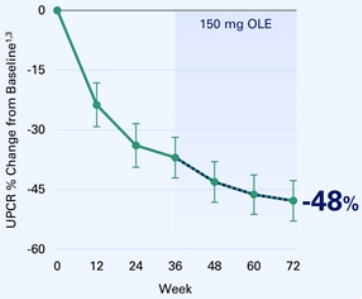
Reduction in Gd-IgA1



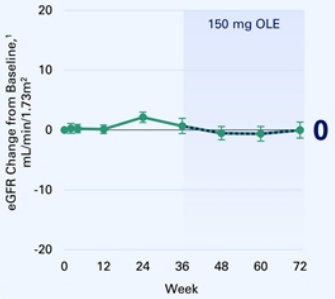
Reduction in patients with hematuria



Reduction in proteinuria

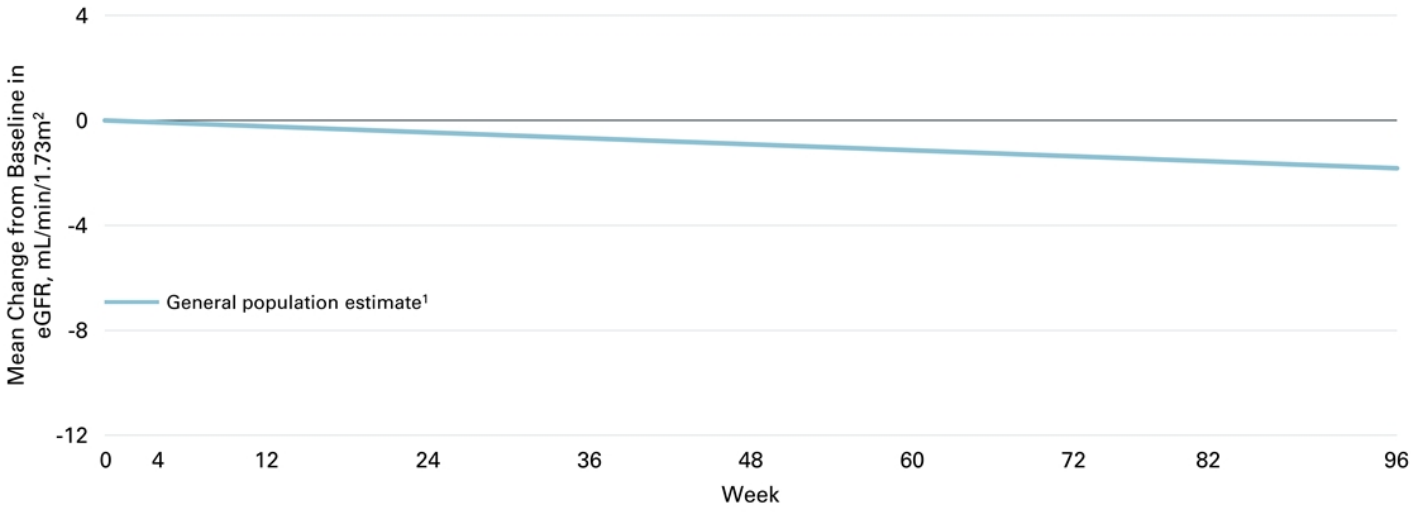


Stabilization of eGFR



1. Mean ± SE; 2. Percentages represent number of participants with hematuria at each visit out of those with baseline hematuria. Data from participants originally randomized to any atacicept group in the double-blind period in the ITT analysis for Gd-IgA1, hematuria, and eGFR, and in week 36 PP analysis for UPCR.

Atacicept Treated Participants Have an eGFR Profile Akin to the *General Population;* Dissimilar to Historical IgAN



This data is based on a cross-trial comparison and not a head-to-head clinical trial; such data may not be directly comparable due to differences in study protocols, conditions and patient populations. Projected eGFR trajectories for general population and IgAN natural history do not represent clinical data and assume a constant eGFR slope over time. Figure cut at week 96 for consistency with Tarpeyo data.

1. Slope estimate from Baba M, et al. PLOS ONE 2015; 2. Average historical placebo slope from 7 clinical trials[3-11]; 3. Lafayette R, et al. Lancet 2023; 4. Travere Corporate Overview January 2024; 5. Li PK-T, et al. Am J Kidney Dis 2006; 6. Manno C, et al. Nephrol Dial Transplant 2009; 7. Lv J, et al. JAMA 2017; 8. Wheeler DC, et al. Kidney Int 2021; 9. Lv J, et al. JAMA 2022; 10. Zhang H, et al. ASN Kidney Week 2023, poster TH-PO1123; 11. Mathur M, et al. N Engl J Med 2023.

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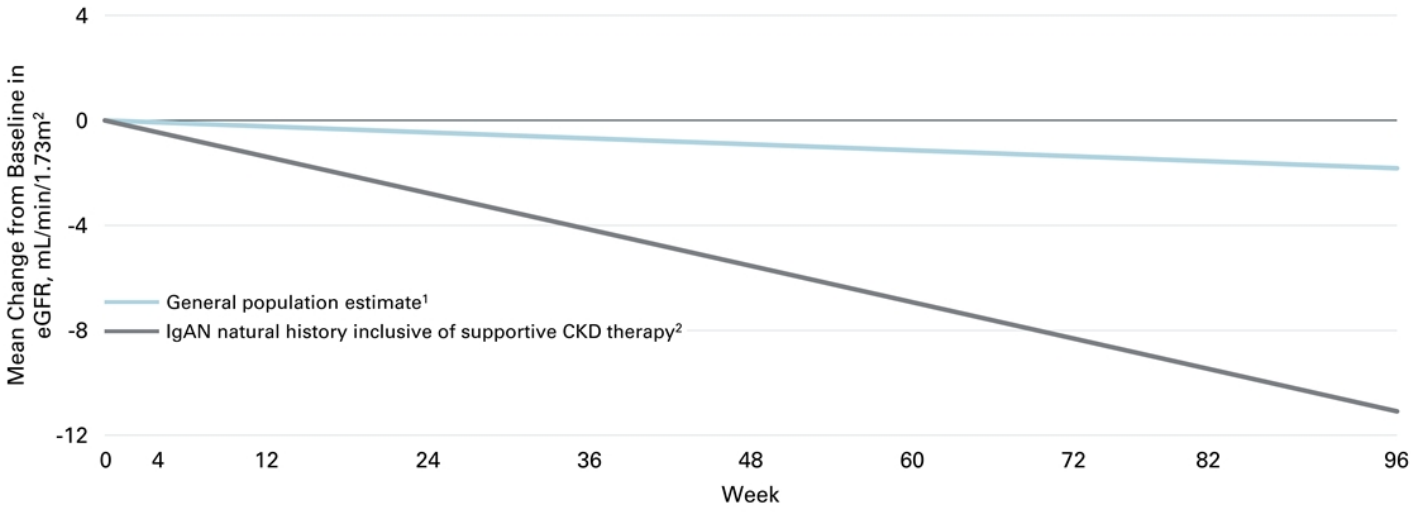

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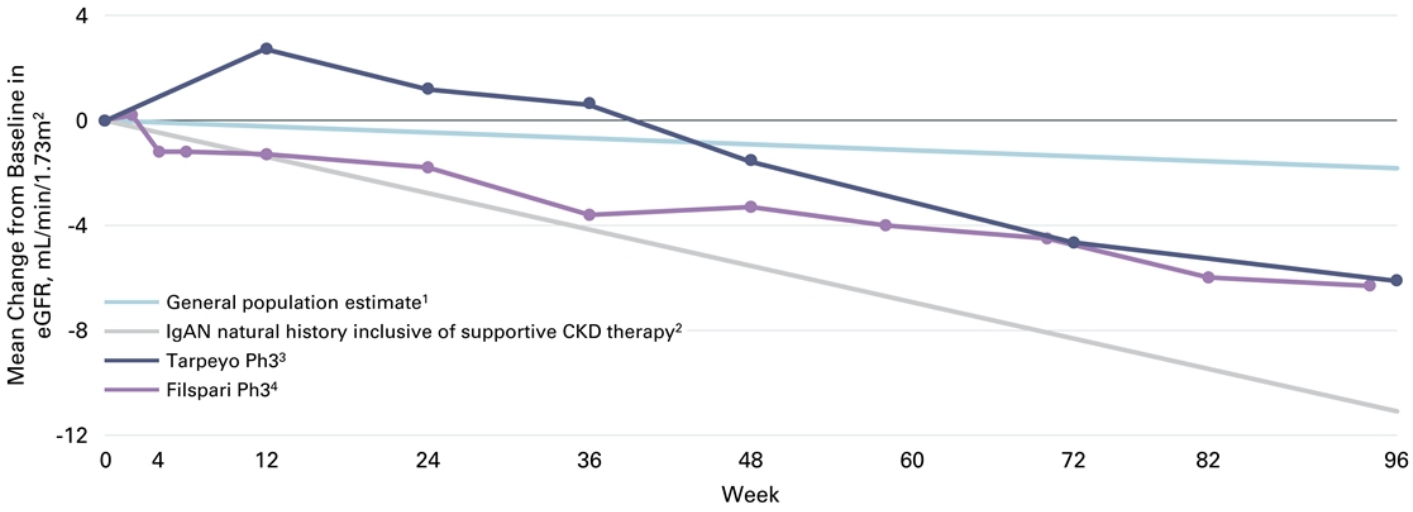

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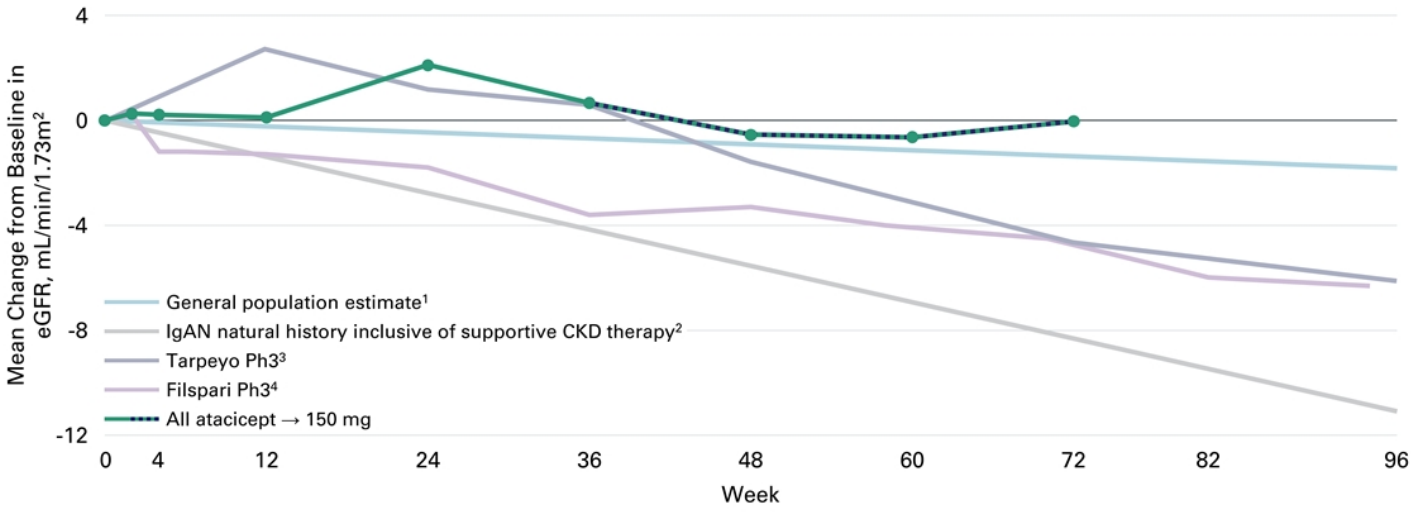

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10. Zhang H, et al. ASN Kidney Week 2023, poster TH-PO1123; 11. Mathur M, et al. N Engl J Med 2023.

Cumulative Atacicept Data Offers Promise For Best-In-Class Potential…

…And Further Supports ORIGIN Phase 3 Design

	Atacicept	Sibeprenlimab[1]	Zigakibart[2]	Telitacicept[3]	Povetacicept[4]
Mechanism	BAFF/APRIL inhibition	APRIL inhibition only	APRIL inhibition only	BAFF/APRIL inhibition	BAFF/APRIL inhibition
Dosing & Administration	25/75/150 mg SC QW (Ph2) 150 mg SC QW (Ph3) 1 x 1 mL self-administered	2/4/8 mg/kg IV (Ph2) 400 mg SC QM (Ph3) 1 x 2 mL in-clinic injection	450mg IV Q2W (Ph2) 600mg SC Q2W (Ph3) 2 x 2 mL in-clinic injection	160/240 mg SC QW (Ph2) 3 x 1 mL injection	80/240 mg SC QM (Ph1b) 1 x TBD mL injection
Development Stage	Ph3	Ph3	Ph3	Ph2 discontinued in US no global development planned	Ph1b
Randomized Controlled Trial Data	✓	✓	✗	✓	✗
N (total pre-Phase 3)	132	155	40	44	20
Gd-IgA1 Reduction	62% at W72	~60% at W52	~70% at W40	50% at W24	~60% at W12
Hematuria	80% resolution at W36	Not reported	Not reported	Not reported	Not reported
UPCR Reduction vs Placebo	Δ 43% (p=0.003) at W36	Δ 43% at W36	No placebo controlled data	No UPCR data (different measure used)	No placebo controlled data
eGFR Duration Data	18 months 24 month pending	12 months	Not reported	6 months	6 months
Projected Commercial Launch	2026	2026	2027	Unknown	Unknown

This data is based on a cross-trial comparison and not a head-to-head clinical trial; such data may not be directly comparable due to differences in study protocols, conditions and patient populations.
Atacicept 150 mg data shown for W36, all-atacicept switch to 150 mg data shown for W72. 1. Mathur M, et al. NEJM 2023, Ph2 4 mg/kg IV Gd-IgA1 data, and Kooienga ASN 2022, TH-PO991, Ph2 pooled sibeprenlimab UPCR data; 2. Barratt J, et al. ERA-EDTA 2023, Ph2 combined cohort data; 3. Lv J, et al. Kidney Int Rep 2023 and Zan J, et al. Kidney Int Rep 2023, Ph2 240 mg data; 4. Tumlin J, et al. ASN 2023, TH-PO1125, Ph1b 80 mg data.

Potential Framework for a Future Treatment Paradigm in IgAN

- In prevalent IgAN patients, initiate disease modifying therapy with dual BAFF/APRIL B cell modulation using atacicept
- In incident IgAN patients with a fresh biopsy, initiate first line disease modifying therapy with dual BAFF/APRIL B cell modulation using atacicept
- Add/continue nonspecific supportive CKD therapy (ACEi, ARB, ERA and SGLT2i) for additional benefit
- With disease modifying therapy, the rationale for steroids and complement inhibitors may not exist

ERA = endothelin receptor antagonist.

Congruency with ORIGIN 2b Instills Greater Confidence in ORIGIN 3; Enrollment On Track

origin3
Initiated in 2023

Inclusion Criteria

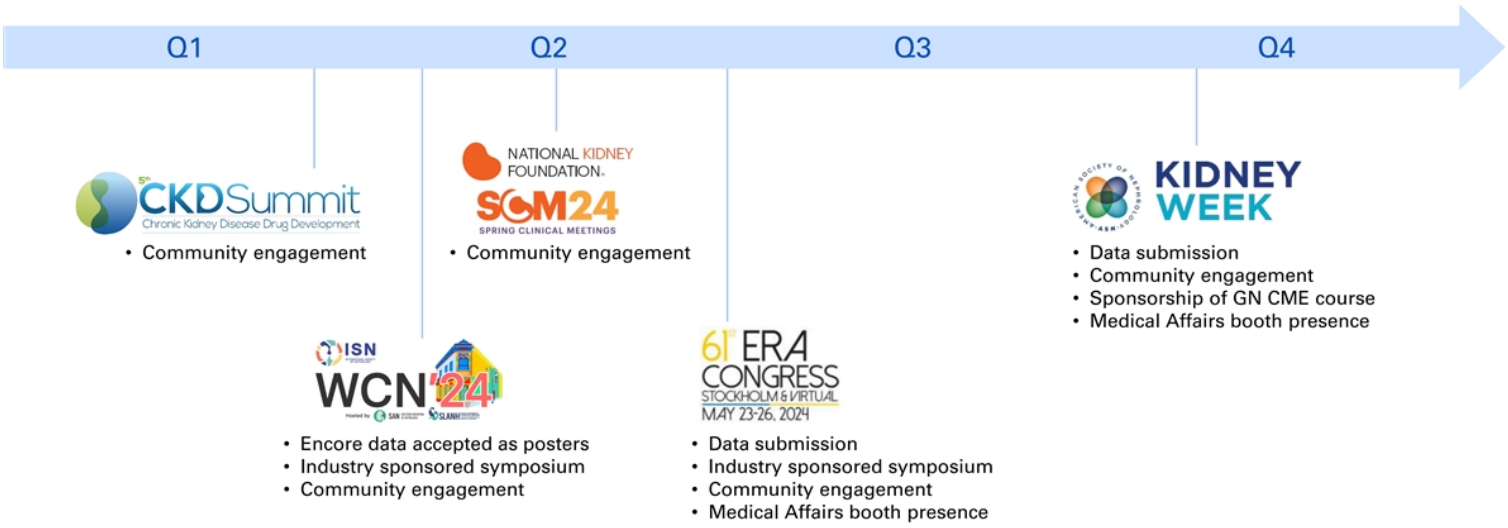
- Patients ≥18 years old with IgAN on kidney biopsy and high risk of disease progression
- Stable and optimized RASi for ≥12 weeks
- Use of SGLT2i allowed
- UPCR-24h ≥1.0 g/g or UP ≥1.0 g per 24h
- eGFR ≥30 mL/min/1.73 m^2
- Blood pressure ≤150/90 mmHg

Endpoints

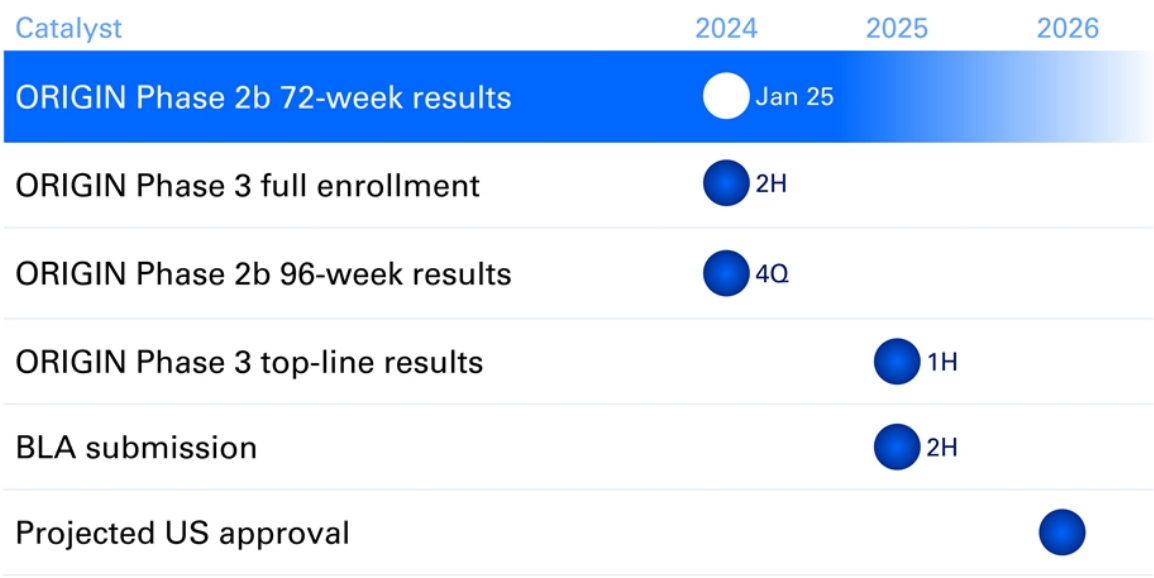
- Primary efficacy: UPCR-24h at week 36 ★ to support potential accelerated approval
  - >90% power at week 36
- Key secondary: eGFR change up to week 104 ☆
  - 90% power for eGFR Δ 4 mL/min at week 104
- Safety

- Operational efficiency leveraging similar study design and worldwide sites as ORIGIN 2b
- Same self-administered SC formulation and dose as used in ORIGIN 2b

2024 Poised To Be An Impactful Year Of Community Engagement



Atacicept: Anticipated Clinical & Regulatory Catalysts Over Next 18 Months



Based on management's current assumptions.

Returning to Vienna in the late 18th Century…

- We have record of a 35-year-old man living in Vienna, with a history of various infections, depression and headache
- Not long before his death, he was noted to have skin rash, gastroenteritis and edema
- He died on December 5, 1791

1763	Scarlet
1763	Erythema nodosum
Starting 1763	Chronic tooth maturation
1764	Angina tonsilaris
1765	Thyphus abdominalis
Starting 1766	Rheumatic fever
1767	Smallpox
1778	Influenza
Starting 1784	Recurrent renal colic
Starting 1784	Hypertension, epistaxis, cluster headache
Starting 1791	Depression, anancasm
5.12.1791	Death of uraemia

Hatzinger M, et al. Acta med-hist Adriat 2013.

In summary, a 35-year-old man died after a fortnight's acute illness characterized by painful and swollen hands and feet at its onset. He was feverish and later developed more generalized swelling, severe weakness, vomiting and diarrhoea. He may have had a rash. He was not dyspnoeic - he could sing - and his consciousness was unclouded until very shortly before death.

For some 2 or 3 months before this illness he had been pale and subject to lapses of consciousness, and had complained of loin pain. For one to two years he had suffered intermittent headaches and depression. He had a history of possible renal colic and, in childhood, typhoid and smallpox. He may have had atypical rheumatic fever and perhaps hepatitis.

Wheater M. J Royal Society Med 1990.

Wolfgang Amadeus Mozart

Perhaps the First Described Individual to Succumb to an IgA Mediated Death

- Mozart displayed signs and symptoms of IgA Vasculitis, and it is believed he ultimately died due to kidney failure
- He remained engaged and composing, working on the Lacrymosa of the *Requiem,* within hours of his ultimate demise
- Imagine if Mozart lived in an era during which B cell modulation of human disease was possible, potentially resulting in 50 more years of his genius composition for the world to enjoy
- More importantly, we recognize that for all current and future Mozarts, the possibility of bringing forward a true disease modifying therapy for IgAN and other B cell mediated diseases is our great collective opportunity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