KYMERA

Revolutionizing Immunology with Oral Medicines

February 2026

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Certain information contained in this presentation and statements made orally during this presentation relate to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company's own internal estimates and research. No head-to-head trials have been conducted comparing KT-621 to dupilumab. Phase 1 clinical data for KT-621 may not be directly comparable to dupilumab's clinical data due to differences in molecule composition, trial protocols, dosing regimens, and patient populations and characteristics. Accordingly, cross-trial comparisons may not be reliable. While the Company believes these third-party studies, publications, surveys and other data to be reliable as of the date of the presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, no independent sources have evaluated the reasonableness or accuracy of the Company's internal estimates or research, and no reliance should be made on any information or statements made in this presentation relating to or based on such internal estimates and research. This presentation contains trademarks, trade names and service marks of other companies, which are the property of their respective owners.

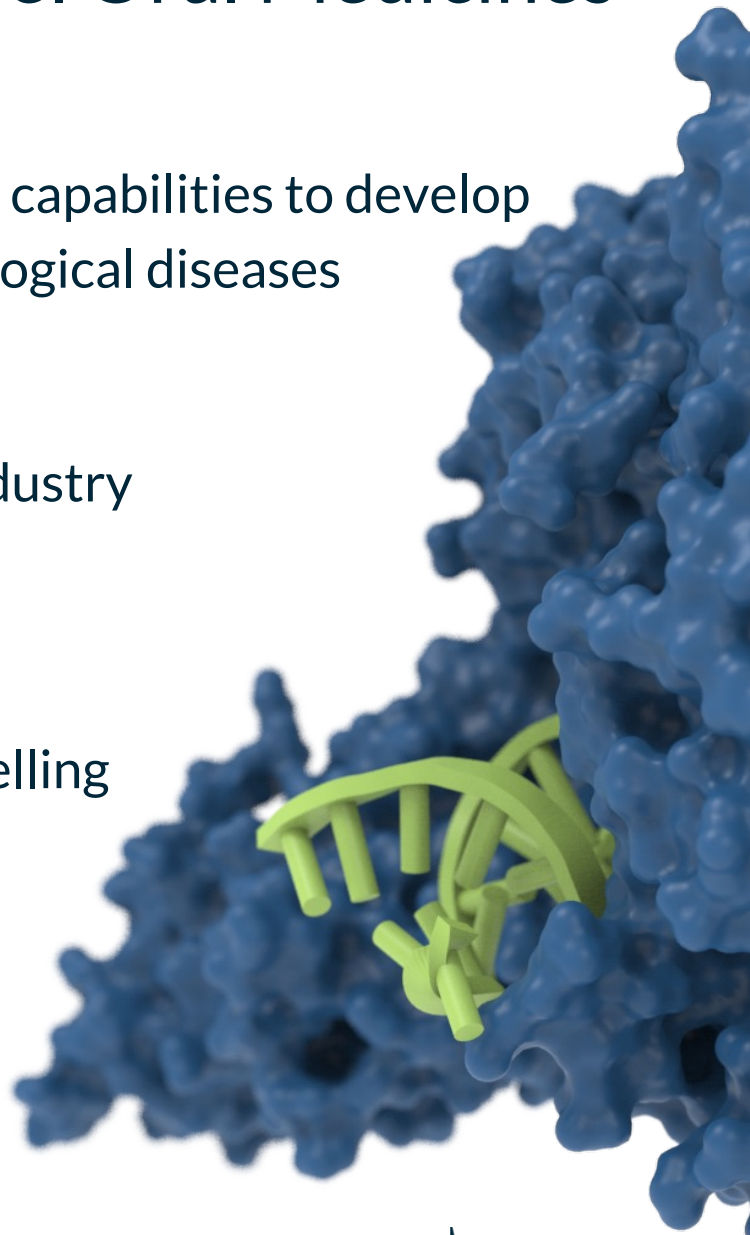
Well Positioned to Redefine Immunology with Novel Oral Medicines

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

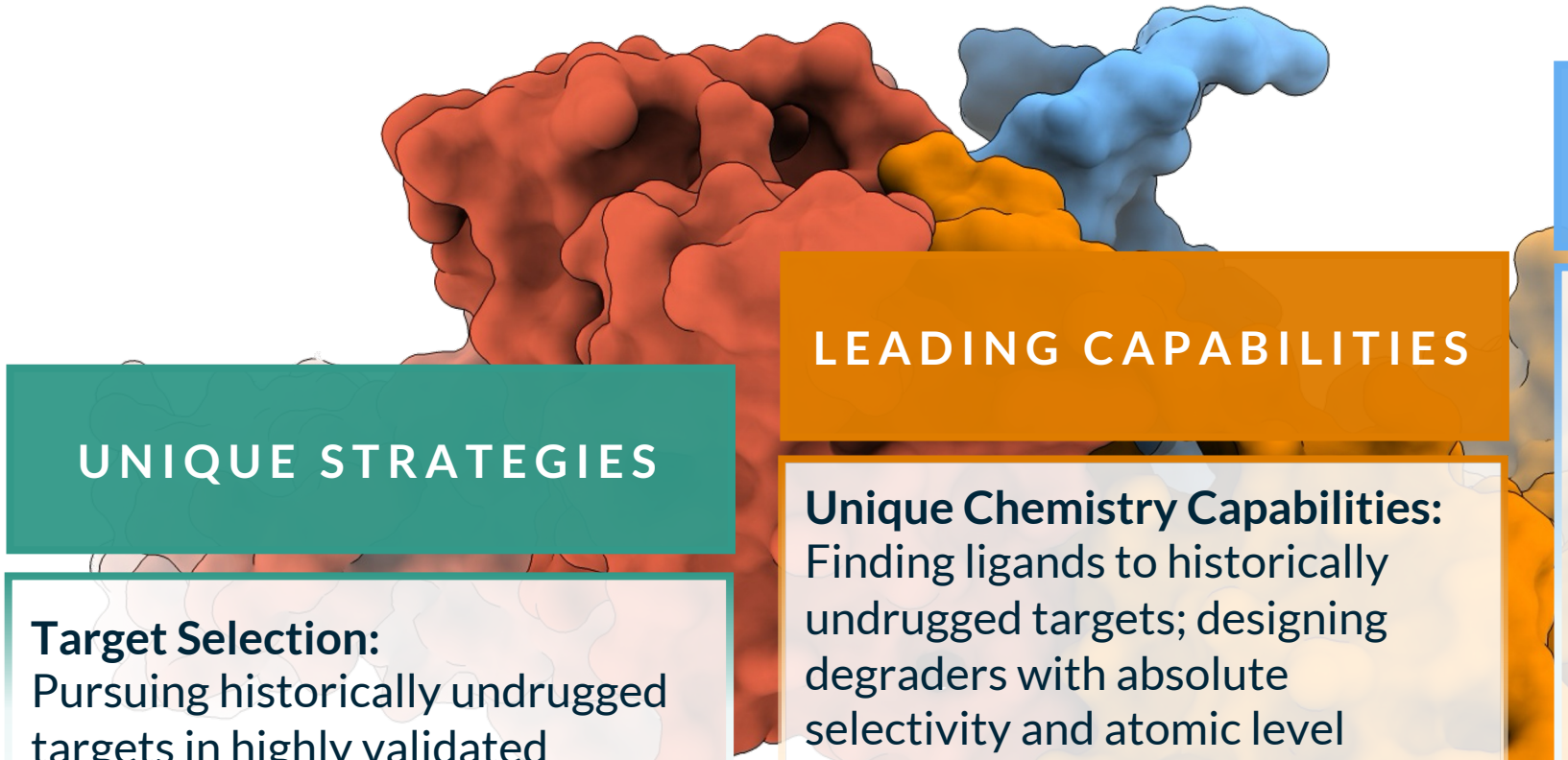
Patient-first mentality, with urgency: Rapidly advancing first-in-industry oral degraders with biologics-like activity

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



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

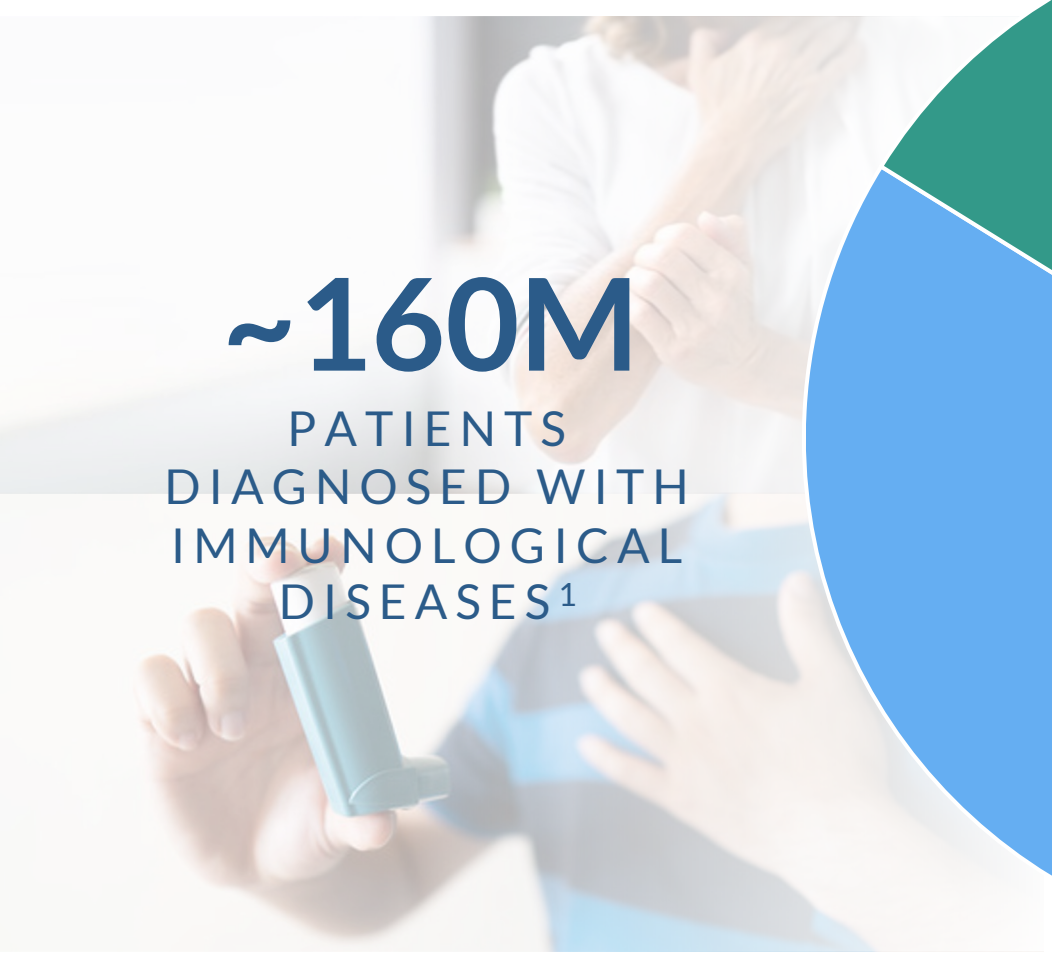
NOVEL INSIGHTS

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

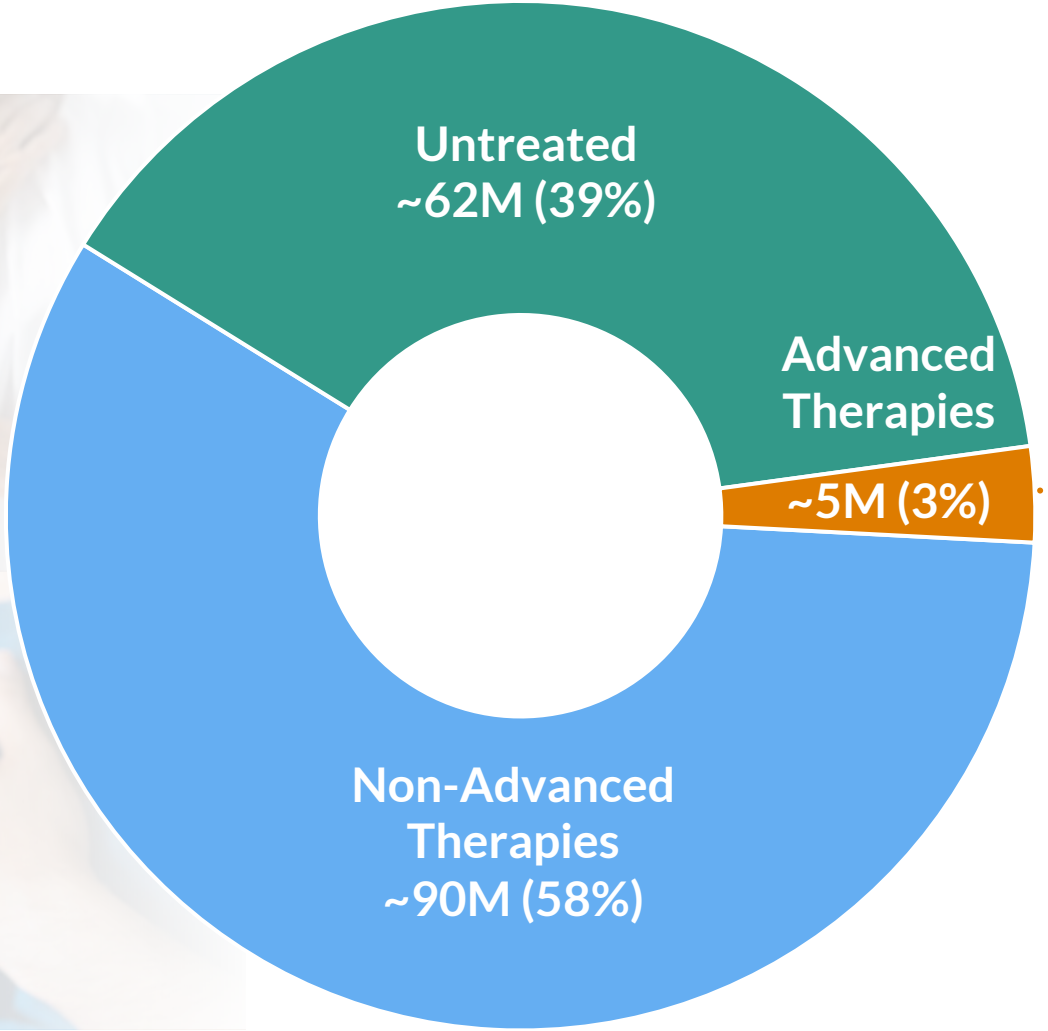
Immunology Remains a Large Underserved Market

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).

Advanced Systemic Therapies Are Mostly Injectable Biologics

Biologics Have Numerous Limitations, Making Orals Preferred by Most Patients

DUPIXENT® (dupilumab) Injection

Skyrizi™ risankizumab-rzaa

VYVGART® (efgartigimod alfa-fcab)

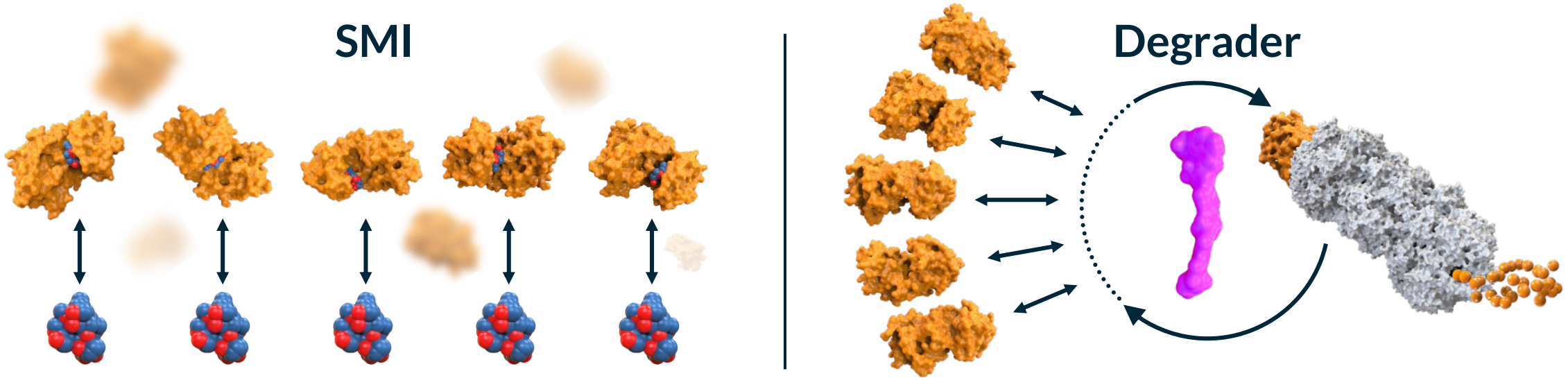
- Inconvenient, burdensome, and often painful for patients
- Immunogenicity risks
- In-office injection training costs HCPs valuable time and resources
- Expensive to manufacture; cold chain delivery/storage

>90%

WOULD SWITCH TO AN ORAL OPTION[1]

[1]Stein Gold et al, Fall Clinical Dermatology Conference, 2025; All trademarks and registered trademarks are the property of their respective owners.

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**), (stoichiometric) requiring continuous high drug exposures to block protein function[1]

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

[1]Mainolfi et al, Annual Reports in Medchem, 2017; PD: Pharmacodynamics; PK: Pharmacokinetics; SMI: Small Molecule Inhibitors.

Unique Target Selection Strategy Drives Best-In-Class Pipeline

Strong genetic/clinical validation

Undrugged or inadequately drugged targets

Large clinical/ commercial opportunities

Clear path to early clinical differentiation

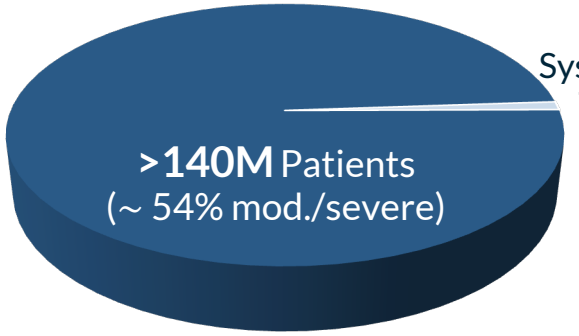
FOCUS ON FIRST- AND BEST-IN-CLASS OPPORTUNITIES

Oral Degraders with Biologics-like Efficacy Can Transform Immunology

Existing Therapies Address 10% or Less of Diagnosed Patients[1]

STAT6
TRANSCRIPTION FACTOR

Key Indications: AD, Asthma, COPD, EoE, CRSwNP, CSU, PN, BP

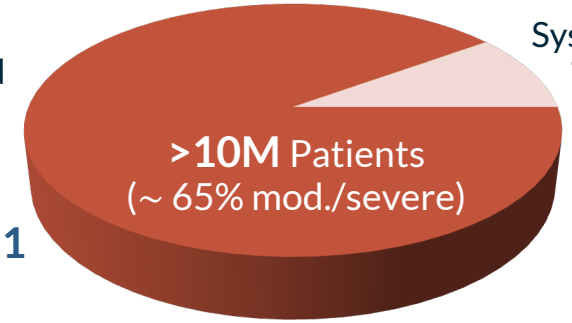


Systemic Advanced Therapies: 1%

>$20B[1]

IRF5
TRANSCRIPTION FACTOR

Key Indications: SLE, RA, UC, CD, Sjögrens, SSc, DM

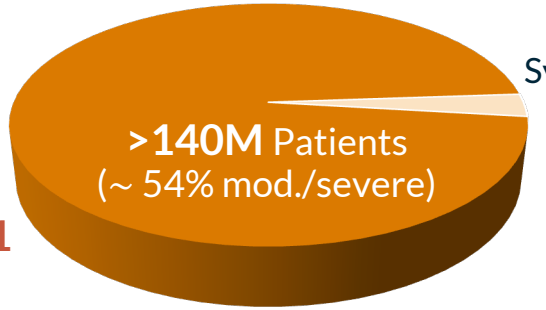


Systemic Advanced Therapies: 7%

>$45B[1]

IRAK4
SCAFFOLDING KINASE

Key Indications[2]: HS, AD, Asthma, COPD, RA, SLE, UC, CD



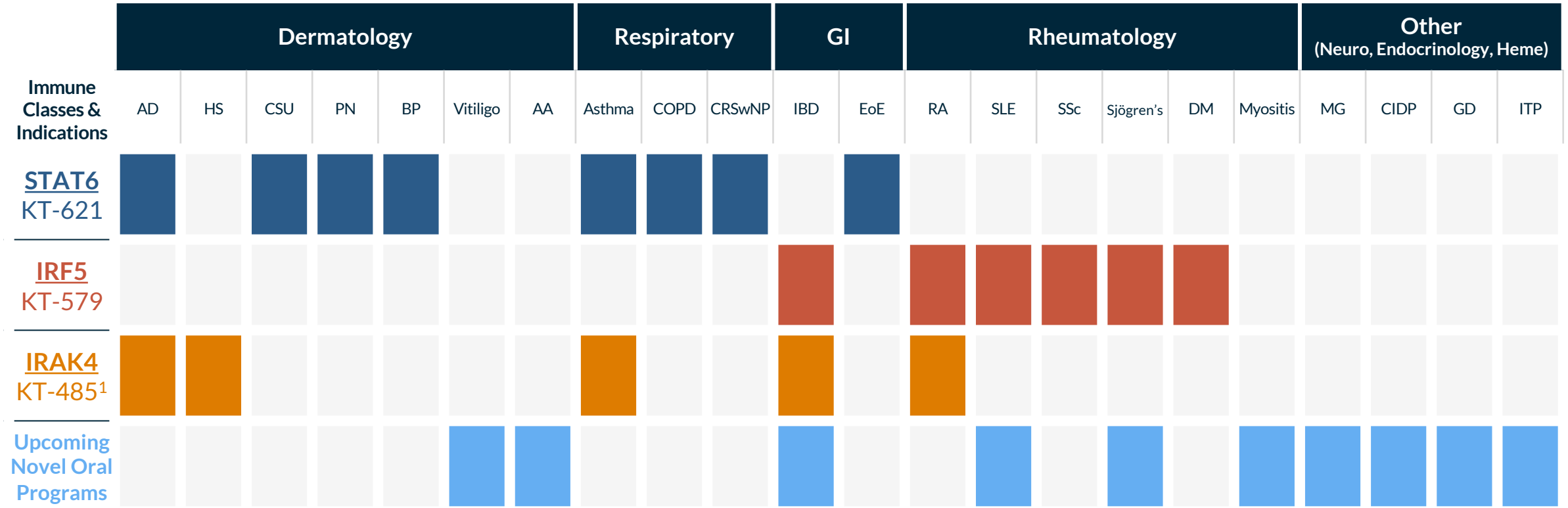
Systemic Advanced Therapies: 3%

>$55B[1]

[1]GlobalData (2023 diagnosed prevalent patient population and forecasted sales for approved systemic advanced therapies in US/EU5/JP; STAT6 estimates include only AD, Asthma, COPD; IRF5 estimates include only SLE, RA, UC, CD; IRAK4 estimates include all noted indications); [2]Diseases where IL-1R/TLR pathway has been implicated in pathogenesis.

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

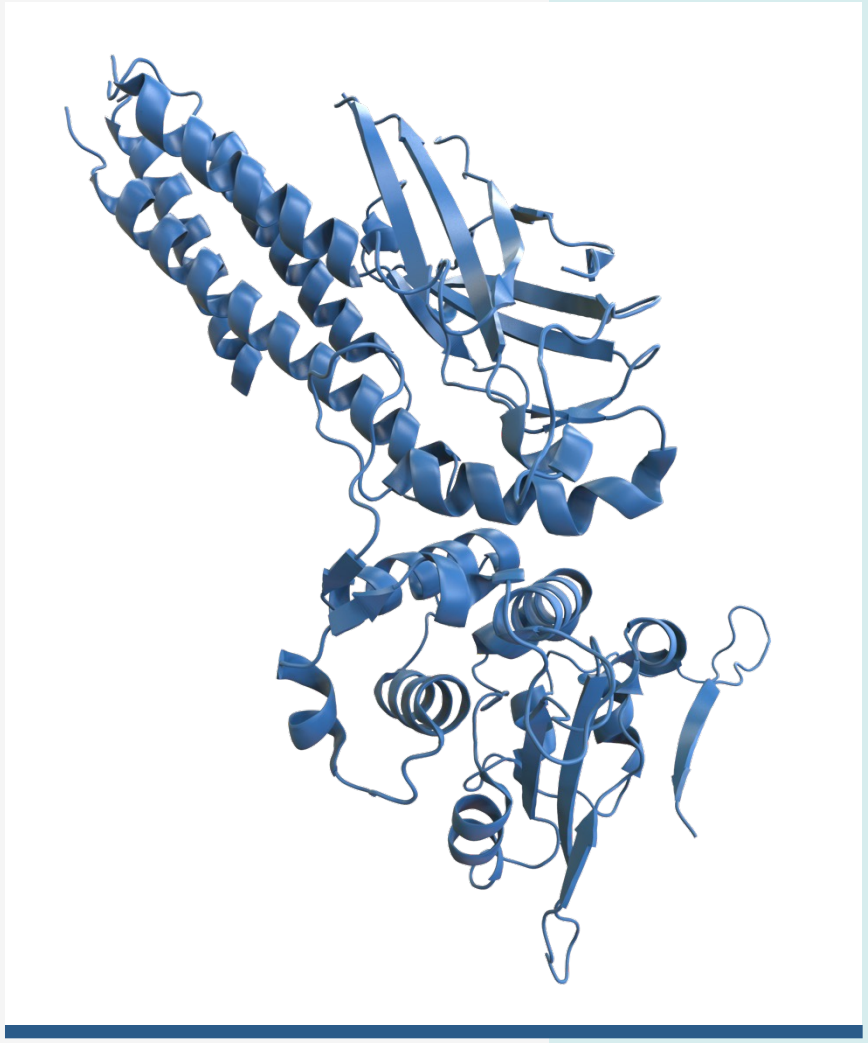
AD: atopic dermatitis; HS: hidradenitis suppurativa; CSU: chronic spontaneous urticaria; PN: prurigo nodularis; BP: bullous pemphigoid; AA: alopecia areata; COPD: chronic obstructive pulmonary disease; CRSwNP: chronic rhinosinusitis with nasal polyps; IBD: inflammatory bowel disease; EoE: eosinophilic 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.

Building a Best-In-Industry Oral Immunology Pipeline

	Potential Indications	Patient Opp.[1]	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	>140M	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	>10M	KT-579				**Ph1 HV Data:** 2H26
Partnered Programs							
IRAK4[2]	HS, AD, RA, Asthma, IBD, others[3]	>140M	KT-485				**Ph1 Start:** 2026 sanofi
CDK2[4]	Breast cancer and other solid tumors	>500K	Molecular Glue				**Option Exercise** 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]GlobalData (2023 diagnosed prevalent patient population in US/EU5/JP; STAT6 estimates include only AD, Asthma, COPD; IRF5 estimates include only SLE, RA, UC, CD; IRAK4 estimates include all noted indications; CDK2 estimates include all HR+/HER2- BC settings); [2]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; [3]Diseases where IL-1R/TLR pathway has been implicated in pathogenesis; [4]Partnered with Gilead, exclusive option and license agreement to accelerate the development and commercialization of a novel molecular glue degrader program.

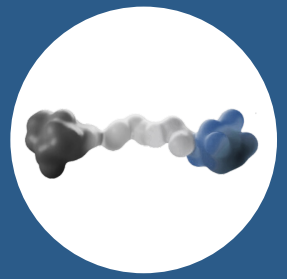


First-in-Class Oral STAT6 Degrader Program

Dupilumab-like activity in a pill

KT-621 Summary

Dupilumab-like Activity in a Pill



KT-621 is the **first highly selective, potent, oral STAT6 degrader** in the clinic

- In preclinical *in vitro and in vivo* studies, it is equal or superior to dupilumab
- Phase 1b AD data showed deep STAT6 degradation in blood and skin, robust reductions in disease-relevant Type 2 biomarkers, meaningful improvements on clinical endpoints and PROs in AD and comorbid asthma and allergic rhinitis, and a favorable safety profile

OPPORTUNITY

- **Over 140M**[1] potential patient impact; only ~1% has access to advanced systemic therapies
- Market (with single digit % penetration) projected to **reach >$27B** by 2030[1]
- An **oral pill with dupi-like efficacy** can transform the treatment paradigm of Type 2 diseases

→ **Potential to access patients beyond biologics-eligible, across all disease severities and ages**

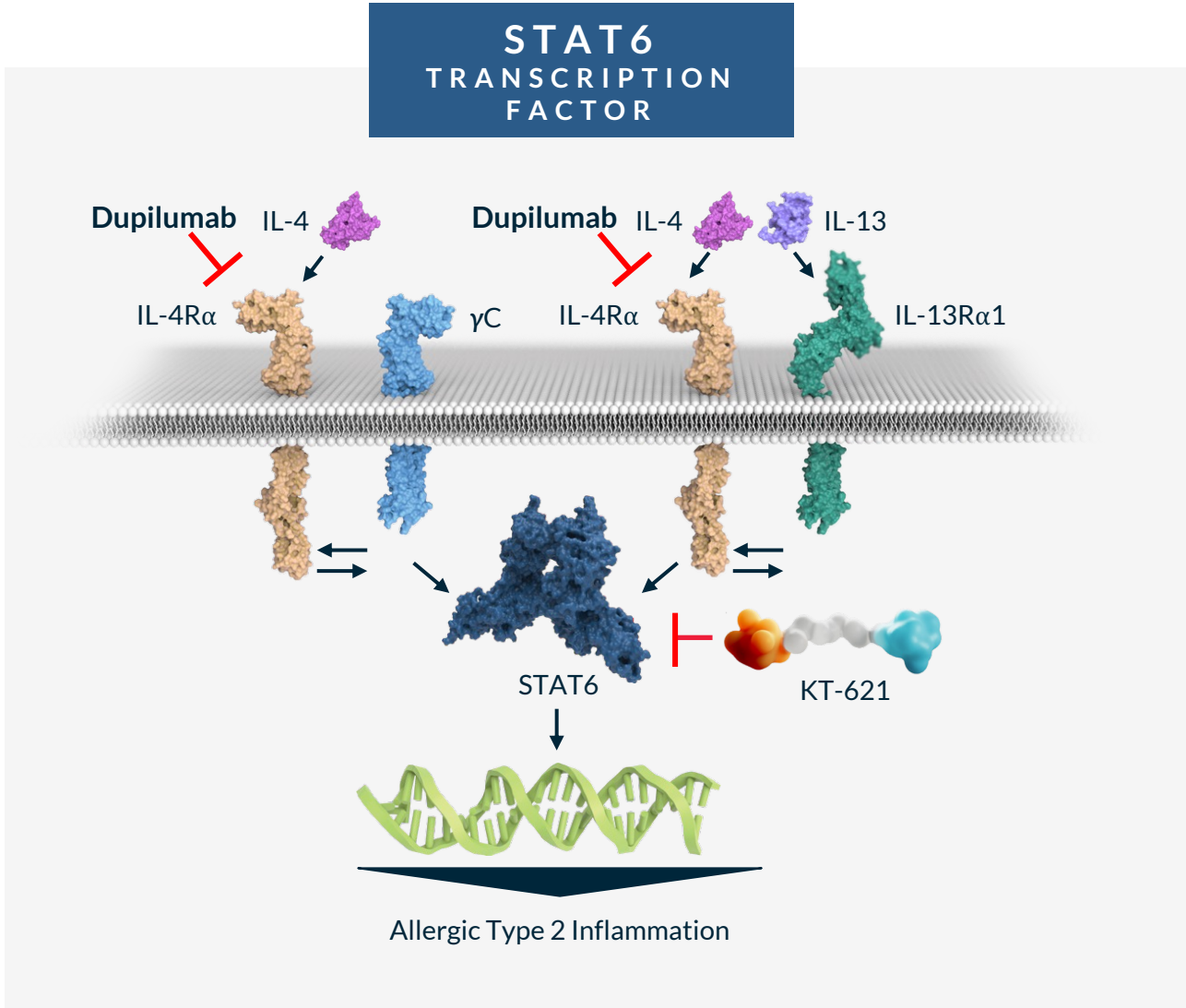
STATUS & UPCOMING MILESTONES

- BROADEN2 Phase 2b AD trial ongoing, with data by mid-2027
- BREADTH Phase 2b asthma trial ongoing, with data in late-2027

[1]GlobalData (2023 diagnosed prevalent patient population and forecasted sales of systemic advanced therapies for AD, Asthma, and COPD only in US/EU5/JP).

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

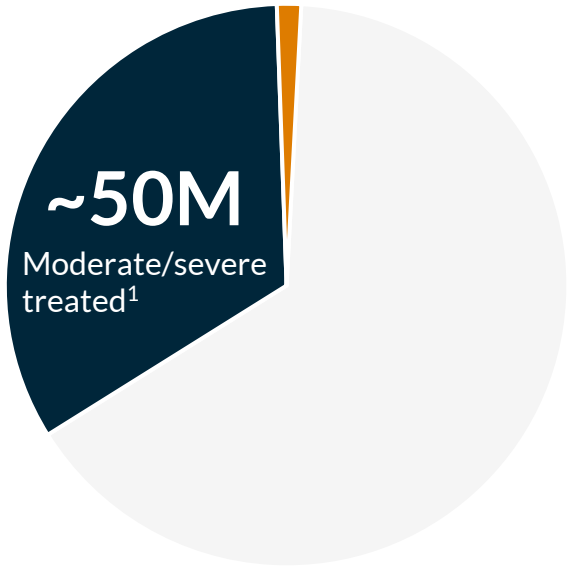


Sharma et al, 2023; Takeda et al, 1996; Kaplan et al, 1996; Junttila, 2018; Kolkhir et al, 2023; AD: Atopic Dermatitis; COPD: Chronic Obstructive Pulmonary Disease; EoE: Eosinophilic Esophagitis; CRSwNP: Chronic Rhinosinusitis with Nasal Polyps; CSU: Chronic Spontaneous Urticaria; PN: Prurigo Nodularis;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

Most Patients with Type 2 Diseases Remain Underserved

~2M

Patients treated with advanced systemic therapy[1]



>140M

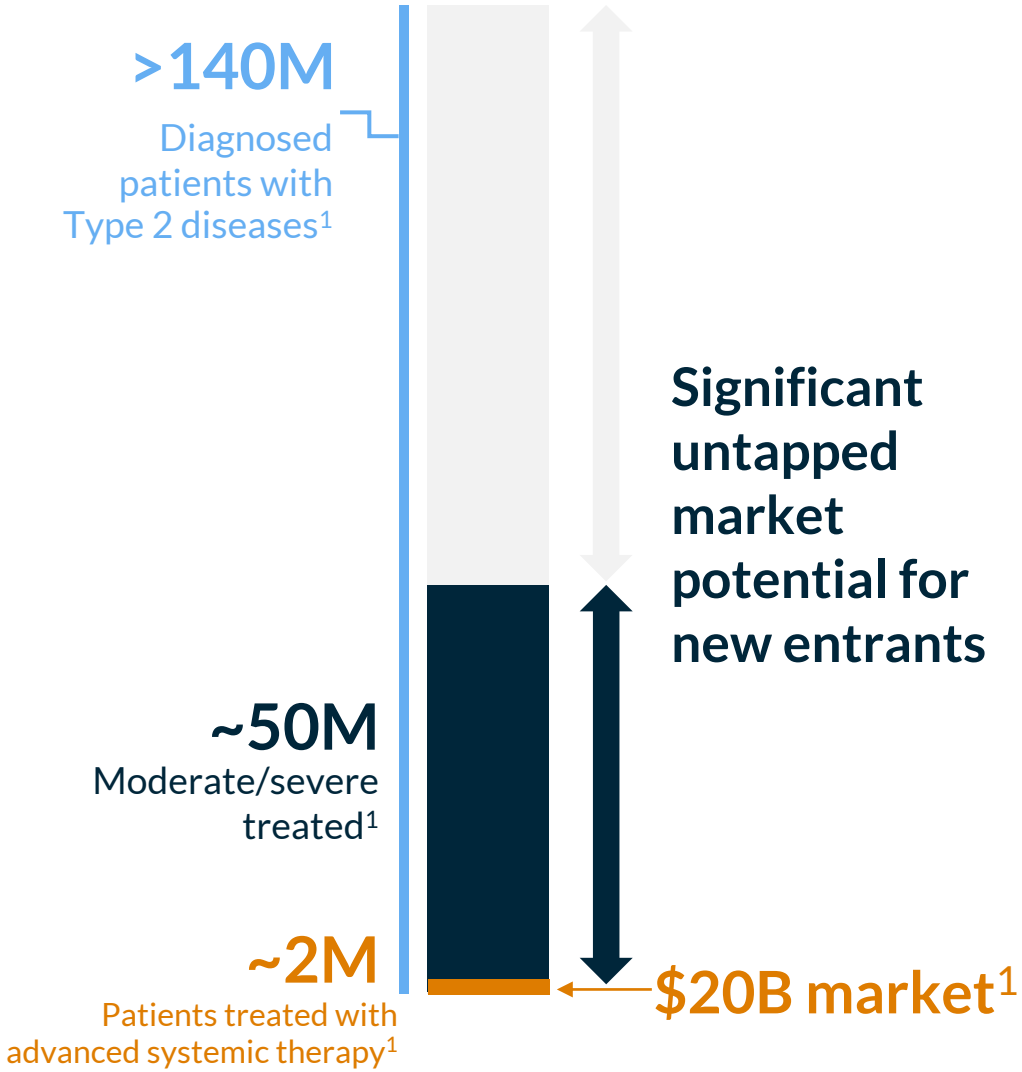
Diagnosed patients with Type 2 diseases[1]

Barriers Built Into the Current Treatment Paradigm

Local Therapies (e.g., topicals, inhalers)	• Do not address underlying drivers of Type 2 disease, only manage symptoms • Insufficient for most moderate/severe patients
Current Oral Therapies (e.g., JAKs, LTRAs, oral steroids)	• Come with serious efficacy and/or safety limitations; black box warnings (cancer, cardiovascular, suicidal thoughts) • Required blood draws for initiation and monitoring
Injectable Therapies (e.g., biologics) *>75% of systemic therapies but associated with high treatment burden*	• Significant injection site pain and needle fear/fatigue • Burdensome loading injections to initiate (4-5 in first month) • Poor persistence (i.e., high drop-off rates) • Cold storage requirement

[1]GlobalData (2023 diagnosed prevalent or treated patient population for AD, Asthma, and COPD in US/EU5/JP.

Unlocking the Full Market Potential in Type 2 Disease



Innovation Drives Growth

- New products and mechanisms have historically expanded the market by reaching more patients

Reducing Patient Burden Could Unlock Access

- An oral therapy that addresses many of the limitations of current therapies while not compromising on safety or efficacy will, for the first time, offer a true alternative for millions of patients of all ages

A Proven Pattern in Expansion

- As demonstrated by the 5x growth of the Psoriasis market over the past ten years thanks to new entrants and orals[2]; AD, asthma, and other Type 2 markets are poised for substantial growth well beyond the current $20B

[1]GlobalData (2023 diagnosed prevalent or treated patient population and forecasted sales for AD, Asthma, and COPD in US/EU5/JP; [2]Evaluate Ltd (Total WW Market Value Top 10 Products 2014-2024).

KT-621 Has Significant Potential to Transform the Treatment of Type 2 Diseases

CURRENT TREATMENT LIMITATIONS

- x Efficacy and incomplete disease control
- x Side effects and safety issues
- x Extremely high treatment burden (multiple, frequent, painful injections, cold storage)
- x Process-intensive care (operational requirements, blood testing, pharmacy hurdles)

KT-621 PARADIGM SHIFT

- ✓ Potential for **biologics-like safety and efficacy** with robust durability
- ✓ Highly validated, **non-immunosuppressive pathway** supporting long-term use
- ✓ Differentiated approach to **address underlying disease** biology
- ✓ Convenience of **once-daily oral administration** with no blood testing/monitoring or injections required

>90%

OF PATIENTS WOULD SWITCH TO AN ORAL OPTION[1]

KT-621 has the opportunity to be the first agent for many Type 2 diseases in areas of vast unmet need for patients of all ages

[1]Stein Gold et al, Fall Clinical Dermatology Conference, 2025.

Opportunity to Shift Treatment Paradigms for Type 2 Diseases

Given the first-in-class profile of KT-621, there is significant opportunity to meet uncontrolled AD and Asthma patients earlier in their treatment journey

Atopic Dermatitis[1]

- **Non-Rx. Management** (Emollients, bathing, trigger avoidance)
- **Topicals** (Steroids, Calcineurin, PDE4, JAK, AHR)
- **Injectables** (IL-4Ra, IL-13, IL-31 biologics)*

OPPORTUNITY WITH KT-621 TO RETHINK EXISTING PARADIGMS

Effective & Safe Oral Therapy (KT-621)

Asthma[2]

GINA 1	GINA 2	GINA 3	GINA 4	GINA 5
LD ICS (As needed)	LD ICS (Maint.)	LD ICS + LABA	MD ICS + LABA	HD ICS + LABA ± LAMA ± Biologic

[1]AD Clinical Guidelines (AAD, 2024); [2]Global Strategy for Asthma Management and Prevention (GINA, 2024); ICS: inhaled corticosteroid; LD: low dose; HD: high dose; LABA: long-acting beta agonist; LAMA: long acting muscarinic antagonist; *Category includes oral JAK inhibitors.

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

Ph1 HV
SAD-MAD

✓ Complete

Ph1b AD
Single Arm, Open Label

✓ Complete

BROADEN STUDY

BROADEN2 TRIAL

Ph2b AD
Dose Ranging

Ongoing
Data expected by mid-2027

Ph3 Dose

Ph3: AD (Initial), EoE, CSU, PN, BP

BREADTH TRIAL

Ph2b Asthma
Dose Ranging

Ongoing
Data expected late-2027

Ph3 Dose

Ph3: Asthma (Initial), COPD, CRSwNP

Initial 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

KT-621 Data Provide Validation and Derisk Future Clinical Trials

Preclinical

POTENCY

- <100 pM DC90 in all relevant human cell types

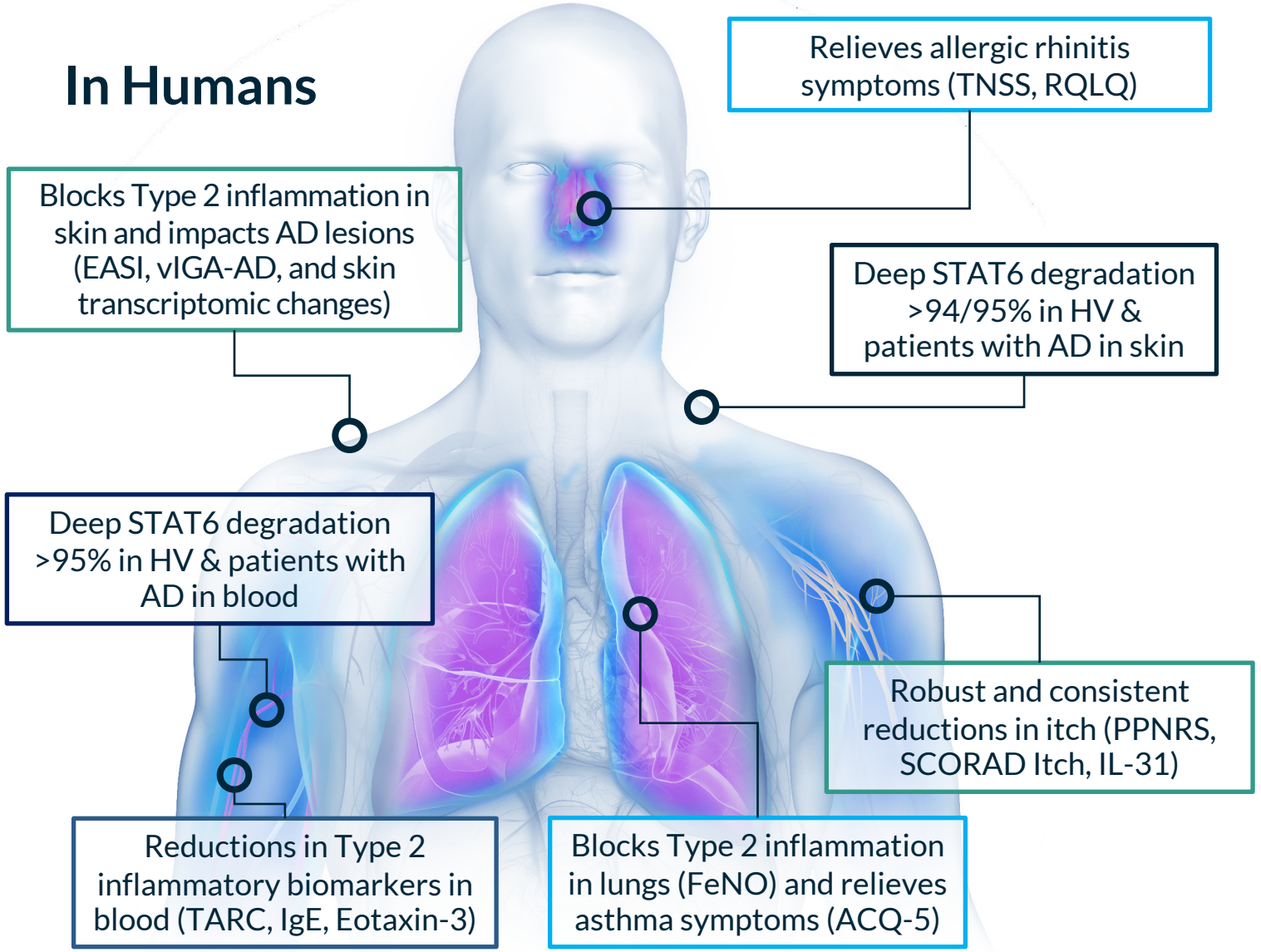
EFFICACY

- 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
  - 6/9 months rat and NHP GLP chronic tox
  - Embryofetal development tox in rat, rabbit and NHP

In Humans



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;
Data expected by mid-2027

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 cells/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: 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

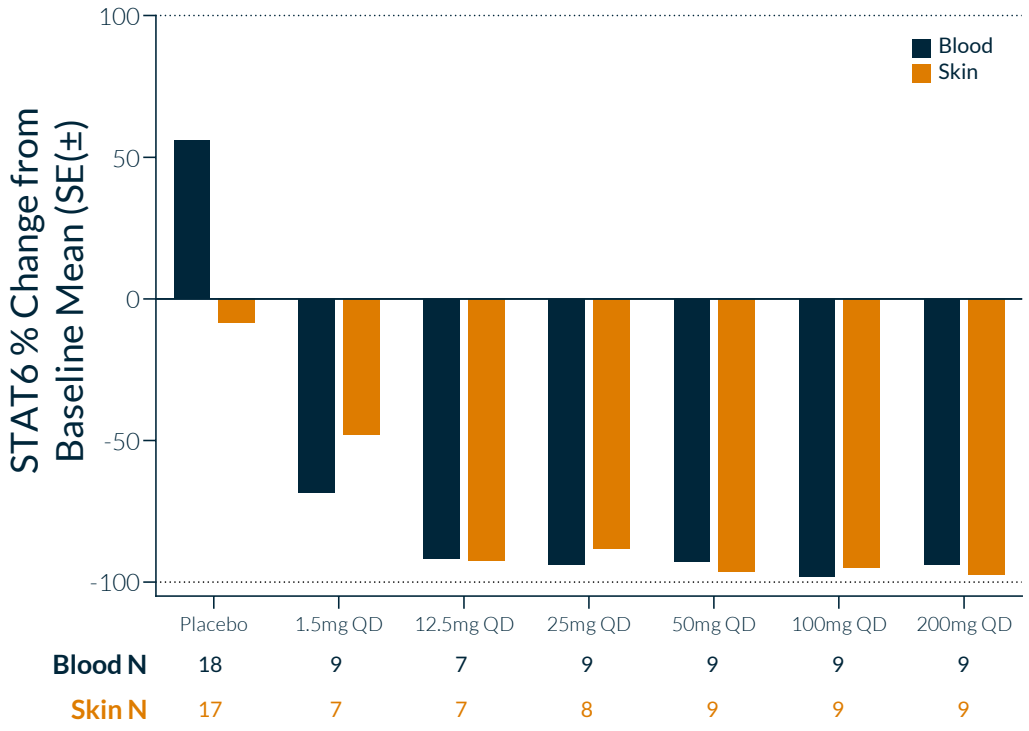
Status update:
Ongoing;
Data expected late-2027

KT-621 Phase 1a Healthy Volunteer and Phase 1b BroADen AD Clinical Trial Data

KT-621: Compelling Clinical Profile

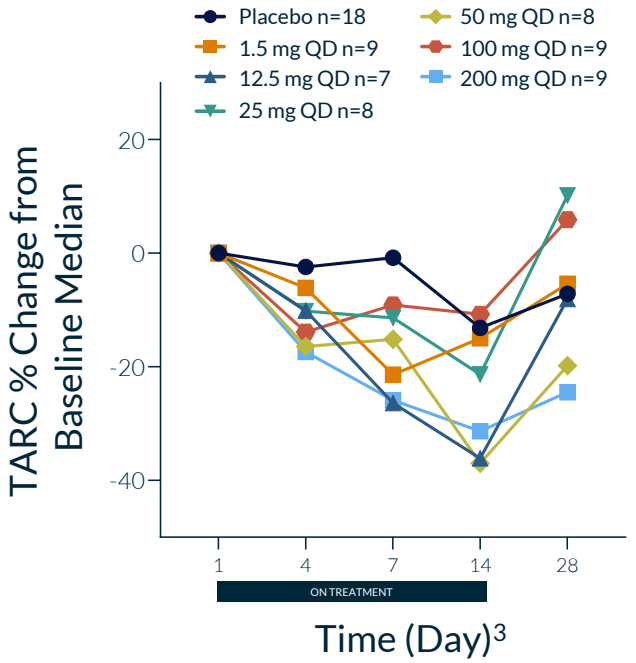
KT-621 Phase 1 Healthy Volunteer Data Suggests Potential for Dupilumab-like Activity in a Pill

Robust STAT6 Degradation in Blood and Skin Following Low Daily Oral Doses

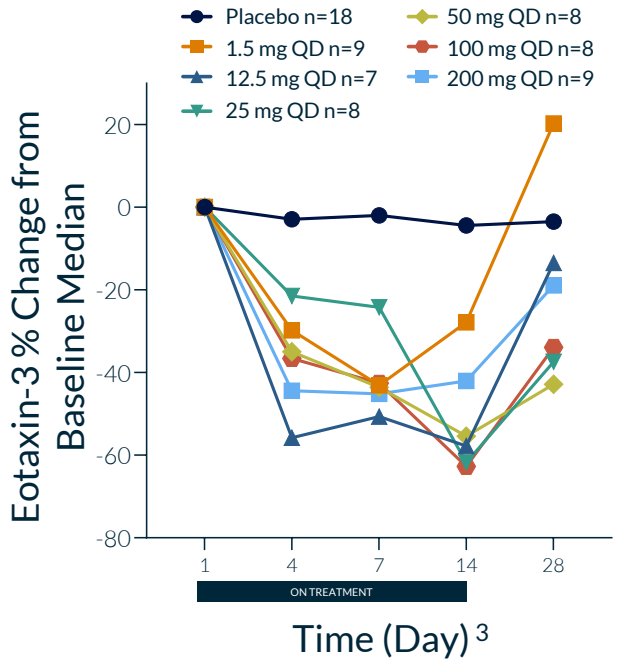


Reductions in Multiple Disease-Relevant Type 2 Biomarkers

Serum TARC[1] with 14-Day KT-621 Dosing



Serum Eotaxin-3[2] with 14-Day KT-621 Dosing



Favorable safety profile: well-tolerated across all dose levels with safety profile undifferentiated from placebo

[1]TARC levels measured in serum using MSD VPLEX; [2]Eotaxin-3 levels measured in serum using MSD VPLEX; [3]Compound dosed on Day 1; For more information on KT-621's Phase 1 healthy volunteer clinical trial, visit Kymera's website.

BroADen KT-621 Phase 1b AD Study Surpassed Objectives

Effects on All Type 2 Biomarkers and Clinical Endpoints Were in Line with or in Some Cases Numerically Exceeded Published Data for Dupilumab at Week 4

Endpoints	BroADen Phase 1b Results Highlights
STAT6 Degradation	✓ Strong fidelity of translation from Phase 1a healthy volunteer study to AD patients with deep STAT6 degradation in blood and skin
Type 2 Biomarkers	✓ Robust reductions in Type 2 biomarkers in blood, skin lesions and lung (FeNO)
Clinical Endpoints	✓ Meaningful improvements of clinical endpoints and patient-reported outcomes in AD, comorbid asthma and allergic rhinitis
Safety	✓ Well-tolerated and favorable safety profile similar to Phase 1a healthy volunteer study

Clinical data continue to support a potential dupilumab-like profile in AD and other Type 2 diseases with a once daily, oral drug

No head-to-head trials have been conducted comparing KT-621 to dupilumab. Phase 1 clinical data for KT-621 may not be directly comparable to dupilumab's clinical data due to differences in molecule composition, trial protocols, dosing regimens, and patient populations and characteristics. Accordingly, cross-trial comparisons may not be reliable.

BROADEN STUDY

Phase 1b in Moderate to Severe Atopic Dermatitis Patients

Baseline entry criteria:

- Adult, moderate to severe AD patients

EASI	**≥16**
vIGA-AD	**≥3**
PPNRS	**≥4**
BSA	**≥10%**

- Documented TCS (Topical Corticosteroid) failure for AD
- Prior biologics allowed, after washout, if patient has responded to treatment
- Concurrent medications for AD not permitted

Design

- Single arm, open label
- 22 patients
- Oral, once daily dose for 28 days
- 14-day follow-up after dosing completed

Dosing

- Two sequential dose cohorts
  - 100 mg (10 patients)
  - 200 mg (12 patients)

Endpoints

- Safety
- Pharmacokinetics
- STAT6 degradation
- Type 2 biomarkers in blood, skin lesions and lung
- Clinical activity (EASI, PPNRS, vIGA-AD, patient-reported outcomes)

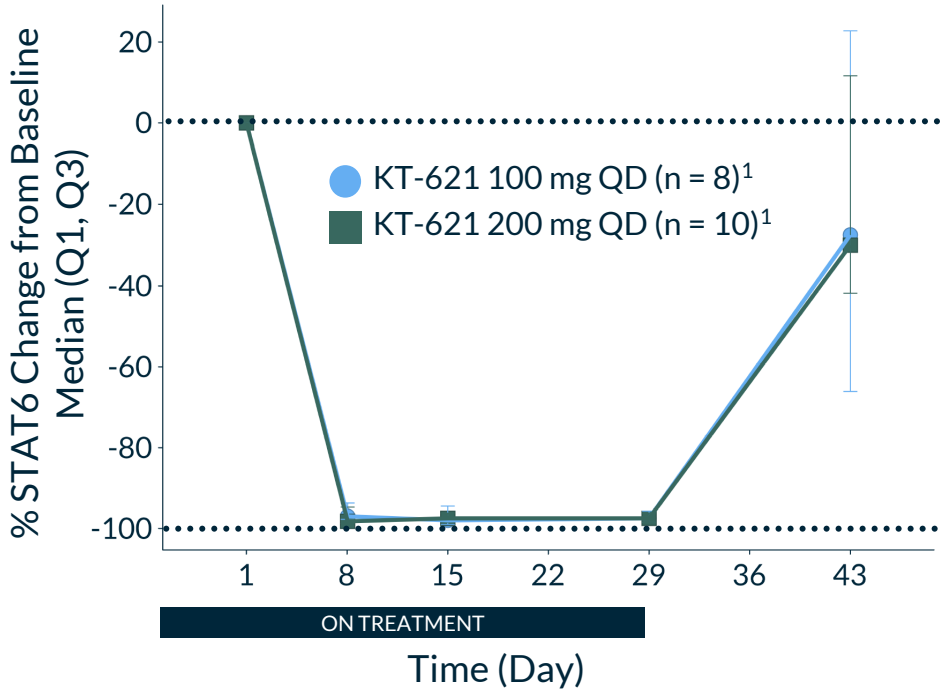
EASI: Eczema Area and Severity Index; vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis; PPNRS: Peak Pruritus Numerical Rating Scale; BSA: Body Surface Area; TCS: Topical Corticosteroid.

KT-621 Achieved Deep STAT6 Degradation in Blood and Skin

Degradation Maintained for 28 Days Across Both Dose Cohorts

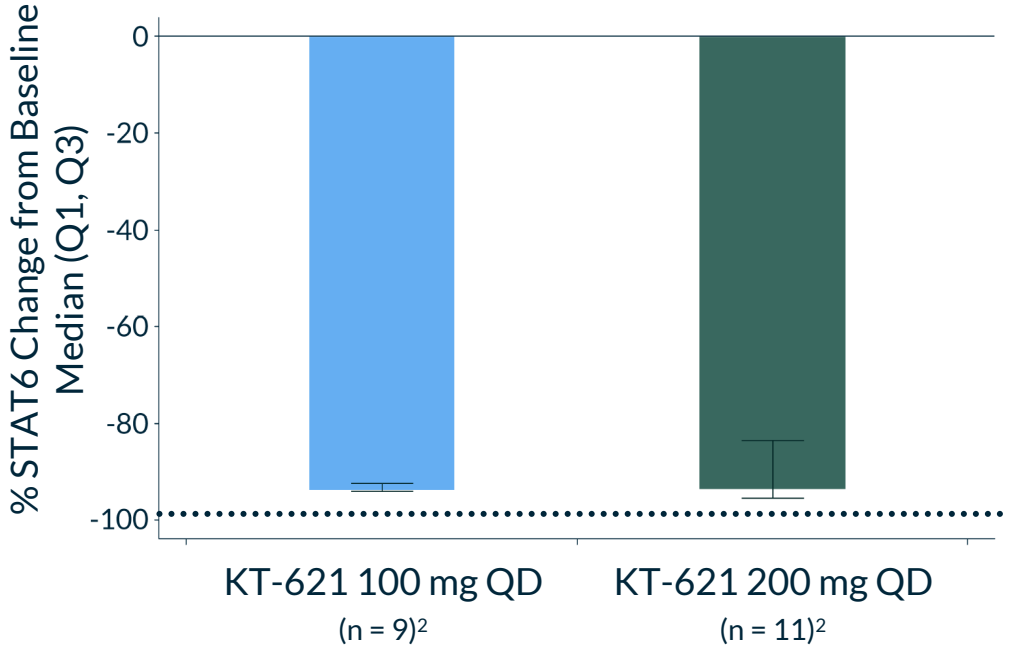
Median STAT6 Degradation in Blood

Method: Flow Cytometry



Median STAT6 Degradation in Skin

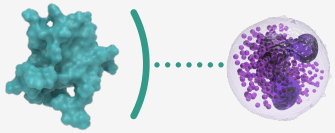
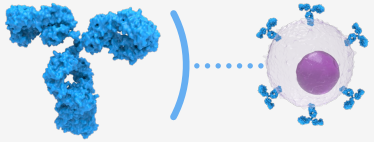
Method: Targeted Mass Spec



- Median STAT6 degradation of 98% in blood in both dose groups maintained throughout the treatment period
- Deep skin degradation of 94% in both dose groups with multiple patients' STAT6 levels below the LLOQ (lower limit of quantification)

Note: N values reflect the number of participants with available samples at Day 29; [1]Two patients (one each in 100 mg and 200 mg dose groups) did not have baseline samples collected, and two D29 samples were unevaluable due to shipping issues that led to loss of stability; [2]Two patients did not consent to D29 biopsies; PK: Pharmacokinetics.

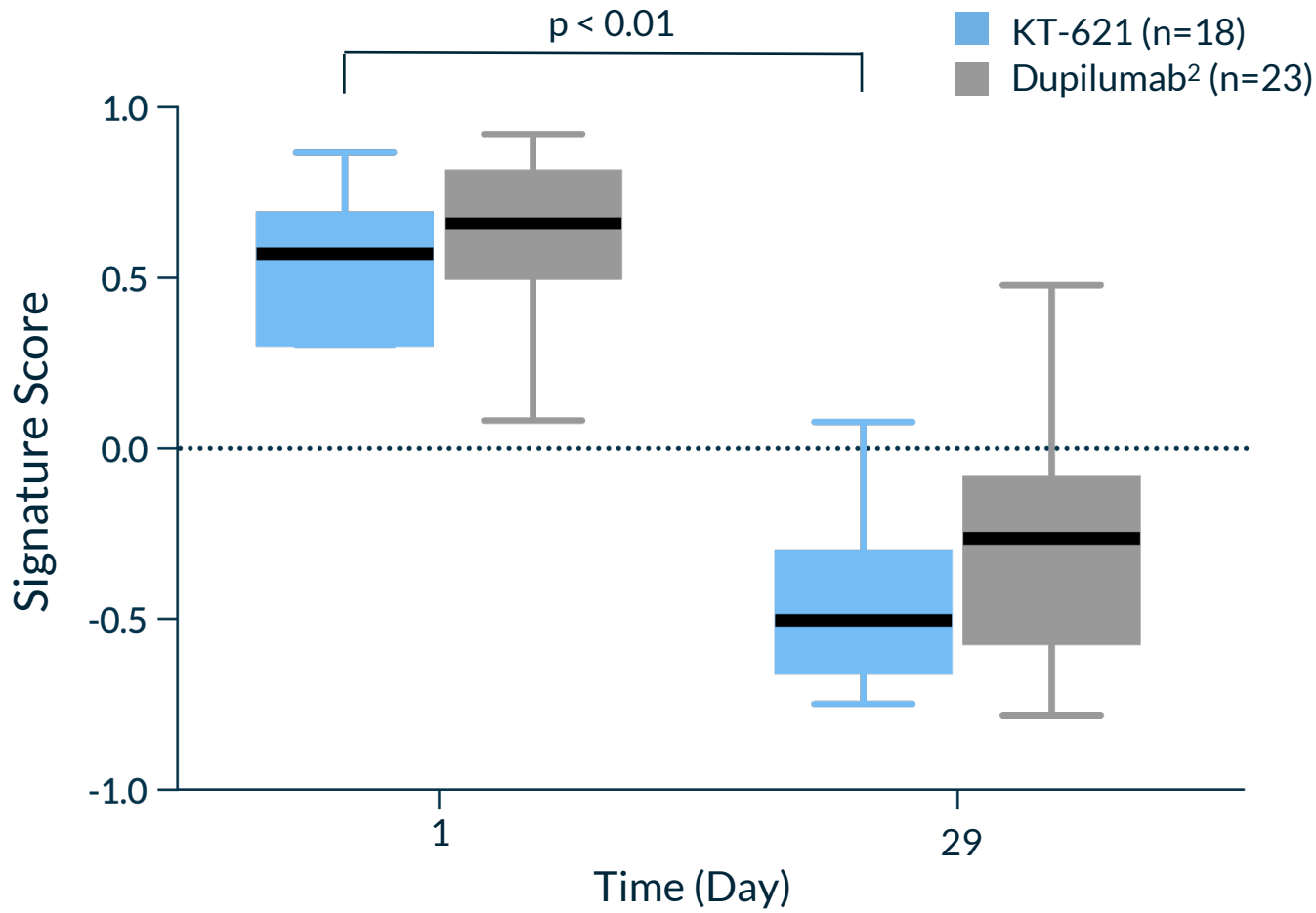
Robust Impact on All Disease-Relevant Biomarkers of Type 2 Inflammation in BroADen Phase 1b AD Trial

	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 Significantly Downregulated Core Type 2 Inflammation Gene Set in Skin Lesions of AD Patients

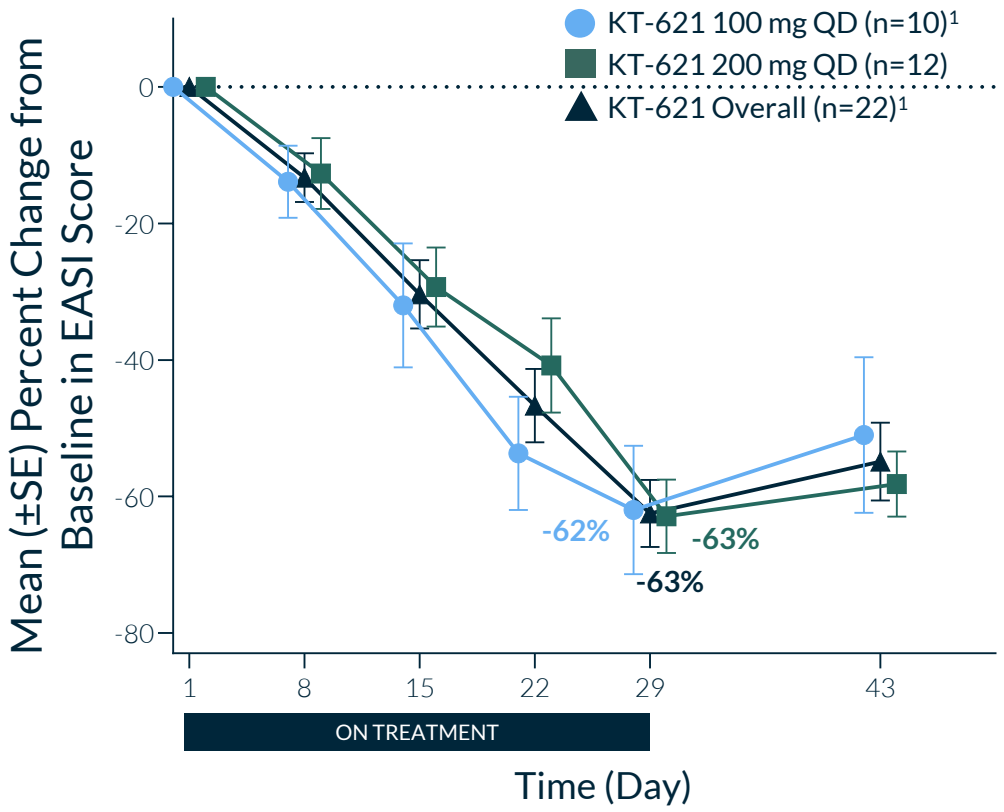
- **Genes in signature[1]:**
  - CCL26/Eotaxin-3
  - CCL17/TARC
  - CCL18/PARC
  - CCL13/MCP-4
- Changes in transcriptome comparable to published data for dupilumab at week 4[2]



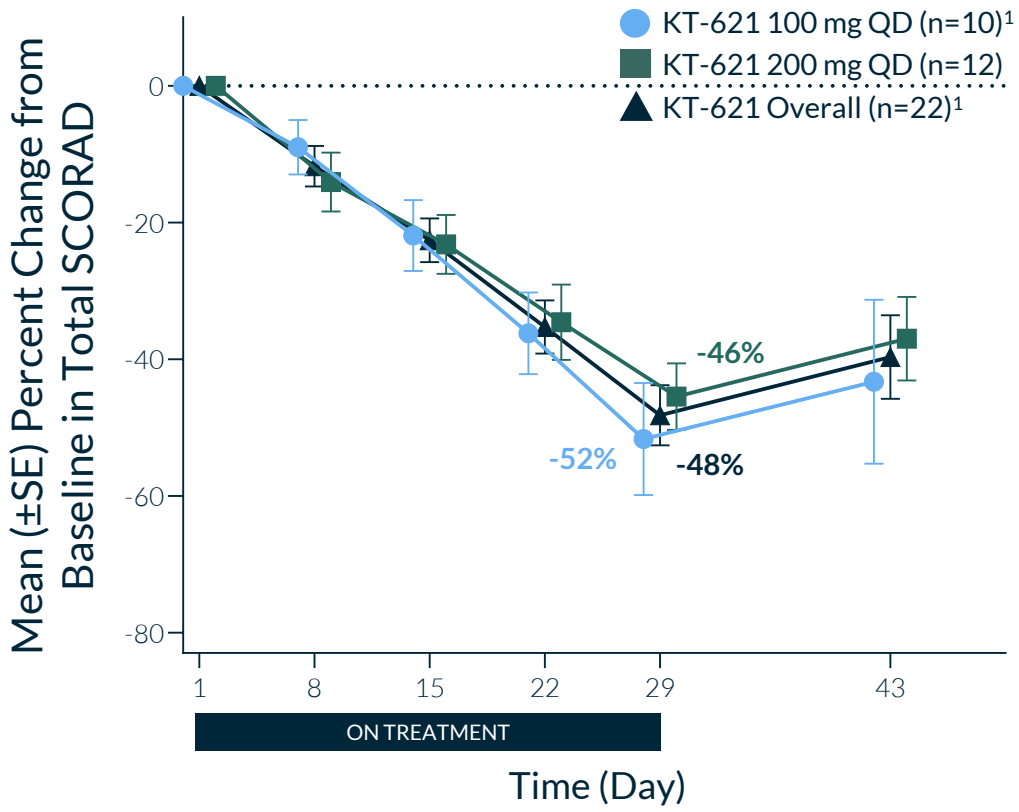
Note: N values reflect the number of participants with available samples at Day 29; In the 100 mg group, one patient did not consent to biopsy; In the 200 mg group, one patient did not consent to D29 biopsy, one patient D29 biopsy was lost, and one patient had poor quality sequencing; [1]Signature scores generated by GSVA (Hänzelmann et al, BMC Bioinformatics, 2013); [2]Dupilumab data from Guttman-Yassky et al, JACI, 2019; P-value < 0.01 for paired t-test between D1 and D29 for both treatments.

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

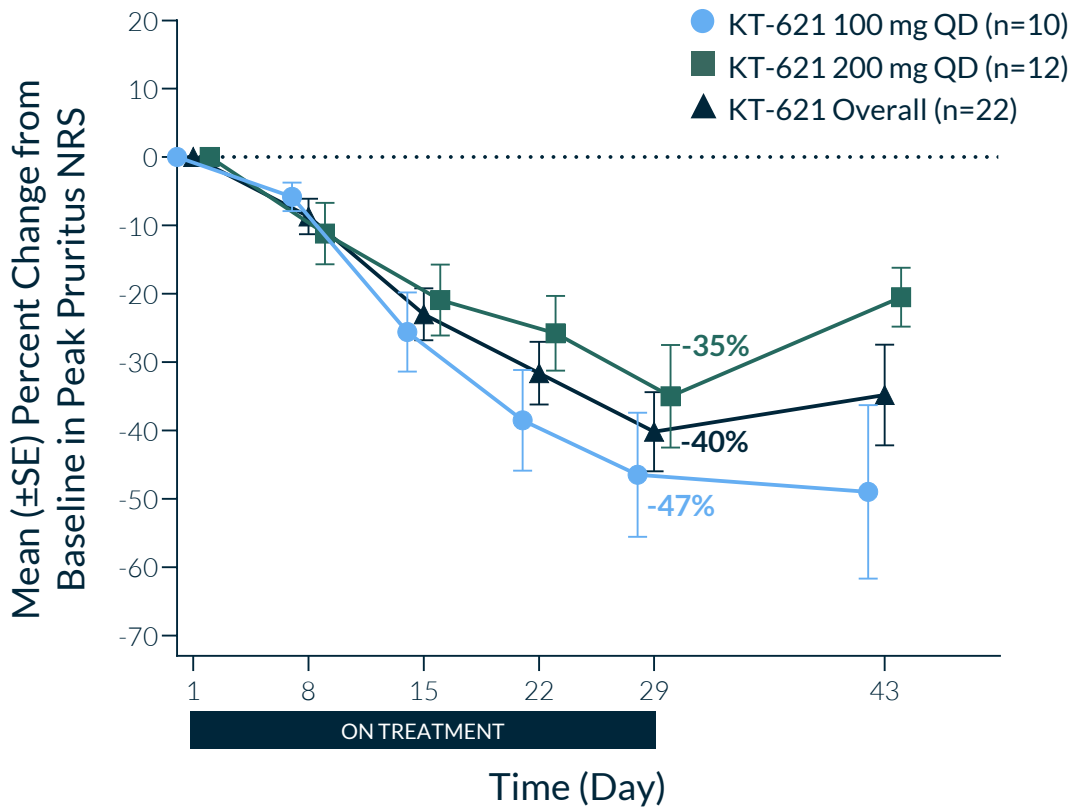


- Reductions seen as early as Day 8 without apparent plateau during treatment duration
- Achieved robust clinical improvement across EASI-50 (76% overall), EASI-75 (29% overall) and vIGA-AD (19% overall)

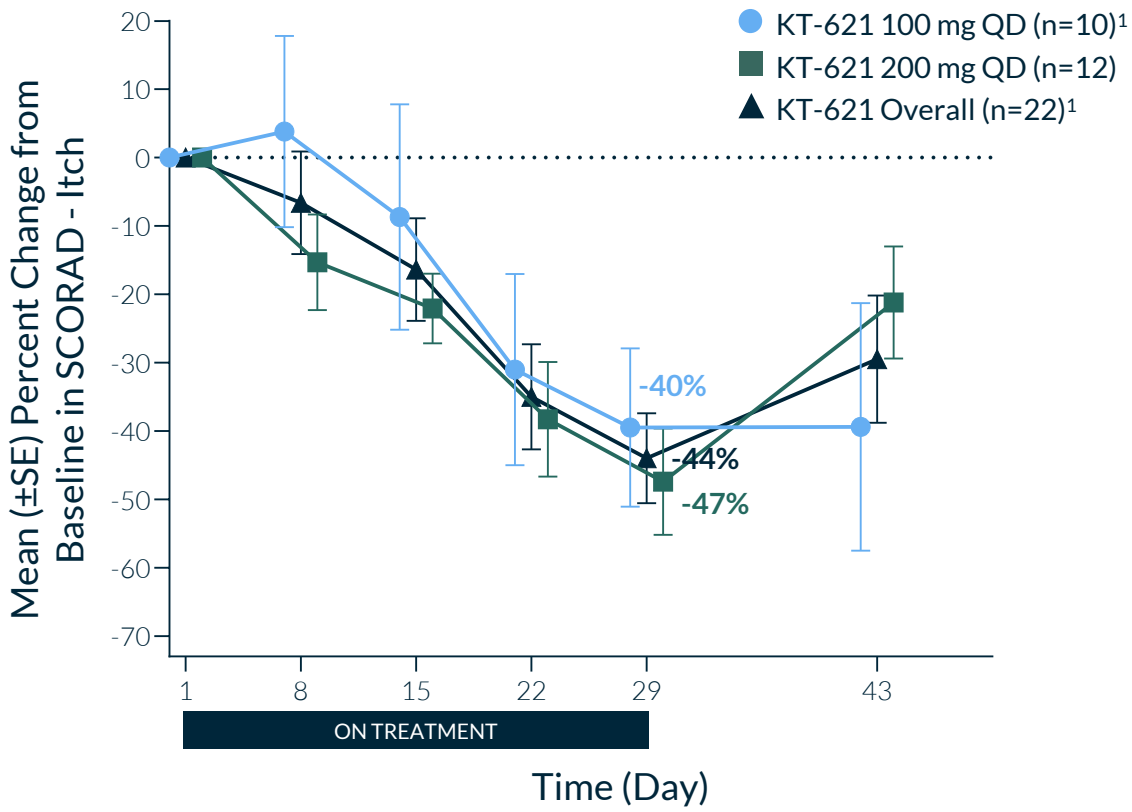
Note: Analysis based on observed cases at each visit; [1]One patient in the 100 mg cohort missed the D29 visit (n=9 for 100 mg and n=21 for Overall at D29); EASI: Eczema Area and Severity Index; SCORAD: SCORing Atopic Dermatitis.

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

Mean % Change in Peak Pruritus NRS



Mean % Change in SCORAD Itch

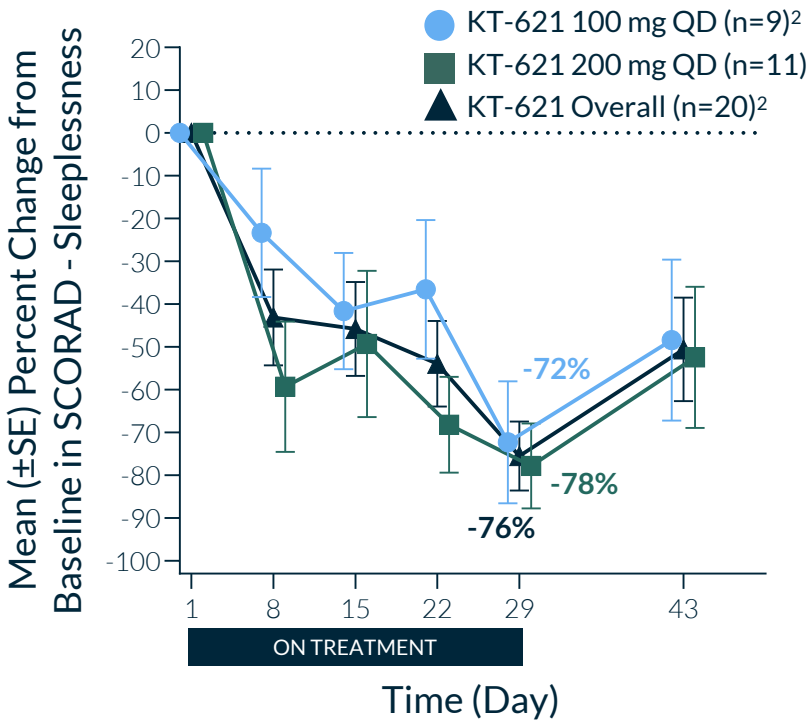


- KT-621 achieved rapid and robust mean Peak Pruritus NRS and SCORAD-Itch reduction without apparent plateau during treatment duration

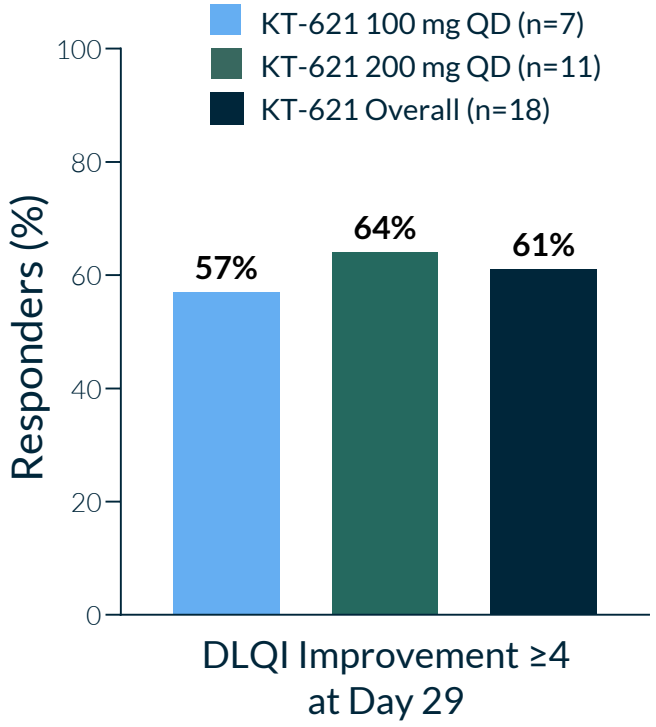
Note: Analysis based on observed cases at each visit; [1]One patient in the 100 mg cohort missed the D29 visit (n=9 for 100 mg and n=21 for Overall at D29); PPNRS: Peak Pruritus Numerical Rating Scale; SCORAD: SCORing Atopic Dermatitis.

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

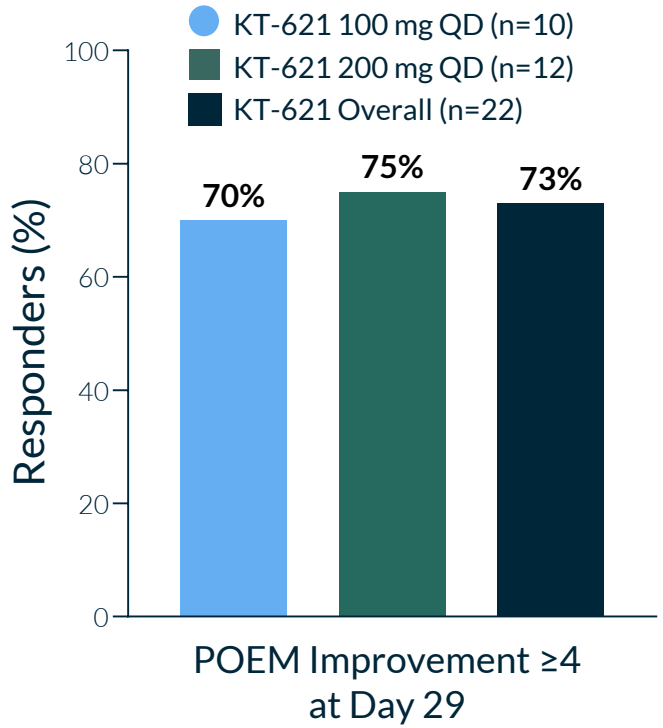
Mean % Change in SCORAD - Sleeplessness[1]



DLQI Responders (Improvement ≥ 4 points)[3]



POEM Responders (Improvement ≥ 4 Points)

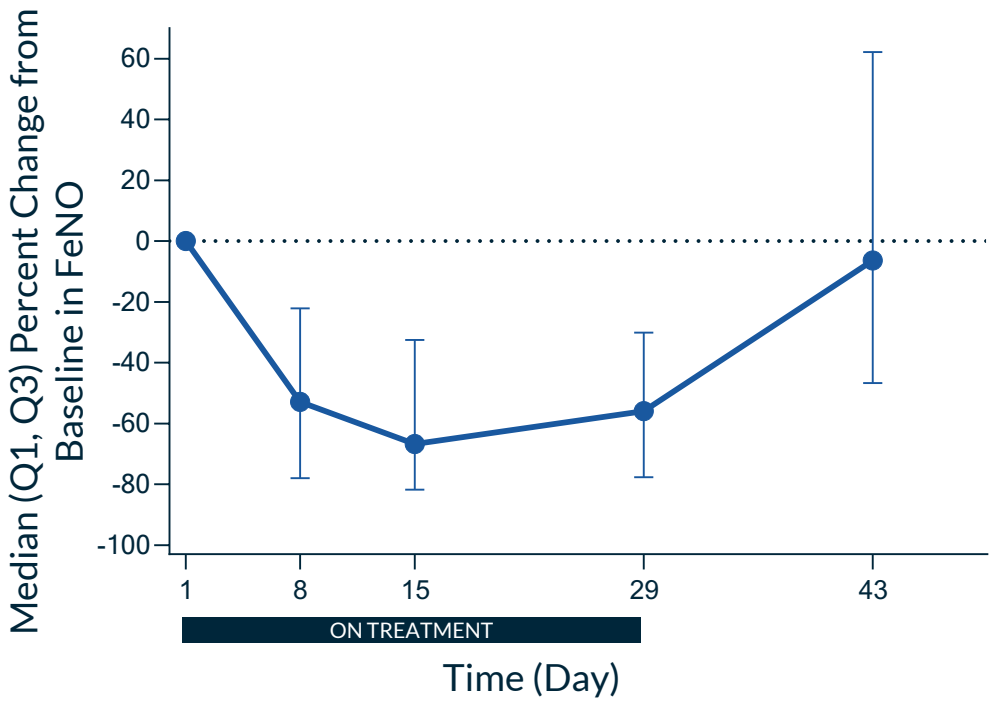


- KT-621 achieved rapid and robust mean SCORAD-Sleeplessness reduction in both dose cohorts
- POEM and DLQI are patient-reported measures evaluating severity, experience, and quality of life; demonstrated improvements are greater than the minimum clinically important difference (MCID)

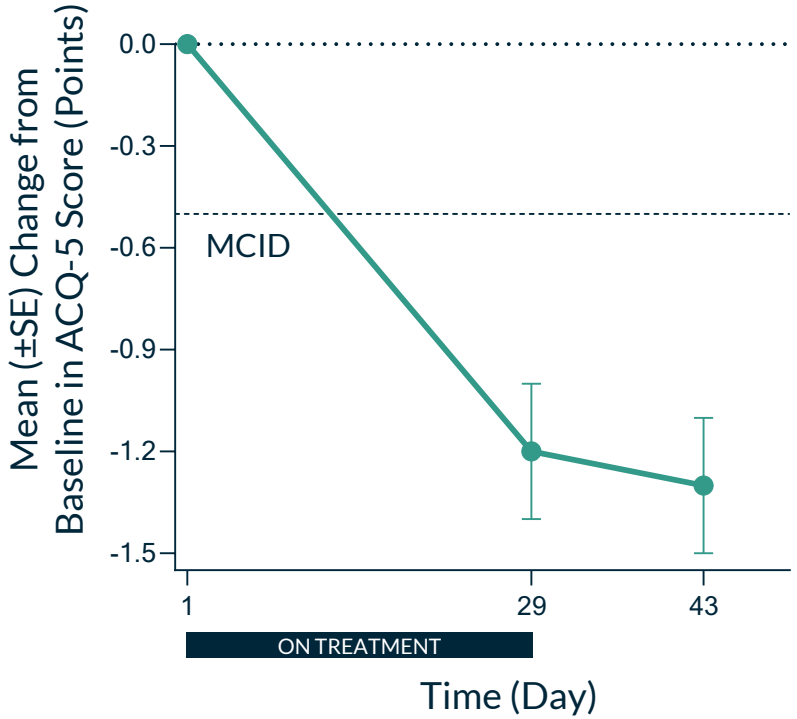
Note: Analysis based on observed cases at each visit. [1]Only patients with non-zero baseline sleeplessness are included; [2]One patient in the 100 mg cohort missed the D29 visit (n=8 for 100 mg and n=19 for Overall at D29); [3]Only patients with baseline DLQI score ≥ 4 included; SCORAD: SCORing Atopic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

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

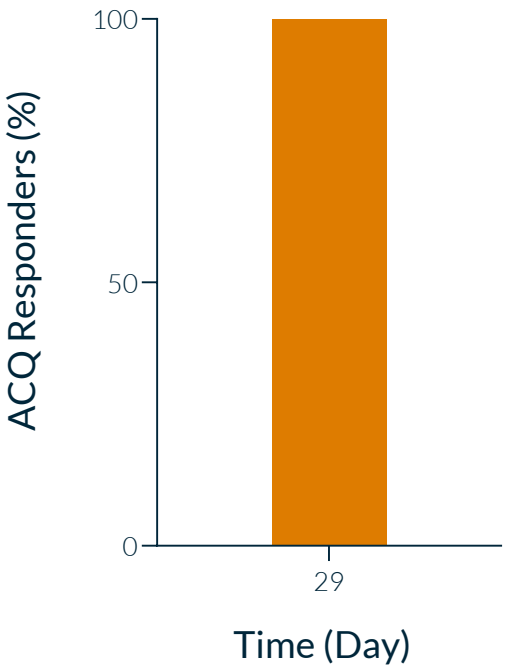
Median % Change from Baseline in FeNO (n=4)



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



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

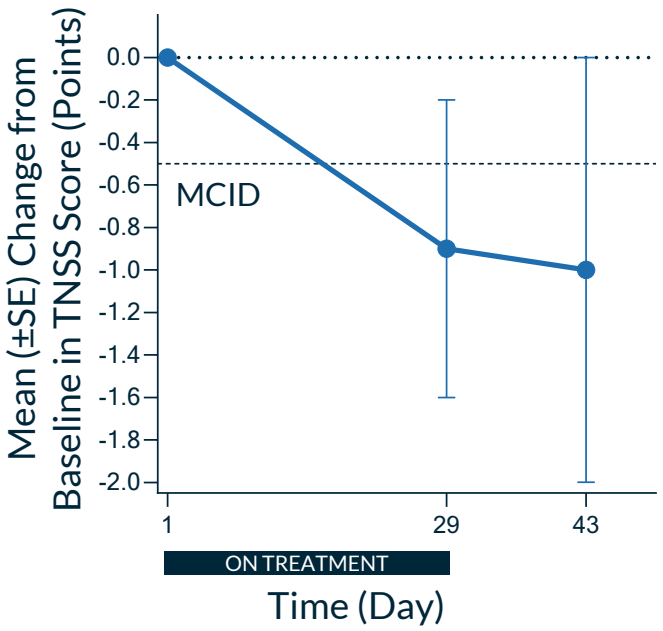


- KT-621 achieved 56% median FeNO reduction at Day 29, exceeding dupilumab (31%) in asthma studies at week 4[1]
- All 4 patients had clinically meaningful reduction in ACQ-5 (mean change of -1.2 points) and a 100% responder rate

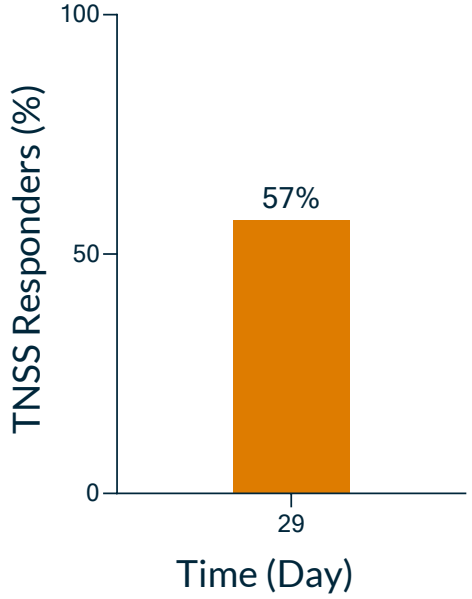
Note: Analysis based on observed cases at each visit; Mean baseline FeNO of 49 ppb and ACQ-5 of 2.5; [1]Pavord et al. JACI. 2024; FeNO: Fractional Exhaled Nitric Oxide; ACQ-5: Asthma Control Questionnaire-5; MCID: Minimum Clinically Important Difference.

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

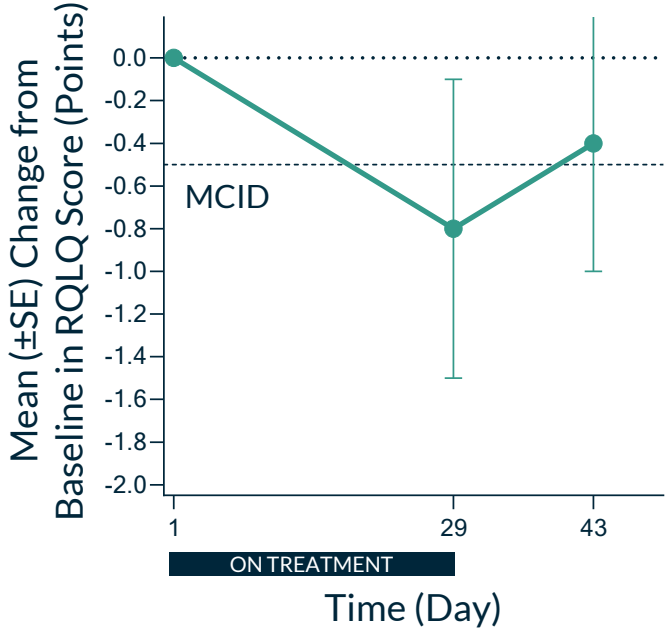
Mean Point Change in TNSS (n=7)[1]



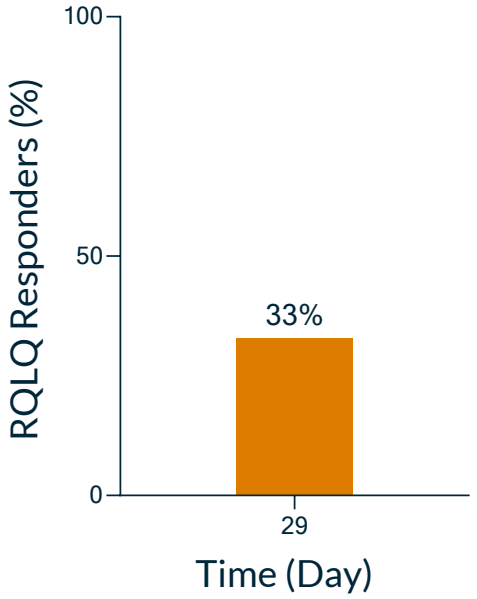
TNSS Responders (Improvement ≥ 0.55, n=7)[1]



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 Demonstrated Robust Improvements Across All Key Clinical Efficacy Endpoints

Results in Line With or in Some Cases Numerically Exceeded Published Data for Dupilumab at Week 4

	KT-621 100 mg QD Day 29 (n=10)	KT-621 200 mg QD Day 29 (n=12)	KT-621 Overall Day 29 (n=22)	Dupilumab 300 mg Q2W D28 Ph3 (n=457)[1]
Mean % Change in EASI	-62%	-63%	**-63%**	**-52%**
EASI-50	67%	83%	**76%**	**57%**
EASI-75	33%	25%	**29%**	**28%**
Mean % Change in PPNRS	-47%	-35%	**-40%**	**-33%**
Mean % Change in SCORAD	-52%	-46%	**-48%**	**-41%[2]**
vIGA-AD 0 and 1	22%	17%	**19%**	**12%**
Mean % Change in % Body Surface Area	-55%	-44%	**-49%**	**-36%[2]**
Mean % Change in SCORAD - Sleeplessness	-72%	-78%	**-76%**	**NR**
Mean % Change in SCORAD - Itch	-40%	-47%	**-44%**	**NR**
POEM Responders	70%	75%	**73%**	**69% at Week 16**
DLQI Responders	57%	64%	**61%**	**69% at Week 16**

[1]Dupilumab Phase 3 data derived or digitized from the pooled data of SOLO1 and SOLO2 studies (Thaci et al, Dermatological Science, 2019; Cather et al, Dermatol Ther, 2022);
[2]Data from 64 patients in dupilumab Phase 2b study (Thaci et al, Lancet, 2016); EASI: Eczema Area and Severity Index; vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis; PPNRS: Peak Pruritus Numerical Rating Scale; SCORAD: SCORing Atopic Dermatitis; NR: Not Reported.

KT-621 BroADen Phase 1b Safety Summary

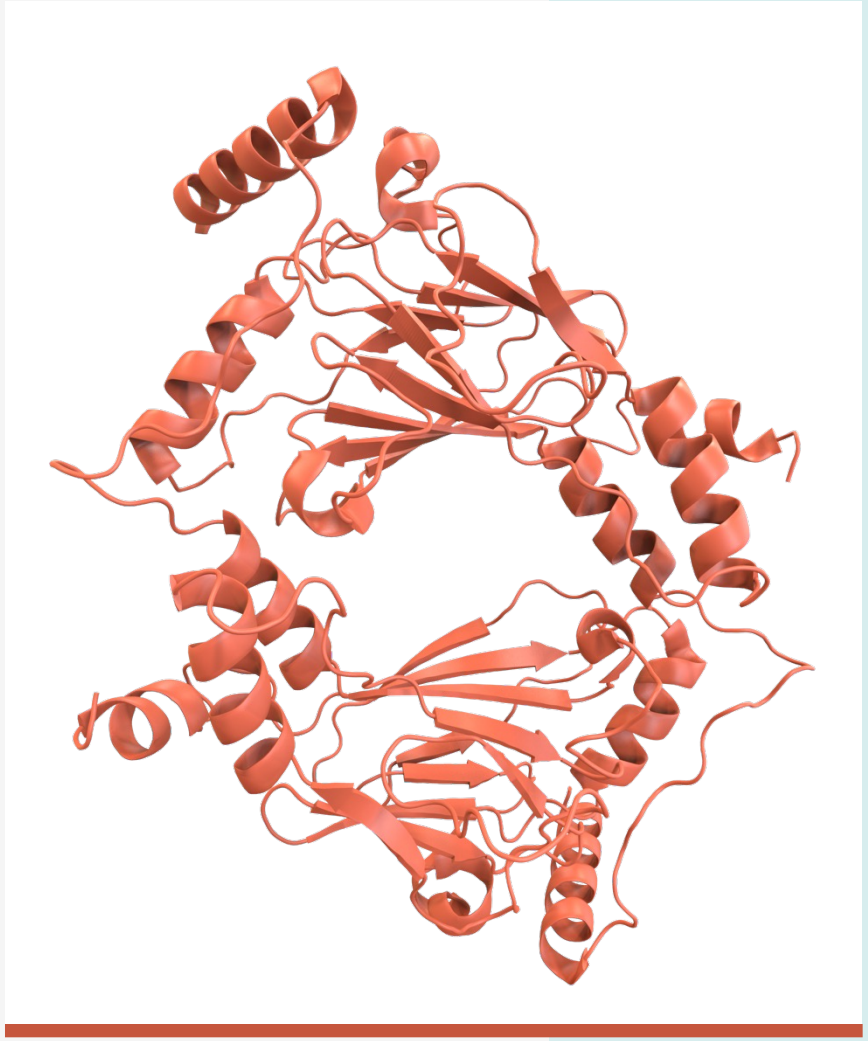
- Well-tolerated with favorable safety at both 100 mg and 200 mg with a profile similar to what was observed in the Phase 1a healthy volunteer trial
- No SAEs or Severe AEs
- No dose-dependent pattern in the TEAEs
- No related TEAEs or TEAEs leading to discontinuation
- No AEs of conjunctivitis (or of any ocular disorder), herpes infections, or arthralgias
- No clinically relevant changes in vital signs, laboratory tests or ECGs

AE: Adverse Event; SAE: Serious Adverse Event; TEAE: Treatment Emergent Adverse Event; ECG: Electrocardiogram.

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

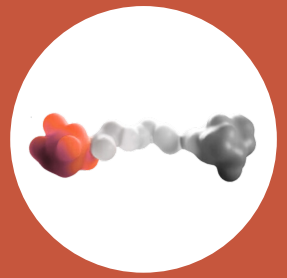


First-in-Class Oral IRF5 Degrader Program

Drugging a Genetically Validated Transcription Factor with an Oral Degrader

KT-579 Overview

Potential First-in-Class Opportunity with Oral Small Molecule Profile



KT-579 is a first-in-class, potent, selective, **oral IRF5 degrader**

- IRF5 has the potential to be the first broad anti-inflammatory to affect immune dysregulation while sparing normal cell function
- Human and mouse genetics de-risk safety and clinical indications
- IRF5 degradation *in vivo* leads to robust cytokine inhibition and *in vivo* efficacy in models of lupus and RA superior to approved drugs in the space
- KT-579 fully degrades IRF5 across multiple preclinical species with a favorable safety profile

OPPORTUNITY

- Over 10M potential patient impact[1]
- WW market for SLE, LN, RA, IBD alone was >$45B in 2023 and **projected to grow to >$55B by 2029**[1]
- Large potential for **oral degrader with biologics-like activity** to block established pro-inflammatory pathways, IFN response, & key pathogenic cell types

→ **Potential to expand access to oral systemic advanced therapies in many diseases with no or suboptimal oral options**

STATUS & UPCOMING MILESTONES

- Phase 1 healthy volunteer trial ongoing, with data in 2H 2026

[1]GlobalData (2023 diagnosed prevalent patient population and forecasted sales for approved systemic advanced therapies in US/EU5/JP).

IRF5: A New Treatment Paradigm for Complex Autoimmune Diseases

Unmet Need: Designed for Disease Complexity

- Heterogenous autoimmune diseases like lupus reflect broad immune dysregulation, not isolated pathway disruption
- Biologics have validated individual pathways (Type I IFN, proinflammatory cytokines, autoantibodies/B cells), but their downstream approach addresses only a narrow scope of disease biology. As a result, many patients experience limited durability and/or inadequate response

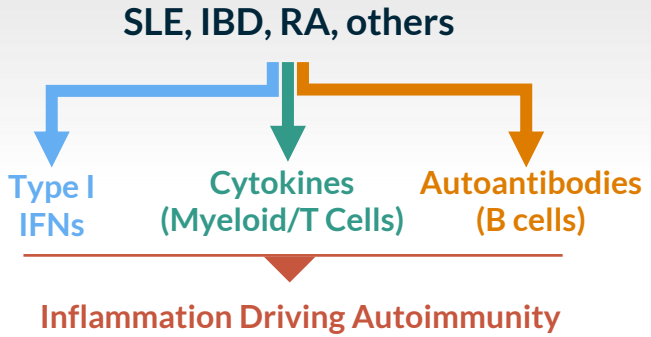
IRF5: Genetically Validated Transcription Factor

- Genetically validated master regulator and amplifier of immune responses in multiple autoimmune diseases
- When dysregulated, it drives skewed transcriptional crosstalk that locks multiple immune pathways into persistent inflammation
- Human risk variants or functional hyperactivation associate with increased signaling in multiple clinically validated pathways
- Not essential for immunity to infectious pathogens, suggesting potential for immune modulation without risk of bacterial or viral infections

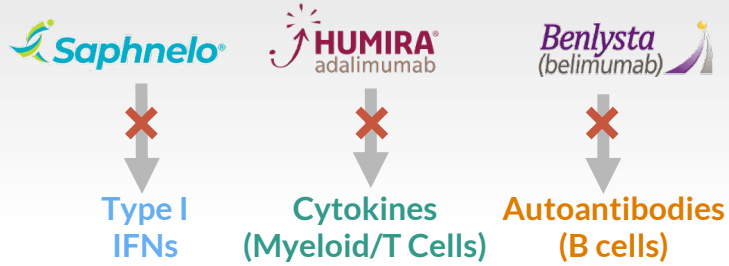
KT-579: First-in-Industry Oral Approach

- Designed to selectively degrade IRF5
- Enables simultaneous modulation of multiple dysregulated disease-defining pathways
- Aims to rebalance immune system and achieve more effective and durable disease control compared to injectable biologics targeting single pathways

Complex diseases are driven by **multiple validated inflammatory pathways**

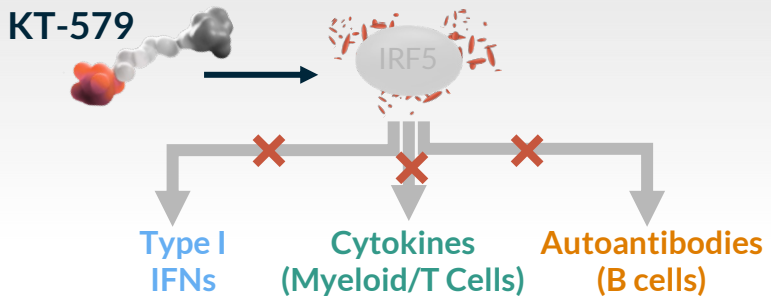


Existing therapies only address a **single pathway** directly at a time

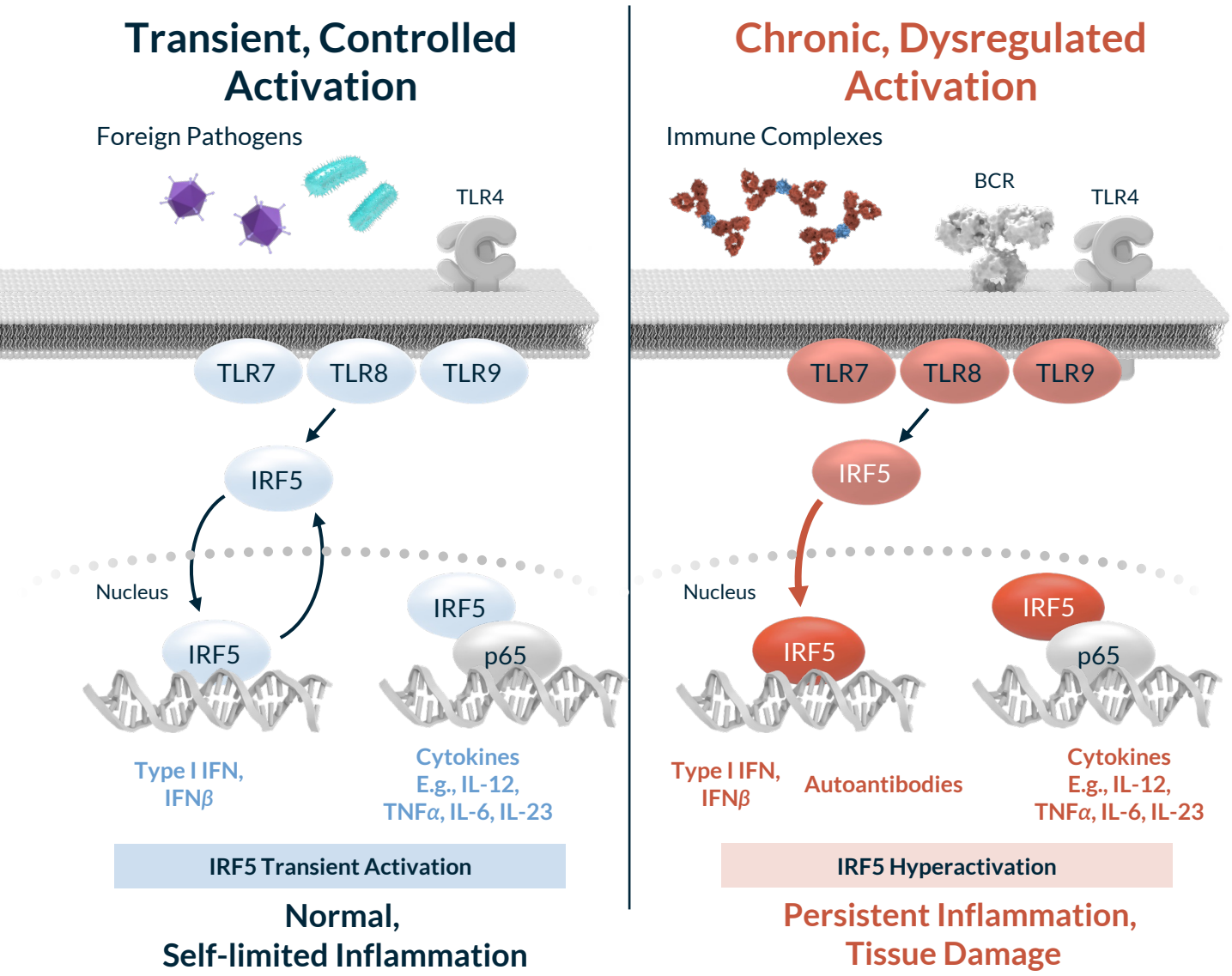


IRF5 hyperactivation impacts multiple disease-defining pathways

IRF5 degradation can address **all pathways with one oral mechanism**



Central Driver of Immune Dysregulation in Autoimmune Diseases



IRF5 Expression, Activation and Function

- IRF5 is **predominantly expressed and activated** in immune cell types: Dendritic Cells (DCs), Monocytes, Macrophages, and B cells
- In normal controlled immune responses, IRF5 is transiently activated by pattern recognition receptors (PRRs) with quick inflammation resolution
- In **autoimmune** diseases, **chronic activation** locks IRF5 into a hyperactive state leading to **amplified** and **skewed** upregulation of **pro-inflammatory and disease-defining pathways**
- IRF5 risk variants and functional hyperactivation associate with increased signaling in multiple clinically validated pathways **(Type I IFN, Cytokines, Autoantibodies)**

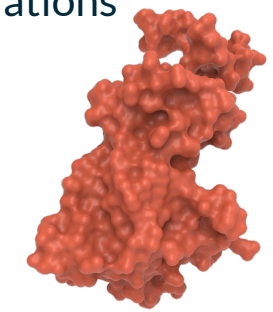
Opportunity to Address a Wide Range of Complex Autoimmune Indications Through Clinically Validated Pathways

Potential to be a First-in-class Oral Advanced Therapy

Individual Pathways Clinically Validated by Biologics

		Type I IFNs	Cytokines (Myeloid/ T Cells)	Autoantibodies (B Cells)
Rheumatology	SLE	Saphnelo®		Benlysta® (belimumab) / GAZYVA® obinutuzumab injection
	Sjogrens			*Ianalumab*
	RA		HUMIRA® adalimumab / ACTEMRA® tocilizumab	Rituxan® Rituximab
GI	UC		HUMIRA® adalimumab / Stelara® (ustekinumab)	
	CD		HUMIRA® adalimumab / Stelara® (ustekinumab)	

Deep degradation of IRF5 will modulate multiple clinically validated pathways (vs suppressing a single pathway) to rebalance the immune system and address heterogenous autoimmune diseases and broad patient populations



IRF5 Genetically Validated in Multiple Autoimmune Diseases

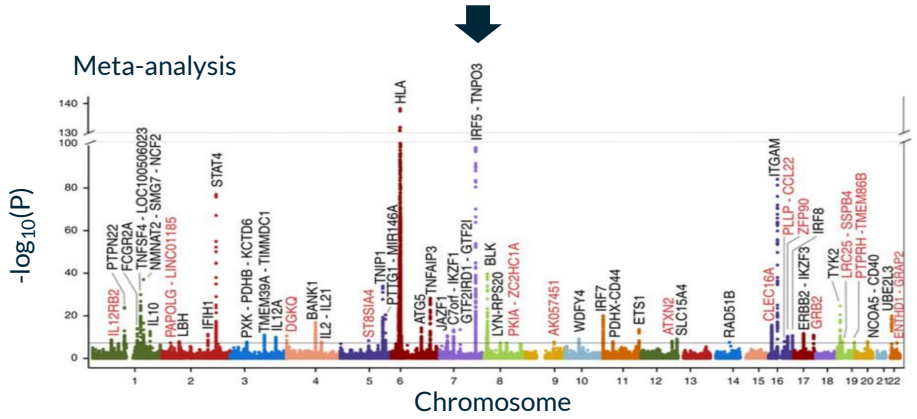
Human Genetics

- Multiple GWAS studies identify IRF5 as an autoimmune susceptibility gene[1]
- IRF5 risk haplotypes and hyperactivated IRF5 levels in SLE patients associated with high serum IFNα levels, anti-dsDNA or anti-RNA binding protein antibodies
- Genetic associations and functional variants also identified in RA, IBD, SSc, and MS

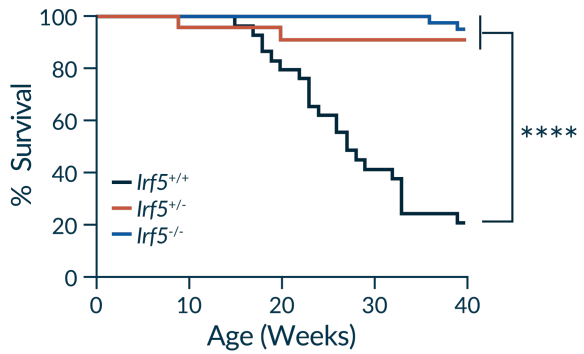
Mouse IRF5 KO Studies

- KO mice are viable and fertile; with normal B cell development
- IRF5 KO mouse can protect or attenuate disease in models of SLE, SSc, RA and IBD
- In a mouse model of lupus (FcγIIB-/-, Yaa) IRF5 plays an essential role in lupus pathogenesis, that is independent of Type I IFN pathways[2]

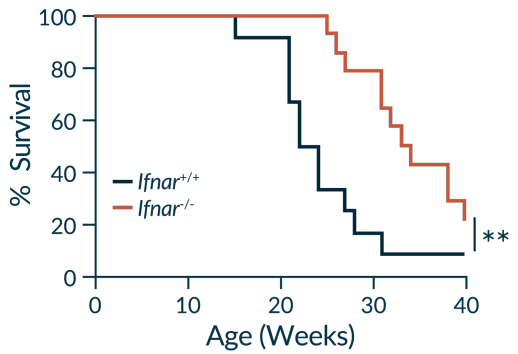
Meta-analyses Identified IRF5 as a Risk Locus for SLE



IRF5 KO Increases Survival and has Essential Role in Lupus



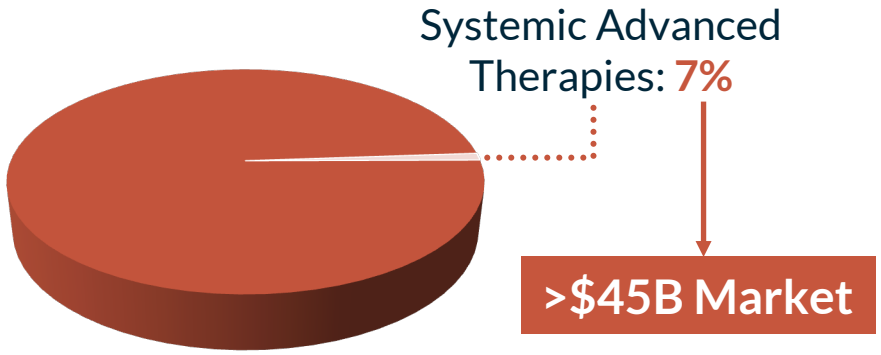
Type I IFN Modestly Protects Against Lupus



[1]Santana-de Anda et al. *Autoimmunity Reviews*. 2011; [2]Richez et al. *J Immunol*. 2010.; ****$p < 0.0001$; **$p = 0.0043$.

IRF5: A Novel Mechanism in High Unmet Need Indications in I&I

>10M

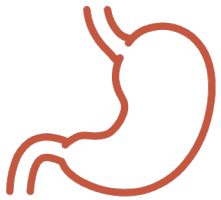


TOTAL POTENTIAL PATIENT IMPACT[1]

IRF5 functional risk variants associate with increased susceptibility to multiple diseases, and IRF5 regulated pathways have been clinically validated by multiple drugs

RHEUMATOLOGY

	PATIENTS[1]
RA	4.7M
Lupus[2]	1.2M
Sjögren's	765K
SSc	200K
DM	>100K



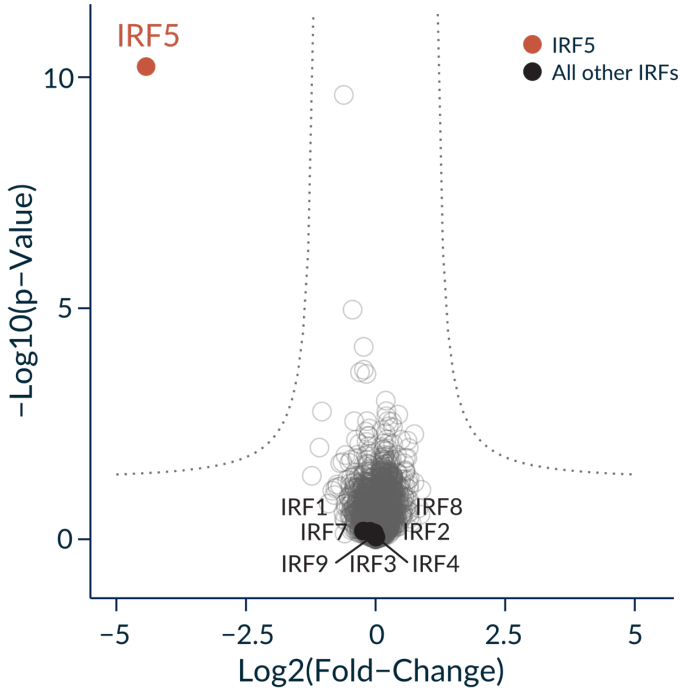
GASTROENTEROLOGY

UC	1.9M
CD	1.5M

[1]GlobalData (2023 diagnosed prevalent patient population US/EU5/JP); [2]Lupus includes SLE, CLE, LN.

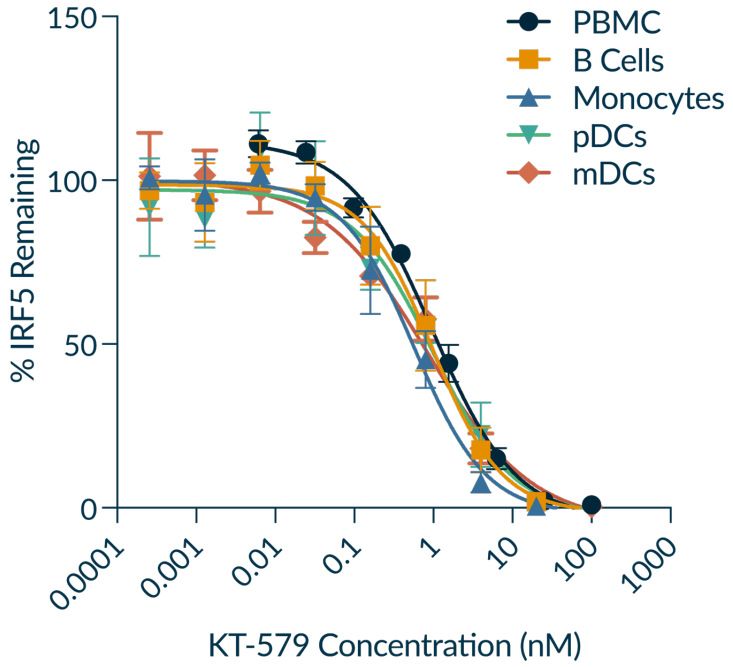
KT-579: An Exquisitely Selective and Picomolar Oral IRF5 Degrader

KT-579: 10xDC$_{90}$ in hPBMCs @ 24 hrs



Selectivity Binding and Cellular Assays	KT-579
IRF3, 4, 6, 7, 8 SPR binding, Kd (nM)	>10,000
IRF3 Degradation DC$_{50}$ (nM)	>10,000
IRF7 Degradation DC$_{50}$ (nM)	>10,000

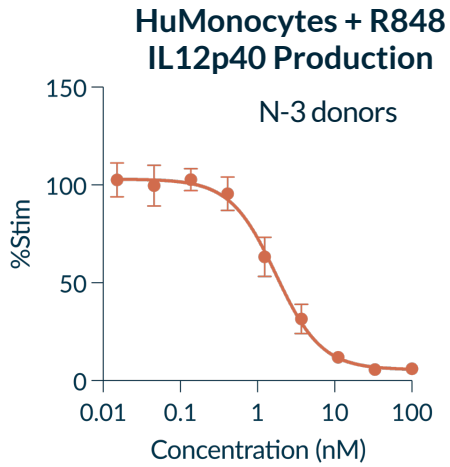
Equipotent Degradation in Relevant hPBMC Subsets @ 24hrs

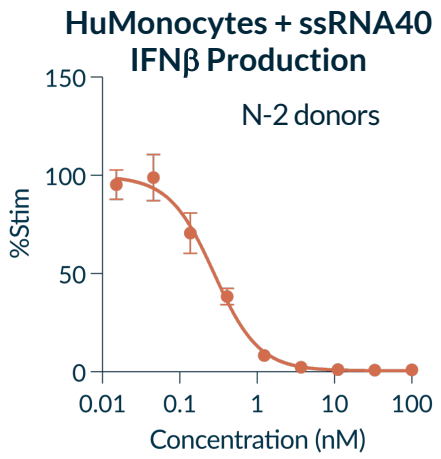


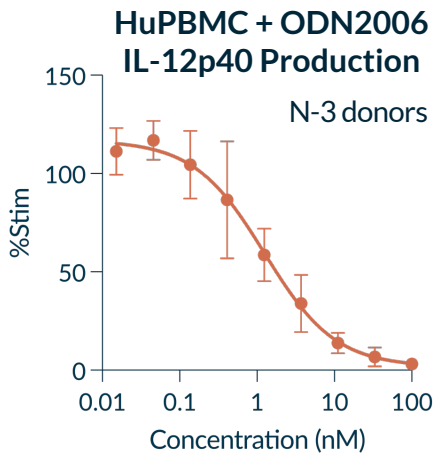
Cell Subsets	KT-579 DC$_{50}$ (nM)
PBMC	0.8
CD19+ B cells	1.0
CD14+ Monocytes	0.6
HLA-DR+CD123+ pDCs	0.9
HLA-DR+CD11c mDCs	0.9

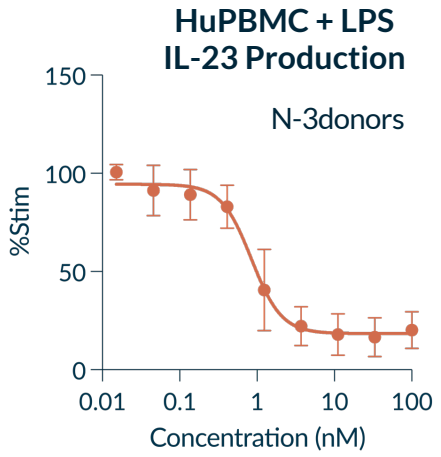
KT-579 Potently Inhibits Production of Key Pro-Inflammatory Cytokines and Type I IFN in Human Primary Cellular Assays

Biology	Receptor	Stim	Cell	Cytokine Readout	KT-579 DC_{50} (nM)
Type I IFN Production	**TLR8**	**ssRNA40**	**Monocytes**	**IFNβ**	**0.3**
	TRL7/8	R848	Monocytes	IFNβ	0.4
	TLR9	ODN2006	PBMC	IFNβ	0.8
Pro-inflammatory Cytokine Production	TLR7/8	R848	PBMC	TNFα	1.5
	TLR7/8	R848	B cells	TNFα	0.4
	TLR8	ssRNA40	Monocytes	TNFα	1.3
	TL7/8	**R848**	**Monocytes**	**IL-12p40**	**0.5**
	TLR9	**ODN2006**	**PBMC**	**IL-12p40**	**1.8**
	TLR7/8	R848	Monocytes	IL-1β	0.15
	TLR4	**LPS**	**PBMC**	**IL-23**	**1.1**



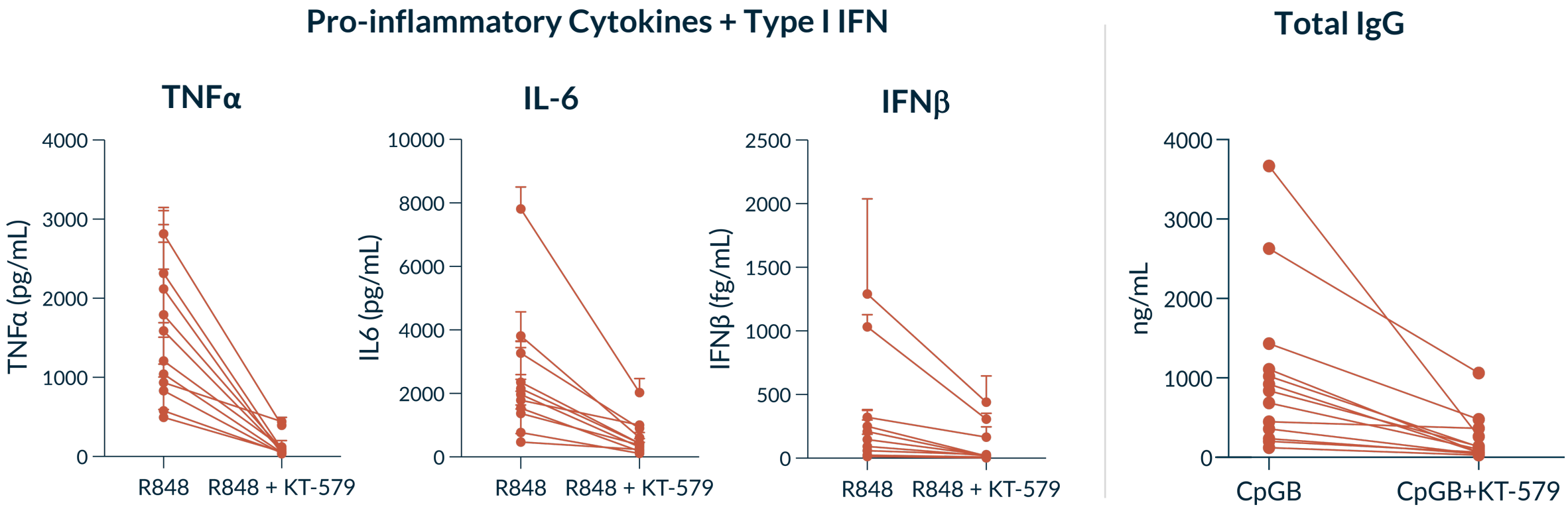






IRF5 degradation inhibits proinflammatory cytokines (TNFα, IL-12, IL-23, IL-1) and Type I IFN (IFNβ) downstream of TLR4, TLR7, TLR8, and TLR9 activation

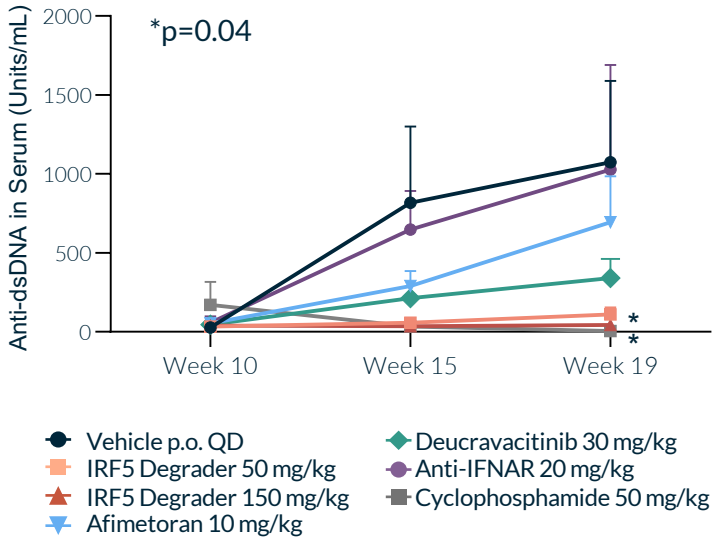
KT-579 Effectively Blocks TLR Induced Pro-inflammatory Cytokines and Type I IFN Induction and Reduces Total IgG levels in SLE PBMC Samples



- KT-579 inhibits TLR7/8 pro-inflammatory cytokines and Type I IFN that are commonly elevated in autoimmune diseases
- KT-579 reduces TLR9 (CpG-B) induced plasmablast differentiation and inhibits IgG production in SLE derived B cells

KT-579 Superior Activity in the MRL/Ipr[1] Model of Lupus

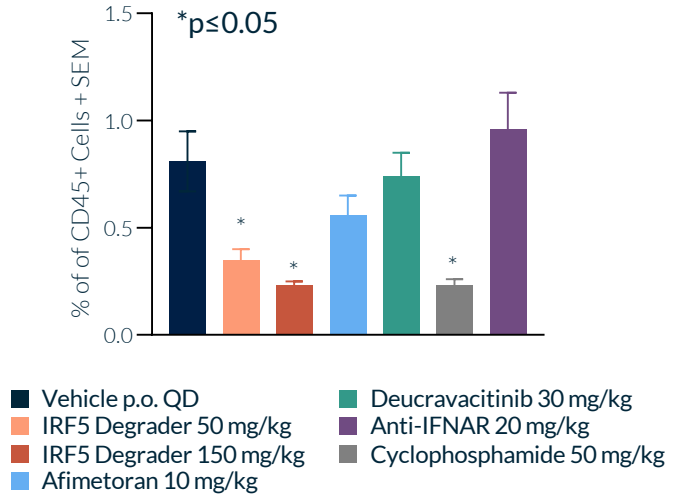
Sustained Reduction of Serum Anti-dsDNA



All KT-579 Treated Mice Survived Length of Study

Treatment	#Total survival
Vehicle	10/15
KT-579 50 mg/kg, p.o.	15/15
KT-579 200 mg/kg, p.o.	15/15
Afimetoran, 10 mg/kg, p.o.	13/15
Deucravacitinib, 30 mg/kg, p.o.	13/15
Cyclophosphamide, 50 mg/kg, i.p.	14/15
Anti-IFNAR mAb, 20 mg/kg, s.c.	9/15

Plasmablasts[2]

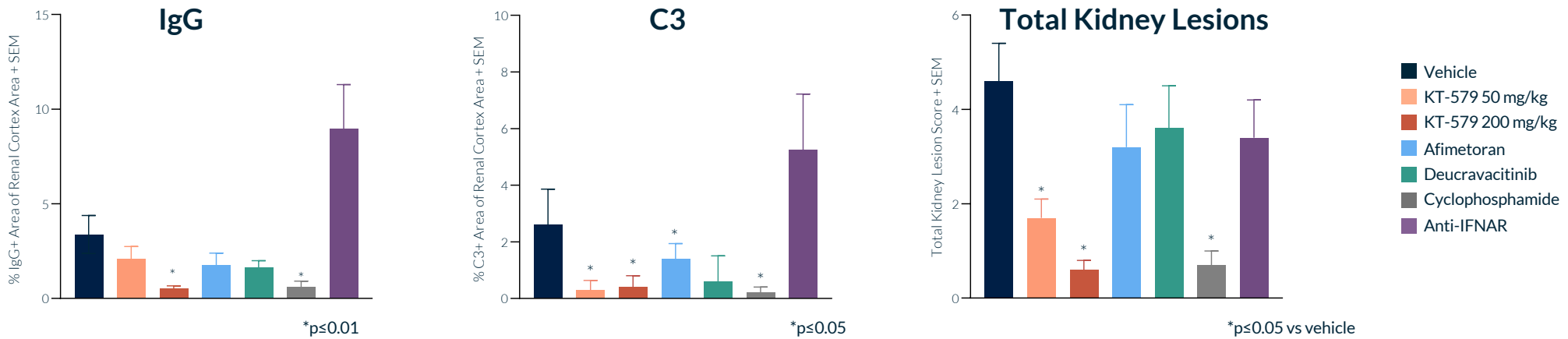


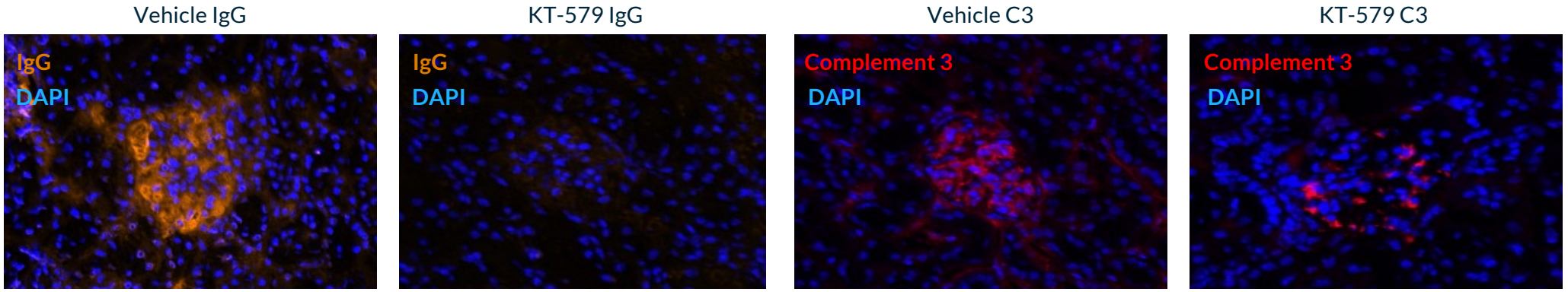
- KT-579 administered once daily for 63 days was well tolerated and prevented mortality more effectively than all other approved or clinically active comparator agents tested
- KT-579 led to sustained reduction of serum anti-dsDNA levels and reduction in splenic plasmablasts, differentiated B cells, and plasma cells, achieving effects comparable to, or better than, standard of care

[1]MRL/lpr mice have a susceptible genetic background and single inactivating mutation in FAS gene, quickly developing lupus-like symptoms and manifestations; [2]Plasmablast phenotype: B220low-CD138hi-CD44-; p.o., oral; s.c., subcutaneous.

KT-579 Significantly Decreases IgG, Complement 3 (C3) and Renal Disease Progression

Assessment of Terminal Kidney Collected from Surviving MRL/lpr Mice at Week 19



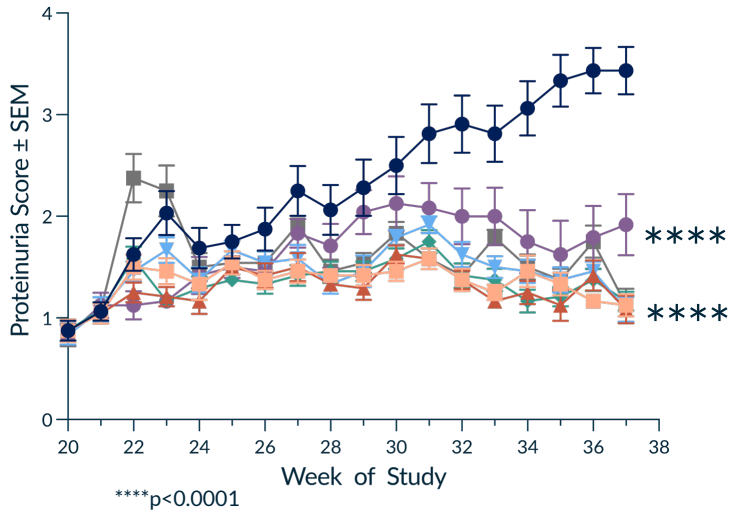


Representative images (10x) of IgG and C3 staining in vehicle vs KT-579 treated MRL/lpr kidney glomerular structures

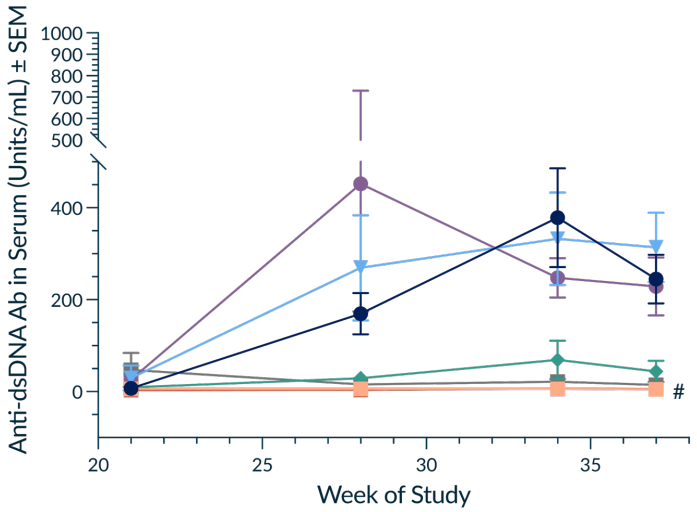
Clear reduction in IgG/complement glomeruli staining indicating reduced kidney disease involvement in SLE MRL/lpr mouse model with IRF5 degrader as compared to standard of care

IRF5 Degradation Demonstrated Generally Better Outcomes than Approved or Clinically Active Drugs in NZBW1 Spontaneous[1] Mouse Lupus Model

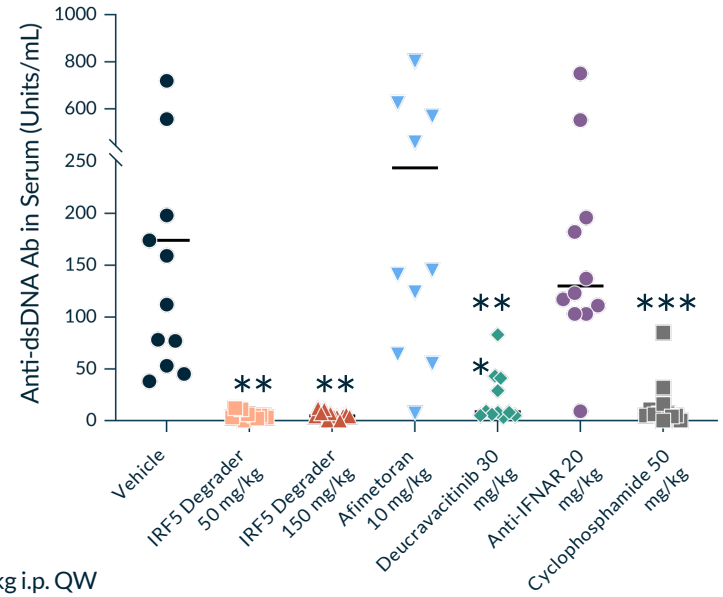
Decreased Proteinuria



Early, Sustained and Complete Reduction in Anti-dsDNA Abs



Superior Activity Reducing Anti-dsDNA at Week 37



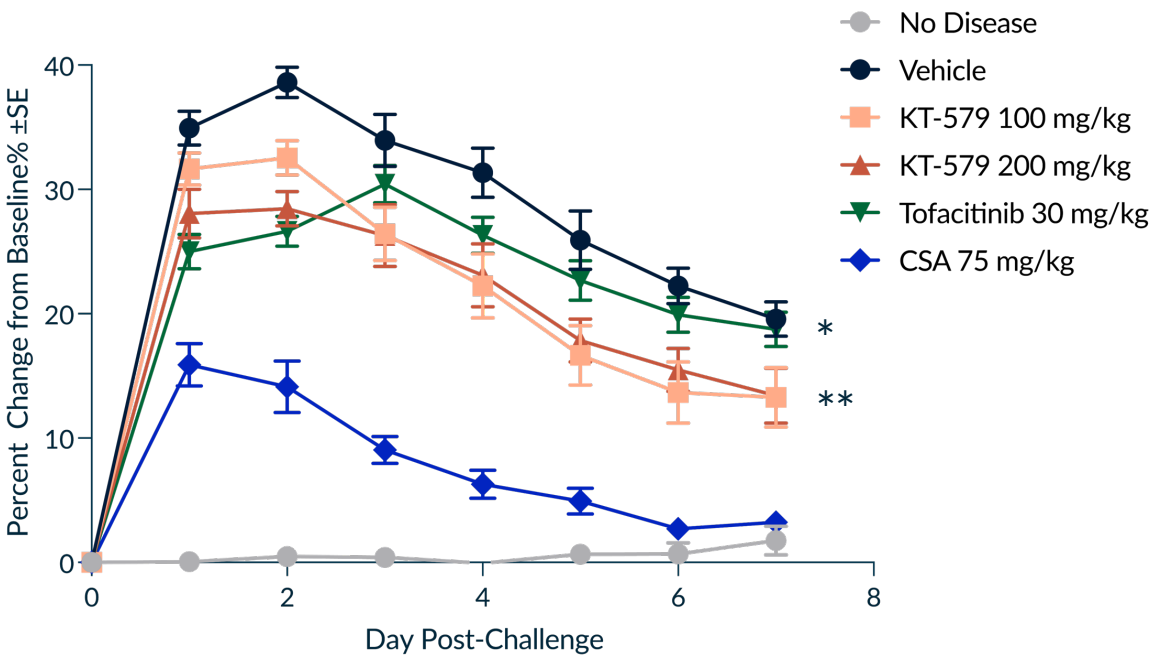
Vehicle p.o. QD
IRF5 Degrader 50 mg/kg p.o. QD
IRF5 Degrader 150 mg/kg p.o. QD
Afimetoran 10 mg/kg p.o. QD
Deucravacitinib 30 mg/kg p.o. QD
Anti-IFNAR 20 mg/kg s.c. 2x/week
Cyclophosphamide 50 mg/kg i.p. QW

- **IRF5 degrader, dosed once a day for 107 days, leading to 80% and >90% IRF5 degradation, led to decreased proteinuria and near complete reduction of serum anti-dsDNA Abs superior to standard of care, approved and clinically active agents tested**
- **Significantly reduces key interferon genes (OAS1, IFIT1, IF44) at 150mg/kg in the blood from NZB.W1 mice comparable to other test agents**

[1]NZBW1 mice develop a similar disease to human SLE, characterized by anti-dsDNA ANA production, and immune complex mediated nephritis due to defects in multiple genes; #IRF5 Degrader 50 mg/kg p.o. and 150 mg/kg p.o. lines are overlapping.

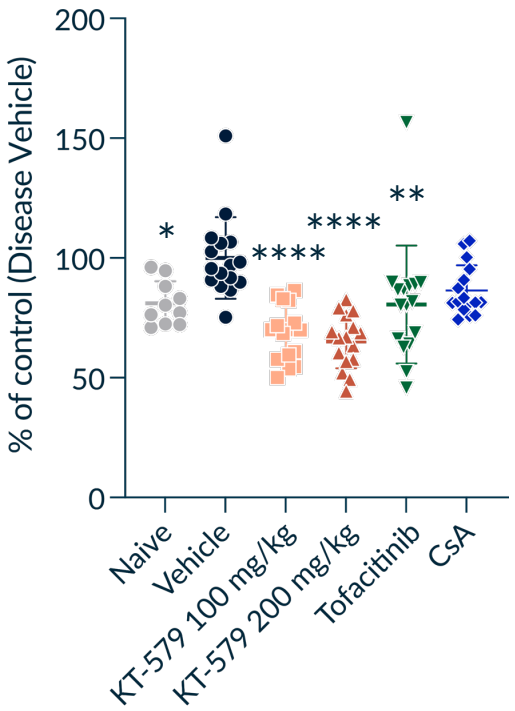
KT-579 Reduces Joint Swelling in a Mouse Model of RA

Treatment with KT-579 Leads to Significant Reduction in Joint Swelling Comparable to Tofacitinib in the AIA[1] Mouse Model of RA

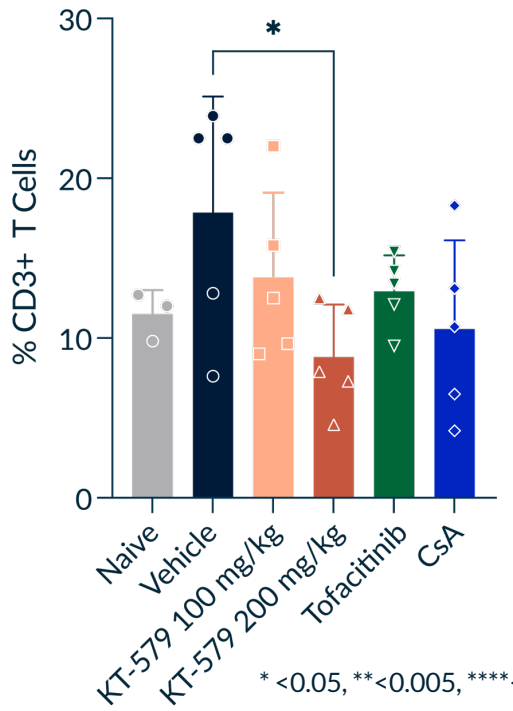


Reduction in Circulating IL-12 Levels and Infiltrating Synovial IFNγ+ Th1 Cells

IL-12p40 Serum Levels Day 2



IFNγ+ CD4+ T Cells Day 7



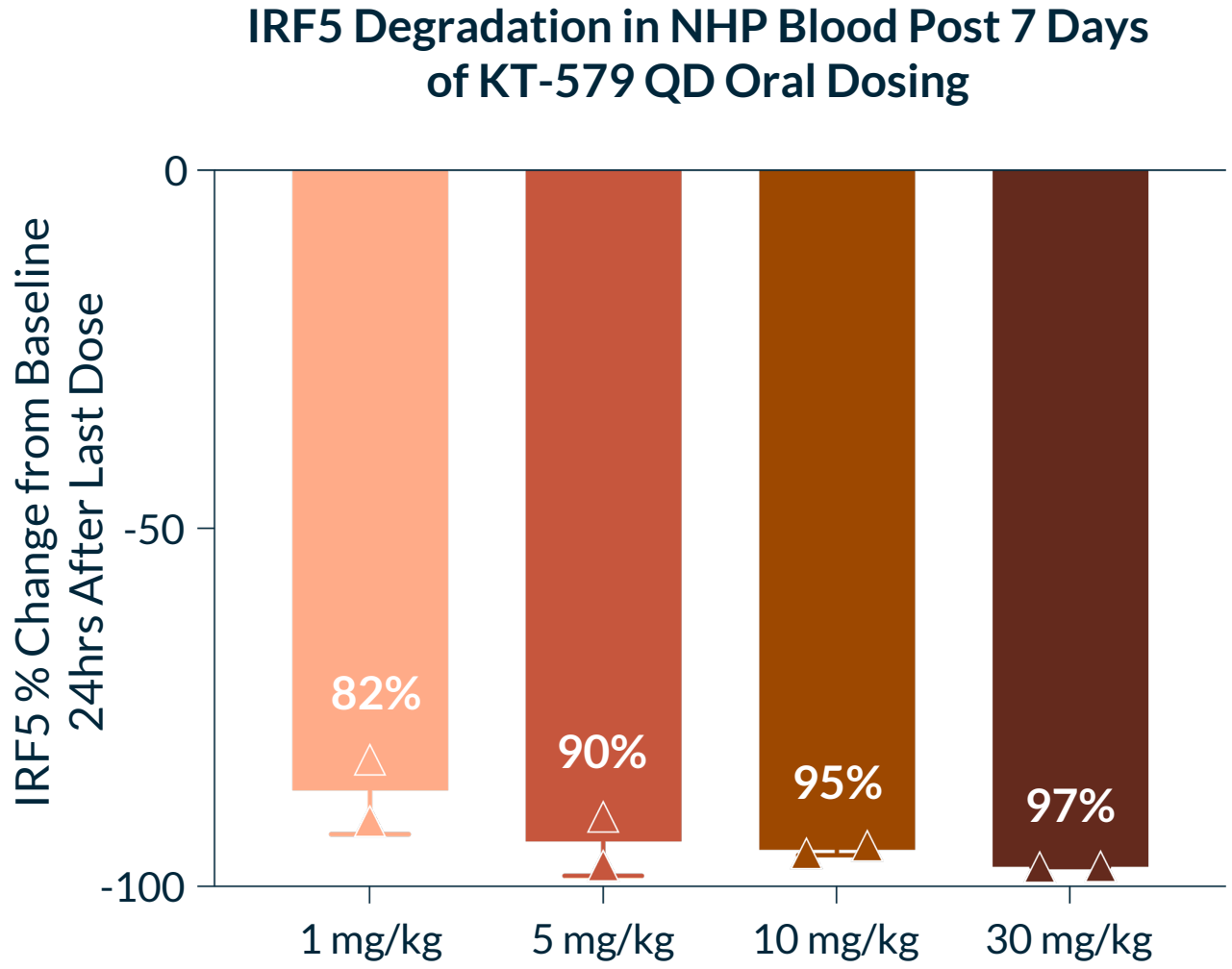
* <0.05, ** <0.005, **** <0.0001

Daily oral dosing of KT-579, leading to ~90% IFR5 degradation, results in significant reduction of circulating pro-inflammatory cytokines, joint swelling and pathogenic infiltrating T cells

[1]Antigen induced arthritis (AIA) model was established by sensitization with methylated bovine serum albumin (mBSA). 14 days later mice were challenged with mBSA (Day 0) that induced rapid inflammation and swelling at site of injection. Samples for analysis collected on Day 2 and Day 7.

KT-579 Potently Degrades at Low Oral Doses in NHP with an Excellent Safety Profile

- KT-579 potently degrades IRF5 across multiple preclinical species with low oral doses
- No adverse findings in non-GLP and GLP toxicity studies in NHP and rodents



KT-579: First IRF5 Agent to Enter Clinical Evaluation

Phase 1 Trial: Double-blind, Placebo-controlled SAD and MAD in Healthy Volunteers

Part A
Single Ascending Dose
(SAD)

Part B
Multiple Ascending Dose
(MAD)
14x daily doses

Primary
- Safety & tolerability of escalating single and multiple doses of KT-579

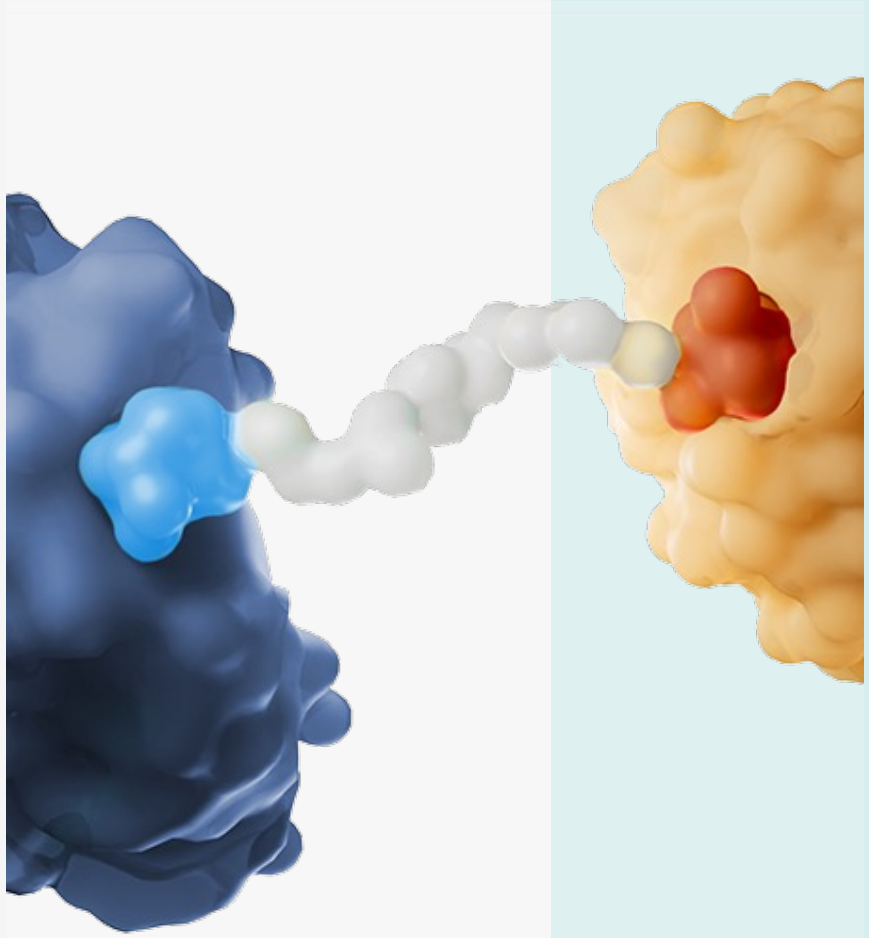
Secondary
- Pharmacokinetic measures

Exploratory
- IRF5 protein levels in blood
- *Ex vivo* blood biomarkers:
  - Proinflammatory cytokines
  - Type I IFNs
  - Inflammatory pathway gene transcripts

Key Trial Aim

Key trial aim is to show that **KT-579 can robustly degrade IRF5 in blood** at doses that are safe and well-tolerated

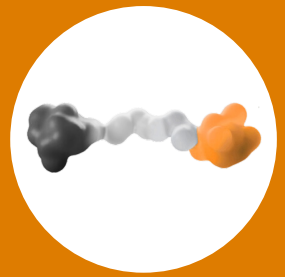
Status update:
Dosing commenced; Data expected 2H26



Partnered Oral Degrader Programs

IRAK4 Overview

Combined Activity of Upstream Biologics (anti-IL-1/18/33/36) in a Pill



KT-485, a second generation IRAK4 degrader, is a highly selective, potent, **oral IRAK4 degrader**

- KT-485 has increased selectivity and potency over KT-474 including absence of any QTc signal and has been prioritized for clinical development by Sanofi
- Learnings from the clinical studies of KT-474 will be applied to accelerate development of KT-485
- Phase 1 studies of KT-485 are expected to initiate in 2026

OPPORTUNITY

- Over **140M** potential patient impact[1]
- **>$55B** in combined global drug sales[2] opportunity
- Large potential for **oral degraders with best-in-pathway efficacy** across Th1-Th17 and Th2 Diseases

→ **Potential to deliver the combined activity of upstream biologics in an oral drug for multiple diseases**

STATUS

- Partnered with Sanofi
- KT-485 completed IND-enabling studies

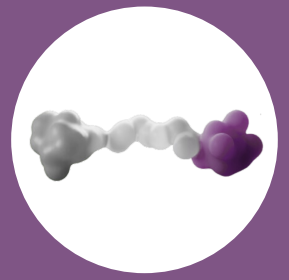
UPCOMING MILESTONES

- Phase 1 start: 2026

[1]GlobalData (2023 diagnosed prevalent patient population for US/EU5/JP); [2]GlobalData (2023 sales for AD, HS, Asthma, COPD, UC, CD, RA, SLE).

CDK2 Overview

Novel Molecular Glue (MG) Degrader



Potential best-in-class profile, with exquisite selectivity for CDK2

- CDK2 is a master regulator of the cell cycle with strong genetic and clinical validation and broad oncology treatment potential including breast cancer and other solid tumors
- Unlike inhibitors, CDK2 molecular glue degraders selectively degrade CDK2 while sparing closely related kinases like CDK1, offering a potentially broader therapeutic index

OPPORTUNITY

- **~500k** potential patient impact in multiple tumor types[1]
- CDK4/6 inhibitor class ~$13B in WW revenue in 2024; projected to grow to $18B by 2030[2]

→ **CDK2 MGs offer an oral therapeutic option with the potential to selectively target cancer cells with minimal impact on healthy tissue**

STATUS

- Partnered with Gilead to accelerate development and commercialization, with up to $750 million in total payments, including $85 million in upfront and potential option exercise payments
- Kymera will lead all research activities for the CDK2 MG program

[1]GlobalData (2025 diagnosed incident patient population for US/EU5/JP); [2]CDK4/6 inhibitor class includes palbociclib, abemaciclib, and ribociclib; Future revenue projection reflects GlobalData's consensus forecast.

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

- Report Phase 1 healthy volunteer data in the 2H 2026

Research

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

Thank You

NASDAQ: KYMR

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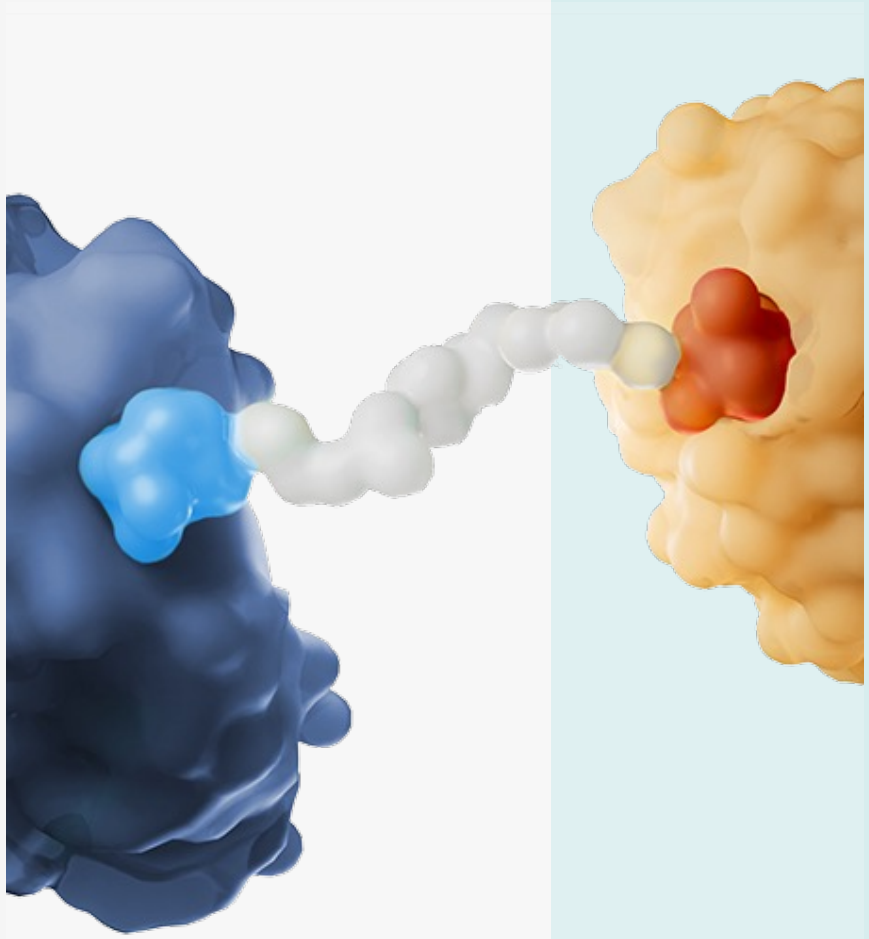
KYMERA

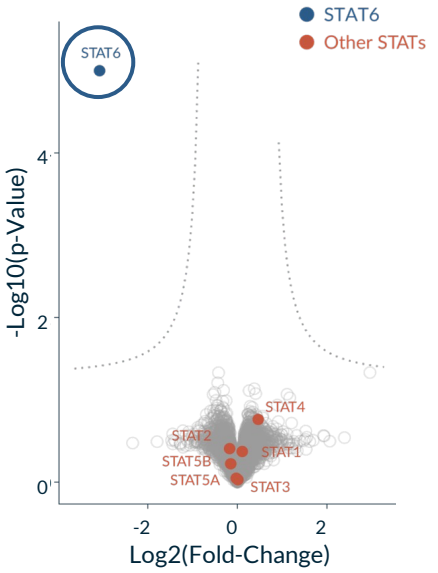


Appendix: First-in-Class Oral STAT6 Degrader Program

KT-621: Compelling Preclinical Profile

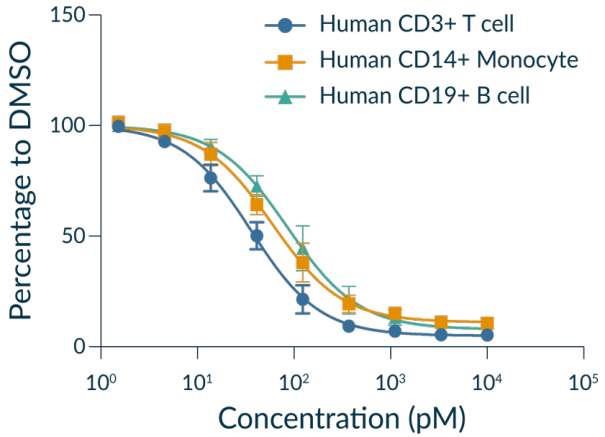
Preclinical Package Suggests Potential for Dupilumab-like Activity in a Pill

Exquisite Selectivity



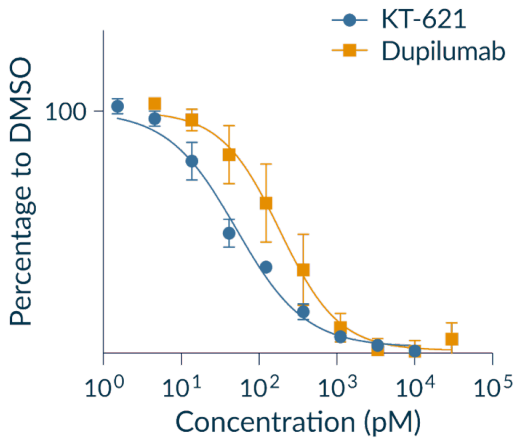
Robust Degradation in Relevant Cell Types

STAT6 Degradation in Immune Cells



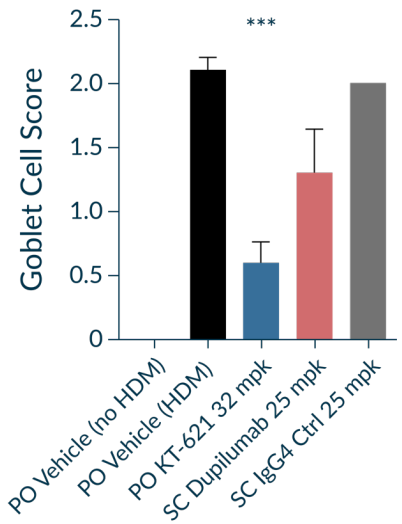
Full Inhibition of IL-4/IL-13 Pathways, More Potent than Dupilumab

IL-4 Induced TARC Release in Human PBMC



Excellent Preclinical Activity Comparable or Superior to Dupilumab

Lung Remodeling Goblet Cell Metaplasia in HDM Model[1]



Favorable safety profile: well-tolerated in multiple preclinical species and safety studies at concentrations 40-fold above efficacious dose with up to 4 months of dosing

[1]A lung inflammation model induced by intranasal house dust mite (HDM) administration with dominant Type 2 inflammation in the IL4/IL4RA humanized mice, Le Floc'h et al, Allergy, 2020; *Significance to PO vehicle (HDM); #Significance to SC IgG4 Ctrl 25 mpk; ***p ≤ 0.001. For more information on KT-621's preclinical profile, visit Kymera's website.

KT-621 Phase 1a Healthy Volunteer Trial: Safety Summary

Well Tolerated Across All Doses Evaluated and Safety Profile Undifferentiated from Placebo

- No Serious Adverse Events
- No Severe Adverse Events
- No dose dependent pattern in Treatment Emergent Adverse Events (TEAEs)
- No Treatment Related AE (TRAE) reported in >1 participant
- No related TEAEs leading to discontinuation
- No clinically relevant changes in vital signs, laboratory tests, and ECGs

TRAEs by Preferred Term: SAD Cohorts

AE Term (severity)	SAD Placebo (n=12)	SAD KT-621 (n=36)
Headache (mild)	1 (8.3%)	0

TRAEs by Preferred Term: MAD Cohorts

AE Term (severity)	MAD Placebo (n=18)	MAD KT-621 (n=52)
Nausea (mild)	1 (5.6%)	0
Asthenia (mild)	0	1 (1.9%)

For more information on KT-621's Phase 1 Healthy Volunteer trial, visit clinicaltrials.gov (NCT06673667); ECG: electrocardiogram.

KT-621 BroADDen Phase 1b Demographics

Generally Well-Balanced Across Treatment Cohorts

	100 mg (n=10)	200 mg (n=12)	Overall (n=22)
Gender, n (%)			
Female	6 (60)	7 (58.3)	13 (59.1)
Male	4 (40)	5 (41.7)	9 (40.9)
Age, years, mean (SD)	30.1 (8.5)	33.0 (11.4)	31.7 (10.1)
BMI, kg/m^2, mean (SD)	32.8 (11.5)	30.8 (9.2)	31.7 (10.1)
Ethnicity, n (%)			
Hispanic or Latino	3 (30)	2 (16.7)	5 (22.7)
Non Hispanic or Latino	7 (70)	10 (83.3)	17 (77.3)
Race, n (%)			
White	4 (40)	3 (25)	7 (31.8)
Black or African American	5 (50)	7 (58.3)	12 (54.5)
Asian	0	1 (8.3)	1 (4.5)
Mixed/Other	1 (10)	1 (8.3)	2 (9.1)

For more information on KT-621's BroADDen Phase 1b atopic dermatitis trial, visit clinicaltrials.gov (NCT06945458); BMI: Body Mass Index; SD: Standard Deviation.

KT-621 BroADen Phase 1b Baseline Disease Characteristics

Generally Well-Balanced Across Treatment Cohorts

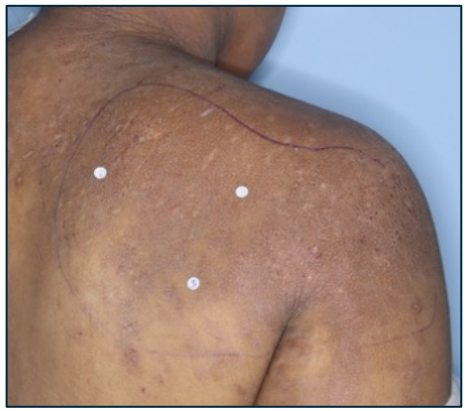
	100 mg (n=10)	200 mg (n=12)	Overall (n=22)
vIGA-AD, n (%)			
Moderate (3)	6 (60)	6 (50)	12 (54.5)
Severe (4)	4 (40)	6 (50)	10 (45.5)
EASI Score, mean (SD)	23.5 (7.5)	26.1 (9)	24.9 (8.3)
Other Disease Characteristics, Mean (SD)			
Peak Pruritus NRS	7.4 (1.2)	7.6 (0.9)	7.5 (1)
SCORAD	55.7 (15.6)	63.8 (13.4)	60.1 (14.7)
BSA (%)	29.1 (9.8)	30.0 (15.1)	29.6 (12.7)
Comorbid Type 2 Diseases, n (%)			
Asthma	1 (10)	3 (25)[3]	4 (18.2)
Allergic Rhinitis	2 (20)	7 (58.3)	9 (40.9)
Prior Systemic Therapy for AD, n (%)	1 (10)[1]	4 (33.3)[2]	5 (22.7)

[1]Patient had prior dupilumab treatment; [2]Two patients had prior dupilumab treatment, one had prior tralokinumab treatment, and one had received both agents; [3]The three patients also have comorbid Allergic Rhinitis; For more information on KT-621's BroADen Phase 1b atopic dermatitis trial, visit clinicaltrials.gov (NCT06945458); EASI: Eczema Area and Severity Index; vIGA-AD: Validated Investigator Global Assessment for Atopic Dermatitis; PPNRS: Peak Pruritus Numerical Rating Scale; BSA: Body Surface Area; SCORAD: SCORing Atopic Dermatitis; SD: Standard Deviation.

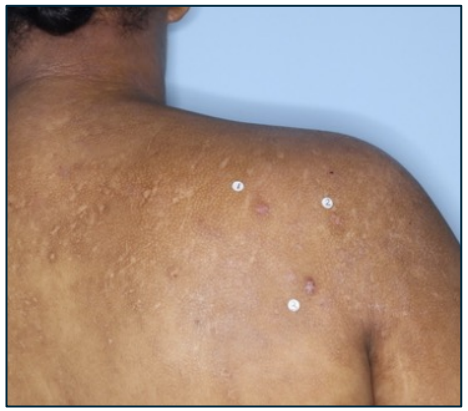
Example of Clinical and Biomarker Response to KT-621 in BroADen Patient with Severe AD and Prior Dupilumab Treatment

Pre and Day 29 Photos for EASI-75 Responder

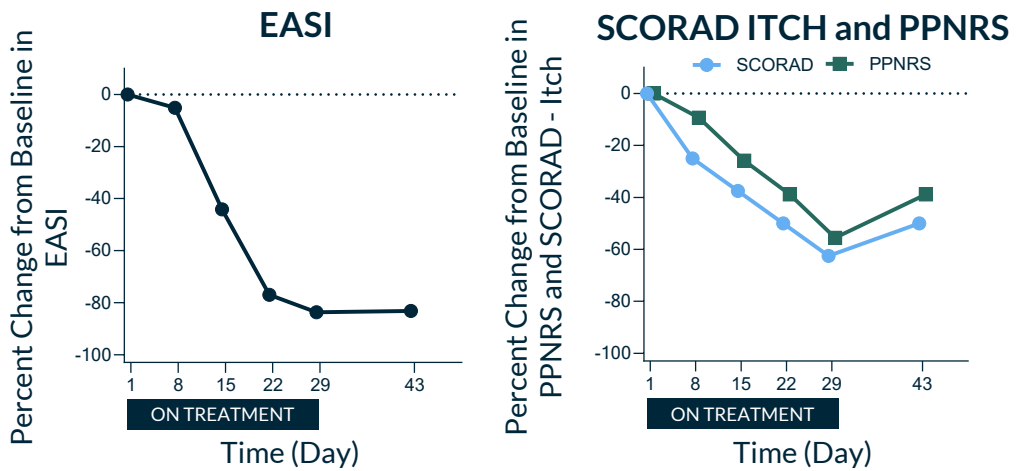
Baseline



Day 29



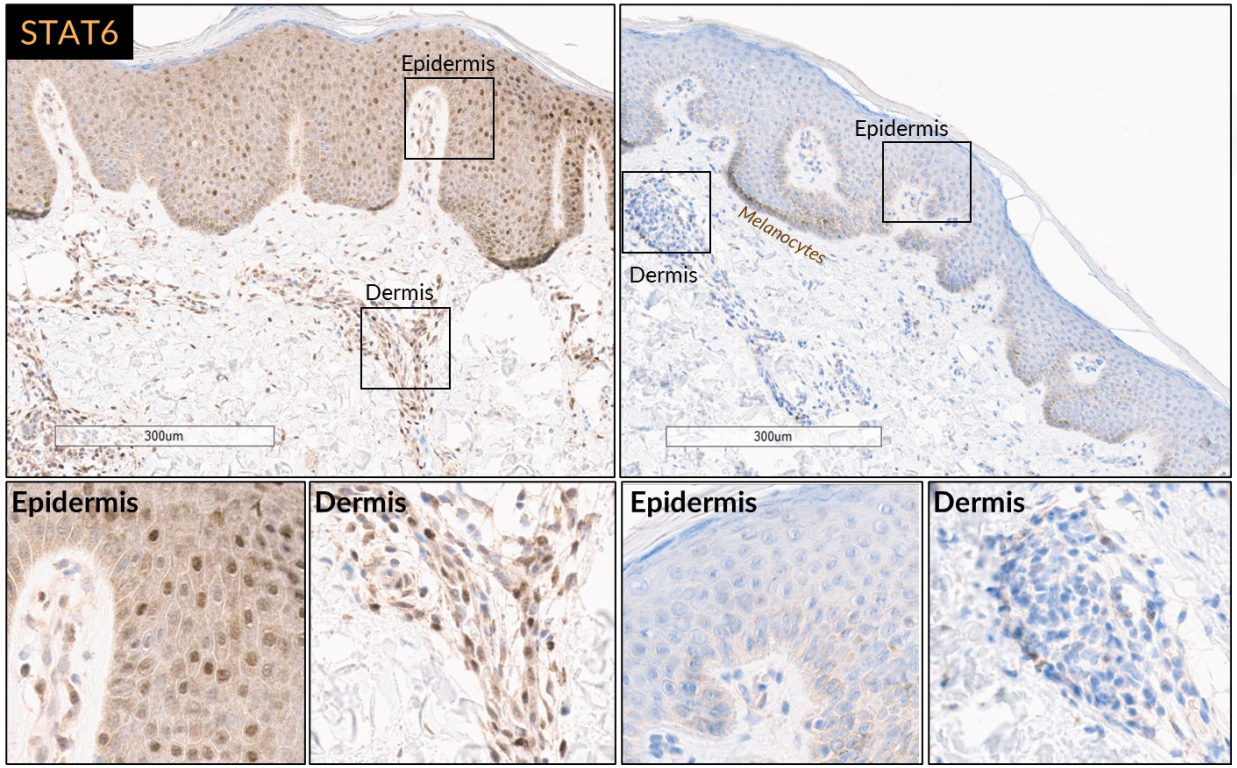
% Change in EASI and Itch[1]



STAT6 Staining IHC

Baseline

Day 29



Biomarker Changes at D29

	Blood STAT6	Skin STAT6	Serum TARC	Serum Eotaxin-3	Serum IgE	Skin Eotaxin-3	Skin KRT16
Change from Baseline	-95%	-94%	-78%	-96%	-19%	-92%	-93%

[1]Baseline EASI = 37.4, Baseline Pruritus NRS = 7.7; For more information on KT-621's BroADen Phase 1b atopic dermatitis trial, visit clinicaltrials.gov (NCT06945458); EASI: Eczema Area and Severity Index; SCORAD: SCORing Atopic Dermatitis; PPNRS: Peak Pruritus Numerical Rating Scale; IHC: Immunohistochemistry.

Atopic Dermatitis: Intensely Pruritic, Chronic and Underserved Disease

Opportunity Exists to Transform the Treatment Paradigm

- Chronic inflammatory disease that causes **inflamed and irritated skin**, making it itchy and painful
- Majority of patients have **disease onset as children** and carry the disease burden into **adulthood**
- Can significantly impact a **patient's quality of life,** disrupting daily activities
- Common comorbidities include **asthma** and **allergic rhinitis**
- Many options only address symptoms, **not underlying Type 2 inflammation**
- Estimated **43 million** adults in US/EU5/JP[1]
- **Less than 1 million** patients currently on dupilumab[2]

"There remains a clear need for new oral therapies that can address the underlying biology of the disease while potentially offering patients greater convenience."

– Eric Simpson, MD, MCR,
Frances J. Storrs Medical Dermatology Professor and Director of CLEAR Eczema Center, Oregon Health & Science University

[1]GlobalData (2023 diagnosed prevalent patient population for AD in US/EU5/JP); [2]GlobalData.

Asthma: Chronic, Inflammatory Lung Disease

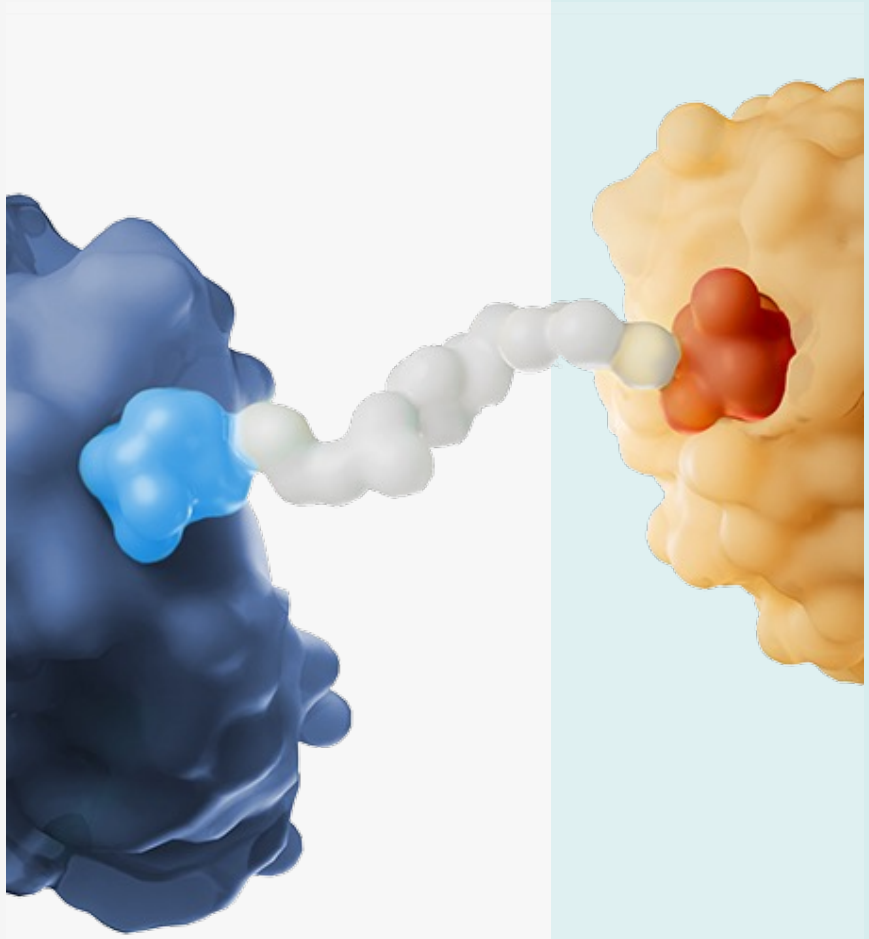
Opportunity Exists to Transform the Treatment Paradigm

- The airways in lungs to become swollen and inflamed, causing coughing, shortness of breath, chest tightness or pain, and **making it harder to breathe**
- **Symptoms may resemble other lung conditions,** called asthma mimics, and can be misdiagnosed as a result
- Can significantly impact a **patient's quality of life** and lead to permanent lung damage
- Many options only address symptoms, **not underlying Type 2 inflammation**
- Estimated **55 million** adults in US/EU5/JP[1]
- **Less than 1 million** patients currently on dupilumab[2]

Participant expressed hope "for future treatments that are easier to use…and better routes of administration than inhalers or injections due to fear of needles."

– Little Airways, Big Voices[3]
Patient Focused Drug Development: Asthma

[1]Diagnosed prevalent patient population for US/EU5/JP (GlobalData; 2023); [2]GlobalData; [3]Little Airways, Big Voices, Patient Focused Drug Development, 2021.



Appendix: Partnered Programs

IRAK4 Biology and Target Rationale

Target Rationale

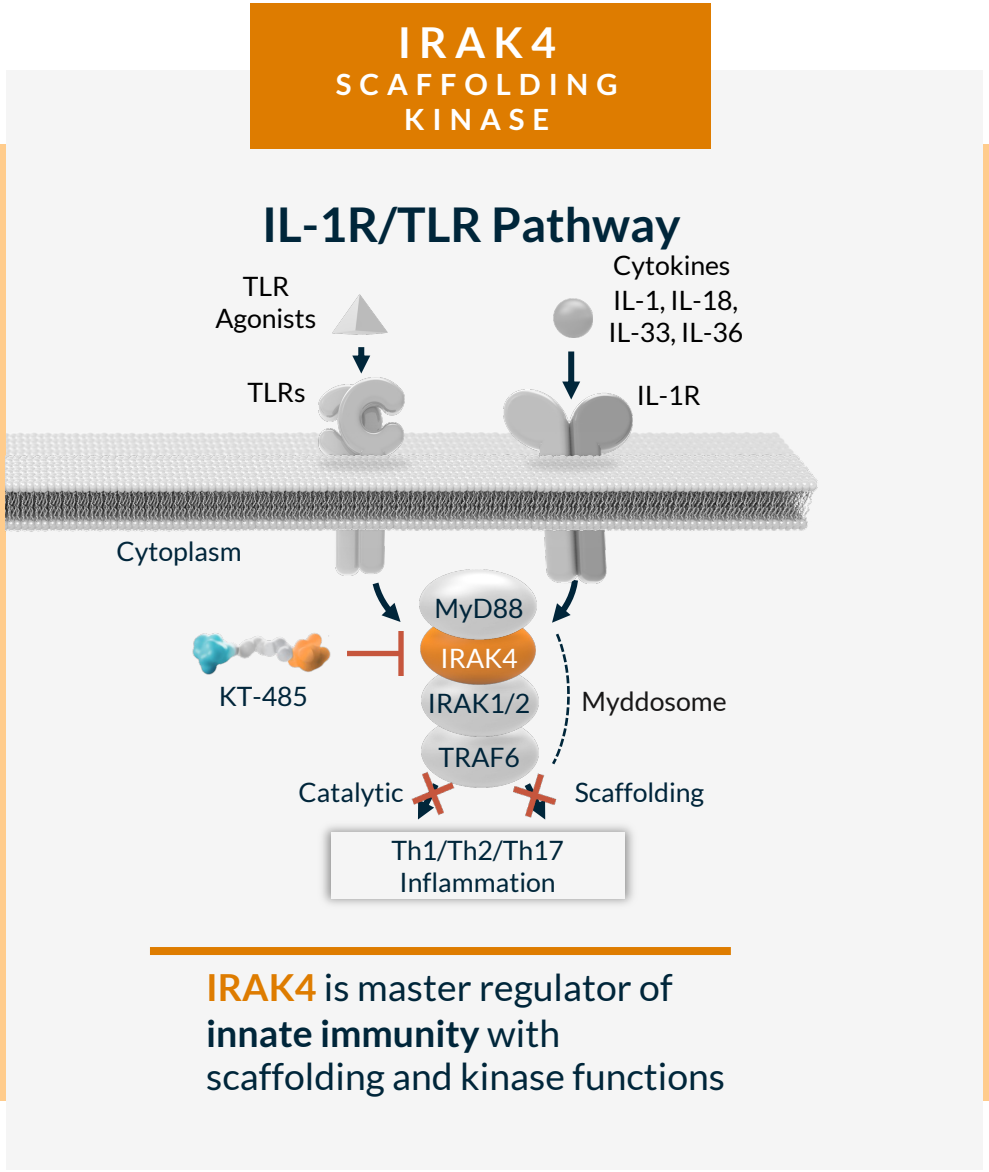
- IRAK4 is an obligate node in IL-1R/TLR signaling, and its degradation is the only approach to fully block the pathway

Human Genetics

- Adult humans with IRAK4 null mutation are healthy

Clinical Pathway Validation

- IRAK4 degradation has the potential to achieve a broad, well-tolerated anti-inflammatory effect
- Multiple development opportunities in immune-inflammatory diseases which signal through MyD88/IRAK4 have been clinically validated[1]:
  - IL-1α/IL-1β: RA, CAPS, HS, AD, RP, Gout
  - IL-18: AD, Macrophage Activation Syndrome
  - IL-36: Generalized Pustular Psoriasis
  - IL-33: Asthma, COPD
  - IRAK4 SMI: RA



[1]Diseases where IL-1R/TLR pathway has been implicated in pathogenesis; Figure adapted from West NT. *Front Immunol* 2019.

CDK2 Molecular Glue Degrader Advantage

Target Biology and Rationale

- Regulates the G1-S transition, with its partner CCNE
- CCNE is often overexpressed or amplified in HGSOC and CDK4/6i resistant breast cancers, driving uncontrolled cell proliferation

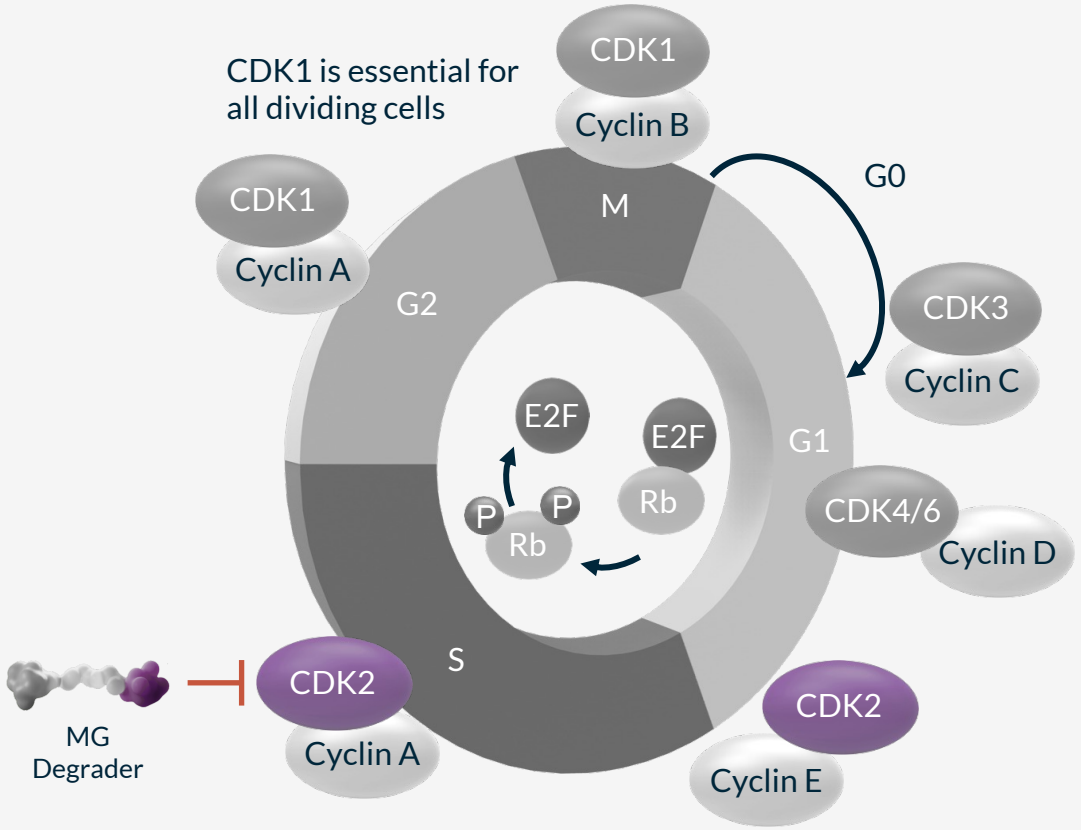
Genetically Validated

- DEPMAP identifies CCNE1 amplification as a biomarker of dependency for CDK2 across cancer cell types
- High CCNE activity is associated to resistance of CDK4/6 therapies

Advantages of MG Degraders

- CDK2 inhibitors face challenges due to similarity with other CDKs, including CDK1, causing toxicities before effective CDK2-pRB pathway inhibition can be achieved
- MG degraders offer greater selectivity, and potentially broader therapeutic index with more complete CDK2 pathway inhibition

CDK2
MASTER CELL CYCLE REGULATOR



Abbreviations

Ab	Antibody
AD	Atopic Dermatitis
AIA	Antigen-Induced Arthritis
ASMS	Affinity Selection Mass Spectrometry
AN Count	Abscess and Inflammatory Nodule Count
BID	Twice a day
BP	Bullous Pemphigoid
BSA	Body Surface Area
CAPS	Cryopyrin-Associated Periodic Syndrome
CD	Crohn's Disease
CDK2	Cyclin-Dependent Kinase 2
COPD	Chronic Obstructive Pulmonary Disease
CRSwNP	Chronic Rhinosinusitis with Nasal Polyps
Cryo-EM	Cryo-Electron Microscopy
Ctrl	Control
CSU	Chronic Spontaneous Urticaria
$DC_{\#}$	Degradation Concentration
DCs	Dendritic Cells
DEL	DNA-Encoded Library
DLQI	Dermatology Life Quality Index
DM	Dermatomyositis
DMSO	Dimethyl Sulfoxide
dsDNA	Double-Stranded DNA
EASI	Eczema Area and Severity Index
EoE	Eosinophilic Esophagitis
EU	European Union
FDA	Food and Drug Administration
FeNO	Fractional Exhaled Nitric Oxide
FIH	First-in-Human
GI	Gastrointestinal
GLP	Good Laboratory Practice
GOF	Gain of Function
GWAS	Genome-Wide Association Study
HD	High Dose
HDM	House Dust Mite
HiSCR	Hidradenitis Suppurativa Clinical Response
hPBMC	Human Peripheral Blood Mononuclear Cells
HS	Hidradenitis Suppurativa
HTS	High Throughput Screening
HV	Healthy Volunteers
I&I	Immunology and Inflammation
IA	Interim Analysis
IBD	Inflammatory Bowel Disease
$IC_{\#}$	Inhibitory Concentration
ICS	Inhaled Corticosteroid
IFN	Interferon
ISG	Interferon-Stimulated Gene
IGA	Investigator Global Assessment
IgE	Immunoglobulin E
IHS4	International Hidradenitis Suppurativa Severity Score
IL	Interleukin
IND	Investigational New Drug Application
IRAK4	Interleukin 1 Receptor Associated Kinase 4
IRF5	Interferon Regulatory Factor 5
JP	Japan
KO	Knockout
LABA	Long-Acting Beta Agonist
LAMA	Long-Acting Muscarinic Antagonist
LD	Low Dose
LLOQ	Lower Limit of Quantification
LOF	Loss of Function
LPS	Lipopolysaccharide Solution
LTRA	Leukotriene Receptor Antagonist
MAD	Multiple Ascending Dose Study
MOA	Mechanism of Action
NF-kB	Nuclear Factor Kappa B
NHP	Nonhuman Primate
nM	Nanomolar
NRS	Numerical Rating Scale
PBMC	Peripheral Blood Mononuclear Cells
Pbo	Placebo
PGA	Physician Global Assessment

Abbreviations

Ph	Phase
PK/PD	Pharmacokinetics/Pharmacodynamics
pM	Picomolar
PN	Prurigo Nodularis
POC	Proof-of-Concept
POEM	Patient-Oriented Eczema Measure
PPNRS	Peak Pruritus Numerical Rating Scale
PRRs	Pattern Recognition Receptors
PsO	Psoriasis
pSTAT	Signal Transducer and Activator of Transcription
QD	Once a day
QoL	Quality of Life
R&D	Research and Development
RA	Rheumatoid Arthritis
RNA	Ribonucleic Acid
ROW	Rest of World
RP	Recurrent Pericarditis
RQLQ	Rhinoconjunctivitis Quality of Life Questionnaire
SAD	Single Ascending Dose study
SAE	Serious Adverse Event
SCORAD	SCORing Atopic Dermatitis
SD	Single Dose
SLE	Systemic Lupus Erythematosus
SMI	Small Molecule Inhibitor
SOC	Standard of Care
SSc	Systemic Sclerosis
STAT	Signal Transducer and Activator of Transcription
STAT6	Signal Transducer and Activator of Transcription 6
TARC	Thymus and Activation-Regulated Chemokine
TEAE	Treatment Emergent Adverse Event
Th1	T Helper 1
Th2	T Helper 2
Th17	T Helper 17
TLR	Toll-like Receptors
TNSS	Total Nasal Symptom Score
TPD	Targeted Protein Degradation
TPP	Target Product Profile
TRAE	Treatment Related Adverse Event
TYK2	Tyrosine Kinase 2
UC	Ulcerative Colitis
US	United States
vIGA-AD	Validated Investigator Global Assessment for AD
WW	Worldwide