KYMERA 10 EST. 2016

# Revolutionizing Immunology with Oral Medicines

October 2026

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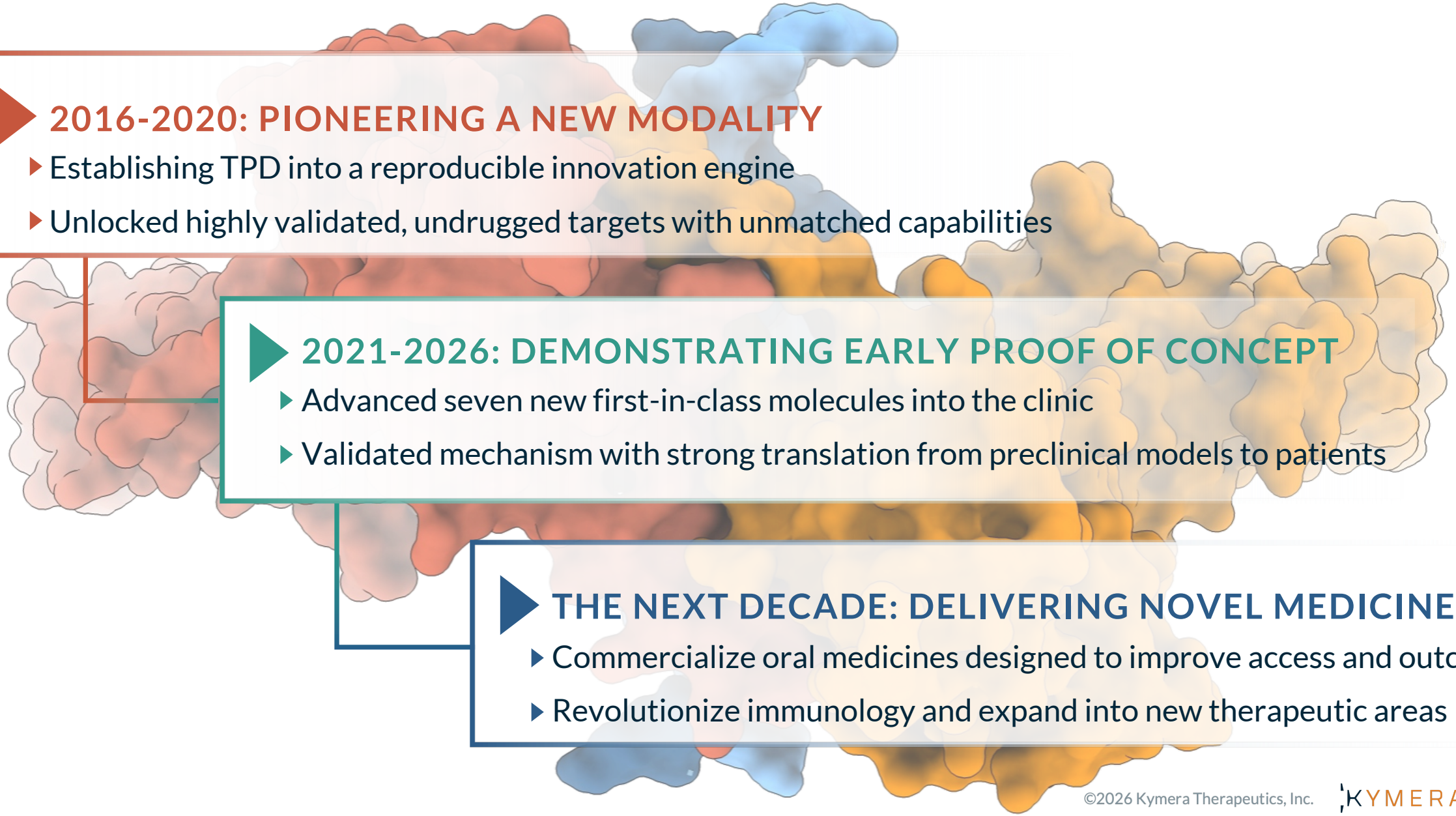
# 2016-2026: A Decade of Discovery and Progress in Motion



## 2016-2020: PIONEERING A NEW MODALITY

- Establishing TPD into a reproducible innovation engine
- Unlocked highly validated, undrugged targets with unmatched capabilities

## 2021-2026: DEMONSTRATING EARLY PROOF OF CONCEPT

- Advanced seven new first-in-class molecules into the clinic
- Validated mechanism with strong translation from preclinical models to patients

## THE NEXT DECADE: DELIVERING NOVEL MEDICINES

- Commercialize oral medicines designed to improve access and outcomes
- Revolutionize immunology and expand into new therapeutic areas

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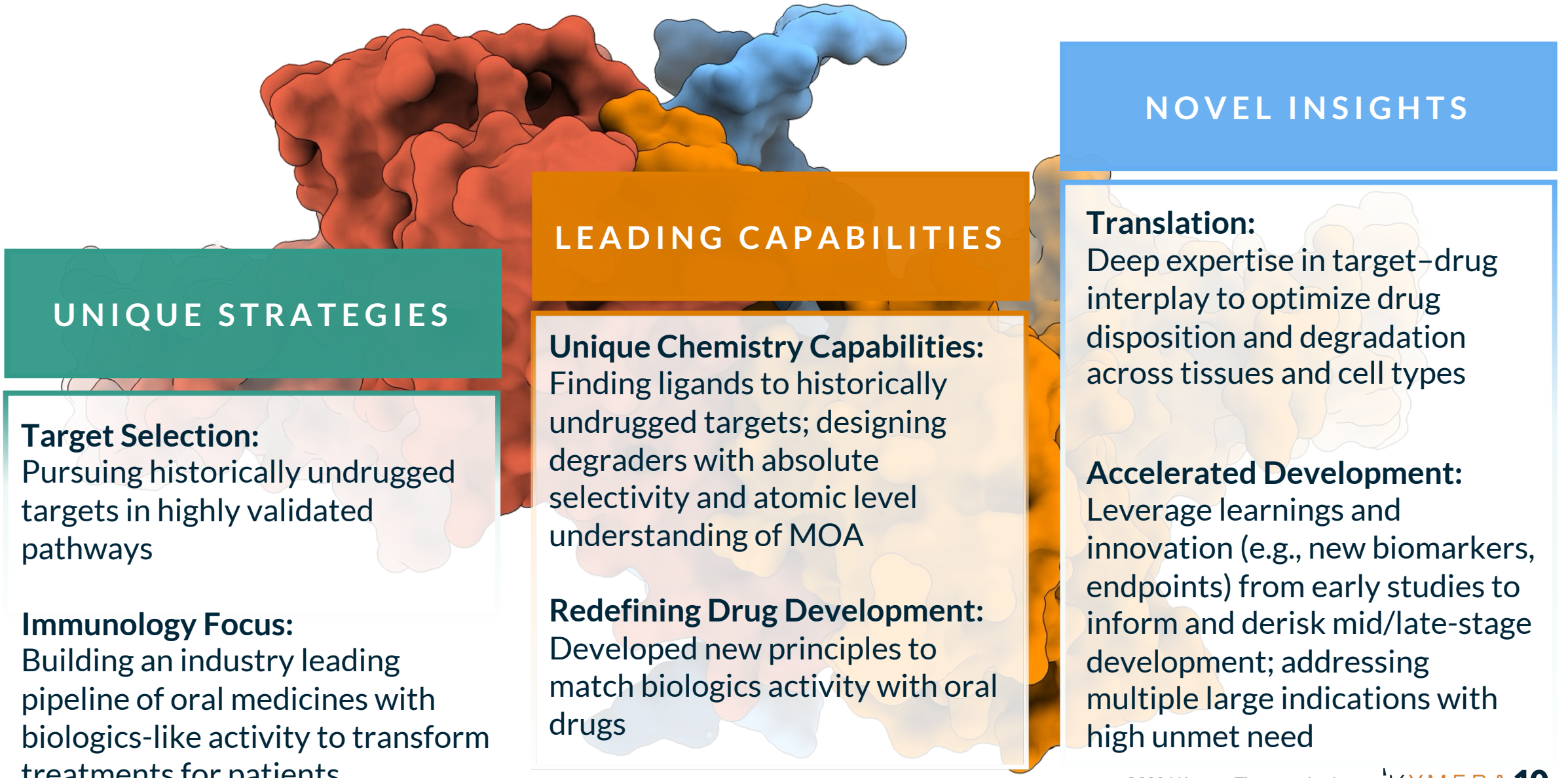
# Kymera: Industry Leader in Developing Oral Degrader Medicines



## 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

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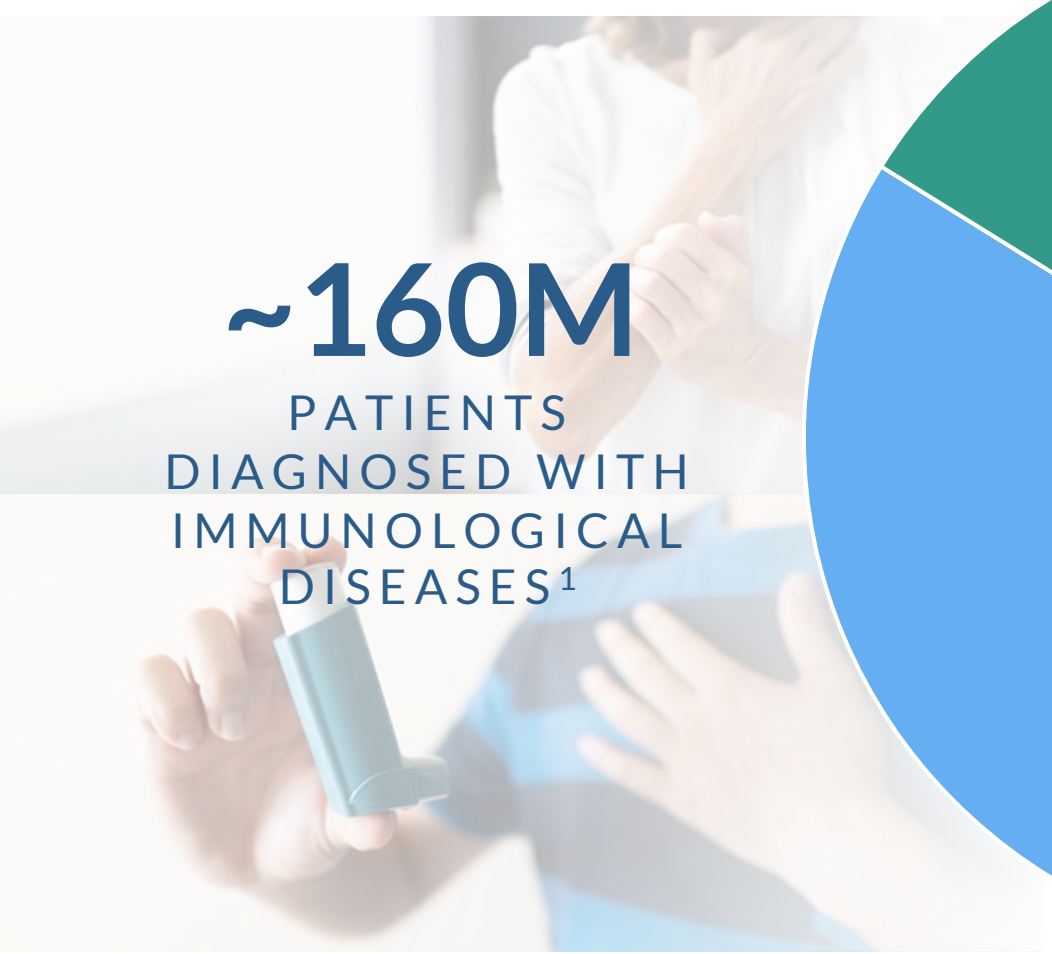
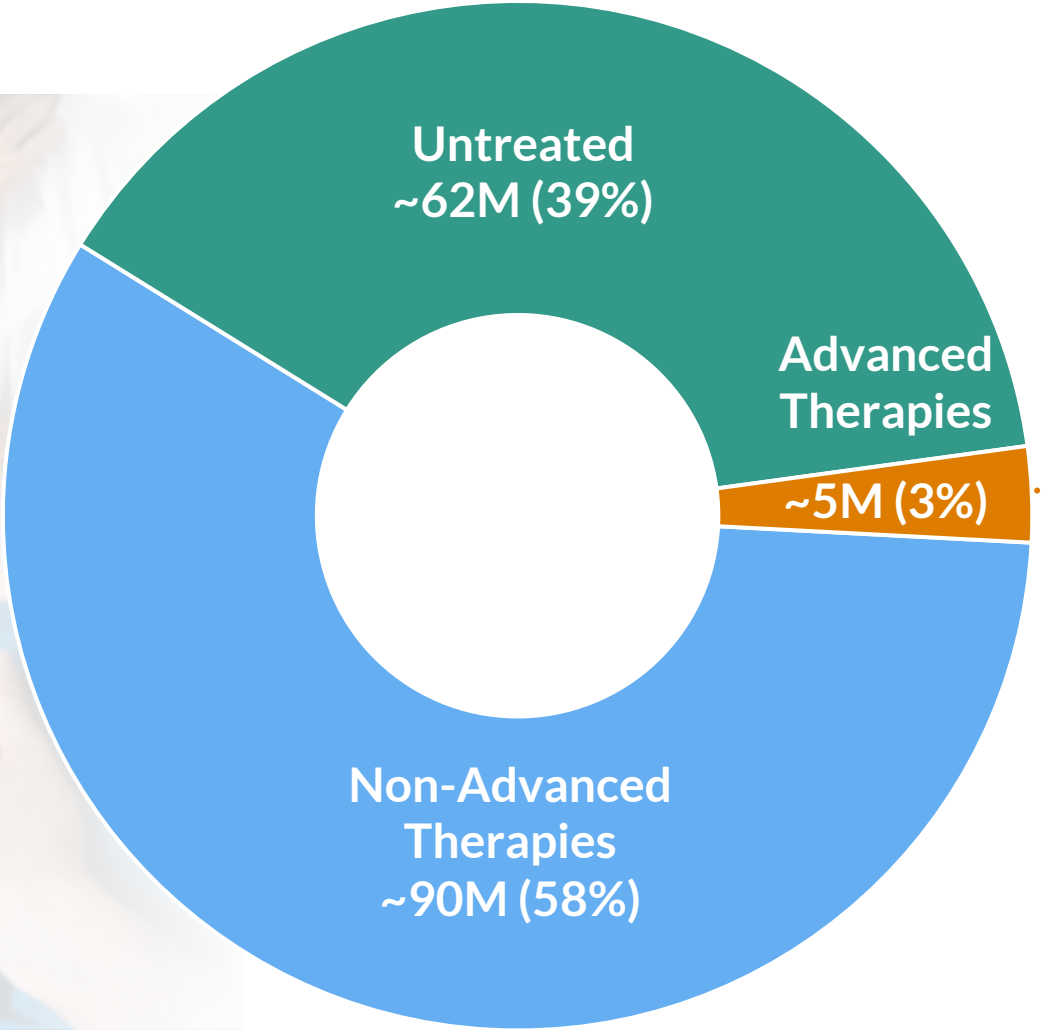
# Immunology Remains a Large Underserved Market

## 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).

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# 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.

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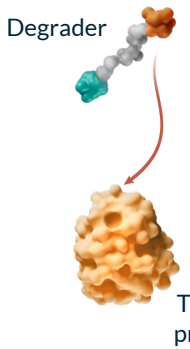
# Targeted Protein Degradation: Achieving Biologics-like Activity with Oral Medicines

## Targeted Protein Degradation (TPD) Mechanism of Action

Harnessing the E3 Ubiquitin Proteasome System

**1**

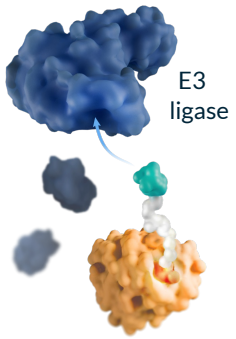
**Catching the Target Protein**




Degrader molecule engages target protein and E3 ubiquitin ligase to bring them in close proximity

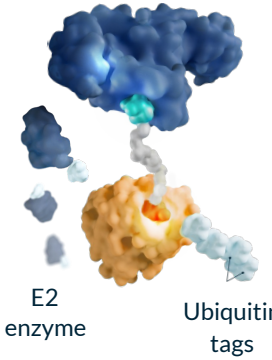
**2**

**Presenting the Target Protein**




**3**

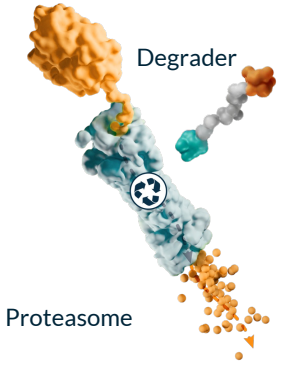
**Tagging the Target Protein**




E3 ligase places a chain of ubiquitin molecules on the target protein

**4**

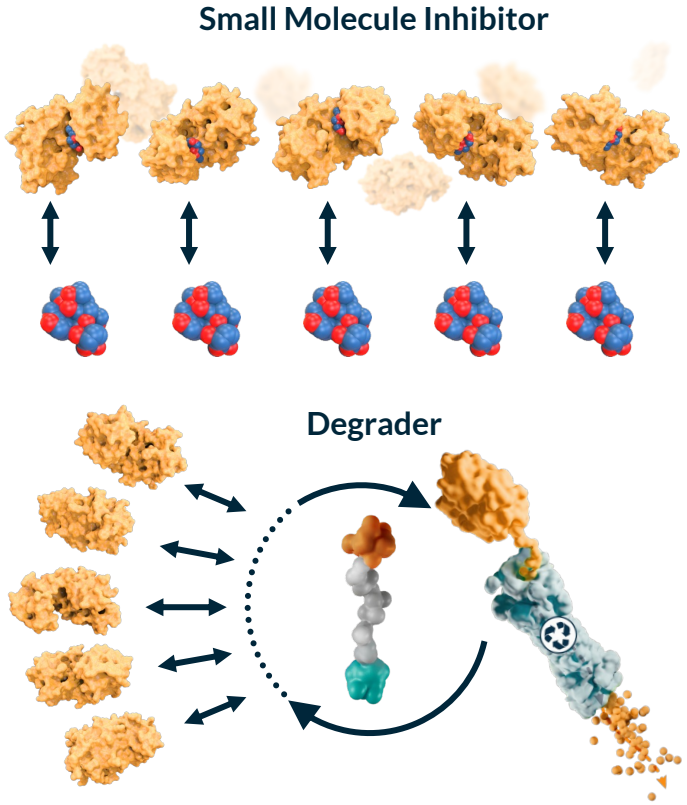
**Degrading the Target Protein**




Ubiquitin is recognized by the proteasome to selectively degrade the target protein

## Degraders Enable Continuous, Complete Pathway Blockade

Superior to Traditional Small Molecule Inhibitors




### STOICHIOMETRIC INHIBITION

Pharmacodynamic effect (PD) correlated to drug exposure pharmacokinetics (PK) (stoichiometric) requiring continuous high drug exposures to block protein function[1]

### CONTINUOUS DEGRADATION

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

Catalytic activity of degraders enables a single molecule to drive degradation of multiple copies of the target protein, delivering deep and continuous pathway blockade with biologics-like activity in a pill

[1]Mainolfi N, Rasmusson T, *Annual Reports Med Chem*, 2017.

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# 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**

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# Building a Best-in-Industry Oral Immunology Pipeline

## Combining the Convenience of Oral Drugs and the Activity of Biologics

| | 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<br>KT-621 – Asthma | | | | **Ph2b AD Data:** By year-end 2026<br>**Ph2b Asthma Data:** Late-2027<br>**Ph3 AD Start:** By mid-2027 |
| **IRF5** | Lupus, Sjögren's, RA, IBD, SSc, DM, others | >10M | KT-579 | | | | **Ph1b Lupus Start:** By early 2027 |
| **Partnered Programs** | | | | | | | |
| **IRAK4[2]** | HS, AD, RA, Asthma, IBD, others[3] | >140M | KT-485 | | | | sanofi |
| **CDK2[4]** | Breast cancer and other solid tumors | >500K | KT-200 | | | | **IND:** 2027<br>GILEAD |

[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.

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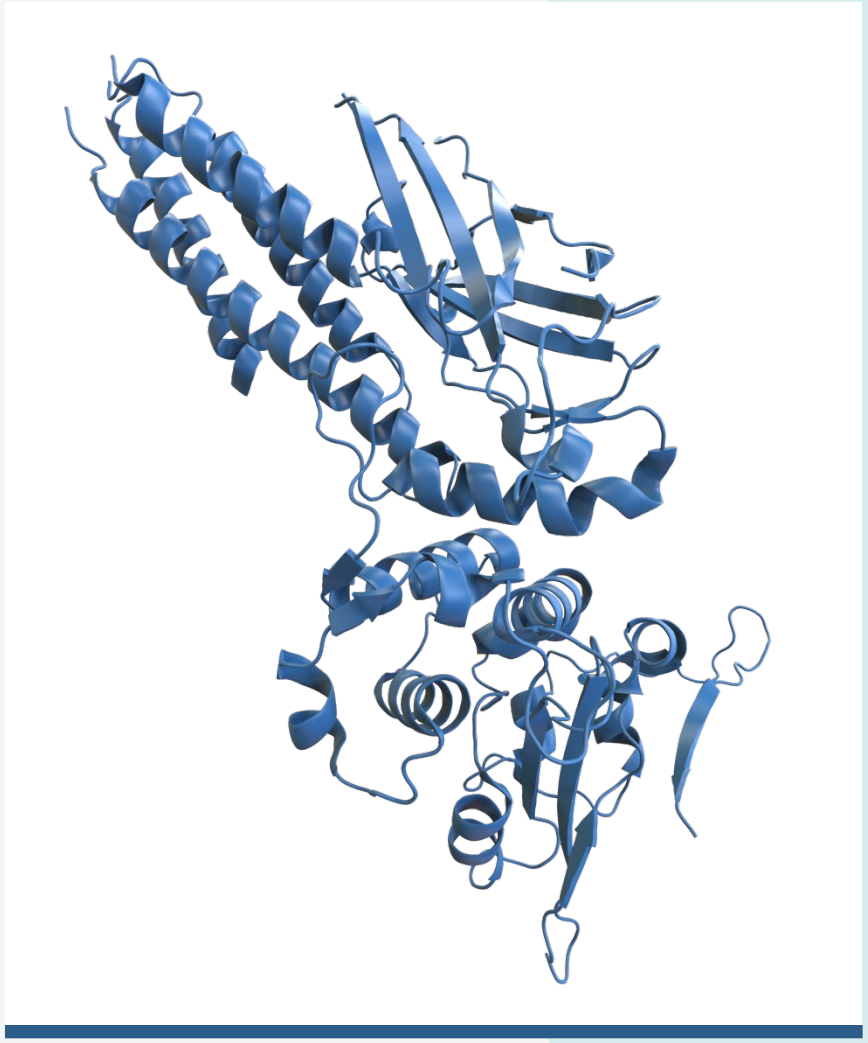

# First-in-Class Oral STAT6 Degrader Program

## Dupilumab-like activity in a pill

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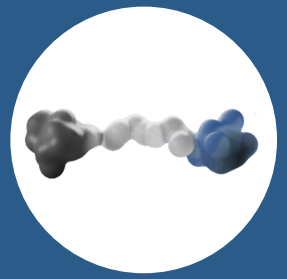
# KT-621 Summary

## 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

- Enrollment complete in BROADEN2 Phase 2b AD trial, with data by year-end 2026
- Phase 3 trials in AD planned to initiate 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).

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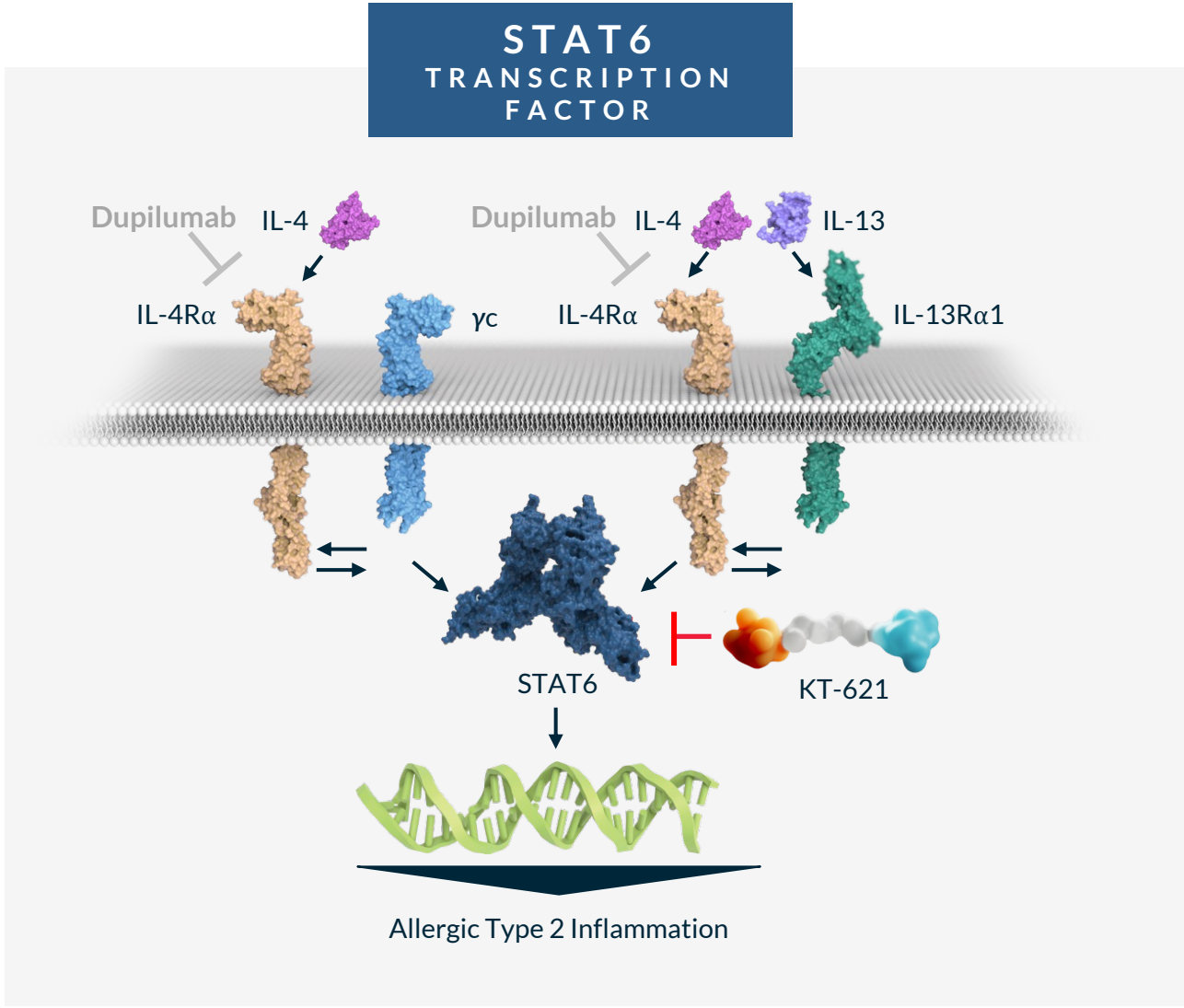
# STAT6 Transcription Factor: Highly Validated but Undrugged Target

- 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.

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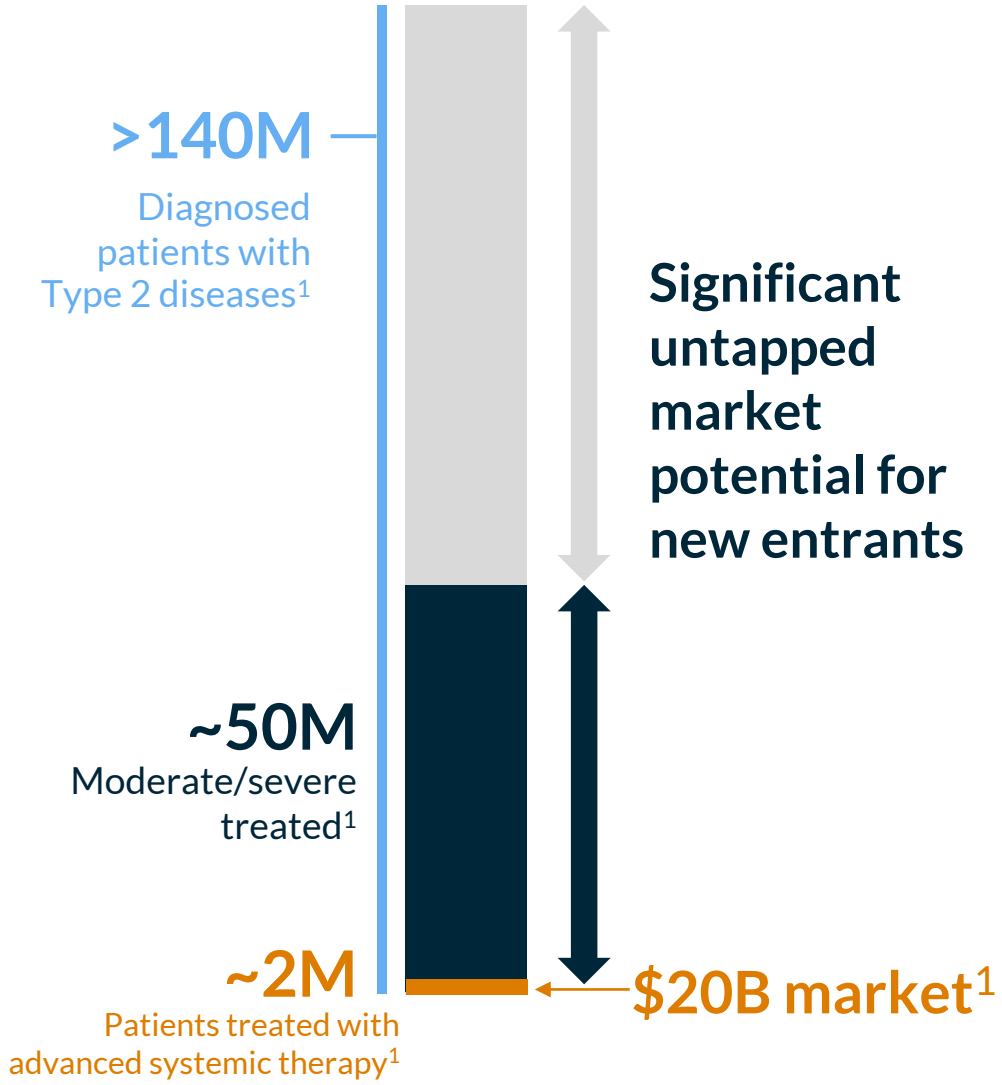
# Unlocking the Full Market Potential in Type 2 Disease




## 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 and forecasted sales for AD, Asthma, and COPD in US/EU5/JP;
[2]Evaluate Ltd (Total WW Market Value Top 10 Products 2014-2024).

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# KT-621: Potential to Transform Type 2 Inflammatory Diseases

KT-621 has the potential to be a novel oral medicine capable of delivering biologics-like efficacy and safety

## KT-621 PARADIGM SHIFT

- ✓ Differentiated approach to **address underlying disease** biology
- ✓ Potential for **meaningful efficacy, favorable safety** profile and robust durability
- ✓ Highly validated, **non-immunosuppressive pathway** supporting long-term use
- ✓ Convenience of a **once-daily oral medicine**

**>90%**

currently treated with an injectable would be willing to try a safe and equally effective oral therapy[1]

**~50%**

of adult psoriasis patients and dermatology providers said they'd prefer an oral therapy over a topical or an injectable[1]

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

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# 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.

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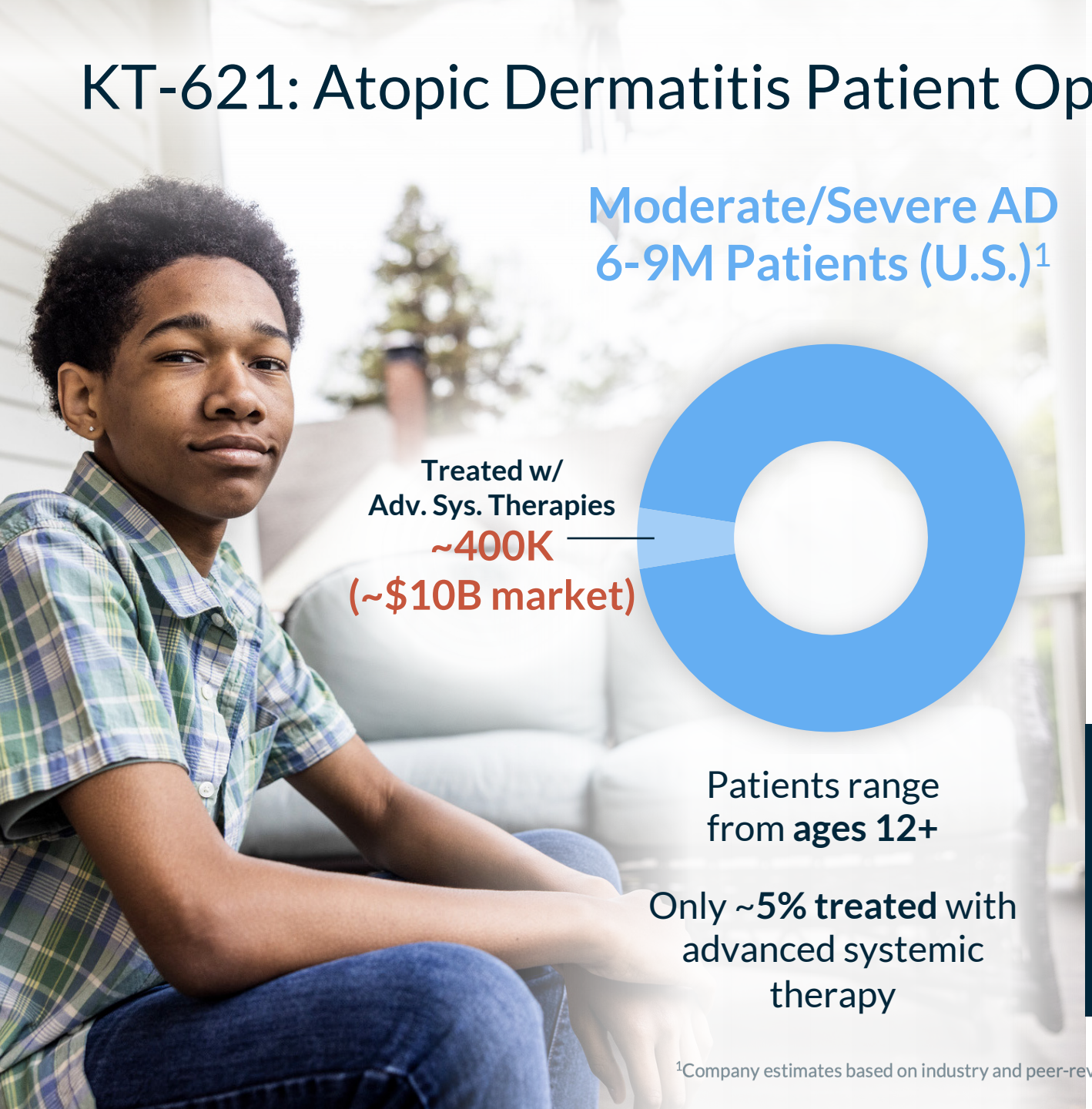
# KT-621: Atopic Dermatitis Patient Opportunity



## Moderate/Severe AD 6-9M Patients (U.S.)[1]




Patients range from **ages 12+**

Only ~**5% treated** with advanced systemic therapy

## Reasons for Low Penetration

- **Reluctance to use Advanced Systemic Therapies**
  Many patients don't accept their disease state to justify an injectable biologic or oral option with boxed safety warnings
- **High Treatment Burden**
  Multiple injections, blood tests, cold storage, access/reimbursement challenges

**KT-621**

- Opportunity to be a first line treatment option
- Potentially the first safe, effective, oral for patients across all Type 2 diseases

[1]Company estimates based on industry and peer-reviewed research.

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

BROADEN2 TRIAL

**Ph2b AD**
Dose Ranging

Enrollment complete
Data expected by year-end 2026

BREADTH TRIAL

**Ph2b Asthma**
Dose Ranging

Ongoing
Data expected late-2027

## PHASE 3 OPPORTUNITIES

- Atopic Dermatitis (initiate by mid-2027)
- Eosinophilic Esophagitis
- Chronic Spontaneous Urticaria
- Prurigo Nodularis
- Bullous Pemphigoid
- Asthma
- Chronic Rhinosinusitis with Nasal Polyps
- Chronic Obstructive Pulmonary Disease

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

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# KT-621 Data Provide Validation and Derisk Future Clinical Trials

## Preclinical

### POTENCY

- <100 pM DC90 in all relevant human cell types

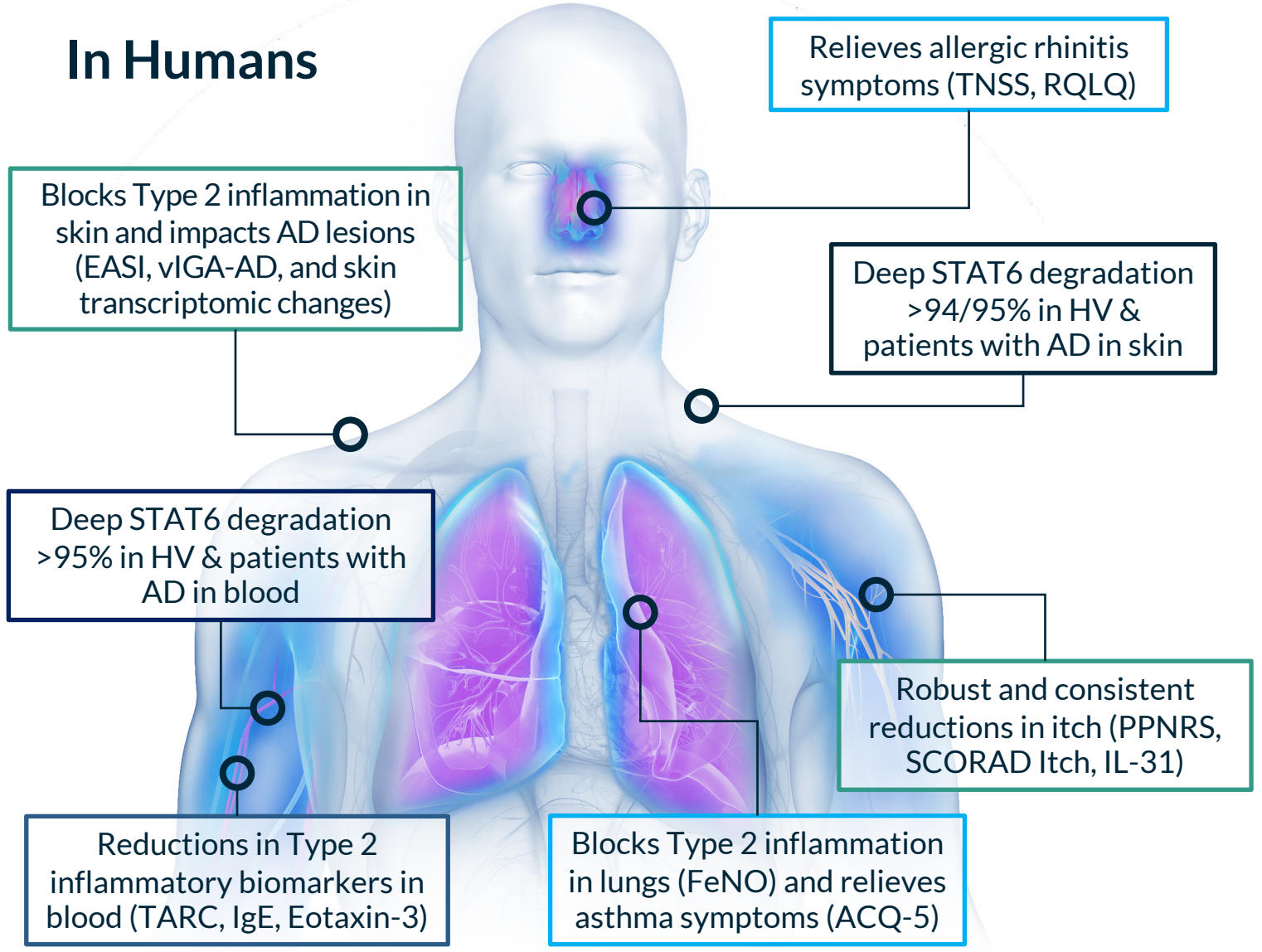
### EFFICACY

- 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

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# KT-621: BROADEN2 Phase 2b Trial

## Accelerated Timeline, KT-621 Program Six Months Ahead of Schedule

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:
**Enrollment complete; Data expected by year-end 2026**

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# 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
- 52-week open label extension

## 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**

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

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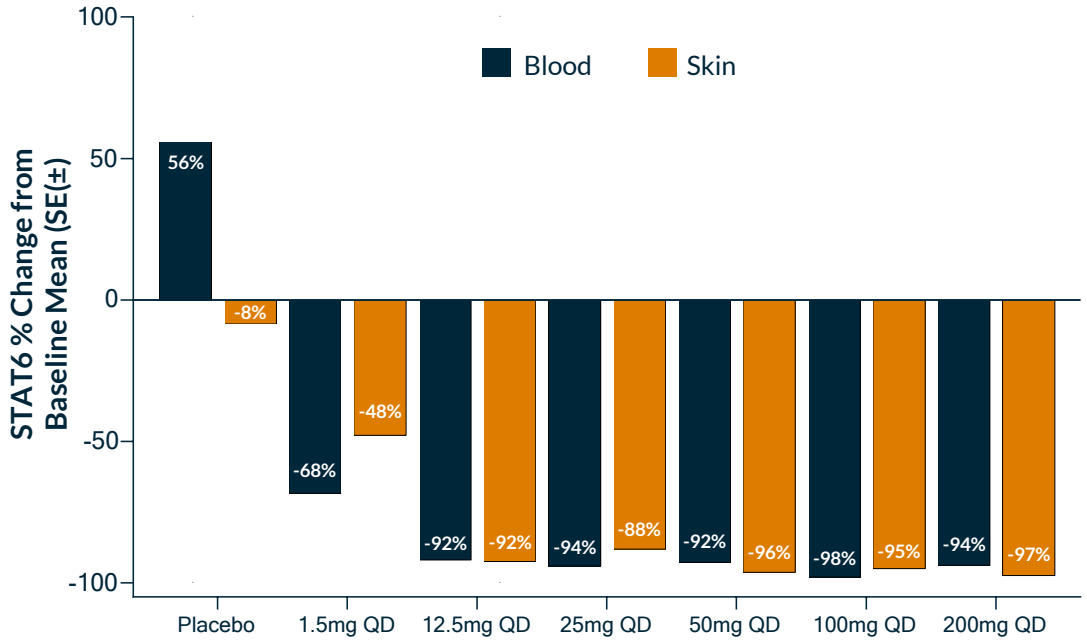
# KT-621: Compelling Clinical Profile

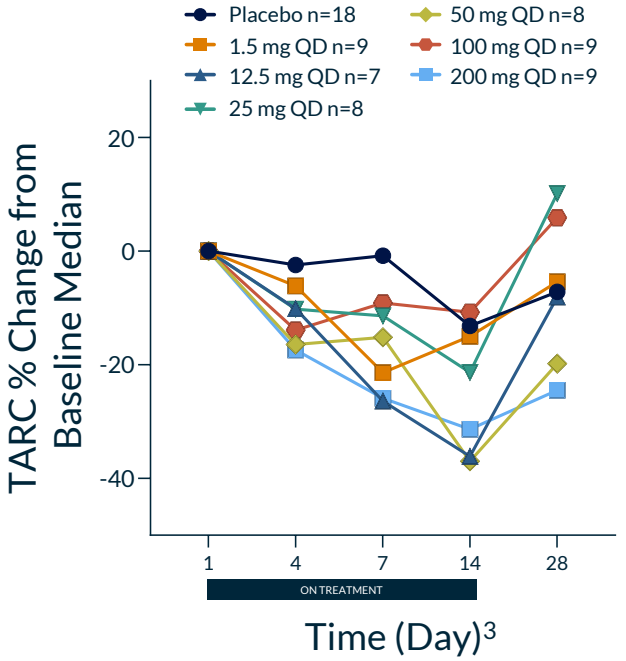
## KT-621 Phase 1 Healthy Volunteer Data Suggests Potential for Dupilumab-like Activity in a Pill

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

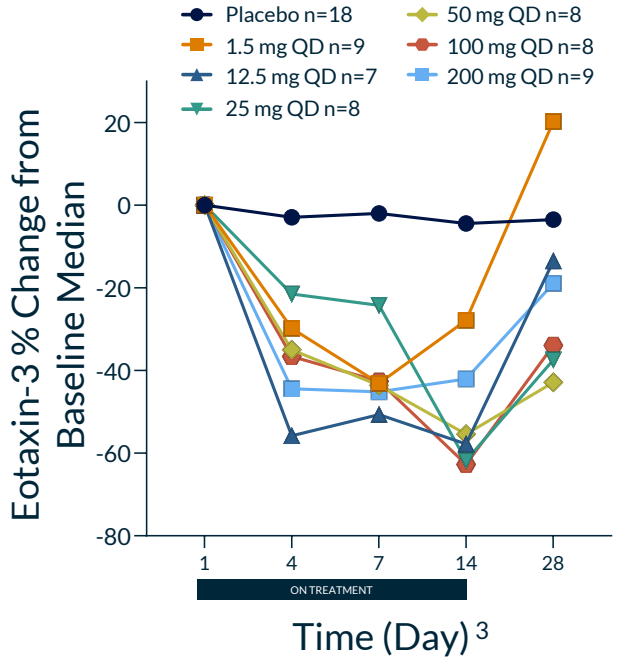



### Reductions in Multiple Disease-Relevant Type 2 Biomarkers

#### 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.

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# 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<br>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.

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# 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.

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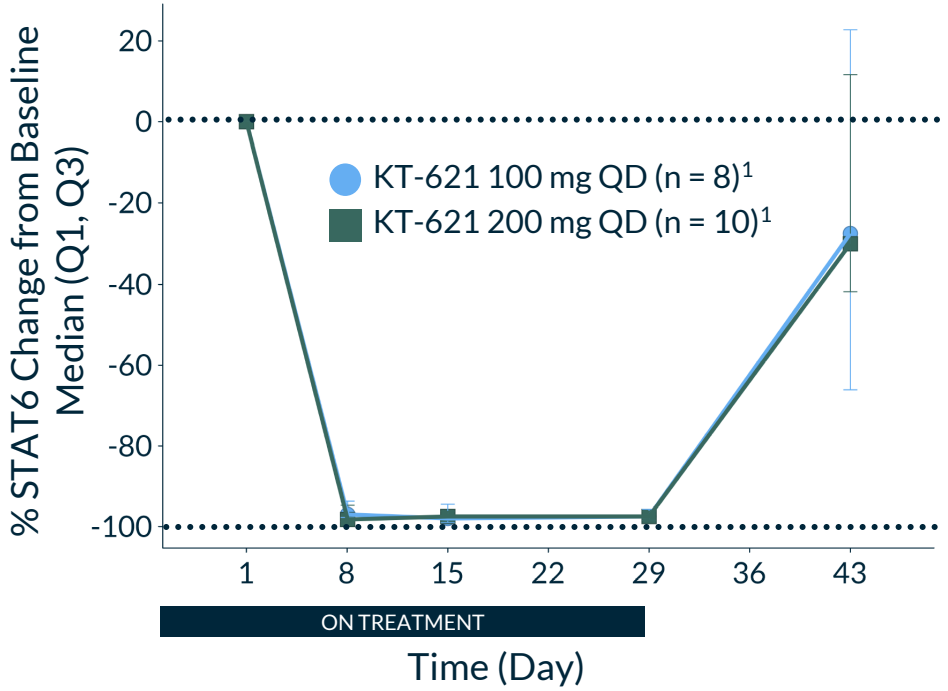
# KT-621 Achieved Deep STAT6 Degradation in Blood and Skin

## Degradation Maintained for 28 Days Across Both Dose Cohorts

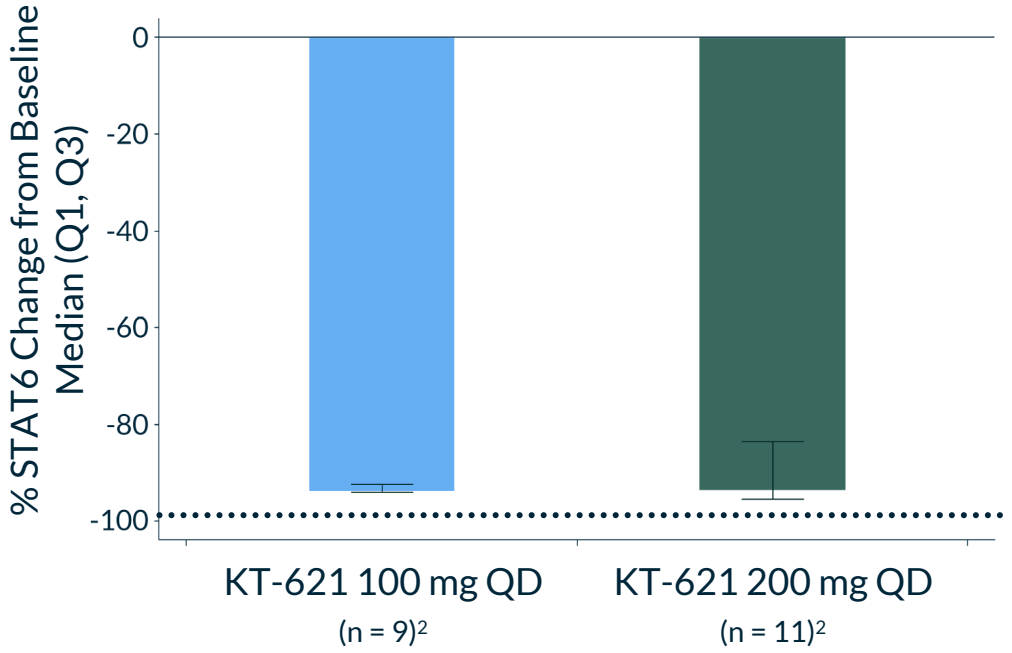
### Median STAT6 Degradation in Blood

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.

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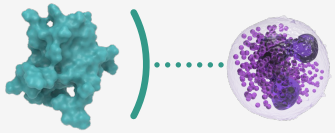
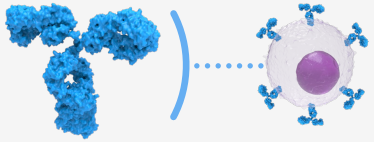
# Robust Impact on All Disease-Relevant Biomarkers of Type 2 Inflammation in BroADen Phase 1b AD Trial

| | TARC (CCL17) | Eotaxin-3 (CCL26) | IgE | IL-31 | FeNO |
|---|---|---|---|---|---|
| | |  |  | | |
| | Validated biomarker of Type 2 inflammation suppression in patients<br><br>Drives chemotaxis of CCR4-expressed T cells to inflammatory sites | Highly specific downstream cytokine of the IL-4/13 pathway<br><br>Drives chemotaxis of CCR3-expressed inflammatory cells to inflamed sites | IL-4 promotes B-cell class switching, amplifying IgE production<br><br>IgE activates mast cells and basophils to release Type 2 cytokines | Key pruritogenic cytokine produced by activated Type 2 cells[1]<br><br>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]<br><br>FeNO reflects airway epithelial iNOS activity driven by IL-4/13 signaling<br><br>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.

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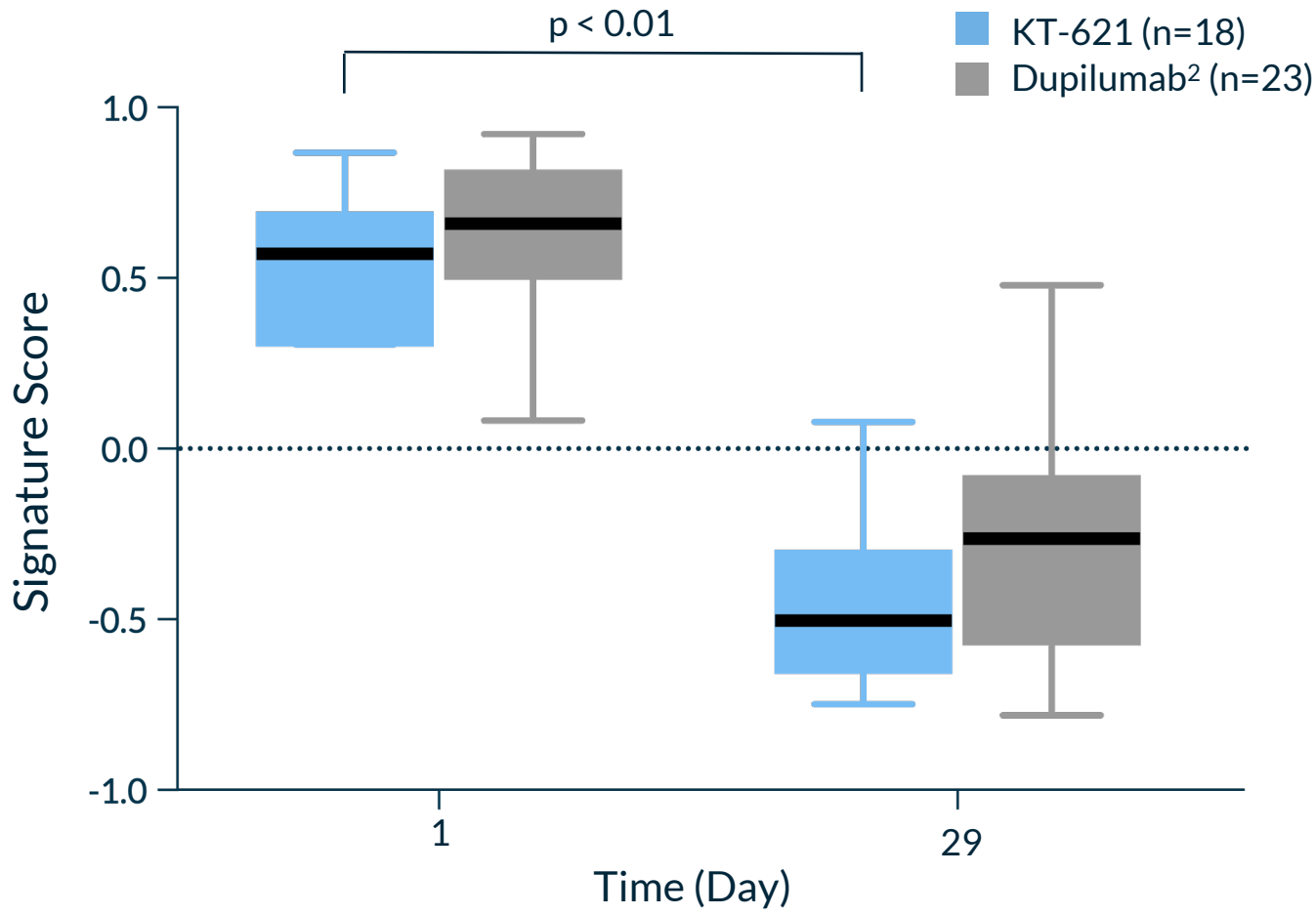
# KT-621 Significantly Downregulated Core Type 2 Inflammation Gene Set in Skin Lesions of AD Patients

- **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.

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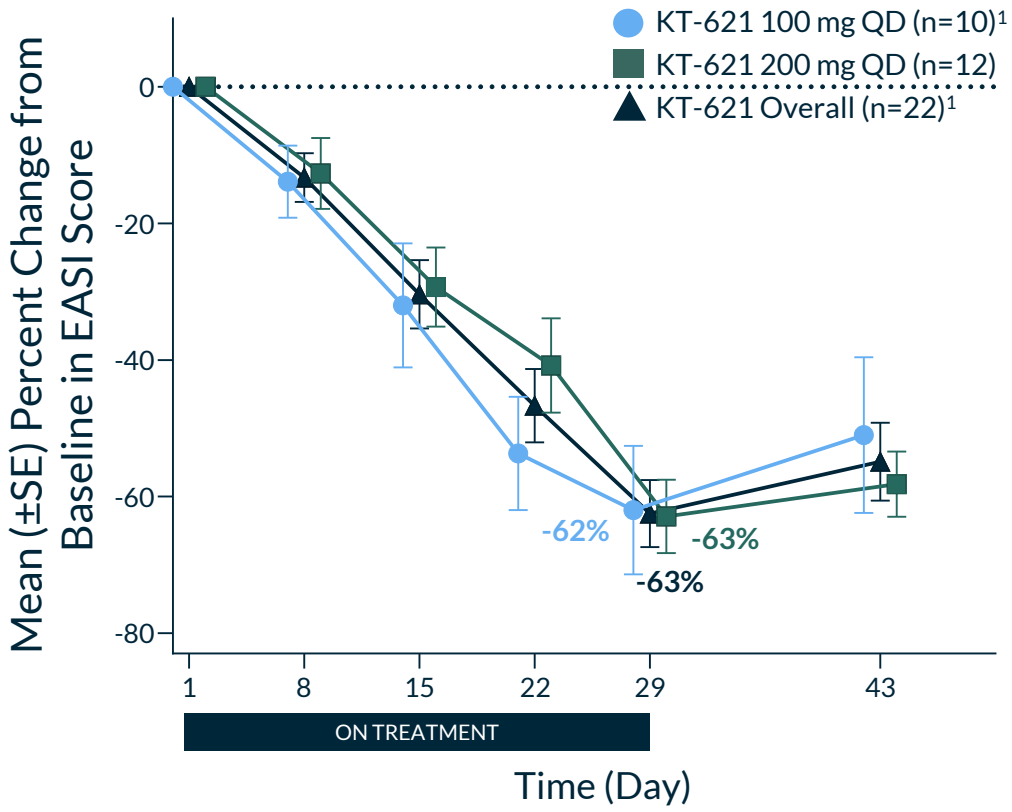
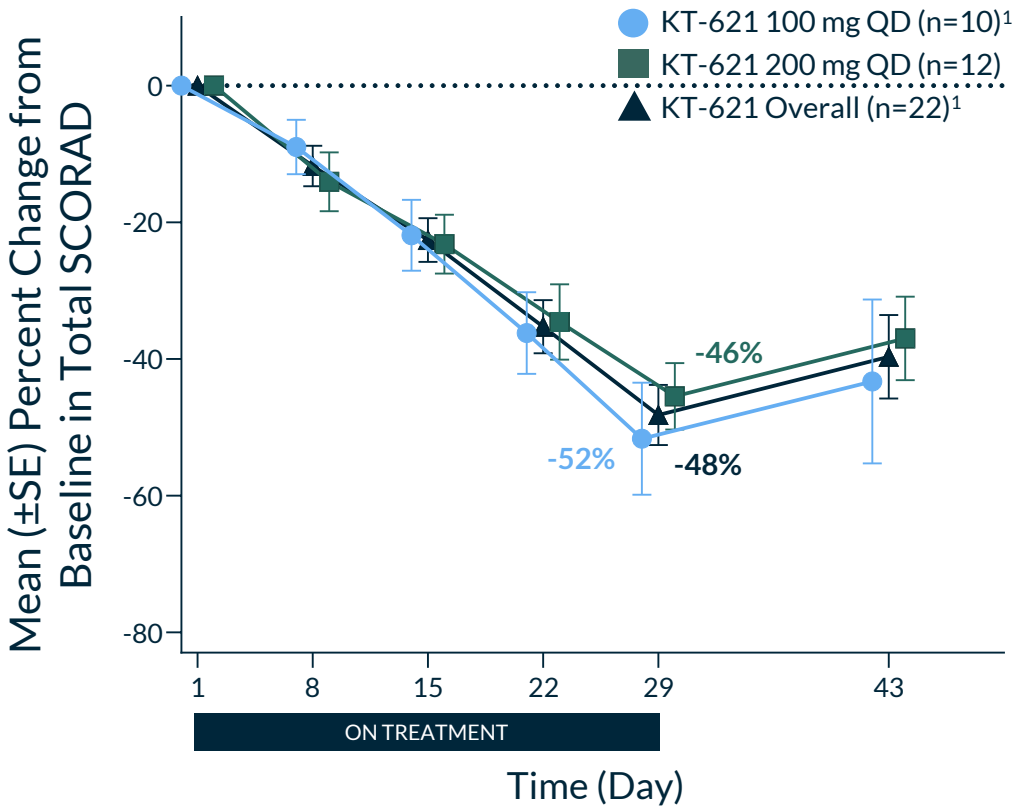
# KT-621 Achieved Rapid and Robust Reductions in EASI and SCORAD Across All Patients

## 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.

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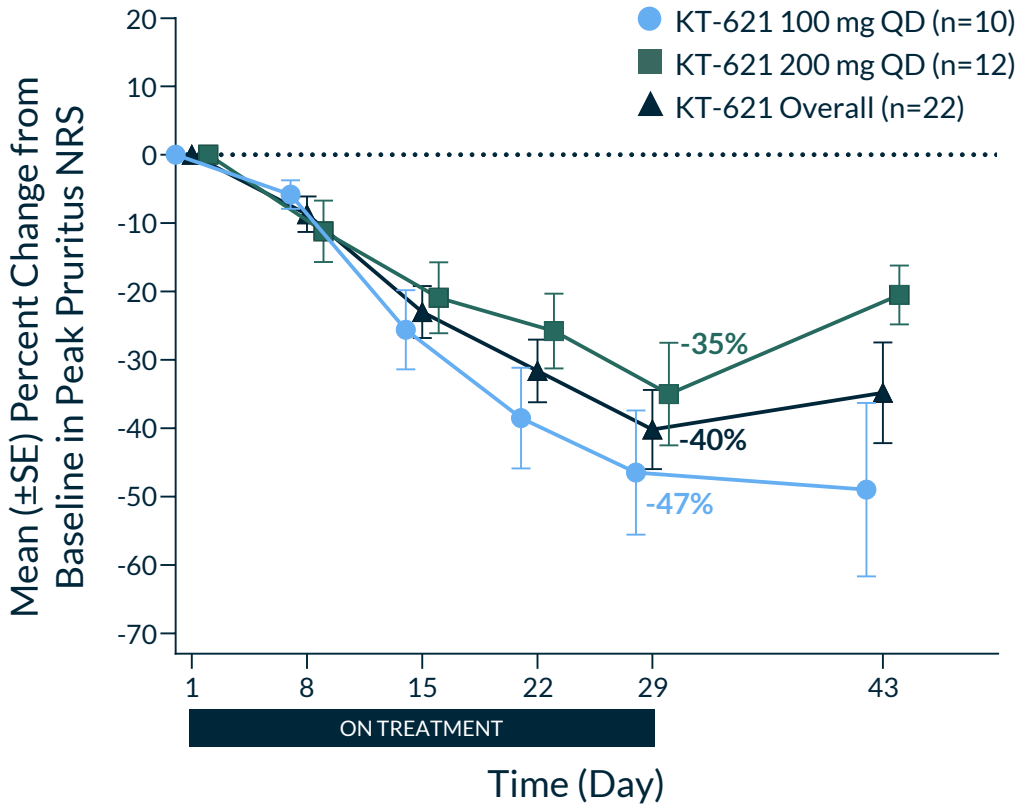
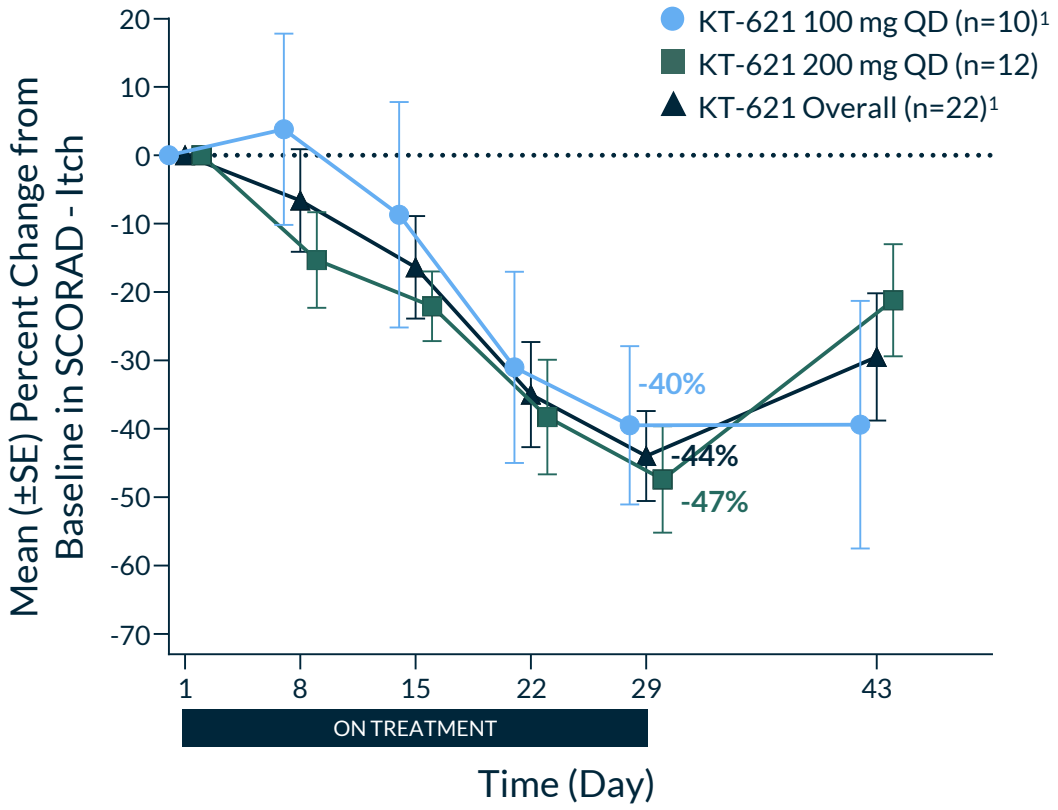
# KT-621 Achieved Robust and Consistent Reductions in Itch Across Independent Clinical Measures

## 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.

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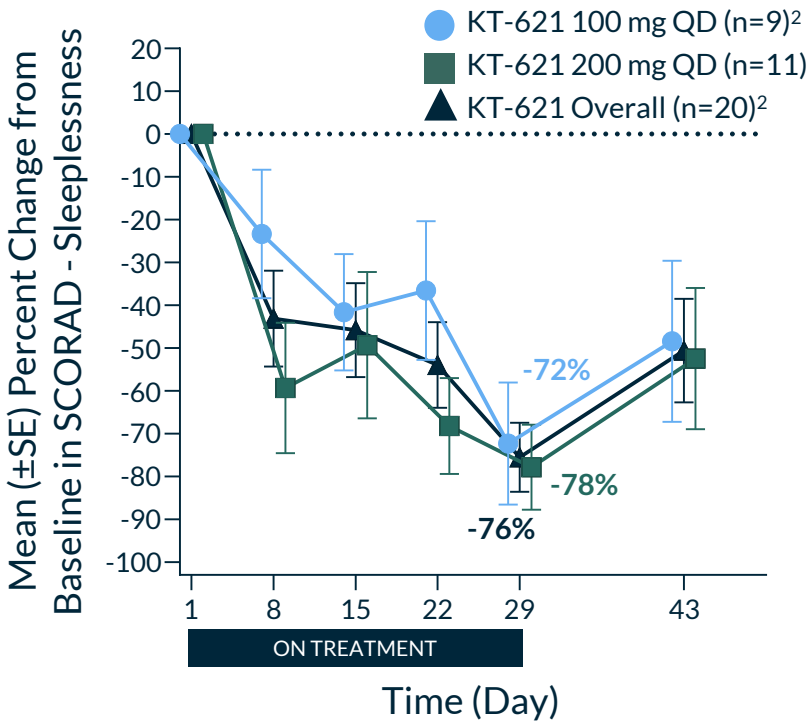
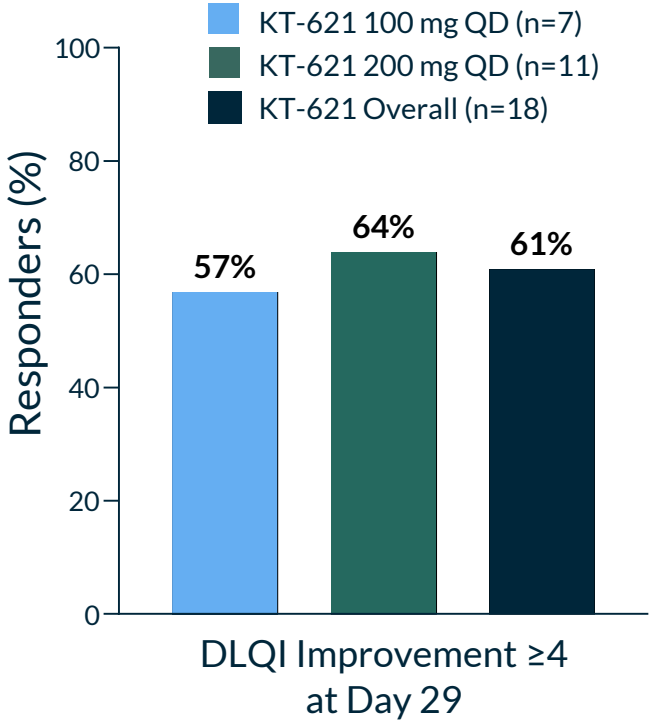
# KT-621 Achieved Robust Improvement in Patient Reported Outcomes and Quality of Life Measures

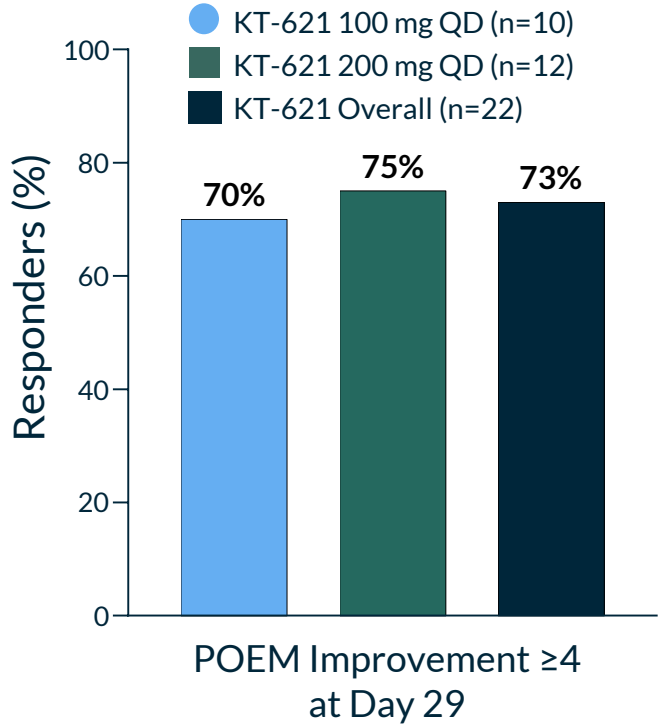
## Mean % Change in SCORAD - Sleeplessness[1]




## 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.

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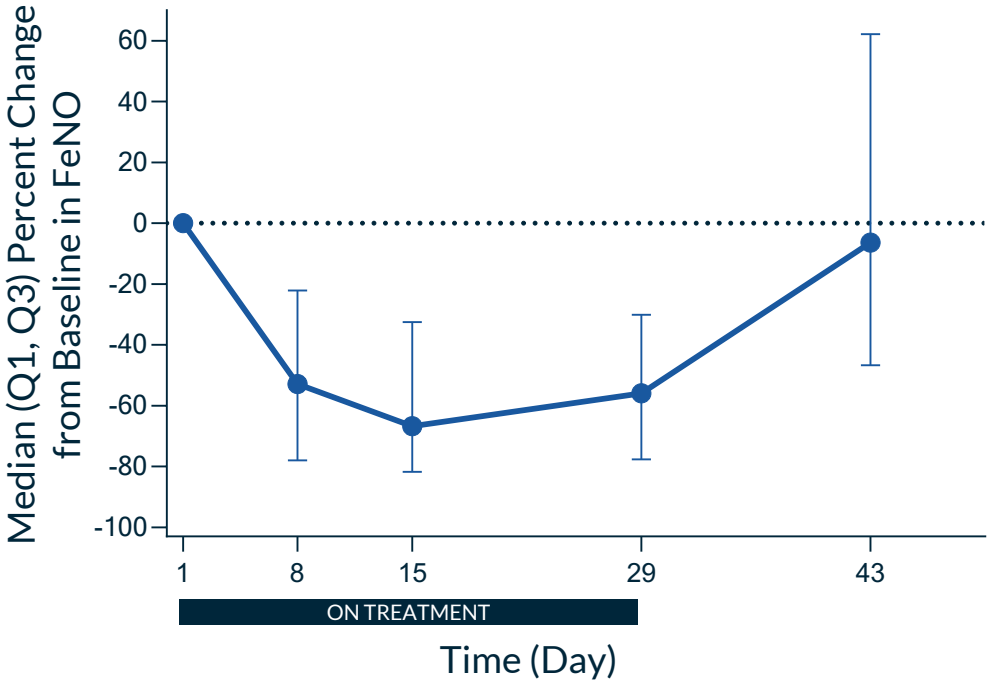
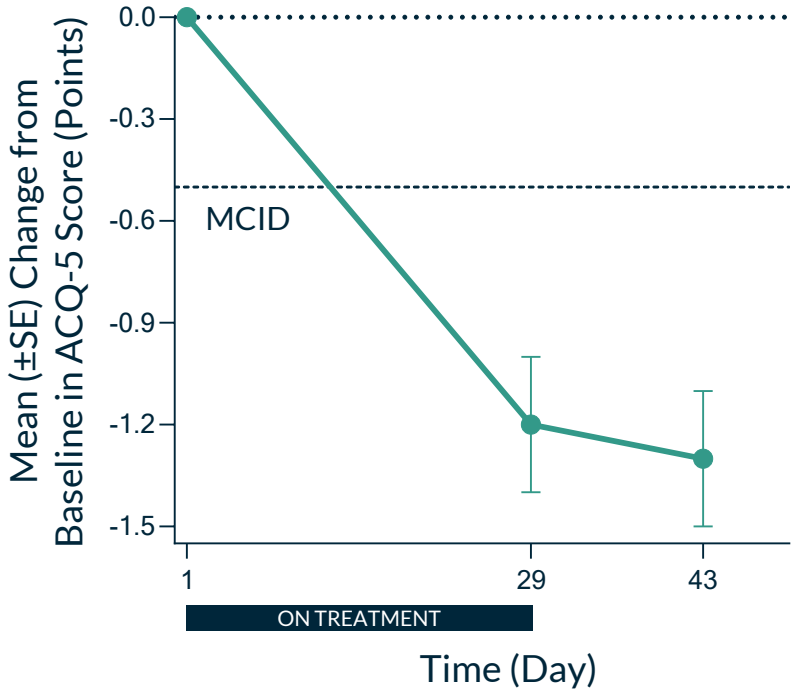
# KT-621 Achieved Robust Impact on FeNO and ACQ-5 in AD Patients with Comorbid Asthma

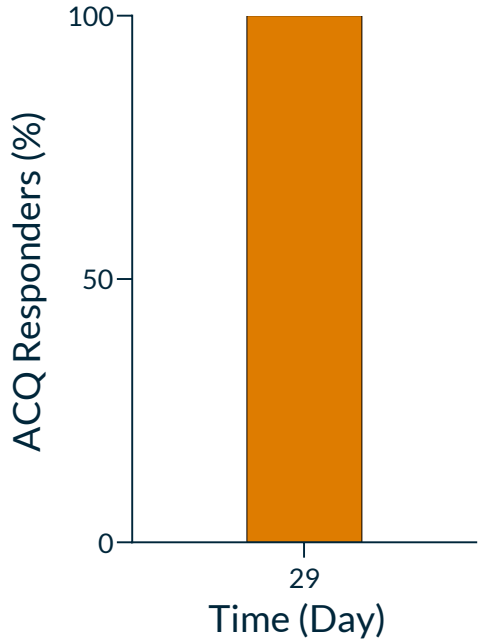
**Median % Change from Baseline in FeNO (n=4)**




**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.

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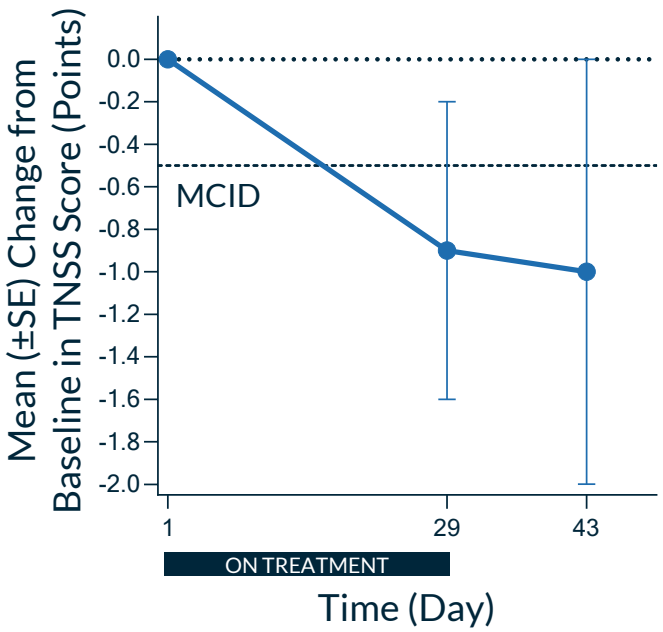
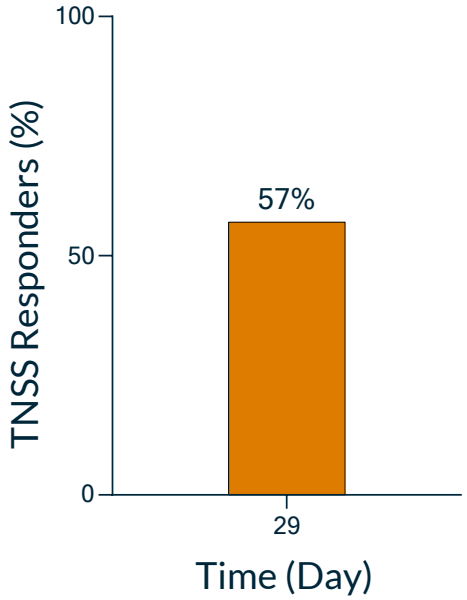
# KT-621 Achieved Robust Impact on TNSS and RQLQ in AD Patients with Comorbid Allergic Rhinitis

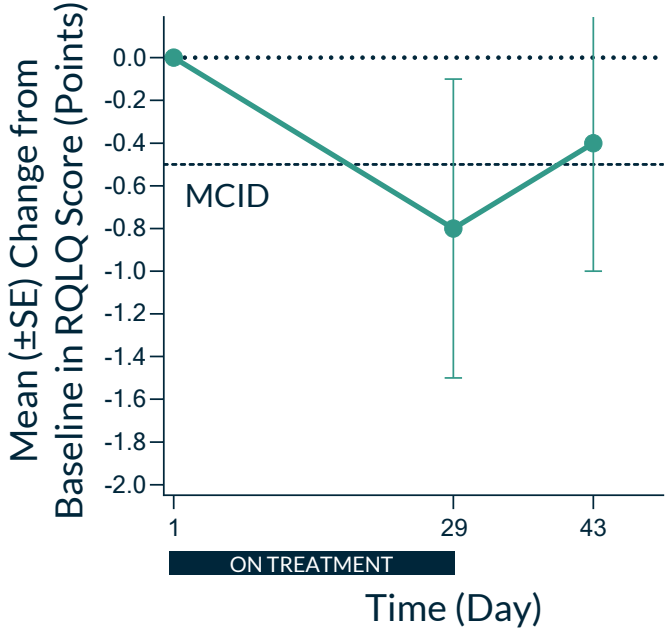
**Mean Point Change in TNSS (n=7)[1]**



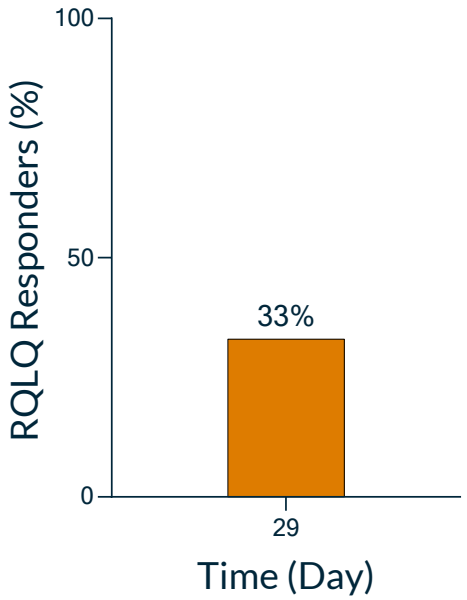

**TNSS Responders (Improvement ≥ 0.55, n=7)[1]**




**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.

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# 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.

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# KT-621 BroADDen 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.

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# 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:<br>- Type 2 biomarkers in blood and skin<br>- FeNO in AD and comorbid asthma patients<br>- Clinical endpoints in patients with AD, comorbid asthma and allergic rhinitis<br>**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

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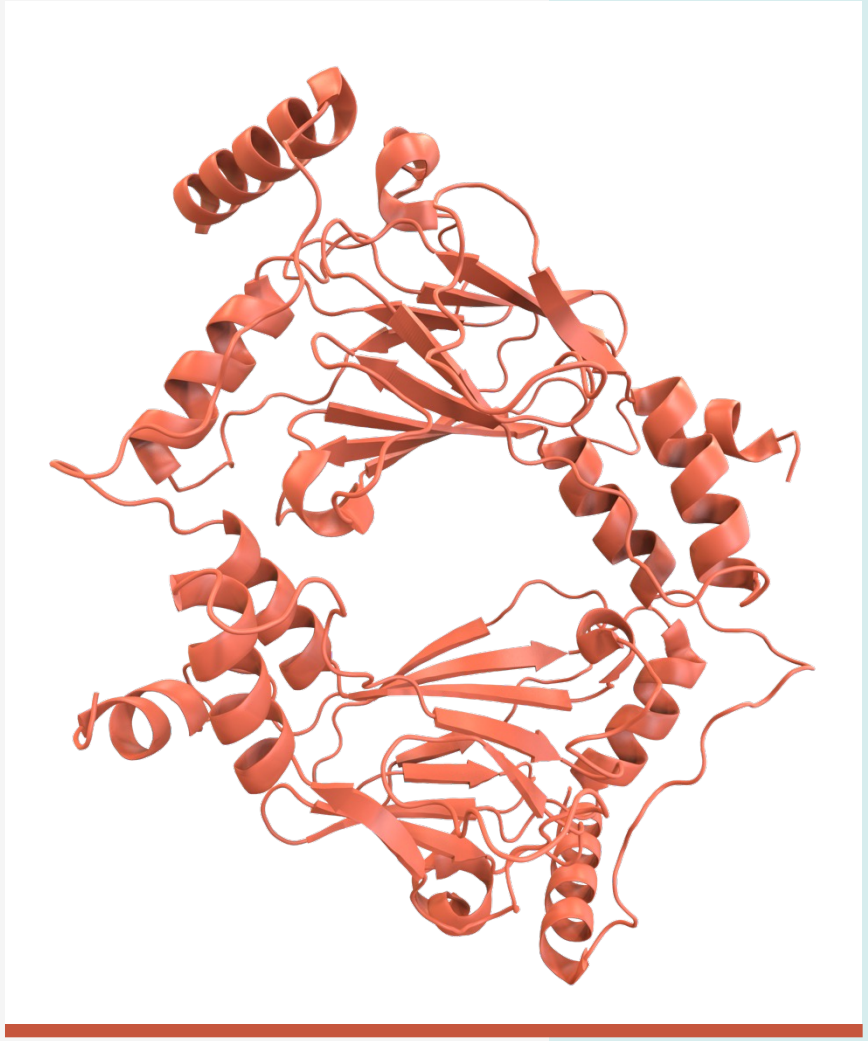

# First-in-Class Oral IRF5 Degrader Program

## Drugging a Genetically Validated Transcription Factor with an Oral Degrader

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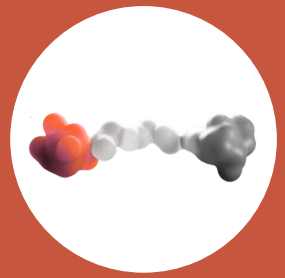
# KT-579 Overview

## 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

- LUMORA Phase 1b lupus trial start by early 2027

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

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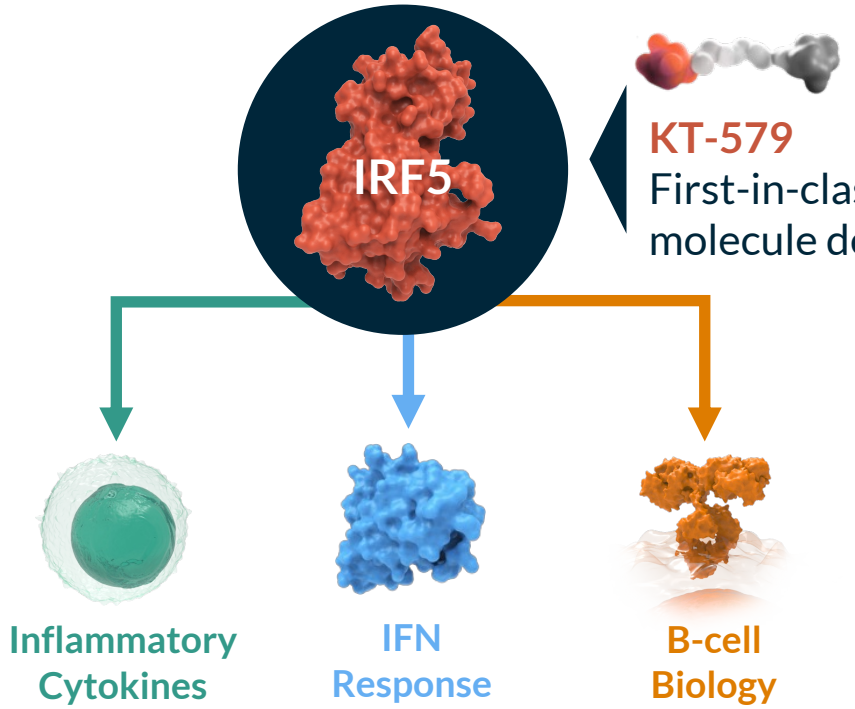
# KT-579: A New Treatment Paradigm for Serious Autoimmune Diseases

**IRF5 hyperactivation** impacts three validated disease-defining pathways




**Immune Complex Formation and Inflammation**
(Lupus, Sjögren's, RA, IBD, others)

## Historically Undrugged Target

IRF5 is a genetically validated transcription factor with impact on multiple clinically validated pathways

## Compelling Preclinical Profile

KT-579 demonstrated robust preclinical activity in primary human cells, patient donor cells and *in vivo* models of autoimmune diseases with a favorable safety profile

## Positive Early Clinical Data

In healthy volunteers, KT-579 showed strong clinical translation with deep degradation, robust reductions of inflammatory biomarkers, and a favorable safety profile

## Broad Patient Impact

Phase 1 data unlocks multiple clinical opportunities starting from upcoming lupus trial

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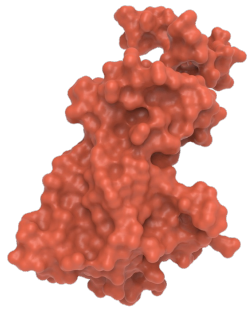
# Opportunity to Address a Wide Range of Serious Autoimmune Indications Through Clinically Validated Pathways

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

**Individual Pathways Clinically Validated by Biologics**

| | | Inflammatory Cytokines | IFN Response | B-cell Biology |
|---|---|---|---|---|
| **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



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# IRF5 Genetically Validated in Multiple Autoimmune Diseases

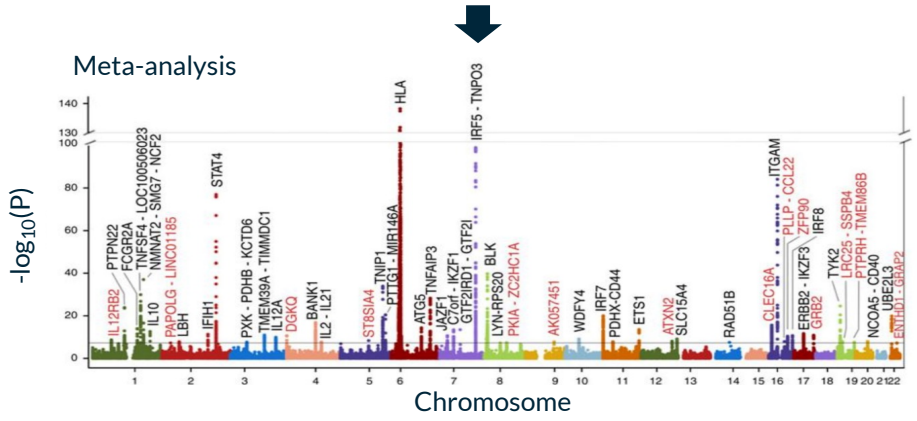
## Human Genetics

- 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

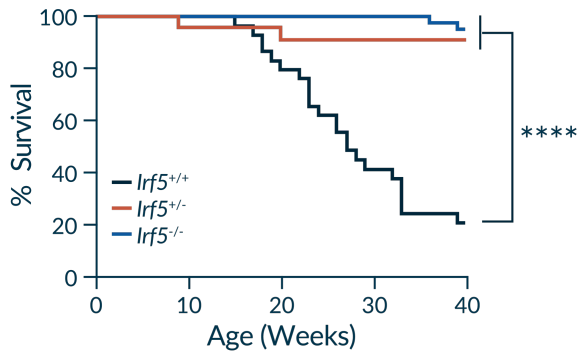
## Mouse IRF5 KO Studies

- 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]

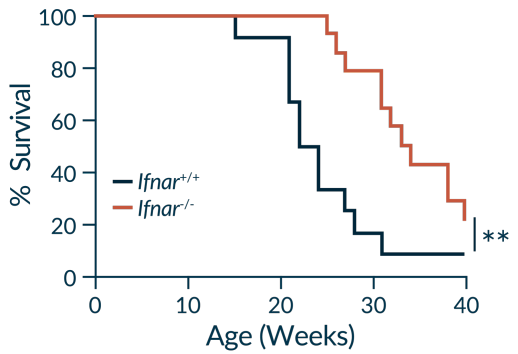
### Meta-analyses Identified IRF5 as a Risk Locus for SLE




### 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.; ****$p < 0.0001$; **$p = 0.0043$.

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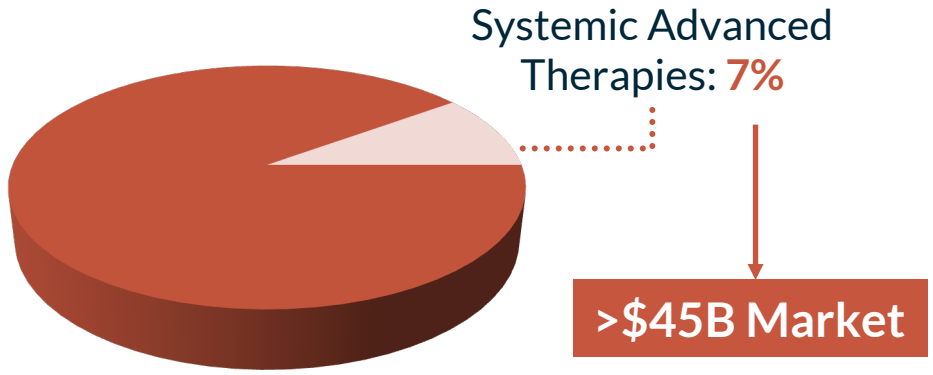
# IRF5: A Novel Mechanism in High Unmet Need Indications in I&I

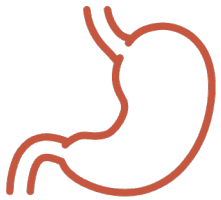
**>10M**




## 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.

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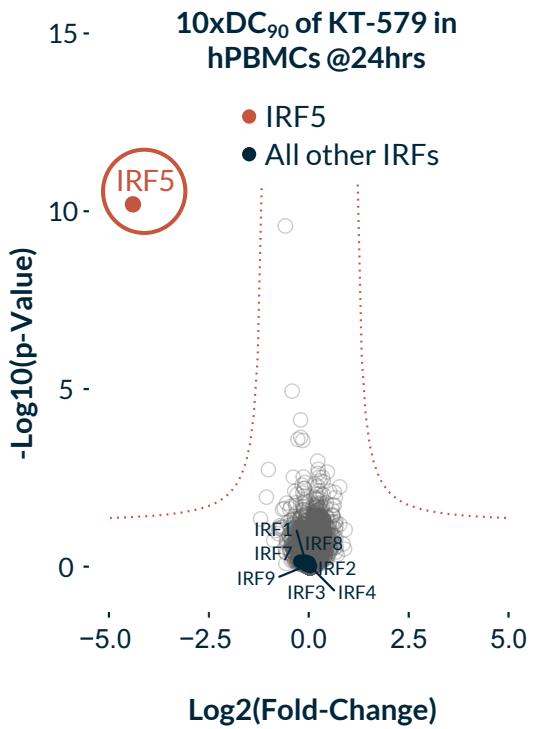
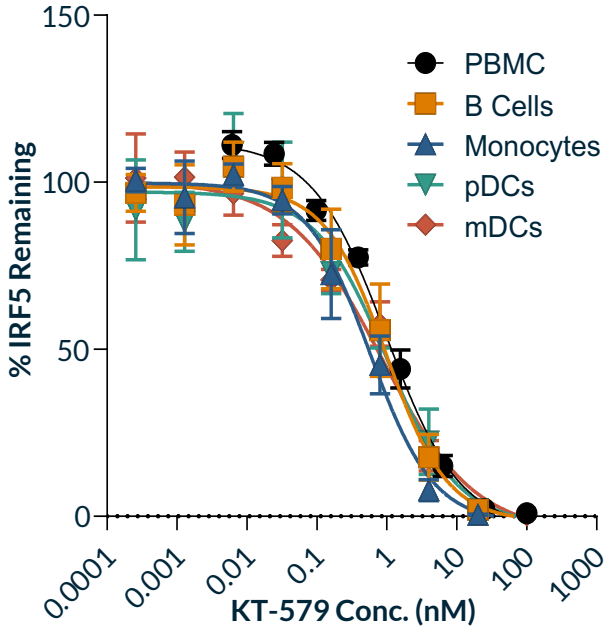
# KT-579: Compelling Preclinical Profile

## Novel oral mechanism to block multiple validated disease-defining pathways

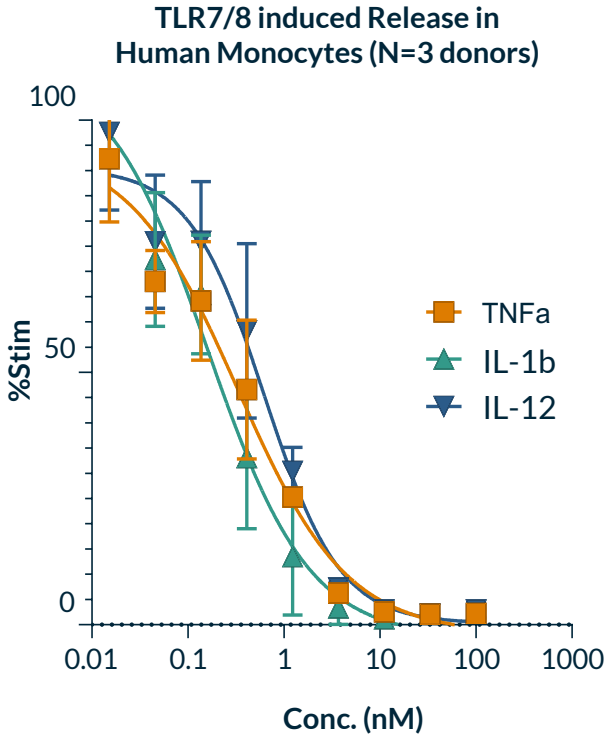
### Exquisite Selectivity



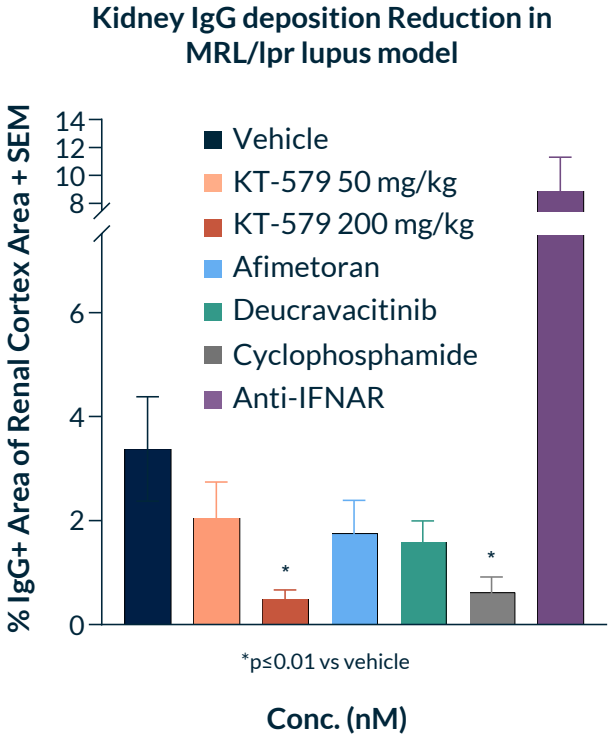

### Robust Degradation in Relevant Cell Types




### Full Inhibition of IRF5 Regulated Cytokines




### Excellent Preclinical Activity Superior to Targeted Agent Comparators




For more information on KT-579's preclinical profile, visit Kymera's website.

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# KT-579 Phase 1 Healthy Volunteer Clinical Trial Data

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# 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)**

Total 48 Healthy Volunteers
8 per cohort
(6:2 KT-579:Placebo)

### PART B
**Multiple Ascending Dose (MAD)**

14x daily doses

Total 72 Healthy Volunteers
12 per cohort
(9:3 KT-579:Placebo)

### Dosing
- Evaluated a range up to 600 mg with multiple daily doses for up to 14 days

### Primary
- Safety & tolerability

### Secondary
- Pharmacokinetic measures

### Exploratory
- IRF5 protein levels in blood
- *Ex vivo* blood biomarkers and pathway gene transcripts:
  - Inflammatory cytokines
  - IFN response
  - B-cell biology

### Key Trial Aim

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

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# Phase 1 SAD/MAD Demographics

| | SAD 1-6: PBO (n=12) | SAD 1-6: KT-579 (n=36) | MAD 1-6: PBO (n=18) | MAD 1-6: KT-579 (n=54) |
|---|---|---|---|---|
| **Gender, n (%)** | | | | |
| Female | 3 (25.0%) | 10 (27.8%) | 7 (38.9%) | 7 (13.0%) |
| Male | 9 (75.0%) | 26 (72.2%) | 11 (61.1%) | 47 (87.0%) |
| **Age, years, median (range)** | 38.0 (19, 53) | 37.5 (22, 55) | 38 (21, 51) | 35.5 (20, 54) |
| **Ethnicity, n (%)** | | | | |
| Hispanic or Latino | 3 (25.0%) | 7 (19.4%) | 4 (22.2%) | 18 (33.3%) |
| Non-Hispanic or Latino | 9 (75.0%) | 29 (80.6%) | 14 (77.8%) | 36 (66.7%) |
| **Race, n (%)** | | | | |
| White | 9 (75.0%) | 25 (69.4%) | 15 (83.3%) | 35 (64.8%) |
| Black or African American | 1 (8.3%) | 8 (22.2%) | 1 (5.6%) | 15 (27.8%) |
| Asian | 1 (8.3%) | 1 (2.8%) | 1 (5.6%) | 2 (3.7%) |
| Multiple | 1 (8.3%) | 2 (5.6%) | 1 (5.6%) | 2 (3.7%) |

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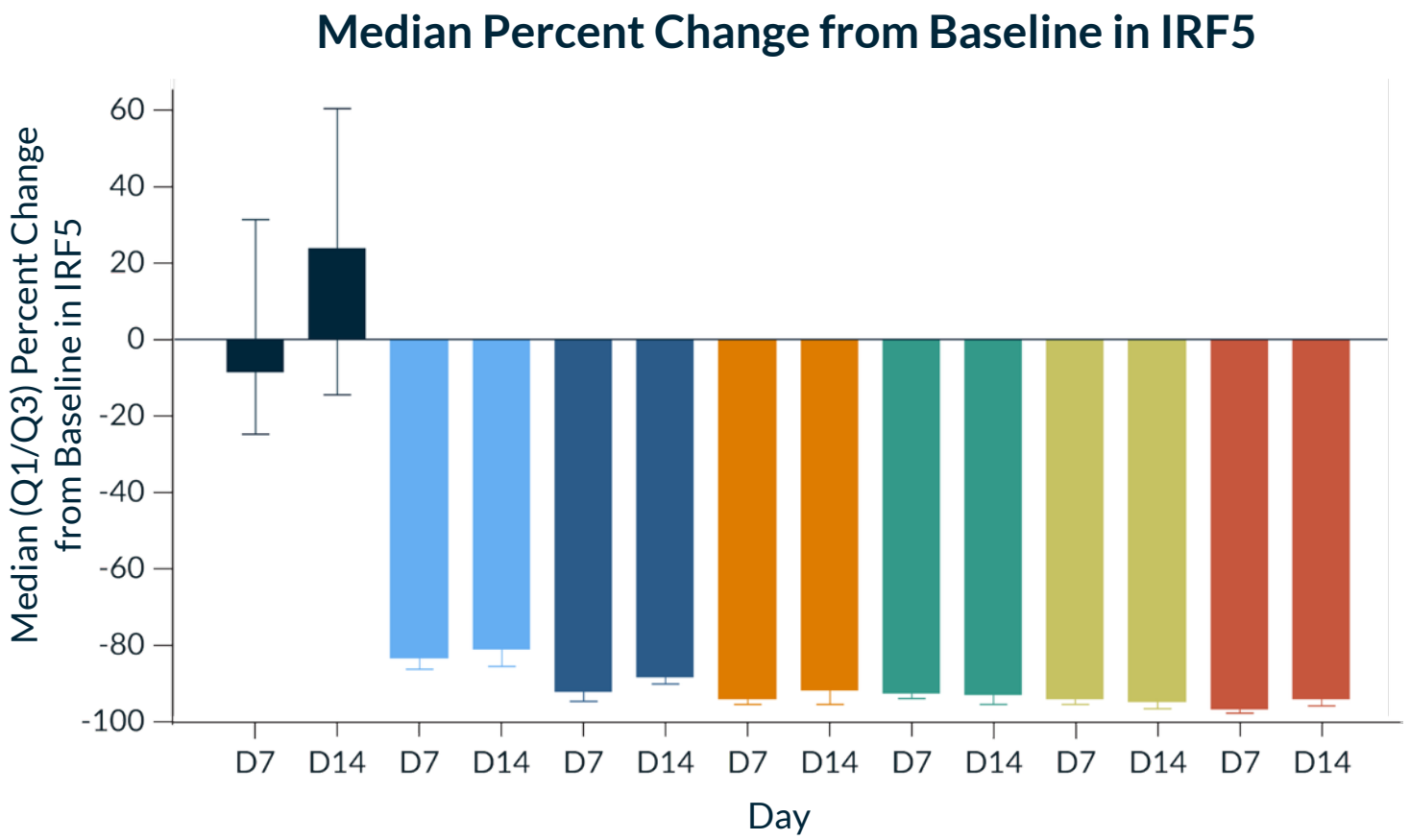
# Multiple Daily Doses of KT-579 Achieved >90% IRF5 Degradation in Blood Across Almost All Dose Levels




| | Median % IRF5 Change | n | D7 | D14 |
|---|---|---|---|---|
| | Placebo | 18 | -9% | 24% |
| | **Dose 1** | **9** | **-83%** | **-81%** |
| | **Dose 2** | **9** | **-92%** | **-89%** |
| | **Dose 3** | **9** | **-94%** | **-92%** |
| | **Dose 4** | **9** | **-93%** | **-93%** |
| | **Dose 5** | **9** | **-94%** | **-95%** |
| | **Dose 6** | **9** | **-97%** | **-94%** |

- KT-579 dosed daily for 14 days showed dose dependent exposure and degradation
- Degradation at every dose by Day 14 was statistically significant ($p \leq 0.0001$ for all comparisons of KT-579 vs. Placebo)

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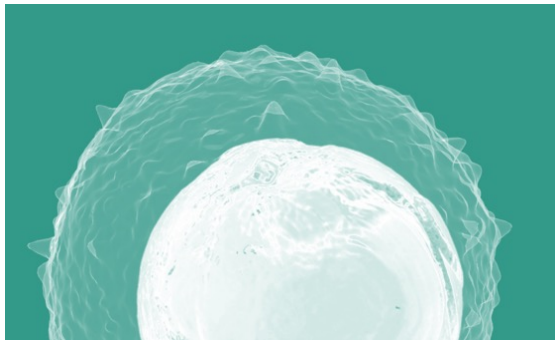
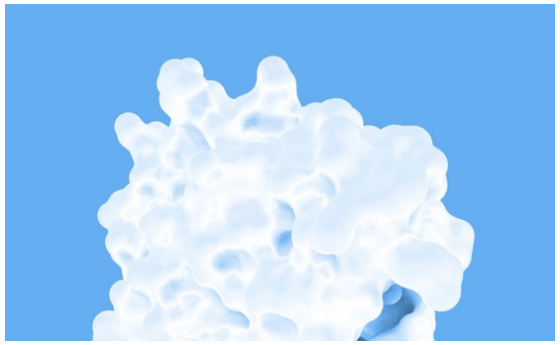
# Complementary Ex Vivo Biomarker Assays Capture the Breadth of IRF5 Biology Across Three Key Immune Pathways[1]

## INFLAMMATORY CYTOKINES[2]



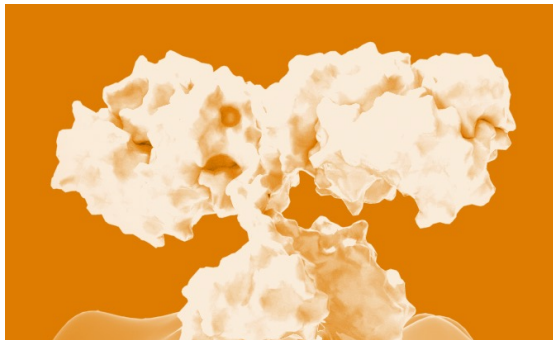
- IRF5 promotes production of key inflammatory cytokines in myeloid cells
- When dysregulated, these responses can sustain chronic inflammation, relevant to several autoimmune diseases

## INTERFERON RESPONSE[3]



- IRF5 connects nucleic-acid sensing with broader interferon-associated transcriptional responses
- Interferon dysregulation is a central feature of several autoimmune diseases, where it amplifies immune activation and contributes to inflammation

## B-CELL BIOLOGY[4]



- IRF5 contributes to immune responses linked to B-cell activation and autoantibody-related biology
- This axis is particularly relevant in several autoantibody driven autoimmune diseases

[1]Wang et al., *Signal Transduction and Targeted Therapy*, 2024; [2]Krausgruber et al., *Nature Immunology*, 2011; [3]Barnes et al., *J Biol Chem*, 2001; [4]De et al., *Frontiers in Immunology*, 2018.

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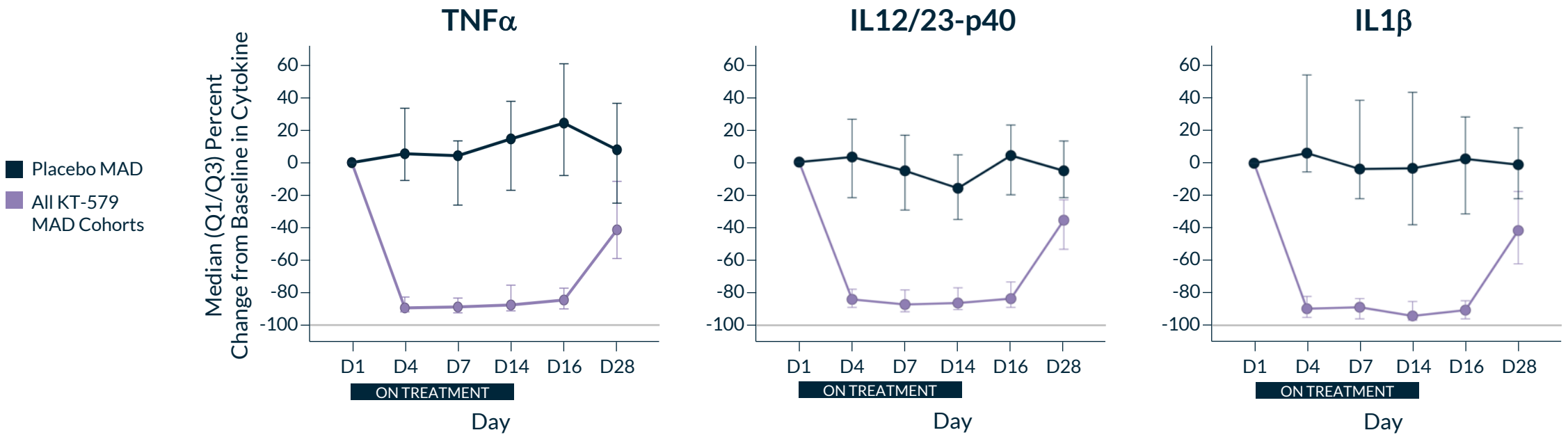
# Multiple Daily Doses of KT-579 Achieved Deep Inhibition of Clinically Validated Inflammatory Cytokines




**Median Cytokine Inhibition Day 14 - R848 (TLR7/8)** Ex Vivo Stimulation

| Median Inhibition Day 14[1] | TNFα | IL12/23-p40 | IL1β |
|---|---|---|---|
| Placebo | 15% | -16% | -3% |
| **All KT-579 MAD Cohorts** | **-88%** | **-87%** | **-94%** |

[1]Placebo, n=18, and all KT-579 MAD cohorts, n=52, two subjects were excluded due to a processing error.

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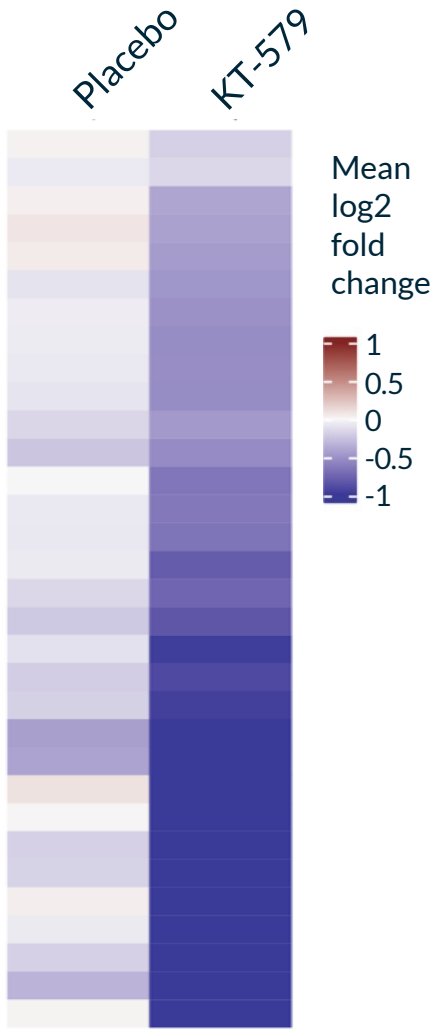
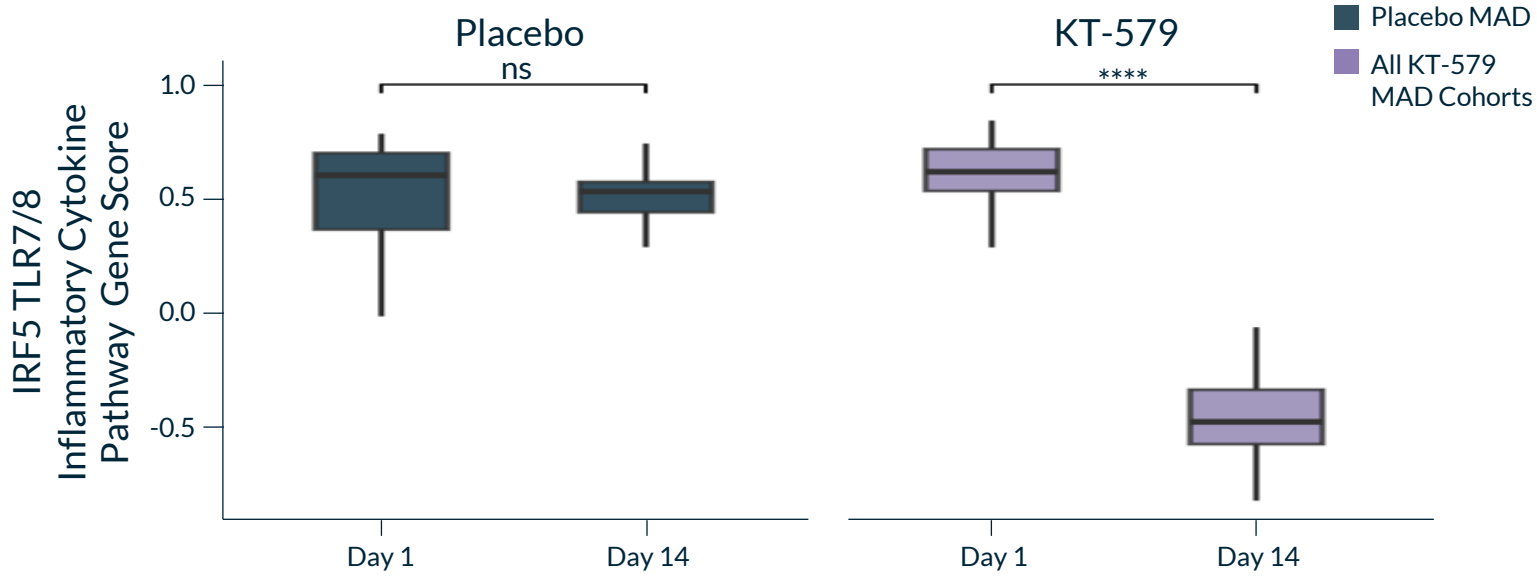
# KT-579 Downregulates Inflammatory Cytokine Pathway Genes




## Genes Significantly Decreased Across MAD Cohorts[1]

Expression at Day 14 compared to pre-dose

### Inflammatory Cytokine Gene Score




- Significant reductions in multiple genes, selected from a core IRF5 mediated TLR7/8 signature focused on inflammatory pathways
- Strong concordance between KT-579 effects on gene and protein reductions in inflammatory cytokines such as IL12β and TNF

[1]Genes from pre-defined gene sets from preclinical KT-579 data across ex vivo stimulations; Names of genes have been removed; Significance defined as downregulated ≥ 1.5x with adj. P value ≤ 0.05; All KT-579 MAD cohorts n=52, Placebo n=18; Signature scores were calculated using GSVA; Paired t-test on GSVA scores; P-values are shown as *** for p < 0.001, **** for p < 0.0001.

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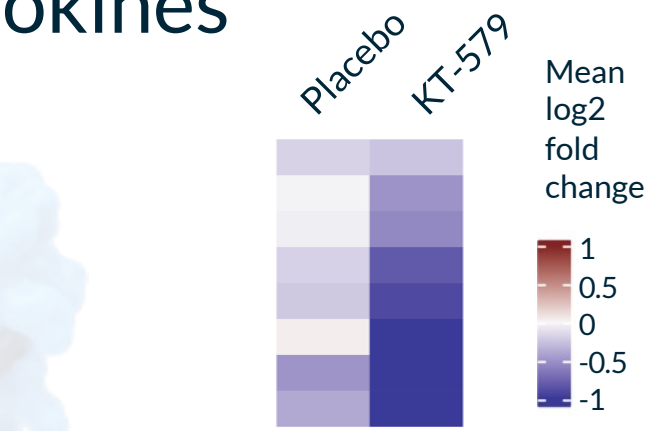
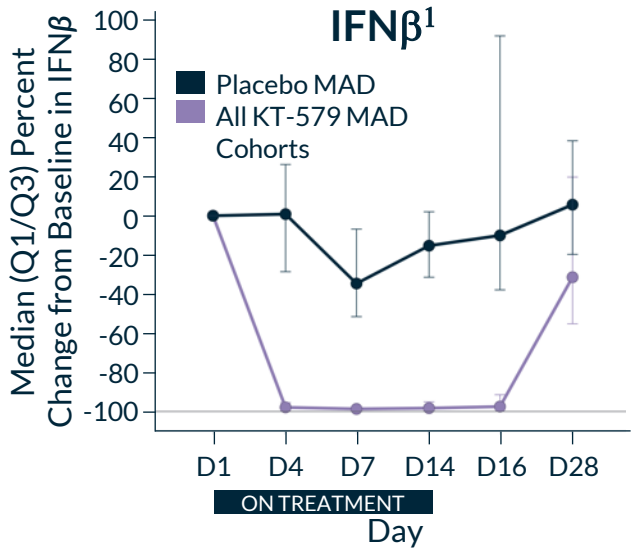
# KT-579 Downregulates Interferon Pathway Genes and Cytokines





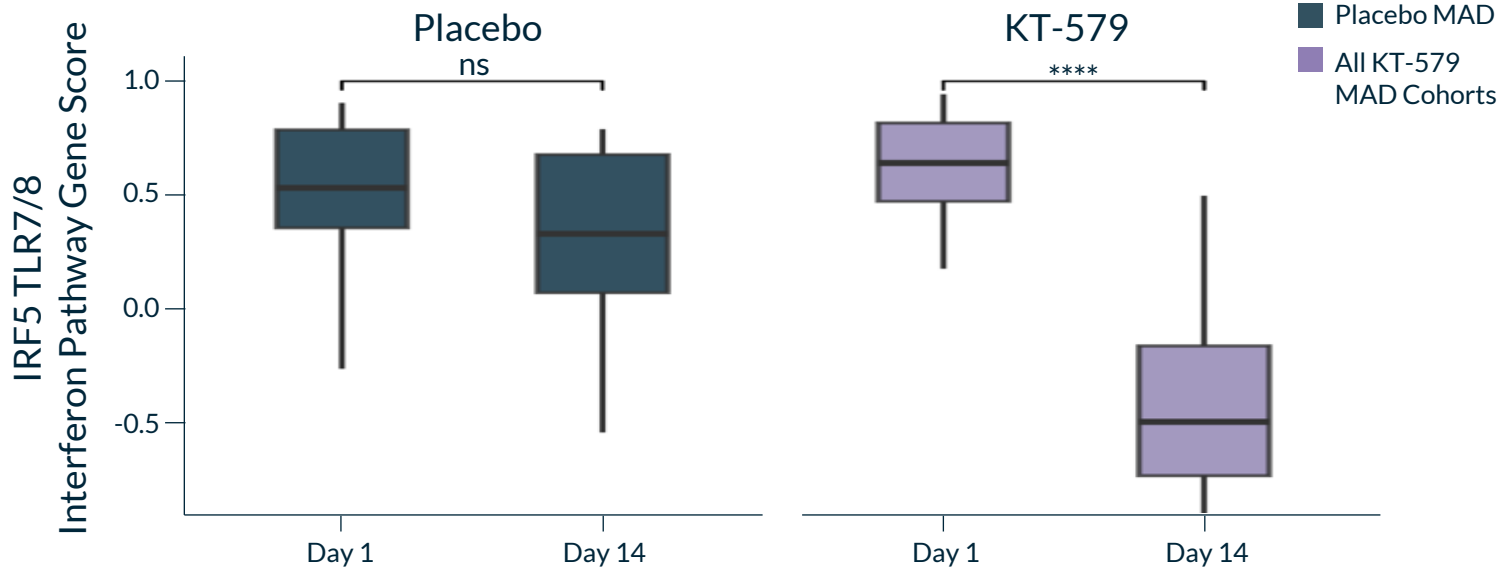




| Median Inhibition Day 14[1] | IFNβ |
|---|---|
| Placebo | -15% |
| **All KT-579 MAD Cohorts** | **-98%** |

## Genes Significantly Decreased Across MAD Cohorts[2]

Expression at Day 14 compared to Pre-dose

### Interferon Gene Score




- Significant reductions in multiple genes selected from a core IRF5 mediated TLR7/8 signature focused on the interferon pathway

[1]All KT-579 MAD cohorts, n=54, Placebo, n=18; [2]Genes from pre-defined gene sets from preclinical KT-579 data across ex vivo stimulations; Names of genes have been removed; Significance defined as downregulated ≥ 1.5x with adj. P value ≤ 0.05; All KT-579 MAD cohorts n=52, Placebo n=18; Signature scores were calculated using GSVA; Paired t-test on GSVA scores; P-values are shown as *** for p < 0.001, **** for p < 0.0001.

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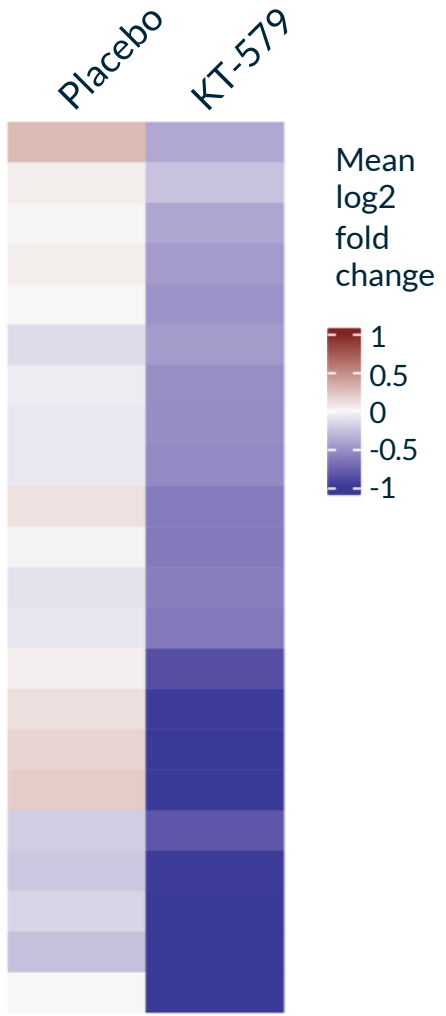
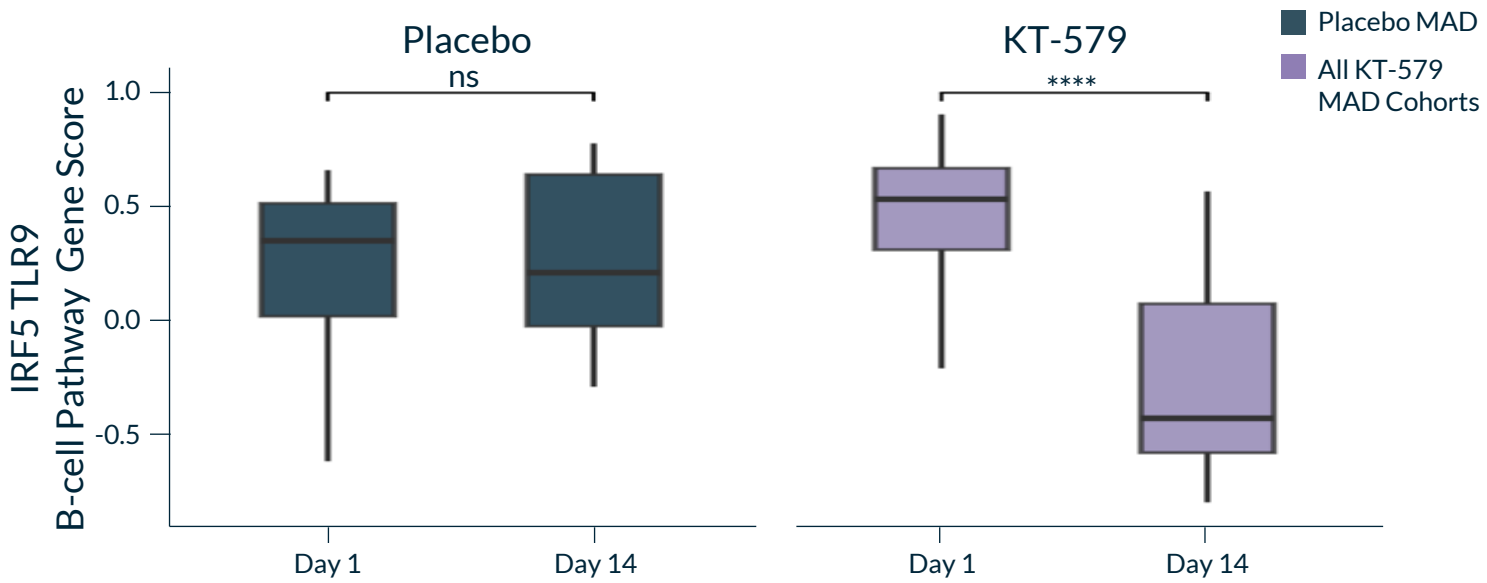
# KT-579 Downregulates B-cell Pathway Genes




## Genes Significantly Decreased Across MAD Cohorts[1]

Expression at Day 14 compared to Pre-dose




- Significant reductions in multiple genes selected from a core IRF5 mediated TLR9 signature focused on DNA-sensing and B-cell activation pathways

[1]Genes from pre-defined gene sets from preclinical KT-579 data across ex vivo stimulations; Names of genes have been removed; Significance defined as downregulated ≥ 1.5x with adj. P value ≤ 0.05; All KT-579 MAD cohorts n=52, Placebo n=18; Signature scores were calculated using GSVA; Paired t-test on GSVA scores; P-values are shown as *** for p < 0.001, **** for p < 0.0001.

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# Healthy Volunteer SAD/MAD Safety Summary

## KT-579 Well Tolerated Across All Doses Evaluated with Safety Profile Comparable to Placebo

- No SAEs
- No Severe AEs
- No AEs leading to study drug discontinuation
- No dose-dependent pattern in the TEAEs
- No imbalances in the rates of infections
- No clinically relevant changes in vital signs, laboratory tests, or ECGs

**TRAEs that Occurred in >1 Participant (MAD/SAD* Cohort)**

| AE Term (*severity*) | MAD 1-6 Placebo (n=18) | MAD 1-6 KT-579 (n=54) |
|---|---|---|
| Headache (*mild*) | 4 (22.2%) | 3 (5.6%) |
| Myalgia (*mild*) | 0 (0.0%) | 2 (3.7%) |
| Constipation (*mild*) | 2 (11.1%) | 1 (1.9%) |

*No TRAEs occurred in more than one participant in the SAD cohorts

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

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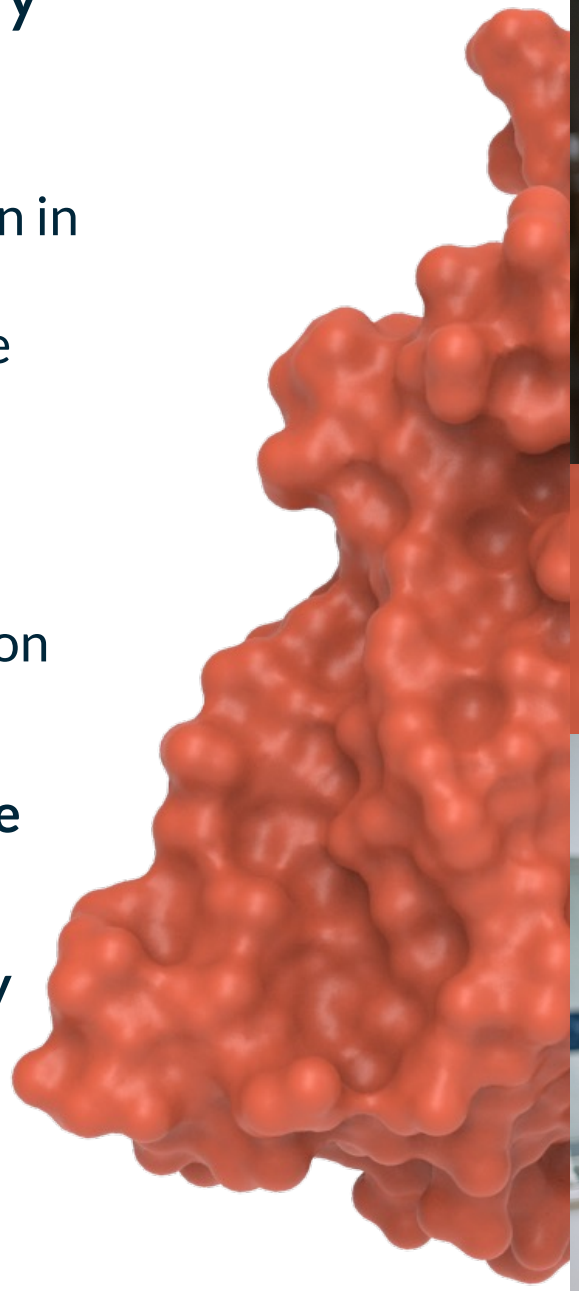
# KT-579 Phase 1 Summary

- Deep, sustained IRF5 degradation in blood, with **≥90% median IRF5 degradation** across multiple dose levels in MAD
- Robust **suppression of disease-relevant blood biomarkers** for inflammatory cytokines, interferon response, and B-cell biology
- Downregulation of **pathway gene transcripts**
- **Favorable safety and tolerability profile in healthy volunteers**



A Novel Oral Mechanism for Serious Autoimmune Diseases

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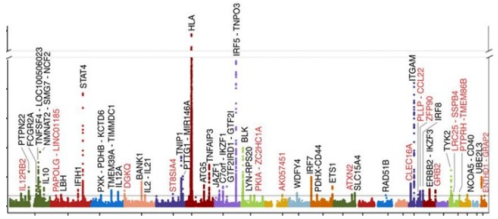
# KT-579: First-in-Industry Opportunity in Lupus

## Novel Oral Mechanism to Address Underlying Biology for High-Burden Disease

### GENETICS/PRECLINICAL VALIDATION

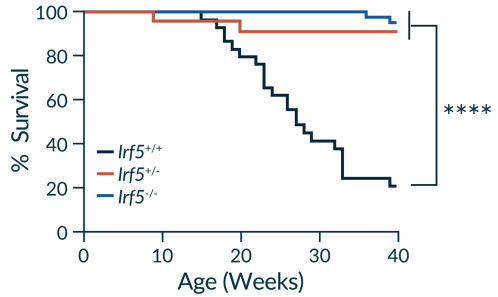
**Meta Analysis**

Meta-analyses identified IRF5 as a risk locus for lupus[1]



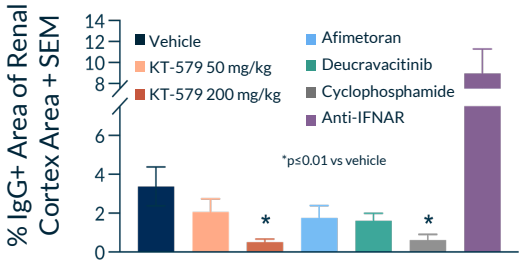
**Survival**

IRF5 KO increases survival in mice and has essential role in lupus[2]




**Disease-modifying Activity**

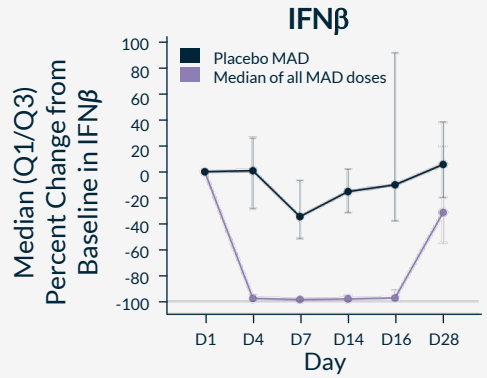
Activity comparable or superior to existing agents in preclinical lupus models




### CLINICAL VALIDATION

**KT-579 Phase 1 Data**

Modulation of key pathway biology in Phase 1 HV trial




**Pathway Validation**

Pathways clinically validated in lupus (mAbs/depletors)

Saphnelo® · Benlysta® (belimumab) · GAZYVA® (obinutuzumab)

[1]Santana-de Anda et al., *Autoimmunity Reviews*, 2011; [2]Richez et al., *J Immunol*, 2010; All trademarks and registered trademarks are the property of their respective owners.

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# KT-579: LUMORA Phase 1b Trial

## Biomarker Study for First-in-Class Mechanism in Lupus Patients

lumora TRIAL

**Adult Patients with Systemic Lupus Erythematosus**

**Baseline entry criteria:**

- ANA ≥1:80
- BILAG-2004 score A or B[1]
- Oral corticosteroid background therapy permitted

### Design

- Randomized, double-blind, placebo-controlled
- ~30 patients

### Dosing

- KT-579 or placebo (2:1)
- Dosing for 16-weeks

### Endpoints

- Safety and tolerability
- Pharmacokinetics
- IRF5 protein levels in blood
- Biomarkers
  - Inflammatory cytokines
  - IFN response
  - B-cell biology
- Exploratory disease endpoints

### Key Trial Aim

Biomarker study to demonstrate that **KT-579 can modulate underlying disease biology** with a favorable safety and tolerability profile

Status update:
**Trial start by early 2027**

[1]In mucocutaneous and/or musculoskeletal domains.

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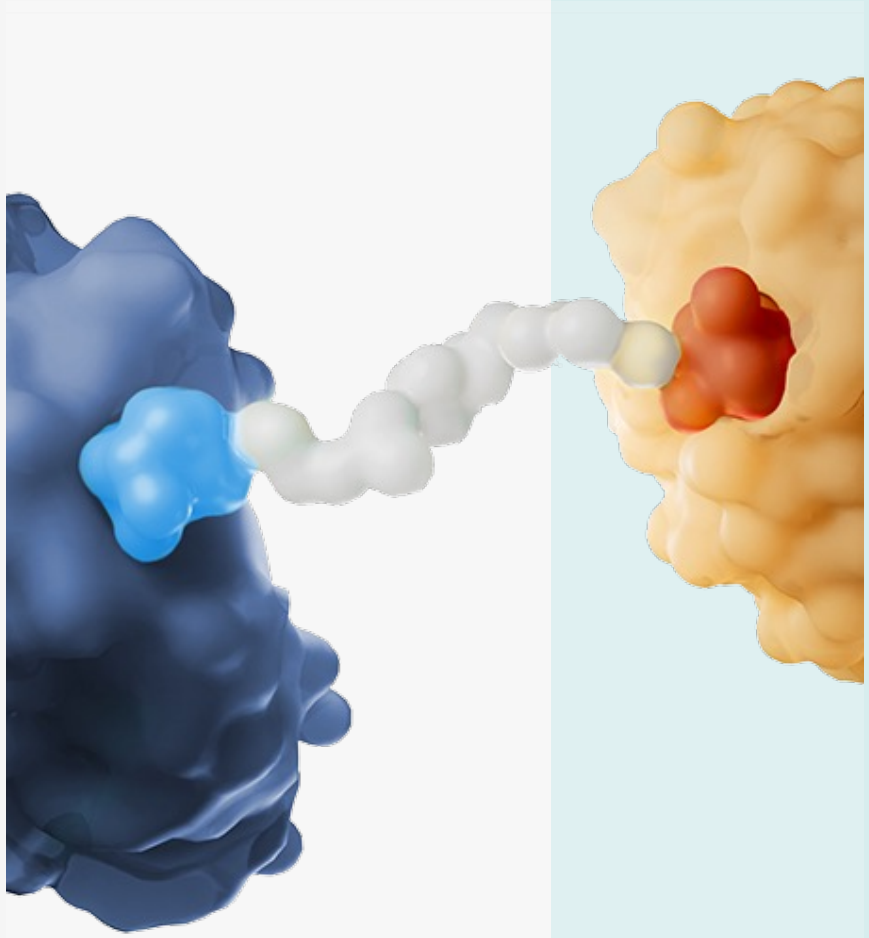

# Partnered Oral Degrader Programs

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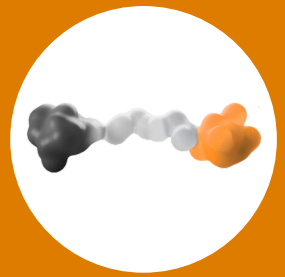
# IRAK4 Overview

## 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

### 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 & UPCOMING MILESTONES

- Partnered with Sanofi
- KT-485 Phase 1 SAD/MAD study in healthy volunteers and hidradenitis suppurativa patients initiated

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

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# KT-485 (SAR447971): Phase 1 First-in-Human Trial

## SAD and MAD in Healthy Volunteers and Hidradenitis Suppurativa Patients

### Three Part Phase 1 Study Design

**SAD**
Healthy Volunteers

**MAD**
Healthy Volunteers

**MAD**
HS Patients

Double-blind, randomized, placebo controlled, sequential single and multiple ascending dose studies

Open-label, sequential multiple ascending dose study

**Primary**

- Safety & tolerability of escalating single and multiple doses of KT-485

**Secondary**

- Pharmacokinetic measures
- IRAK4 protein levels in skin (MAD patient cohort)

More information on the KT-485 Phase 1 trial is available on clinicaltrials.gov (NCT07629336).

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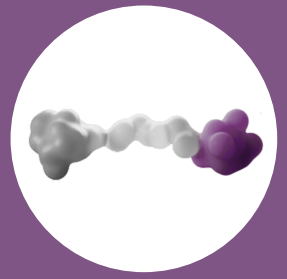
# CDK2 Overview

## Novel Oral Molecular Glue (MG) Degrader



**KT-200** is a first-in-class, oral CDK2 molecular glue degrader

- CDK2 is a master regulator of the cell cycle with strong genetic/ clinical validation and broad oncology treatment potential
- Unlike inhibitors, CDK2 molecular glue degraders selectively degrade CDK2 while sparing closely related kinases like CDK1
- In preclinical testing, KT-200 demonstrated low-nanomolar degradation of CDK2, robust activity in CCNE1 amplified, overexpressed cell lines and in vivo tumor models, brain penetrant potential, and a favorable safety profile

### 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]

.....►

**Potential to provide more precise, safe, effective, oral treatments for breast cancer and other solid tumors**

### STATUS & UPCOMING MILESTONES

- Partnered with Gilead to accelerate development and commercialization, with up to $750 million in total payments, including $85 million in upfront and option exercise payments
- Gilead exercised option to license KT-200, and will progress the program into IND-enabling studies to support an IND filing in 2027

[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.

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# Catalysts for Growth

## KT-621
**First-in-Class Oral STAT6 Degrader**

- Report BROADEN2 Phase 2b AD data by year-end 2026
- Initiate Phase 3 trials in AD by mid-2027
- Advance BREADTH Phase 2b asthma study; report data in late-2027

## KT-579
**First-in-Class Oral IRF5 Degrader**

- Start LUMORA Phase 1b lupus trial by early 2027

## Partners & Pipeline

- Sanofi advancing KT-485, oral IRAK4 degrader, in Phase 1 clinical testing in healthy volunteers and HS patients
- Gilead to advance KT-200, oral CDK2 molecular glue degrader, to support an IND filing in 2027
- Advance an early pipeline of novel programs with a goal to deliver at least one new DC per year

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# Thank You

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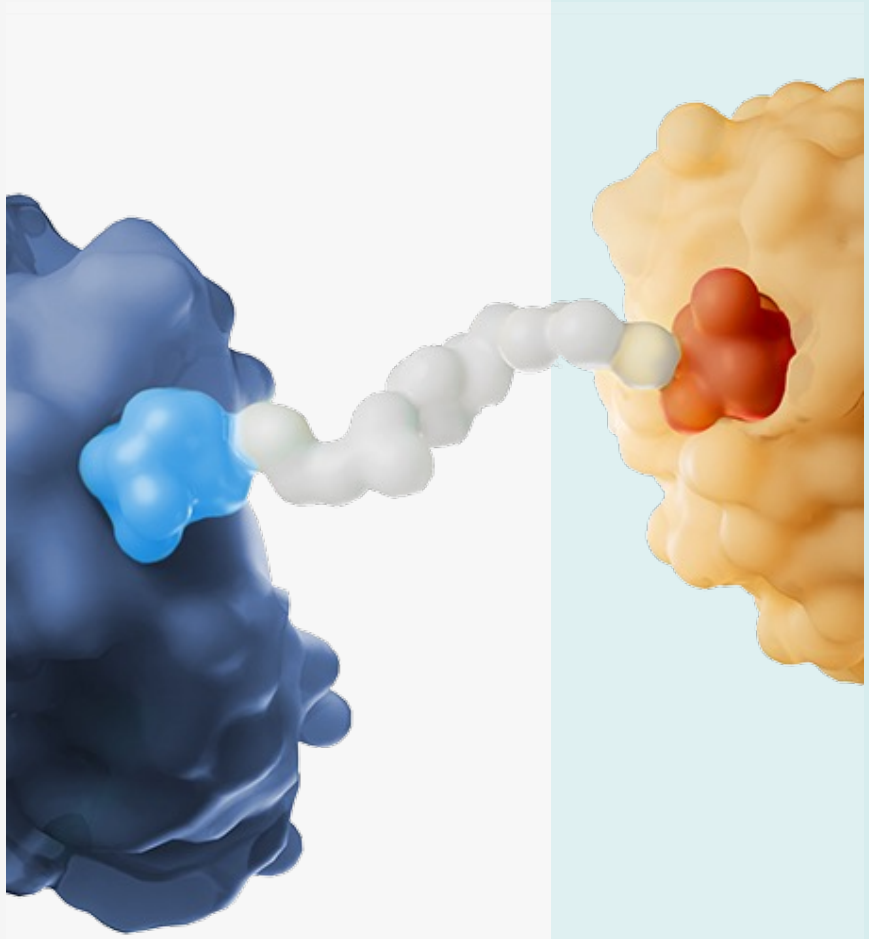

# Appendix: First-in-Class Oral STAT6 Degrader Program

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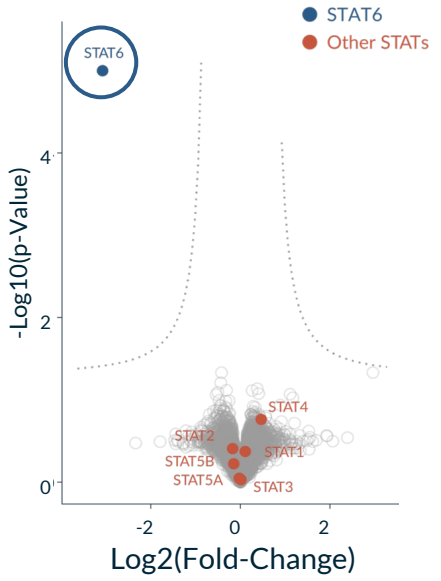
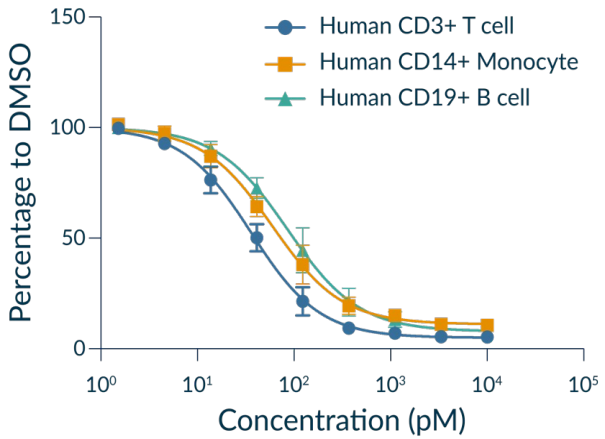
# KT-621: Compelling Preclinical Profile

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

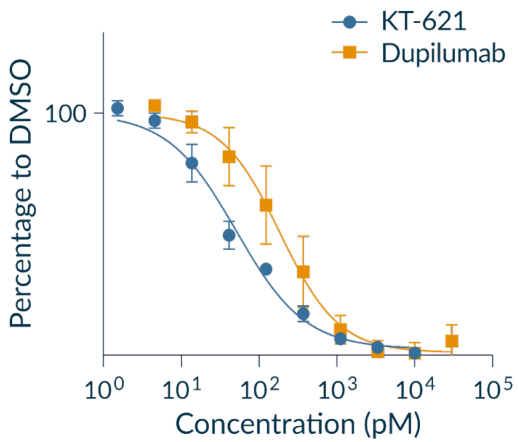
### Exquisite Selectivity



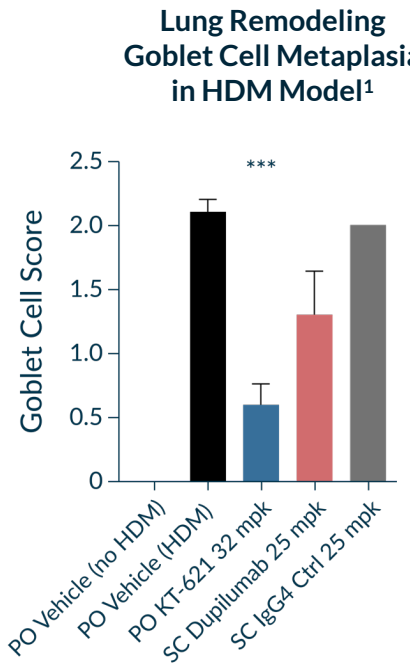

### Robust Degradation in Relevant Cell Types




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




### Excellent Preclinical Activity Comparable or Superior to Dupilumab




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.

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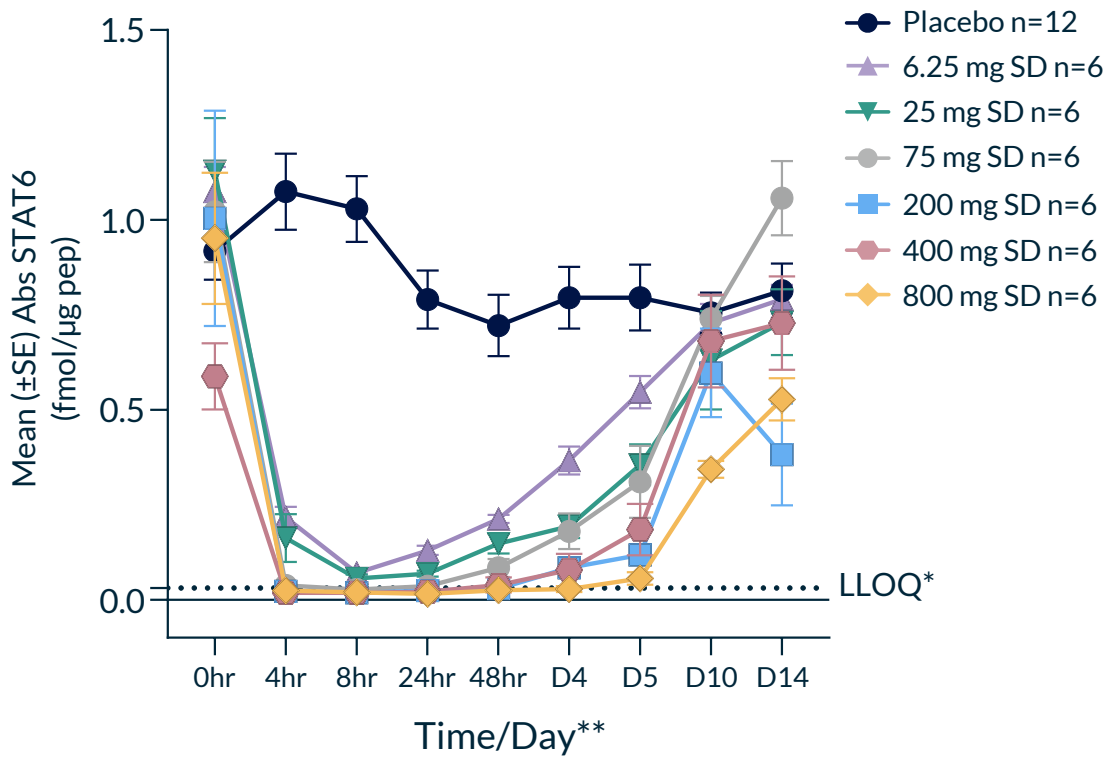
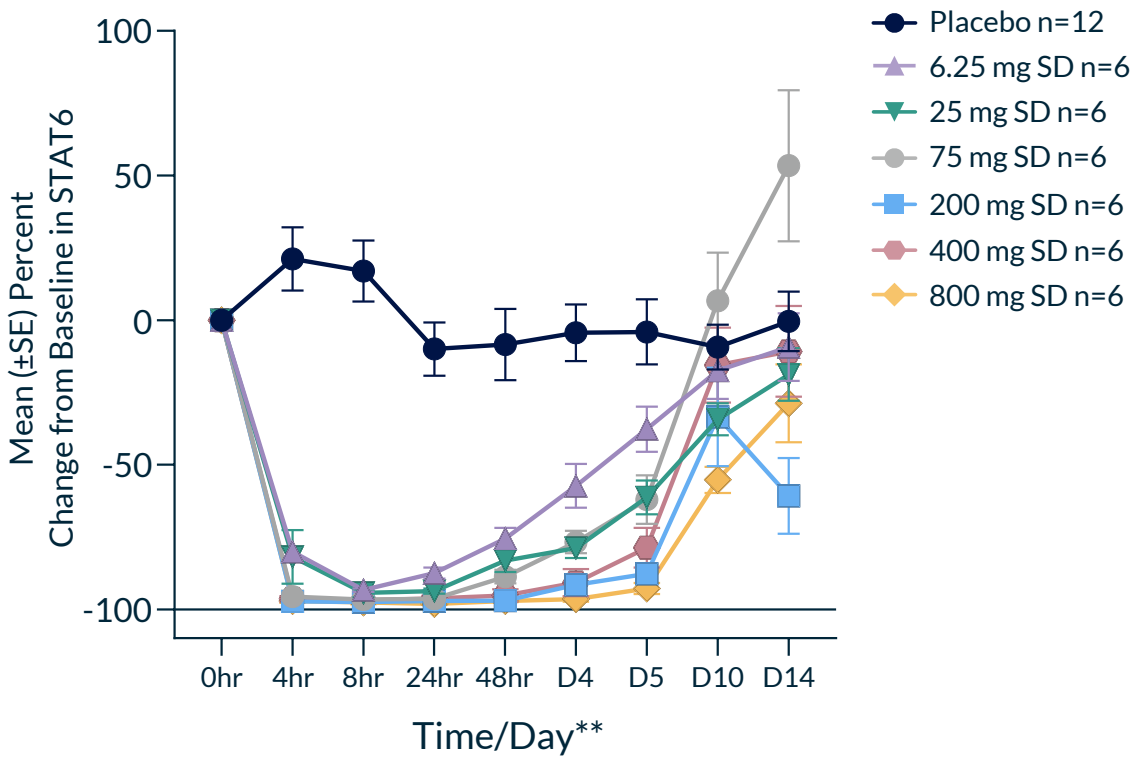
# Single Doses of KT-621 Achieved Rapid, Deep and Prolonged STAT6 Degradation in Blood

## Absolute STAT6 Levels[1]




## Mean Percent Change from Baseline in STAT6




- Maximal degradation seen as quickly as 4 hours after single dose
- Robust degradation maintained as long as 4 days after single dose with recovery by Day 14
- Multiple subjects with STAT6 levels below LLOQ at doses of 75 mg or greater

[1]STAT6 levels measured in isolated PBMC using targeted mass spectrometry;
*Lower Limit of Quantification (LLOQ): Below this level, only estimated value and imputed LLOQ to ½ the value;
**Compound dosed on Day 1; For more information on KT-621's Phase 1 healthy volunteer clinical trial, visit Kymera's website.

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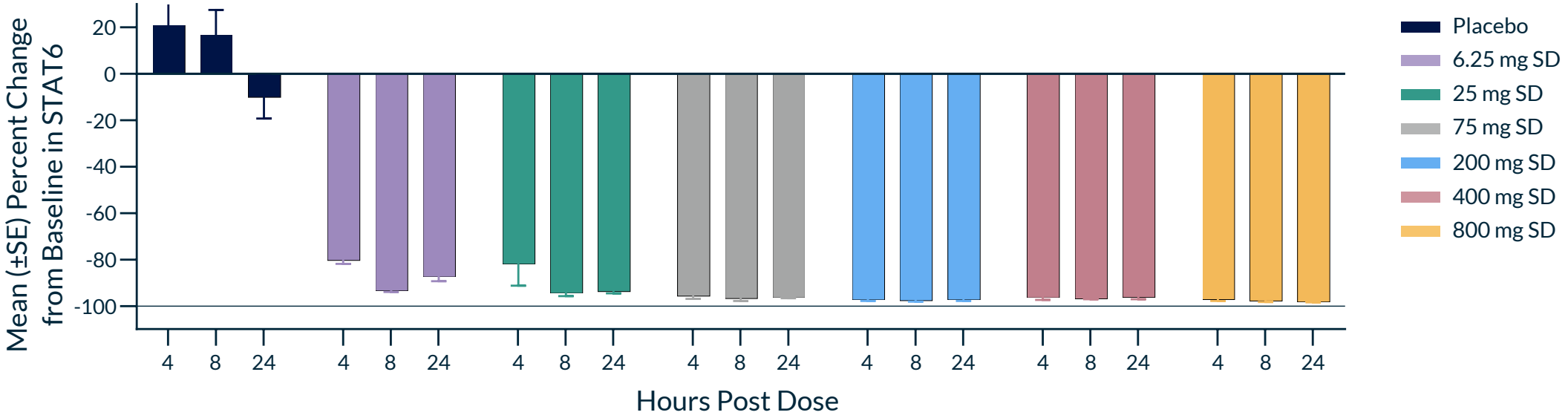
# Single Dose Administration of KT-621 Achieved >90% STAT6 Degradation in Blood Across All Dose Levels

## >95% Mean STAT6 Degradation Achieved at Doses of 75 mg or Greater




| Mean % STAT6 Change | N | 4hr | 8hr | 24hr |
|---|---|---|---|---|
| Placebo | 12 | 21% | 17% | -10% |
| **6.25 mg** | 6 | **-80%** | **-93%** | **-87%** |
| **25 mg** | 6 | **-82%** | **-94%** | **-94%** |
| **75 mg** | 6 | **-96%** | **-97%** | **-96%** |
| **200 mg** | 6 | **-97%** | **-98%** | **-97%** |
| **400 mg** | 6 | **-96%** | **-97%** | **-96%** |
| **800 mg** | 6 | **-97%** | **-98%** | **-98%** |

- At doses ≥75 mg, >95% mean STAT6 degradation was achieved with STAT6 levels below LLOQ in multiple subjects
  - $p < 0.0001$ for all comparisons (KT-621 vs. Placebo)

Note: Not all time points have the same N; For more information on KT-621's Phase 1 healthy volunteer clinical trial, visit Kymera's website.

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# 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.

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# KT-621 BroADen 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 BroADen Phase 1b atopic dermatitis trial, visit clinicaltrials.gov (NCT06945458); BMI: Body Mass Index; SD: Standard Deviation.

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# 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.

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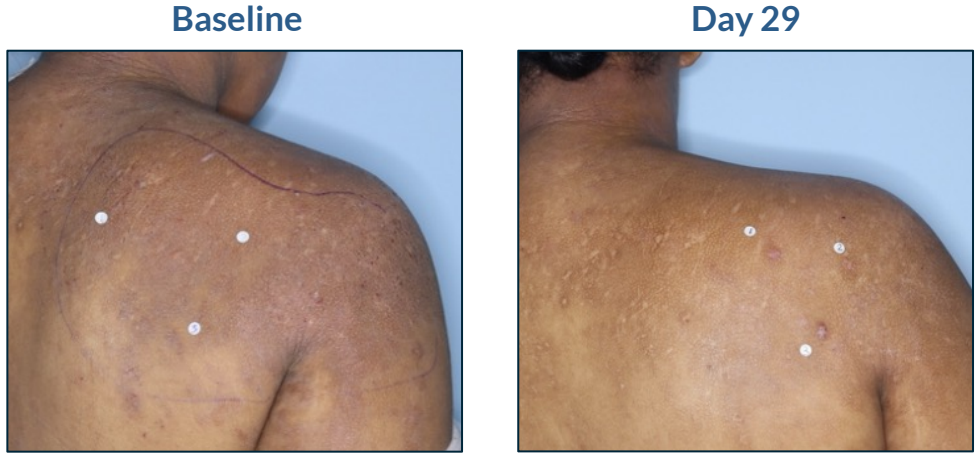
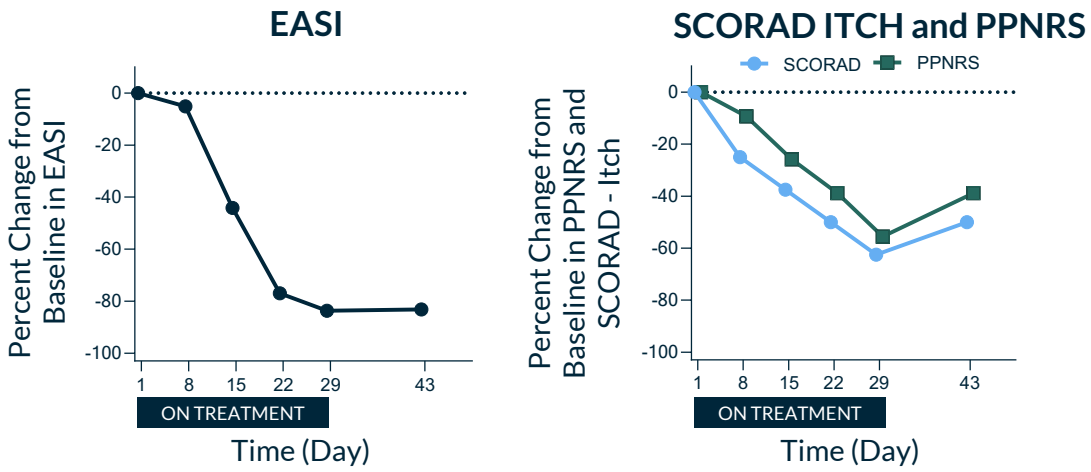
# Example of Clinical and Biomarker Response to KT-621 in BroADen Patient with Severe AD and Prior Dupilumab Treatment

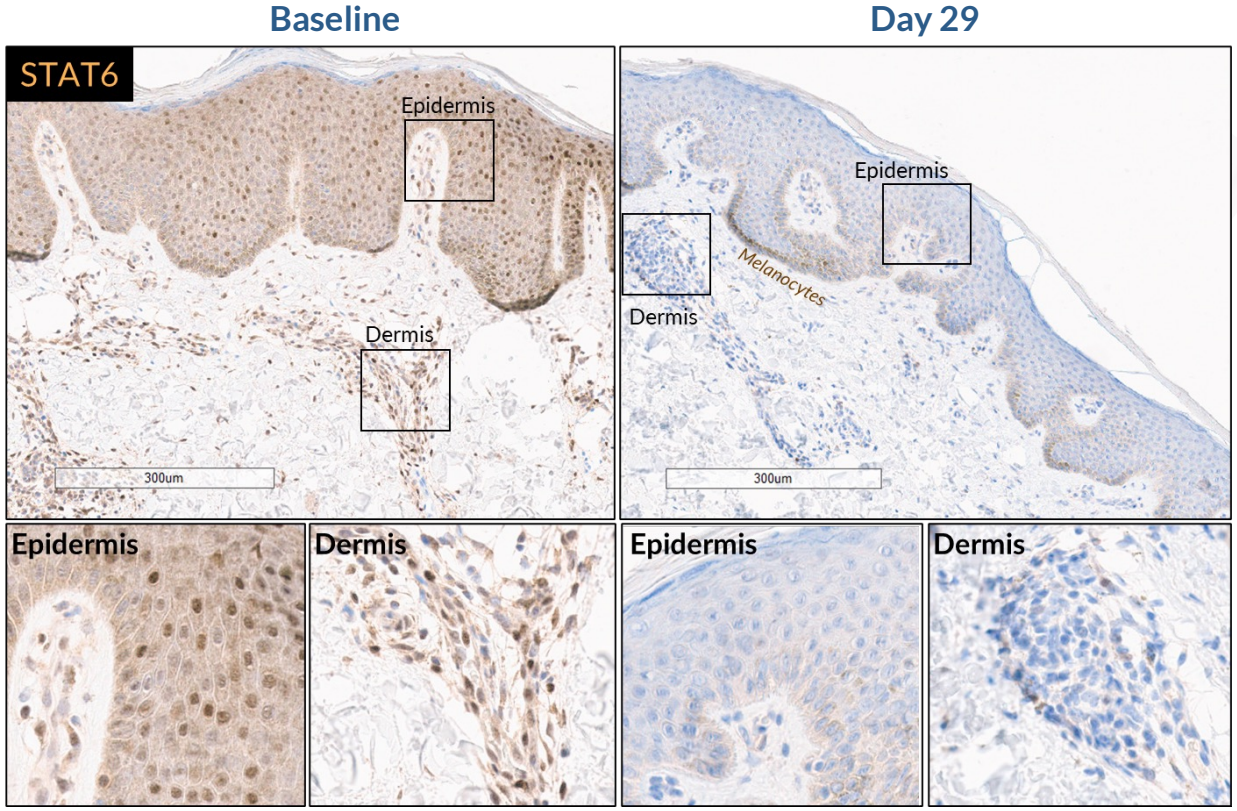
## Pre and Day 29 Photos for EASI-75 Responder




## % Change in EASI and Itch[1]




## STAT6 Staining IHC




## 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.

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# 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.

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# 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.

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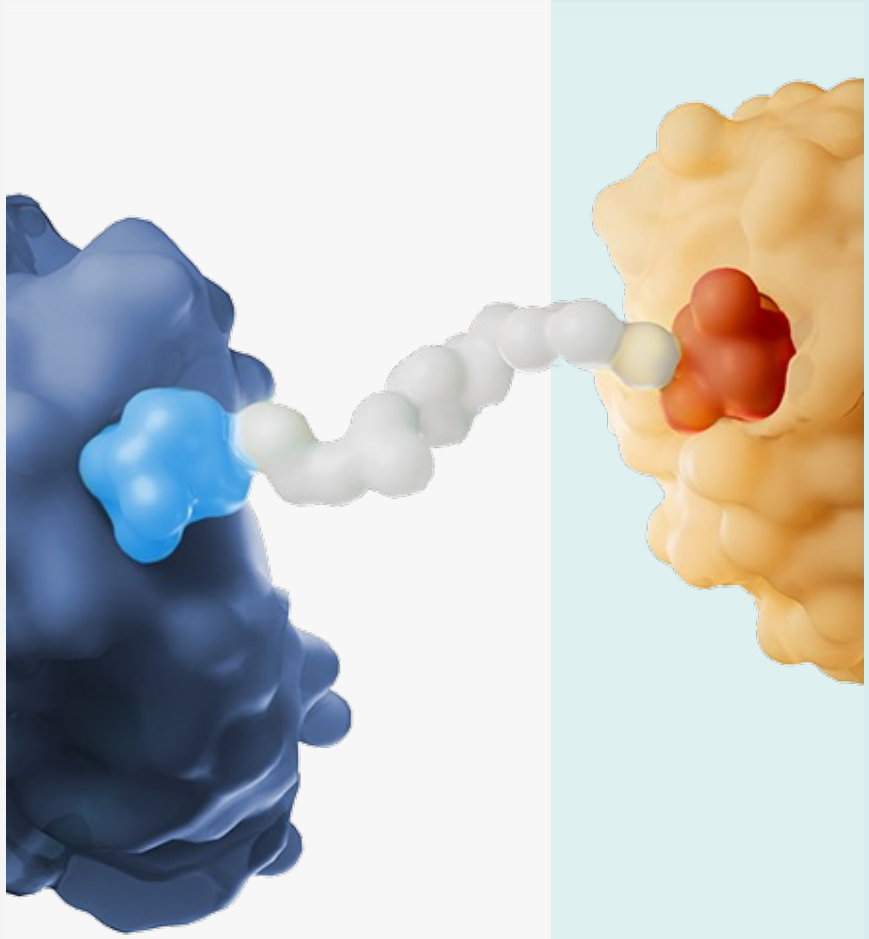

# Appendix: First-in-Class Oral IRF5 Degrader Program

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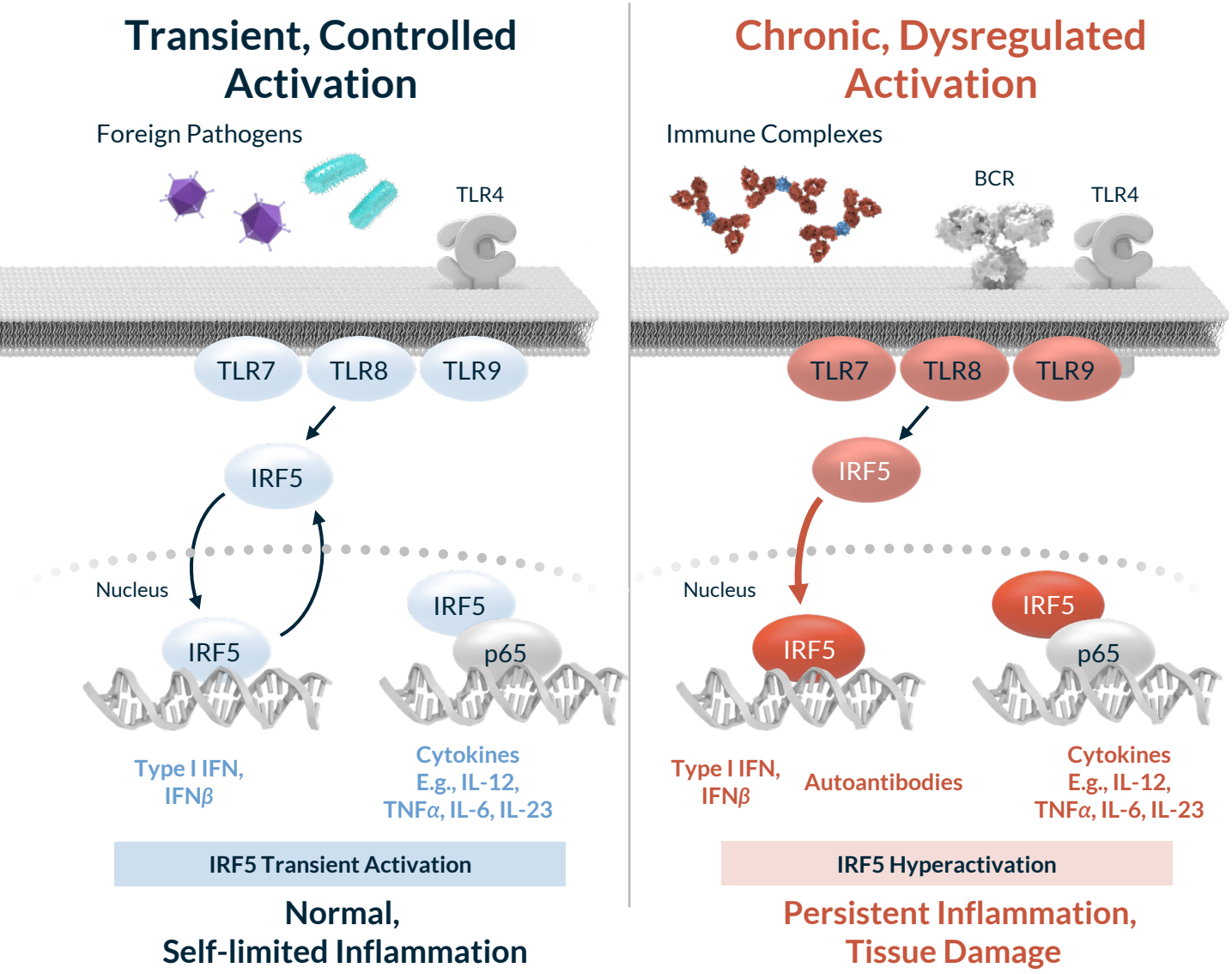
# Central Driver of Immune Dysregulation in Autoimmune Diseases




## 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)**

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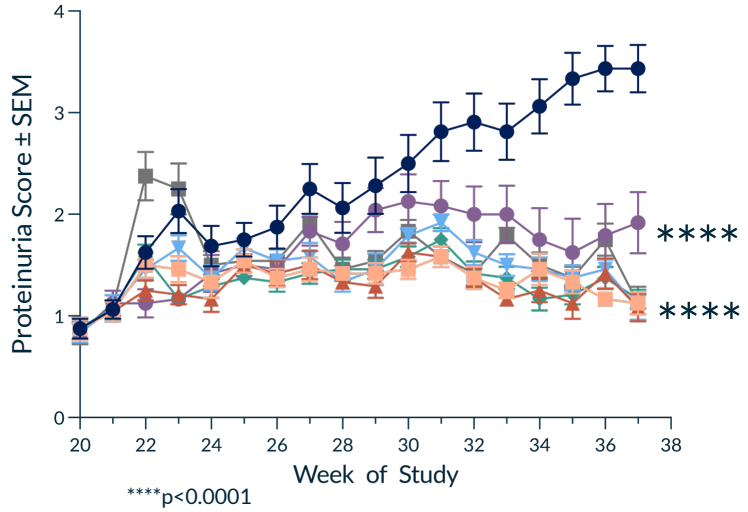
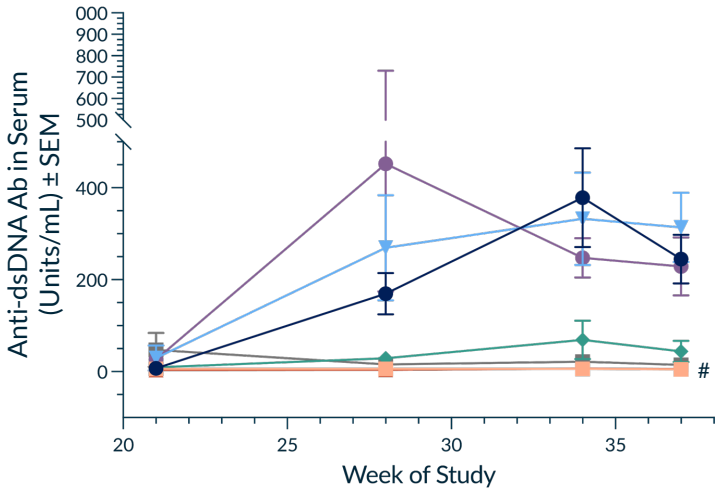
# IRF5 Degradation Demonstrated Generally Better Outcomes than Approved or Clinically Active Drugs in NZBW1 Spontaneous[1] Mouse Lupus Model

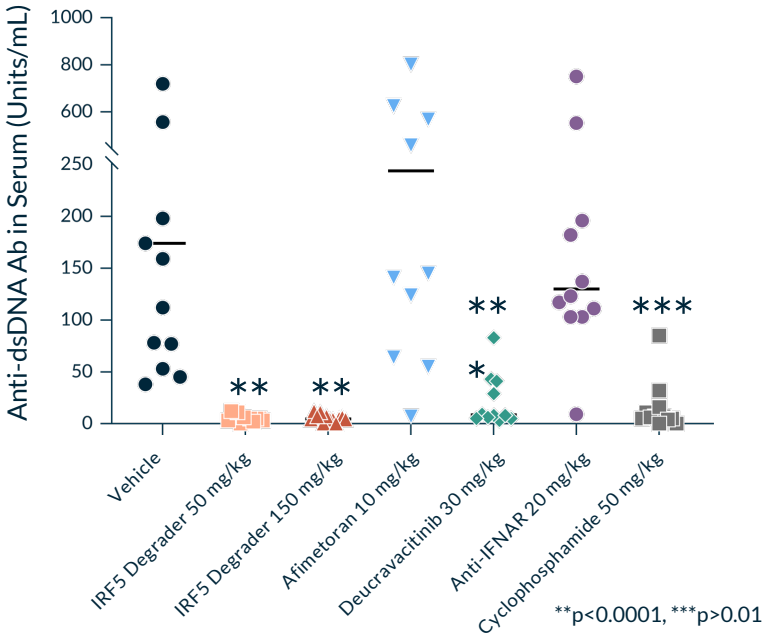
## Decreased Proteinuria




## Early, Sustained and Complete Reduction in Anti-dsDNA Abs




## Superior Activity Reducing Anti-dsDNA at Week 37




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

- **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.

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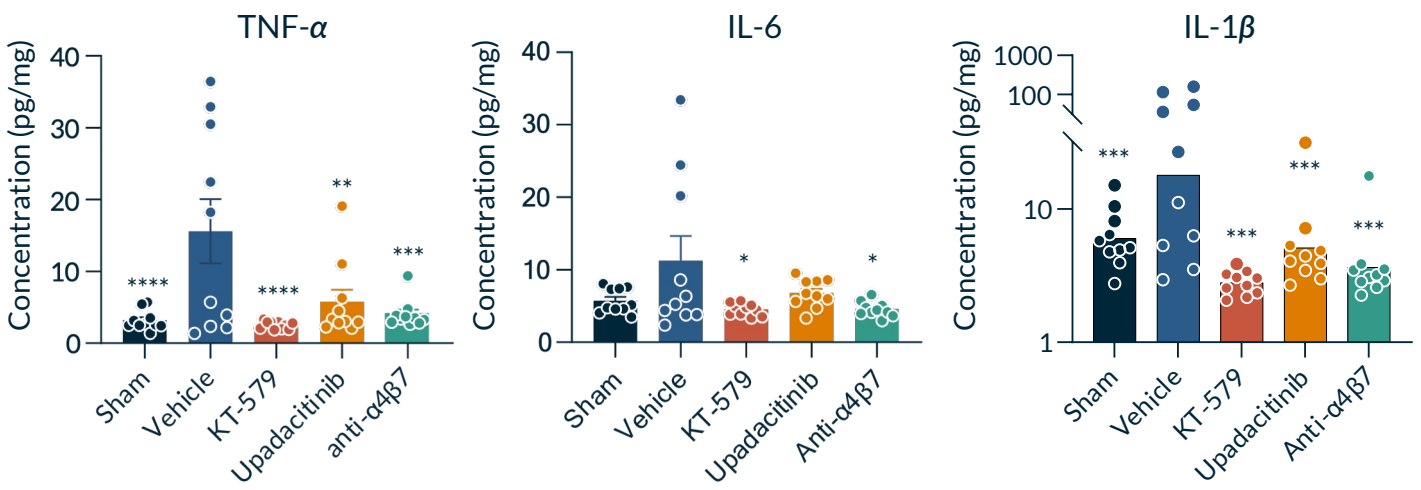
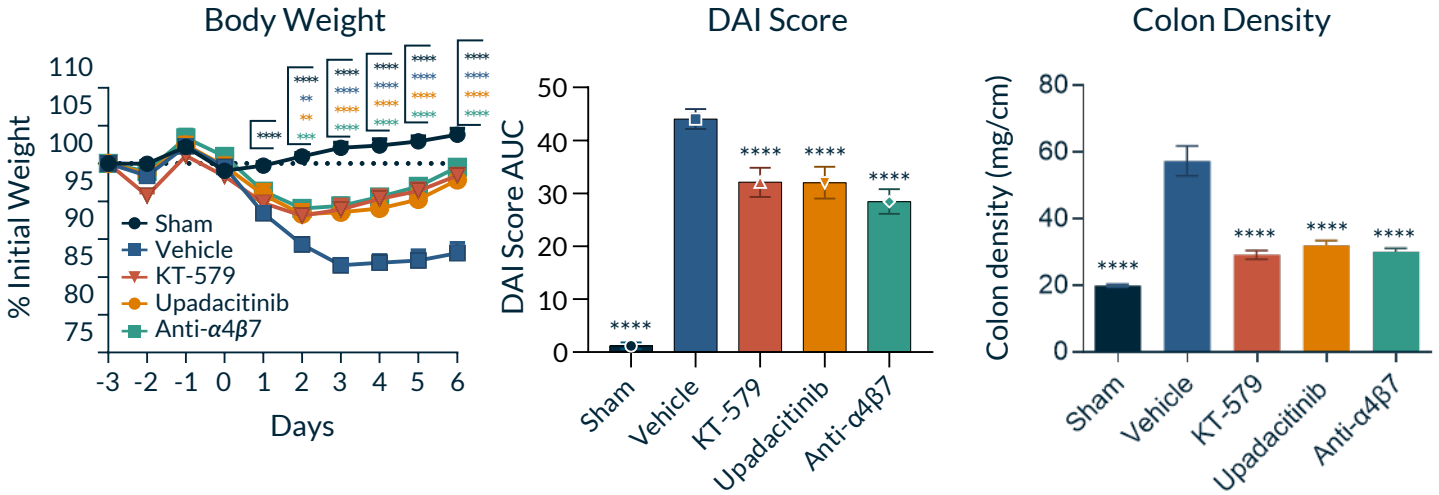
# KT-579 Reduces Disease Progression, Fibrosis, and Intestinal Damage in the Preclinical TNBS Mouse Model of IBD

## KT-579 Reduces Inflammatory Cytokines in the Colon

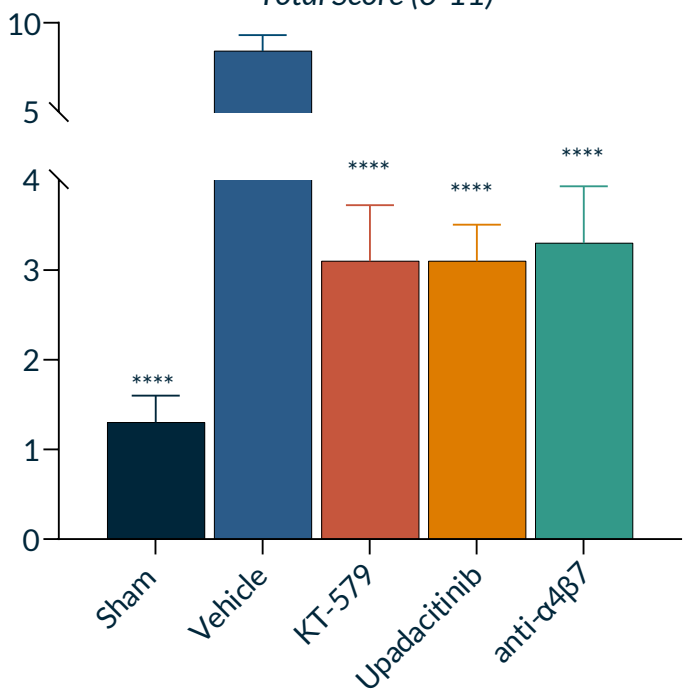



## KT-579 Reduces Disease Activity




## H&E Pathological Scoring

*Total Score (0-11)*



**KT-579 demonstrated disease-modifying activity comparable or superior to approved and clinically active therapies**

KT-579 results in significant reduction of pro-inflammatory cytokine (TNF-α, IL-6, IL-1β) levels in the colon comparable to naïve at terminal day 6 collection. ANOVA, *<0.05, **<0.005, ***<.0001. KT-579 treatment in the TNBS model results in significant reduction of A) body weight loss on Days 2-6 and B) maintenance of colon density (mg/cm) at terminal Day 6 collection. C) Pathological ex vivo analyses of the colons demonstrated KT-579 significantly reduced disease severity resulting in fewer inflammatory infiltrates, improved crypt structure integrity and lack of crypt abscesses. ANOVA, *<0.05, **<0.005, ***<0.001, ****<.0001.

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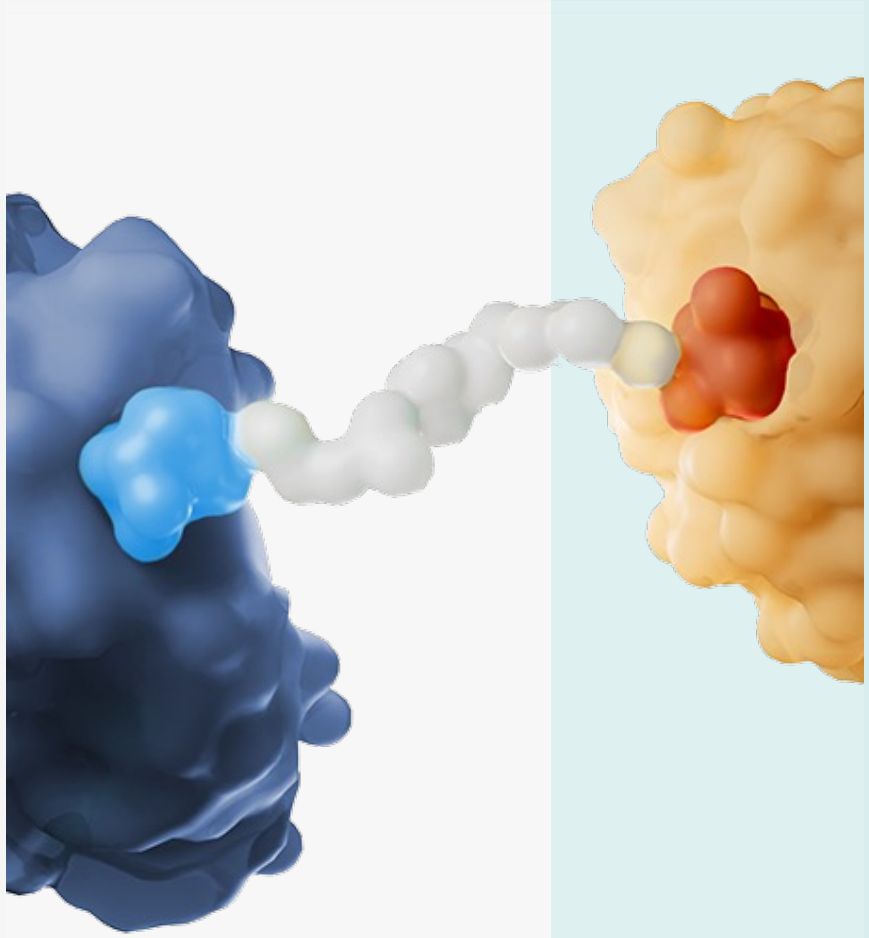

# Appendix: Partnered Programs

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# IRAK4 Biology and Target Rationale

## Target Rationale

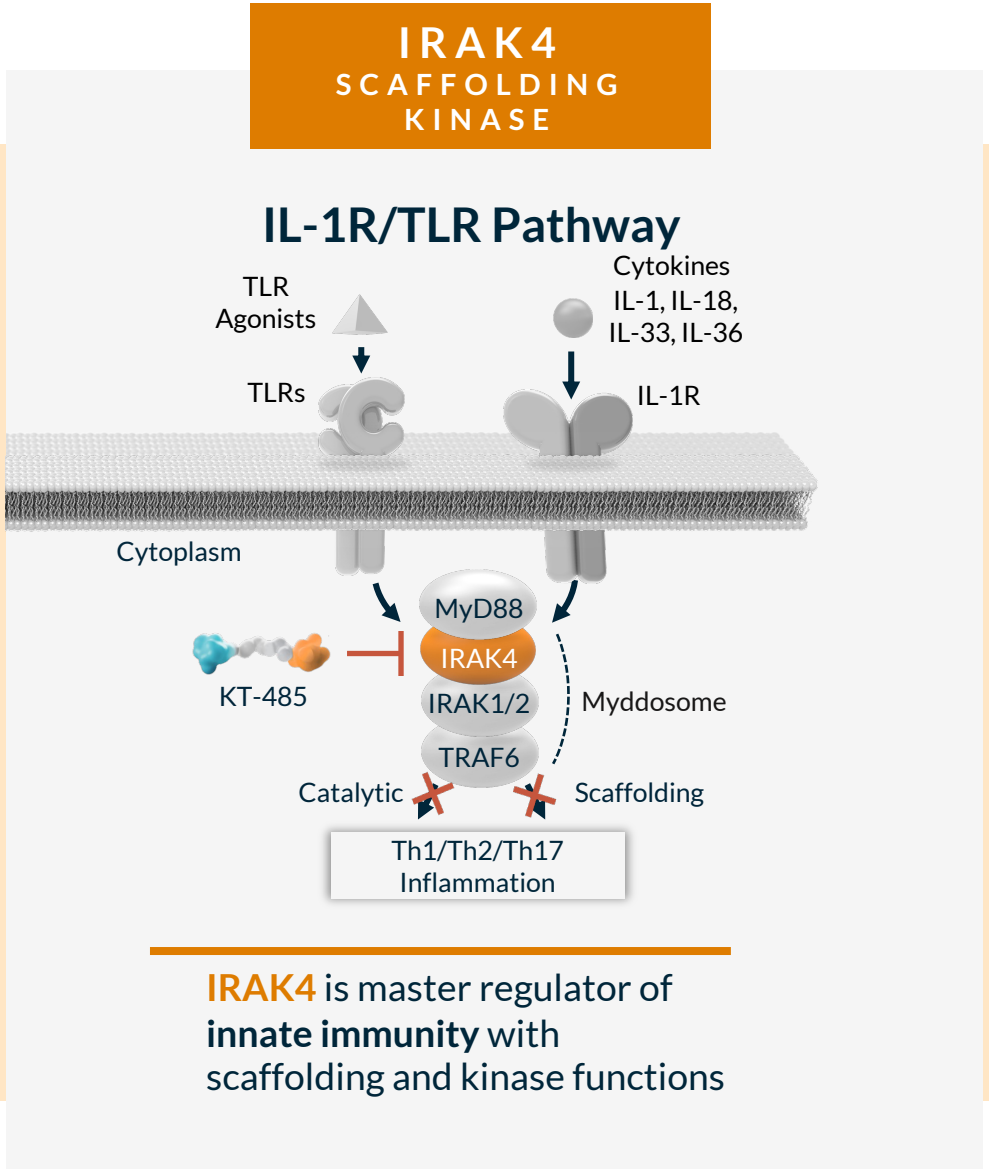
- IRAK4 is an obligate node in IL-1R/TLR signaling, and its degradation is the only approach to fully block the pathway

## 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.

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# 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

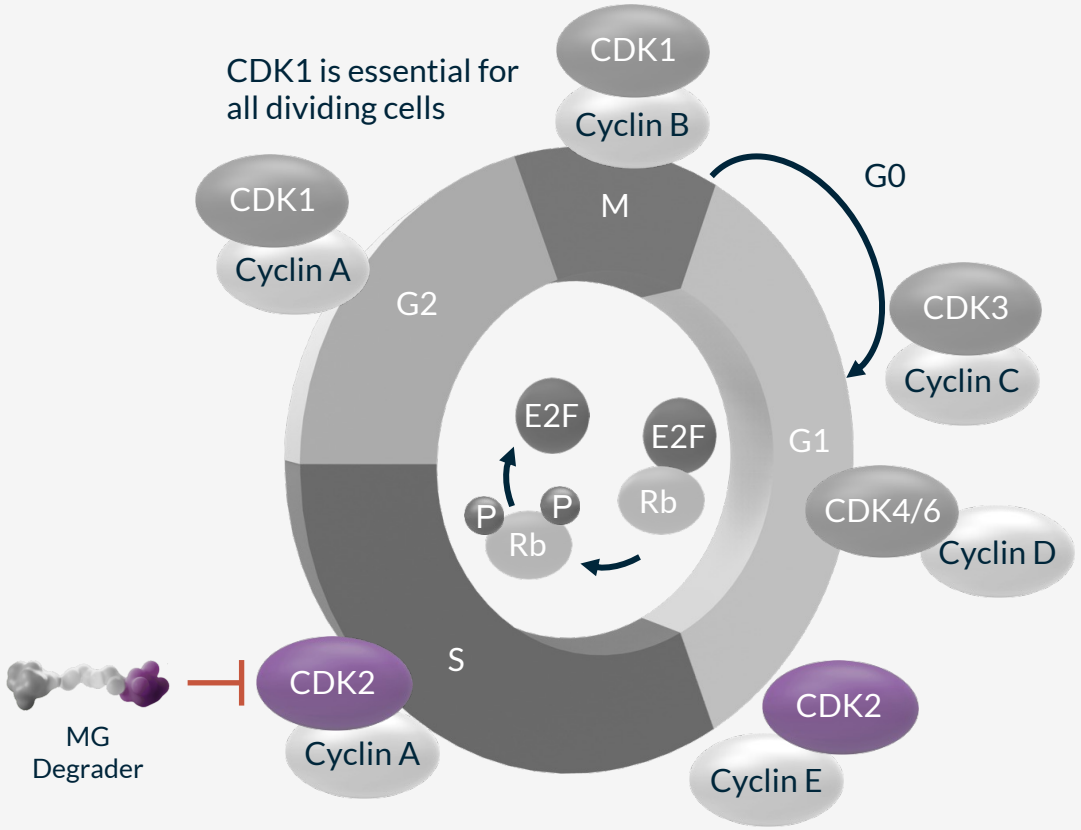
**Genetically Validated**

- 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**

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# 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 |

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# 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 |

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