KYMERA

Revolutionizing Immunology with Oral Medicines

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Kymera Therapeutics

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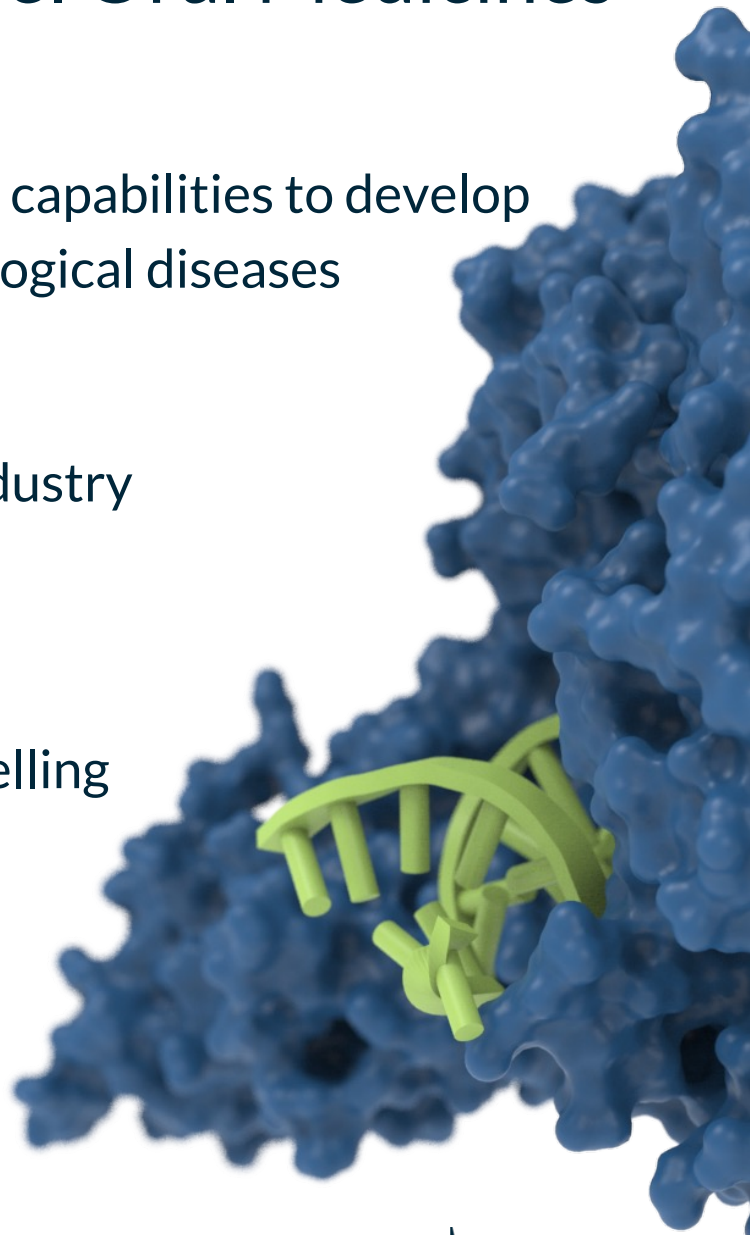
Well Positioned to Redefine Immunology with Novel Oral Medicines

Unrivaled science and expertise: Innovative, experienced team and capabilities to develop revolutionary oral therapies to transform the treatment of immunological diseases

Patient-first mentality, with urgency: Rapidly advancing first-in-industry oral degraders with biologics-like activity

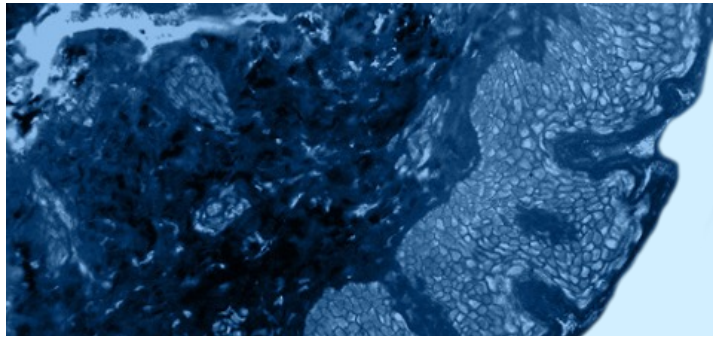
Defining and surpassing clinical benchmarks: Demonstrated compelling clinical translation of degraders' impact on clinical endpoints

Scaling for the future: Building a fully integrated global company to deliver a new class of immunology medicines



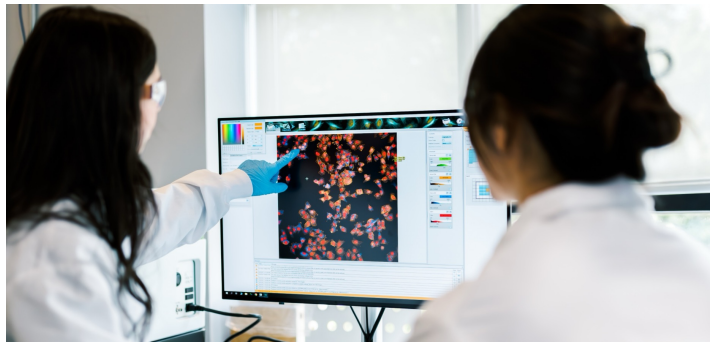
2025: A Defining Year for Kymera

Committed to Driving Patient Impact



FIRST-IN-CLASS: STAT6/KT-621

- ✓ Positive Phase 1 Healthy Volunteer study (June 2025)
- ✓ Positive BroADen Phase 1b AD study (December 2025)
- ✓ Commenced dosing in BROADEN2 Phase 2b trial in AD patients
- ✓ Enabled January 2026 initiation of BREADTH Phase 2b asthma study



FIRST-IN-CLASS: IRF5/KT-579

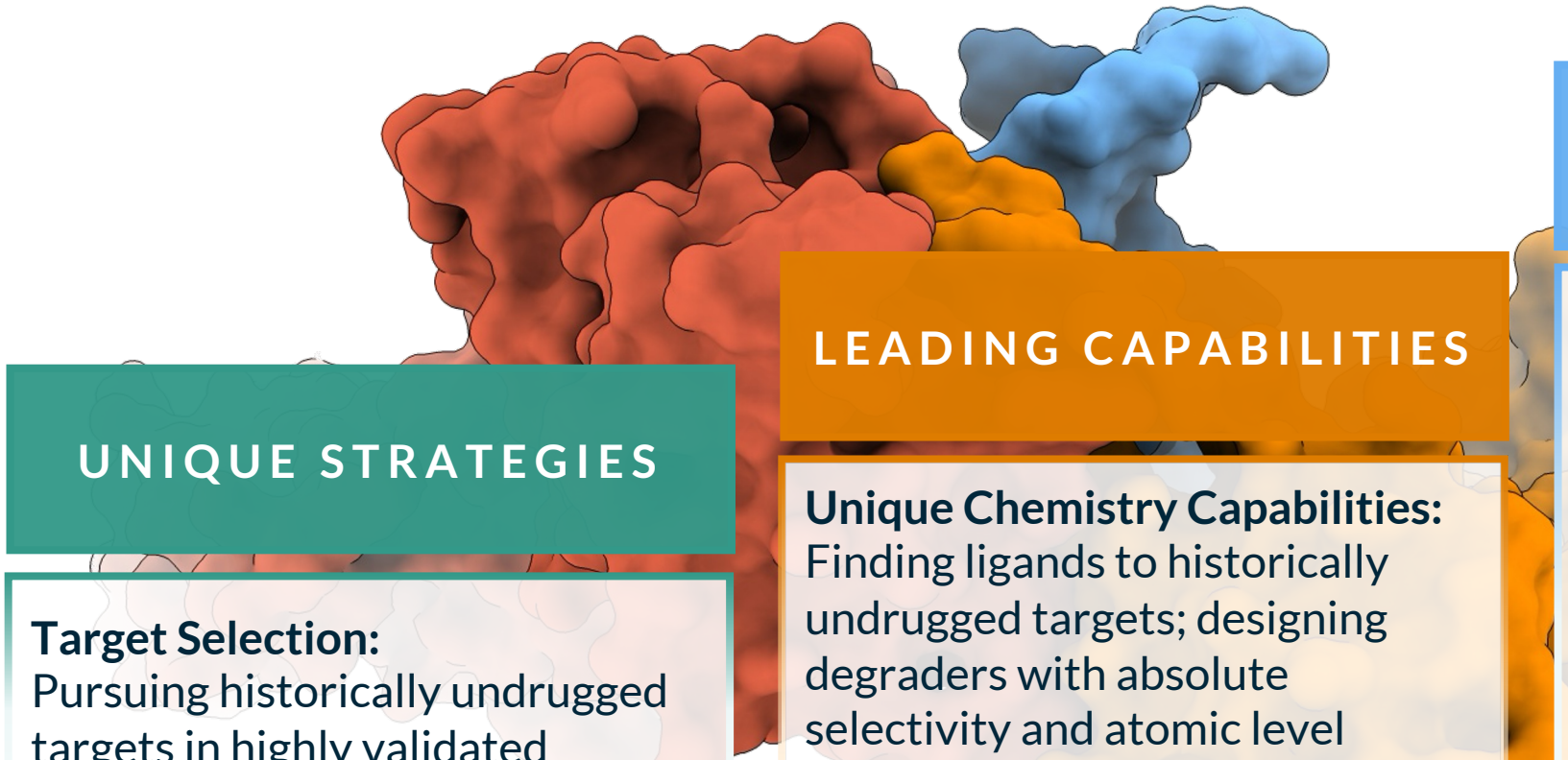
- ✓ Unveiled IRF5 program with compelling preclinical profile
- ✓ Completed IND-enabling studies
- ✓ Presented new preclinical data in SLE and RA efficacy models at ACR

CORPORATE

- ✓ Entered collaboration with Gilead to develop CDK2 molecular glue degraders
- ✓ Sanofi opted-in to KT-485 with plans to advance into Phase 1 testing in 2026
- ✓ Capitalized to execute on goals with $1.6B in cash and runway into 2029[1]

[1]Unaudited, estimated cash as of December 31, 2025.

Kymera: Industry Leader in Developing Oral Degrader Medicines



UNIQUE STRATEGIES

Target Selection:
Pursuing historically undrugged targets in highly validated pathways

Immunology Focus:
Building an industry leading pipeline of oral medicines with biologics-like activity to transform treatments for patients

LEADING CAPABILITIES

Unique Chemistry Capabilities:
Finding ligands to historically undrugged targets; designing degraders with absolute selectivity and atomic level understanding of MOA

Redefining Drug Development:
Developed new principles to match biologics activity with oral drugs

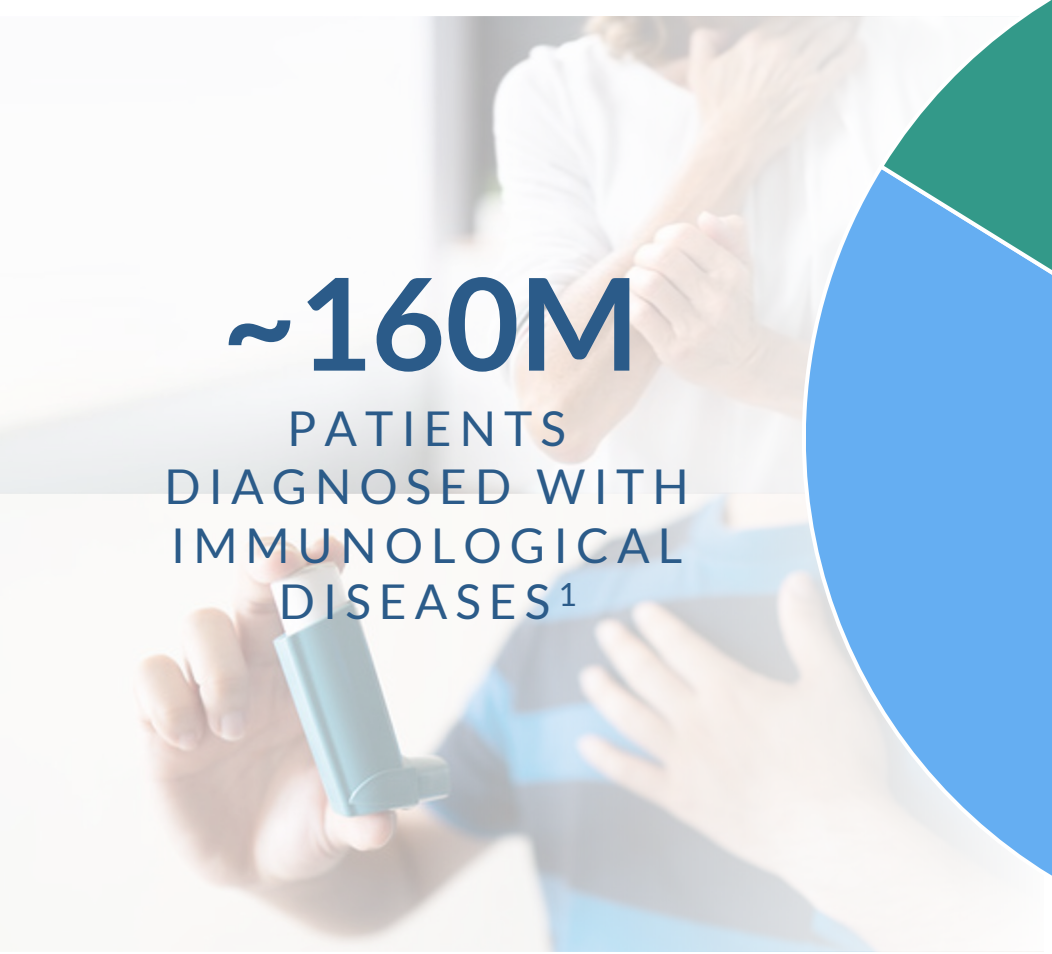
NOVEL INSIGHTS

Translation:
Deep expertise in target–drug interplay to optimize drug disposition and degradation across tissues and cell types

Accelerated Development:
Leverage learnings and innovation (e.g., new biomarkers, endpoints) from early studies to inform and derisk mid/late-stage development; addressing multiple large indications with high unmet need

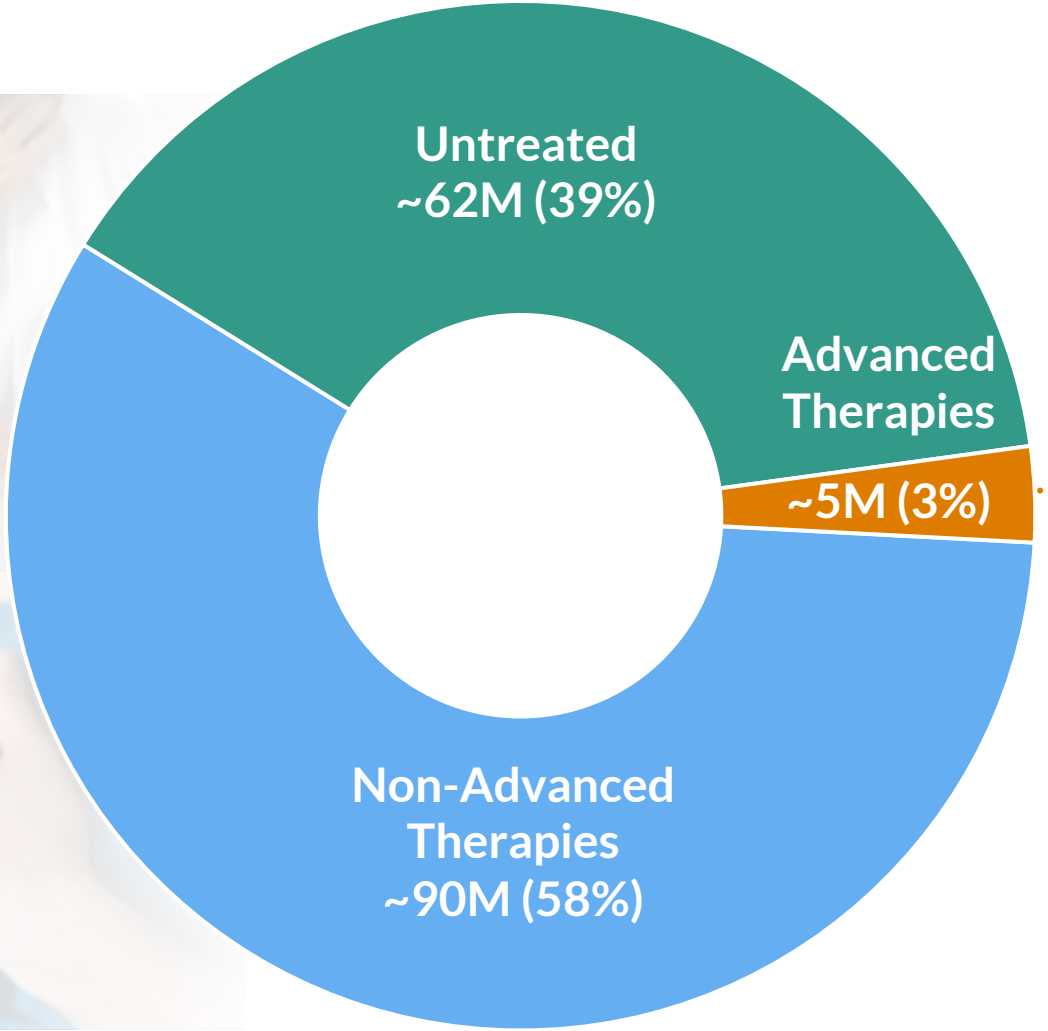
Immunology Remains a Large Underserved Market

Millions of Patients Do Not Have Access to Advanced Systemic Therapies



~160M

PATIENTS DIAGNOSED WITH IMMUNOLOGICAL DISEASES[1]

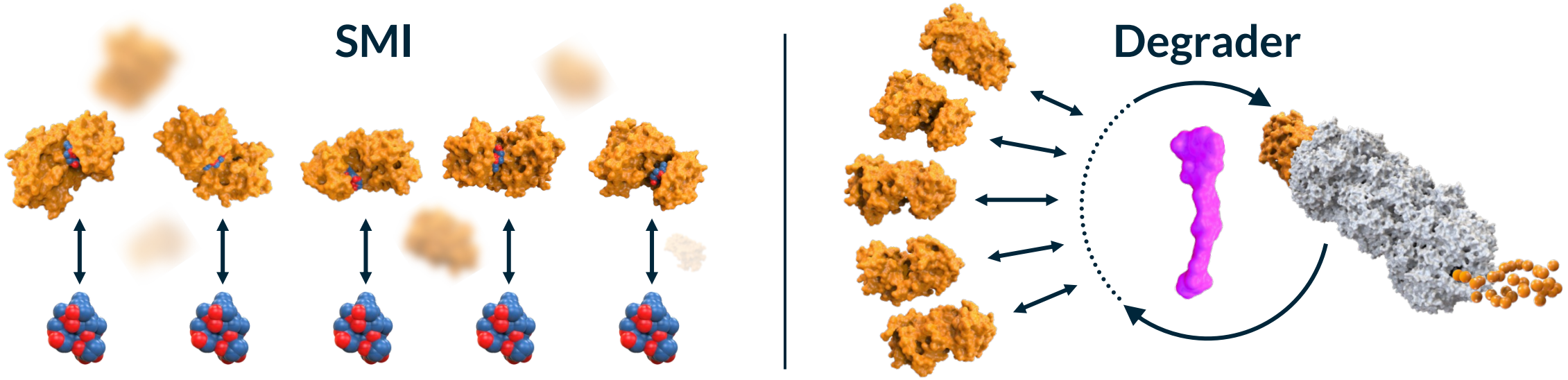


>$100B

IN ANNUAL SALES FOR ADVANCED THERAPIES[2]

[1]Diagnosed prevalent patient population for US/EU5/JP (GlobalData; 2023); key immunologic diseases includes AD, Asthma, CD, COPD, HS, MS, PsA, PsO, RA, SLE, UC;
[2]Market Forecasts for US/EU5/JP (GlobalData; 2023).

Degraders Allow for Continuous, Biologics-like, Complete Pathway Blockade Unlike Traditional Small Molecule Inhibitors (SMI)



STOICHIOMETRIC INHIBITION

Pharmacodynamic effect **(PD) correlated** to drug exposure (**PK**), (stochiometric) requiring continuous high drug exposures to block protein function

CATALYTIC DEGRADATION

PD NOT directly correlated to **PK**, (catalytic) allowing fast, complete protein elimination and pathway blockade with low/short drug exposures

- **Oral drugs** with **biologics-like** activity, **unlocking target classes** unreachable by other modalities
- **High selectivity, strong potency, and catalytic mechanism** enable full and constant target suppression with low doses
- Achieves **full pathway blockade**, matching biologics-like depth of immune modulation

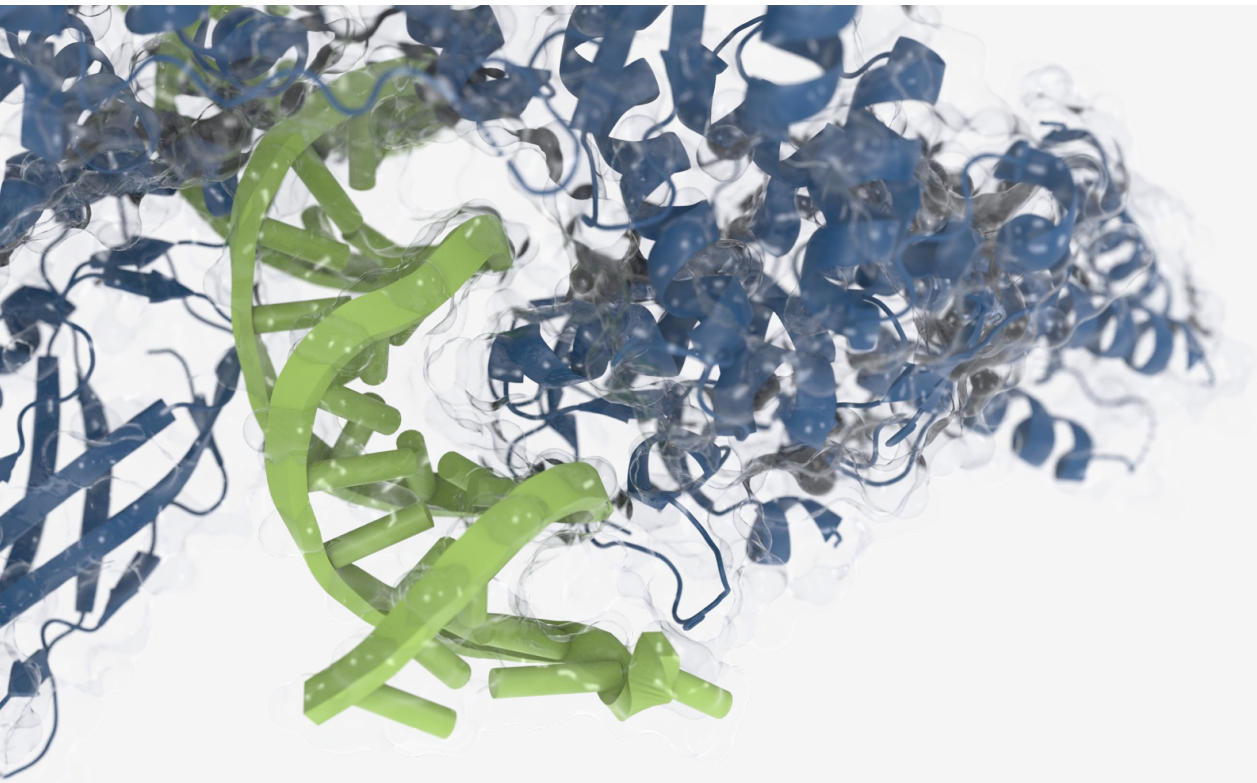
Oral degraders that combine the activity of injectable biologics with the convenience of oral drugs have the potential to transform current treatment paradigms

Building a Best-In-Industry Oral Immunology Pipeline

	Potential Indications	IND-Enabling	Phase 1	Phase 2	Phase 3	Upcoming Milestones
Immunology - Wholly-Owned Oral Small Molecule Degraders						
STAT6	AD, Asthma, COPD, EoE, CRSwNP, CSU, PN, BP, others	KT-621 – AD KT-621 – Asthma				**Ph2b AD Data:** By mid-2027 **Ph2b Asthma Data:** Late-2027
IRF5	Lupus, Sjögren's, RA, IBD, SSc, DM, others	KT-579				**Ph1 HV Start:** 1Q26 **Ph1 HV Data:** 2H26
Partnered Programs						
IRAK4	HS, AD, RA, Asthma, IBD, others[2]	KT-485[1]				sanofi **Ph1 Start:** 2026
CDK2[3]	Breast cancer and other solid tumors	Molecular Glue				GILEAD

Combining the convenience of oral drugs and the activity of biologics to expand access to systemic advanced therapies for millions of patients around the world

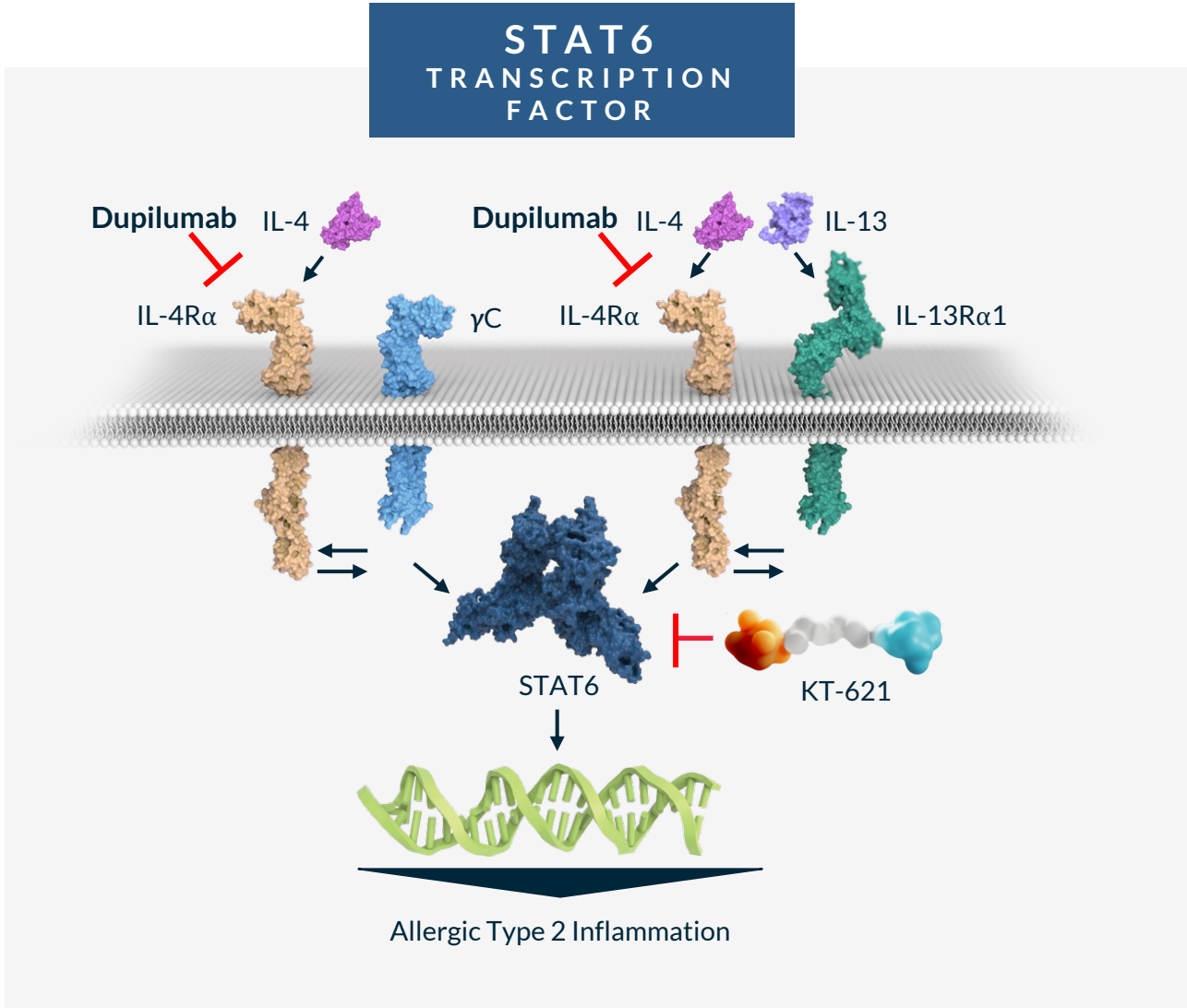
[1]KT-485 (SAR447971) partnered with Sanofi, with Kymera option to participate in the development and commercialization, and 50/50 profit split, in the United States. Double digit tiered royalties in ROW; [2]Diseases where IL-1R/TLR pathway has been implicated in pathogenesis.[3]Partnered with Gilead, exclusive option and license agreement to accelerate the development and commercialization of a novel molecular glue degrader program.



KT-621
Potential to Redefine the Treatment of Type 2 Diseases

STAT6 Transcription Factor: Highly Validated but Undrugged Target

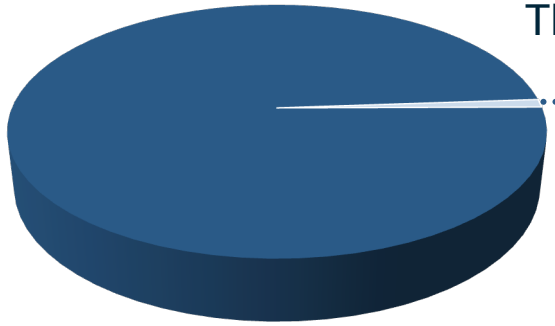
- STAT6 is the specific transcription factor in the IL-4/IL-13 pathway
- IL-4/IL-13 is clinically validated by dupilumab across multiple Type 2 diseases:
  - AD, asthma, COPD, EoE, CRSwNP, CSU, PN, BP
- STAT6 is genetically validated by human GoF and heterozygous LoF alleles
- While several therapies target the upstream IL-4/IL-13 receptors, there are no known drugs that selectively target this pathway with oral delivery potential



Sharma et al, 2023; Takeda et al, 1996; Kaplan et al, 1996; Junttila, 2018; Kolkhir et al, 2023; AD: Atopic Dermatitis; COPD: Chronic Obstructive Pulmonary Disease; EoE: Eosinophilic Esophagitis; CRSwNP: Chronic Rhinosinusitis with Nasal Polyps; CSU: Chronic Spontaneous Urticaria; PN: Prurigo Nodularis; BP: Bullous Pemphigoid; GoF: Gain of Function; LoF: Loss of function.

STAT6 Opportunity to Serve Millions of Patients with Type 2 Inflammation

>140M



Systemic Advanced Therapies: **1%**

>$20B Market

TOTAL POTENTIAL PATIENT IMPACT[1]

An oral STAT6 degrader has the potential to transform the treatment paradigm for Type 2 diseases and tap into large underserved markets

	PATIENTS[1]
DERMATOLOGY	
AD	43M
CSU	4.4M
PN	305K
BP	166K
GASTROENTEROLOGY	
EoE	1.6M
RESPIRATORY	
Asthma	55M
COPD	34M
CRSwNP	2.8M

[1]Diagnosed prevalent patient population for US/EU5/JP (GlobalData; 2023); [2]Estimate based on GlobalData 2023 market forecasts for AD, Asthma, and COPD in US/EU5/JP then extrapolated to remaining Type 2 indications; AD: Atopic Dermatitis; COPD: Chronic Obstructive Pulmonary Disease; EoE: Eosinophilic Esophagitis; CRSwNP: Chronic Rhinosinusitis with Nasal Polyps; CSU: Chronic Spontaneous Urticaria; PN: Prurigo Nodularis; BP: Bullous Pemphigoid.

Current Treatments Do Not Address Patients' Needs

>55% of Adults with Moderate/Severe AD and 60% of Adult Asthma Patients Report Inadequate Disease Control[1,2]

INADEQUATE SYSTEM OF CARE

- **Topical treatments (AD) and inhalers (asthma)** are limited to the mildest cases
- **Biologics** associated with high treatment burden and cost/access concerns
- **Current oral therapies (JAKs and oral steroids)** come with serious efficacy and/or safety limitations

CURRENT TREATMENT LIMITATIONS

- Needle fear/fatigue
- Burdensome dosing (up to 4 injections in 1st month)
- Complex treatment initiation (blood testing)
- Inconsistent adherence
- Side effect and safety issues

PATIENTS WANT

- Rapid onset of relief and durable efficacy
- **Convenience of a daily pill;** >90% of patients on biologics would switch to an oral option[3]
- No treatment initiation requirements
- No side effects or safety issues
- HCP confidence in prescribing

KT-621 has the potential to offer "what patients want", transforming the treatment of AD, asthma, and other Type 2 diseases

[1]National Eczema Association; [2]U.S. Centers for Disease Control and Prevention; [3]Stein Gold et al, Fall Clinical Dermatology Conference, 2025.

KT-621 Key Data: Validation of STAT6 Targeting and Derisking Late Development

KT-621 Data Provide Validation and Derisk Future Clinical Trials

Preclinical

POTENCY

- < 100 pM DC90 in all relevant human cell types

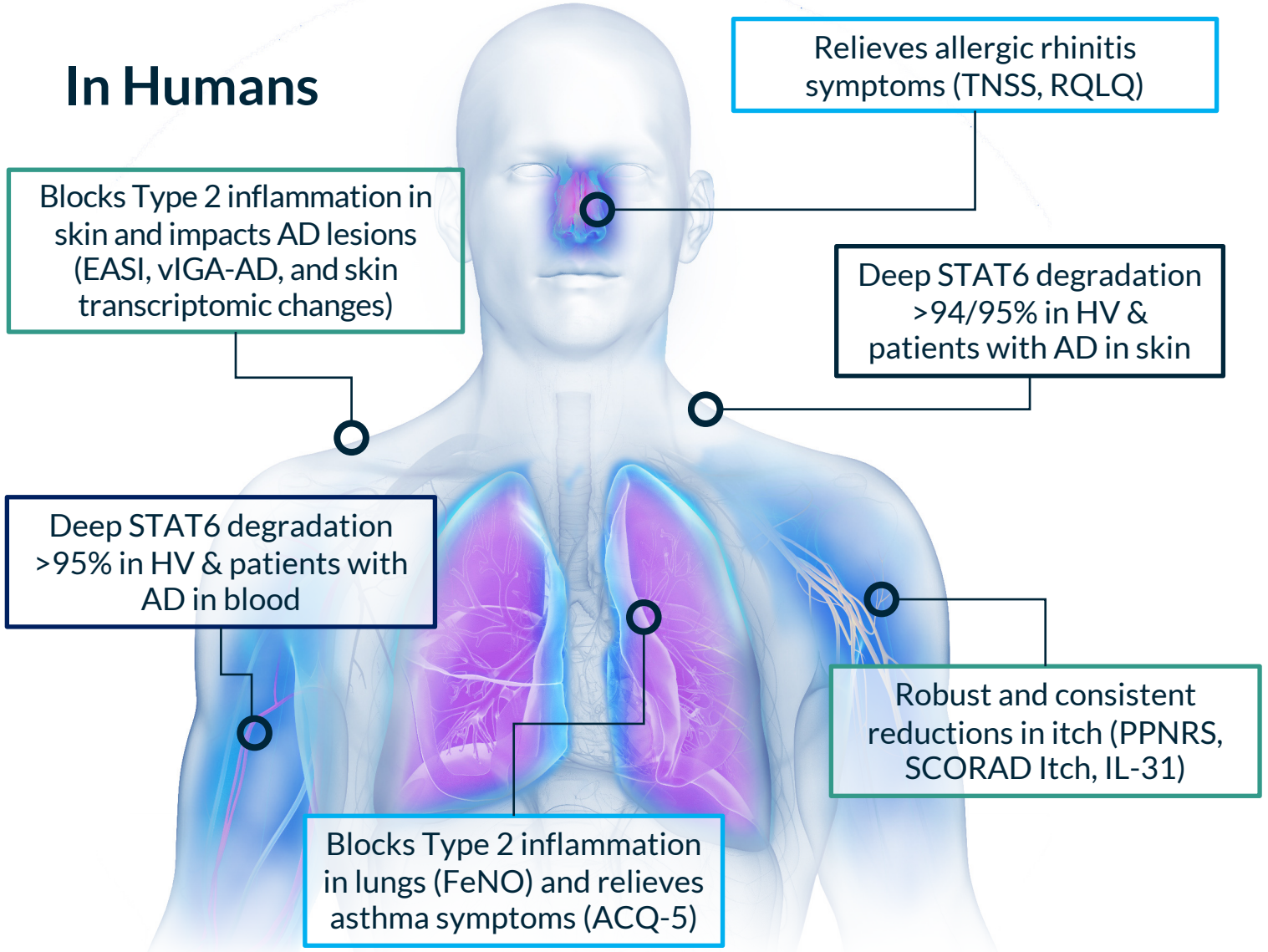
EFFICACY

- Blocked IL-4 and IL-13 signaling in human cells and *in vivo* systems equally or more potently than dupilumab
- Robust activity in asthma and AD in mouse models

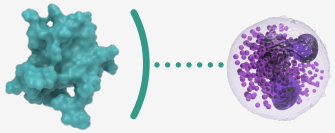
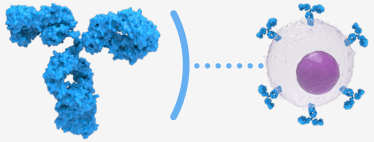
SAFETY

- No AEs at any doses in:
  - 4 weeks rat and NHP GLP tox
  - 4 months rat and NHP GLP tox
  - Embryofetal development tox in rat, rabbit and NHP

In Humans



Robust Impact on All Disease-Relevant Biomarkers of Type 2 Inflammation in BroADen Phase 1b AD Trial

	TARC (CCL17)	Eotaxin-3 (CCL26)	IgE	IL-31	FeNO
					
	Validated biomarker of Type 2 inflammation suppression in patients Drives chemotaxis of CCR4-expressed T cells to inflammatory sites	Highly specific downstream cytokine of the IL-4/13 pathway Drives chemotaxis of CCR3-expressed inflammatory cells to inflamed sites	IL-4 promotes B-cell class switching, amplifying IgE production IgE activates mast cells and basophils to release Type 2 cytokines	Key pruritogenic cytokine produced by activated Type 2 cells[1] Signals through the IL-31RA/OSMR complex on neurons, keratinocytes, and immune cells, linking immune activation to itch	Marker of Type 2 airway inflammation in asthma[2] FeNO reflects airway epithelial iNOS activity driven by IL-4/13 signaling Historically not measured in AD patients
Median % Inhibition at Day 29					
KT-621	**74%[3]**	**73%**	**14%**	**54%**	**33%**
Dupilumab[4]	74%	51% (in Asthma)	~15%	Not measured	Not measured

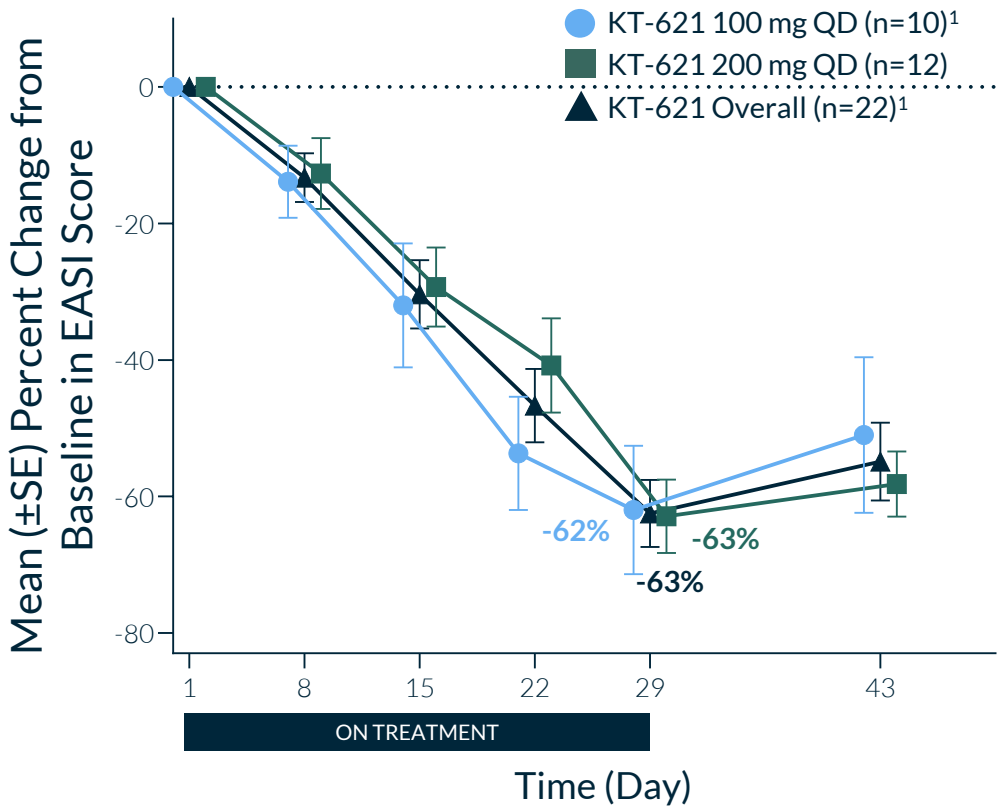
[1]Raap et al, *Journal of Allergy and Clinical Immunology*, 2008; [2]Chung et al, *Lancet*, 2021; [3]Represents TARC reduction in patients with elevated baseline TARC levels, defined as the lower bound of the 95% confidence interval for median baseline TARC levels from the dupilumab SOLO1-2 AD studies; [4]Hamilton et al, *Clinical & Experimental Allergy*, 2021.

KT-621 Impact on Atopic Dermatitis in BroADen Phase 1b Trial

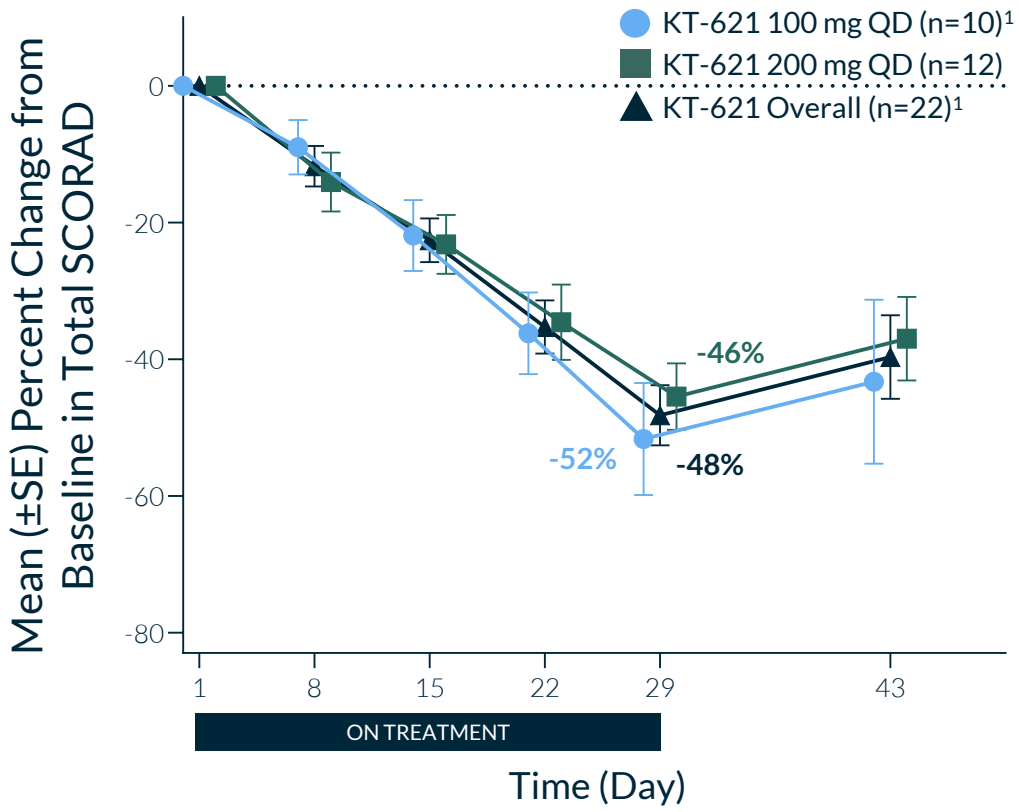
A single-arm, open-label study of KT-621 evaluating two dose levels (100 mg and 200 mg) administered orally once daily for 28 days in 22 patients with moderate to severe AD. For more information, visit Kymera's website.

KT-621 Achieved Rapid and Robust Reductions in EASI and SCORAD Across All Patients

Mean % Change from Baseline in EASI



Mean % Change in Total SCORAD

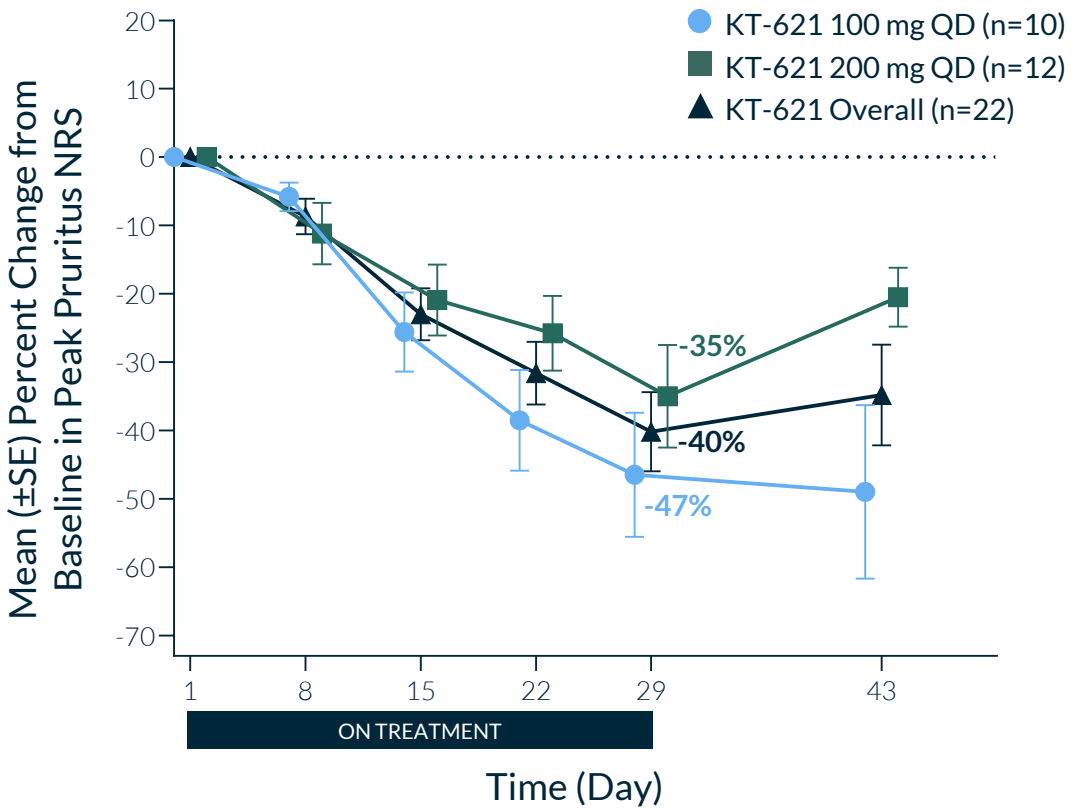


- Reductions seen as early as Day 8 without apparent plateau during treatment duration
- Achieved robust clinical improvement across EASI-50 (76% overall), EASI-75 (29% overall) and vIGA-AD (19% overall)

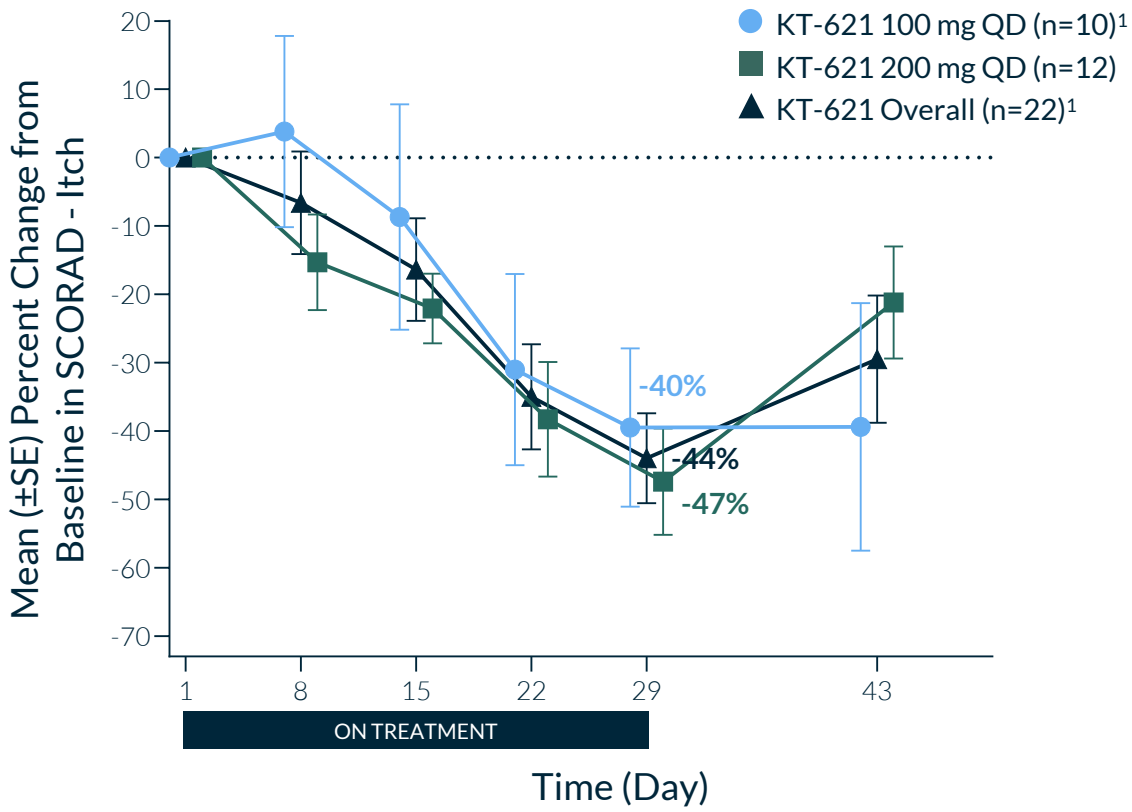
Note: Analysis based on observed cases at each visit; [1]One patient in the 100 mg cohort missed the D29 visit (n=9 for 100 mg and n=21 for Overall at D29); EASI: Eczema Area and Severity Index; SCORAD: SCORing Atopic Dermatitis.

KT-621 Achieved Robust and Consistent Reductions in Itch Across Independent Clinical Measures

Mean % Change in Peak Pruritus NRS



Mean % Change in SCORAD Itch

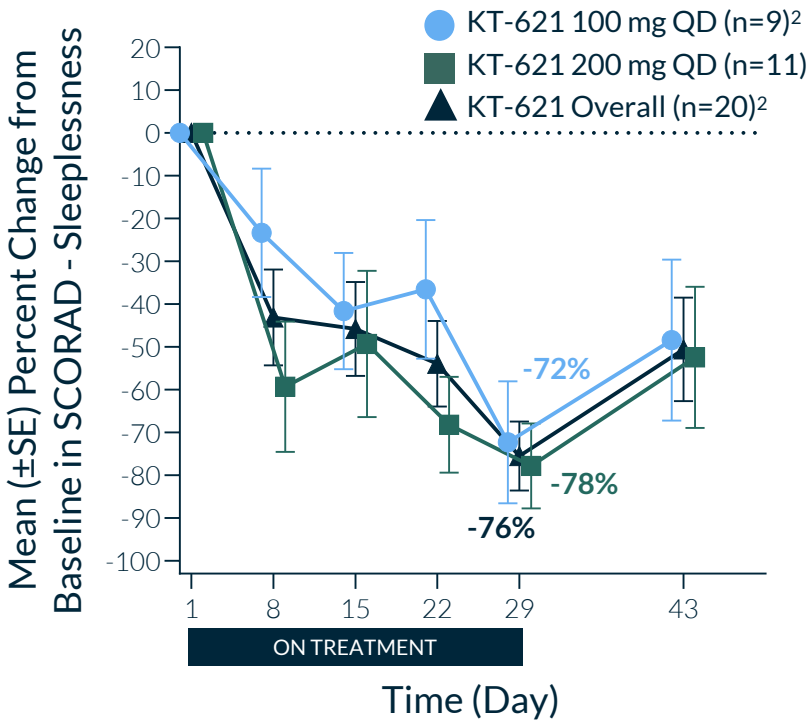


- KT-621 achieved rapid and robust mean Peak Pruritus NRS and SCORAD-Itch reduction without apparent plateau during treatment duration

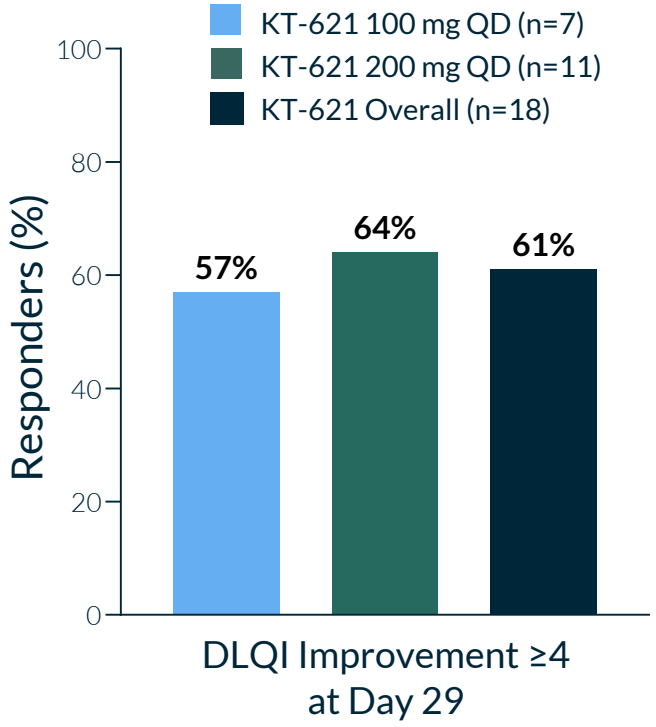
Note: Analysis based on observed cases at each visit; [1]One patient in the 100 mg cohort missed the D29 visit (n=9 for 100 mg and n=21 for Overall at D29); PPNRS: Peak Pruritus Numerical Rating Scale; SCORAD: SCORing Atopic Dermatitis.

KT-621 Achieved Robust Improvement in Patient Reported Outcomes and Quality of Life Measures

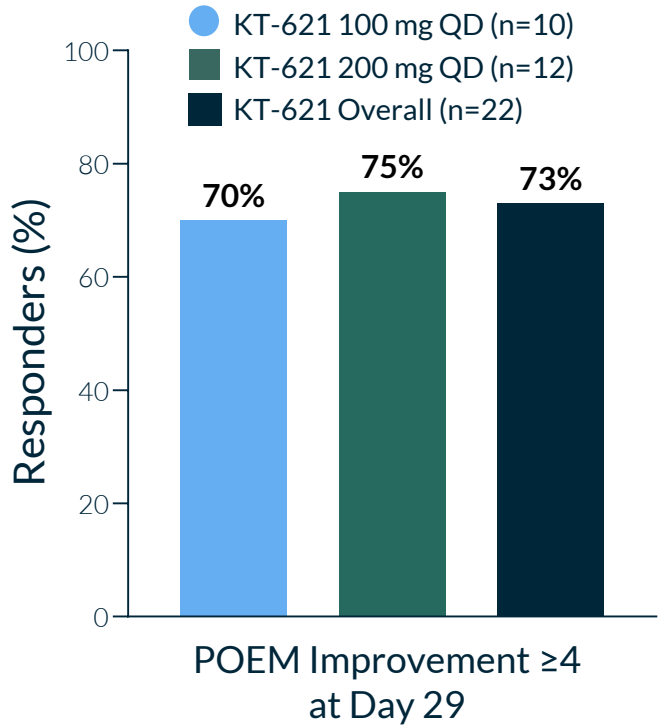
Mean % Change in SCORAD - Sleeplessness[1]



DLQI Responders (Improvement ≥ 4 points)[3]



POEM Responders (Improvement ≥ 4 Points)



- KT-621 achieved rapid and robust mean SCORAD-Sleeplessness reduction in both dose cohorts
- POEM and DLQI are patient-reported measures evaluating severity, experience, and quality of life; demonstrated improvements are greater than the minimum clinically important difference (MCID)

Note: Analysis based on observed cases at each visit. [1]Only patients with non-zero baseline sleeplessness are included; [2]One patient in the 100 mg cohort missed the D29 visit (n=8 for 100 mg and n=19 for Overall at D29); [3]Only patients with baseline DLQI score ≥ 4 included; SCORAD: SCORing Atopic Dermatitis. DLQI: Dermatology Life Quality Index; POEM: Patient-Oriented Eczema Measure.

KT-621: BROADEN2 Phase 2b Trial

Randomized, Double Blind, Placebo-controlled, Parallel-group, Multicenter Dose-ranging

BROADEN2 TRIAL

Adult & Adolescent Moderate to Severe AD Patients Ages 12-75

Baseline entry criteria:
EASI ≥ 16;
vIGA-AD ≥ 3;
Peak Pruritus NRS ≥4;
BSA ≥10%;
Documented TCS failure for AD

Design

- Randomized, double-blind, placebo-controlled
- ~200 patients
- Daily dose for 16-weeks; 52-week open label extension

Dosing

- Three KT-621 doses + one placebo (1:1:1:1)

Endpoints

- Primary endpoint: Percent change from baseline in EASI score at week 16
- Secondary endpoints include:
  - EASI-50, EASI-75, vIGA-AD 0/1
  - At least a 4-point improvement from baseline in Peak Pruritus NRS

Key Trial Aim

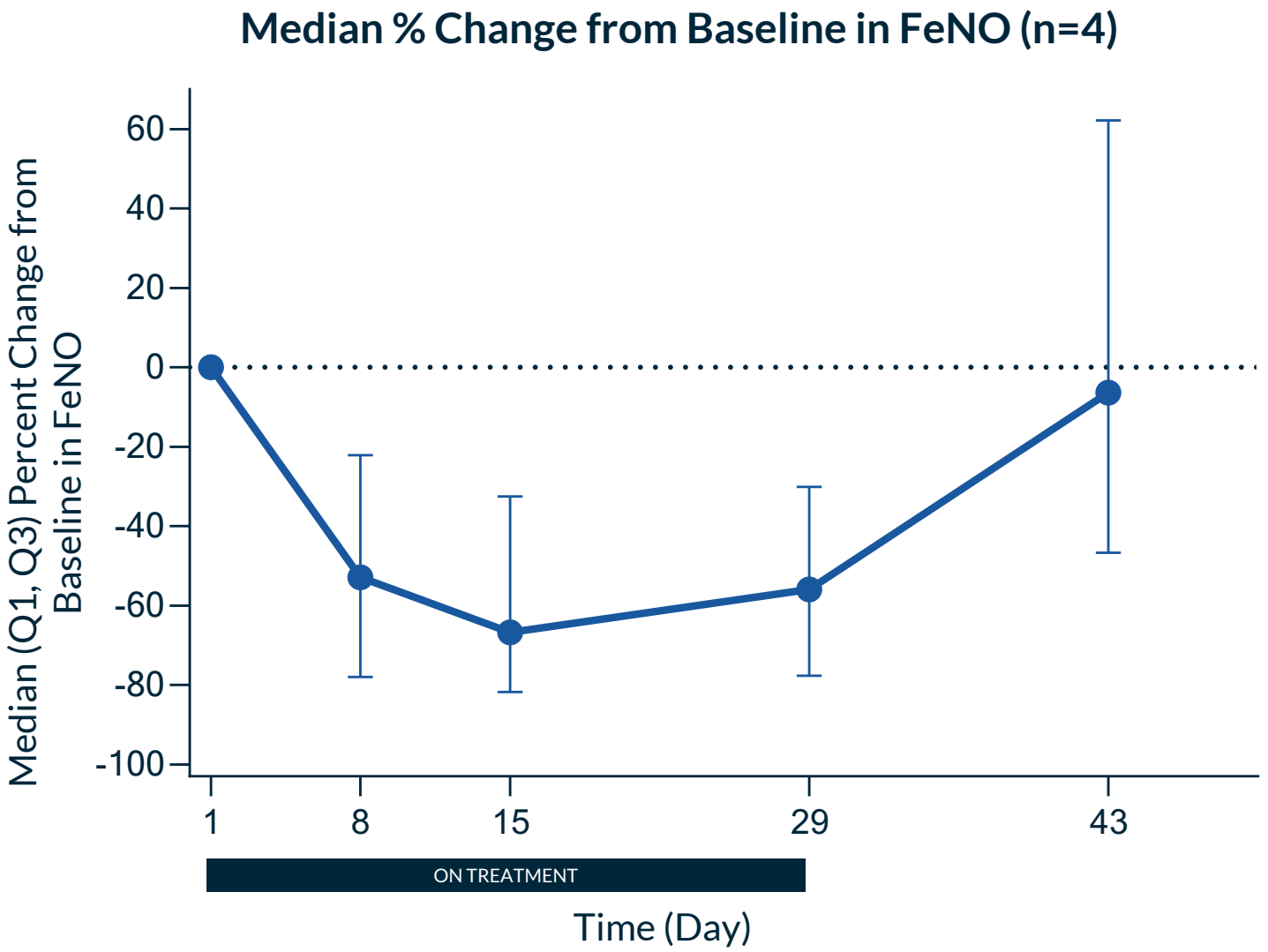
Establish clinical activity and safety in **AD to select Phase 3 dose to support registrational studies** in multiple dermatological and gastrointestinal indications

Status update:
Ongoing, also including adolescents;
Data expected by mid-2027

KT-621 Impact on Comorbid Type 2 Respiratory Diseases in BroADDen Phase 1b Trial

KT-621 Achieved Robust Impact on FeNO in AD Patients with Comorbid Asthma

- KT-621 achieved 56% median FeNO reduction at Day 29, exceeding dupilumab (31%) in asthma studies at week 4[1]

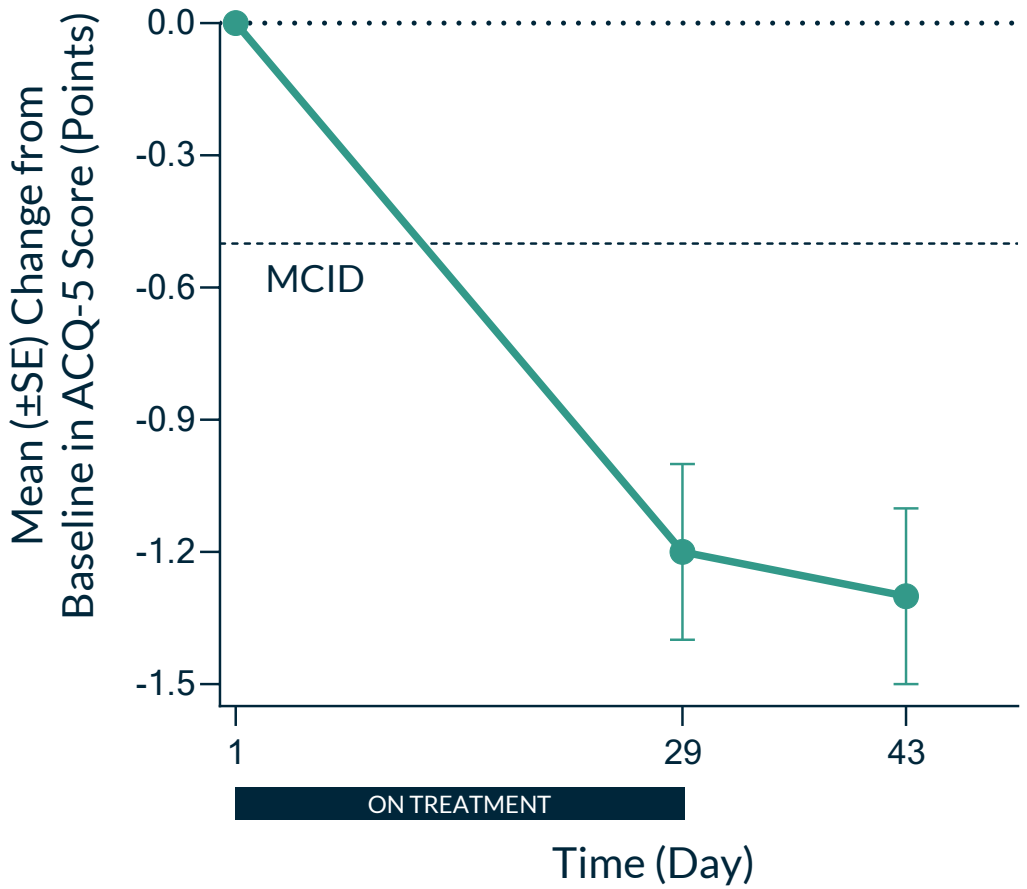


Note: Analysis based on observed cases at each visit; Mean baseline FeNO of 49 ppb; [1]Pavord et al. JACI. 2024; FeNO: Fractional Exhaled Nitric Oxide

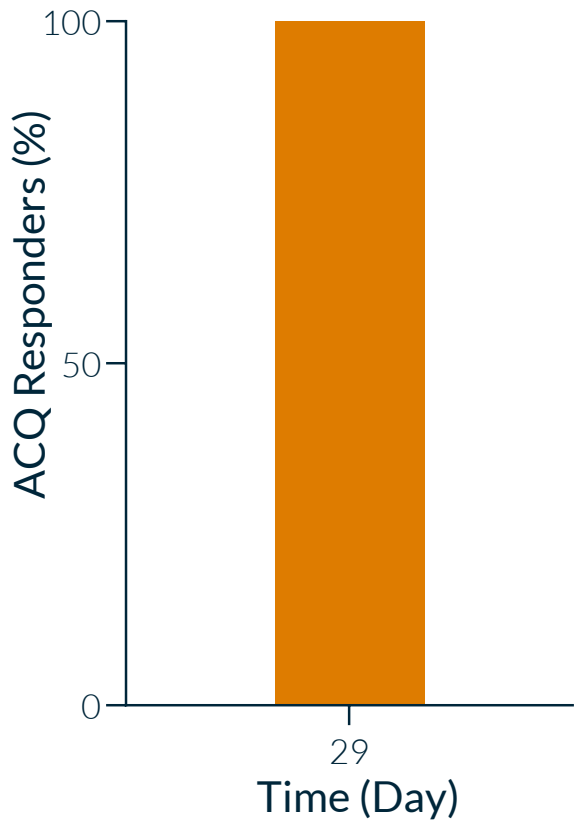
KT-621 Achieved Robust Impact on ACQ-5 in AD Patients with Comorbid Asthma

- All 4 patients had clinically meaningful reduction in ACQ-5 (mean change of -1.2 points) and a 100% responder rate

Mean Point Change in ACQ-5 (n=4)



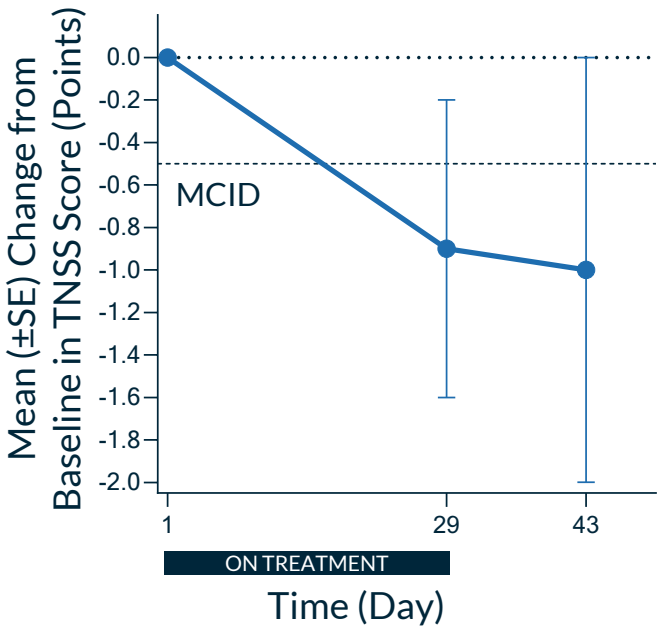
ACQ-5 Responders (Improvement ≥ 0.5, n=4)



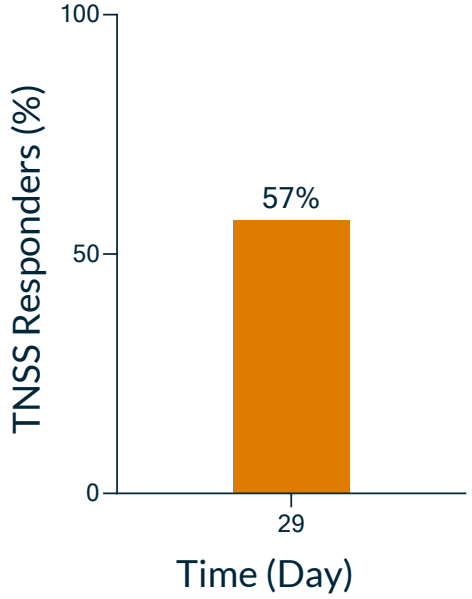
Note: Analysis based on observed cases at each visit; Mean baseline ACQ-5 of 2.5; ACQ-5: Asthma Control Questionnaire-5; MCID: Minimum Clinically Important Difference.

KT-621 Achieved Robust Impact on TNSS and RQLQ in AD Patients with Comorbid Allergic Rhinitis

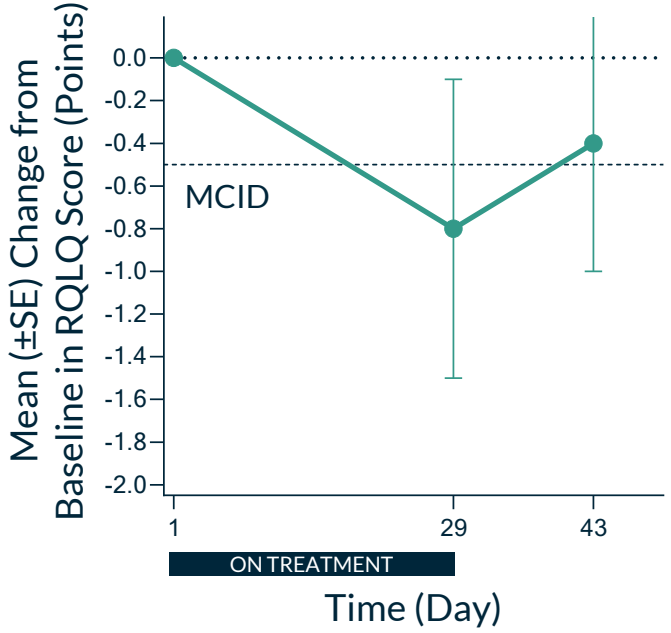
Mean Point Change in TNSS (n=7)[1]



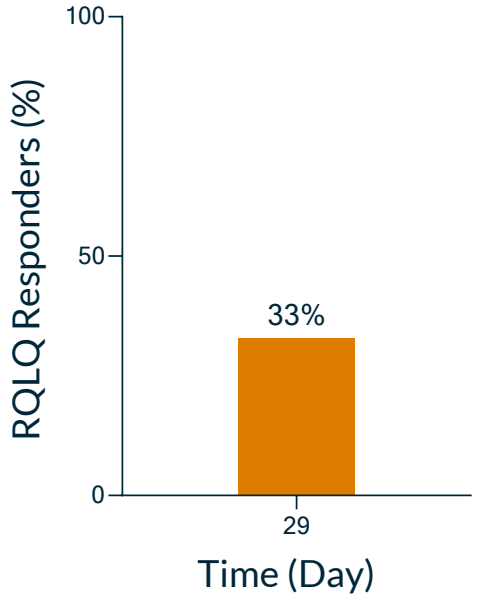
TNSS Responders (Improvement ≥ 0.55, n=7)[1]



Mean Point Change in RQLQ (n=6)[2]



RQLQ Responders (Improvement ≥ 0.5, n=6)[2]



- At Day 29, KT-621 achieved mean changes of –0.9 and -0.8 points in TNSS and RQLQ, respectively
- TNSS and RQLQ responder rates were 57% and 33%, respectively

Note: Analysis based on observed cases at each visit; [1]Only patients with baseline TNSS score ≥ 0.55 are included; [2]Only patients with baseline RQLQ score ≥ 0.5 are included; Mean baseline TNSS and RQLQ of 4.4 and 2.4, respectively; MCID: Minimum Clinically Important Difference; TNSS: Total Nasal Symptom Score; RQLQ: Rhinoconjunctivitis Quality of Life Questionnaire.

KT-621: BREADTH Phase 2b Trial

Randomized, Double Blind, Placebo-controlled, Parallel-group, Multicenter Dose-ranging

BREADTH TRIAL

Adult, Moderate to Severe Eosinophilic Asthma Patients

Baseline entry criteria:
Blood eosinophils ≥ 300 cell/uL
FeNO ≥ 25 ppb
Pre-bronchodilator FEV1 40-80% of predicted normal

Design

- Randomized, double-blind, placebo-controlled
- ~264 patients
- Daily dose for 12-weeks

Dosing

- Three KT-621 doses + one placebo (1:1:1:1)

Endpoints

- Primary endpoint: Percent change from baseline in pre-bronchodilator FEV1 at week 12
- Secondary endpoints include:
  - Change from baseline in ACQ-5, AQLQ

Key Trial Aim

Establish clinical activity and safety in **asthma to select Phase 3 dose to support registrational studies** in multiple respiratory indications

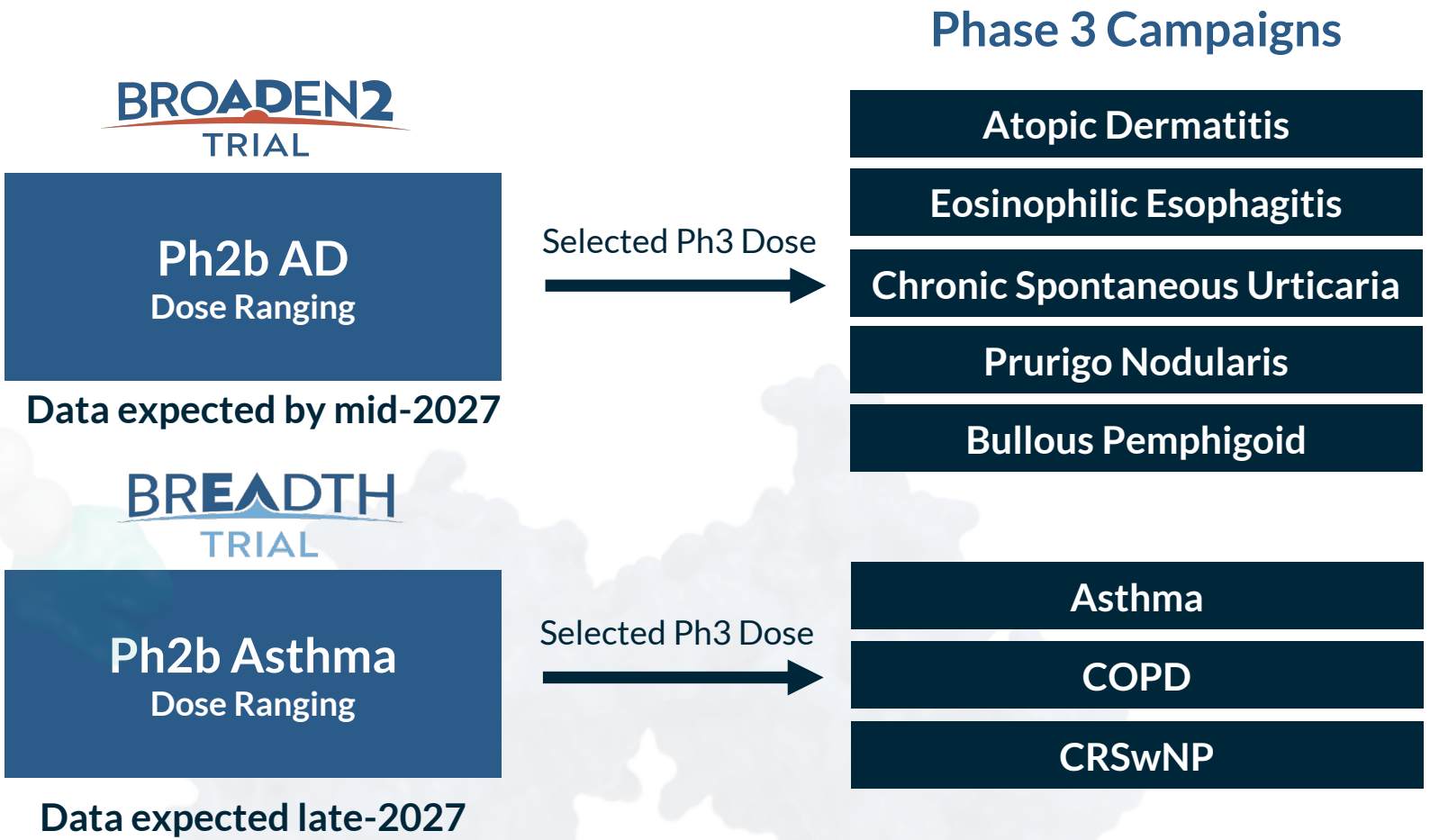
Status update:
Initiated January 2026; Data expected late-2027

Kymera Completes the Clinical Translation of STAT6 Degradation

Study	Data	Potential for "Dupilumab-in-a-pill" Profile
Human Genetics	STAT6 is a key driver of Type 2 inflammation	✓
Preclinical	KT-621 degraded STAT6 and blocked IL-4/13 Type 2-driven inflammation *in vitro/in vivo* as effectively as dupilumab	✓
Phase 1a Healthy Volunteers	KT-621 safely and deeply degraded STAT6, blocking IL-4/13 biomarkers equally or numerically better than dupilumab	✓
BroADen Phase 1b AD Patients	KT-621 safely and deeply degraded STAT6 and demonstrated meaningful improvements on: - Type 2 biomarkers in blood and skin - FeNO in AD and comorbid asthma patients - Clinical endpoints in patients with AD, comorbid asthma and allergic rhinitis **Results in line with or in some cases numerically exceeded published data for dupilumab at week 4**	✓

KT-621 clinical data continues to support STAT6 degradation as a potentially transformative approach for Type 2-driven inflammatory diseases, with a once-a-day, oral drug

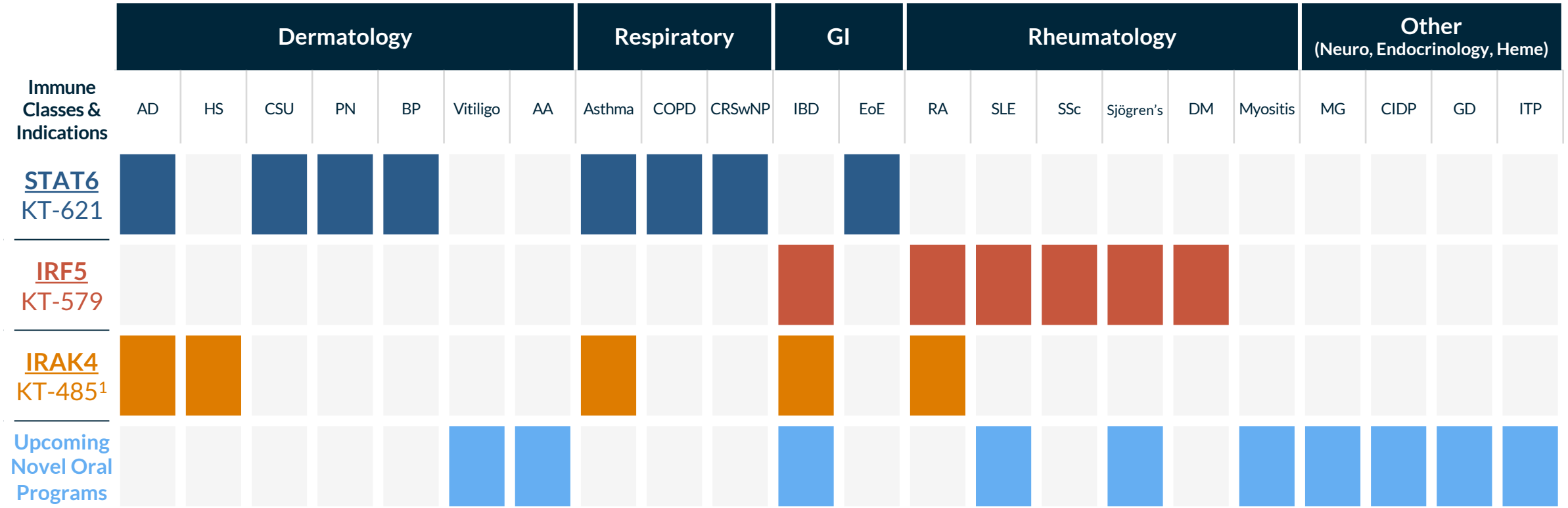
KT-621 Development Plan Enables Efficient Path to Registration Across All Type 2 Diseases



Current parallel Phase 2b trials in moderate to severe AD and asthma have the potential to support subsequent Phase 3 trials across multiple dermatology, GI, and respiratory indications

Opportunity to Address a Wide Range of Immunology Indications

Orals with Biologics-Like Activity to Transform Immunology



Synergies and know-how **across key immunological pathways** creates multiple development and combination opportunities and positions Kymera to expand access to systemic advanced therapies for broad patient populations

AD: atopic dermatitis; HS: hidradenitis suppurativa; CSU: chronic spontaneous urticaria; PN: prurigo nodularis; BP: bullous pemphigoid; AA: alopecia areata; COPD: chronic obstructive pulmonary disease; CRSwNP: chronic rhinosinusitis with nasal polyps; IBD: inflammatory bowel disease; EoE: eosinophilic esophagitis; RA: rheumatoid arthritis; SLE: systemic lupus erythematosus; SSc: systemic sclerosis; DM: dermatomyositis; MG: myasthenia gravis; CIDP: chronic inflammatory demyelinating polyneuropathy; GD: Graves' disease; ITP: immune thrombocytopenic purpura; [1]Diseases where IL-1R/TLR pathway has been implicated in pathogenesis.

2026: Catalysts for Growth

Unlocking high value targets to **revolutionize immunology with oral degrader medicines**

KT-621
First-in-Class Oral STAT6 Degrader

- Complete enrollment in BROADEN2 Phase 2b AD study in 2026; report data by mid-2027
- Advance BREADTH Phase 2b asthma study; report data in late-2027

KT-579
First-in-Class Oral IRF5 Degrader

- Initiate Phase 1 HV trial in Q1 2026
- Report Phase 1 HV data in 2H 2026

Research

- Advance at least one new development candidate towards IND for a first-in-class, oral immunology program in 2026

Partnered Programs

- Collaborate with Sanofi to advance KT-485, oral IRAK4 degrader, into a Phase 1 clinical trial in 2026
- Collaborate with Gilead to advance oral CDK2 molecular glue degrader program in preclinical studies