Biogen

June 11, 2025

BIOGEN THEMATIC PIPELINE SEMINAR

RARE KIDNEY DISEASE

FORWARD-LOOKING STATEMENTS

This presentation and the discussions during this conference call contains forward-looking statements, relating to: our strategy and plans; potential of, and expectations for, our commercial business and pipeline programs; capital allocation and investment strategy; clinical development programs, clinical trials, and data readouts and presentations; regulatory discussions, submissions, filings, and approvals; the potential benefits, safety, and efficacy of our and our collaboration partners' products and investigational therapies; the anticipated benefits and potential of investments, optimization of the cost structure including our "Fit for Growth" program, actions to improve risk profile and productivity of R&D pipeline, collaborations, and business development activities; intellectual property; litigation and disputes; our future financial and operating results; 2025 financial guidance. These forward-looking statements may be accompanied by such words as "aim," "anticipate," "assume," "believe," "contemplate," "continue," "could," "estimate," "expect," "forecast," "goal," "guidance," "hope," "intend," "may," "objective," "outlook," "plan," "possible," "potential," "predict," "project," "prospect," "should," "target," "will," "would," and other words and terms of similar meaning. Drug development and commercialization involve a high degree of risk, and only a small number of research and development programs result in commercialization of a product. Results in early-stage clinical trials may not be indicative of full results or results from later stage or larger scale clinical trials and do not ensure regulatory approval. You should not place undue reliance on these statements.

Given their forward-looking nature, these statements involve substantial risks and uncertainties that may be based on inaccurate assumptions and could cause actual results to differ materially from those reflected in such statements. This presentation and the discussions during this conference call includes, among others, forward-looking statements including: that Biogen is building on a new foundation with the goal of long-term sustainable growth in its commercial portfolio; the multi-billion dollar potential of its late-stage pipeline; that we believe there remains a significant long-term opportunity for our ongoing product launches including LEQEMBI; that we believe that continued execution against these key strategic elements, as well as a disciplined approach to business development, will allow us to generate long-term value for our shareholders by bringing innovative medicines to patients; and all statements and information under the heading "Full Year 2025 Financial Guidance". These forward-looking statements are based on management's current beliefs and assumptions and on information currently available to management. Given their nature, we cannot assure that any outcome expressed in these forward-looking statements will be realized in whole or in part.

We caution that these statements are subject to risks and uncertainties, many of which are outside of our control and could cause future events or results to be materially different from those stated or implied in this document, including, among others, factors relating to: our substantial dependence on revenue from our products and other payments under licensing, collaboration, acquisition or divestiture agreements; uncertainty of long-term success in developing, licensing, or acquiring other product candidates or additional indications for existing products; expectations, plans and prospects relating to product approvals, approvals of additional indications for our existing products, sales, pricing, growth, reimbursement and launch of our marketed and pipeline products; the potential impact of increased product competition in the biopharmaceutical and healthcare industry, as well as any other markets in which we compete, including increased competition from new originator therapies, generics, prodrugs and biosimilars of existing products and products approved under abbreviated regulatory pathways; our ability to effectively implement our corporate strategy; the successful execution of our strategic and growth initiatives, including acquisitions; the drivers for growing our business; difficulties in obtaining and maintaining adequate coverage, pricing, and reimbursement for our products; the drivers for growing our business, including our dependence on collaborators and other third parties for the development, regulatory approval, and commercialization of products and other aspects of our business, which are outside of our full control; risks associated with current and potential future healthcare reforms; risks related to commercialization of biosimilars, which is subject to such risks related to our reliance on third-parties, intellectual property, competitive and market challenges and regulatory compliance; failure to obtain, protect, and enforce our data, intellectual property, and other proprietary rights and the risks and uncertainties relating to intellectual property claims and challenges; the risk that positive results in a clinical trial may not be replicated in subsequent or confirmatory trials or success in early stage clinical trials may not be predictive of results in later stage or large scale clinical trials or trials in other potential indications; risks associated with clinical trials, including our ability to adequately manage clinical activities, unexpected concerns that may arise from additional data or analysis obtained during clinical trials, regulatory authorities may require additional information or further studies, or may fail to approve or may delay approval of our drug candidates; the occurrence of adverse safety events, restrictions on use with our products, or product liability claims; risks relating to technology, including our incorporation of new technologies such as artificial intelligence into some of our processes; risks related to use of information technology systems and potential impacts of any breakdowns, interruptions, invasions, corruptions, data breaches, destructions and/or other cybersecurity incidents of our systems or those of connected and/or third-party systems; problems with our manufacturing capacity, including our ability to manufacture products efficiently or adequately address global bulk supply risks; risks relating to management, personnel and other organizational changes, including our ability to attracting, retaining and motivating qualified individuals; risks related to the failure to comply with current and new legal and regulatory requirements, including judicial decisions, accounting standards, and tariff or trade restrictions; the risks of doing business internationally, including geopolitical tensions, acts of war and large-scale crises; risks relating to investment in our manufacturing capacity; risks relating to the distribution and sale by third parties of counterfeit or unfit versions of our products; risks relating to the use of social media for our business, results of operations and financial condition; fluctuations in our operating results; risks related to investment in properties; risks relating to access to capital and credit markets to finance our present and future operations and business initiatives and obtain funding for such activities on favorable terms; risks related to indebtedness; the market, interest, and credit risks associated with our investment portfolio; risks relating to share repurchase programs; change in control provisions in certain of our collaboration agreements; fluctuations in our effective tax rate and obligations in various jurisdictions in which we are subject to taxation; environmental risks; and any other risks and uncertainties that are described in other reports we have filed with the U.S. Securities and Exchange Commission.

These statements speak only as of the date of this presentation and the discussions during this conference call and are based on information and estimates available to us at this time. Should known or unknown risks or uncertainties materialize or should underlying assumptions prove inaccurate, actual results could vary materially from past results and those anticipated, estimated or projected. Investors are cautioned not to put undue reliance on forward-looking statements. A further list and description of risks, uncertainties and other matters can be found in our Annual Report on Form 10-K for the fiscal year ended December 31, 2024 and in our subsequent reports on Form 10-Q and Form 10-K, in each case including in the sections thereof captioned "Note Regarding Forward-Looking Statements" and "Item 1A. Risk Factors," and in our subsequent reports on Form 8-K. Except as required by law, we do not undertake any obligation to publicly update any forward-looking statements whether as a result of any new information, future events, changed circumstances or otherwise.

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CALL PARTICIPANTS

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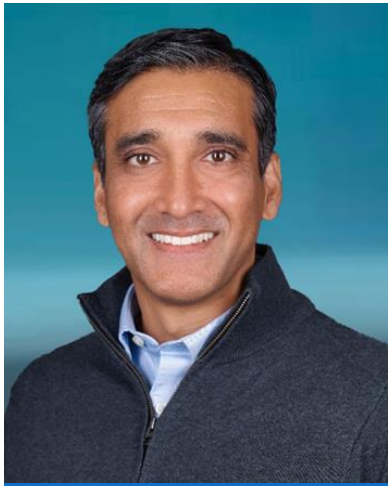
Travis Murdoch, M.D

Head of Biogen
West Coast Hub



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Biogen West Coast Hub

Priya Singhal, M.D., M.P.H.

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OPENING REMARKS

Christopher A. Viehbacher

President and
Chief Executive Officer

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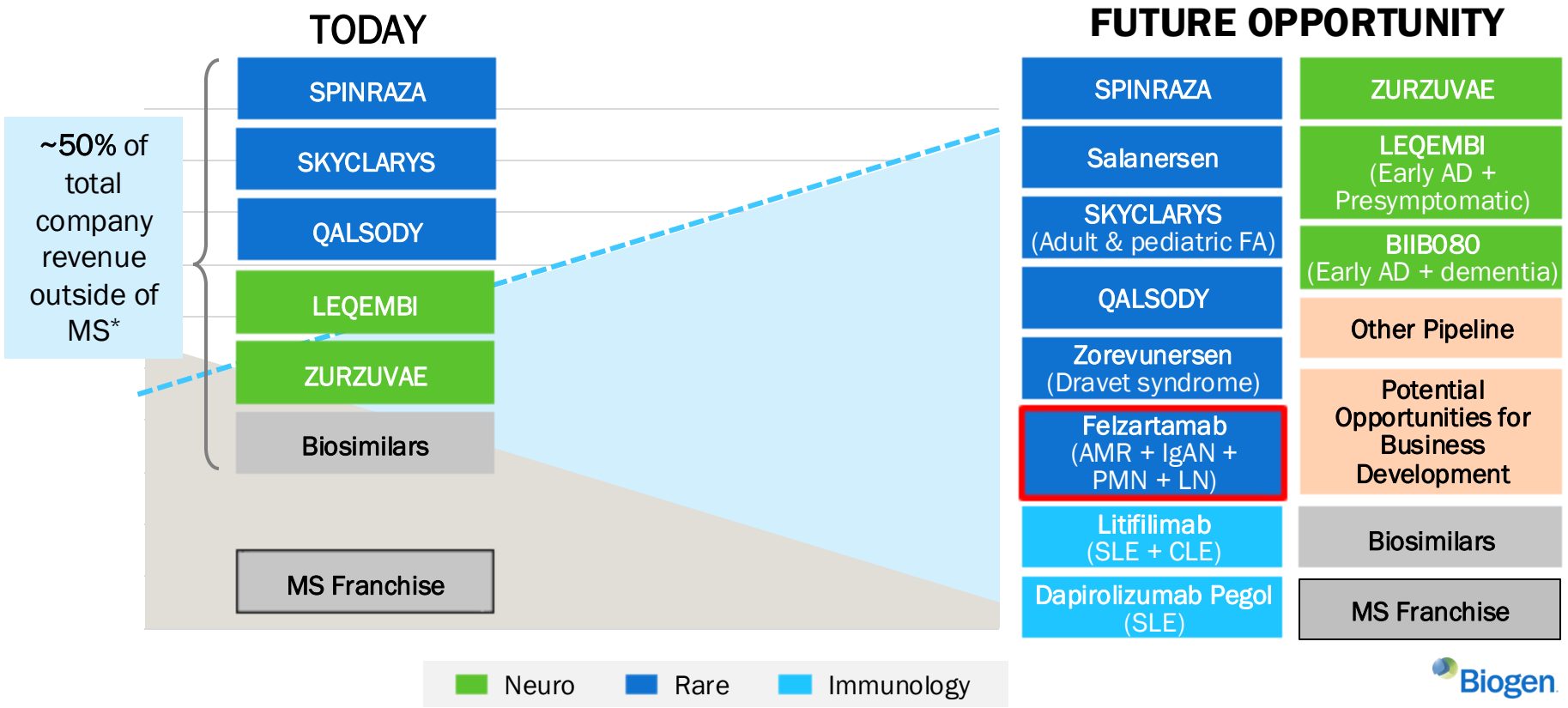
WE ARE ON A JOURNEY TO BUILD THE NEW BIOGEN

Broadening our portfolio across Neuro, Immunology & Rare Disease



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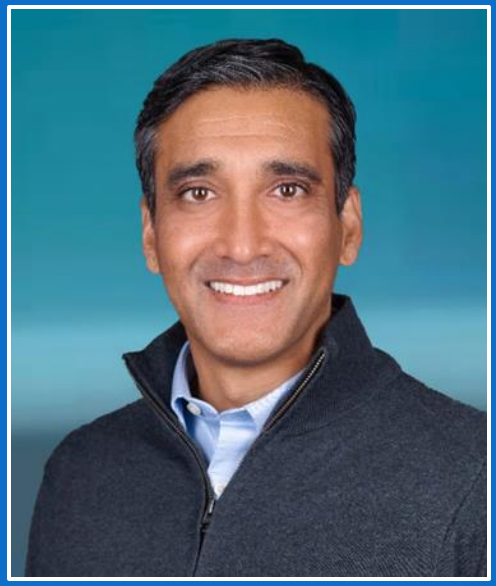
*MS product revenue plus royalty revenue on sales of OCREVUS in Q1 2025; 2. Consists of current products and potential products

FELZARTAMAB

Travis Murdoch, M.D.

Head of Biogen West Coast Hub / Founder and former CEO of HI-Bio



Uptal Patel, M.D.

Head of Development, Biogen West Coast Hub / Former Chief Medical Officer of HI-Bio

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BUILDING OUR NEPHROLOGY EXPERTISE FROM INTEGRATING HI-BIO AS OUR NEW BIOGEN WEST COAST HUB

- HI-Bio – a leading precision immunology company that developed novel targeted therapies focusing on the pathogenic drivers of immune disease
- Biogen West Coast Hub includes ~45 HI-Bio colleagues with deep expertise in nephrology and rare immunology
- We have retained >90% of the team since last year's acquisition, and have grown the team to >100 employees

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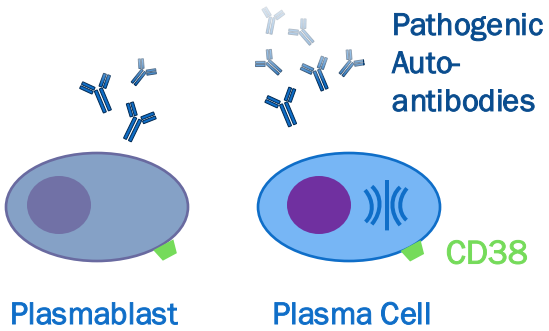
ANTIBODY PRODUCING B CELLS THAT EXPRESS CD38 ARE IMPLICATED IN MULTIPLE AUTOIMMUNE DISEASES

Antibody-Mediated Diseases: Diseases caused by antibodies produced by the immune system attacking the body's own tissues. These antibodies are produced mainly by CD38-expressing plasmablasts and plasma cells.

Plasmablasts and Plasmacells are the key antibody producing B-cells, and both express CD38

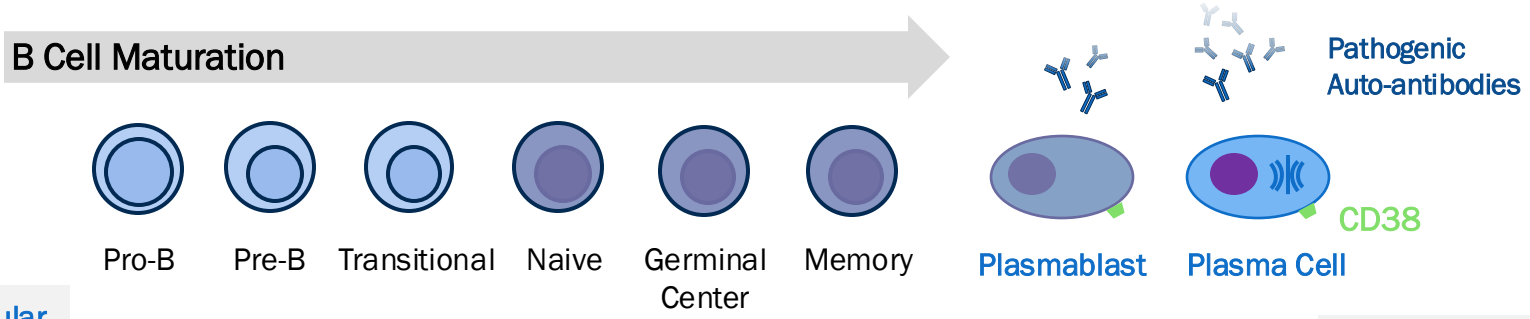


Nephrology	Neurology	Hematology/ Dermatology	Rheumatology
Membranous Nephropathy	Autoimmune Encephalitis	Antiphospholipid Syndrome	SLE
Antibody Mediated Rejection	Myasthenia Gravis	AIHA	Myositis
IgA Nephropathy	CIDP	ITP	Systemic Sclerosis
Lupus Nephritis	NMOSD	Bullous Pemphigoid	Sjogren's Syndrome
ANCA Associated Vasculitis	Guillain-Barre Syndrome	Pemphigus Vulgaris	Rheumatoid Arthritis

Non-exhaustive list

AIHA = Autoimmune hemolytic anemia; ANCA = Anti-Neutrophil Cytoplasmic Antibody; CIDP = chronic inflammatory demyelinating polyneuropathy; ITP = Immune thrombocytopenic purpura; NMOSD = Neuromyelitis Optica Spectrum Disorder; SLE = systemic lupus erythematosus

TARGETING CD38 REPRESENTS AN ATTRACTIVE APPROACH FOR TREATING ANTIBODY-MEDIATED DISEASES



Molecular Target	Pro-B	Pre-B	Transitional	Naive	Germinal Center	Memory	Plasmablast	Plasma Cell	Key Points
CD38							✓	✓	*Directly depletes plasmablasts and long-lived plasma cells*
APRIL/ BAFF			●	●	●	●	●	●	Inhibits plasmablasts and plasma cells
CD20		●	●	●	●	●			Depletes only precursor B cells but not plasmablasts or plasma cells
CD19	●	●	●	●	●	●	●		

APRIL = A proliferation-inducing ligand; BAFF = B-cell activating factor

FELZARTAMAB'S NOVEL BINDING TO CD38 MAY PROVIDE A DIFFERENTIATED PROFILE

Felzartamab is hypothesized to have a safety profile that is more appropriate for long term use in immunologic indications

1

Low potential for undesirable effects of targeting CD38

- *Depletes CD38 expressing cells in a manner that does not involve complement-mediated cytotoxicity allowing for faster infusions and potential lower instance of infusion-related reactions*
- *Binding to CD38 in a manner that does not inhibit CD38 ectoenzyme activity*

2

Differentiated long-term preclinical safety established

- *Felzartamab is a CD38 with a complete toxicology package in non-human primates; results show that felzartamab was well tolerated*

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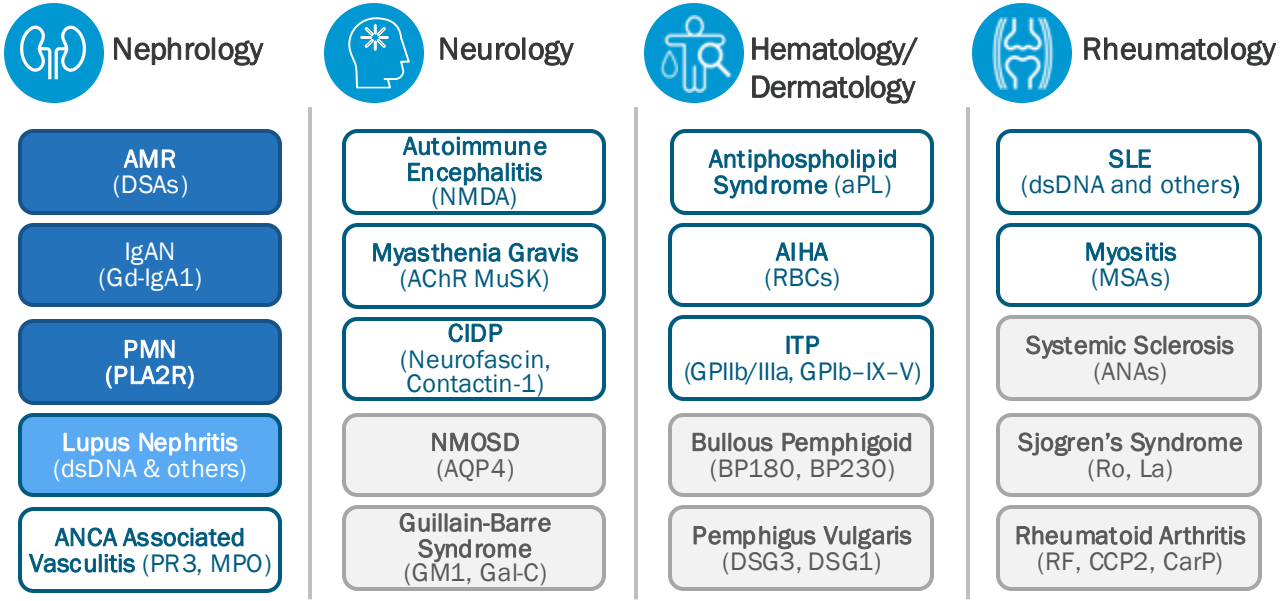
WE ARE TARGETING A SELECT SET OF INDICATIONS THAT COULD BE ADDRESSED BY AN ANTI-CD38 APPROACH



- Select nephrology indications with significant unmet need
- Attractive commercial potential
- Opportunity to provide first and/or differentiated treatment

Felzartamab Phase 3 program

Felzartamab early-stage development

Clinical experience with anti-CD38*

Disease Name (pathogenic antibody)

*off-label use of Darzalex, Sarclisa

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AMR

ANTIBODY MEDIATED REJECTION IN KIDNEY TRANSPLANT

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LATE AMR IS A LEADING CAUSE OF KIDNEY TRANSPLANT LOSS

AMR is mediated by CD38-expressing plasma cells and plasmablasts production of donor-specific antibody and CD38-expressing natural killer cell-mediated damage

Pathology	Diagnosis	Current SoC	Patient Impact
Microvascular inflammation (MVI) and persistent donor-specific antibodies	Diagnosis is made based on routine post-transplant surveillance or symptomatic presentation and confirmed via biopsy AMR can occur within the first few months (early AMR) or >6 months (late AMR) post-kidney transplant	Off label use of broad immunosuppressants characterized by limited efficacy in late AMR and potential for severe side effects	Late AMR carries an elevated risk of kidney transplant rejection: >75% transplant loss with a median graft survival of ~2 years after diagnosis[1]

High unmet need for disease modifying agents that preserve kidney function

Significant market opportunity with ~11k late AMR patients in the U.S[2]

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1. Redfield RR, et al., 2016; 2. Calculated from annual transplant incidence (Source: https://optn.transplant.hrsa.gov/data/view-data-reports/national-data/#), AMR incidence (Schinstock, C.A., et. al.), 5-year patient survival (Ciancio et al https://onlinelibrary.wiley.com/doi/abs/10.1111/ctr.13392) and assessments of early vs. late AMR (Hart, clin. Transplant., 2021)

LATE AMR CONSTITUTES A HIGH UNMET NEED AND SIGNIFICANT COMMERCIAL OPPORTUNITY

There are roughly 90k patients on the waitlist for a kidney transplant each year in the U.S.[1]

- ~40% of kidney transplant recipients are between 50-64 years of age[2]
- Annual cost of managing end-stage kidney disease is $230k[3]

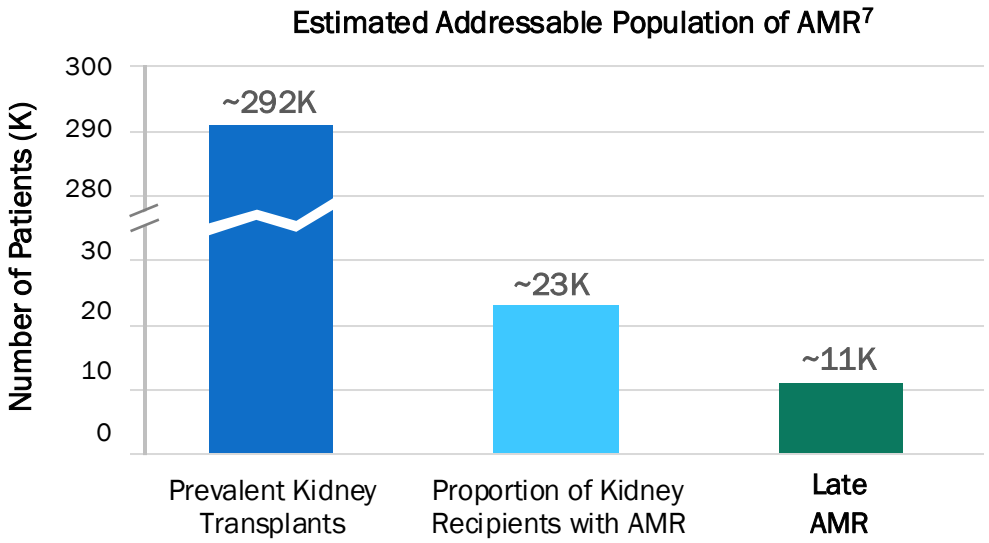
Transplanted kidneys are scarce and expensive

- There are ~28k kidney transplants in the U.S. each year[4]
- Only ~1/3 of patients on the wait list receive a transplant each year[4]
- Cost of a transplant is ~$450k[5]

There is a high burden to late AMR

- >75% of late AMR patients lose their kidney transplant[6]
- 11k patients diagnosed with Late AMR in the U.S.[7]
- Cost of treating AMR is ~$160k/year[8]

The development of Late AMR is an important risk for the transplanted kidney population



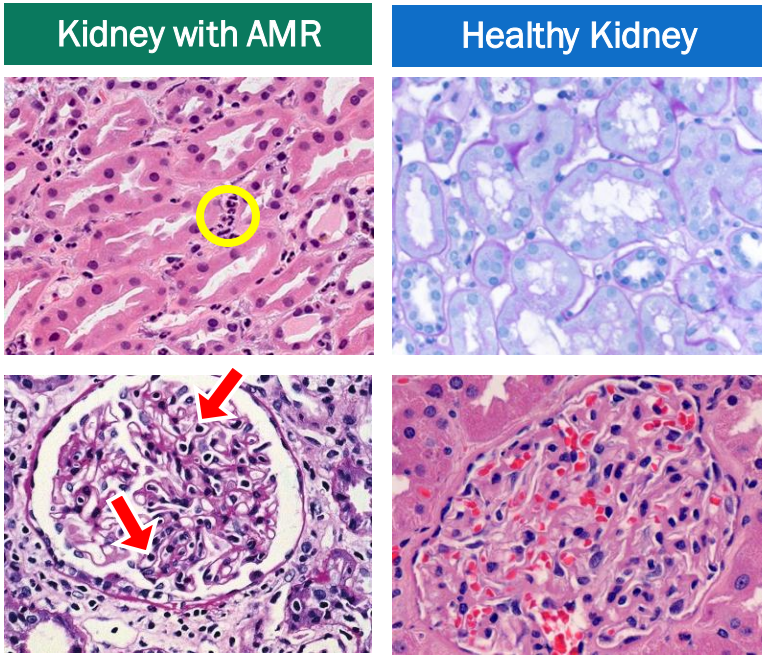
1. https://www.kidneyregistry.com/for-donors/kidney-donation-blog/is-there-a-living-kidney-donor-waiting-list-for-kidney-transplants;. 2. Brooks, Ned. National Kidney Registry, 2023. 3. League et al., 2022; 4. Increasing organ transplant access (iota) model. CMS.gov. (n.d.); 5. Wang JH, Hart A.. Kidney360. 2021; 6. Redfield RR, et al, 2016; 7. Calculated from annual transplant incidence (Source: https://optn.transplant.hrsa.gov/data/view-data-reports/national-data/#), AMR incidence (Schinstock, C.A., et. al.), 5-year patient survival (Ciancio et al https://onlinelibrary.wiley.com/doi/abs/10.1111/ctr.13392) and assessments of early vs. late AMR (Hart, clin. Transplant., 2021); 8. Banga et al., American Transplant Congress, 2015

AMR IS DEFINED BY ACTIVE FEATURES OF MICROVASCULAR INFLAMMATION IN BIOPSY

Microvascular inflammation is central to AMR and is defined by peritubular capillaritis and glomerulitis

Peritubular capillaritis: inflammatory cells within the capillary lumina (yellow circle)

Glomerulitis: Infiltration of leukocytes in capillary loops (red arrows) and endothelial swelling



AMR biopsy resolution is based upon Banff criteria and is achieved when active features of microvascular inflammation (MVI), including peritubular capillaritis and glomerulitis, and other factors resolve

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BANFF Foundation for Transplant Pathology. Banff classification for Renal Transplant Pathology, 2022 https://banfffoundation. org/central-repository-for-banff-classification-resources-3

MULTIPLE AGENTS IN DEVELOPMENT FAILED TO MEET THE EFFICACY THRESHOLD FOR PRESCRIBERS

	Clazakizumab[1]	Bortezomib[2]	Rituximab (aCD20)[3]
Sample size	N = 20	N = 44	N = 25
Biopsy Resolution	20%	19%	0%

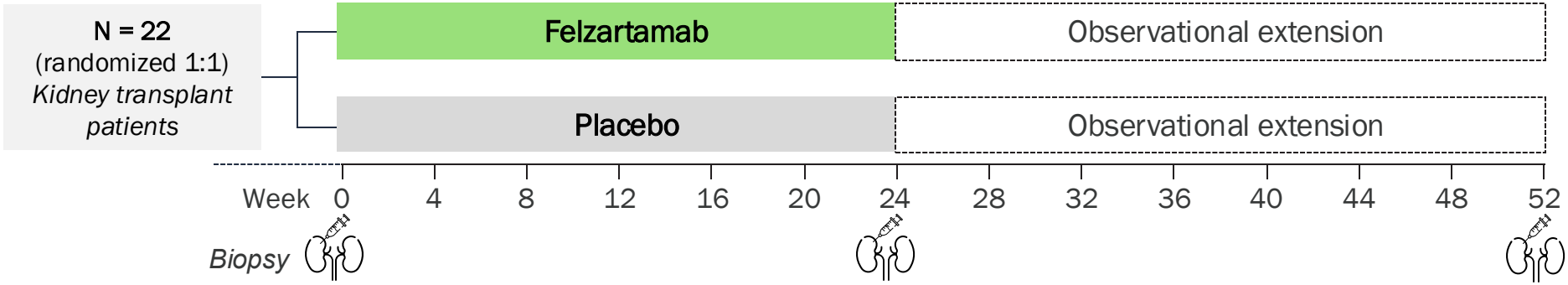
KOL aspiration for future treatment[4]

>45%
Clinically Meaningful

>75%
Transformational

Biopsy resolution defined as MVI<2; Analysis time points: Clazakizumab (52w), Bortezomib (24m), Rituximab (12m)
1. Böhmig et al, JASN 2021; 2. Eskandary et al., JASN 2018; 3. Moreso et al, Am. J Transplant 2017; 4. Blinded primary market research among KOLs in the United Stated, conducted in collaboration with Clearview Healthcare Partners (4Q 2023); n=10. MVI = microvascular inflammation

A PHASE 2 STUDY WAS CONDUCTED TO DETERMINE WHETHER FELZARTAMAB COULD RESOLVE LATE AMR IN KIDNEY TRANSPLANT



Primary outcome: Safety and side effect profile of felzartamab

Key prespecified safety outcomes were:

- Incidence of serious AEs
- IRRs
- Infections or infestations

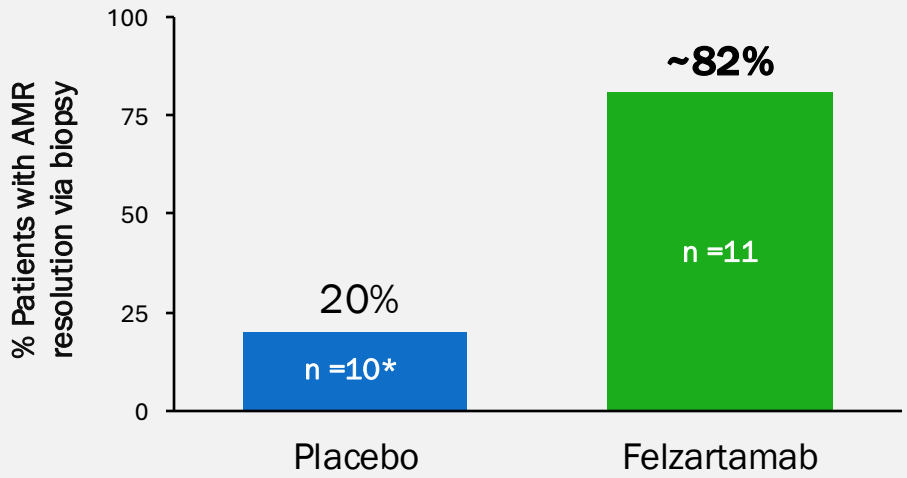
Selected secondary outcomes:

- Morphologic and molecular AMR activity
- Resolution of MVI
- DSA characteristics
- NK and plasma cell counts
- dd-cfDNA
- eGFR slope
- Torque teno viral load

AE = adverse event; dd-cfDNA = Donor-derived cell-free DNA; DSA = Donor-specific antibodies; eGFR = estimated glomerular flow rate; IRR = infusion-related reactions; NK = natural killer cell; RCT = randomized controlled trial

FELZARTAMAB PHASE 2 STUDY SHOWED THE POTENTIAL FOR TRANSFORMATIONAL EFFICACY IN LATE AMR

Felzartamab treatment resulted in >80% biopsy late AMR resolution at week 24



Meaningful improvement in kidney function

1-year eGFR improvement of 4.14ml/min (-3.20 to 11.48)

Results published in NEJM May 2024

ORIGINAL ARTICLE

A Randomized Phase 2 Trial of Felzartamab in Antibody-Mediated Rejection

K.A. Mayer, E. Schrezenmeier, M. Diebold, P.F. Halloran, M. Schatzl, S. Schranz, S. Haindl, S. Kasbohm, A. Kainz, F. Eskandary, K. Doberer, U.D. Patel, J.S. Dudani, H. Regele, N. Kozakowski, J. Kläger, R. Boxhammer, K. Amann, E. Puchhammer-Stöckl, H. Vietzen, J. Beck, E. Schütz, A. Akifova, C. Firbas, H.N. Gilbert, B. Osmanodja, F. Halleck, B. Jilma, K. Budde, and G.A. Böhmig

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*Placebo patient (n=1) lost graft at 14w (biopsies not collected)
AMR = antibody-mediated rejection; NEJM = New England Journal of Medicine

MAJORITY OF ADVERSE EVENTS WERE MILD TO MODERATE IN SEVERITY WITH NO TREATMENT DISCONTINUATIONS

Adverse Events*

Event	Felzartamab (N=11)		Placebo (N=11)	
	No. of patients %	No. of events	No. of patients %	No. of events
Any adverse event – no. (%)	11 (100)	119	11 (100)	81
Mild	11 (100)	61	9 (82)	37
Moderate	11 (100)	55	11 (100)	42
Severe	2 (18)	3	1 (9)	2
Treatment-related – no. (%)†	10 (91)	27	7 (64)	11
Infusion-related reaction – no. (%)‡	8 (73)	8	0	0
Serious event	1 (9)	2	4 (36)	7
Covid-19 pneumonia	0	0	2 (18)	2
Urinary tract infection	0	0	2 (18)	2
Hyponatremia	0	0	1 (9)	1
RSV infection	0	0	1 (9)	1
Clostridium difficile diarrhea	0	0	1 (9)	1
Acute kidney injury	1 (9)	1	0	0
Viral keratoconjunctivitis	1 (9)	1	0	0

*Covid-19 denotes coronavirus disease 2019, and RSV respiratory syncytial virus.
† The determination that an adverse event was related to felzartamab or placebo was made by investigators.
‡ Infusion-related reaction was a predefined adverse event of special interest. In the felzartamab group, such events were classified as mild (in 2 patients) or moderate (in 6 patients).
Source: Mayer et al., N Engl J Med 2024;391:122-13

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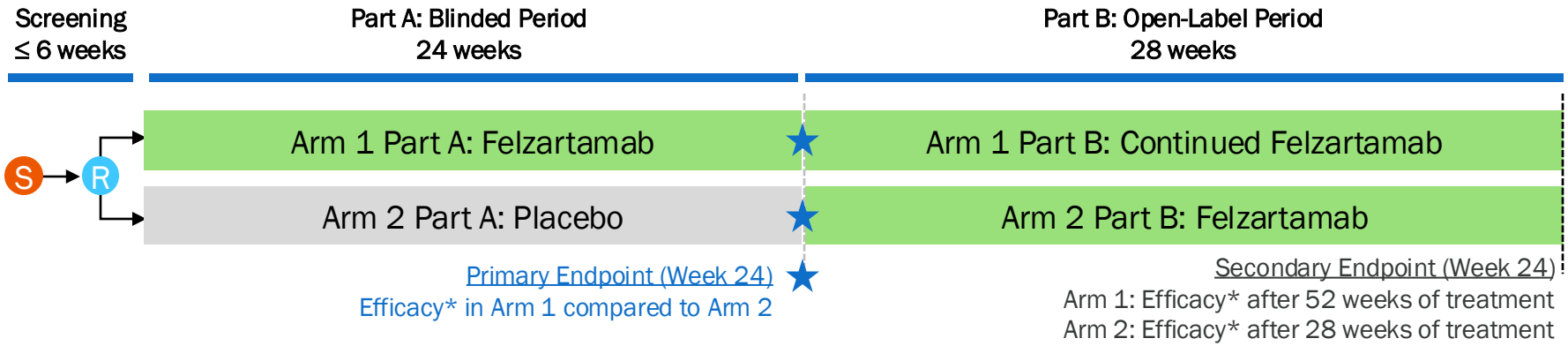
PHASE 3 STUDY FOR FELZARTAMAB IN LATE AMR IN KIDNEY TRANSPLANT IS UNDERWAY WITH DATA EXPECTED IN 2027

TRANSCEND study is a double-blind, placebo-controlled, multicenter, randomized Phase 3 trial with a targeted enrollment of 120 participants

Population: Kidney Transplant Recipients with Late AMR

Primary Endpoint: Percentage of Participants Who Achieve Biopsy-proven Histologic Resolution at week 24

Expected Readout: 2027



Potential for chronic treatment to manage relapse risk

* Efficacy consists of biopsy-proven histologic resolution of AMR

FELZARTAMAB IN AMR: POTENTIAL TO BE THE FIRST APPROVED TREATMENT WITH TRANSFORMATIONAL EFFICACY

- Late AMR is the most common driver of kidney transplant loss
- There are currently no approved treatments and prior agents in development have failed to meet the efficacy threshold for prescribers
- Felzartamab phase 2 data showed transformational >80% resolution of AMR*

Phase 3 data expected in 2027

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*Transformational efficacy defined by internal KOL market research as having biopsy resolution greater > 75%


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AMR:
QUESTION & ANSWERS

IGAN

IGA NEPHROPATHY

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IGAN IS A LEADING CAUSE OF END STAGE RENAL DISEASE WITH A NEED FOR NEW TREATMENT OPTIONS WITH DURABLE DISEASE REMISSION

IgAN is driven by immune complexes composed of antibodies produced by CD38-expressing plasma cells

Pathology	Diagnosis	Current SoC	Patient Impact
Immune complexes comprised of galactose deficient-IgA1 and anti-IgA autoantibodies are deposited in the kidney resulting in tissue damage	Typically **presents at ages 20 – 40**, with males twice as likely to develop IgAN Outside of countries with regular screening (e.g., Japan) patients are typically diagnosed through incidental findings and confirmed through biopsy	Current and emerging standard of care involves steroids and chronic immunosuppression and/or non-specific approaches to manage proteinuria (e.g., UPCR)	Up to 40% of patients reach end-stage kidney disease within 20 years of diagnosis[1]

High unmet need for disease modifying agents that provide durable disease remission

Significant market opportunity with ~130k patients in the U.S.[2]

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1. Yexin Liu, et. Al, Prediction of ESRD in IgA Nephropathy Patients from an Asian Cohort: A Random Forest Model. Kidney Blood Press Res 20 December 2018; 43 (6): 1852–1864; 2. Based upon Kwon. JHEOR. 2021; Jarrick. Am Soc of Neph. 2019

MARKET RESEARCH SUGGESTS A NEED FOR A DIFFERENTIATED TREATMENT OPTION

In addition to enhanced efficacy and acceptable safety, physicians and patients are looking for a novel therapy that delivers durable remission

Physicians

"9 injections, even if the first ones are weekly is fine, it's totally acceptable on healthcare and patients."
– FR Nephrologist KOL

"The shorter the therapy is the better…"
– UK Nephrologist KOL

"There'll be less need for any further immunosuppressive treatment, which means there'll be less infections and treatment related complications."
– UK Nephrologist HVP

Patients

"If you don't have to think about it, you'll never forget to take your medication."
– U.S. Patient

"You know, the fact that I would have to take [a product] every 2 weeks, it would be a reminder that I have this…"– U.S. Patient

"But I think the [treatment] holiday is much more important because then you're not doing anything at all."
– U.S. Patient

Physicians and patients have defined a clear ***preference for non-chronic treatment*** in IgAN

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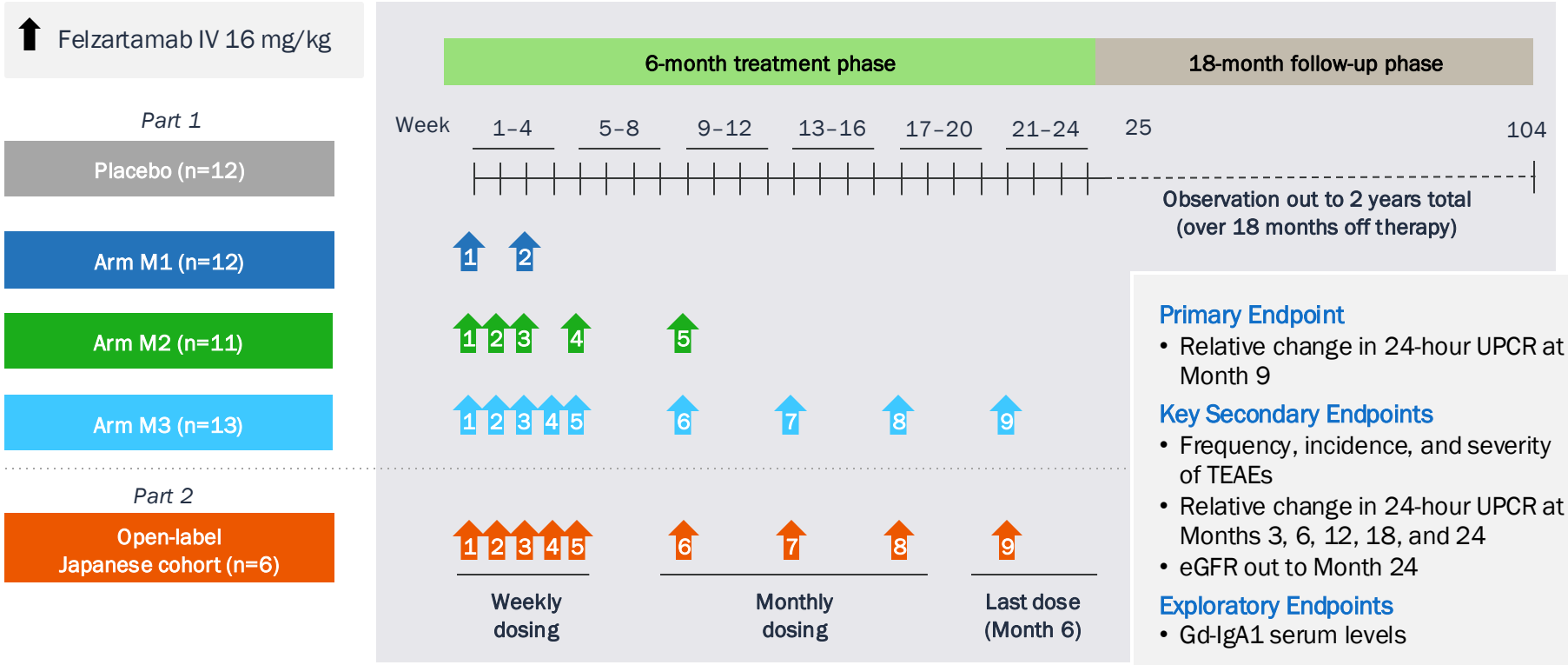
Source: ClearView primary market research in IgAN

A PHASE 2 STUDY TESTED FELZARTAMAB'S BENEFIT IN IGAN OUT TO 2 YEARS ACROSS DIFFERENT DOSE REGIMENS



Primary Endpoint

- Relative change in 24-hour UPCR at Month 9

Key Secondary Endpoints

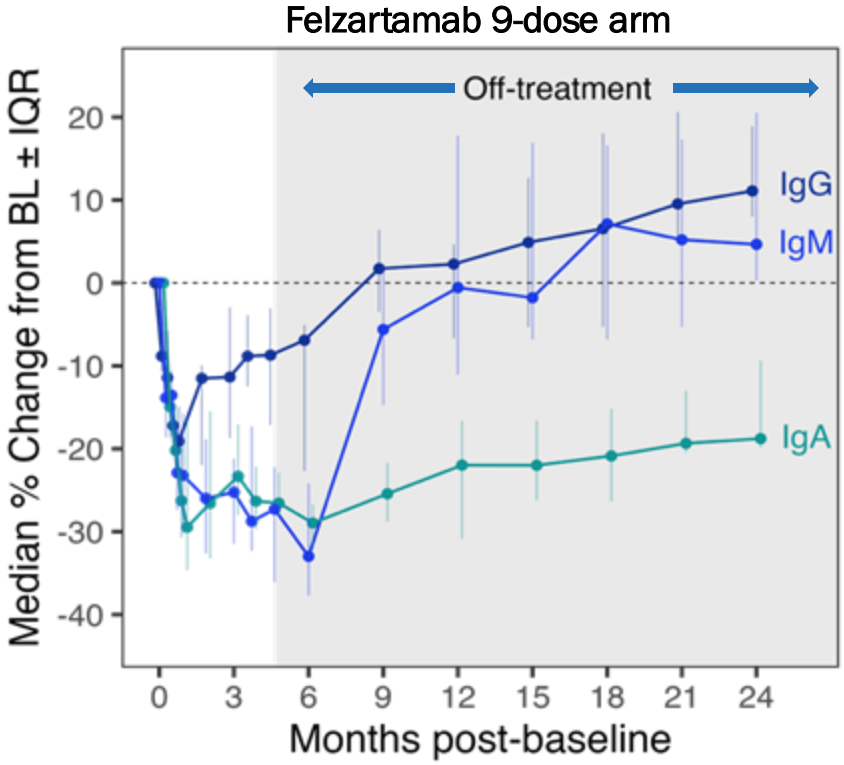
- Frequency, incidence, and severity of TEAEs
- Relative change in 24-hour UPCR at Months 3, 6, 12, 18, and 24
- eGFR out to Month 24

Exploratory Endpoints

- Gd-IgA1 serum levels

Floege J., et al. *Kidney International* 2025 (in press)
ADA = anti-drug antibodies; eGFR = estimated glomerular flow rate; Gd-IgA1 = galactose deficient IgA1; TEAE = treatment emergent adverse events; UPCR = urine protein creatine ratio

FELZARTAMAB DIFFERENTIATED PHASE 2 DATA SHOWED A SUSTAINED REDUCTION IN PATHOLOGIC ANTIBODY WITH A REBOUND IN PROTECTIVE ANTIBODIES



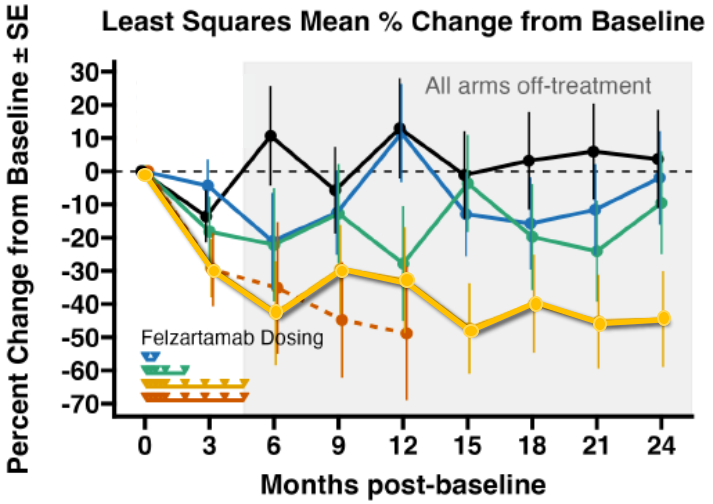
Rapid rebound of protective IgG and IgM observed with cessation of treatment

Sustained suppression of pathologic IgA persists at 18-months off treatment

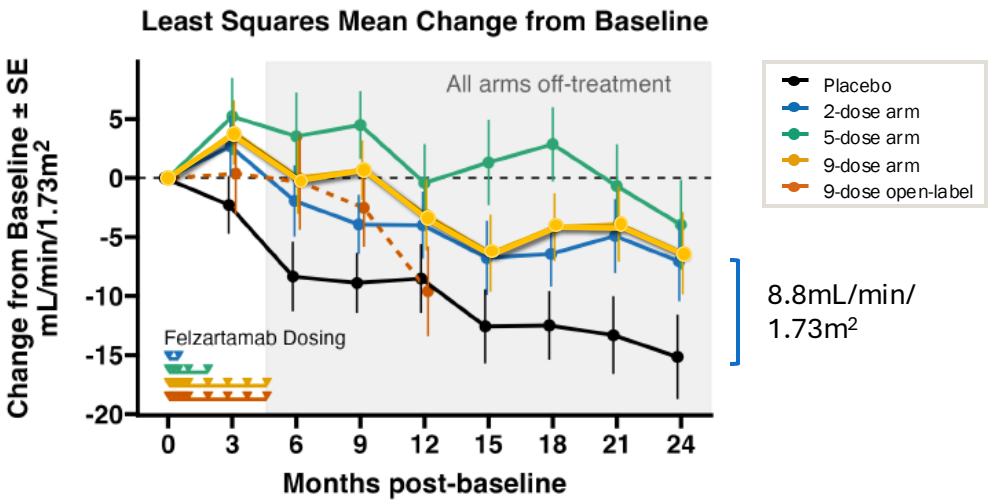
Source: Floege et al., ERA Congress, 2024

PHASE 2 DATA SHOWS THAT WITH 5 MONTHS OF TREATMENT PATIENTS HAVE SUSTAINED CLINICAL BENEFIT OUT TO 2 YEARS

Proteinuria (UPCR) % Change from Baseline



eGFR Change from Baseline



Sustained Efficacy out to 24 months

In the 9-dose group (i.e., 5 months of treatment) felzartamab demonstrated **~50% UPCR reduction at 24 months and durable pbo-adjusted stabilization of eGFR (>18 months off treatment)**

Observed safety profile

Administration of felzartamab was generally well tolerated with a safety profile generally consistent with prior studies

Source: Floege et al., Kidney week, 2024
BL = baseline; IgAN = IgA nephropathy; eGFR = estimated glomerular filtration rate; SE = standard error; UPCR = urine protein creatinine ratio

CLINICAL DATA SUPPORTS A DIFFERENTIATED PROFILE FOR FELZARTAMAB IN IGAN

	Anti-CD38[1] (felzartamab)	APRIL/BAFF[2]
Disease-specific MoA	✓ Depletion of auto-antibody producing plasma cells	✗ Inhibition of naïve & memory B-cells
Proteinuria (UPCR)	~50% reduction at 24 months (18 months off drug) **Off drug**	42-67% reduction @ 11-12 months (chronic therapy) **On treatment**
eGFR	Stable @ 24 months	Stable @ 11-12 months
Non-chronic treatment	✓ Phase 2 data shows 5-month dosing shows durable response out to 2 years	✗ Phase 2 studies utilized continuous treatment in the 48-52-week studies

1. Floege et al., ERA Congress, 2024; 2. Data ranges include Phase 2 data from 1) atacicept (42% proteinuria reduction @ 48 wk): ORIGIN phase 2b NCT04716231, J Am Soc Nephrol . 2024 Oct 26:10.1681; 2) zigakibart (67% proteinuria reduction @ 52 wk): phase 1/2 NCT03945318, press release 2024 Jun; 3) sibeprenlimab (62% proteinuria reduction @ 52 wk): ENVISION phase 2 NCT04287985, n engl j med 390;1 2024 Jan; and 4) povetacicept (66% proteinuria reduction @ 48 wk): RUBY-3 phase 1/2, NCT05732402, press release 2024 Nov, 2023 Nov
Note: Direct comparisons should not be made as these were different studies, with different patients and study designs

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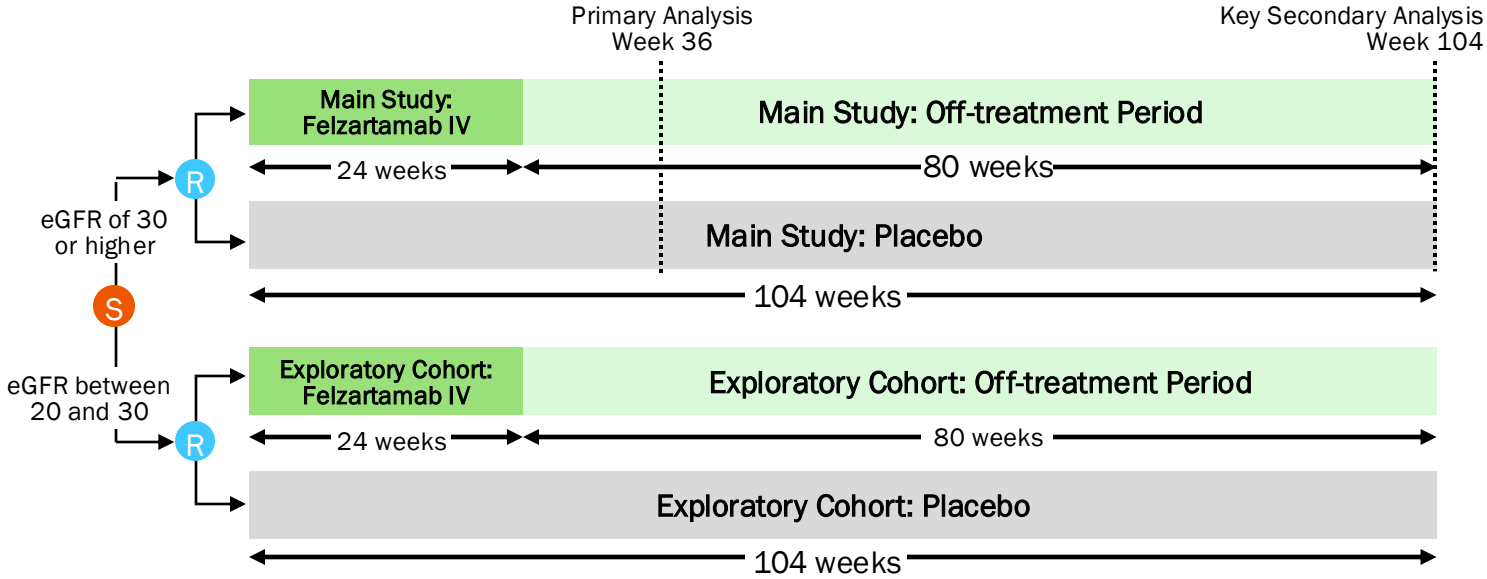
PHASE 3 STUDY IS DESIGNED TO DEMONSTRATE IMPROVEMENT IN KIDNEY FUNCTION WITH POTENTIAL FOR NON-CHRONIC TREATMENT

PREVAIL study is a Phase 3, Randomized, Double-blind, Placebo-controlled trial with a targeted enrollment of 450 participants

Population: Adults with IgA Nephropathy

Primary Endpoint: Percent Change From Baseline in Proteinuria as Measured by the UPCR at week 36

Expected Readout: 2029



S Screening R Randomization

FELZARTAMAB IN IGAN: POTENTIAL TO BE THE ONLY OPTION WITH DURABLE TREATMENT-FREE REMISSION

- Up to 40% of patients reach end-stage kidney disease within 20 years of diagnosis
- High unmet need for treatments that provide durable treatment free disease remission
- Felzartamab Phase 2 data shows 5 months of treatment resulted in sustained clinical benefit while off therapy for up to 24 months

Phase 3 data expected in 2029

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IGAN:
QUESTION & ANSWERS

PMN

PRIMARY MEMBRANOUS NEPHROPATHY

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PMN IS A LEADING CAUSE OF NEPHROTIC SYNDROME WITH NO APPROVED THERAPIES

Majority of PMN cases are driven by PLA2R auto-antibodies generated by CD38-expressing plasma cells

Pathology	Diagnosis	Current SoC	Patient Impact
In ~80% of PMN cases, anti-PLA2R autoantibodies accumulate in the kidney, forming deposits that result in kidney damage and reduced filtration	Disease onset and diagnosis typically occurs at **40 – 50 years old**, and predominantly impacts Caucasians Most patients present with Nephrotic syndrome (i.e., edema/swelling, proteinuria) and diagnosis is often confirmed through biopsy	No approved therapies Patients mostly cycle through immunosuppressant therapies, anti-CD20s or chemotherapy drugs	Up to 40% of patients progress to end-stage kidney disease within 15 years[1]

High unmet need for disease modifying agents that treat high-risk and relapsed patients

Important market opportunity with ~36k PMN patients in the U.S[2]

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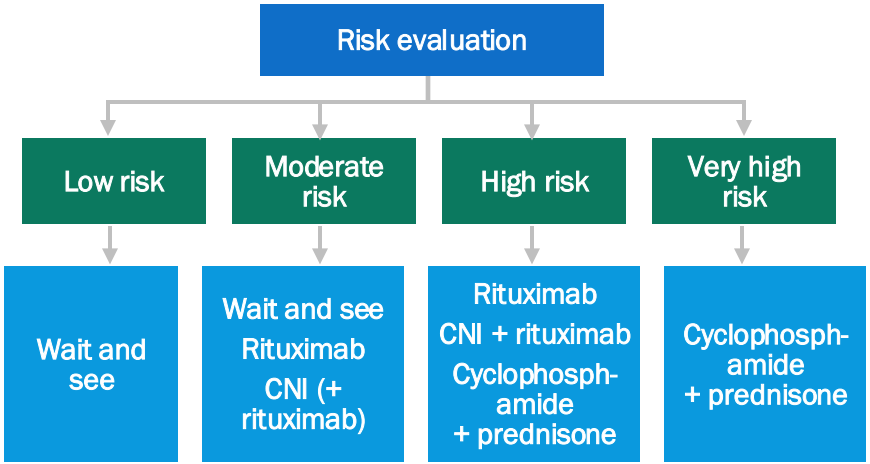
1. Lai WL, et al., Membranous nephropathy: a review on the pathogenesis, diagnosis, and treatment. J Formos Med Assoc. 2015 Feb;114(2):102-11; 2. Based upon Kanigicherla. Nephrol. Dial. Transplant. 2016; McGrogan. Nephrol Dial Transplant. 2011; 36k represents the total number of diagnosed patients who are actively being managed.

THERE IS A CLEAR UNMET NEED FOR SAFER AND MORE EFFECTIVE OPTIONS FOR TREATING PMN

Treatment is based on risk evaluation



Risk assessment based on GFR and proteinuria

Off-label treatment options have high risk of relapse and/or toxicity[1, 2]

Cyclophosphamide + prednisolone
- 20%–30% relapse rate
- Lowers rate of ESKD but dosing is limited by toxicity

Calcineurin inhibitor (CNI)
- 40%–50% relapse rate
- Unclear if CNIs prevent ESKD

Rituximab
- 20%–40% of patients will not respond to initial treatment
- ~30% relapse rate

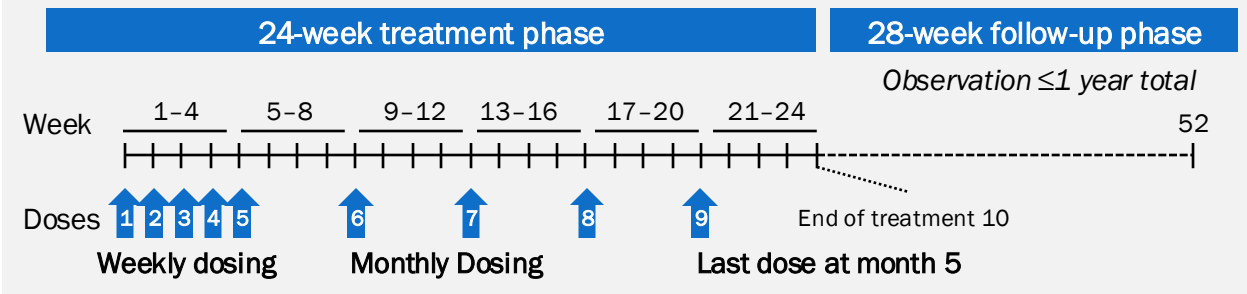
ESKD = end-stage kidney disease

1.. Couser WG. Clin J Am Soc Nephrol. 2017;12(6):983–997; 2. Teisseyre M et al. Front Immunol. 2022;13:859419.

AN OPEN-LABEL PHASE 1B/2A STUDY WAS CONDUCTED TO TEST THE HYPOTHESIS THAT FELZARTAMAB COULD IMPROVE OUTCOMES FOR PATIENTS

Cohort 1; n=18
Newly diagnosed & relapsed

Cohort 2; n=13
Refractory



Primary objective:

- To assess the safety and tolerability of felzartamab treatment in subjects with anti-PLA2R autoantibody positive membranous nephropathy

Primary endpoint:

- Incidence and severity of TEAEs

Key secondary and exploratory endpoints

- Reduction of serum anti-PLA2R autoantibody titer
- Serum concentrations of felzartamab and anti-drug antibodies
- Clinical efficacy based on proteinuria reduction
- Changes in quality of life
- Incidence and severity of AEs during follow-up phase

Cohort 1: newly diagnosed patients and those who relapsed on prior therapy; patients with serum anti-PLA2R antibodies ≥50 RU/ml determined at screening (central laboratory, Euroimmun ELISA)
Cohort 2: patients with anti-PLA2R antibody-positive MN refractory to a prior IST and requiring another IST; patients with serum anti-PLA2R antibodies ≥20 RU/ml measured at screening (central laboratory, Euroimmun ELISA)

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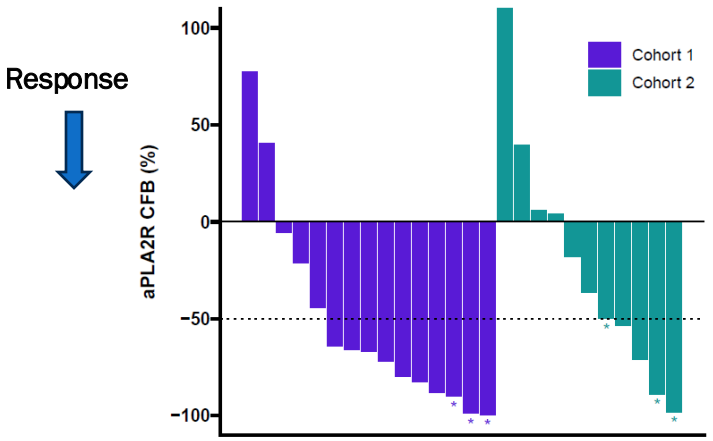
FELZARTAMAB DRIVES ROBUST AND SUSTAINED REDUCTIONS IN ANTI-PLA2R IN BOTH NDR AND REFRACTORY PATIENTS

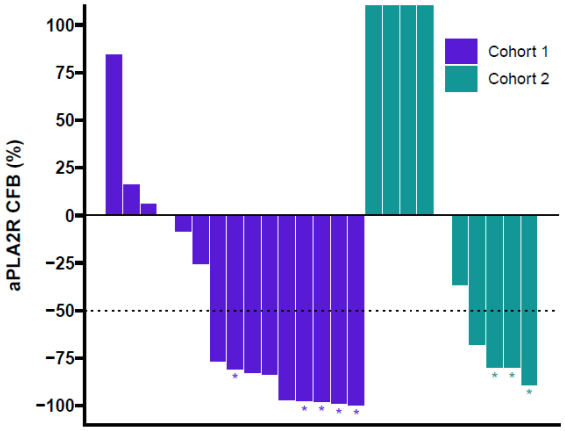
Depth of Response

6 month timepoint (1 month after final dose)



Durability of Response

12 month timepoint (7 months after final dose)



Cohort 1

- Newly diagnosed & relapsed
- Serum anti-PLA2R >50.0 RU/mL

Cohort 2

- Refractory subjects to prior immunosuppressive therapy
- Serum anti-PLA2R >20.0 RU/mL

Depletion of PLA2R autoantibodies is sustained out to 1-year after a 5-month course of felzartamab

ICR: immunologic complete remission, <14 RU/mL; iPR: immunologic partial remission, reduction of ≥50%

Rovin BH, Ronco PM, Wetzels JFM, et al. Phase 1b/2a Study Assessing the Safety and Efficacy of Felzartamab in Anti-Phospholipase A2 Receptor Autoantibody–Positive Primary Membranous Nephropathy. Kidney Int Rep. 2024;9(9):2635-2647. doi:10.1016/j.ekir.2024.06.018.

NDR = newly diagnosed and relapsed; RU = relative units

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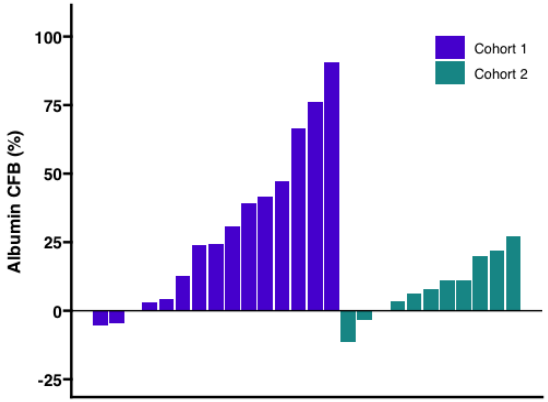
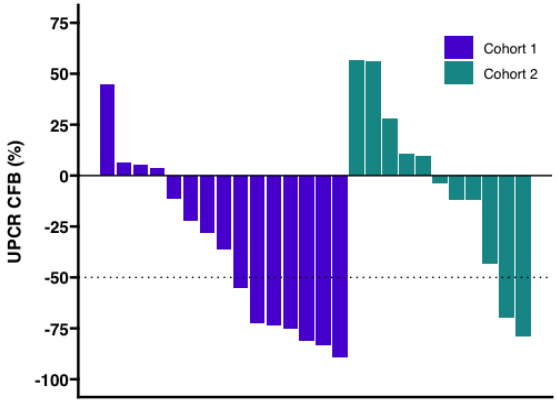
REDUCTIONS IN ANTI-PLA2R RESULTED IN IMPROVEMENTS IN KIDNEY FUNCTION AS COMPARED TO BASELINE AT 12 MONTHS

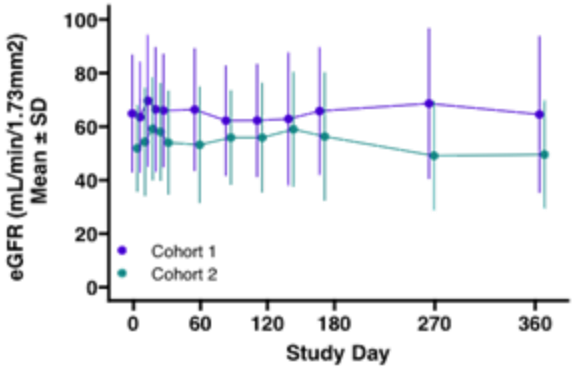
Recovery of serum albumin



Emerging proteinuria responses



Stable eGFR through end of study



Cohort 1
- Newly diagnosed & relapsed
- Serum anti-PLA2R >50.0 RU/mL

Cohort 2
- Refractory subjects to prior immunosuppressive therapy
- Serum anti-PLA2R >20.0 RU/mL

Administration of felzartamab was generally well tolerated with a safety profile generally consistent with prior studies

Rovin BH, Ronco PM, Wetzels JFM, et al. Phase 1b/2a Study Assessing the Safety and Efficacy of Felzartamab in Anti-Phospholipase A2 Receptor Autoantibody–Positive Primary Membranous Nephropathy. Kidney Int Rep. 2024;9(9):2635-2647. doi:10.1016/j.ekir.2024.06.018.

TEAE = treatment emergent adverse event

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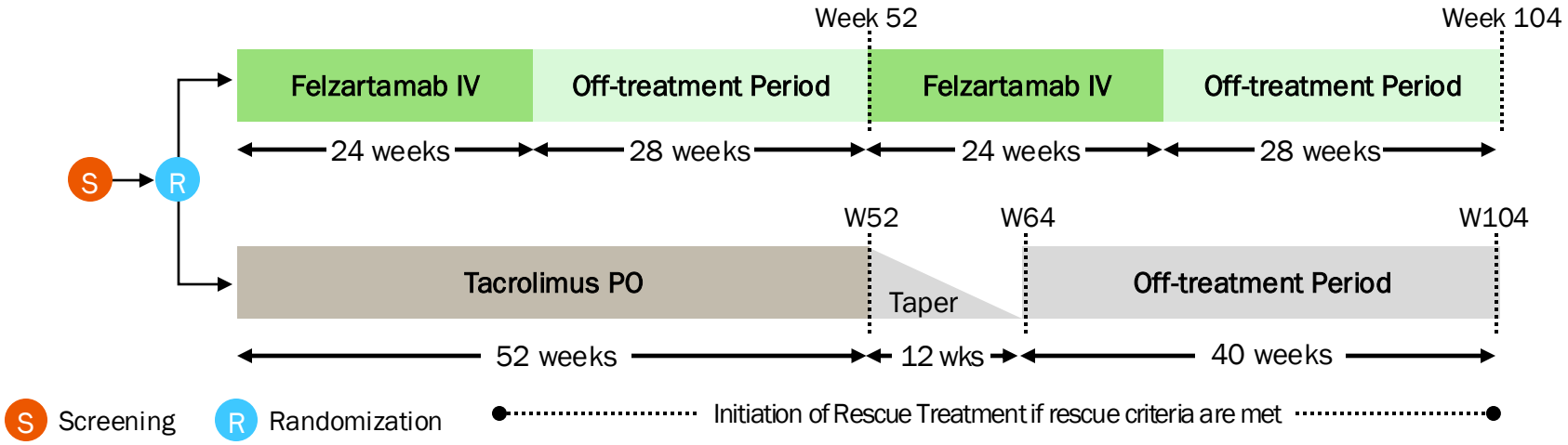
PHASE 3 STUDY IS DESIGNED TO DEMONSTRATE COMPLETE REMISSION OF PROTEINURIA IN PMN

PROMINENT is an Open-label, Multicenter, Randomized Phase 3 trial with a targeted enrollment of 180 participants
Population: Adults with Primary Membranous Nephropathy that are newly diagnosed of have relapsed
Primary Endpoint: Percentage of participants who achieve complete remission at Week 104
Expected Readout: 2029



Felzartamab has the potential to transform the patient journey in PMN and become the first treatment for PMN that specifically targets plasma cells

FELZARTAMAB IN PMN: POTENTIALLY A PREFERRED AGENT FOR HIGH-RISK FIRST 1L AND 2L PATIENTS

- Up to 40% of patients progress to end-stage kidney disease within 15 years
- Current treatment options have limited efficacy and/or severe (chemotherapy) side effects
- Felzartamab phase 2 data showed robust and sustained reductions in anti-PLA2R and improvements in both NDR and refractory patients

Phase 3 data expected in 2029

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PMN:
QUESTION & ANSWERS

FELZARTAMAB CONCLUSION

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OUR FELZARTAMAB PHASE 3 PROGRAMS SPAN THREE IMPORTANT OPPORTUNITIES

AMR	IgAN	PMN
Potential first approved treatment with transformational efficacy	Potential to be the only option with durable treatment-free remission	Potential to be the preferred agent for high-risk first- and second-line patients
Phase 3 data expected in 2027	Phase 3 data expected in 2029	Phase 3 data expected in 2029

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EACH OF THESE INDICATIONS PRESENT COMMERCIALLY ATTRACTIVE OPPORTUNITIES

Relative Value Drivers	Late AMR	IgAN	PMN
Estimated U.S. Market Size	11k[1]	130k[2]	36k[3]
Potential Average annualized number of dose administrations over first 2 years	9* (Ongoing continuous dosing)	4.5	9
Potential Competitive Context	First approved treatment with transformational efficacy	Differentiated therapy with non-chronic dosing in an increasingly competitive market	Preferred agent for relapsing patients and potential first-line therapy for high-risk newly diagnosed patients

*Less frequent dosing during maintenance but is expected to continue indefinitely

1. Calculated from annual transplant incidence (Source: https://optn.transplant.hrsa.gov/data/view-data-reports/national-data/#), AMR incidence (Schinstock, C.A., et. al. Kidney transplant with low levels of DSA or low positive B-flow crossmatch: An underappreciated option for highly sensitized transplant candidates. Transplantation. 2017;101:2429–2439.), 5-year patient survival (Ciancio et al https://onlinelibrary.wiley.com/doi/abs/10.1111/ctr.13392)
2. Based upon Kwon. JHEOR. 2021; Jarrick. Am Soc of Neph. 2019
3. Based upon Kanigicherla. Nephrol. Dial. Transplant. 2016; McGrogan. Nephrol Dial Transplant. 2011; 36k represents the total number of diagnosed patients who are actively being managed.

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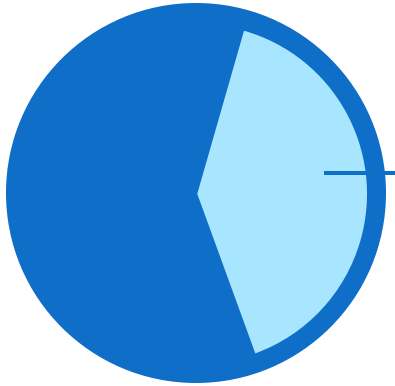
PHASE 3 THERAPEUTIC AREAS PRESENT COMMERCIALLY ATTRACTIVE OPPORTUNITIES WITH A FOCUSED COMMERCIAL FOOTPRINT

AMR

250 U.S. transplant centers[1]

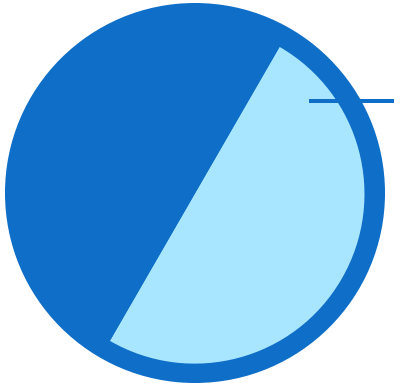


50-100 manage majority of patients

Community nephrologists monitor for symptoms of AMR and refer to transplant nephrologist for treatment

IgAN and PMN

Roughly 10,000 U.S. Nephrologists[2]



Roughly 5,000 physicians see the majority of patients

1. Wang JH, Hart A. Global Perspective on Kidney Transplantation: United States. Kidney360. 2021; 2. Centers for Medicare & Medicaid Services. National downloadable file. May 21, 2022. https://data.cms.gov/provider-data/dataset/mj5m-pzi6

LUPUS NEPHRITIS IS AN EXAMPLE OF FELZARTAMAB'S BROADER POTENTIAL IN IMMUNOLOGY

- Anti-CD38 potentially delivers durable benefits without the AEs associated with CAR T or T-cell engagers (e.g. CRS)
- Existing clinical data demonstrates that anti-CD38 is active in LN*
- We are executing a Phase 1 study for felzartamab in LN, first data expected by end of 2026
- Early-stage study results to inform the potential for a registrational study

We are evaluating additional expansion indications for Felzartamab

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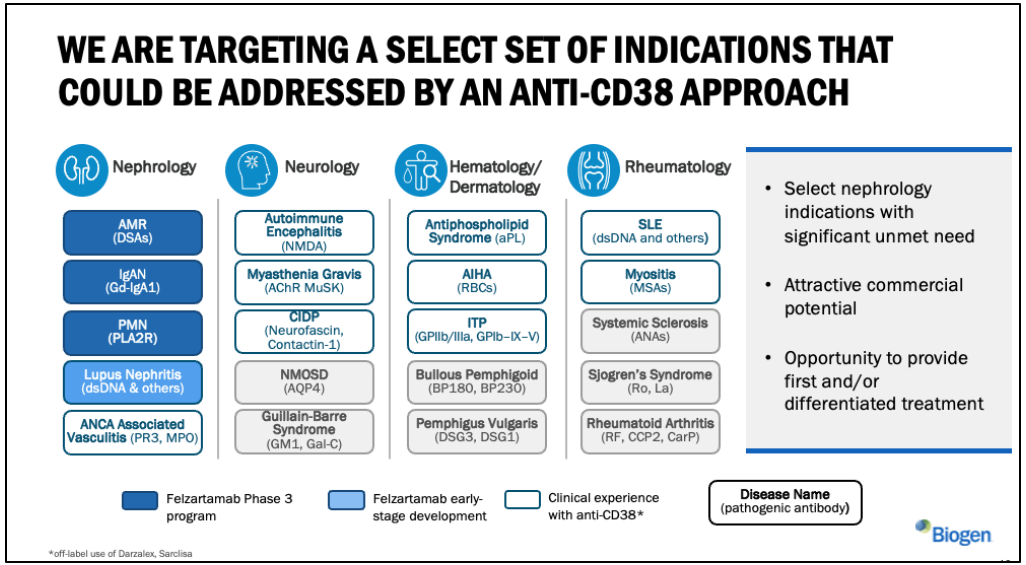
* Roccatello, D., Fenoglio, R., Caniggia, I. et al. Daratumumab monotherapy for refractory lupus nephritis. Nat Med 29, 2041–2047 (2023). https://doi.org/10.1038/s41591-023-02479-1

AE = adverse event; CAR = chimeric antigen receptor; CRS = cytokine release syndrome; LN = lupus nephritis

WE HAVE THE OPPORTUNITY TO FURTHER BUILD OUR CD38 FRANCHISE



Opportunities:

1. Deliver a **subcutaneous formulation of felzartamab** to allow dosing flexibility

2. Advance a **next-generation anti-CD38 antibody** to provide a distinct profile for future development as a follow-asset or in a separate set of indications

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FELZARTAMAB HAS THE POTENTIAL TO BE A FOUNDATION OF AN ANTI-CD38 FRANCHISE IN MULTIPLE IMMUNE-MEDIATED DISEASES

- **Best-in-class opportunity:** Novel aCD38 targeting plasma cells producing pathogenic antibodies and NK cells
- **Compelling clinical data:** Proof of concept data generated across multiple indications demonstrating durable disease modifying effects with strong safety & tolerability profile
- **Broad Application:** Potential to expand to other indications where an anti-CD38 approach may address key drivers of disease
- **Strong execution:** Phase 3 studies ongoing for AMR and IgAN with an additional Phase 3 study in PMN expected to start in 2025; phase 1 study in LN underway

Opportunity to expand our anti-CD38 franchise beyond felzartamab

CONCLUDING REMARKS

Priya Singhal, M.D., M.P.H.

Head of Development

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BUILDING AND STRENGTHENING OUR PIPELINE TO SUPPORT OUR LONG-TERM GROWTH OBJECTIVE

Phase 1	Phase 2	Phase 3		Regulatory Review in Certain Markets
Felzartamab (anti-CD38 mAb) – LN	BIIB080 (tau ASO)^ Early AD	Lecanemab (Aβ mAb)* SC-AI Initiation Early AD	Felzartamab (anti-CD38 mAb) – AMR	Lecanemab (Aβ mAb)* SC-AI Maintenance Early AD
Izastobart (C5aR1 mAb) – complement mediated disease	BIIB122 (LRRK2 inhibitor)* – PD *Now fully enrolled*	Lecanemab (Aβ mAb)* Preclinical AD	Felzartamab (anti-CD38 mAb) – IgAN	HD Nusinersen (SMN2 splice modulator) SMA
SKYCLARYS (Nrf2 activator) – Pediatric FA *Phase 3 planned in 2025*	BIIB091 (peripheral BTK Inhibitor) – MS	Dapirolizumab pegol (anti-CD40L)* – SLE	Felzartamab (anti-CD38 mAb) – PMN *Sites activated; screening initiated*	Zuranolone ($GABA_A$ PAM)* – PPD
Salanersen (BIIB115) (SMN ASO)^ – SMA Phase 1b	Zorevunersen (SCN1A ASO)* – Dravet syndrome *Phase 3 planned in 2025*	Litifilimab (BDCA2 mAb) – SLE		
		Litifilimab (BDCA2 mAb) – CLE		

Programs discussed today

AD and Dementia | *Immunology* | *Neuromuscular disorders* | *Neuropsych* | *Parkinson's disease* | *MS* | *Neurodevelopmental*

Pipeline Updates: Added = Zorevunersen in Dravet syndrome; Advanced = Felzartamab in AMR and IgAN to Phase 3; Felzartamab Phase 3 in PMN started screening.*Collaboration program; # Collaboration and option agreement; ^ Licensed from Ionis Pharmaceuticals, Inc.; AD = Alzheimer's disease; AMR = antibody mediated rejection; ASO = antisense oligonucleotide; CLE = cutaneous lupus erythematosus; DPNP = diabetic peripheral neuropathic pain; FA = Friedreich ataxia; GABA = γ-Aminobutyric acid; HD = higher dose; IgAN = IgA nephropathy; LN = lupus nephritis; LRRK2 = leucine rich repeat kinase 2; MS = multiple sclerosis; PAM = positive allosteric modulator; PD = Parkinson's disease; PMN = primary membranous nephropathy; PoC = proof-of-concept; PPD = postpartum depression; SC-AI = subcutaneous autoinjector; SLE = systemic lupus erythematosus; SMA = spinal muscular atrophy

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OUR DISCIPLINED SCIENTIFIC APPROACH IS POISED TO DELIVER KEY EXPECTED MILESTONES OVER THE NEXT 18 MONTHS

4 Phase 3 Starts

- Zorevunersen Phase 3 in Dravet syndrome*
- ***Felzartamab Phase 3 in IgAN***
- Felzartamab Phase 3 in PMN
- SKYCLARYS Phase 3 in pediatric FA

5 Clinical Trial Readouts

- Litifilimab Phase 3 in SLE
- Litifilimab Phase 3 in CLE#
- BIIB080 Phase 2 in Early AD
- Salanersen (BIIB115) Phase 1 in SMA
- Felzartamab Phase 1 in LN

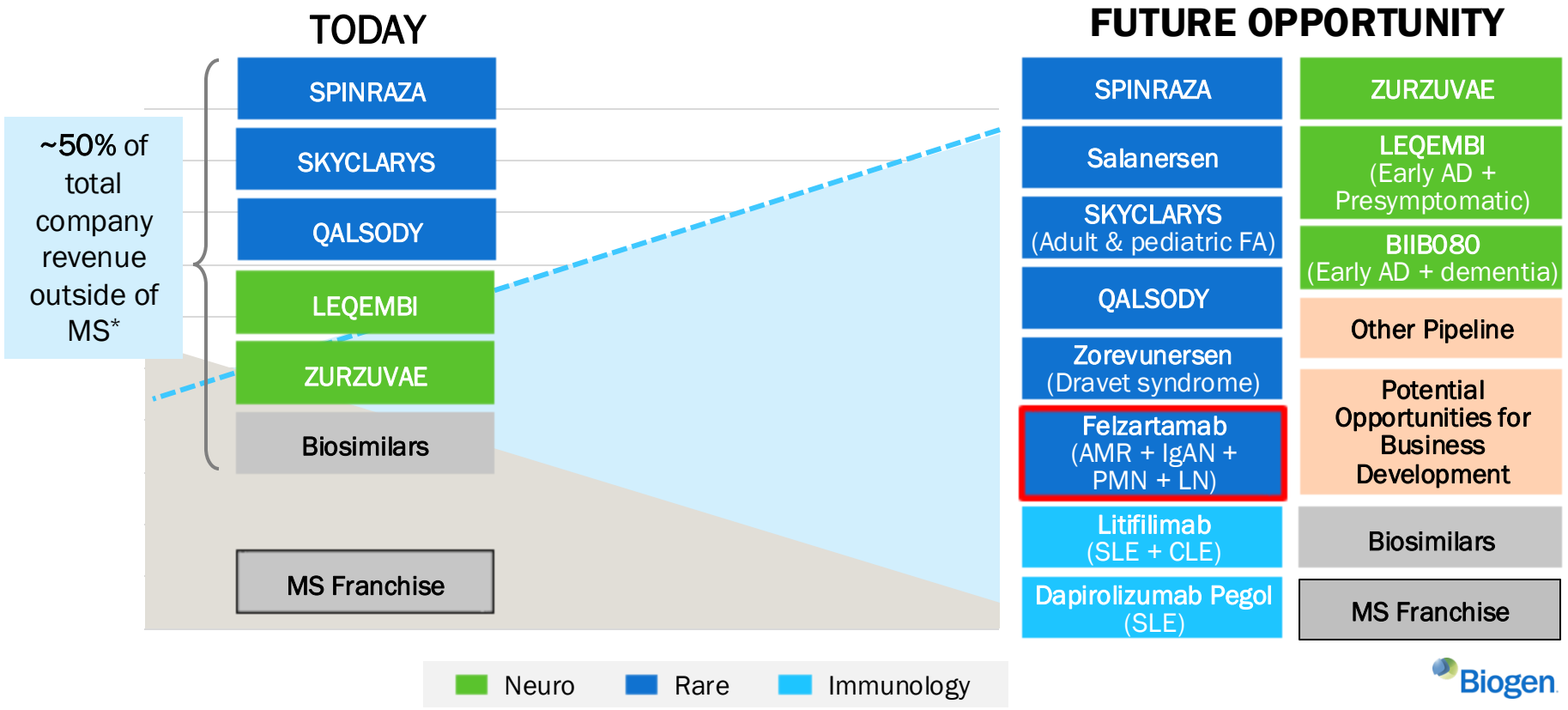
3 Regulatory Decisions

- LEQEMBI SC-AI maintenance in Early AD
- LEQEMBI SC-AI initiation in Early AD
- Nusinersen (SPINRAZA) higher dose in SMA

LEQEMBI (lecanemab-irmb) is being developed in collaboration with Eisai Co; BIIB080 is licensed from Ionis Pharmaceuticals, Inc.; Zorevunersen is being developed in collaboration with Stoke therapeutics; *Stoke has disclosed that first sites have initiated in the U.S. in May 2025; AD = Alzheimer's disease; AI = autoinjector; CLE = cutaneous lupus erythematosus; DS = Dravet syndrome; FA = Friedreich's ataxia; IgAN = IgA nephropathy; LN = lupus nephritis; PD = Parkinson's disease; PMN = primary membranous nephropathy; SC = subcutaneous; SLE = systemic lupus erythematosus; SMA = spinal muscular atrophy; # Readout expected H2 2026 to H1 2027

WE ARE ON A JOURNEY TO BUILD THE NEW BIOGEN

Broadening our portfolio across Neuro, Immunology & Rare Disease



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*MS product revenue plus royalty revenue on sales of OCREVUS in Q1 2025; 2. Consists of current products and potential products


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QUESTION &
ANSWERS