Building The Future of Immune Therapeutics

Company Overview

July 2024

Forward Looking Statements

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Q32 Mission: Building The Future of Immune Therapeutics

IL-7Rα Antagonist Antibody	• Bempikibart (ADX-914): Dual inhibitor of IL-7 and TSLP signaling, designed to provide broad control of T cell-mediated pathological processes in autoimmune diseases • Ph1 data show attractive PK/PD profile, favorable tolerability w/Q2W SC dosing; Ph2 PK run in confirmed Ph2 dose • Currently in two ongoing Ph2 clinical trials: Atopic Dermatitis (AD) and Alopecia Areata (AA)
Novel Tissue-targeted Complement Platform with Clinical Asset	• Differentiated, proprietary approach to address complement dysregulation directly at the site of impacted tissue • ADX-097: Designed to catalytically degrade alternative pathway convertases, gaining control of the amplification loop and all 3 complement pathways • Ph1 ADX-097 data show attainment of dose-dependent target PK/PD, favorable tolerability and good immunogenicity profile with Q1W SC dosing • Initiating in two Ph2 trials: Renal basket (1H'24) and ANCA-associated vasculitis (1H'25)
Near Term Value Creation Potential	• **Q4'24 - Bempikibart AD Ph2:** Topline results (including 14-week placebo-controlled efficacy data) • **Q4'24 - Bempikibart AA Ph2:** Topline results (including 24-week placebo-controlled efficacy data) • **2H'25 - ADX-097 Renal basket Ph2:** Topline results (initial data YE'24) • **2H'25 - ADX-097 AAV Ph2 Part A:** Topline results
Exceptional Team and Investors	• Management team with extensive public biotech experience • Deep complement therapeutics and inflammatory/autoimmune expertise • Blue chip investor syndicate

ATLAS VENTURE ABINGWORTH OrbiMed Healthcare Fund Management acorn BIOVENTURES OSAGE UNIVERSITY PARTNERS

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Q32 Pipeline: Poised to Deliver Multiple Near-term Clinical Readouts

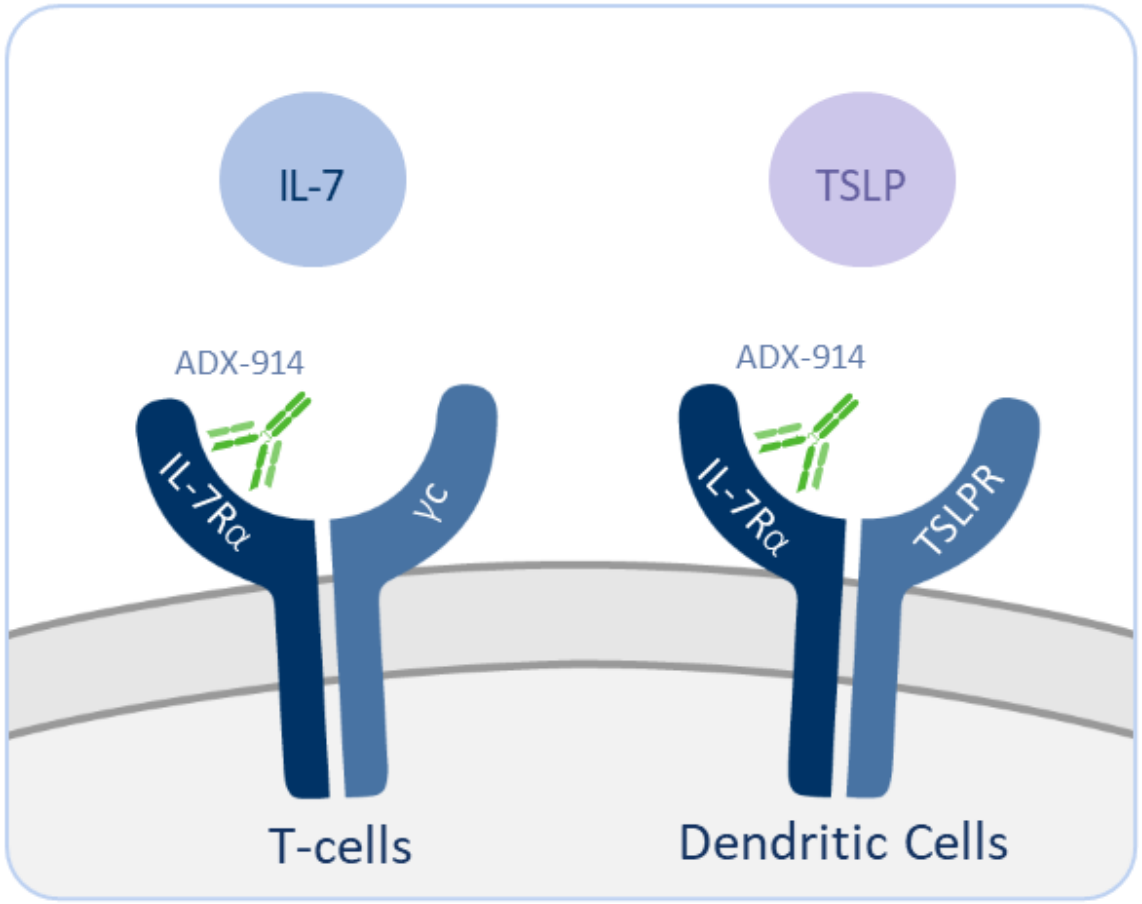
Program	Indication	Discovery/ Preclinical	Phase 1	Phase 2	Anticipated Milestones
IL-7/TSLP PROGRAM					
Bempikibart[1] (ADX-914)	Atopic Dermatitis				*AD enrollment complete; Ph2 topline results Q4'24*
	Alopecia Areata				*AA enrollment complete; Ph2 topline results Q4'24*
COMPLEMENT INHIBITOR PLATFORM					
ADX-097	Renal Basket (IgAN, LN, C3G)				*Renal Basket: topline results 2H'25*
	AAV				*AAV: Part A topline results 2H'25*

Note: AAV = Anti-Neutrophil Cytoplasmic Autoantibody (ANCA)-Associated Vasculitis; IgAN = IgA Nephropathy; LN = Lupus Nephritis; C3G = C3 Glomerulopathy.

Bempikibart (ADX-914)
(IL-7 / TSLP Receptor Inhibitor)

Bempikibart: Novel Investigational Therapy for T-cell Mediated Inflammatory and Autoimmune Diseases Delivering Two Phase 2 Readouts in 2024

Bempikibart: IL-7Rα antagonist antibody blocks IL-7 and TSLP signaling



IL-7

- Potent regulator of pathogenic T_{eff} / T_{mem} survival and proliferation
- Suppresses T_{reg} cells
- Activates TfH cells to induce B-cell mediated antibody production

TSLP

- Central regulator of DC differentiation and TH2 cytokine production
- Activates TH1, sensory neurons, mast cells, eosinophils, basophils and ILC2

Clinical Data Completed to Date

- Ph1: Durable subcutaneous (SC) PK/PD and tolerability
- Ph2 AD Part A: Confirmatory PK/PD (clinical data remains blinded)

Topline Clinical Efficacy Data Expected Q4'24

- AD Ph2 (Part A and B)
- AA Ph2

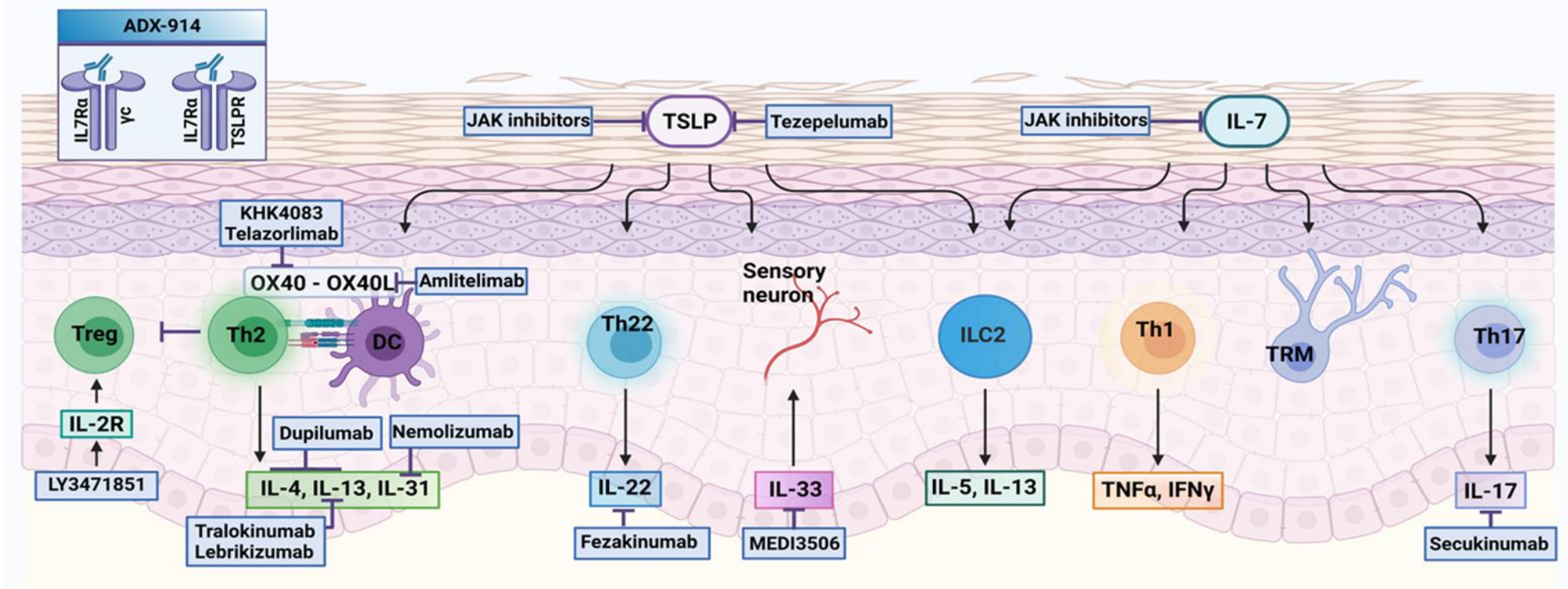
IL-7 and TSLP are Central Drivers of Inflammation and Autoimmunity

Pathogenic Immune Response	Ligand/Receptor Activation	Preclinical Evidence
• Induction of pathogenic T-eff/ T-mem and ILC2 cells • Inhibition of T-reg function • Increased Th-helper cell mediated antibody production • Activation of TH2 immune response	• Elevated IL-7 and sIL-7Rα in disease • Increased TSLP signaling in disease • Increased IL-7 and TSLP transcriptional signature in disease	• Overexpression of IL-7 or TSLP recapitulates disease pathology • Blocking IL-7 & TSLP pathways exerts protective effects in multiple models • Potential for long-term, durable responses and remittive therapy

Blockade of IL-7 and TSLP has therapeutic potential in a broad range of inflammatory and autoimmune diseases

AD – atopic dermatitis, AA – alopecia areata, RA – rheumatoid arthritis, NIU – non-infectious uveitis, MS – multiple sclerosis, UC – ulcerative colitis

Bempikibart in AD: Dual Mechanism Intended to Block Th2 and Th1 for Potential to Impact Both Acute and Chronic Pathogenesis

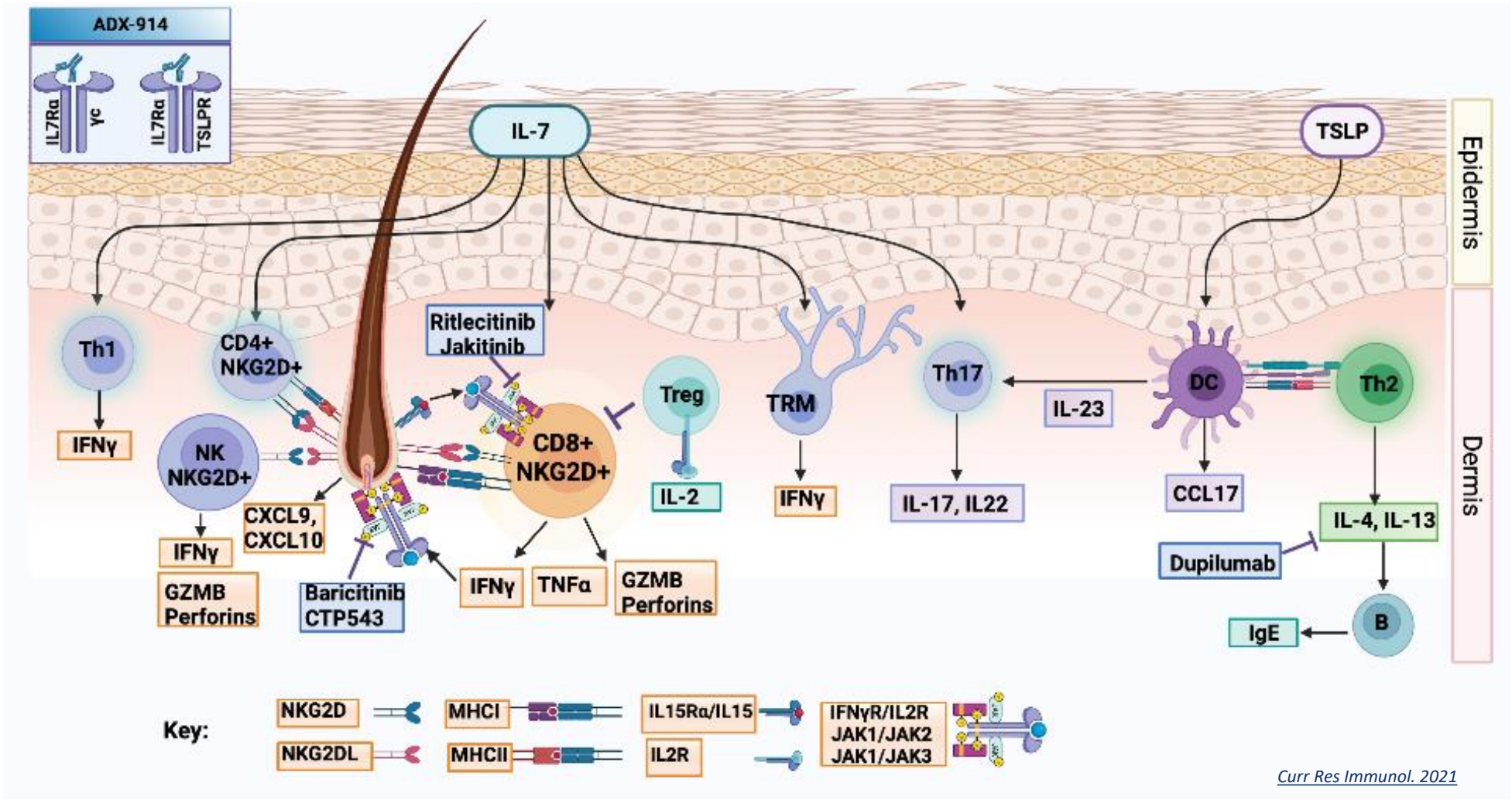


Existing therapies focused on early Th2-driven disease. Mature, chronic increasingly Th1-driven disease remains an unmet need

Adapted from Bieber, T. *Nat Rev Drug Disc 2022*

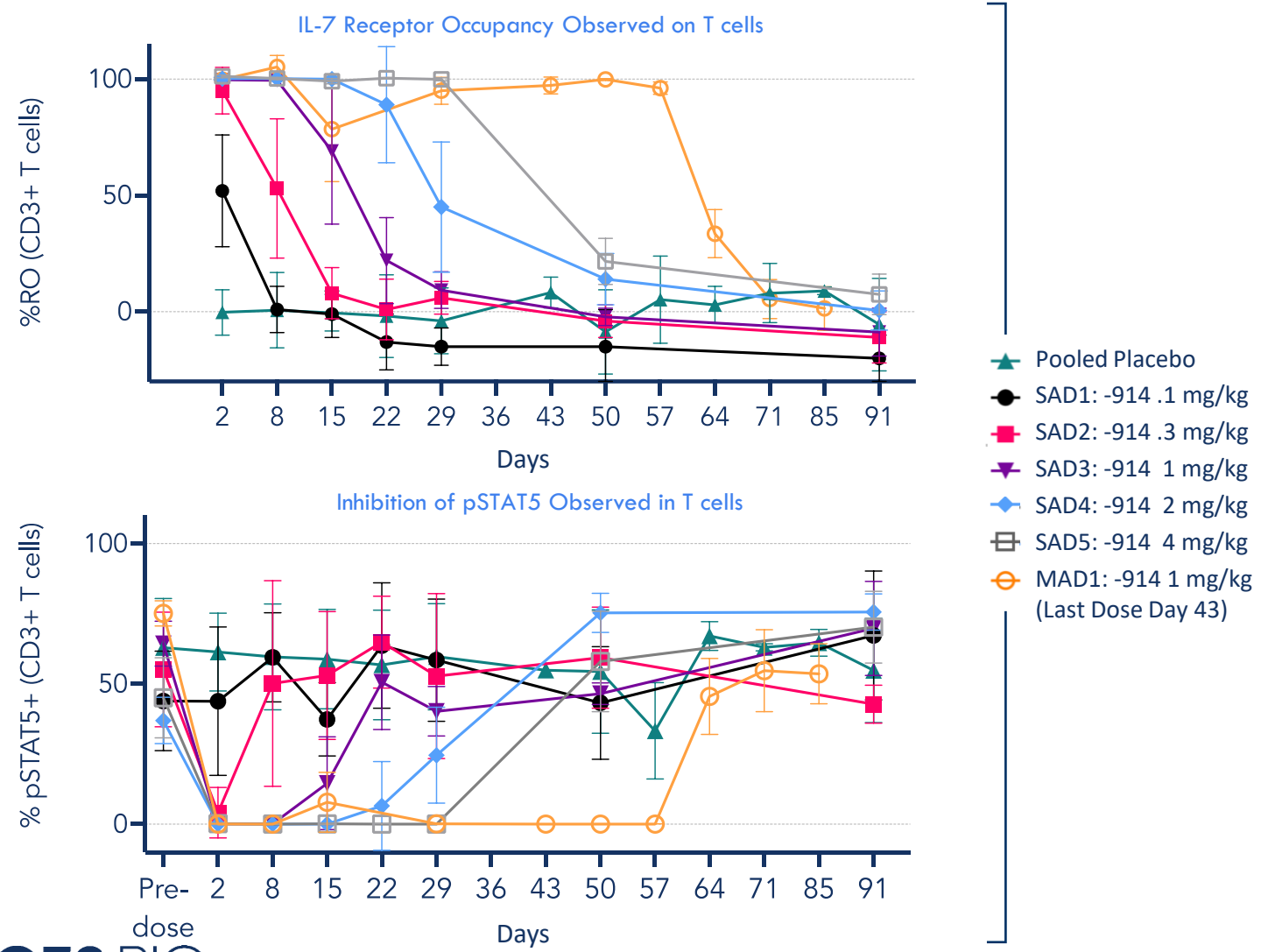
Bempikibart in AA: IL-7 Inhibition is Believed to Block CD8+ T cell-Driven Inflammation

Hair Follicle Immune Dysregulation in Alopecia



Curr Res Immunol. 2021

Bempikibart Phase 1 Clinical Data Supported Further Clinical Advancement



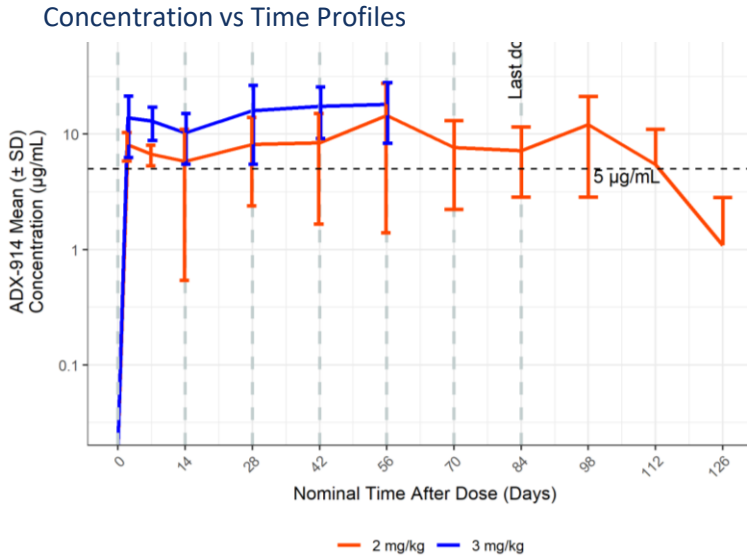
- Well tolerated up to 4 mg/kg
- Full RO & pSTAT5 inhibition at 1 mg/kg SC Q2W (MAD: 4 doses through Day 43)
- PK/PD profile evaluated in patients; full RO at < 3mg/kg SC observed
- Reversible decrease in T_{eff} cells and no effect on T_{regs} observed
- No ADA impact on pharmacology or safety observed
- Attenuation of T-cell dependent Ab response observed

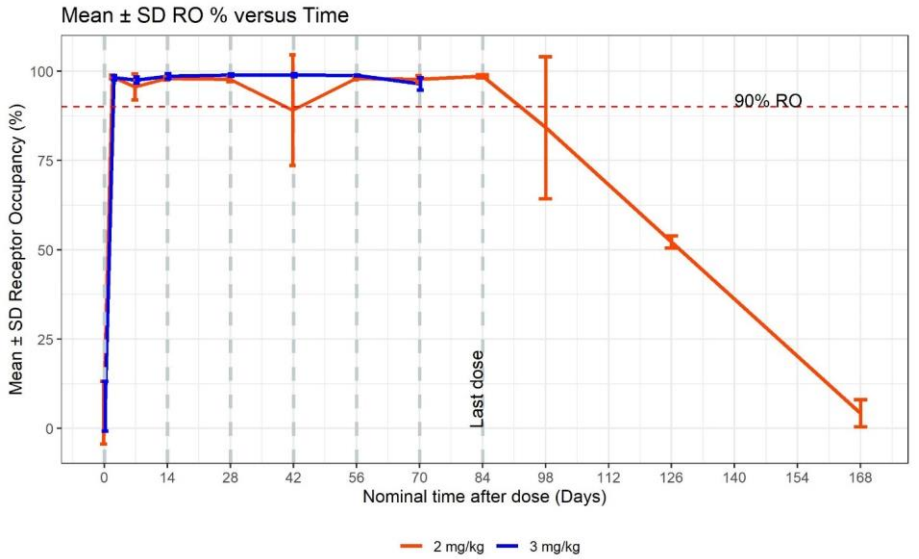
RO: Receptor occupancy; pSTAT5: phosphorylated STAT5; Q2W: once every 2 weeks; NHV: normal healthy volunteers; SC: subcutaneous; Teff: T effector; Treg: T regulatory; ADA: anti-drug antibody; Ab: antibody

Bempikibart Phase 2 AD Part A: Supported Selection of 200 mg SC Flat Dose (~2.7mg/kg) -- Third Party Reviewed PK; Trial Otherwise Remains Blinded

Part A Evaluated 2 mg/kg and 3 mg/kg Q2W SC which informed dose selection in Part B

- **PK/Dosing:** Both dose groups maintained target PK threshold > 5 ug/ml (dose predicted to achieve full target engagement in tissue); supports administration of 200 mg SC flat dosing (~2.7 mg/kg)[1] in AD Part B & AA
- **PD:** 100% of both dose groups achieved maximal RO (>90%) by day 3, maintained through entirety of dosing period (graph below)
- **Absolute lymphocyte counts:** No CTCAE grade ≥3 lymphopenia observed
- **ADA:** Low titer ADA, with no impact on PK observed
- **Safety:** To date, no lymphopenia associated AEs including viral infections observed
- **Efficacy:** Part A remains blinded. Part B blinded through topline data readout.





1. Equivalent to ~2.7 mg/kg for a 75 kg individual.

Bempikibart Phase 2 Clinical Trial in Atopic Dermatitis
ADX-914-202 SIGNAL-AD

Atopic Dermatitis

Atopic dermatitis is the most common chronic inflammatory skin disease

- Prevalence in children ranges from 2%-20%[1,2,3]
- Prevalence in adults ranges from 2% to 5%[1,2]
- Multiple pathways beyond the classical "T helper cell (TH)2" pathway are important in pathogenesis[4-7]

Despite recent therapeutic advances, a large AD population remains sub-optimally treated

- Dupilumab was a breakthrough and set benefit/risk precedent for systemic biologic therapy, but up to 30% of patients fail to achieve adequate disease control[8,9]
  - Does not address importance of Th1/Th17 pathway in more mature, chronic disease
- JAK inhibitors have shown efficacy, but also associated with significant adverse events (i.e. black box warnings)
- Development of remittive therapies gaining interest
  - ADX-914 has the potential to affect OX40L through TSLP and exert remittive effect via IL-7 axis

Design/Timeline

12-week treatment (n= ~120 patients in Part A and B), 12-week follow-up

- **Part A Key assessments: completed, but remains blinded**
  - Safety and PK
  - Evaluated two doses: 2 mg/kg and 3 mg/kg Q2W for dose selection in Part B and AA: 200mg SC (~2.7mg/kg) flat dose selected for Part B
- **Part B Key assessments: 200mg SC Q2W vs placebo (1:1)**
  - Primary: Mean % change from baseline in EASI score at week 14
  - Key Secondaries: Time to EASI change, mean % change from baseline in SCORAD, proportion of patients achieving EASI thresholds, proportion of patients achieving specified vIGA-AD improvements
  - Proportion of patients achieving an AD-IGA of 0 or 1 with a ≥2 grade improvement

Timeline: Enrollment completed; topline data expected in Q4'24

1. Weidinger et al. Nat Rev Dis Primers. 2018; 2. Bieber et al. Nat Rev Drug Discov 2022; 3. Based on studies conducted globally 4. Sims et al. Exp Dermatol 2021; 5.Thijs et al. J Allergy Clin Immunol 2017; 6. Gittler et al. J Allergy Clin Immunol 2012; 7. Czarnowicki et al. J Allergy Clin Immunol 2020. 8. Bieber et al. Nat Rev Drug Discov 2022; 9. Patrizi et al. J Dermatolog Treat 2021.

Bempikibart Phase 2 Clinical Trial in Alopecia Areata
ADX-914-203 SIGNAL-AA

Alopecia Areata

Alopecia Areata is common, and psychosocially debilitating; scalp and face commonly impacted

- Approximate prevalence of 2%, most before age 50
- Up to 40% of patients develop chronic disease; 10% progress to complete loss of scalp* and/or body hair**

Psychological comorbidities common and result in major impact on patients' lives

Despite recent JAK approvals, there remains significant medical need

- Current lack of options for inducing remission, avoiding life-long treatment (JAK inhibitors require chronic treatment and hair loss reoccurs with treatment cessation or taper)
- JAK inhibitors have shown efficacy, but also associated with significant adverse events (i.e. black box warnings)

Design/Timeline

24-week treatment (n= ~40 patients), 12-week follow-up

- **Key assessments: 200mg SC Q2W vs placebo (3:1)**
  - Primary: Mean % change from baseline in SALT score at Week 24
  - Key Secondaries: Time to SALT change, proportion of patients achieving SALT thresholds
  - Proportion of patients achieving an AA-IGA of 0 or 1 with a ≥2 point improvement
  - Change from Baseline in Clinician Reported Outcome (ClinRO) for Eyebrow and Eyelash Hair Loss

Timeline: Enrollment completed; topline data expected in Q4'24

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*Alopecia Totalis; **Alopecia Universalis

Tissue Targeted Platform: Building The Future of Complement Therapeutics

Proprietary Tissue-targeted Platform: Building The Future of Complement Therapeutics

The Evolution of Complement Therapeutics

1st Gen: Pioneer

Alexion

2nd Gen: Continued Systemic Applications

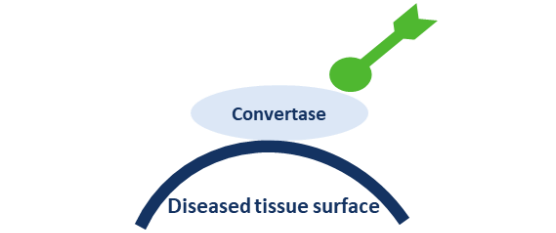
Apellis *InflaRx* *Kira*

Novartis *UCB* *Omeros*

Akari *Sanofi* *Alnylam*

The Future: Tissue Targeted

Q32 BIO



Directed inactivation in diseased tissue

Q32 Tissue-targeted Platform Value Proposition: Designed to Enable Clinical Profile Superior to Systemic Complement Inhibitors

The Unmet Need	The Opportunity
• **Limited activity:** Reliant on systemic blockade for impact on affected organ	• **Enhanced activity through tissue targeting:** Differentiated approach to driving efficacy by inactivating convertases directly at site of destruction
• **High doses, frequent administration required:** High abundance, rapid turnover of most target complement proteins	• **Reduced treatment burden**: SC route with QW dosing; potential for Q2W
• **Infection risk:** Complement plays critical role in combating infection; systemic blockade increases risk	• **Improved risk/benefit profile**: Designed to maximize therapeutic index while maintaining intact immune surveillance; broader indication potential

ADX-097
(C3d targeted antibody – fH_{1-5} fusion protein)

ADX-097: A Novel Tissue-Targeted Approach to Regulating the Complement System

ADX-097 Design: C3d antibody – fH_{1-5}

ADX-097 Construct: humanized anti-C3d mAb linked to two moieties of a negative regulatory protein (fH_{1-5})

- Designed to be active at the site of complement activation in the tissue without systemic impact
- Inactivation of alternative pathway convertases gains control of amplification loop and all 3 complement pathways

Clinical Data Completed to Date:

- Ph1 completed: supported dose selection and continued advancement
- 450mg SC QW selected: Ph1 confirmed SC dosing (with possibility to further reduce frequency), demonstrated exposures above predicted range for clinical activity, while below systemic inhibition

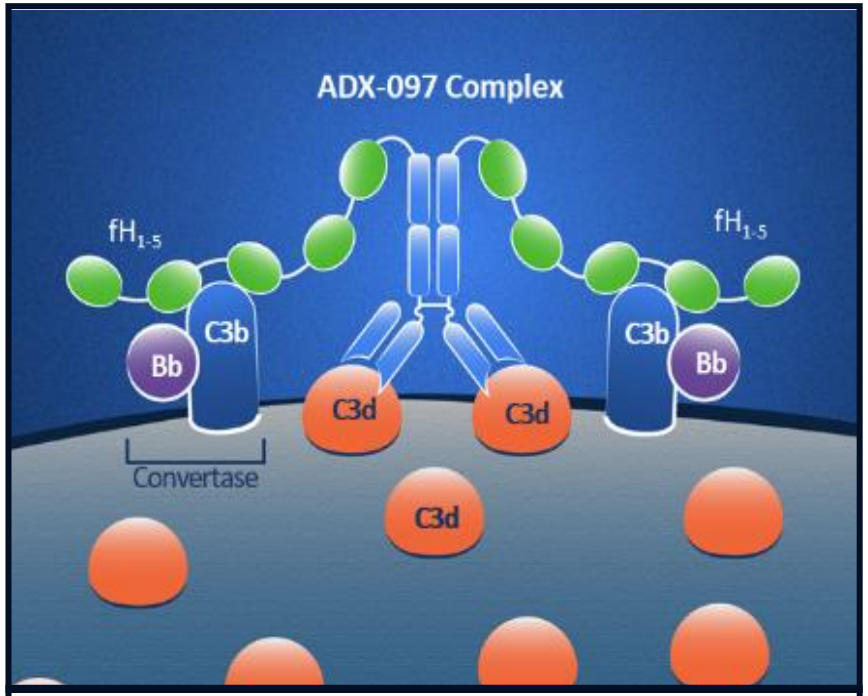
Topline Data Expected 2H'25

- Renal basket Ph2 topline data (initial data YE'24)
- AAV Ph2 Part A topline data

ADX-097 Lead Bivalent Fusion Protein: Designed with Unique MOA to Drive Localized, Complement Re-regulation For Enhanced Activity and Tolerability

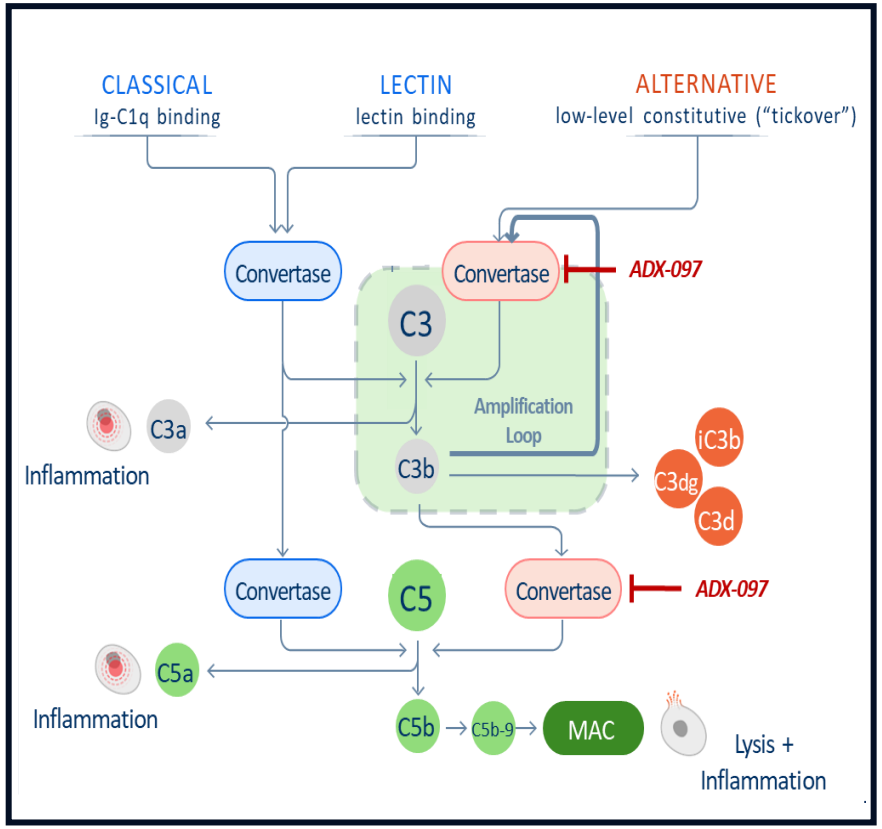
ADX-097 Design: C3d antibody – fH_{1-5}

Designed to be held at site of tissue complement activity allowing catalytic degradation of alternative pathway convertases



ADX-097: Fusion protein
Humanized anti-C3d mAb linked to two moieties of a negative regulatory protein (fH_{1-5})

Inactivation of alternative pathway convertases gains control of amplification loop and all 3 complement pathways



ADX-097 Preclinical and Ph1 Data: Robust Data Package Supports Desired PK and PD with Favorable Tolerability and Immunogenicity Profile

Preclinical Data

- Tissue distribution and target binding
- Durable (>7 days) tissue PK/PD after SC dosing
- Reduction in key proof of mechanism (POM)/proof of concept (POC) biomarkers including proteinuria and albuminuria
- >40X safety margin for planned Ph2 clinical dosing

Ph1 Clinical Data

- Favorable tolerability and good immunogenicity profile across all SAD/MAD doses
- Weekly SC dosing met desired exposures for predicted complete tissue inhibition (based on preclinical modeling) with no systemic inhibition
- Proximal POM supports in-vivo ADX-097 integrity

ADX-097 Preclinical Data: Supports POM, POC, PK/PD Dosing Model and Indication Rationale

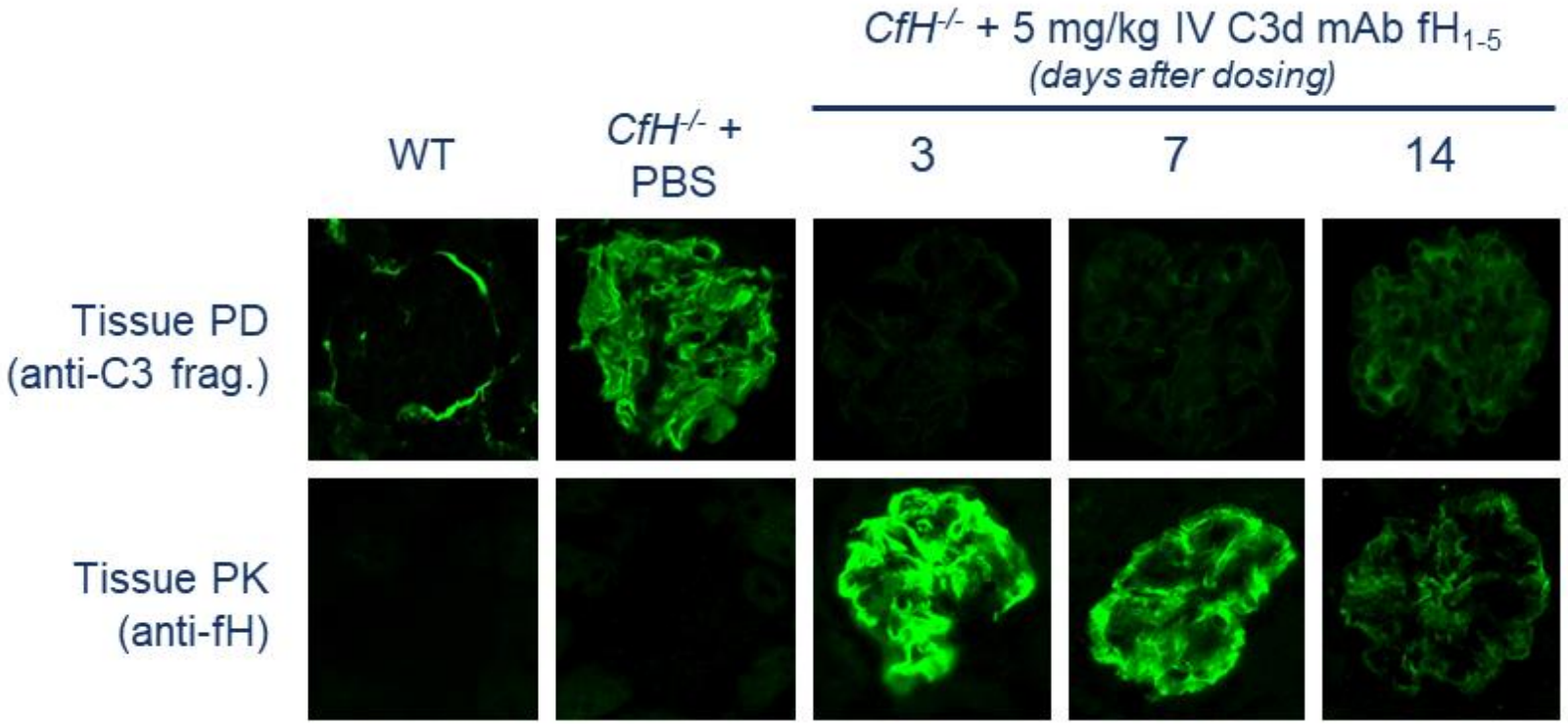
Milestone[1]	Organ System	Species	Model
Target validation in human disease	✓ Kidney ✓ Skin ✓ Liver	✓ Human	IHC of human disease biopsies (multiple disease-types for kidney, skin & liver)
Biodistribution of drug to tissue	✓ Kidney ✓ Skin ✓ Liver	✓ Mouse ✓ Rat ✓ NHP	fH -/- mice, EBA mouse model, PHN rat model, NHP UVB
Proof of Mechanism (POM): Durable inhibition of complement in tissue, absent systemic blockade	✓ Kidney ✓ Skin ✓ Liver	✓ Mouse ✓ Rat ✓ NHP	fH -/- mice, EBA mouse model, PHN rat model, NHP UVB
Proof of Concept (POC): Targeted activity at low mg/kg SC administration	✓ Kidney	✓ Rat	PHN rat model

Drug levels of 0.3 - 3.2 ug/ml predicted to provide maximal tissue targeted complement inhibition and activity based on preclinical data

1. Not all milestones were assessed in every organ system in every species

fH -/- Mouse Model of Human C3G with Uncontrolled Complement Activation: Showed Robust and Durable Tissue PK/PD in Absence of Circulating Inhibition of Complement

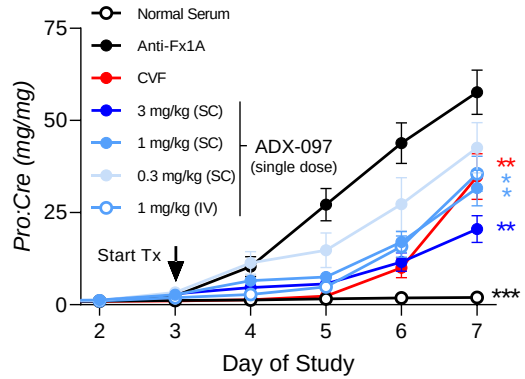
Potent Tissue PK/PD



- ✓ **Robust tissue PK/PD and activity at doses of 1 – 3 mg/kg, SC or IV**
- ✓ **Tissue PD EC90 = circulating concentration of 0.3 ug/mL**
- ✓ **Long term, durable kidney PK/PD in absence of systemic complement inhibiting activity**
- ✓ **Supports dosing every 1 to 2 weeks**

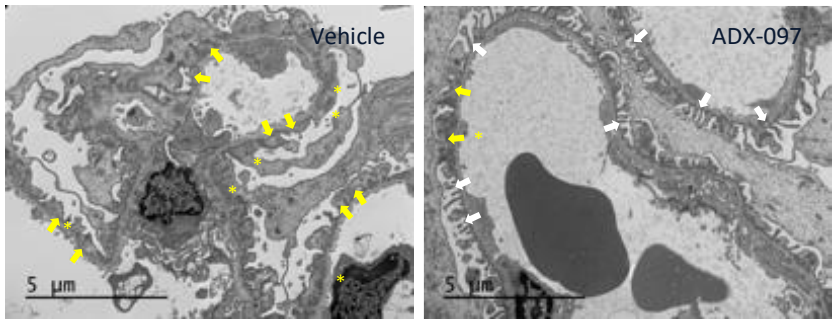
Passive Heymann Nephritis Rat Model of Human Membranous Nephropathy: Showed Robust Effect on POM and POC Endpoints

Proteinuria



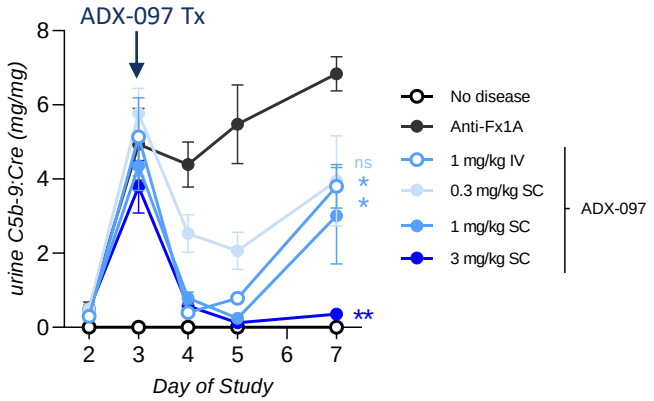
Potent inhibition of proteinuria equivalent to full systemic complement depletion (cobra venom factor)

Podocyte Structures



Podocyte Injury - Key driver of protein leakage in kidney disease

Urine sC5b-9



Potent inhibition of urine sC5b-9, a non-invasive approach to monitor complement inhibition in kidney

- ✓ **Robust tissue PK/PD and activity at doses of 1 – 3 mg/kg, SC or IV**
- ✓ **Tissue PD, urine sC5b-9 and proteinuria EC70-EC90 = circulating concentration of 0.3 ug/mL**
- ✓ **Protection of podocytes**

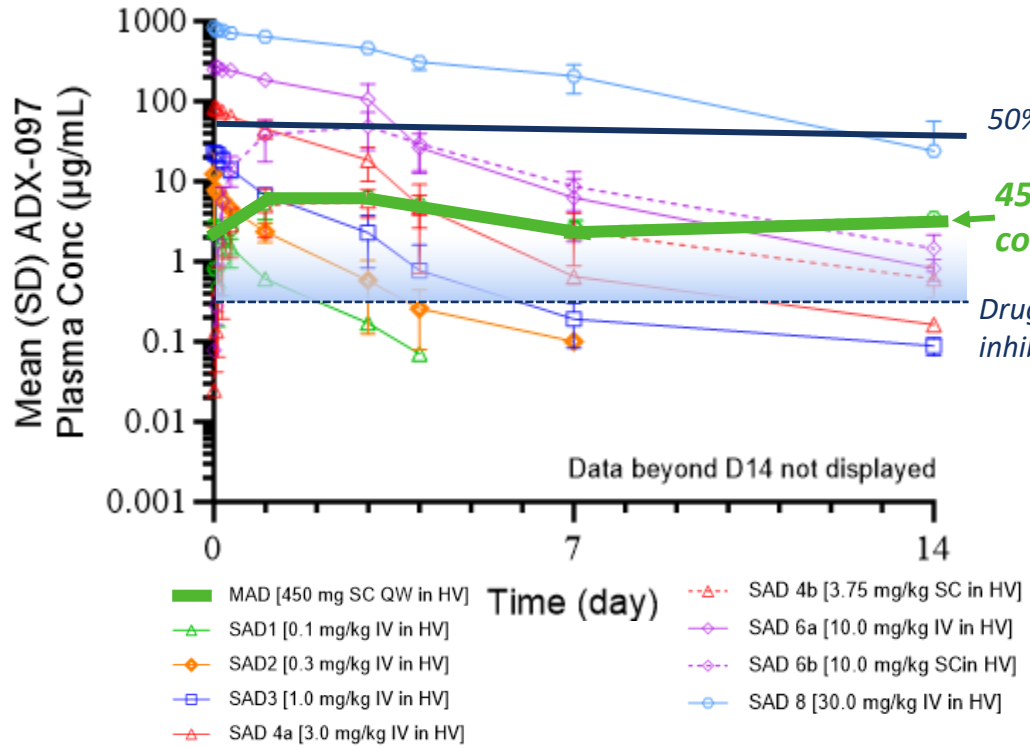
ADX-097-001 Phase 1 Study: Complete with Primary Goals Achieved

ADX-097-001 SAD/MAD (n= 56 Healthy Volunteers)
Explored Single Doses of 0.1 - 30 mg/kg IV and/or SC and 450 mg SC Multiple Dose Cohort (~6 mg/kg)

Primary Goals	Achieved	Results
Confirm planned Ph2 dose/route/schedule	✓	• Attained expected dose-dependent PK/PD • Once weekly SC dosing provided desired exposure for predicted complete tissue inhibition with no concomitant systemic inhibition
Evaluate proximal POM to establish *in-vivo* ADX-097 integrity	✓	• PK levels aligned with predicted Wieslab alternative pathway inhibition
Characterize safety profile	✓	• No serious or severe AEs or discontinuations due to AEs
Characterize immunogenicity risk	✓	• No AEs related to immunogenicity • Minimal anti-drug antibodies (ADA) detected across SAD/MAD; low level titers

ADX-097 Phase 1 PK Data: Weekly SC Dosing Met Desired Exposures for Predicted Complete Tissue Inhibition With No Concomitant Systemic Inhibition

Phase 2 Dose Identified: 450 mg SC Met Key Exposure Goals



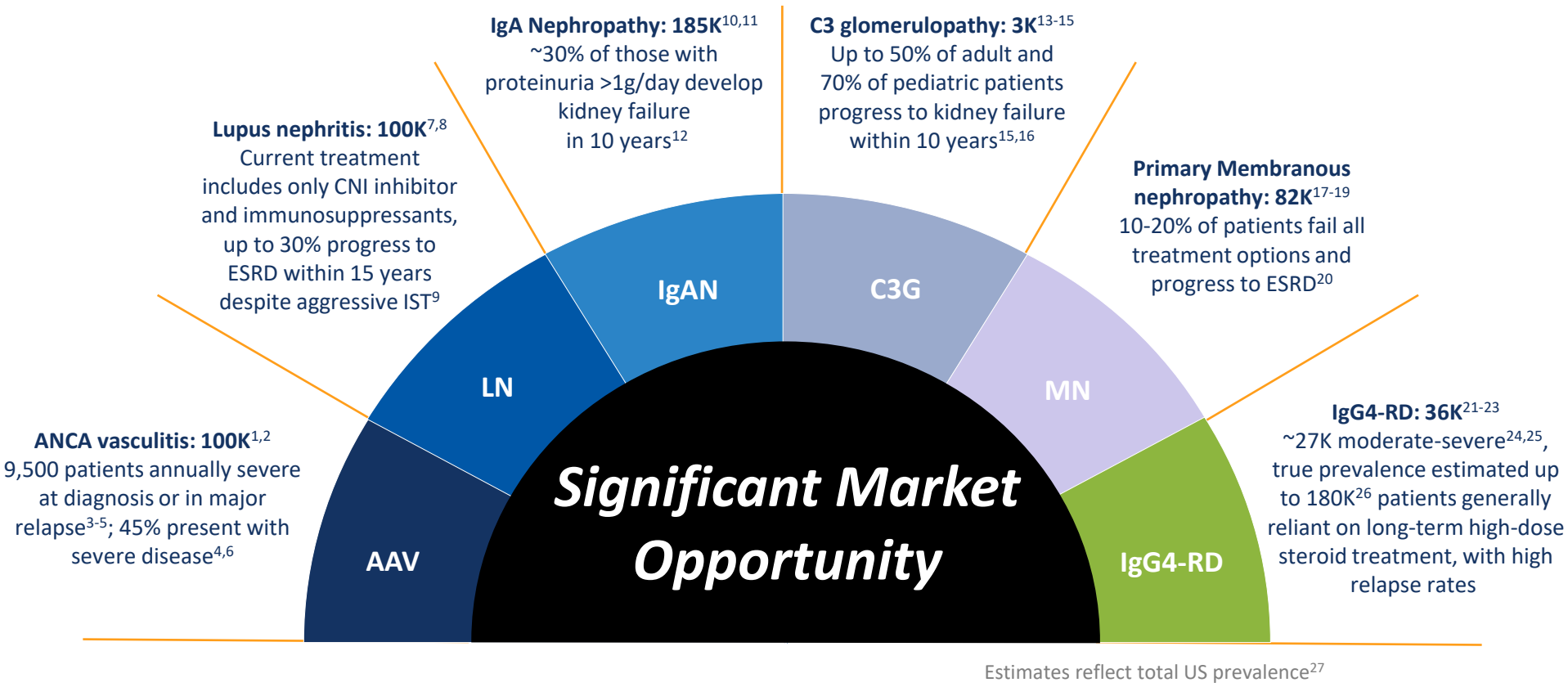
50% systemic complement inhibition

450mg SC achieved exposures below systemic inhibition and above conservative end of predicted range for clinical activity

Drug levels predicted to provide maximal tissue complement inhibition and activity based on mouse, rat and NHP preclinical data

Updated to include cohorts SAD 6b and SAD 8

ADX-097:
Significant Market Potential in Priority High Unmet Need Indications



Estimates reflect total US prevalence[27]

1. Watts et al. Nat Rev Rheum 2022; 2. Estimated using U.S. and Norway incidence study results, and Norway prevalence study results as applied to U.S. population; 3. Berti et al. Arthritis Rheum 2017; 4. Q32 qualitative research; 5. Specks et al. N Engl J Med. 2013; 6. Severe disease patients are those with a high risk of progression to ESRD, among other factors; 7. Hoover et al. Kidney Int 2016; 8. Pryor et al. Rheum Dis Clin North Am. 2021; 9. Pryor et al. Rheum Dis Clin North Am. 2021; 10. Kwon et al. J Health Econ Outcomes Res. 2021; 11. Swaminathan et al. Clin J Am Soc Nephrol 2006; 12. Berthoux FC, et al. Semin Neph 2008; 13. Bomback et al. Kidney Int. 2018. 14. Smith et al. Natur Rev Nephrol. 2019; 15. Servais et al. Kidney Int 2012; 16. Smith RJH et al. J. Am. Soc. Neph 2007; 17. Ronco et al. Nat Rev Dis Primers 2021; 18. Swaminathan et al. Clin J Am Soc Nephrol 2006; 19. Hanko et al. Nephrol Dial Transplant 2009; 20. Couser et al. Clin J Am Soc Neph 2017; 21. Umehara et al. Mod Rheum 2012; 22. Uchida et al. Int J Rheum 2012; 23. Estimated using Japan prevalence study results as applied to U.S. population; 24. Brito-Zerón et al. Medicine 2016; 25. Moderate-severe patients are those who require pharmacological, primarily glucocorticoid treatment; 26. ACR clinical guidelines 2018; 27. Based on 2020 Census population

LN, IgAN, C3G Basket:
Designed to Provide Building Blocks for Renal Expansion

Renal Disease (LN, IgAN, C3G)	Design/Timeline
Lupus Nephritis (LN) **6-fold mortality risk** increase vs general population[1,2] Up to 30% develop **kidney failure** requiring dialysis or kidney transplant **within 15 years of diagnosis**[3,4] **IgA Nephropathy (IgAN)** **Up to 40%** develop ESRD w/in 20 years of diagnosis[5,6], and patients have **10 years** reduced life expectancy[7,8] **~70% not adequately controlled** w/supportive care[5,9] **C3 glomerulopathy (C3G)** **Up to 50% of adult, 70% of pediatric** patients progress to **kidney failure** within 10 years[10-13] **>70%** experience **recurring disease**; ~50% experience allograft loss w/in 10 years of kidney transplant[10,14-17]	**Basket design (n= up to 30), 24-week treatment** • Designed to assess safety, tissue pharmacology and magnitude/timing of treatment effect with focused dose-ranging • Open-label with interim data readouts • SC dosing with duration of treatment TBD (prior regulatory discussions support up to 24 weeks) • Key assessments: Drug localization and impact in tissue, biomarkers (including surrogate endpoint biomarkers proteinuria and eGFR) for assessment of ADX-097 activity • Anticipated to provide data for key regulatory discussions **Planning to initiate trial in 1H'24, with topline results expected in 2H'25 and initial open-label data by YE'24**

1. Mahajan et al. Lupus 2020; 2. Cervera et al. Medicine 2002; 3. Maroz et al. Am J Med Sci 2013. 4. Ward et al. J Rheumatol 2009; 5. Habas et al. Medicine (Baltimore) 2022. 6. Berthoux et al. Semin Nephrol 2008; 7. Pitcher et al. Clin Jour of Amer Soc Neph 2023. 8. Hastings et al. Kidney Int Rep 2018; 9. Raun et al. N Engl J Med 2015; 10. Heiderscheit et al. Am J Med Genet C Semin Med Genet 2022. 11. Smith et al. J Am Soc Nephrol 2007; 12. Servais et al. Kidney Int 2012; 13. Rabasco et al. Kidney Int 2015; 14. Smith et al. Nat Rev Nephrol 2019. 15. Welte et al. BMC Nephrology 2018; 16. Salvadori et al. WJT 2016. 17. Regunathan-Shenk et al. AJKD 2019 18. Hoover et al. Kidney Int 2016; 19. Pryor et al. Rheum Dis Clin North Am. 2021; 20. Braun et al. Int Urol Nephrol 2011; 21. McQuarry et al. Kidney Int 2013; 22. Bomback et al. Kidney Int. 2018.

AAV Two-part Phase 2:
Expected to Deliver Key Biomarker Data & Evaluation of Reduction of Steroids

AAV	Design/Timeline
More effective induction and maintenance • With treatment, 5-year mortality 10-30% overall[1-3] • 5-year mortality with renal disease – 20-50%[1-3] • Relapse is substantial issue: Up to 50% of patients relapse within 5 years, often 12-18 months of IST discontinuation[4-8] **Reduction/Elimination of Glucocorticoids (GCs)** • IST, particularly GC side effects, account for significant early treatment related morbidity and mortality, primarily due to infection[9] • Avacopan approved in 2021, but due to limitations in development program, label states, “does not eliminate GC use” and guides to avoid use in "active, serious infection“[10]	**Part A: 12-week treatment (n= up to 20 patients)** • Designed to evaluate safety and early treatment effect • IV loading dose followed by SC dosing as adjunct to SOC • Key assessments: ADX-097 localization and impact in tissue, BVAS, biomarkers for early assessment of ADX-097 activity • Anticipated to provide data for key regulatory discussions • Ph2 design informed by avacopan Ad Com roadmap **Planning to initiate trial in 1H’25; topline results expected in 2H’25**

1. Tan et al. Ann Rheum Dis 2017; 2. Heil et al. RMD Open 2017; 3. Q32 internal estimates. 4. Samman et al. Int J Rheumatol 2021; 5. Tan et al. Ann Rheum Dis 2017; 6. Walsh et al N Engl J Med 2020; 7. Demiselle et al. Ann Intensive Care 2017; 8. Kitching et al. Nat Rev Dis Primers 2020; 9. Titeca-Beauport et al. BMC Nephrol 2018 10. Tavneos FDA label. 11. Berti et al. Arthritis Rheumatol 2017; 12. Watts et al. Nephrol Dial Transplant 2015. 13. Watts et al. Nat Rev Rheumatol 2022

IST: immunosuppressive therapy
BVAS: Birmingham Vasculitis Activity Score (planned Phase 3 endpoint)

Q32 Bio Has Significant Potential to Unlock Near-term Value-creation

Runway through multiple Phase 2 clinical readouts and into mid-2026

Financial Overview

- Q1 cash balance of $135.3M, providing expected cash runway **to mid 2026**
- Approximately 11.9M shares outstanding

Merger Supports Potential Achievement of Numerous Anticipated Milestones

- **4Q'24:** AD Ph2 topline results (including 14-week placebo-controlled efficacy data)
- **4Q'24:** AA Ph2 topline results (including 24-week placebo-controlled efficacy data)
- **2H25:** Renal basket Ph2 topline results (initial data YE'24)
- **2H25:** AAV Ph2 Part A topline results