Building The Future of Immune Therapeutics

Company Overview

December 2024

Forward Looking Statements

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This presentation contains forward-looking statements within the meaning of the U.S. Private Securities Litigation Reform Act of 1995. Forward-looking statements can be identified by words such as "may," "will," "should," "would," "expect," "anticipate," "plan," "likely," "believe," "estimate," "project," "intend," "potential," and similar expressions regarding future periods. These forward-looking statements include, but are not limited to, our beliefs, observations, expectations and assumptions regarding the topline data from the SIGNAL-AA Phase 2a and the safety, tolerability, clinical activity including biomarker data, potential efficacy and potential benefits of bempikibart, plans and expectations for Part B of the SIGNAL-AA Phase 2a clinical trial statements regarding expected cash and the sufficiency of the Company's cash to fund operations into mid- 2026, the expectations surrounding the potential, safety, efficacy, and regulatory and clinical progress of Q32's product candidates, including bempikibart and ADX-097, and anticipated milestones, data readouts and timing, among others. Statements that are not historical facts are forward-looking statements. Forward-looking statements are based on current beliefs and assumptions that are subject to risks and uncertainties and are not guarantees of future performance. Actual results could differ materially from those contained in any forward-looking statement as a result of various factors, including, without limitation: the Company's need for additional funding, which may not be available; failure to identify additional product candidates and develop or commercialize marketable products; the early stage of the Company's development efforts; potential unforeseen events during clinical trials could cause delays or other adverse consequences; risks relating to the regulatory approval process; interim, topline and preliminary data may change as more patient data become available, and are subject to audit and verification procedures that could result in material changes in the final data; Q32's product candidates may cause serious adverse side effects; inability to maintain our collaborations, or the failure of these collaborations; our reliance on third parties, including for the manufacture of materials for our research programs, preclinical and clinical studies; failure to obtain U.S. or international marketing approval; ongoing regulatory obligations; effects of significant competition; unfavorable pricing regulations, third-party reimbursement practices or healthcare reform initiatives; product liability lawsuits; securities class action litigation; the impact of the COVID-19 pandemic and general economic conditions on our business and operations, including our preclinical studies and clinical trials; the possibility of system failures or security breaches; risks relating to intellectual property and our ability to protect our patents and other proprietary rights; significant costs incurred as a result of operating as a public company; as well as those risk and uncertainties set forth more fully under the caption "Risk Factors" in our Quarterly Report on Form 10-Q for the quarter ended September 30, 2024, as well as other risks detailed in our subsequent filings with the United States Securities and Exchange Commission. Any forward-looking statement made by us is based only on information currently available to us and speaks only as of the date on which it is made. We undertake no obligation to publicly update any forward-looking statement, whether written or oral, that may be made from time to time, whether as a result of new information, future developments or otherwise.

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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; potent inhibition observed by clinical biomarkers, with potential to treat both Th1 and Th2 mediated diseases • Ph2a data show attractive PK/PD profile, favorable safety and tolerability profile w/Q2W subcutaneous dosing • SIGNAL-AA demonstrated clinical activity of bempikibart in patients with alopecia areata (AA), including improvement from baseline on SALT score and meaningful achievement of SALT-20 response • Currently in ongoing Ph2 clinical trials: Alopecia Areata (AA) advancing into Part B
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 • Currently in a Ph2 renal basket trial
Near Term Value Creation Potential	• **1H'25 – Bempikibart AA Ph2a:** Initiate enrollment in SIGNAL-AA Part B • **1H'25 – ADX-097 Renal basket Ph2:** Initial data • **2H'25 – ADX-097 Renal basket Ph2:** Topline results
Exceptional Team and Investors	• Management team with extensive public biotech experience • Deep complement therapeutics and inflammatory/autoimmune expertise

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

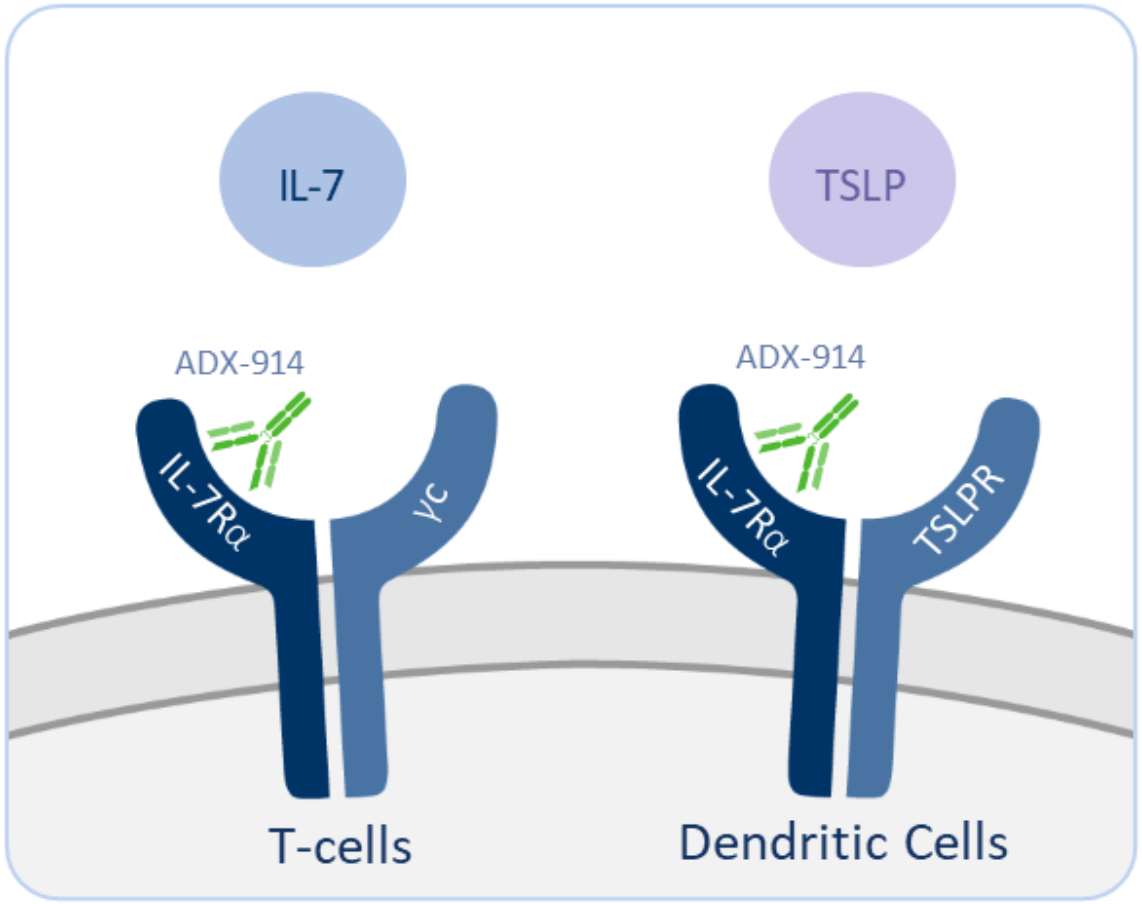
Program	Indication	Discovery/ Preclinical	Phase 1	Phase 2	Anticipated Milestones
IL-7/TSLP PROGRAM					
Bempikibart (ADX-914)[1]	**Alopecia Areata**				***Initiate Part B enrollment 1H'25***
COMPLEMENT INHIBITOR PLATFORM					
ADX-097	**Renal Basket (IgAN, LN, C3G)**				***Topline results 2H'25***

Note: IgAN = IgA Nephropathy; LN = Lupus Nephritis; C3G = C3 Glomerulopathy.

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

Bempikibart: Investigational Therapy for T-cell Mediated Inflammatory and Autoimmune Diseases With Demonstrated Clinical Activity

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 Generated to Date

- Ph1: Durable SC PK/PD and tolerability
- Ph2 AA Part A: Demonstrated encouraging clinical activity; well-tolerated safety profile; PK/PD demonstrated desired exposures, target engagement and inhibition of Th2 and Th1 biomarkers
- Ph2 AD: Well-tolerated safety profile; PK/PD demonstrated desired exposures, target engagement and inhibition of Th2 and Th1 biomarkers

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

Bempikibart Shows Potential to be Best in Class IL-7Rα Antibody

	Bempikibart **Q32 Bio** (IgG1 Effector-less)	**Lusvertikimab (OSE-127)** **OSE** (IgG4)	**ZB-168** **Zura Bio (formerly Pfizer)** (IgG1)	**GSK-2618960** **GSK** (IgG1)
Active in development	✓	✓	**Not currently funded[1]**	✗
Antagonist	✓	✓	--	✗
PK/PD supports current single-injection SC formulation	✓	✗	✗	✗
Fully human	✓	✗	✓	✗
Antibody generation	Medarex (fully human)	Rat mAb (humanized)	Phage library	Murine mAb (humanized)
IL-7R binding on cells ($EC_{50,}$ nM)[2]	0.08	0.24	0.04	0.32
IL-7Rα binding affinity, biacore (K_D, nM)[2]	0.09	0.16	0.13	0.23
Inhibition of IL-7 induced pSTAT5 in T-cells (IC_{50} nM)[2]	0.22	0.31	0.37	0.41
Inhibition of TSLP induced signaling in monocytes (IC_{50} nM)[2]	2.88	1.07	0.20	7.47

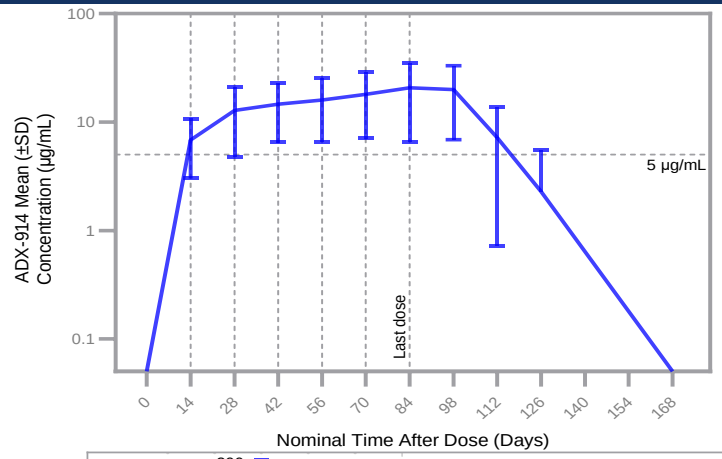
Bempikibart at 200 mg clinically demonstrated potent IL-7 and TSLP inhibition via changes in Th2 biomarkers and T-cells[3]

[1]Zura Bio, https://investors.zurabio.com/news-events/presentations; development dependent on additional financing and pending topline data from other programs
[2]Company data; data were generated by Q32 Bio in side-by-side assay of bempikibart vs comparator IL-7Rα mAb analogues
[3]Results from Phase 2a SIGNAL-AA and SIGNAL-AD clinical trials

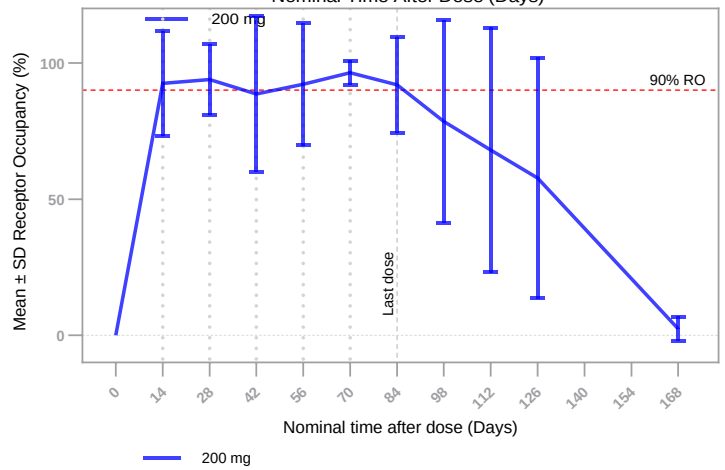
Favorable PK and Receptor Occupancy (RO) Achieved in Phase 2a Clinical Trials

SIGNAL-AD

PK

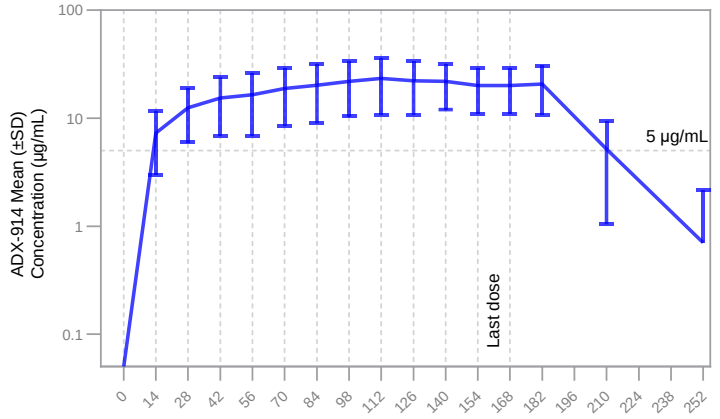


RO

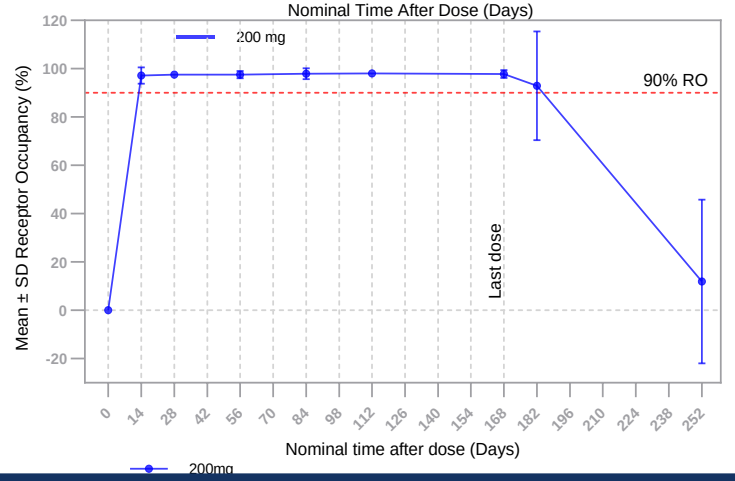


SIGNAL-AA

PK



RO



Bempikibart 200mg achieved >5µg/mL mean concentration and >90% RO in 85%-90% of patients, consistent with Phase 1 results

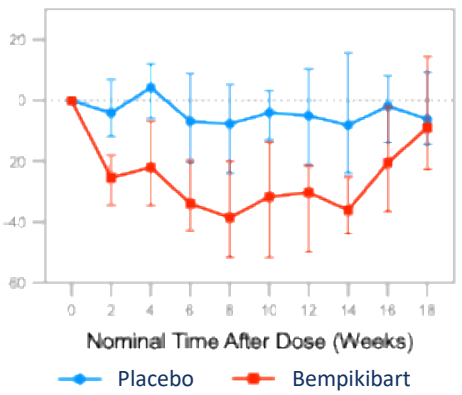
Figures depict PK and RO for all enrolled patients in SIGNAL-AD and SIGNAL-AA except samples impacted by missed dose (PK) or samples impacted by missed dose or assay deviation (RO)

Biomarker Results in SIGNAL-AD: Substantial Reductions Observed in Phase 2a Clinical Trials Suggesting Potent Inhibition of TSLP and IL-7 Mediated Signaling

Th2 Biomarkers: SIGNAL-AD

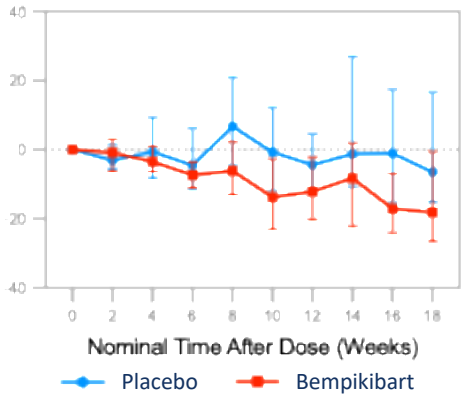
Median Changes Over Time (95% CI)

TARC



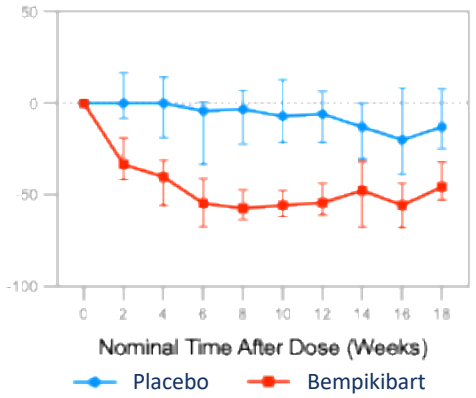
Statistically significant difference
wks 6-10 ($p < 0.05$)

IgE



Treatment resulted in up to -18% reduction at wk 18

Eosinophils

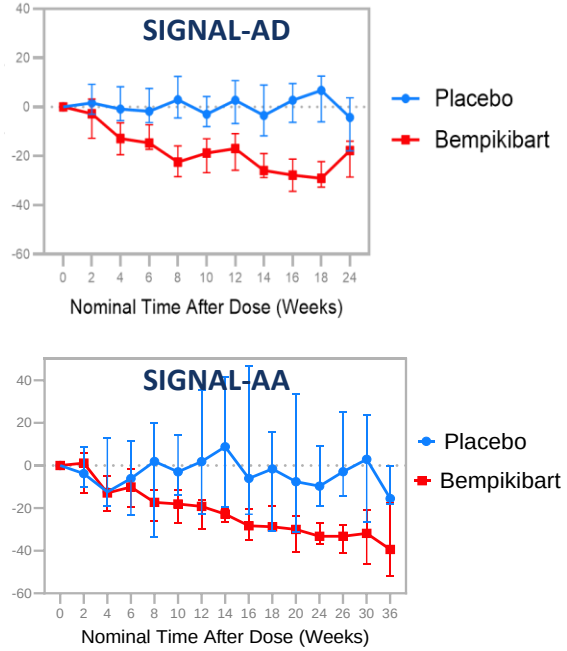


Statistically significant difference
wks 2-12, 16, 18 ($p < 0.05$)

Th1 Biomarkers: SIGNAL AD and AA

Median Changes Over Time (95% CI)

Absolute Lymphocytes



SIGNAL-AD: significant difference wks 4-18 ($p < 0.05$); SIGNAL-AA: significant difference wks 10, 24, 26 ($p < 0.05$)

Patients treated for 12 weeks in SIGNAL-AD, 24 weeks in SIGNAL-AA; biomarker and absolute lymphocyte analyses included all subjects, excluding samples impacted by protocol deviations

SIGNAL-AA: Bempikibart Phase 2a Clinical Trial in Alopecia Areata

Alopecia Areata

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

- Autoimmune disease, often associated with atopic disorders (atopic dermatitis, asthma, allergic rhinitis)
- Affects ~2% of the population, often manifesting before age 50
- Up to 40% become chronic, including complete loss of scalp* and/or body hair**, severity of disease and long duration of episode each associated with more gradual and lower rates of treatment response

Despite JAKi 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

Part A and B

- **Part A (n=44) Key Assessments: 200mg SC Q2W vs placebo (3:1) 24-week treatment (completed), 12-week follow up:**
  - Primary: Mean % change from baseline in SALT score at Week 24
  - Key Secondaries: Time to SALT change, proportion of patients achieving SALT thresholds
  - Change from Baseline in Clinician Reported Outcome (ClinRO) for Eyebrow and Eyelash Hair Loss
- **Planned Part B:**
  - Further evaluate bempikibart in AA to expand upon encouraging activity observed to date
  - Intend to enroll ~20 additional patients in an open-label expansion, expected to include a loading regimen
  - Changes in SALT from baseline
  - **Timeline: Enrollment expected to initiate in 1H25**

*Alopecia Totalis; **Alopecia Universalis

SIGNAL-AA: Baseline Characteristics

mITT (n=44)		
	Bempikibart 200 mg (n=33)	**Placebo (n=11)**
Gender (n, %)	Female (27, 81.8%)	Female (7, 63.6%)
Age (years, Mean ± SD)	48.8 ± 10.2	47.1 ± 14.2
Race (n, %)	White (19, 57.6%) Black /African American (10, 30.3%) American Indian/Alaska Native (1, 3.0%) Asian (1, 3.0%) Missing (2, 6.0%)	White (4, 36.4%) Black/African American (6, 54.5%) Asian (1, 9.1%)
Body weight (kg, Mean ± SD)	82.7 ± 13.9	85.1 ± 16.9
Baseline SALT Scores (Mean ± SD)	75.0 ± 20.3	75.5 ± 21.6
Duration of current episode (months Mean ± SD)	68.5 ± 36.2	51.7 ± 36.5

Revised Per Protocol[1] (n=27)		
	Bempikibart 200 mg (n=23)	**Placebo (n=4)**
Gender (n, %)	Female (18, 78.3%)	Female (2, 50.0%)
Age (years, Mean ± SD)	47.7 ± 11.3	59.8 ± 11.9
Race (n, %)	White (14, 60.9%) Black/African American (7, 30.4%) Other (2, 8.7%)	White (3, 75.0%) Black/African American (1, 25.0%)
Body weight (kg, Mean ± SD)	81.9 ± 14.2	82.3 ± 12.2
Baseline SALT Scores (Mean ± SD)	75.4 ± 20.7	88.4 ± 22.5
Duration of current episode (months, Mean ± SD)	58 ± 37.2	36.5 ± 21.2

[1] Table reflects Revised Per-Protocol Population (defined as pre-specified per-protocol population removing 3 placebo patients from one site excluded for marked protocol violations of entry criteria)

SIGNAL-AA: Key Efficacy Findings

Endpoint (Post-Hoc Analysis)	Bempikibart 200 mg (N=23)	Placebo (N=4)
Mean reduction in SALT score (Week 24)[1] *Wilcoxon Rank Sum p-value 0.045*[2]	16%	2%
SALT-20 (Week 24)	9%	0%
SALT-20 (Week 26)	13%	0%

[1]Table reflects Revised Per-Protocol Population. Results for Revised mITT Population (defined as pre-specified mITT population removing 3 patients with no evaluable post-baseline SALT score and 3 placebo patients from one site excluded for marked protocol violations of entry criteria): 12% bempikibart (n=32) vs. 5% placebo (n= 6), p-value NS (not shown on table)

[2]Due to resulting sample size following removal of the excluded site patients, normality and equal variance assumptions were not met for the planned statistical analyses. Given lack of normality caused by small sample size, Wilcoxon Rank Sum test was selected as most appropriate to compare the responses in each group (p= 0.045). A randomized permutation test with 10,000 permutations further confirmed the statistical significance of treated response over placebo by Wilcoxon Rank-Sum test (p=0.0432). Welch's t-test was also considered (p-value of 0.0318) assuming normality to be met with a larger sample size

SIGNAL-AA: SALT Improvement Over Time

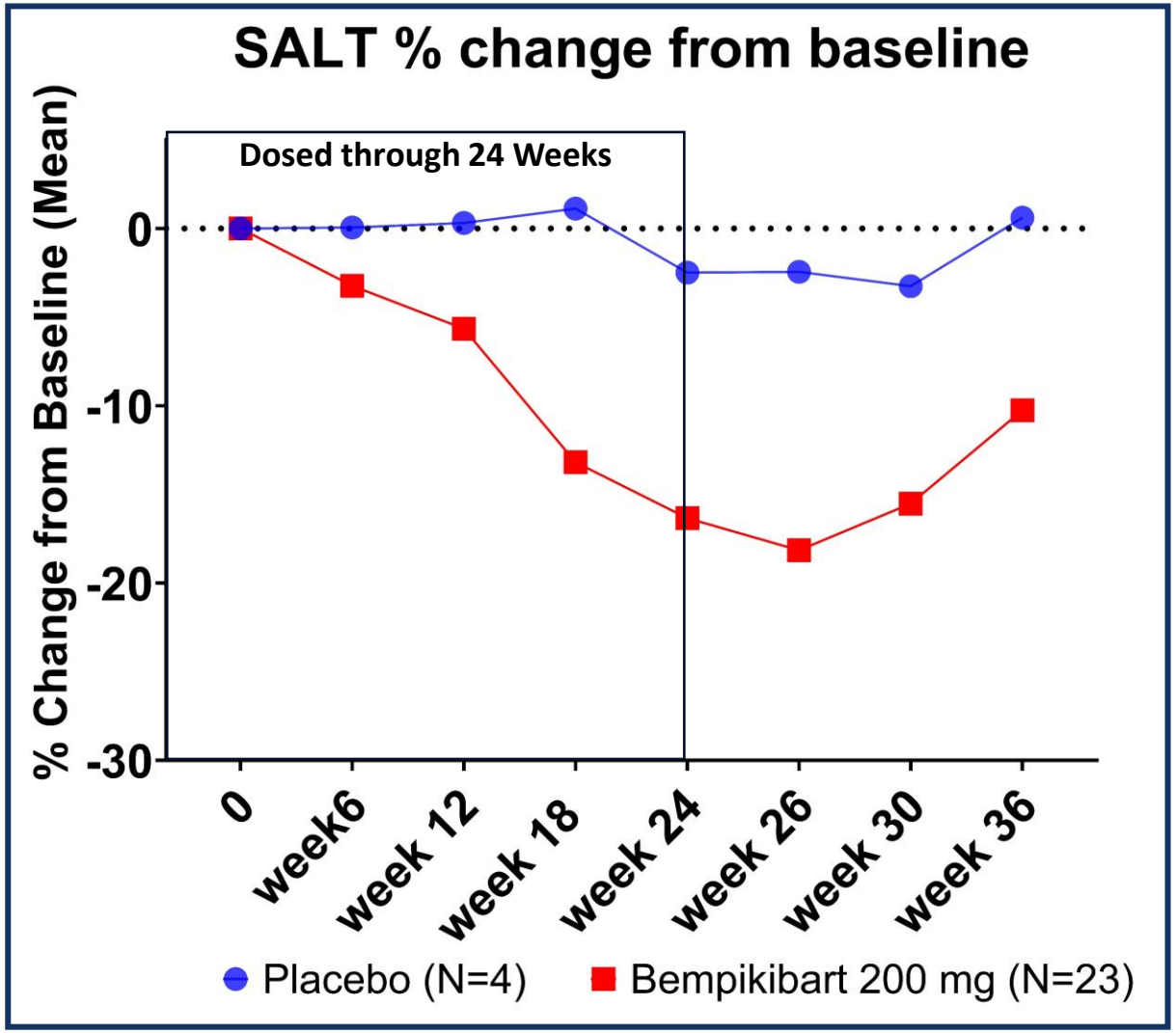


Figure reflects Revised Per-Protocol Population

KEY FINDINGS:

Clinical Activity Observed in SIGNAL-AA

- Mean change in SALT 16% vs 2% for placebo (Week 24)
- Mean change in SALT 18% vs 2% for placebo (Week 26)
- SALT-20 achieved in 9% at Week 24, 13% at Week 26

Activity Observed Despite Difficult to Treat Population

- Mean duration of current episode 58 months (~5 yrs)
- Duration of current episode (>4 yrs) associated with more gradual responders, late responders, and lower percentage of patients achieving SALT-20[1]

1 King, B World Congress of Dermatology 2023

SIGNAL-AA: Change in SALT Score by Patient at Week 24

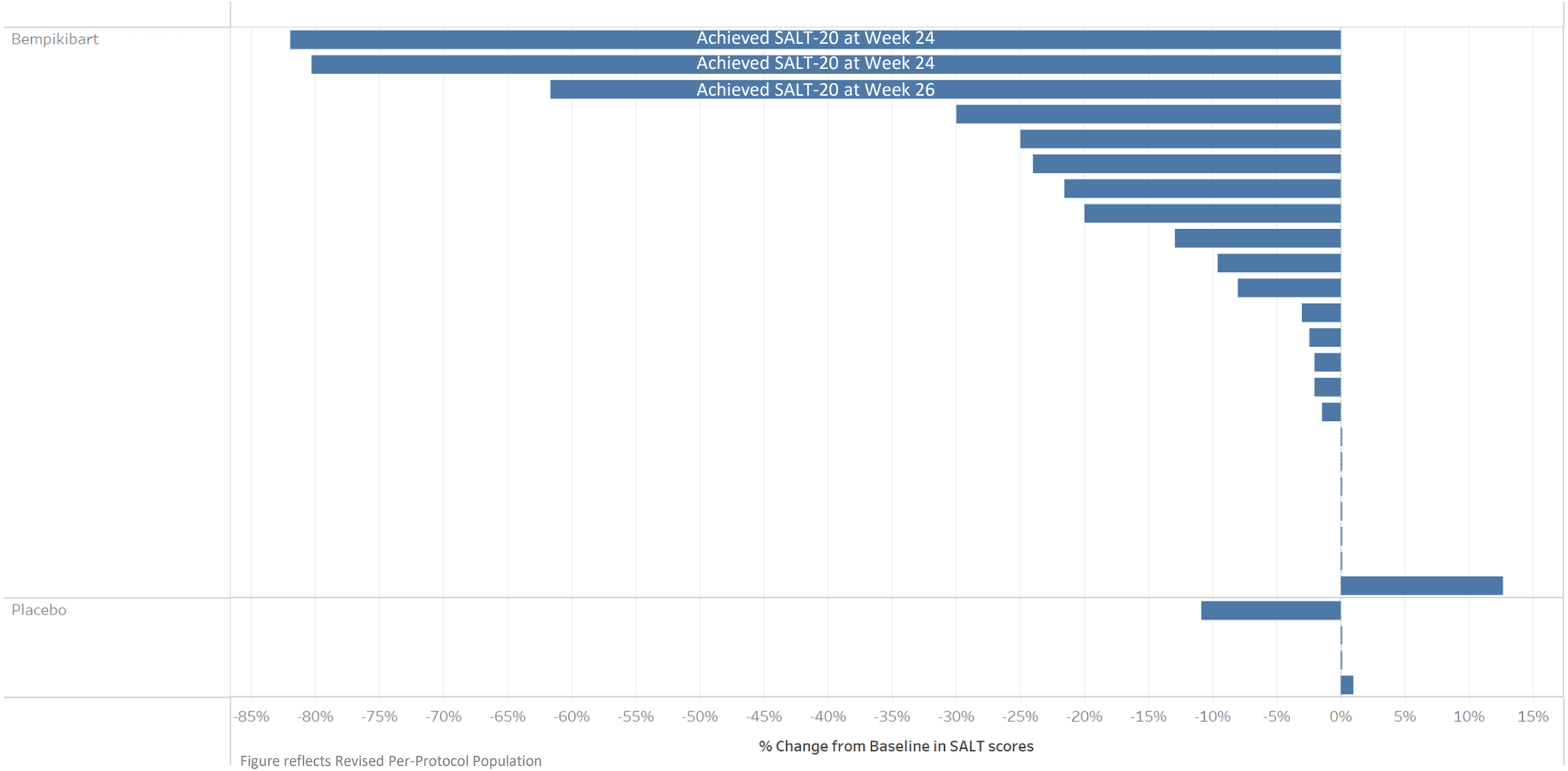


Figure reflects Revised Per-Protocol Population

SIGNAL-AA and SIGNAL-AD: Favorable Safety and Tolerability Profile

	Bempikibart 200 mg (N=96) n (%) [E]	Placebo (N=69) n (%) [E]
Participants with at least one TEAE	55 (57.2) [154]	30 (43.5) [76]
Participants with at least one TEAE by greatest reported relationship with study treatment [a]		
Not related	28 (29.2) [43]	23 (33.3) [47]
Related	27 (28.1) [68]	7 (10.1) [12]
Participants with at least one TEAE by worst reported severity CTCAE grade [b]		
Grade 1 - Mild	30 (31.2) [49]	13 (18.8) [27]
Grade 2 - Moderate	20 (20.8) [21]	15 (21.7) [22]
Grade 3 - Severe	4 (4.1) [6]	2(2.9) [2]
Grade 4 - Life threatening	1 (1) [1]	0 [0]
Grade 5 - Death	0 [0]	0 [0]

No Grade 3 or Higher Bempikibart Related Adverse Events

Grade 3 (n=6): 5 not related; 1 possibly related to study treatment (placebo arm)

Grade 4 (n=1): 0 related to study treatment

SIGNAL-AA and SIGNAL-AD: Adverse Events of Special Interest (Infections)

SIGNAL-AD PART A & B

	Bempikibart N=63		Placebo N=58	
	Related n (%)	Not Related n (%)	Related n (%)	Not Related n (%)
Infections and infestations	3 (4.8)	12 (19.0)	0	9 (15.5)
Events				
Upper respiratory tract infection	1 (1.6)	5 (7.9)	0	2 (3.4)
Nasopharyngitis	0	4 (6.3)	0	2 (3.4)
Herpes virus infection	0	1 (1.6)	0	1 (1.7)
Acute sinusitis	0	1 (1.6)	0	0
COVID-19	0	1	0	1 (1.7)
Candida infection	0	1 (1.6)	0	0
Conjunctivitis	0	1 (1.6)	0	0
Ear infection	1 (1.6)	0	0	0
Folliculitis	1 (1.6)	0	0	0
Gastrointestinal viral infection	0	1 (1.6)	0	0
Gastroenteritis	0	0	0	1 (1.7)
Hordeolum	0	1 (1.6)	0	0
Influenza	0	0	0	1 (1.7)
Oral herpes	0	1 (1.6)	0	0
Otitis media	0	1 (1.6)	0	0
Paronychia	0	0	0	1 (1.7)
Skin infection	0	0	0	1 (1.7)
Streptococcal infection	0	1 (1.6)	0	0
Urinary tract infection	0	2 (3.2)	0	1 (1.7)

n=1 Lymphocyte Count Decreased SIGNAL AD; Grade 2

SIGNAL-AA

	Bempikibart N=33		Placebo N=11	
	Not Related n (%)	Related n (%)	Not Related n (%)	Related n (%)
Infections and infestations	12 (36.4)	1 (3.0)	1 (9.1)	2 (18.2)
Events				
Urinary tract infection	3 (9.1)	0	1 (9.1)	1 (9.1)
COVID-19	3 (9.1)	0	0	0
Viral upper respiratory tract infection	2 (6.1)	0	1 (9.1)	0
Folliculitis	0	1 (3.0)	0	1 (9.1)
Nasopharyngitis	2 (6.1)	0	0	0
Cellulitis	0	0	1 (9.1)	0
Diverticulitis	1 (3.0)	0	0	0
Gastroenteritis viral	1 (3.0)	0	0	0
Herpes simplex	1 (3.0)	0	0	0
Hordeolum	1 (3.0)	0	0	0
Otitis externa	1 (3.0)	0	0	0
Pulpitis dental	1 (3.0)	0	0	0
Upper respiratory tract infection	1 (3.0)	0	0	0

n=7 Lymphocyte Count Decreased in SIGNAL AA; All grades 1/2

Current Landscape of Marketed Agents for AA

- **Olumiant (baricitinib) approved in 2022, Litfulo (ritlecitinib) approved in 2023**
- **Both carry classwide Black Box Warning:**

WARNING: SERIOUS INFECTIONS, MORTALITY, MALIGNANCY, MAJOR ADVERSE CARDIOVASCULAR EVENTS (MACE), and THROMBOSIS

See full prescribing information for complete boxed warning.

- **Increased risk of serious bacterial, fungal, viral, and opportunistic infections that may lead to hospitalization or death, including tuberculosis (TB). Interrupt treatment if serious infection occurs until the infection is controlled. LITFULO should not be given to patients with active tuberculosis. Test for latent TB before and during therapy; start treating latent TB prior to use. Monitor all patients for active TB during treatment, even patients with initial negative, latent TB test. (5.1). Monitor all patients for signs and symptoms of infection during and after treatment with LITFULO. (5.1)**
- **Higher rate of all-cause mortality, including sudden cardiovascular death with another Janus kinase inhibitor (JAK) vs. TNF blockers in rheumatoid arthritis (RA) patients. LITFULO is not approved for use in RA patients. (5.2)**
- **Malignancies were reported in patients treated with LITFULO (5.3). Higher rate of lymphomas and lung cancers with another JAK inhibitor vs. TNF blockers in RA patients.**
- **Higher rate of MACE (defined as cardiovascular death, myocardial infarction, and stroke) with another JAK inhibitor vs. TNF blockers in RA patients. (5.4).**
- **Thrombosis has occurred in patients treated with LITFULO. Increased incidence of pulmonary embolism, venous and arterial thrombosis with another JAK inhibitor vs. TNF blockers. (5.5)**

- **Olumiant approved recommended dose for AA: 2mg once daily**
  - Phase 3 SALT-20 at Week 24: 13% (BRAVE-AA1: 7% placebo-adjusted, BRAVE-AA2: 11% placebo-adjusted)[1]
- **Litfulo approved dose for AA: 50mg once daily**
  - Phase 3 SALT-20 at Week 24: 23% (21% placebo adjusted)[2]
- **Doctors and patients seeking alternatives to currently approved agents[3]**
  - Desire for safer options to currently available treatments

1 King B, et al. Two Phase 3 Trials of Baricitinib for Alopecia Areata, N Engl J Med 2022
2 https://www.accessdata.fda.gov/drugsatfda_docs/label/2023/215830s000lbl.pdf
3 Source: Wells Fargo Research, "Takeaways from Our Investor Lunch with Management and Alopecia Areata KOL" Oct 31, 2024

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

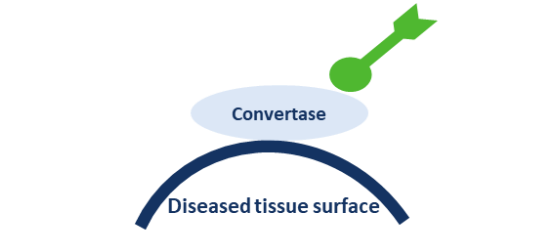
Apellis *InflaRx* *Kira*

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

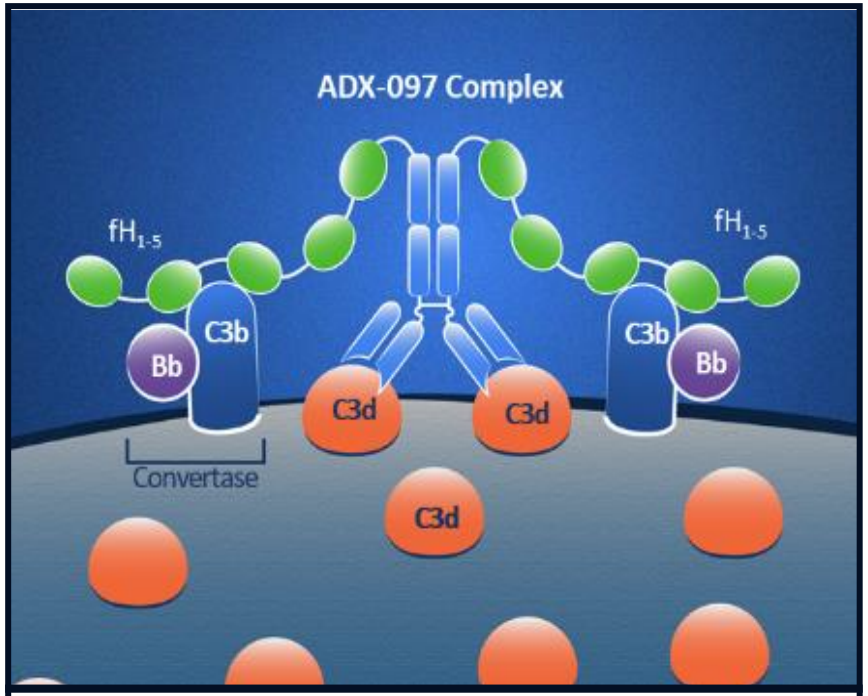
Topline Data Expected 2H'25

- Renal basket Ph2 topline data (initial data 1H'25)

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

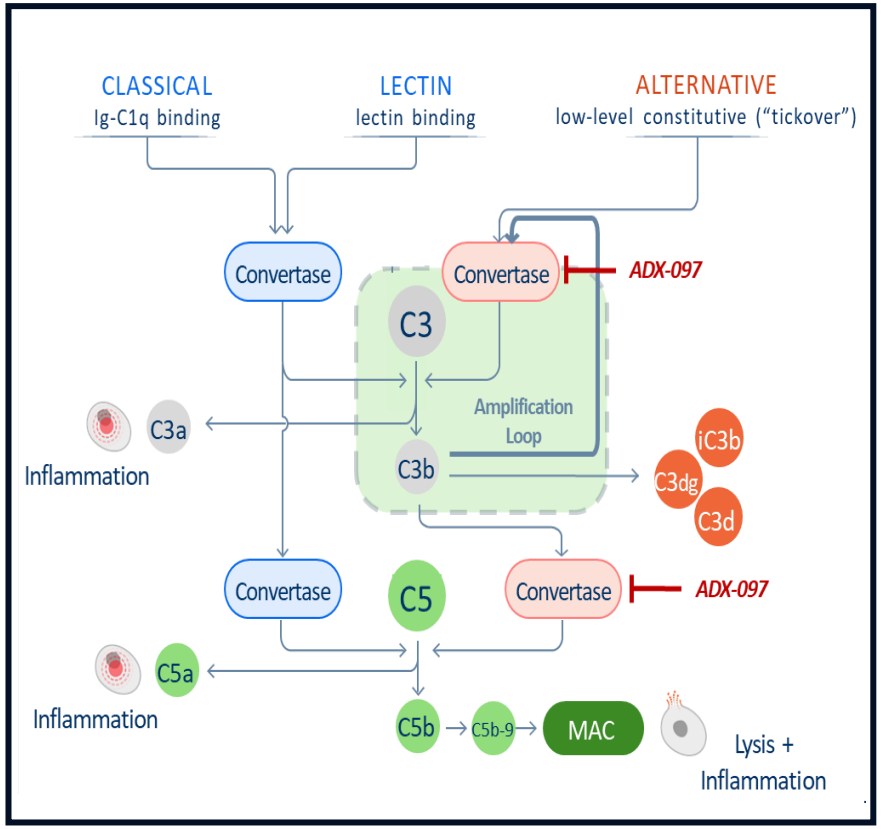
ADX-097 Design: C3d antibody – fH_{1-5}

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

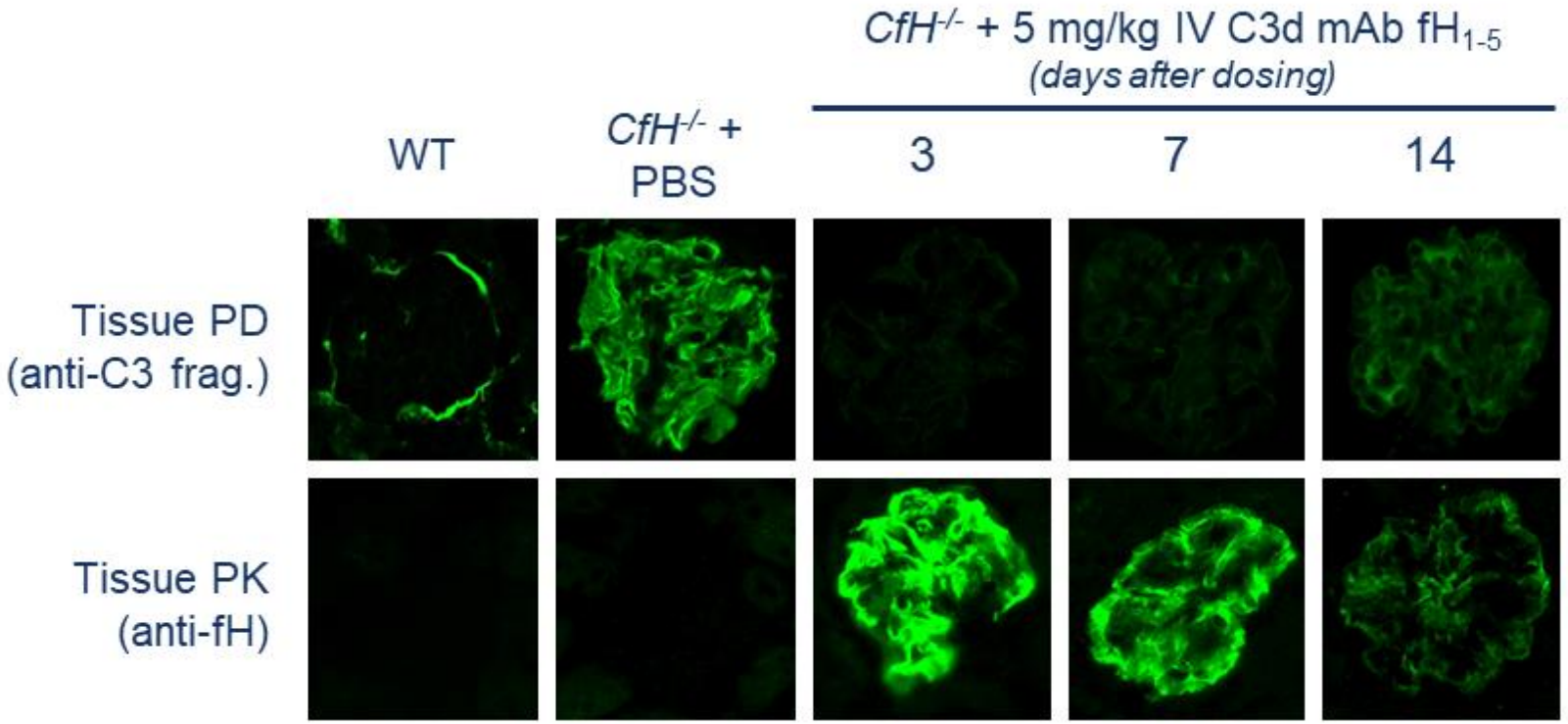
Milestone[1]	Organ System	Species	Model
Target validation in human disease	✓ Kidney ✓ Skin ✓ Liver	✓ Human	IHC of human disease biopsies (multiple disease-types for kidney, skin & liver)
Biodistribution of drug to tissue	✓ Kidney ✓ Skin ✓ Liver	✓ Mouse ✓ Rat ✓ NHP	fH -/- mice, EBA mouse model, PHN rat 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

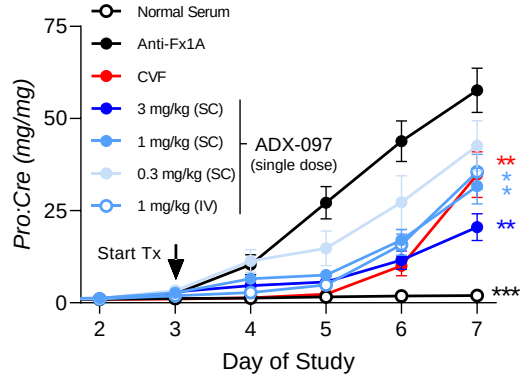
Potent Tissue PK/PD



- ✓ **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**

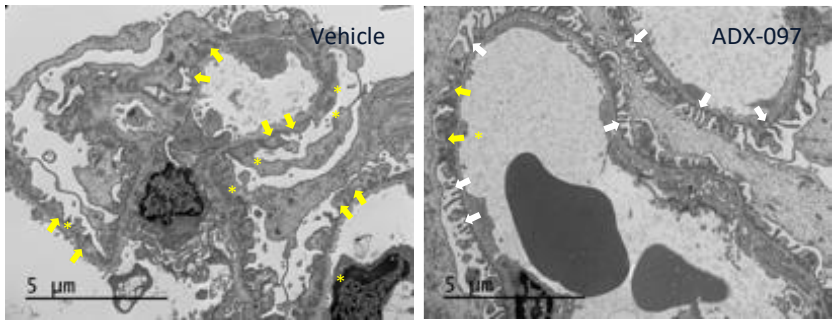
Passive Heymann Nephritis Rat Model of Human Membranous Nephropathy: Showed Robust Effect on POM and POC Endpoints

Proteinuria



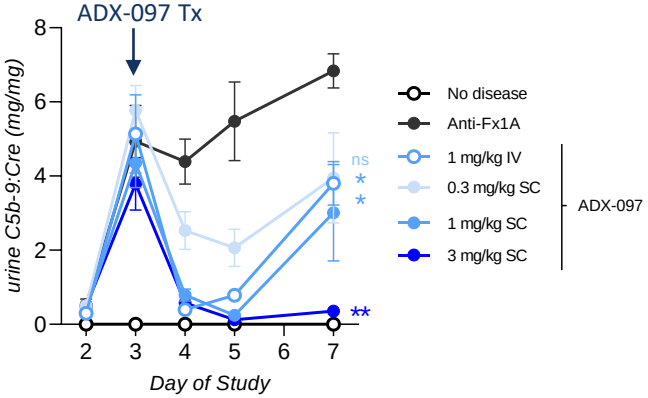
Potent inhibition of proteinuria equivalent to full systemic complement depletion (cobra venom factor)

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**

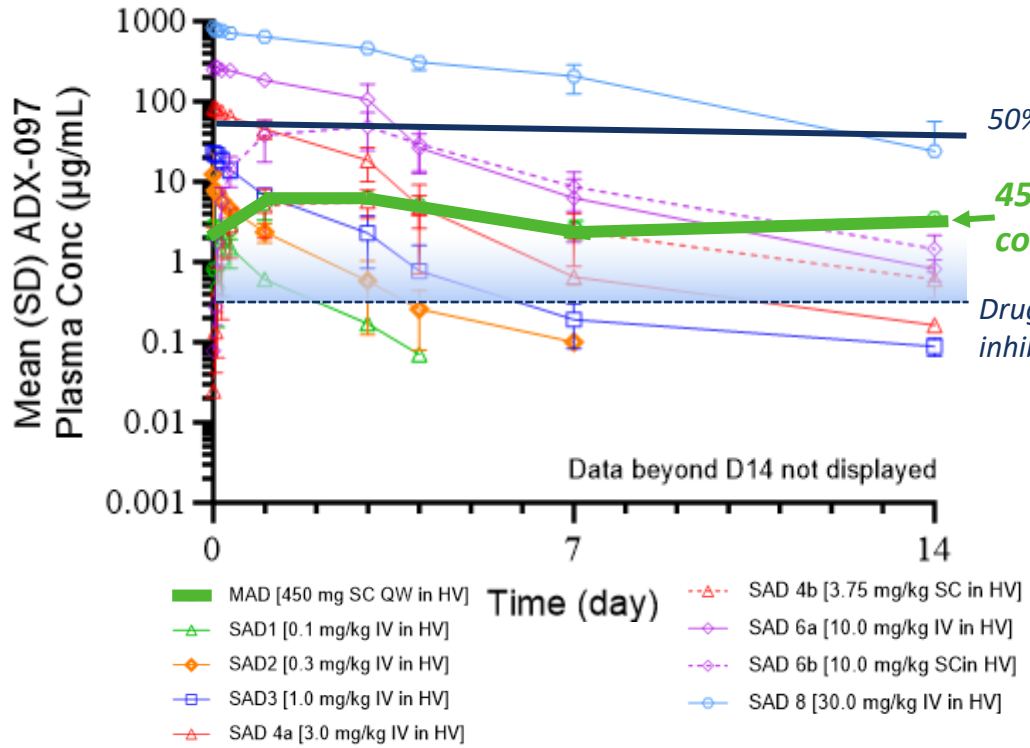
ADX-097-001 Phase 1 Study: Complete with Primary Goals Achieved

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



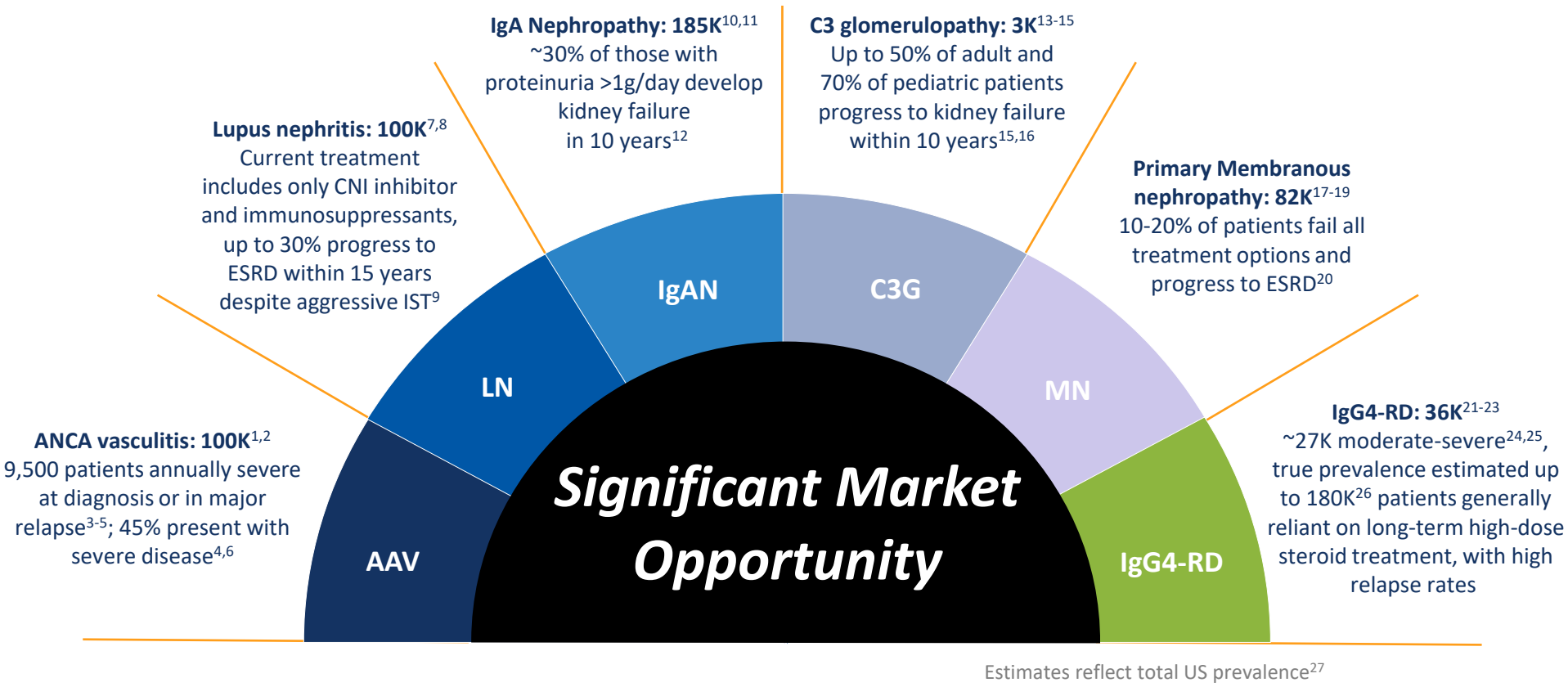
50% systemic complement inhibition

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 **Trial initiated; topline results expected in 2H'25, with initial open-label data by 1H'25**

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.

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

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

Financial Overview

- Q3 cash balance of $89.1M, providing expected cash runway **to mid 2026**
- Approximately 12.1M shares outstanding

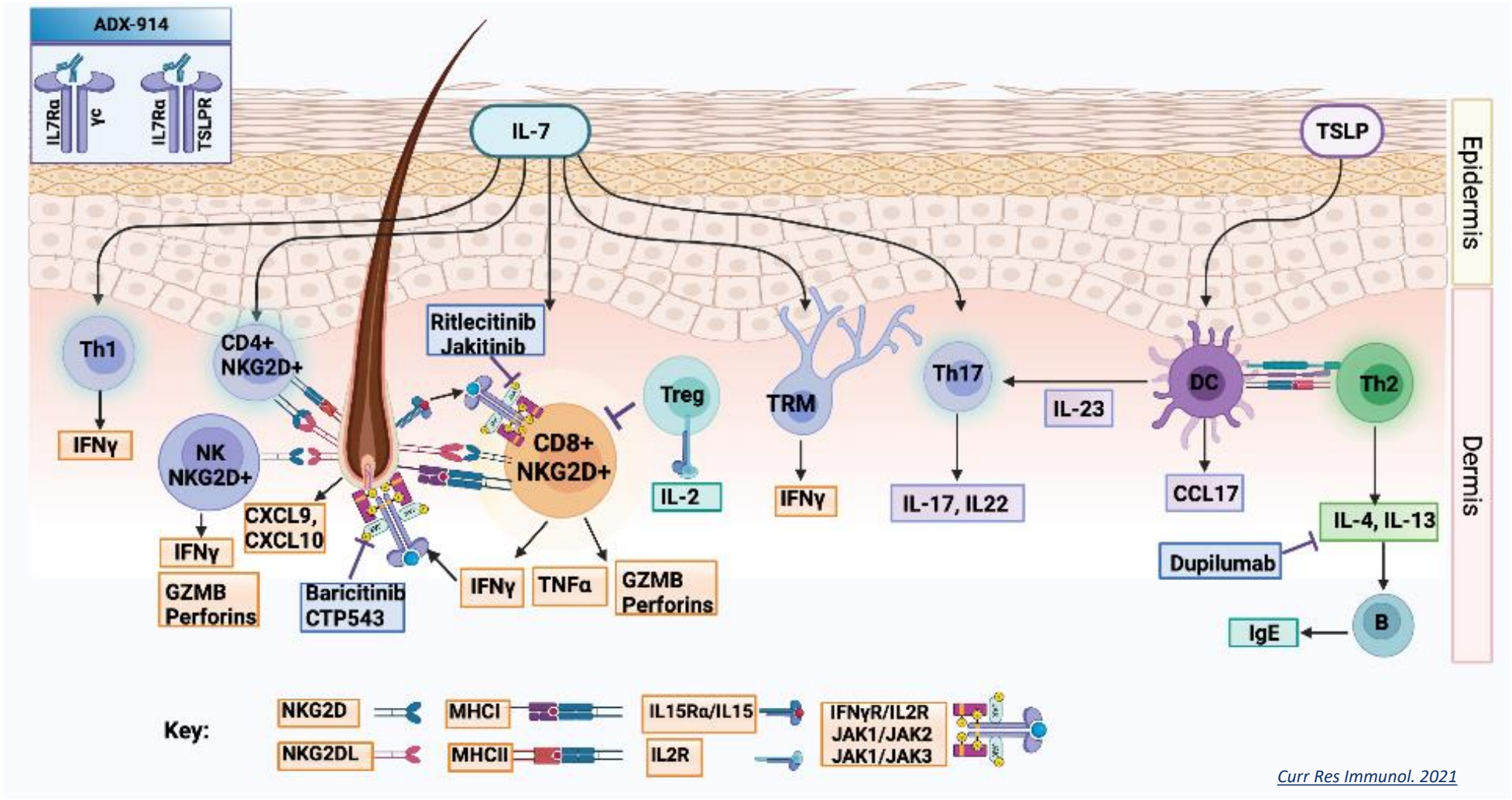
Numerous Anticipated Milestones in 2025

- **1H25:** Bempikibart AA Ph2 Part B initiation
- **1H25:** Renal basket Ph2 initial data
- **2H25:** Renal basket Ph2 topline results

APPENDIX

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 2 Clinical Trial in Atopic Dermatitis Topline Results
ADX-914-202 SIGNAL-AD

Part A

12-week treatment (n= 15 patients in Part A), 12-week follow-up

- **Part A Key Assessments**
  - 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

- **Part A Topline Results at Week 14[1]**
  - At 2 mg/kg Q2W, mean % change in EASI score from baseline was 58.0%; at 3 mg/kg Q2W, mean % change in EASI score from baseline was 83.9%
  - On a pooled basis, mean % change in EASI score from baseline was 72.3%
  - Mean % change in EASI score was 38.3% for placebo

Part B

12-week treatment (n= ~100 patients in B), 12-week follow-up

- **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

- **Part B Topline Results at Week 14[2]**
  - Patients treated with bempikibart showed a 74.4% mean % change in EASI from baseline, compared to 76.2% for placebo (p= NS)
  - Results of the primary endpoint were generally consistent when stratified for pre-specified baseline entry criteria
  - Results from analysis of key secondary endpoints were generally consistent with findings from the primary endpoint

1 Actual Percent Change from Baseline EASI (modified Full Analysis Set)

2 Least-Squares Mean Change as per pre-specified primary endpoint